Carla Sharp
Jennifer L. Tackett
Editors

Handbook of

Borderline Personality Disorder in Children and Adolescents

Foreword by John M. Oldham

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Carla Sharp: For Milla and Christian—for your love, patience, and support.

Jennifer Tackett: For my brother, Josh, who gave me infinity.

Foreword

Great progress has been made in our understanding of borderline personality disorder (BPD), a prevalent condition in clinical treatment settings that is characterized by significant impairment in functioning and by disabling, high-risk patterns of behavior. Persuasive data are emerging that clarify the moderate heritability of BPD, the nature of its most relevant heritable endophenotypes (e.g., emotion dysregulation and impulsivity), and the importance of epigenetics and the principle of bidirectional gene/environment interaction, all of which help us know some of the things to look for as we try to understand a given patient with BPD. Studies utilizing sophisticated brain imaging technologies are revealing patterns of neuropathology and pathophysiology in patients with BPD that may, at least in part, explain specific behaviors. For example, volume abnormalities in the limbic system and deficient connectivity between the limbic system and the prefrontal cortex have been observed in patients with BPD, which could correlate with emotional hyper-reactivity and impaired ability to down-regulate emotions. These findings and many others, such as altered pain processing, neuropeptide abnormalities, and abnormal immune responses, are being steadily reported in patients with BPD, and there is a growing recognition that BPD is fundamentally a brain disorder, conceptually similar to what until recently were referred to as "Axis I" disorders.

Although the personality disorders (PDs) have been in every edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) of the American Psychiatric Association (APA), the terms used to classify them have changed through the years. In 1980, a multiaxial system was introduced in DSM-III, in which the personality disorders (PDs) were classified on Axis II, along with only one other category, "Specific Developmental Disorders," and the rationale for placing these categories on a separate axis was to insure "that consideration is given to the possible presence of disorders that are frequently overlooked. . ." (APA, 1980, p. 23). Intentionally or not, I believe that the decision to locate the PDs on a separate axis from the "Clinical Syndromes" on Axis I, such as affective disorders, schizophrenic disorders, and anxiety disorders, reflected a common view that many Axis I conditions were "biogenic," i.e., heritable episodic conditions, whereas the personality disorders were "psychogenic," largely the result of early developmental misfortune, and they were not thought likely to have favorable responses to treatment. Nonetheless, even if at least partially so motivated, the decision to

place the PDs on Axis II did have the intended result, launching a groundswell of research that has led to important advances in our understanding of these conditions, particularly BPD.

There has been a parallel explosion of progress on the treatment frontier. I had the good fortune to chair the APA Work Group to develop a Practice Guideline for the Treatment of Patients with Borderline Personality Disorder, which was published in 2001 (APA, 2001). The primary, or core, evidence-based treatment recommended for BPD was psychotherapy, combined with symptom-targeted adjunctive pharmacotherapy, as needed. The two specific types of psychotherapy that had then been reported to be effective in patients with BPD, based on randomized controlled trials (RCTs), were dialectical behavior therapy (DBT) (Linehan, 1987) and mentalization-based therapy (MBT) (Bateman & Fonagy, 1999). In more than a decade since then, RCTs have demonstrated the effectiveness of other types of psychotherapy, including transference-focused psychotherapy (TFP) (Clarkin et al., 2007), schema-based therapy (SBT) (Young & Klosko, 2005), cognitive behavioral therapy (CBT) (Davidson, 2006), systems training for emotional predictability and problem solving (STEPPS) (Blum et al., 2008), and a number of others. Furthermore, evidence-based practice guidelines for the treatment of patients with BPD have now been published in The Netherlands (Trimbos Instituut, 2008), the United Kingdom (Kendall et al., 2009), and Australia (NHMRC, 2012), and a large Cochrane review has been published as well (Lieb, Vollm, Rucker, Timmer, & Stoffers, 2010), all of which present similar recommendations that psychotherapy is the primary treatment for BPD. These worldwide analyses of clinical studies of treatment of BPD are enormously encouraging, endorsing hope, and signaling that patients with BPD can benefit from treatment and need not fear that a diagnosis of BPD represents the pronouncement of a "life sentence."

Most of the work summarized above, however, has been carried out in adult patients with BPD. Interestingly, DSM-III stated that PDs "by definition begin in childhood or adolescence and are characteristic of most of adult life" (APA, 1980, p. 306). In 1994, DSM-IV stated that "Personality Disorder categories may be applied to children or adolescents in those relatively unusual instances in which the individual's particular maladaptive personality traits appear to be pervasive, persistent, and unlikely to be limited to a particular developmental stage or an episode of an Axis I disorder. It should be recognized that the traits of a Personality Disorder that appear in childhood will often not persist unchanged into adult life. To diagnose a Personality Disorder in an individual under age 18 years, the features must have been present for at least 1 year" (APA, 1994, p. 631), and this language is essentially unchanged in DSM-5 (APA, 2013, p. 647). As a result, there has been a general assumption, even though it is incorrect, that clinicians were not to diagnose PDs in anyone under the age of 18, which may have contributed, until recently, to the slow pace of research on PDs in adolescents.

Fortunately, the critical importance of prevention, early identification, and early intervention has gained traction for all forms of illness, and mental disorders in particular have been referred to as the chronic diseases of the young. Brain development during childhood and adolescence is complex

under normal circumstances, as the pre-programmed process of change transforms early high levels of neuroplasticity and cellular redundancy into maturing states of greater efficiency, resulting in the emergence of abstract thinking, executive function, cognitive control, and emotion regulation. Successful navigation of these neurodevelopmental waters is enhanced by a stable psychosocial environment and relies especially on the presence of caring and available attachment figures. If a child has a moderate level of heritable risk to develop an illness such as BPD, the presence of stable, caring, and predictable caretaking figures may offset that risk and the PD may not develop. Conversely, however, even a lower level of risk may set the stage for the development of BPD in the context of caretaker inconsistency, neglect, or even frank abuse.

As our understanding of early development has evolved, a broad consensus has emerged that disruptions in attachment and experiences of early life stress can derail these complex and delicate maturational processes in ways that can be disabling and persistent. Adolescence is a critical developmental window when pathological patterns of identity formation and behavior can begin to appear as harbingers of future personality disorders, and recognition of these patterns is the key to protective early intervention.

A great deal of progress has been made in our understanding of emerging patterns of BPD during adolescence. This volume, the Handbook of Borderline Personality Disorder in Children and Adolescents, is an invaluably important compendium, contributing a wealth of new information. Carla Sharp and Jennifer Tackett have assembled a remarkable cast of authors (they among them) to cover with breadth and depth the latest conceptual thinking, clinical work, and research findings, richly filling in what have been large gaps in our knowledge about the biopsychosocial scaffolding that can lead to the emergence of BPD. A comprehensive resource, the Handbook, presents sophisticated analyses of trait models of borderline pathology; reviews of neurobiological, genetic, and social-cognitive (e.g., "hypermentalizing") factors in borderline patients; descriptions of the early developmental course of emerging BPD; and evidence-based treatment recommendations.

The final section of the Handbook includes a description of the Alternative Model for the Personality Disorders, published in Section III ("Emerging Measures and Methods") of DSM-5. As a member of the Work Group on Personality and Personality Disorders for DSM-5, I was involved in the development of this Alternative Model (AM), which re-frames the personality disorders, including BPD, as moderate or greater impairment in personality functioning (defined as impairment in a sense of self [identity and self-direction] and impairment in interpersonal relationships [empathy and intimacy]), along with the presence of pathological personality traits. In the case of BPD, there is no stipulation regarding age of onset at age 18. In addition to the presence of moderate or greater impairment in personality functioning, BPD is defined by the presence of four or more of seven pathological personality traits. Four of these are in the trait domain of Negative Affectivity (emotional lability, anxiousness, separation insecurity, and depressivity), two are in the trait domain of Disinhibition (impulsivity

and risk-taking), and one is in the trait domain of Hostility (antagonism). This new trait-based model for BPD aligns well with a growing literature that recognizes the utility and conceptual advantage of a dimensional approach to BPD, reflected in a great deal of the content of this Handbook. The field is indebted to Sharp and Tackett for compiling this outstanding volume, which will serve as an invaluable resource to researchers and clinicians, as the importance of the emergence of BPD during adolescence becomes increasingly recognized.

Houston, TX John M. Oldham

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Jennifer L. Tackett received her Ph.D. from the University of Minnesota in 2006 and holds faculty appointments at the Universities of Houston and Toronto. She is an active researcher in the areas of child personality, personality disorder, and externalizing psychopathology. Dr. Tackett served as a developmental consultant to the Personality and Personality Disorders Work Group for DSM-5. She has over 60 publications, co-edited *Personality and Psychopathology* (The Guilford Press, 2006), and received early career awards from the Society for Research in Psychopathology, the Society for Personality Assessment, and the Ontario Ministry of Research and Innovation. Dr. Tackett has extensive editorial experience and currently serves as the Associate Editor at *Assessment*, *Journal of Research in Personality*, *Journal of Personality Disorders*, and *Journal of Psychopathology and Behavioral Assessment*.

Part I

Introduction

Introduction: An Idea Whose Time Has Come

1

Carla Sharp and Jennifer L. Tackett

Five years ago, Chanen and McCutcheon (2008) provocatively titled a paper "Personality disorder in adolescence: The diagnosis that dare not speak its name" to describe a 16-year-old female with Borderline Personality Disorder (BPD). This title reflected the fact that—for many years—diagnosing BPD in youth has encountered a great deal of reluctance. This is particularly problematic, given the long-standing general consensus that BPD has its roots in childhood and adolescence (American Psychiatric Association, 2000). Explanations for this reluctance have included the notion that a BPD label connotes severity and nonmalleability, which may negatively affect a developing child's self-concept or bias others' perceptions of the child (Kernberg, Weiner, & Bardenstein, 2000). Some have questioned diagnosing BPD prior to the onset of puberty and prior to the completion of identity formation (Shapiro, 1990), based on the idea that personality may lack cohesiveness and stability prior to age 18 (Crick, Murray-Close, & Woods, 2005). And others have emphasized the problem of distinguishing borderline features from the normal developmental trajectory of adolescence (Meijer, Goedhart, & Treffers, 1998; Miller, Muehlenkamp, & Jacobson, 2008). These questions are all valid and over the last 15 years, we have seen an unprecedented increase in research activity aimed at evaluating these and other hypotheses related to BPD in youth. In this volume, we have brought together many researchers and clinicians who have spearheaded this transformation in the field, which, in turn, now enables us to characterize juvenile BPD not as a diagnosis that dare not speak its name, but as an idea whose time has come.

That the time was right to bring together the stellar group of researchers and clinicians who have contributed to this volume can be quantified by the results of a literature search of empirical studies via *PSYCInfo* and *Web of Science* with search terms of (1) Borderline Personality Disorder, Pathology or BPD and (2) Adolescent(s), Child(ren), Youth(s), Juvenile(s), Girl(s), or Boys(s) spanning 1990–2013. The results of this literature search are visually presented in Fig. 1.1 and demonstrate a five-fold increase in empirical studies examining BPD in youth in 2013, as compared to 1993.

It also shows that any attempt at publishing an edited volume even 5 years ago may not have yielded the strong empirical contributions that we were able to obtain at this point in time. Some of the early momentum in the field was already observable in 2004 with a special issue of review articles in *The Canadian Journal of Child and Adolescent Psychiatry* (Guile & Greenfield, 2004), followed by a special issue of largely theoretical papers of developmental models of BPD in 2005 in *Development and Psychopathology* (Lenzenweger & Cicchetti, 2005). In the editorial to their special issue, Lenzenweger and Cicchetti

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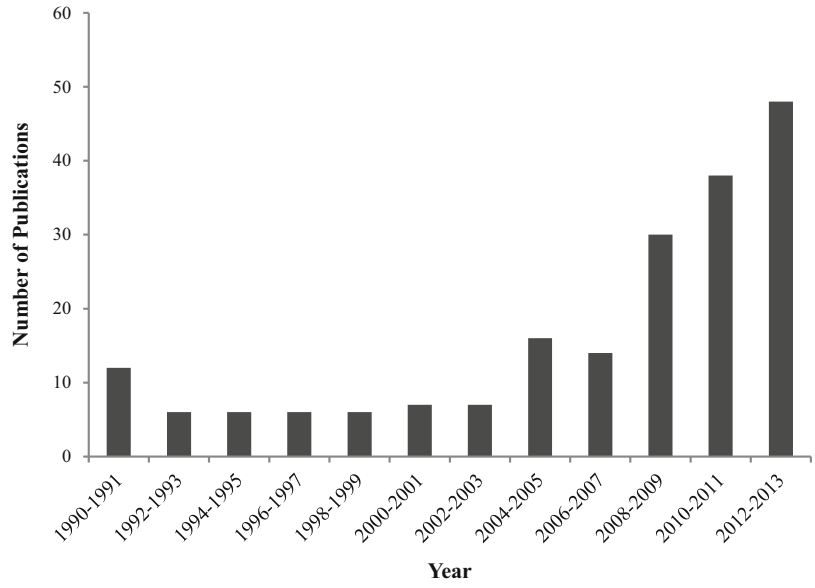

Fig. 1.1 Published research articles on BPD in Youth

Literature searches conducted via *PSYCInfo* and *Web of Science* with search terms of Borderline Personality Disorder, Pathology or BPD and Adolescent(s), Child(ren), Youth(s), Juvenile(s), Girl(s), or Boys(s). Search results yielded 196 published empirical articles from 1990 to 2013. Thanks to Allison Kalpakci who created this graph.

(2005) emphasized that the next generation of BPD research should take place within a developmental context. This work has begun and has spawned renewed energy in the field—for example, a recent special issue on BPD in youth in *Journal of Abnormal Child Psychology* (Stepp, 2012). It has also made possible this volume, which, for the first time, brings together the empirical research that sheds light on important remaining topics in the field that are best addressed through a developmental lens. These are: (1) the phenomenology of BPD across the lifespan, including its underlying structure and natural organization; (2) empirical support for and integration of long-standing theoretical approaches regarding the ontogeny of BPD; (3) the life-course trajectory of BPD; (4) endophenotypes that may underlie behavioral variation at the phenotypic level; and (4) clinical approaches to the early treatment of BPD. These topics have guided the organization of this volume. We briefly describe them in this chapter, which serves as the first of two introductory chapters of Part I (Introduction). Chapter 2 by Joel Paris provides an overview of broad research strategies employed over the years to examine juvenile BPD—a theme that John Gunderson returns to in the final chapter of the volume.

Part II of the current volume (Issues in Conceptualization and Assessment) deals with questions of phenomenology, natural organization, and assessment of youth BPD. This section exemplifies in many ways the recent transformation represented in Section 3 of DSM-5, namely, a move towards a trait-based approach to the conceptualization of personality pathology. In Chap. 3, Andrea Fossati sets the stage for this discussion by providing a rich overview of the phenomenology and validity of the borderline construct in a developmental context. Next, in Chap. 4, Jared Michonski tackles the topic of the underlying factor structure of DSM criteria in youth BPD and suggests that a unidimensional model appears to offer the most parsimonious conceptualization of BPD in both adult and youth samples.

Chapters 5–7 each cover modern approaches to the assessment of borderline pathology in

youth as operationalized by three commonly used measures: the Personality Assessment Inventory for Adolescents (Morey & Meyer, Chap. 5), the Minnesota Multiphasic Personality Inventory—Adolescent version (Martin Sellbom & Matthew Jarrett, Chap. 6), and the Dimensional Personality Symptom Itempool (Barbara De Clercq, Mieke Decuyper & Elien De Caluwé, Chap. 7). Part I concludes with a chapter by Jennifer Tackett and Shauna Kushner discussing the relevance of normal personality trait perspectives for understanding the development of youth BPD. Together, the chapters in Part I underscore the immense potential that dimensional trait-based approaches hold for the study of borderline features in youth and highlight cutting-edge perspectives on conceptualization and assessment of the youth BPD construct. The categorical approach embodied in DSM-IV and DSM-5 focuses narrowly on clinically relevant symptoms, but it does not allow for the study of the entire range of borderline symptoms and related characteristics, which allows for the identification of not only those individuals who are demonstrating "clinically significant" levels of symptomatology but also those who may be considered "at risk" (Crick et al., 2005). The inclusion of at-risk individuals is especially important for the study of the etiology of BPD in order to better understand not only those who go on to meet full diagnostic criteria for BPD but also those youth with high-risk profiles who ultimately follow more adaptive trajectories. Such etiological processes, which interact in complex reciprocal ways, are discussed next in Part III (Etiology and Core Components).

Part III includes empirically based, integrative theoretical approaches regarding the emergence and development of BPD as well as critical reviews of endophenotypes that may underlie behavioral variation at the phenotypic level. Part III begins with Chap. 9 by Marianne Goodman, Mercedes Perez-Rodrigues, and Larry Siever, who provide a theoretically agnostic overview of the neurobiological basis of BPD, including recent findings in adolescents. Chapter 10 by Marina Bornovalova, Brittany Jourdan-Arthur, and Anahi Collado-Rodriquez reviews behavior genetic studies of youth BPD, which supports the familial transmissions of BPD and begins to characterize genetic and environmental effects on BPD over time. Together, Chaps. 9 and 10 provide evidence for what John Oldham refers to in the Foreword as the "biogenic" nature of youth BPD.

The rest of the chapters in Part III cover and extend the two most prominent etiological models of BPD. Chapters 11–14 all extend Linehan's (1993) influential developmental model of BPD. In Chap. 11, Sheila Crowell, Erin Kaufman, and Theodore Beauchaine extend Linehan's model by providing a more central role for the interaction of trait impulsivity with family-level risk factors to constitute the dysregulation of emotion that lies at the core of BPD across the lifespan. In Chap. 12, Ed Selby, Amy Kranzler, and Emily Panza present their Emotional Cascade Model which also builds on Linehan's biosocial model by delineating in precise detail how people with BPD may experience frequent and intense elevations in negative emotion, which may lead to subsequent behavioral dysregulation, as a result of emotional cascades through ruminative processes. In Chap. 13, Alexis Matusiewicz, Grace Weaverling, and Carl Lejeuz put some of these ideas to the test in their review of the empirical evidence in support of affective dysfunction and emotion regulation problems in adolescents with BPD. And in Chap. 14, Kim Gratz, Katherine Dixon-Gordon, and Matthew Tull apply Linehan's theory and Crowell et al.'s (Crowell, Beauchaine, & Linehan, 2009) extension thereof to the problem of self-injurious behavior in adolescents with BPD.

Part III concludes with Chap. 15 in which Carla Sharp reviews the literature in support of another prominent developmental model of BPD, namely Fonagy's mentalization-based model of BPD (Fonagy, 1991; Fonagy & Luyten, 2009). She extends this model by presenting a hypermentalizing theory of BPD which suggests

that mentalizing dysfunction is present in BPD, but not in the form of failure or suppression as previously thought, but in the form of excess (hypermentalizing). While this may still be seen as an ultimate failure of mentalizing, it does provide a more parsimonious account of mentalizing dysfunction in BPD that can be readily accessed.

In Part IV, we grapple with problems of the life-course trajectory of BPD and shed further light on etiological models of BPD by considering the developmental course and psychosocial correlates of BPD. Part IV begins with Chapter 16 by Amanda Venta, Kathrin Herzhoff, Patricia Cohen, and Carla Sharp, who review prospective studies on the developmental course of BPD. This sets the stage for Chap. 17, in which Stephanie Stepp, Diana Whalen, and Sarah Pedersen describe longitudinal work which has led to a developmental formulation of BPD through an externalizing pathway characterized by an interaction between emotional impulsivity and deleterious family environments. The early family environment is explored further in Chap. 18 by Mary Zanarini and Michelle Wedig, who review prospective and retrospective studies on early childhood adversity and the development of BPD, thereby supporting their tripartite model of the etiology of BPD (Zanarini & Frankenburg, 1994). This model states that three factors—one environmental in nature, one constitutional in nature, and one representing the interaction of the other two or a triggering factor—are necessary (but perhaps not sufficient) for the development of BPD. The focus on the early family environment continues in Chap. 19, in which Jenny Macfie and Jennifer Strimpfel review empirical research on parenting and BPD—especially in at-risk children by virtue of being the biological offspring of mothers with BPD. Their research informs a developmentally sensitive model of parenting and the development of BPD as a function of parent temperament, child temperament, the environmental context, and the representations of the parent's own childhood experiences. Part IV concludes with Chap. 20 by Chris Hopwood and Aaron Pincus which informs the preceding chapters on the child's social environment and which also holds promise for integration into social-cognitive models of BPD as reviewed in Chap. 15. Specifically, they show how integrative interpersonal theory (Pincus, 2005) can be useful for conceptualizing borderline personality development and present empirical evidence in support of their model.

With Part V, we complete the translational spectrum by focusing on treatment. Readers may be surprised to find that incredible advances have occurred in the development of treatment approaches for youth with borderline traits. In Part V, these approaches are discussed and vignettes with real-life treatment session extracts are provided to bring each treatment approach to life. The first three chapters in Part V cover treatments that are informed largely by psychodynamic approaches, while the last three chapters cover treatments informed by cognitive-behaviorally based approaches—although all current treatments reflect cross-fertilization that has occurred in recent years to advance more integrative approaches to the treatment of personality pathology. Chapter 21 by Peter Fonagy, Trudie Rossouw, Carla Sharp, Anthony Bateman, Liz Allison, and Clare Farrar describes the principles and structure of *mentalization-based treatment for adolescents (MBT-A)*, which incorporates monthly sessions of *mentalization-based treatment for families* (MBT-F). They also discuss the particular relevance of the mentalization construct for understanding self-harm in adolescence, and describe the results of a recent pragmatic randomized superiority trial comparing MBT-A with treatment as usual for adolescents with self-harm. Chapter 22 by Lina Normandin, Karin Ensink, Frank Yeomanns, and Otto Kernberg report on the adaptation of adult *Transference Focused Psychotherapy to personality disorders in adolescents* (TFP-A). TFP is organized around the principle that disturbances in the organization of the self are central to the development of BPD and these disturbances will manifest in the core dimensions of the self, involving negative effect, self-regulation, motivation, and reward, as well as control systems involving attention and reflectiveness, interpersonal interaction, and affiliation. This chapter is followed by Chap. 23 in which

Andrew Chanen, Louise McCutcheon, and Ian Kerr describe their *Helping Young People Early* (HYPE) program, a comprehensive and integrated indicated prevention and early intervention program for youth (15–25 years of age). *HYPE* includes both a service model and an individual therapy and incorporates the principles of cognitive analytic therapy (CAT) into both components. Empirical data in support of HYPE is provided, and an important paradigm shift is recommended towards a model of early intervention for BPD.

The next three chapters describe approaches to the treatment of youth BPD from a cognitive-behavior perspective—specifically, *Dialectical Behavior Therapy* (DBT). In Chap. 24, Alec Miller, Mary Carnesale, and Elizabeth Courtney describe the modifications to standard DBT that were made to provide treatment that is developmentally appropriate and relevant for adolescents and their families. They also report on the empirical findings in support of the downward extension of DBT to youngsters. Next, in Chap. 25, Blaise Aguirre, Janna Hobbs, and Michael Hollander describe the adaption of DBT to the family therapy context, and in Chap. 26, Renee Harvey, Nancee Blum, Donald Black, Jo Burgess, and Paula Henley-Cragg describe their *Systems Training for Emotional Predictability and Problem Solving* (STEPPS) program. While STEPPS has been used for adults in a variety of settings with a high degree of success, this chapter describes an adaptation of the program for an adolescent population and provides feedback from a pilot group recently completed in the United Kingdom.

Part VI concludes the edited volume with three important chapters regarding the broader context of the scientific and clinical endeavors reflected in this volume and charts future research. In Chap. 27, Merav Silverman and Bob Krueger describe and reflect on the main challenges to DSM-IV personality disorders and the process of developing the personality disorder sections in DSM-5. They pay special attention to the ways that these changes may impact research and treatment of BPD and BPD symptoms in children and adolescents and emphasize that the fundamental goal of these proposed changes is to provide a more precise method of describing and diagnosing BPD in order to improve the quality of research and to aid clinicians in their diagnosis and treatment of the disorder. Tyrer summarizes the framework used to guide revision of the personality disorders in ICD-11 and the decision to shift from a focus on diagnostic categories to an overall continuum of severity. He further discusses the implications of these changes for borderline PD, in particular.

Finally, in Chap. 29, John Gunderson provides an overview of the field as a context for the current volume. He outlines three important goals for future research: first, to uncover the implications of BPD's heritability; second, the identification of risk markers; and third, the potential to customize home environments that might derail BPD's onset. To achieve these goals, Gunderson calls for large scale prospective studies of children at risk for developing BPD that tests potentially preventive interventions.

On reflection, and in line with Gunderson's closing chapter, the collection of chapters in this volume has underscored for us the value of the developmental psychopathology approach. In many ways, this book testifies to the incredible tools that developmental psychopathology has to offer when trying to understand and examine a complex behavioral phenotype like BPD. This framework breaks down intellectual barriers between traditional disciplines by integrating clinical, developmental psychology, child/adolescent psychiatry, genetics, neurology, public health, and philosophy of science into a multidisciplinary effort. Such an integrative framework is critical to organize and propel future efforts, as exemplified in this volume. We urge researchers, clinicians, and students to be guided by the developmental psychopathology principles in tackling the problems associated with BPD (and other personality pathology). This will require longitudinal studies of large samples of children across the full latent trait of borderline pathology, alongside more selected samples of children with severe dysfunction. It will require studies across multiple levels of analyses that aim to integrate genetic and neurobiological findings with findings at the level of the behavioral phenotype. It will also require experimental work with novel approaches to

characterizing parent–child and peer interactions, and it will require complex and sophisticated analytic approaches to understand change across time, and to understand the underlying structure of BPD across different developmental epochs. This agenda can be realized only through collaborative strategizing.

We hope that this volume provides researchers, clinicians, and their students with the tools they need to build on the work described here. We are incredibly grateful to the authors who have generously and enthusiastically agreed to contribute to this volume: from the giants in the field on whose shoulders we all stand, to current leaders in the field, to the upcoming young stars. We say thank you for each and every patient and their family who has shared with us their most private thoughts and feelings—at the Adolescent Treatment Program of the Menninger Clinic and the University of Houston. We are also indebted to our students who inspire and teach us every day, and our families for their patience, love, and support.

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A History of Research on Borderline Personality Disorder in Childhood and Adolescence

2

Joel Paris

Introduction

Borderline personality disorder (BPD) was first described 75 years ago (Stern, 1938), but only became accepted in the classification of mental disorders decades later (American Psychiatric Association, 1980); but it was at that point that research on the disorder took off. Formal investigation of BPD also led to a reformulation of its nature, moving from a condition lying on a "border" between neurosis and psychosis to a personality disorder rooted in underlying traits but associated with prominent symptoms. Thus BPD emerges from multiple risk factors (Crowell, Beauchaine, & Linehan, 2009; Paris, 2007). There is no single explanation for its cause, and risks are not the same in all patients. The pathways to BPD demonstrate equifinality, with different pathways capable of leading to the same outcome (Cicchetti & Rogosch, 2002).

One of the most important findings from research has been that BPD begins in adolescence and usually remits by middle age (Paris, 2008). Consistent childhood precursors, such as those established for antisocial personality, have not been identified, but, as we will see, there is evidence that both internalizing and externalizing symptoms in childhood precede the development of overt BPD. Adolescent BPD is not different from the clinical picture in young adults and needs to be identified and treated in much the same way.

BPD Begins in Adolescence

BPD emerges from complex and interactive biological and psychosocial risk factors (Crowell et al., 2009; Paris, 2007). There is no single explanation for its cause, and risks do not seem to be the same in all patients. The pathways to BPD demonstrate equifinality, with different pathways capable of leading to the same outcome (Cicchetti & Rogosch, 2002).

While BPD is a common clinical presentation in young adult populations, its symptoms usually remit by age 40 (Gunderson et al., 2011; Paris, 2008; Zanarini, Frankenburg, Reich, & Fitzmaurice, 2012). In this respect, its course resembles that of many other mental disorders, such as schizophrenia and substance abuse, although like these other disorders, residual psychosocial problems can continue even when the most prominent symptoms remit.

Research on BPD has the aim of recognizing the disorder and diagnosing it as early as possible. BPD symptoms usually first become apparent in adolescence (Chanen, 2012; Shiner, 2009). Zanarini, Frankenburg, Khera, and Bleichmar (2001) found the mean of first clinical presentation

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to be age 18, with a standard deviation of 5–6 years. But the true age of onset was probably closer to 13, since most patients experienced symptoms for 5 years before entering the mental health system. Other data supporting the validity of BPD in adolescence come from the presence of similar psychosocial risk factors to those found in adults (Chanen, 2012; Goldman, D'Angelo, & DeMaso, 1993). Since schizophrenia and mood disorders also tend to begin after puberty, there is no reason to see BPD in a different light. Research has also documented typical cases of BPD in the adolescent years, which possess all the characteristic features of adult BPD (Chanen, 2012). The emergence of this clinical disorder is marked by wide mood swings and a wide variety of impulsive behaviors, including self-cutting, overdoses, and substance abuse—precisely the clinical picture found in adult BPD.

Even when a disorder emerges at a clinical level in adolescence, it can have precursors earlier in development. What were these patients like before puberty? We know the answer to this question for antisocial personality disorder (ASPD), which is often preceded by severe conduct disorder. But in BPD, precursors are not as obvious. While ASPD is essentially a continuation of conduct disorder, one does not see the symptoms of BPD in childhood, since children rarely cut themselves, overdose, abuse substances, or have the kind of stormy relationships that characterize adult patients. While children do sometimes think about or threaten suicide (Pfeffer, 2002), actual attempts are uncommon prior to puberty (Brent, 2001). These questions could only be answered by direct observation of populations identified as at risk, with longitudinal follow-up to determine which children develop BPD during adolescence.

Precursors of BPD in Childhood

Even if childhood symptoms are different from those seen in adolescence, personality traits are reasonably stable by mid-childhood (Shiner, 2009), and could reflect underlying predispositions for BPD, predicting its emergence later in development. Again, identifying childhood precursors of BPD is more difficult than in ASPD, in which a pattern of uncontrolled aggression can be identified as early as age three, associated with the development of antisocial behavior at age 18 (Caspi, Moffitt, Newman, & Silva, 1996). But early-onset conduct disorder does not always lead to ASPD, and adolescent-onset conduct disorder is a separate condition with a more favorable prognosis (Moffitt, Caspi, Rutter, & Silva, 2004).

Prospective research is most practical in high prevalence disorders like depression or alcoholism, but is more difficult in BPD, which is not nearly as frequent. The best estimates of community prevalence for adult BPD are around 1 %, about the same as schizophrenia (Lenzenweger, Lane, Loranger, & Kessler, 2007). While data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) has suggested that adult BPD occurs in 6 % of adults (Grant et al., 2008), these high numbers are contradicted by most other published reports (Paris, 2010). The NESARC researchers set the bar too low, and reanalysis with a higher threshold reduced prevalence at least by half (Trull, Jahng, Tomko, Wood, & Sher, 2010). Thus if we take 1 % as the most accurate estimate, if one followed 1,000 children over time, only 10 would develop BPD. Moreover, prospective studies of children always suffer from attrition, and those most at risk for BPD during adolescence could be particularly likely to drop out. For these reasons, even the best research, such as the large-scale children in the community study (Cohen, 2008), or the long-term follow-up of children at risk by Carlson, Egeland, and Sroufe (2009) have failed to find enough cases to draw firm conclusions.

Researchers have therefore had to rely on number of symptoms rather than a full diagnosis as an outcome variable. Johnson, Cohen, Brown, Smailes, and Bernstein (1999) reported a relationship between childhood adversity and BPD symptoms, while Carlson et al. (2009) reported that attachment disorganization and parental hostility during childhood predicted symptoms in young adulthood. The problem is that even if

there is a degree of continuity between BPD features and full disorder, these results may or may not be representative of clinical populations.

Given these problems, researchers need alternative strategies, one of which would be to study a high-risk sample. Some researchers have reported on symptoms in children of mothers with BPD (Sharp & Romero, 2007; Weiss et al., 1996). However, samples have been small because of the difficulty of recruitment. In any case, parent to child transmission of the disorder is rare (Links, Steiner, & Huxley, 1988), and these cohorts are at risk for a variety of other mental disorders, not necessarily BPD.

An alternative strategy would be to measure personality traits in children and then follow them to see if they develop BPD. In adults, the primary dimensions that underlie the disorder are emotional dysregulation (Linehan, 1993), also called affective instability (Koenigsberg, 2010); impulsivity or disinhibition (Gunderson & Links, 2008; Siever & Davis, 1991), and cognitive dysfunction (Zanarini, Gunderson, & Frankenburg, 1990). An additional domain of pathology, problems in interpersonal relationships (Gunderson et al., 2011), is probably a consequence of the others and references content not well represented in broad personality trait taxonomies. All these traits, which include both externalizing and internalizing problems, might describe a temperamental vulnerability to BPD.

Symptoms in children can also be divided into externalizing and internalizing problems that can be measured by standard instruments (Achenbach & McConaughy, 1997), and children at risk might show this pattern. The hypothesis that high levels of both externalizing and internalizing problems in childhood precede BPD has not been directly tested, but prospective data from a large-scale community study shows that children with this pattern are more likely to endorse suicidal ideation as young adults (Brezo et al., 2006). It is also possible that children who develop BPD might have a predominance of internalizing symptoms before puberty (explaining why children at risk do not come to clinical attention), accompanied by externalizing behaviors that become severe only after puberty (and are associated with referral to the mental health system).

Although the idea that all cases of BPD are associated with adversity and trauma has influenced clinical thinking, the relationship is far from consistent (Paris, 2008). Moreover, conclusions from retrospective data are limited by the fact that adult patients tend to have a negative view of their childhood, perceptions colored by current symptomatology (Hardt & Rutter, 2004). Child abuse and neglect are risk factors for BPD, but risk factors of this kind are seen only in a minority of cases, with only about a third reporting severe childhood adversity, about a third describing mild trauma, and another third reporting no traumatic events at all (Paris, 2008). Trauma is neither necessary nor sufficient, and these patterns are again consistent with equifinality and multifinality (Cicchetti & Rogosch, 2002).

Gender complicates the identification of developmental precursors of BPD. While most clinical cases are female, most epidemiological surveys find that, in contrast to antisocial personality, the disorder is equally common in men and women (Paris, 2010). This could be explained if men with BPD either have a less severe disorder or are not treatment-seeking. In childhood, externalizing symptoms (such as attention-deficit hyperactivity disorder and conduct disorder) drive referrals, so that the mental health system cares mostly for boys. Girls have less obvious symptoms, and might present a more internalizing picture prior to adolescence. Thus whatever drives the development of BPD could be in dormancy prior to puberty.

Again, the ideal research strategy would be one in which traits are measured in children at risk, and in which cohorts are studied prospectively (Tackett, Balsi, Oltmann, & Krueger, 2009; Tackett et al., 2012). This approach has illuminated the childhood precursors of many mental disorders. For example, children who later develop schizophrenia have minor symptoms from an early age (Baum & Walker, 1995). Depression in adults can be preceded by subclinical dysphoria during childhood (Cicchetti & Toth, 1998). Antisocial personality disorder is preceded by an early and severe onset of conduct disorder (Zoccolillo, Pickles, Quinton, & Rutter, 1992). But as we have seen, methodological and practical

problems have thus far prevented researchers from conducting this kind of definitive study in BPD.

Some alternative strategies could be considered. One is to study children in clinical settings who have symptoms that resemble adult BPD. The literature on these "borderline children" (Kernberg, Weiner, & Bardenstein, 2000) describes a population with a mixture of impulsive and affective symptoms (behavioral problems, suicidal threats, and mood instability), as well as cognitive phenomena similar to those seen in adults with BPD (micropsychotic phenomena such as hallucinations and paranoid trends), and problematic relationships. Our own research group identified this syndrome among children whose mothers meet criteria for a diagnosis of BPD (Weiss et al., 1996), and later conducted a study of a clinical cohort (Guzder, Paris, Zelkowitz, & Feldman, 1999; Guzder, Paris, Zelkowitz, & Marchessault, 1996). That study made use of a structured interview developed for diagnosis in children (Greenman, Gunderson, Cane, & Saltzman, 1986), with subscales for impulsivity, depression suicidality, and micropsychotic symptoms. It found similar risk factors in this cohort to those identified in adults: family dysfunction, abuse, and parental mental disorder.

The problem is that borderline pathology in childhood has limited relevance to adult BPD. The clinical picture is a general precursor for adult personality disorders rather than being specific to this diagnosis (Sharp & Romero, 2007). First, most cases involve boys, not girls, limiting the likelihood of BPD as an outcome. Second, when these cohorts are followed, they tend to develop a wide range of personality disorders, but not necessarily BPD (Lofgren, Bemporad, King, Lindem, & O'Driscoll, 1991; Zelkowitz et al., 2004).

Using a population-based approach, Zanarini et al. (2011) looked for BPD in prepubertal children on the cusp of adolescence. The sample was a birth cohort of 6,330 British 11-year-olds, the Avon Longitudinal Study of Parents and Children (ALSPAC). The findings showed that over 3 % met criteria for BPD. However diagnosis was based on the same broad criteria as the NESARC study, and may therefore be an overestimate. In a birth cohort longitudinal study of British twins, Belsky et al. (2012) examined "borderline personality related characteristics" (BPRC) in 1,116 pairs of twins aged 12. The findings showed that BPRC was both heritable and associated with harsh parental treatment, suggesting a stress-diathesis model of development. In a sample of 1,233 girls followed longitudinally, Stepp, Burke, Hipwell, and Loeber (2012) found that high levels of scores on attention-deficit hyperactivity disorder and oppositional defiant disorder at age 8 predicted BPD symptoms at age 14. However, since none of these studies is definitive, it will be interesting to see what children in these cohorts will look like on follow-up into young adulthood.

One advantage of studying children in the community with features that resemble BPD is to see whether the same risk factors are present. Thus, Winsper, Zanarini, and Wolke (2012), in the ALSPAC sample, found high levels of family dysfunction and maladaptive parenting associated with BPD features. This seems to provide further support for a relationship between psychosocial adversity and borderline psychopathology. However, these methods cannot tease out environmental from genetic risks. In a twin study, Boronolova, Hicks, Iacono, and McGue (2009) found that borderline traits in childhood are heritable, and that childhood abuse had no specific causal relationship to BPD features in children (Bornovalova et al., 2012). These findings, similar to results seen in sibling designs applied to adult BPD (Laporte, Paris, Russell, & Guttman, 2011), support a stress-diathesis theory for the developmental psychopathology of BPD features.

In summary, research on prepubertal children provides clues to the childhood precursors of BPD. However we do not know enough at this point to determine who is at risk and who is not. Even if the largest community follow-up studies

have had difficulty identifying cases, research faces a difficult challenge.

BPD in Adolescence

Adolescence is a trigger for the onset of many major mental disorders, including schizophrenia, bipolar disorder, and substance abuse (Paris, 2003). Many patients with BPD report affective and behavioral changes at puberty, and Zanarini et al. (2001) have confirmed this as a common age of onset for symptoms. Given that puberty is associated with so many major biological and psychosocial changes, this observation should not come as a surprise.

While a degree of moodiness and impulsivity is associated with normal adolescence, these problems only reach clinical significance in a minority of cases (Iliffe et al., 2009). Also, some of the behaviors that characterize BPD, including self-harm (Moran et al., 2012) and substance abuse (Copeland, Shanahan, Costello, & Angold, 2009), are commonly the subject of adolescent experimentation but remit by young adulthood. Those who continue to show these behaviors over time may be more likely to meet criteria for a diagnosis of adolescent BPD.

Yet clinicians are often reluctant to diagnose personality disorders in adolescence. This is a bias, based on the idea that all adolescents are "a little bit borderline", and one only need to wait until they grow out of this phase. Moreover, clinicians often consider the diagnosis to be stigmatizing. But avoidance of formal diagnosis might be reduced if it were more widely known that BPD tends to remit with time, and that the condition is treatable. Another problem is the overlap in symptoms between bipolar disorder and BPD in adolescents (Sharp, Ha, Michnski, & Venta, 2012). Many typical cases of BPD are being called bipolar, even without hypomania, largely on the basis of mood instability, a problem that cuts across all ages (Paris, 2012). Follow-up studies of children with mood dysregulation severe enough to be called "bipolar" shows that they show a similar picture in mid-adolescence to BPD youth (Geller, Tillman, Bolhofner, & Zimerman, 2008), and there is no evidence that adolescents with unstable mood go on to develop classical forms of bipolarity.

More broadly, the widely held belief that adolescence is a time of troubles is not supported by research. Most teenagers do not experience "adolescent turmoil", and this phenomenon is not universal among those with a high risk for adult mental disorders (Cicchetti & Rogosch, 2002). For this reason, BPD in adolescence cannot be written off as a transient phenomenon. In fact, adolescents who meet formal criteria for this disorder continue having serious symptoms in early adulthood (Bernstein et al., 1993).

Gender differences affect the timing of symptoms, in that aggressive behavior appears earlier in boys (Crick & Zahn-Waxler, 2003), consistent with the earlier onset of ASPD and the later onset of BPD. Using data from the large-scale children in the community study, Crawford, Cohen, and Brook (2001) found that among adolescents with BPD symptoms, externalization in males predicted continuing psychopathology, whereas a combination of externalizing and internalizing symptoms in females was most predictive. Finally, while there is a relationship of BPD to lower socioeconomic levels (Cohen et al., 2008), it has not been shown that family income or education affects outcome in young adulthood.

The BPD diagnosis in adolescence is not necessarily stable into young adulthood (Bernstein et al., 1993). That is another reason for counting symptoms in long-term follow-up of cohorts at risk (Cohen et al., 2008). Moreover, as shown by prospective studies of BPD in young adults (Gunderson et al., 2011; Zanarini et al., 2012), some patients with personality disorders, including, BPD, can remit within only a few years. These observations reflect, at least in part, an imprecision of classification, since diagnostic criteria often depend on acute symptoms rather than on stable personality traits. In this light, diagnostic instability should not necessarily be seen as showing recovery. Behavioral patterns can shift to move patients from one diagnosis to

another, which does not exclude continuation of dysfunction.

Conclusion

It is now clear that BPD begins in adolescence and can be treated in adolescence. However the mystery of its childhood precursors remains unsolved. Even if we could identify early risk factors, they might not be specific or sensitive to BPD as an outcome. The recipe for cooking BPD during childhood and adolescence requires many ingredients: abnormal temperament, psychosocial stressors, and social disadvantage.

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Suggested Reading

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Part II

Issues in Conceptualization and Assessment

Borderline Personality Disorder in Adolescence: Phenomenology and Construct Validity

3

Andrea Fossati

Definitions and core constructs

Borderline personality disorder (BPD) is a debilitating disorder that occurs in approximately 1–3 % of the general population (Leichsenring, Leibing, Kruse, New, & Leweke, 2011; Lenzenweger, 2008). It is characterized by distressful, impairing, and pervasive dysregulation of (1) affect (chronic fear of abandonment, affective instability, intense and inappropriate anger), (2) self-concept and attention (dissociative experience), of cognition (distorted thoughts and perceptions), (3) interpersonal relationships (intense, volatile), and (4) behavior (impulsivity and repetitive self-destructive behaviors). Individuals with BPD often engage in self-injurious and suicidal behavior, gambling, compulsive shopping, substance or alcohol use, binge eating, and reckless driving (American Psychiatric Association [APA], 2000, 2013; Leichsenring et al., 2011). DSM-5 (APA, 2013) diagnostic criteria for BPD are listed in Table 3.1. Given that these types of impulsive, self-destructive behaviors may lead to psychiatric hospitalization and/or incarceration, the rate of BPD in psychiatric settings is approximately 20 % and the rate in incarceration settings is even higher (APA, 2000, 2013; Leichsenring et al., 2011).

Thus, BPD is not only relatively prevalent but also associated with significant public health and security concerns; the clinical and social burden of adult BPD diagnosis raised the interest for early BPD diagnosis, in order to implement early intervention programs (Chanen, 2011; Miller, Muehlenkamp, & Jacobson, 2008; Sharp et al. 2009) which may promote more adaptive developmental pathways and averting many of the outcomes that were briefly summarized above. In the last three decades, several clinicians and researchers started to raise doubts as to whether it is plausible to suggest that BPD "jumps out of the blue" in a person as he or she turns 18 (Bleiberg, 2001; Bleiberg, Rossouw, & Fonagy, 2011; Chanen et al., 2004; Ludolph et al., 1990; Sharp et al., 2009; Winograd, Cohen, & Chen, 2008). Indeed, adolescents with poor social and academic functioning are frequently described as showing a constellation of symptoms of emotion dysregulation, instability in self-image and interpersonal relationships, and impulsivity that can be hardly differentiated from the clinical picture that would suggest a BPD diagnosis in adults (Bleiberg et al., 2011; Kernberg, Weiner, & Bardenstein, 2000). Moreover, the array and complexity of symptoms associated with BPD have inspired numerous etiological hypotheses of developmental antecedents of BPD, including deprivation of early socialization, constant exposure to chaotic and traumatic environments, deviant family

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Table 3.1 Diagnostic and statistical manual diagnostic criteria and proposed (i.e., Section III) criteria, 5th Edition (DSM-5; APA, 2013), for borderline personality disorder

DSM-5 diagnostic criteria for BPD	DSM-5 proposed (i.e., Section III) diagnostic criteria for BPD
A. Borderline Personality Disorder is manifested by a pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity beginning by early adulthood and present in a variety of contexts, as indicated by *five* (or more) of the following:	Typical features of borderline personality disorder are instability of self-image, personal goals, interpersonal relationships, and affects, accompanied by impulsivity, risk taking, and/or hostility. Characteristic difficulties are apparent in identity, self-direction, empathy, and/or intimacy, as described below, along with specific maladaptive traits in the domain of Negative Affectivity, and also Antagonism and/or Disinhibition
1. Frantic efforts to avoid real or imagined abandonment. Note: Do not include suicidal or self-mutilating behavior covered in (5)	**A**. Moderate or greater impairment in personality functioning, manifested by characteristic difficulties in two or more of the following four areas:
	1. ***Identity***: Markedly impoverished, poorly developed, or unstable self-image, often associated with excessive self-criticism; chronic feelings of emptiness; dissociative states under stress
	2. ***Self-direction***: Instability in goals, aspirations, values, or career plans
2. A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation. This is called "splitting"	
3. Identity disturbance: markedly and persistently unstable self-image or sense of self	3. ***Empathy***: Compromised ability to recognize the feelings and needs of others associated with interpersonal hypersensitivity (i.e., prone to feel slighted or insulted); perceptions of others selectively biased towards negative attributes or vulnerabilities
	4. ***Intimacy***: Intense, unstable, and conflicted close relationships, marked by mistrust, neediness, and anxious preoccupation with real or imagined abandonment; close relationships often viewed in extremes of idealization and devaluation and alternating between over involvement and withdrawal
4. Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating). Note: Do not include suicidal or self-mutilating behavior covered in (5)	**B**. Four or more of the following seven pathological personality traits, at least one of which must be (5) Impulsivity, (6) Risk taking, or (7) Hostility:
5. Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior	1. ***Emotional lability*** (an aspect of **Negative Affectivity**): Unstable emotional experiences and frequent mood changes; emotions that are easily aroused, intense, and/or out of proportion to events and circumstances
	2. ***Anxiousness*** (an aspect of **Negative Affectivity**): Intense feelings of nervousness, tenseness, or panic, often in reaction to interpersonal stresses; worry about the negative effects of past unpleasant experiences and future negative possibilities; feeling fearful, apprehensive, or threatened by uncertainty; fears of falling apart or losing control
	3. ***Separation insecurity*** (an aspect of **Negative Affectivity**): Fears of rejection by—and/or separation from—significant others, associated with fears of excessive dependency and complete loss of autonomy

(continued)

Table 3.1 (continued)

DSM-5 diagnostic criteria for BPD	DSM-5 proposed (i.e., Section III) diagnostic criteria for BPD
	4. ***Depressivity*** (an aspect of **Negative Affectivity**): Frequent feelings of being down, miserable, and/or hopeless; difficulty recovering from such moods; pessimism about the future; pervasive shame; feeling of inferior self-worth; thoughts of suicide and suicidal behavior
6. Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days)	5. ***Impulsivity*** (an aspect of **Disinhibition**): Acting on the spur of the moment in response to immediate stimuli; acting on a momentary basis without a plan or consideration of outcomes; difficulty establishing or following plans; a sense of urgency and self-harming behavior under emotional distress
	6. ***Risk taking*** (an aspect of **Disinhibition**): Engagement in dangerous, risky, and potentially self-damaging activities, unnecessarily and without regard to consequences; lack of concern for one's limitations and denial of the reality of personal danger
7. Chronic feelings of emptiness	7. ***Hostility*** (an aspect of **Antagonism**): Persistent or frequent angry feelings; anger or irritability in response to minor slights and insults
8. Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights)	**Specifiers.** Trait and level of personality functioning specifiers may be used to record additional personality features that may be present in borderline personality disorder but are not required for the diagnosis. For example, traits of Psychoticism (e.g., cognitive and perceptual dysregulation) are not diagnostic criteria for borderline personality disorder (see Criterion B) but can be specified when appropriate. Furthermore, although moderate or greater impairment in personality functioning is required for the diagnosis of borderline personality disorder (Criterion A), the level of personality functioning can also be specified
9. Transient, stress-related paranoid ideation or severe dissociative symptoms	

Note. BPD: Borderline Personality Disorder. BPD may be applied to children or adolescents when the individual's particular maladaptive personality traits appear to be pervasive, persistent, and unlikely to be limited to a particular developmental stage or an episode of an axis I disorder. Moreover, to diagnose a personality disorder in an individual *under 18* years of age, the features must have been present for at least *1 year* (this does not apply to Section III criteria which can be applied to adolescents; APA, 2013)

interactional patterns, and relatively subtle forms of neuropsychological and biochemical impairment (Leichsenring et al., 2011); these hypothetical developmental antecedents are thought to lead to maladaptive behaviors in adolescence, or even in childhood, which in turn are thought as a predictive of a BPD diagnosis in adulthood.

Notwithstanding these considerations, applying BPD (as well as any PD) diagnosis to adolescents is still a controversial topic (Miller et al., 2008). Personality disorders are defined in the DSM-5 (APA, 2013) as relatively stable, enduring, and pervasively maladaptive patterns of coping, thinking, feeling, regulating impulses, and relating to others; by contrasts, adolescents are usually involved in fluid developmental processes, in which dramatic changes take place and little appears stable or enduring (Bleiberg et al., 2011). During adolescence, boys

and girls have to face relevant body changes, dramatic increase in the intensity of affective and emotional drives, deep reorganization of the self in the context of peer-directed norms and interactions, and pressures towards autonomy and the assumption of adult roles. Moreover, DSM-5 (APA, 2013) indicates that BPD symptomatology in adolescence has to be severe enough such that behavioral manifestations persistently interfere with an adolescent's daily functioning over the course of 1 year or longer. Assuming a comprehensive clinical assessment is conducted, the DSM-IV-TR\-5 definition permits the diagnosis of BPD for adolescents; however it does remain vague, leaving much to clinical judgment (Miller et al., 2008).

Adolescence as a Key Developmental Stage for the Emergence of BPD

Adolescence may not represent a smooth transition to adulthood for many youngsters, and the transformations which physiologically take place during adolescence may give adolescents and their families a hard time (Bleiberg et al., 2011). Indeed, adolescence is usually considered as a developmental stage characterized by impulsivity, emotional and psychological turmoil, rapid mood swings, and increased vulnerability to breakdowns in adaptive behaviors (Irwin, 1989; Irwin, Burg, & Uhlercart, 2002). Empirical studies consistently show that sensation-seeking and risk-taking behaviors dramatically increase during adolescence, together with a rise in the conflicts with parents and an increase in the interest for peer-directed interpersonal relationships and social transactions (Irwin, 1989). Driving while intoxicated by alcohol, use of illicit drugs, binge drinking, fights, unsafe sexual intercourses, etc. are frequently observed in samples of US adolescents (Irwin et al., 2002); up to one-third of adolescents (particularly male adolescents) may experience limited capacity to meet adaptive demands, impairment in relationships, maladaptive impulsive behavior, self-harm, and relevant problems with identity and self-esteem (Offer & Offer, 1975; Westen, Betan, & Defife, 2011; see also Cohen et al., 2010 for perspective based on neuroscience). Impulsive and reckless, risk-taking behaviors are thought to be highly frequent in adolescence because they could help promoting adaptive fitness; together with peer-directed relationships, they facilitate exploration and promote the transition from childhood dependency from caregivers to independence and individuation in adulthood (Spear, 2007; Tucker & Moller, 2007).

From a different perspective, studies based on neurosciences consistently evidenced that neurodevelopmental changes which occur during adolescence bear close similarities to the neurobiological markers of BPD. For instance, adolescents are markedly less able than adults to recruit the frontal and prefrontal cortex when reading emotions; in particular, the activity of the dorsal medial prefrontal cortex has been consistently shown to decrease from adolescence to adulthood (Wang, Lee, Sigman, & Dapretto, 2006). Social perspective taking is also disturbed during adolescence (Choudhury, Blakemore, & Charman, 2006). The ability to determine if words match the expression of emotions declines in both speed and accuracy (Monk et al., 2003; by contrast, amygdala activation in response to pictures of faces expressing emotions is significantly greater in adolescents (Killgore, Oki, & Yurgelun-Todd, 2001). Amygdala hyperreactivity in adolescents has been reported also in response to fearful faces during a go/no go task (Hare et al., 2008). Thus, as a whole, neuroscientific studies consistently reported a phase-specific compromise in mentalistic functions during adolescence. Interestingly, amygdala hyper-reactivity has been described in adults with BPD (New et al., 2007), together with poor functioning of the lateral prefrontal cortex, medial prefrontal cortex, medial parietal cortex, medial temporal lobe, and rostral anterior cingulated cortex (Fonagy & Luyten, 2009)—all structures that undergo massive reorganization during adolescence. Far from suggesting that all adolescents may show BPD difficulties to some extent, findings from neuroscientific studies may help to understand the underlying mechanisms that lead vulnerable children—including attachment

disorganization as a vulnerability factor—to manifest BPD symptoms when they enter adolescence (Bleiberg et al., 2011).

Empirical Evidence on the Reliability of BPD Diagnosis in Adolescence

All available data indicate that adolescence is a critical point for early identification and therapeutic treatment of BPD. Thus, it is not surprising that the relevance of DSM-IV-TR\-5 (APA, 2000, 2013) criteria for differentiating emerging BPD in adolescence from normal adolescent development and stress has become a relevant clinical topic.

When semi-structured interviews were used to diagnose DSM-symptom criteria in adolescence, empirical studies consistently reported adequate inter-rater reliability values for BPD diagnosis, usually in the 0.85–0.88 range (Becker et al., 1999; Blais, Hilsenroth, & Fowlder, 1999; Garnet, Levy, Mattanah, Edell, & McGlashan, 1994). Currently, the Childhood Interview for DSM-IV Borderline Personality Disorder (CI-BPD; Zanarini, 2003a) represents the only published interview-based measure specifically adapted for use in children and adolescents. Zanarini, Horwood, Wolke, Waylen, Fitzmaurice, and Grant (2011) used it in a sample of 6,410 11-year-old children in the United Kingdom. Inter-rater reliability using taped interviews of 30 children revealed κ values ranging from 0.36 to 1.0, with a median value of 0.88. Overall, 86 % of the κ values were in the excellent range of greater than 0.75; two independent studies reported similar findings (Chang, Sharp, & Ha, 2011; Sharp et al., 2011). Recently, Sharp, Ha, Michonski, Venta, and Carbone (2012) showed that using the CI-BPD the inter-rater reliability of the DSM-IV BPD diagnosis in adolescent was excellent, as it was indicated by a Cohen κ value of 0.89.

Although findings are far from being conclusive, the internal consistency of the DSM-symptom criteria for BPD in adolescence seems also adequate ($\alpha = 0.76$) and comparable to the internal consistency observed in adult participants ($\alpha = 0.74$) (Becker et al., 1999). Sharp and colleagues (2012) showed that the Cronbach α value of the DSM-IV BPD criteria in a large sample ($N = 245$) of adolescent inpatients was 0.80 when the CI-BPD (Zanarini, 2003a) was used to assess BPD. Recently, Michonski, Sharp, Steinberg, and Zanarini (2013) demonstrated using item response theory approach that in a large, population-based sample ($n = 6{,}339$) of young adolescents from the United Kingdom (ages 11–12) a single underlying dimension adequately accounted for covariation among the BPD criteria; moreover, each criterion was found to be discriminating to a degree comparable to what has been reported in adult studies.

As a whole, psychometric data clearly indicate that BPD could be reliably diagnosed in adolescence using descriptive diagnostic criteria (Miller et al., 2008). The lower values that were observed for the internal consistency of BPD criteria when compared with values of inter-rater reliability for BPD diagnosis suggest that the clinical presentation of BPD in adolescence may be as heterogeneous as it is in adulthood; however, diagnostic agreement between independent clinician/research for BPD diagnosis in adolescence is good.

Although some studies reported disproportionally high prevalence rates for DSM-based BPD diagnosis, with values ranging from roughly 11 % (Bernstein et al., 1993) to 14 % (Chabrol, Montovany, Chouicha, Callahan, & Mullet, 2001), these findings were likely to be the result of unreliable assessment of BPD features and sampling bias (Miller et al., 2008). Recent studies based on a rigorous methodology of BPD assessment and large community samples reported prevalence rates for BPD diagnosis in adolescence were less suspect than previous findings. Zanarini (2003b), using the CI-BPD in a community sample of 6,330 11-year-old participants who were interviewed after their 11th birthday, reported a base rate of 3.27 % for DSM-IV-TR BPD diagnosis. This finding closely matched the epidemiological data on the prevalence of BPD in general population samples of adult participants (Lenzenweger, 2008). Interestingly, when Zanarini and colleagues (2011)

compared the prevalence of BPD in the community sample of 6,330 11-year-old participants with the prevalence of BPD in a community sample of 34,653 American adults, they reported that a significantly higher percentage of adults than children met DSM-IV criteria for BPD (5.9 % vs. 3.2 %).

Thus, although BPD criteria may be over-inclusive of symptoms that characterize the developmental period of adolescence (Miller et al., 2008), prevalence rates of thoroughly assessed BPD diagnosis did not advise against diagnosing BPD in adolescence (and even in late childhood).

Researchers considered stability as a key defining feature of BPD and emphasized the persistence (i.e., temporal stability) of BPD diagnosis over time as the "gold standard" regarding the reliability of the BPD diagnosis in adolescence (Miller et al., 2008). Some research, based primarily on community samples, suggested that BPD may have concurrent validity in adolescence (i.e., is a valid indicator of distress and dysfunction), but is relatively unstable over time (e.g., Bernstein et al., 1993; Bondurant, Greenfield, & Tse, 2004; Korenblum, Marton, Golembeck, & Stein, 1990; Mattanah, Becker, Levy, Edell, & McGlashan, 1995); this suggests that BPD in adolescence may reflect a point-in-time disturbance rather than chronic impairment (Levy et al., 1999). However, current research indicates that BPD is not particularly stable in adult samples either, and that symptoms are likely to be reduced through treatment efforts to a subclinical or non-clinical level of dysfunction. A relatively recent 10-year follow-up study of adult patients with BPD showed that 88 % of adult patients obtained remission of BPD symptoms over a 10-year period and once remitted, recurrence of symptoms occurred in only 6 % (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2006). Similar findings were reported by the Collaborative Longitudinal Study on Personality Disorders in the Collaborative, in which it was found that 85 % of borderline patients achieved a remission lasting 12 months or longer (Gunderson et al., 2011); however, in this study only 20 % of borderline patients attained a Global Assessment of Functioning score of 71 or higher for a period of 2 months or longer (Gunderson et al., 2011). These findings suggested that BPD may not be as constant a dysfunction as originally believed (Zanarini et al., 2006).

Interestingly, longitudinal studies consistently support the hypothesis that DSM-based diagnostic criteria for BPD in adults represent an admixture of acute symptoms and trait-like or temperament-like dysfunctional personality features (McGlashan et al., 2005; Zanarini et al., 2006). In particular, 10-year follow-up data based on adult patients with BPD diagnosis suggest that chronic dysphoria, thinking/unusual perceptual experiences and paranoid ideation, and intolerance of aloneness, abandonment/engulfment/annihilation concerns, counterdependency, and dependency/masochism represent BPD features that are relatively slow to remit (Zanarini et al., 2006). Interestingly, longitudinal studies on the course over time of BPD diagnosis in adolescence show that despite reports of low temporal stability, there appears to be a subset of adolescents for whom the diagnosis remains over time, as with adults (see also Chaps. 16and 18). Moreover, recent data from a study based on 47 adolescent girls assessed over a 10-year period suggest a pattern of enduring functional and psychopathological impairments associated with poor outcomes in individuals once diagnosed with BPD, even if symptomatically "remitted" (Biskin, Paris, Renaud, Raz, & Zelkowitz, 2011). This finding is highly consistent with the results of Zanarini and colleagues' (2012) 16-year follow-up study on adults with BPD which indicated that sustained symptomatic remission is substantially more common than sustained recovery from BPD, and that sustained remissions (defined as no longer meeting study criteria for BPD or another personality disorder (DSM-III-R criteria; APA, 1987) for a period of 2 years or longer, or one follow-up period) and recoveries (defined as a Global Assessment of Functioning score of 61 or higher) are substantially more difficult for BPD patients to attain and maintain than for patients with other forms of personality disorder.

In summary, available research data indicate that BPD can be reliably diagnosed in adolescence; rather, BPD diagnosis itself is likely to be less stable than it was previously thought both in adolescence and adulthood, and BPD diagnosis seems to be composed of acute symptoms and trait-like—or temperament-like—dysfunctional features. The relevance of an early diagnosis of BPD in adolescence is stressed by the fact that poor outcomes have been observed in young adults who were diagnosed as BPD in adolescence, even in the case of "remission" of BPD.

Empirical Evidence on the Construct Validity of BPD Diagnosis in Adolescence

A number of research studies tried to address the construct validity of BPD in adolescents—i.e., whether the BPD diagnosis in adolescence actually measures what is intending to measure—pointing to the consistent relationships found between BPD and associated areas of dysfunction and distress as evidence of the validity of the BPD diagnosis (Becker, Grilo, Edell, & McGlashan, 2002; Becker, McGlashan, & Grilo, 2006; Blais et al., 1999; Chabrol & Leichsenring, 2006; Chabrol, Montovany, Callahan, Chouicha, & Ducongé, 2002; Grilo, Becker, Edell, & McGlashan, 2001; Levy et al., 1999; McManus, Brickman, Alessi, & Grapentine, 1984; Meijer, Goedhart, & Treffers, 1998; Pinto, Grapentine, Francis, & Picariello, 1996). As a whole, adolescents with BPD diagnosis when compared to non-BPD controls show at follow-up: (a) lower GAF score and higher scores on self-report measures of acute psychiatric symptoms—e.g., anxiety, depression, etc. (Levy et al., 1999); (b) social impairment, e.g., fewer and shorter friendships, less enjoyment of others, a lack of a confidant, the absence of a romantic relationship, and fewer social activities (Becker et al., 1999); (c) school or work problems, for instance, repeating grades or dropping out of school (Becker et al., 1999); (d) higher comorbidity of axis I diagnoses; (Becker et al., 2006; Chabrol et al., 2002; Grilo et al., 2001; Pinto et al., 1996) and (e) higher frequency of contact with the police for antisocial behavior (Becker et al., 1999; Chabrol & Leichsenring, 2006), and higher frequency of drug use (Bornovalova, Hicks, Iacono, & McGue, 2013).

Becker and colleagues (1999) reported that mean inter-criterion correlations for BPD criteria were low, but also similar across adolescent and adult groups (0.28 for adolescents, 0.26 for adults). Discriminant validity was adequate and similar between the adolescents and adults as evidenced by low diagnostic overlap measured through inter-category mean inter-criterion correlations (0.07 for adolescents, 0.06 for adults). In a study on the diagnostic efficiency of BPD criteria between adolescent and adult inpatients, Becker et al. (2002) found no significant differences in the base rates of BPD between the adolescents and adults, nor any of the BPD criteria; similar findings were obtained also in Segal-Trivitz et al. (2006) study.

Although some studies reported a substantial overlap among the individual symptom criteria of BPD and other axis I and axis II disorders (Becker, Grilo, Edell, & McGlashan, 2000; Crawford, Cohen, & Brook, 2001), several studies indicated that some BPD in adolescents can be reliably distinguished from other axis I disorders (Becker et al., 2006; Chabrol et al., 2002; Grilo et al., 2001; Pinto et al., 1996).

Studies showed that adolescents diagnosed with BPD at baseline continued to experience some level of affective disturbance or behavioral disruption even when they "remitted" from BPD—i.e., when they did not meet the criteria for BPD diagnosis any longer (Biskin et al., 2011; Meijer et al., 1998). The findings that functional impairments persisted among adolescents who no longer met BPD criteria, particularly for affective disturbances, is consistent with findings in the adult literature.

As a whole, available scientific evidence, together with recent studies based on thorough assessment of DSM-IV BPD criteria in a large, community-based sample of adolescents (Michonski et al., 2013), indicates that a dimensional perspective may be particularly important for conceptualizing BPD pathology among youth because it is better able to account for the

developmental fluctuations and increased heterogeneity that have been reported in younger samples.

The high comorbidity between BPD and several axis I disorders—in particular, mood disorders, anxiety disorders, substance abuse, eating disorders, posttraumatic stress disorder, and attention deficit/hyperactivity disorder—that was frequently reported in the scientific literature lead some author to call into question the diagnostic specificity of BPD in adolescence (Bleiberg et al., 2011). However, it should be observed that the comorbidity rate between axis I disorders and BPD that is observed in adolescent samples is consistent with the epidemiological data that were obtained in adult BPD samples; usually, co-occurrence base rates between BPD and axis I diagnoses among adults are in the range of 10–30 % for bipolar I/II disorders, 30–50 % for eating disorders and posttraumatic stress disorder, 50 % for substance use disorder, and 50–60 % for anxiety disorders (Kutcher, Marton, & Korenblum, 1990; Lewinsohn, Zinbarg, Seeley, Lewinsohn, & Sack, 1997; Muratori, 2003). The differential diagnosis between BPD and mood disorders may be problematic as mood disorders are themselves poorly characterized in adolescence; this may be particularly difficult in the case of bipolar II disorder. Indeed, adolescents with mood disorders are usually described as moody, irritable, affectively labile with anger outbursts and poor tolerance to frustration. However, it should be stressed that, different from bipolar II disorder (and bipolar I disorder as well), the affective shifts of BPD oscillate between anger and dysphoria rather than elation and depression, and tend to be *rapidly* reversible and exquisitely *reactive* to the relationship context rather than endogenously driven and episodic (Henry et al., 2001; Koenigsberg et al., 2002). On the contrary, episodic manifestation, lasting days or weeks, of elevated mood, increased energy/activity, disinhibition, accelerated thinking, decreased need for sleep, poor critical judgment—or at the opposite excessive need for sleep, psychomotor retardation, and irritability/impulsivity, and psychotic symptoms strongly suggest to consider a mood disorder diagnosis, particularly in the presence of a family history of mood disorder (Bleiberg et al., 2011).

The "Warning Signs" of Emerging BPD in Adolescence

Research evidence indicates that there is no single symptom which is predictive of later BPD diagnosis in adolescence (Bleiberg et al., 2011); rather, a pattern of 2–3 selected BPD symptoms in adolescence seemed to be highly predictive of BPD diagnosis at follow-up. Early studies reported that symptoms with the highest predictive power (i.e., most stable symptoms) for adolescents were chronic feelings of emptiness and inappropriate, intense anger (Garnet et al., 1994). Additional studies have also consistently identified symptoms of identity disturbance, affective instability, and inappropriate, intense anger as having the greatest predictive power for BPD in adolescents (Becker et al., 2002; McManus et al., 1984; Meijer et al., 1998; Pinto et al., 1996). The positive predictive power of these three symptoms is almost identical to those identified in the adult BPD literature (Becker et al., 2002; Blais et al., 1999), suggesting that apparent key symptom criteria are valid across age groups. The role of identity disturbance as a core diagnostic feature of BPD in adolescence was strongly supported by Westen et al. (2011) findings; in this study identity disturbance in adolescents appeared as a multidimensional construct—it was based on four distinct factors, lack of normative commitment, role absorption, painful incoherence, and lack of consistency—which was highly similar to identity disturbance in adults and significantly predictive of BPD symptoms (even controlling for the effect childhood abuse; Westen et al., 2011).

Recently, Michonski and colleagues (2013) reported that a single underlying dimension adequately accounted for covariation among the BPD criteria. Each criterion was found to be discriminating to a degree comparable to what has been reported in adult studies. Interestingly, consistent with adult findings (Aggen, Neale,

Røysamb, Reichborn-Kjennerud, & Kendler, 2009; Jane, Oltmanns, South, & Turkheimer, 2007), five BPD criteria were found to exhibit differential item functioning—i.e., differences in the relation of an item to the latent trait across population subgroups—between boys and girls. In Michonski and colleagues' (2013) study, paranoid ideation and identity disturbance were the most discriminating DSM-IV BPD criteria in boys and girls, respectively; BPD characteristics dealing with emotional reactivity or poor impulse control (i.e., inappropriate, intense anger or difficulty controlling anger, affective instability, and impulsivity) were the easier to endorse compared with suicidal behaviors (girls) and abandonment fears (boys) that were the more "difficult" (i.e., requiring the highest level of BPD liability) to endorse.

Deliberate, nonsuicidal self-harm deserves a particular consideration. Although deliberate self-harm is a common feature of BPD in adolescence (e.g., Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006), it is neither necessary nor sufficient symptom for diagnosing BPD in adolescence (e.g., Siever, Torgersen, Gunderson, Livesley, & Kendler, 2002). However, deliberate self-harm in adolescence should be carefully assessed in adolescence because of four key issues: (a) deliberate self-harm is highly addictive because it releases opiates that relieve the pain and sensitivity to abandonment, rejection, or difficulties in attunement associated with the reduced opiates shown to be implicated in self-injurious behavior in BPD (Stanley et al., 2010); (b) in adolescence, nonsuicidal, deliberate self-harm shows a significant overlap with suicidal behavior, including instances of unintended, accidental death or near death in the course of deliberate self-harm (Nock et al., 2006); (c) nonsuicidal, deliberate self-harm is a particularly obnoxious instance of nonmentalizing behavior (Bleiberg et al., 2011), consistently associated with emotional neglect (Sar, Akyuz, Kugu, Ozturk, & Ertem-Vehid, 2006) and dissociation triggered by abandonment, rejections, or lapses in attunement (Stiglmayr et al., 2008); (d) nonsuicidal, deliberate self-harm is a marker of mentalizing collapse which is strongly associated with dissociation and evocative of intense, albeit frequently chaotic and problematic reactions in others—e.g., acute hospitalizations and desperate efforts of parental control, mixed with feelings of guilt, shame, rage, and despair that spur coercive cycles leading to further impairment in the mentalistic abilities of both BPD adolescents and their parents.

Recent data indicate that selected childhood disorders and behavioral problems may represent childhood antecedents of emerging BPD features in adolescence. Burke and Stepp (2011) using prospective data from the Developmental Trends Study (Loeber, Green, Lahey, Frick, & McBurnett, 2000), a clinic-referred sample of 177 boys, found that childhood and adolescent symptoms of ODD and ADHD as well as marijuana use predicted BPD symptoms at age 24. Interestingly, in Burke and Stepp (2011) study, conduct disorder (CD), depression, and anxiety were not related to BPD symptoms in young adulthood. This finding was replicated by Stepp, Burke, Hipwell and Loeber (2011) in a study based on data from 1,233 girls spanning ages 8–14 years. The authors found that ADHD and ODD symptoms at age 8 predicted BPD symptoms at age 14; moreover, the rate of growth in ADHD symptoms from ages 8–10 years and the rate of growth in ODD symptoms from age 10–13 predicted BPD symptoms at age 14. These patterns of prospective associations were not found for CD and depression at age 14 (Stepp et al., 2011).

Clinical considerations and research data also point to other childhood markers of vulnerability for the emergence of BPD in adolescence. Children at risk for manifesting BPD in adolescence are characterized by relational aggression, a hostile, distrustful view of the world, and affective instability in relational contexts (Crick, Murray-Close, & Woods, 2005), controlling and coercive behaviors towards attachment figures, impulsivity, poorly defined sense of the self, and a propensity towards inappropriate and intense outbursts of anger (Allen, Fonagy, & Bateman, 2008; Bleiberg et al., 2011). Although recent longitudinal data seem to be inconsistent with

Table 3.2 Core diagnostic features of borderline personality disorder in adolescence, childhood markers of vulnerability to BPD, and positive outcome ("resilience") factors

	Borderline personality disorder in adolescence
Core diagnostic features	Identity disturbance (particularly for girls)
	Inappropriate, intense anger
	Paranoid ideation (boys)
	Chronic feelings of emptiness (deliberate self-harm, dissociation proneness)
Childhood markers	
Childhood disorders	Attention deficit/hyperactivity disorder
	Oppositional defiant disorder
Childhood problem behavior	Controlling and coercive behaviors towards attachment figures
	Poorly defined sense of the self
	Hostile, distrustful view of the world
	Relational aggression
	Intense outbursts of anger
	Affective instability
Resilience factors	Reflection
	Agency
	Relatedness

Note: No single diagnostic element is suggestive of a BPD diagnosis in adolescence, rather a pattern of 2–3 diagnostic criteria may be suggestive of emerging BPD in adolescence; diagnostic criteria between brackets indicate BPD characteristics that frequently occur in the clinical presentation of BPD in adolescence, although they lack diagnostic specificity

the widely held assumption that childhood abuse causes BPD, suggesting that BPD traits in adulthood are better accounted for by heritable vulnerabilities to internalizing and externalizing disorders (Bornovalova et al., 2013), it should be considered that when such vulnerable children are exposed to childhood abuse or neglect in attachment relationships, they become engulfed in a vicious cycle: trauma activates the attachment system and efforts to seek proximity and protection. However, attachment also activates intense distress, in both children and their caregivers, and this escalating distress—and the associated increase in the chances of further trauma—intensifies attachment activation (Bleiberg et al., 2011).

Finally, empirical data suggest the existence of *resilience* factors which should not be overlooked by the clinicians treating adolescents. Aiming at identifying protective factors from persistent "turmoil" in adulthood, Hauser, Allen and Golden (2006) followed a sample of 150 adolescents, half of whom had been admitted to psychiatric hospitals in their early adolescence; interestingly, they identified a group of "surprising" participants who were former patients and were functioning in the top half of all young adults—both former patients and never hospitalized participants—in measures of psychiatric symptoms, antisocial behavior, quality of relationships, and social and emotional functioning. According to Hauser and colleagues (2006) findings, the "surprisingly" resilient participants were characterized by three protective factors: (a) *reflection*—i.e., the capacity and willingness to recognize, experience, and reflect on one's own thoughts, feeling, and motivations; (b) *agency*—i.e., a sense of oneself as effective and responsible for one's actions; (c) *relatedness*—i.e., a valuing relationship that takes the form of openness to the other's perspective and of efforts to engage with others (using contemporary terminology, it could be said that Hauser and colleagues' (2006) protective factors are markers of effective mentalizing).

Childhood markers of vulnerability to BPD manifestation in adolescence, most discriminating BPD features (i.e., core diagnostic features) in adolescence, and protective factors are summarized in Table 3.2.

Future Directions

In summary, notwithstanding the controversies that historically surrounded the diagnosis of BPD in adolescence, available scientific evidence indicates that BPD can be reliably and validly diagnosed in adolescent clients. Empirical studies suggest that while there is a subgroup of severely affected adolescents for whom the BPD diagnosis remains stable over time, there appears to be a less severe subgroup that moves in and out of the diagnosis. Rate of diagnostic stability of BPD in adolescents is comparable to that in adults and a select few symptom criteria have consistently emerged as significant predictors of BPD retention in both adolescents and adult samples (e.g., Zanarini et al., 2006; Zanarini, Frankenburg, Hennen, Reich, & Silk, 2005). Available evidence recommends that BPD in adolescents be conceptualized from a dimensional/continuous rather than categorical approach, as a dimensional approach may better account for the developmental variability and the heterogeneity found among adolescents (Miller et al., 2008).

Consistent with Miller and colleagues' (2008) suggestions, current literature indicate that mental health practitioners should strongly consider formally assessing for BPD, either categorically or continuously, when working with adolescents. Regardless of presence of a full-fledged diagnosis, BPD symptoms (even if fewer than 5) in adolescents may indeed accurately reflect significant distress and dysfunction that requires intervention (Miller et al., 2008). Reliable and valid self-report measures—for instance, the Borderline Personality Inventory (BPI; Chabrol et al., 2004; Leichsenring, 1999), Borderline Personality Disorder Features Scale for Children (BPFSC; Crick et al., 2005), Personality Assessment Inventory (Morey, 2007), and the Millon Adolescent Clinical Inventory (Millon & Davis, 1993; Millon, Millon, & Davis, 1993)—and semi-structured interviews/observer-rated measures—for instance, the CI-BPD (Zanarini, 2003a)—are currently available to clinicians and researchers to assess BPD in adolescence. Thus, although it is known that there may be negative stigma associated with BPD diagnosis and that this poses a serious concern with regard to using the diagnosis, available data strongly indicate that stigma should not preclude clinicians from assessing for BPD and carefully considering BPD diagnosis when warranted (Miller et al., 2008).

The impact of the changes to personality disorder general criteria and BPD diagnostic criteria that are incorporated in Section III DSM-5 will significantly affect future research in this area. Although the DSM-5 model of personality disorders is not without criticism (see, for instance, the position summarized in Krueger & Eaton, 2010) and has been added to the provisional section of the DSM 5 (i.e., Section III), the proposed removal from DSM-5 Section III criteria of any reference to adult age in order to diagnose personality disorders (Skodol, 2011) will be likely to prompt the assessment of BPD in adolescence. The shift to a hybrid dimensional-categorical model for personality and personality disorder assessment and diagnosis in the DSM-5 is highly consistent with Michonski and colleagues' (2013) findings concerning the need for flexible diagnostic systems for assessing BPD in adolescence. The emphasis placed by current DSM-5 characterization of personality disorder, including BPD, as the failure to develop coherent sense of self or identity (identity and self-direction) and chronic interpersonal dysfunction (empathy and intimacy) as core features (Skodol, 2011), is in line with the findings of longitudinal studies indicating that identity disturbances and disturbed relationships represent key elements of the early manifestations of BPD in adolescence. Interestingly, in Section III of the DSM-5 the traits of Negative Affectivity—namely, Emotional lability, Anxiousness, Separation insecurity, and Depressivity-, Disinhibition—namely, Impulsivity and Risk taking, and Antagonism—namely, Hostility—as defining features of the BPD dysfunctional trait profile is in close agreement with findings showing that paranoid ideation (Michonski et al., 2013), inappropriate anger outbursts, and affective instability (Becker et al.,

2002; Garnet et al., 1994; McManus et al., 1984; Meijer et al., 1998; Pinto et al., 1996) are key elements of the clinical picture of BPD in adolescence.

The development and refinement of diagnostic criteria and assessment instruments specifically designed to capture BPD manifestations in adolescence and childhood is clearly among the priorities of the agenda for future research on BPD. Current evidence indicates that BPD does not appear "out of the blue" in adolescence; rather, symptoms of attention deficit/hyperactivity disorder and oppositional defiant disorder in childhood or childhood problem behavior usually precede the emergence of BPD in adolescence. Of course, this data does not mean that all children with attention deficit/hyperactivity disorder or oppositional defiant disorder will develop BPD later in their lives. Hopefully, future studies will illuminate the developmental pathways that lead some children to develop BPD in adolescence or adulthood, highlighting both risk factor and protective factors.

The co-occurrence of other axis II personality disorders (as well as of other axis I disorders) with BPD in adolescence, and its impact on course, outcome and treatment response of BPD, is likely to receive special attention in the next future. In adult participants, BPD has been reported to be characterized by high co-occurrence rates with other disorders, particularly with bipolar, narcissistic personality disorder and schizotypal personality disorder (Grant et al., 2008). Interestingly, Zanarini et al. (2004) in a 6-year perspective study on based 290 patients meeting both DIB-R (Zanarini, Frankenburg, Chauncey, & Gunderson, 1987; Zanarini, Frankenburg, & Vujanovicm, 2002) and DSM-III-R (APA, 1987) criteria for BPD reported that in adult participants both remitted and non-remitted BPD patients experienced declining rates of most types of axis II disorders over time. However, the rates of avoidant, dependent, and self-defeating personality disorders remained high among non-remitted borderline patients. Additionally, the absence of these three disorders was found to be significantly correlated with a borderline patient's likelihood of remission and time-to-remission. Zanarini and colleagues' (2004) findings clearly highlight the need for studying the impact of co-occurring personality disorders on the clinical curse of BPD also in adolescent samples.

Last but not least, the possibility to identify very early manifestations of BPD in childhood is likely to represent a major research task in the next future. Research data on adolescents point to the fact that early identification of BPD allows to implement empirically based early intervention programs (Chanen, 2011); in a sense, the earlier the diagnosis and treatment of BPD, the better the outcome. Interestingly, It has been suggested that enduring personality patterns are apparent by the end of preschool (Kernberg et al., 2000). Moreover, patterns of inflexible coping strategies and insecure attachment in the preschool years have been found to develop into persistent childhood characteristics that manifest themselves as depression, drug use, and criminal behaviors later in life (National Advisory Mental Health Council, 1995).

Considering BPD properly, some studies suggested that BPD symptoms are likely to be detectable at an early age, showing that specific features of BPD, such as self-harm or traits of impulsivity and affective instability present during childhood, are predictive of receiving a BPD diagnosis as an adult (e.g., Siever et al., 2002; Zanarini et al., 2006). Siever and colleagues (2002) found that parents of patients with BPD reported their children's development was characterized by a pattern of unusual sensitivity, moodiness, and self-soothing that differentiated these children who were received a BPD diagnosis as adults from their non-BPD siblings; this pattern increased ninefold the likelihood of subsequently receiving a BPD diagnosis. In the same study, the authors reported the manifestation in early adolescence of promiscuity, impulsivity, suicidal behavior, verbal aggression, and deliberate self-harm in girls who subsequently received a BPD diagnosis, and of substance abuse, suicide attempts, impulsive behavior, aggression, and self-cutting in boys who will be given a BPD diagnosis as adults. These few studies do not allow to draw any firm conclusion about the

construct validity of the BPD diagnosis in childhood, but indicate the relevance of this research topic and represent a path for future studies.

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The Underlying Factor Structure of DSM criteria in Youth BPD

4

Jared D. Michonski

Factor Analysis as a Tool for Understanding BPD

Factor analysis is a statistical procedure that aims to identify from a set of observed variables a smaller number of underlying dimensions (latent factors) that account for covariation among the observed variables. For the purposes of this chapter, the observed variables consist of items—or more precisely, diagnostic criteria as, for instance, found in the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Publishing, 2013)—as opposed to subscale or test scores (see Chaps. 5–8 for a discussion of other approaches to assessing borderline personality disorder (BPD) in youth). When lacking a priori hypotheses regarding the latent structure, exploratory factor analysis (EFA) is undertaken. When fitting the variables to a hypothesized latent structure, the approach constitutes confirmatory factor analysis (CFA). Both approaches have been used extensively to investigate the dimensional structure of BPD, particularly in adults.

Factor analysis is useful for addressing several questions relevant to the evaluation of psychiatric diagnoses. These include questions pertaining to how well each criterion (or item) discriminates among varying levels of the latent factor(s) (i.e., size of factor loadings), what level of the latent factor(s) is required for an individual to be expected to endorse a given criterion (i.e., threshold locations), and whether the criteria are invariant across population subgroups, such as gender (Aggen, Neale, Roysamb, Reichborn-Kjennerud, & Kendler, 2009; Feske, Kirisci, Tarter, & Pilkonis, 2007; Michonski, Sharp, Steinberg, & Zanarini, 2013). Of greatest importance to the present discussion, however, is the issue of construct validity. Floyd and Widaman (1995) describe CFA as a technique that speaks primarily to the question of construct validity: Does the instrument's factor structure reflect the hypothesized structure of the construct that the instrument is intended to measure?

The issue of the presumed structure of the BPD construct is not a simple one—perhaps especially in youth. On the one hand, criteria were selected to define a single diagnostic entity (DSM-III; APA, 1980). As such, one would expect for the BPD criterion set to constitute a coherent combination of traits and symptoms that "hang together," as would be evidenced by a single common factor adequately accounting for covariation among the criteria. In other words, the diagnostic criteria could be represented by a single dimension of severity, such that the higher one's standing on this latent severity dimension the greater the likelihood that the individual would be rated as positive for *any* of the given criteria.

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On the other hand, the heterogeneity of BPD has been a well-documented problem (e.g., Lenzenweger, Clarkin, Yeomans, Kernberg, & Levy, 2008; Sanislow & McGlashan, 1998; Zanarini et al., 1998). In adolescents, BPD has been noted to constitute an even "more diffuse range of psychopathology" (Becker et al., 1999; Becker, Grilo, Edell, & McGlashan, 2000, 2002; Becker, McGlashan, & Grilo, 2006). If it is true that BPD is a heterogeneous condition, then one might expect that a multidimensional factor structure underlies the diagnostic criteria. In this case, separable (although likely correlated) dimensions of substantive meaning (e.g., "disturbed relatedness," "behavioral dysregulation," and "affective dysregulation"; Sanislow, Grilo, & McGlashan, 2000) would permit characterization of BPD individuals in terms of their profile with respect to these BPD factors. For instance, one person meeting criteria for BPD may be characterized by high affective and behavioral dysregulation but low disturbed relatedness, while another is characterized by elevations across all three factors. Incidentally, such a depiction is roughly consistent with the tenth edition of the International Classification of Diseases' (ICD-10; World Health Organization, 1992) parsing of BPD symptoms into *impulsive* and *borderline* subtypes. That is, the impulsive subtype is characterized by emotional instability and poor impulse control, whereas the borderline subtype is characterized by emotional instability, unstable self-image, emptiness, unstable relationships, and suicidal/self-harm behaviors.

Diagnostic Schemes for Defining BPD in Youth

A number of attempts have been made to delineate youth-specific diagnostic criteria for defining borderline personality (e.g., Bemporad, Smith, Hanson, & Cicchetti, 1982; Cohen, Paul, & Volkmar1986; Cohen, Shaywitz, Young, & Shaywitz, 1983; Kernberg, 1982; Towbin, Dykens, Pearson, & Cohen, 1993; Vela, Gottlieb, & Gottlieb, 1983; see Ad-Dab'Bagh & Greenfield, 2001 and see also Chap. 3, Fossati, in the current volume for reviews). However, these proposals have resulted in few follow-up studies (e.g., Lofgren, Bemporad, King, Lindem, & O'Driscoll, 1991), and none of the criterion sets have been subjected to factor analysis. Rather, most investigations have simply relied upon adult diagnostic criteria for identifying BPD in youth. Mirroring the adult literature (see Linehan, 1993, Ch. 1), two diagnostic schemes have been utilized: (1) BPD as defined by the DSM criteria (versions III through IV; APA, 1980, 1987, 1994) and (2) as defined by the Diagnostic Interview for Borderlines-Revised (DIB-R; Zanarini, Gunderson, & Frankenburg, 1989). The latter includes several studies conducted by the research group at SMBD-Jewish General Hospital in Montreal, who used a chart review version of the DIB (Armelius, Kullgren, & Renberg, 1985; Greenman, Gunderson, Cane, & Saltzman, 1986) to examine what has been termed "borderline pathology of childhood" (e.g., Zelkowitz, Guzder, & Paris, 2001; Guzder, Paris, Zelkowitz, & Feldman, 1999; Guzder, Paris, Zelkowitz, & Marchessault, 1996; Zelkowitz et al., 2004; 2007). However, the majority of factor analytic studies of BPD criteria, both in adult and youth samples, have relied on the DSM-derived conceptions of BPD (reviewed below).

To date, only one study has factor analyzed the DIB-R in youth (Chabrol, Montovany, Callahan, Couicha, & Ducongé, 2002). The DIB-R is a semi-structured interview, consisting of 186 questions that tap four domains of functioning relevant to BPD: affect, cognition, impulsive behaviors, and interpersonal relationships. Information gathered from the interview questions are used to provide ratings for 22 summary statements that reflect BPD-relevant features (Zanarini et al., 1989). Chabrol, Montovany, et al. (2002) administered the DIB-R to a sample of 118 French high school students and subjected the summary scores to principal component analysis (PCA) and CFA. PCA results revealed three tenable solutions consisting of 1, 2, and 3 factors. CFA results favored the 3-factor structure, with moderate factor correlations (ranging from .45 to .60).

However, this finding should be viewed cautiously. The sample size was rather small, and results have not been replicated. Additionally, the three factors were conceptually messy. Factor 1 (labeled "affectivity/cognition") comprised ten features (depression, helplessness/hopelessness, anxiety, loneliness/emptiness, odd thinking/unusual perceptual experiences, quasipsychotic experiences, suicidal behavior, abandonment concerns, intolerance of aloneness, and dependency); factor 2 (labeled "impulsivity") comprised five features (substance abuse, sexual deviance, other impulsive actions, counterdependency, and stormy relationships); and factor 3 (labeled "aggressiveness in interpersonal relationships") comprised five features (anger, hypomania, devaluation/manipulation, demand/entitlement, and self-mutilation). At present, no studies in the adult BPD literature have examined the factor structure of the DIB or DIB-R (cf. Andión et al., 2011).

As such, a discussion of the factor structure of BPD in children and adolescence must necessarily be restricted to BPD as defined by the DSM criteria. In what follows, theoretical and empirical support for the multidimensionality of the DSM criteria will be reviewed. Subsequently, evidence favoring a unidimensional factor structure will be considered, and it will be suggested that the bulk of the empirical literature supports a unidimensional factor structure. Findings from both adult and youth literatures will be reviewed, but with greater attention to the latter. Finally, future areas for investigation will be suggested in service of clarifying our understanding of the dimensional structure of child and adolescent BPD.

Theoretical Support for Multidimensionality of DSM BPD

There are a number of theoretical reasons for presuming that the DSM-5 criteria for BPD measure a multidimensional construct. For one, conceptually, the nine criteria have been viewed as tapping multiple domains of dysregulation. Linehan (1993) organized the criteria into five such patterns: (1) emotional dysregulation (affective instability, inappropriate anger), (2) interpersonal dysregulation (unstable relationships, efforts to avoid abandonment), (3) behavioral dysregulation (suicidal/self-injurious behaviors, self-damaging impulsivity), (4) cognitive dysregulation (stress-related paranoid ideation/dissociation), and (5) self-dysregulation (identity disturbance, emptiness). Similarly, Lieb, Zanarini, Schmahl, Linehan, and Bohus (2004) suggested four domains of disturbance based on the finding that patients who exhibit symptoms in each of these areas can be adequately discriminated from other forms of personality disorder. Their four groupings included (1) affective disturbance (affective instability, inappropriate anger, emptiness), (2) disturbed cognition (stress-related paranoid ideation/dissociation, identity disturbance), (3) impulsivity (suicidal/self-injurious behaviors, self-damaging impulsivity), and (4) interpersonal disturbance (unstable relationships, efforts to avoid abandonment). Given such theoretically meaningful criterion groupings, it is reasonable to expect that certain groups of criteria correlate more strongly with one another versus others such that factor analysis would reveal that multiple factors best account for covariation among the DSM criteria.

Another reason to expect multidimensionality for the DSM BPD criteria comes from recent findings from longitudinal studies regarding the remission rate of personality disorders. BPD has been found to be less stable over time than what is expected from the DSM definition of personality disorder ("...an enduring pattern of...is pervasive and inflexible...is stable over time..."; APA, 2000, p. 685). In the Collaborative Longitudinal Personality Study (CLPS; Grilo et al., 2004), 42 % of participants originally diagnosed with BPD remitted at 2-year follow-up (defined as 2 consecutive months with two or fewer criteria met). In the McLean Study of Adult Development (MSAD; Zanarini, Frankenburg, Hennen, & Silk, 2003), 34.5 % of BPD participants remitted at 2-year follow-up (defined as no longer meeting either DIB-R or DSM-III-R diagnostic criteria). What may account for these remission rates are improvements in certain,

more acute BPD symptoms. Authors from both studies have concluded that the DSM personality diagnoses represent hybrids consisting of two components: (1) more stable personality traits (e.g., affective instability, inappropriate anger) and (2) intermittently expressed symptomatic behaviors, representing efforts to cope with, defend against, or compensate for pathological personality traits (e.g., self-injurious behaviors, efforts to avoid abandonment) (Skodol et al., 2005; Zanarini, Frankenburg, Hennen, Reich, & Silk, 2005). This distinction between stable and unstable aspects of personality disorders suggests additional, meaningful criterion groupings that might be supported by factor analysis.

Among children and adolescents, the likelihood of multidimensionality may be even greater. A series of studies conducted by the Yale Psychiatric Institute group compared BPD in adolescents to BPD in adults. The authors summarized their findings by stating that, among youth, BPD appears to constitute a more "diffuse range of psychopathology" (Becker et al. 1999, 2000, 2002, 2006). For example, inter-criterion correlations were examined across personality disorder categories. Whereas in adults the largest correlations for BPD were with respect to other Cluster B disorders (i.e., antisocial, histrionic, and narcissistic), in adolescents BPD criteria correlated more broadly with other personality disorder criteria (e.g., paranoid and dependent in addition to Cluster B disorders) (Becker et al., 1999). Similarly, when evaluating comorbidity frequencies for BPD in relation to other personality disorders, Becker et al. (2000) reported a broader pattern of co-occurrence for adolescents. Specifically, in the adolescent sample, BPD was significantly associated with schizotypal and passive–aggressive personality disorders, while in the adult sample BPD was significantly associated only with antisocial personality disorder. Adolescent BPD was also shown to exhibit greater diffusiveness with regard to the diagnostic efficiency (e.g., positive predictive power) of its symptom criteria compared to adults (Becker et al., 2002). Among adults, each BPD criterion performed at a similar level in predicting BPD diagnosis. However, among adolescents, certain criteria showed superior predictive capacity, indicating that the diagnostic efficiency of BPD criteria may have greater variability in adolescence. For these reasons, one might expect even greater heterogeneity to exist among the BPD criteria in youth, perhaps as evidenced by a greater number of factors underlying the criteria or by factor correlations of lesser magnitude than what has been reported in adults.

Factor Analytic Studies of DSM BPD

Adult Studies

Factor analytic investigations of BPD have a longer tradition in the adult literature compared to the child and adolescent literature. To date, 14 published studies have examined the factor structure of the BPD criteria with adult samples using a diagnostic interview (Aggen et al., 2009; Becker, Añez, Paris, & Grilo, 2010; Clarkin, Hull, & Hurt, 1993; Clifton & Pilkonis, 2007; Eaton et al., 2011; Feske et al., 2007; Fossati et al., 1999; Hallquist & Pilkonis, 2012; Johansen, Karterud, Pedersen, Gude, & Falkum, 2004; Nestadt et al., 2006; Rosenberger & Miller, 1989; Sanislow et al., 2000, 2002; Taylor & Reeves, 2007). Two others have been performed using retrospective clinician ratings (Blais, Hilsenroth, Castlebury, 1997; Rusch, Guastello, & Mason, 1992), and six studies have relied on self-report ratings of the diagnostic criteria (Benazzi, 2006; Chen, Zhong, Liu, & Lu, 2011; Gardner & Qualter, 2009; Selby & Joiner, 2009; Wang, Leung, & Zhong, 2008; Whewell, Ryman, Bonanno, & Heather, 2000). Because these studies have influenced the hypothesized factor structures tested in youth samples, they will be reviewed here briefly. Additionally, that certain factor structures have been replicated consistently in the adult literature adds credence to the robustness of findings in the youth literature that emulate these adult findings.

Across the 14 studies using structured interviews with adults, results were mixed with regard to dimensionality. However, the bulk of

studies supported either a 3-factor or unidimensional structure. Only three studies provided evidence of two factors (Eaton et al., 2011; Feske et al., 2007; Rosenberger & Miller, 1989), but in two of these the authors favored a single-factor solution in light overall goodness of fit, model parsimony, factor interpretability, and/or high factor correlations (Eaton et al., 2011; Feske et al., 2007). Of further note, the composition of the two factors was not consistent across these studies.

Four other studies favored a 3-factor solution (Clarkin et al., 1993; Sanislow et al., 2000, 2002; Taylor & Reeves, 2007). Of these, only the solution by Sanislow et al. (2000, 2002) has been replicated. Sanislow et al. (2000) conducted PCA with varimax rotation on DSM-III-R criteria in a sample of adult inpatients. The first factor ("disturbed relatedness") contained unstable relationships, identity disturbance, and chronic feelings of emptiness. The authors interpreted this factor as capturing a disturbed sense of self and relatedness to others, suggesting that it may represent the core personality features of BPD that underpin much of the symptomatic interpersonal behaviors seen in these patients. The second factor ("behavioral dysregulation") included the criteria of impulsivity and suicidal behaviors. Sanislow et al. viewed this factor as capturing *behaviors* typical of BPD patients, as opposed to *symptoms* or personality *traits*. The third factor ("affective dysregulation") consisted of affective instability, uncontrolled anger, and abandonment fears. They regarded this dimension as reflecting the physiological temperament of BPD patients—i.e., how they moderate their responses to stress.

Sanislow et al. (2002) replicated this 3-factor structure using CFA on the DSM-IV criteria. They hypothesized that the additional criterion (stress-related paranoid ideation/dissociation) represented in the newer edition of the DSM would load on the disturbed relatedness factor. Results supported the 3-factor structure, both when fitted to the baseline data and when fitted to the 2-year follow-up data. A χ^2 difference test suggested that the fit for the 3-factor model was significantly better than a unidimensional model. Sanislow et al.'s conceptualization has also been found to exhibit acceptable fit in three other patient studies using diagnostic interviews (Clifton & Pilkonis, 2007; Feske et al., 2007; Johansen et al., 2004).

Despite the above evidence in support of multiple dimensions underlying the BPD criteria, there is good reason to believe that a single dimension adequately accounts for the covariation among the criteria. For one, nine studies have supported a unidimensional solution, including both clinical (Becker et al., 2010; Clifton & Pilkonis, 2007; Feske et al., 2007; Fossati et al., 1999; Hallquist & Pilkonis, 2012; Johansen et al., 2004; Sanislow et al., 2002) and non-clinical samples (Aggen et al., 2009; Eaton et al., 2011). In four of these (Clifton & Pilkonis, 2007; Feske et al., 2007; Johansen et al., 2004; Sanislow et al., 2002), a unidimensional model was directly compared to Sanislow et al.'s 3-factor model. With one exception (Sanislow et al., 2002), authors of these studies favored the unidimensional model due to model parsimony and high factor correlations (r's $\geq$ .84). Even in Sanislow et al. (2002), factor correlations were notably high, nearing unity (r's ranged from .90 to .99); and RMSEA, an index of model fit that penalizes for lack of model parsimony (see Brown, 2006), favored the unidimensional solution.

Child and Adolescent Studies

Six published studies have examined the factor structure of the DSM criteria for BPD using youth samples (Table 4.1). Five of these studies assessed BPD using a diagnostic interview (Becker et al. 2006; Chabrol, Choicha, et al., 2002; Michonski et al., 2013; Sharp, Ha, Michonski, Venta, & Carbone, 2012; Speranza et al., 2012). The remaining study examined the factor structure in youth using a self-report measure of the BPD criteria (Leung & Leung, 2009). These will be described in detail below.

All but one of these studies (Becker et al., 2006) examined the structure of the nine DSM-IV criteria. Becker and colleagues performed

Table 4.1 Factor analytic studies of the DSM criteria for borderline personality disorder in youth

Study	Sample	Method	Results
Assessed via diagnostic interview			
Chabrol, Choicha et al. (2002)	60 French high school students	PCA with varimax rotation and CFA testing 1-, 2-, and 3-factor models, using DSM-IV criteria (SIDP)	PCA revealed three components. CFA favored a 1-factor model, which showed acceptable fit
Becker et al. (2006)	123 adolescent psychiatric inpatients	PCA with varimax rotation of DSM-III-R criteria (PDE)	Four components: (1) suicidal behaviors, emptiness; (2) affective instability, inappropriate anger, identity disturbance; (3) unstable relationships, fear of abandonment; (4) impulsivity, identity disturbance
Sharp et al. (2012)	245 adolescent psychiatric inpatients	Ordinal CFA testing 1-factor model, using DSM-IV criteria (CI-BPD)	1-factor model showed acceptable fit
Speranza et al. (2012)	107 outpatient adolescents with BPD symptomatology from Europe	PCA with promax rotation and CFA testing (uncorrelated) 3-factor model, using DSM-IV criteria (SIDP)	PCA revealed two components: (1) paranoid ideation, identity disturbance, emptiness, affective instability, fear of abandonment; (2) inappropriate anger, impulsivity, affective instability, suicidal behaviors, unstable relationships. CFA showed acceptable fit for 3-factor model (Sanislow et al., 2002)
Michonski et al. (2013) (see also Table 4.2)	6,339 11- to 12-year-olds from the general population in the United Kingdom	Graded-response IRT model (analogous to 1-factor CFA model), using DSM-IV criteria (CI-BPD)	1-factor models, fitted separately for girls and boys, both showed acceptable fit
Assessed via self-report			
Leung and Leung (2009)	5,224 (time 1) and 5,461 (time 2) high school students in Hong Kong	Five CFA models (1-factor, two 3-factor, and two 4-factor models) were fitted to ten items from MSI-BPD	All models showed acceptable fit. A modified 3-factor model based on Sanislow et al. (2002) and Lieb et al.'s (2004) was favored: (1) affective instability, inappropriate anger; (2) suicidal behaviors, impulsivity; (3) emptiness, identity disturbance, dissociation, unstable relationships, fear of abandonment, paranoid ideation

PCA using varimax (uncorrelated) factor rotation on the eight DSM-III-R criteria in a sample of 123 psychiatrically hospitalized adolescents. They regarded a 4-factor solution, with eigenvalues ranging from 2.47 to 0.90, as offering the most conceptual appeal (Table 4.1). They described these factors as representing (1) self-negation or depression, (2) affective dysregulation or irritability, (3) interpersonal dysregulation, and (4) impulsiveness. Notably, these four dimensions were found to exhibit relatively distinct patterns of association across Axis I conditions. For instance, factor 1 was the only factor associated with major depression and dysthymia; factor 2 was associated with anxiety and oppositional defiant disorders; and factor 4 was associated with conduct, oppositional defiant, alcohol use, and drug use disorders. Thus, conceptually this factor structure offers some appeal. However, results have not been replicated; their findings are limited by small size; and the authors did not employ robust model estimation procedures appropriate for ordinal items, which can result in underestimation of inter-item correlations, creation of "pseudofactors" that emerge as artifacts

Table 4.2 Goodness of fit statistics for three CFA models of DSM-IV BPD criteria in 11- to 12-year-old youth

Model	χ^2	*df*	$\Delta\chi^2$	Δ_{df}	RMSEA	CFI	TLI
Boys							
Model 1: unidimensional	140.22***	27			.037	.987	.982
Model 2: two factors (Feske et al., 2007)	132.08***	26	9.42**	1	.036	.987	.983
Model 3: three factors (Sanislow et al., 2002)	98.91***	24	38.88***	3	.032	.991	.987
Girls							
Model 1: unidimensional	126.85***	27			.034	.988	.985
Model 2[a]: two factors (Feske et al., 2007)	126.39***	26	1.03	1	.034	.988	.984
Model 3: three factors (Sanislow et al., 2002)	121.22***	24	7.90*	3	.035	.989	.983

[a]The latent variable covariance matrix was not positive definite (factor correlation exceeded 1.0)
*$p < .05$. **$p < .01$. ***$p < .001$

due to item difficulty, and incorrect estimation of test statistics and standard errors (see Brown, 2006, Ch. 9; Muthen & Kaplan, 1985).

Chabrol, Choicha, et al. (2002) examined the factor structure of DSM-IV BPD in a small sample of French high school students ($N = 60$). They conducted PCA with varimax rotation, as well as CFA comparing 1-, 2-, and 3-factor models. PCA results revealed three components with eigenvalues of 3.70, 1.06, and 1.01. All three CFA models showed acceptable fit. The authors favored the unidimensional model due to what they regarded as high factor correlations in the 2- and 3-factor models (r's ranging from .70 to .78). However, Chabrol et al. did not provide goodness of fit indices for the multidimensional models, thus making it difficult to evaluate the adequacy of fit for these two models. Further caution is warranted in interpreting their findings in light of their very small sample size and failure to employ robust model estimation procedures.

More recently, Speranza et al. (2012) conducted PCA using promax (correlated) factor rotation, as well as CFA to test an uncorrelated version of Sanislow et al.'s (2002) 3-factor model. In contrast to the two previous studies, Speranza appeared to address the ordinal response format of the criterion scores, using a polychoric correlation matrix (appropriate for ordered categorical indicators; see Brown, 2006, Ch. 9) as input for the factor analyses, rather than treating the criteria as continuous variables. Their sample consisted of 107 outpatient adolescents in Europe exhibiting BPD symptomatology. PCA revealed two components that the authors interpreted as reflecting internally and externally oriented criteria. For instance, paranoid ideation, identity disturbance, and chronic emptiness loaded on the first component (internalizing); whereas inappropriate anger, self-damaging impulsivity, suicidal/self-injurious behavior, and unstable relationships loaded on the second component (externalizing). CFA results showed acceptable fit for the 3-factor model. Both the PCA and CFA results provide support for BPD as a multidimensional construct in adolescents. However, as with the previous two studies, their findings are limited by small sample size. Additionally, Speranza et al. did not test a unidimensional CFA model; thus, the relative superiority of the 3-factor over a single-factor model could not be determined. Furthermore, that the three factors would be uncorrelated is difficult to reconcile with previous findings showing very high factor correlations (Clifton & Pilkonis, 2007; Feske et al., 2007; Johansen et al., 2004; Sanislow et al., 2002).

In a larger sample of inpatient adolescents ($N = 245$), Sharp et al. (2012) tested a single-factor CFA model for the BPD criteria. Sharp et al. appropriately addressed the ordinal response format of the criterion scores, using a robust model estimation procedure and polychoric correlation matrix as input. The 1-factor model showed acceptable fit. As additional support for unidimensionality, the standardized factor loadings were substantial (exceeding .60), and the magnitude of the first eigenvalue (4.58) extracted from the polychoric correlation matrix

was large relative to subsequent eigenvalues (1.08 and 0.93 for the second and third eigenvalues, respectively). However, this study did not test for alternative, multidimensional solutions and, thus, cannot speak as directly to the superiority of a single-factor over multifactor solutions.

One final study to date has examined the factor structure of BPD in youth as assessed via diagnostic interview (Michonski, 2011a, 2011b). This study directly compared unidimensional and multidimensional models using a nationally representative sample ($N = 6{,}339$) of 11–12-year-old children in Great Britain. A portion of this dissertation work was recently published, emphasizing the item response theory (IRT) analyses (Michonski et al., 2013). Additional analyses not reported in Michonski et al. (2013), however, will be presented here that speak more explicitly to the possibility of multidimensional factor structure.

Three ordinal CFA models using a robust estimation procedure (WLSMV) were fitted in MPlus (Muthen & Muthen, 1998–2010). These included a unidimensional model, a 2-factor model based on Feske et al. (2007) shown to exhibit good fit, and Sanislow et al.'s (2002) 3-factor model, which has been the most replicated multidimensional model in the adult literature (Table 4.2). Models were fitted separately for boys and girls. With the exception of the 2-factor model for girls, all models exhibited excellent fit, as evidenced by global indices of goodness of model fit (CFI and TLI $> .95$; RMSEA $< .05$). The best fitting model for both genders was Sanislow et al.'s 3-factor solution, as evidenced by significant χ^2 difference tests. However, other results favored unidimensionality. Factor correlations were notably high. For boys, the correlation between the disturbed relatedness and behavioral dysregulation factors was .85, between the disturbed relatedness and affective dysregulation factors was .93, and between the behavioral dysregulation and affective dysregulation factors was .97. For girls, the correlation between the disturbed relatedness and the behavioral dysregulation factors was .89, between the disturbed relatedness and affective dysregulation factors was .98, and between the behavioral dysregulation and affective dysregulation factors was .94. As further evidence of a unidimensional conceptualization, the magnitude of the first eigenvalue (5.29 for boys; 5.32 for girls) relative to the second eigenvalue (0.78 for boys; 0.78 for girls) extracted from the polychoric correlation matrix was substantial for both genders, and standardized factor loadings were high (.61 to .81 for boys; .58 to .83 for girls).

One additional study examined the factor structure of BPD in adolescents (Leung & Leung, 2009). Unlike those reviewed above, this study employed a self-report measure of the DSM-IV criteria. Leung and Leung (2009) administered a Chinese version of the McLean Screening Instrument for BPD (MSI-BPD; Wang et al., 2008; Zanarini, Vujanovic, et al., 2003) to a large sample of high school students in Hong Kong (aged 12–20), collected over two time points ($N = 5{,}224$ and 5,461, respectively). In contrast to interview-based measures of DSM-IV BPD, which provide individual scores for each of the nine criteria, the MSI-BPD contains ten item scores. The additional item results from breaking up the stress-induced paranoid ideation or dissociation criteria into two questions: (a) "Have you often been distrustful of other people?" and (b) "Have you frequently felt unreal or as if things around you are unreal?" They subjected five different models to CFA: a unidimensional model, two 3-factor models based on Sanislow et al. (2002), and two 4-factor models based on Lieb et al.'s (2004) conceptual groupings.

All five models exhibited adequate fit at both time points. The unidimensional model was found to provide the worst overall fit in terms of global goodness of fit indices. The best fitting solution was a modified version of Lieb et al.'s (2004) 4-factor model, with the following structure: (1) affective disturbance (affective instability, inappropriate anger), (2) impulsivity (suicidal/self-injurious behaviors, self-damaging impulsivity), (3) self-disturbance (emptiness, identity disturbance, dissociative symptoms), and (4) interpersonal disturbance (unstable

relationships, efforts to avoid abandonment, paranoid ideation). Factor correlations for this model ranged from .76 to .97, with the highest correlation between the self-disturbance and interpersonal disturbance factors. A modified 3-factor model in which these two factors were combined was found to be the second best-fitting model and offered a more parsimonious solution. Factor correlations for this model ranged from .76 to .83.

Summary of Factor Analytic Findings

Taken together, several general conclusions can be made regarding the factor structure of BPD as defined by the DSM criteria. First, across both the adult and youth literatures, there is more support for a single-factor solution than for any other factor model. In youth, four of six studies reported an acceptably fitting unidimensional solution (Chabrol, Choicha, et al., 2002; Leung & Leung, 2009; Michonski et al., 2013; Sharp et al., 2012; see also Table 4.2), and the remaining two studies did not directly test the adequacy of a unidimensional model (Becker et al., 2006; Speranza et al., 2012). Further, in studies where multidimensional models showed superior fit, factor correlations tended to be high, in some cases nearing unity (Johansen et al., 2004; Sanislow et al., 2002). Thus, a unidimensional model appears to offer the most parsimonious conceptualization, as has been advocated by several other authors (Clifton & Pilkonis, 2007; Feske et al., 2007; Johansen et al., 2004).

In addition to a 1-factor solution, one other model has been replicated within both adult and youth samples—Sanislow et al.'s 3-factor model. Previous authors have suggested that such findings need not necessarily be viewed as incompatible (Sanislow et al. 2002; Skodol et al., 2005). Whereas a good-fitting unidimensional model indicates that the BPD criteria define a unitary diagnosis, a good-fitting multidimensional model highlights factors that may reflect the heterogeneity that has been observed among BPD patients. This notion, although appealing, warrants some caution. For one, the multidimensional models reported thus far tend to be limited in their conceptual tightness. For instance, the loading of abandonment fears along with affective instability and inappropriate anger in Sanislow et al.'s (2000, 2002) model is theoretically confusing, especially when considering that affective instability and inappropriate anger have been found to be the most stable BPD criteria, while abandonment fears has been found to be the least stable (McGlashan et al., 2005). Second, the magnitude of the factor correlations that have been reported makes it difficult to reconcile parsing the BPD criteria.

However, results from factor analysis of the MSI-BPD in a sample of Chinese adolescents (Leung & Leung, 2009) may offer some promise for identification of an empirically and conceptually tenable multidimensional model. As reviewed above, these authors found support for a factor model that resembles the conceptually derived criterion groupings suggested by Lieb et al. (2004). In their refitted 3-factor model, factor correlations were not excessively high (i.e., unique variance in the factors ranged from .31 to .42). Similar results were reported in clinical and non-clinical samples of Chinese adults (Chen et al., 2011; Wang et al., 2008).

Future Research

The above review reveals several areas for further investigation. First, the addition of criteria for defining BPD may aid discovery of multiple dimensions that many presume underlie the DSM criteria (e.g., Lieb et al., 2004; Linehan, 1993; Paris, 2007). Recall that findings from the one study to factor analyze the DIB-R criteria for BPD (which consists of a larger criterion set) favored a 3-factor model, with only moderate factor correlations (Chabrol, Montovany, et al., 2002). Moreover, factor analyses of the MSI-BPD, which contains ten instead of nine items, have supported multiple dimensions (Chen et al., 2011; Leung & Leung, 2009; Wang et al. 2008; cf. Gardner & Qualter, 2009). These findings suggest that the addition of a single criterion

may enable empirically and conceptually sound multifactor CFA models. However, further research is needed to rule out whether these findings are attributable to self-report administration, are unique to Chinese samples, or are specific to the MSI-BPD.

Another area for future research that may uncover the supposed heterogeneity of BPD is to fit the criteria to more complex latent variable models. One possibility is to consider bifactor models, which have shown tenability with other personality disorders (e.g., psychopathy; Patrick, Hicks, Nicohl, & Krueger, 2007). Another possibility is to consider factor mixture models, a hybrid approach that combines categorical (latent class analysis) and continuous (CFA) latent variables (Lubke & Muthén, 2005), as was recently done to investigate the latent structure of BPD in adults (Hallquist & Pilkonis, 2012). Hallquist and Pilkonis found that the BPD criteria were best represented by a hybrid model in which the criteria measured a single latent dimension but where two latent classes (symptomatic and asymptomatic) were distinguished by their locations (i.e., mean differences) along this latent dimension. Furthermore, they discovered that meaningful subtypes of BPD could be identified but that doing so required incorporating additional indicators of psychopathology beyond the DSM criteria. This finding is consistent with the view that BPD is heterogeneous but "that the search for variability of BPD should move beyond the DSM diagnostic criteria to examine a range of alternative interpersonal and emotional constructs" (Clifton & Pilkonis, 2007, p. 77).

A third—and probably most important—area for future investigation is to identify childhood and adolescent indicators of borderline pathology. As reviewed above (see also Chap. 2, Fossati, in the current volume), few attempts have been made to delineate childhood and adolescent descriptions of borderline pathology. All factor analytic investigations to date have relied on criteria that reflect downward extensions of adult BPD criteria (i.e., the DSM and, to a much less extent, the DIB-R).

Furthermore, researchers who identify good-fitting multifactor conceptualizations of BPD criteria should seek to further substantiate the validity of the separate factors by evaluating them with respect to external correlates. If the multiple factors are truly meaningfully separable, then they should exhibit unique patterns of correlations with external criteria. Sanislow et al. (2002) made this suggestion over a decade ago, but few studies have responded to the call (Becker et al., 2006; Chmielewski, Bagby, Quilty, Paxton, & McGee Ng, 2011). Becker et al. (2006) examined four BPD components identified using PCA in relation to Axis I pathology. Using an extended criterion set (15 items) assessed via self-report, Chmielewski et al. (2011) evaluated the three factors prescribed by Sanislow et al. (2002) with respect to normal personality traits. In line with these studies, future research might test the utility of separate factors by examining their associations with such variables as Axis I pathology, personality traits, functional impairment, social adjustment, substance abuse, conduct problems, responsiveness to different treatments, and change over time.

Finally, although the DSM-IV BPD criteria remain unchanged in DSM-5, Part III of DSM-5 presents an alternative model in which personality disorders are represented as a hybrid of categorical and dimensional elements (see Chap. 27). This alternative model may alter the way BPD is conceptualized in the future and, therefore, its factor structure. Future research will need to examine the factor structure of BPD as defined by the criteria outlined in Part III. Differences from what has been reported in the present review may emerge given the proposed changes to the criterion set. For example, "anxiousness" and "depressivity" have been added, while self-harming behavior has been collapsed into a more general feature of "impulsivity" that reflects acting without planning. However, considering that Part III defines BPD according to only seven pathological personality traits, statistically it may be unlikely for more than one factor to be found to account for the covariation among these traits. Nonetheless, factor analysis may prove useful in helping to refine the criterion set, thereby informing our understanding of BPD in subsequent editions of the DSM.

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Suggested Reading

Brown, T. A. (2006). *Confirmatory factor analysis for applied research*. New York: Guilford Press. (A reader-friendly book on CFA. Provides sample code and examples using multiple software packages.)

Hallquist, M. N, & Pilkonis, P. A. (2012). Refining the phenotype of borderline personality disorder: Diagnostic criteria and beyond. *Personality Disorders: Theory, Research, and Treatment, 3,* 228–246. (An example of moving beyond factor analysis to more complex latent variable models. In spite of its statistical sophistication, the article is still fairly readable.)

Michonski, J. D., Sharp, C., Steinberg, L., & Zanarini, M. C. (2013). An item response theory analysis of the DSM-IV borderline personality disorder criteria in a population-based sample of 11- to 12-year-old children. *Personality Disorders: Theory, Research, and Treatment,* 4(1), 15–22. (Application of a latent trait

model (IRT) in a young adolescent sample that is analogous to a 1-factor CFA model and that goes beyond merely evaluating factor structure (i.e., considers item difficulty, item discrimination, and measurement invariance across gender).)

Sanislow, C.A., Grilo, C.M., Morey, L.C., Bender, D.S., Skodol, A.E., Gunderson, J.G., et al. (2002). Confirmatory factor analysis of DSM-IV criteria for borderline personality disorder: findings from the Collaborative Longitudinal Personality Disorders Study. *American Journal of Psychiatry, 159*, 284–290. (Provides evidence for a 3-factor model of the BPD criteria that has substantially influenced subsequent factor analytic investigations of BPD.)

Conceptualizing Youth Borderline Personality Disorder Within a PAI Framework

5

Leslie C. Morey and Justin K. Meyer

The assessment of features related to borderline personality disorder (BPD) among youth represents an interesting challenge, as is made apparent by the extensive discussion of important maturational and developmental aspects of these features presented throughout this volume. Many of these challenges are common to other efforts to assess personality and psychopathology constructs among children and adolescents. For example, strategies that may be suitable for the assessment of a 30-year-old, such as self-report questionnaires that describe psychological phenomena and nuanced emotions, are likely to be of questionable applicability to a 4-year-old. As a result, problems in younger children are typically assessed with behavior checklists which are completed by informants such as parents, teachers, or observers. In contrast, most commonly used assessments of problems in adulthood have self-report at their core, using information gathered either through questionnaire or through structured interview. In adolescence, there tends to be a transition between approaches, as both informant-based and questionnaire-based methods are commonly used in this age range.

While part of the popularity of self-report measures lies in their convenience and simplicity of use, it is also the case that obtaining information about the phenomenology of the individual being evaluated is central to many forms of mental disorder, including BPD. Examining diagnostic criteria for BPD reveals that most of these center around disruptions in the experience of the individual—confusion around identity, unstable perceptions of others, feelings of emptiness, or boredom (American Psychiatric Association, 1994). Self-report questionnaires provide a highly standardized and reliable means with which to assess these experiences, and accordingly are popular techniques for use with respondents throughout most of the life span. However, it is important to examine the suitability of such techniques for use with youth, and this chapter provides such an examination with respect to one such personality questionnaire—the Personality Assessment Inventory (PAI: Morey, 1991; Morey, 2007a, 2007b). The chapter will first provide a broad overview of the PAI and related measures that are particularly salient for assessment of children and adolescents. Then, it will examine research applications of the PAI in this population, to provide an indication of the utility of this approach for assessing constructs of particular relevance to BPD in this age range.

The Personality Assessment Inventory: An Overview

The PAI is a self-report questionnaire, first published in 1991, that was designed to provide

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psychological assessment information pertaining to psychopathology, personality, and psychosocial environment. As an inventory, it has broad coverage of several important psychological constructs, including BPD, that make it applicable to diverse types of settings. As a result, surveys of practicing clinicians indicate that it has become one of the most popular clinical measures in mental health, forensic/correctional, screening, and training contexts (Archer, Buffington-Vollum, Stredy, & Handel, 2006; Lally, 2003).

The PAI includes 344 questions that can be completed either with paper and pencil or administered via computer. Typically completion time for the PAI is between 45 and 60 min, although this can vary depending upon the clinical presentation. The respondent provides answers to questions on a 4-point scale that ranges from "totally false, not at all true" to "very true." The response selected corresponds to an item score (ranging from 0 to 3), and these scores are summed into total scores. These items are arranged into 22 full scales: 4 Validity scales, 11 Clinical scales, 5 Treatment scales, and 2 Interpersonal scales. Ten of the scales contain conceptually driven subscales designed to facilitate interpretation and coverage of the full breadth of complex clinical constructs. These scales are described in more detail in the succeeding section.

The original PAI is designed for use with individuals aged 18 and older; as will be described below, there is an Adolescent Version (PAI-A: Morey, 2007b) that includes items and norms suited for use with those aged 12–18. Because the PAI is a self-report instrument, it requires that the respondent be capable of understanding written test items and understand the use of the response scale. Analyses of the PAI and PAI-A items indicate that the statements are worded at a fourth-grade reading level, which is lower than comparable psychopathology inventories (Schinka & Borum, 1993). As a result, there are unlikely to be age-specific constraints related to reading comprehension among adolescent respondents, although such problems might be found in particular adolescents (or adults). Even so, the valid administration of the PAI or PAI-A assumes that respondents are capable of meeting the demands associated with completing a self-report instrument. Thus, care should be taken in testing clients who, particularly given the nature of BPD, tend to be highly distractible, confused, disoriented, or might be manifesting extreme psychomotor retardation or agitation. Particular caution should be exercised in testing a client whose native language is not English because educational attainment and even apparent spoken English fluency may bear little relationship to reading level. The instruments do include validity scales that are designed to assist in determining whether the profile validly represents client experience. Such validity scales are often useful in detecting factors that might distort the test results in patients with prominent features of BPD.

The raw scores for the PAI and PAI-A scales and subscales are transformed to *T*-scores (mean of 50, standard deviation of 10) in order to provide interpretation relative to standardization samples of community-dwelling respondents. For the original PAI, this involved a sample of 1,000 adults aged 18–89 that was selected to match 1995 US census characteristics on the basis of gender, race, and age. That sample had an educational level of the standardization sample (mean of 13.3 years) designed to be representative of a community group with the required fourth-grade reading level. For the PAI-A, the standardization sample involved a national census-matched community sample from the United States that included 707 adolescents, stratified in different age bands by race and gender using 2003 US Census Department data. For both the PAI and PAI-A, the *T*-scores for each scale and subscale are linear transformations using the means and standard deviations derived from the respective standardization samples. Unlike several similar instruments, the PAI does not calculate *T*-scores differently for gender, instead using combined norms. In developing the PAI, item selection parameters included several procedures intended to eliminate items that might be biased due to demographic features, and items that displayed any signs of being interpreted differently as a function of these features were eliminated in the course of selecting final items for the test. As it turns out,

with relatively few exceptions, differences as a function of demography were negligible in the community sample. Of particular note, no gender differences are found on the PAI Borderline Features (BOR) scale in community samples (Morey, 2007a).

Personality Assessment Inventory-Adolescent (PAI-A)

The introduction of the original PAI resulted in considerable use in personality and psychopathology assessment, including hundreds of studies in college-age samples facilitated by the availability of college student norms for the PAI (Morey, 1991). Some researchers felt that the structure and constructs represented by the PAI were also well-suited to assessment in early adolescence, and studied the utility of the instrument despite the absence of norms for this age range (e.g., Hoekstra, 1998). As such evidence mounted, the PAI-A grew out of the expressed interest of many professionals who wished to use the PAI with adolescents in clinical settings. The intent of the development work on the PAI-A was to provide an instrument that would closely parallel the adult version of the inventory, rather than develop an entirely new instrument targeted specifically at an adolescent population. As a result, the PAI-A attempts to retain the structure and—as much as possible—the items of the adult inventory and to demonstrate clinical utility for adolescents in various settings.

The PAI-A is a 264-item questionnaire that was developed and standardized for use in the clinical assessment of adolescents 12–18 years of age. For those individuals aged 18 years, appropriate norms are available for either the PAI-A or the PAI adult version. The latter test may be more informative for those 18-year-olds who are no longer in school and/or who live outside their parental home. As noted above, the clinical constructs assessed by the PAI-A are identical to those of the PAI; these constructs were selected on the basis of the stability of their importance within the nosology of mental disorder and their significance in contemporary diagnostic practice, not because they were specifically pertinent to diagnostic concepts applicable only to adolescents. Rather, the constructs assess experiences (e.g., suicidal ideation, depression, anxiety) that are expressed with reasonable consistency across the lifespan. In some instances, PAI-A items were revised from the PAI to be particularly applicable to the experiences of adolescents, but similar results would be anticipated from the application of either instrument if applied to respondents close to the 18-year cutoff for the two instruments.

Given the structural similarity of the PAI-A to the PAI, the largest distinction between the two instruments involves the age of the normative reference sample. For the parent instrument, it had been established that there were age differences observed with some of the scales, with ANT, AGG, BOR, and PAR demonstrating the largest effects across the 18–89 age range sampled by the PAI (Morey, 1991). However, use of an adolescent normative sample for the PAI-A provides a reference point for respondents in this age range, thus addressing such normative age differences. Of perhaps greater interest for the development of the PAI-A than such normative differences was the assumption that responses to these items would carry similar implications for both adults and adolescents. Thus, items were examined for differential item functioning (e.g., Holland & Wainer, 1993) across adolescent and adult samples, and PAI items that appeared to have different characteristics for adolescents than for adults were eliminated from the final version of the PAI-A. This process eliminated 80 items and reduced the 344-item PAI to the 264-item PAI-A.

PAI/PAI-A Areas of Assessment

As noted previously, the PAI and PAI-A (which are comprised of the same scales) are organized into four broad sections. The *Validity* scales were developed to provide an assessment of the potential influence of certain response tendencies on PAI test performance, including both random and systematic influences upon test responding. The *Clinical* scales measure a variety of important

diagnostic and psychopathological constructs, and most include subscales that measure the core facets of these constructs. The *Treatment Consideration* scales measure various factors central in treatment planning, such as gauging treatment motivation and intensity and type of intervention needed. Finally, the *Interpersonal* scales provide a more normative (i.e., independent of psychopathology) description of the respondent's characteristic style of relating to others. The following paragraphs provide a brief description of the scales included in each of these sections.

Validity scales. The assessment of profile validity is an important component of any method intended for use in an evaluative context, but it is particularly relevant for assessment of borderline personality features. The PAI validity scales were developed to provide an assessment of the potential influence of certain response tendencies on PAI test performance, including both random and systematic influences upon test responding. The extent to which such scales and indicators are useful for clarifying interpretation of psychological tests has been a point of some controversy (McGrath et al., 2010; Morey, 2012), but there is substantial evidence supporting utility of the various PAI validity markers; for example, a meta-analysis of negative distortion detection research (Hawes & Boccaccini, 2009) concluded that all reviewed negative distortion indicators on the PAI demonstrated large average effect sizes across several studies.

Non-systematic or random response influences on the PAI can be identified by elevations on the ICN and INF scales. Such influences might include noncompliance or random responding, idiosyncratic interpretation of items, reading or language comprehension problems, or confusion, perhaps associated with organicity, psychosis, or the effects of intoxication or detoxification. If there is significant non-systematic distortion in the profile, the results are unlikely to be interpretable and it is likely that the participant will need to be assessed with methods other than self-report questionnaire.

Systematic profile distortion involves response patterns that affect the test results in predictable ways, and can occur in positive or negative directions, and even in both directions in a given profile (with different scales influenced by different factors). Such distortion can also be either effortful/intentional or non-effortful. Effortful distortion occurs when respondents intentionally present themselves in a manner that is at odds with their experience or historical fact (i.e., they might malinger, or fake good). In non-effortful distortion, respondents may present themselves in manner consistent with their subjective experience, but which an experienced clinician might see as an exaggeration (if overly negative) or a lack of insight (if overly positive). Such distortion can be related to specific forms of psychopathology. For example, cognitive symptoms of depression often involve a magnification of personal difficulties, while narcissistic or manic disorders can manifest limited capacity for a critical appraisal of one's real abilities and prospects. PAI indices of systematic profile distortion were developed to assess self- and other-deception, with the PIM scale measuring distortions in a positive direction and NIM assessing negative distortions.

The detection of negative profile distortion is particularly salient for the assessment of borderline personality because such individuals often present in a crisis state, overwhelmed by current and recurring stresses. This can lead to considerable negative distortion in the self-report as a "cry for help" that can complicate interpretation—leading to elevated scores on nearly every indicator of psychopathology. Such negative coloration of experience may well be a central feature of borderline pathology (Kurtz & Morey, 1998), but it can also lead to false positive results where such patients endorse virtually any negative symptom about which they are asked. In fact, elevations on these types of negative distortion indicators on the MMPI, such as the F scale, are one of the most characteristic features of the borderline MMPI profile (Morey & Smith, 1988). Elevations on NIM are also common in adult borderline patients on the PAI (Morey, 1991), underscoring the need to evaluate self-reports of severe and diverse psychopathology with caution in this population.

Substantive scales. The remaining PAI scales, shown in Table 5.1, were assembled to provide information about clinical and personality constructs relevant in a variety of different contexts. Of the clinical scales, the BOR scale is most obviously salient for the assessment of borderline features in youth (i.e., PAI-A BOR) and adults (i.e., PAI-BOR). Although personality disorders in general tend to become apparent during adolescence, personality disorder diagnoses in youth are typically made cautiously to be certain that any issues observed are not limited to a particular developmental stage. For BPD, issues such as identity concerns that are salient for the disorder may be encountered in typical adolescents and young adults, and normative data on the PAI does suggest that elevations on the BOR scale are more frequent in respondents in the 18–25 year range (Morey, 1991). Furthermore, studies have noted that the features of BPD are fairly unstable over time during adolescence (Johnson et al., 2000), although similar observations have been noted for adults (Gunderson et al., 2011) and this instability may be characteristic of the features themselves rather than reflecting a developmental process. In either case, it is important to note that the PAI-A BOR scale does not demonstrate a significant association with age for the period from 12 to 18 years (Morey, 2007b). As such, it appears that the items of the BOR scale are tapping maladaptive variants of issues related to identity and interpersonal behavior, rather than experiences that are developmentally normative for adolescents (which would be accounted for in the norming of the PAI-A).

The full scale score on BOR can considered an indicator of a certain global immaturity in one's approach to the world with average scores reflecting a person who reports being emotionally stable and who also has stable relationships with partners, friends, and family. Scores that are 1–2 standard deviations above community norms are indicative of an adolescent who may be seen as moody, sensitive, and having some fundamental uncertainty about life goals that exceeds the normal ambiguity of adolescence. As scores become markedly elevated, the severity of these issues increasingly reflects a level of personality functioning within the borderline range. Such youth are typically in a state of crisis, often regarding difficulties in relationships. There is considerable hostility and feelings of betrayal by others. Symptomatically, such individuals report being very depressed and anxious in response to current and ongoing circumstances. The respondent will be impulsive, acting in ways that appear to others to be self-destructive, seeming to sabotage his or her own best intentions with acting-out behaviors that might include alcohol or drug abuse, risky sexual behavior, suicidal gestures, or aggressive outbursts.

The BOR scale includes four subscales assessing different core elements of borderline personality functioning. These subscales are (a) Identity Problems (BOR-I), tapping uncertainty about self-definition and a poorly integrated concept of both the self and of significant others; (b) Affective Instability (BOR-A), assessing poor modulation of affective reactions, resulting in intense emotions such as despair, panic, and rage; (c) Negative Relationships (BOR-N), assessing interpersonal ambivalence and a mistrust of intimacy, marked by the concurrent existence of profound dependency on other people, combined with an expectation of abandonment or exploitation by these same people; and (d) Self-Harm (BOR-S), that taps the tendency to engage in impulsive, self-destructive behaviors. Each of these reflected conceptually distinct but correlated features of borderline personality, and each may reflect a particular presumed etiological pathway. As an example, using twin studies Distel et al. (2010) not only estimated a heritability of 0.42 for the full scale BOR scale but also identified genetic effects specific to each of the subscales, particularly for BOR-S.

The BOR scale has been widely studied in a variety of domains related to borderline personality functioning, including depression, personality traits, coping, Axis I disorders, and interpersonal problems; some of this research as particularly relevant to the assessment of youth will be described later in this chapter. At this point, it is important to note that BOR elevations have demonstrated strong diagnostic efficiency against

Table 5.1 PAI scales and subscales

Scale		Content measured
Validity scales		
ICN	Inconsistency	Inconsistent responding to similar items
INF	Infrequency	Idiosyncratic responses to items
NIM	Negative impression management	Negative response set due to cognitive distortions and/or feigning of psychopathology
PIM	Positive impression management	Positive response set due to lack of insight and/or intentional dissimulation
Clinical scales		
SOM	Somatic complaints	
SOM-C	Conversion	Rare sensorimotor symptoms associated with conversion disorders
SOM-S	Somatization	Frequent, common physical symptoms or vague complaints of ill health or fatigue
SOM-H	Health concerns	Preoccupation with physical functioning
ANX	Anxiety	
ANX-C	Cognitive	Ruminative worry, impaired concentration and attention
ANX-A	Affective	Tension, difficulty relaxing, nervousness, and fatigue
ANX-P	Physiological	Physical signs such as sweating, tremors, palpitations
ARD	Anxiety-related disorders	
ARD-O	Obsessive-compulsive	Intrusive thoughts, compulsive behaviors, perfectionism, affective constriction
ARD-P	Phobias	Common phobic fears
ARD-T	Traumatic stress	Enduring effects of trauma exposure
DEP	Depression	
DEP-C	Cognitive	Worthlessness, hopelessness, difficulty concentrating
DEP-A	Affective	Feelings of sadness, dysphoria
DEP-P	Physiological	Lowered drive, disruptions in sleep and eating patterns
MAN	Mania	
MAN-A	Activity level	Disorganized over-involvement, accelerated behavior
MAN-G	Grandiosity	Inflated self-esteem, expansiveness
MAN-I	Irritability	Impatience, low frustration tolerance
PAR	Paranoia	
PAR-H	Hypervigilance	Tendency to closely monitor environment for threat
PAR-P	Persecution	Belief that others intentionally obstruct respondent
PAR-R	Resentment	Bitterness and cynicism, externalization of blame
SCZ	Schizophrenia	
SCZ-P	Psychotic experiences	Unusual perceptions and ideas, magical thinking
SXZ-S	Social detachment	Social isolation, discomfort, and awkwardness
SCZ-T	Thought disorder	Confusion, concentration difficulties, and disorganization
BOR	Borderline features	
BOR-A	Affective instability	Poor modulation of emotional responses
BOR-I	Identity problems	Uncertainty about major life issues, lack of purpose
BOR-N	Negative relationships	History of intense, ambivalent relationships
BOR-S	Self-harm	Impulsivity with disregard for negative consequences
ANT	Antisocial features	
ANT-A	Antisocial behaviors	History of antisocial and illegal behavior
ANT-E	Egocentricity	Lack of empathy, exploitative approach to relationships
ANT-S	Stimulus-seeking	Cravings for excitement, low boredom tolerance, recklessness

(continued)

Table 5.1 (continued)

Scale		Content measured
ALC	Alcohol problems	Use of and problems with alcohol
DRG	Drug problems	Use of and problems with drugs
Treatment consideration scales		
AGG	Aggression	
AGG-A	Aggressive attitude	Hostility, easily aroused anger
AGG-V	Verbal aggression	Assertiveness, readiness to express anger to others
AGG-P	Physical aggression	Tendency and history for physical aggression
SUI	Suicidal ideation	Frequency and intensity of thoughts of self-harm or suicide
STR	Stress	Perception of an uncertain and unstable environment
NON	Nonsupport	Perception that others are not available or willing to provide support
RXR	Treatment rejection	Low motivation for treatment, little readiness to change
Interpersonal scales		
DOM	Dominance	Desire and tendency for control in relationships; low scores suggest meekness and submissiveness
WRM	Warmth	Interest and comfort with close relationships; low scores suggest hostility, anger, or mistrust

diagnoses of BPD established using other methods such as structured interviews (Bell-Pringle, Pate, & Brown, 1997; Distel, Hottenga, Trull, & Boomsma, 2008; Jacobo, Blais, Baity, & Harley, 2007; Stein, Pinsker-Aspen, & Hilsenroth, 2007). Furthermore, BOR has also been shown to be a sensitive measure of response to treatments targeting BPD, such as Dialectical Behavior Therapy (Harley, Blais, Baity, & Jacobo, 2007; Stepp, Epler, Jahng, & Trull, 2008) or Manual-Assisted Cognitive Therapy (Morey, Lowmaster, & Hopwood, 2010). Although there have been fewer diagnostic studies using the adolescent version, available data suggest similar conclusions for the PAI-A. For example, Ha, Sharp, Michonski, Venta, and Carbone (2011) demonstrated that the PAI-A BOR scale, and all four of its subscales, demonstrated large effects in distinguishing adolescent inpatients (ages 12–17) diagnosed with the Childhood Interview for BPD (CI-BPD: Zanarini, 2003) from youth from the same unit receiving other diagnoses. Similar analyses from the same group demonstrated a correlation of 0.66 between PAI-A BOR and the continuous symptom count score resulting from the CI-BPD interview (Sharp, Ha, Michonski, Venta, & Carbone, 2012). Such studies demonstrate that the BOR scale from the PAI instruments provide a solid assessment of both diagnostic features and of treatment response.

One interesting application of the BOR scale for assessment of borderline features in youth was begun by Crick, Murray-Close, and Woods (2005), who developed an adaptation of the PAI-BOR scale, called the Borderline Personality Features Scale for Children (BPFS-C), in consultation with the author of the PAI. This scale modified each of the 24 items of the PAI-BOR scale to include age-appropriate wording for children ages 9 and older, but thus still assessing the four subscale components of the full scale. In the original (Crick et al. 2005), the internal consistency of the BPFS-C was high, exceeding 0.76 at three different assessment intervals. Subsequent research has confirmed the diagnostic utility of the BPFS-C gauged against diagnoses assigned to youth using structured interviews. For example, Chang, Sharp, and Ha (2011) administered the Child Interview for DSM-IV BPD (CI-BPD: Zanarini, 2003) to an inpatient sample of adolescents ranging from ages 12–18 and found that the BPFS-C had high accuracy (Sensitivity of 0.856; Specificity of 0.840) referenced against structured interview results. Chang et al. (2011) also examined a parent-report version of the BPFS, and found that it correlated reasonably

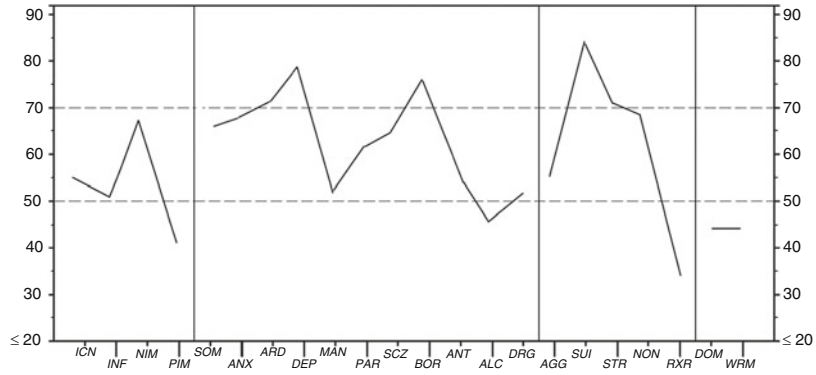

Fig. 5.1 Mean profile for borderline patients, adapted from Bell-Pringle et al. (1997)

well with the child self-report BPFS ($r = 0.687$) but was less accurate with respect to CI-BPD diagnosis (Se = 0.733; Sp = 0.720). Both parent (alpha = 0.885) and child (alpha = 0.892) versions demonstrated adequate internal consistency in that study.

Although the BOR scale obviously merits particular attention when using the PAI/PAI-A to assess borderline personality phenomena, it should be apparent that many of the other constructs measured by the PAI are salient for this diagnosis. Indeed, research demonstrates that individuals diagnosed with borderline personality often demonstrate elevations on many other PAI scale in addition to BOR (Bell-Pringle et al., 1997; Morey, 1991). For example, the mean profile of a sample of patients diagnosed with borderline personality (Bell-Pringle et al., 1997) is presented in Fig. 5.1. This figure reveals that 10 of the 18 substantive scales of the PAI were elevated at least one standard deviation above community norms (i.e., 60 t) while 5 were elevated at least two standard deviations (70 t). While BOR is prominently elevated, so are ARD, DEP, SUI, and STR, while ANX, SOM, and NON also display prominent problems in these areas. Thus, a brief mention of some salient research on these scales is warranted, as these additional scales could help to clarify other aspects of the typical complex symptom picture that characterizes a borderline presentation. For example, the SOM scale, particularly SOM-C, has proven useful in several studies of the distinction between epileptic and non-epileptic seizures (e.g., Locke et al., 2011), providing an example of how the PAI might be useful in cases where functional vs. organic origins of physical complaints are an issue. The ARD scale (particularly ARD-T) has been found to be related to a variety of traumatic stress reactions, differentiating women psychiatric patients who were victims of childhood abuse from other women patients who did not experience such abuse (Cherepon & Prinzhorn, 1994) and demonstrates marked elevations in samples of combat-related PTSD (e.g., Calhoun, Boggs, Crawford, & Beckham, 2009). The DEP and ANX scale, while related to other commonly used measures of negative affect (Morey, 2007a, 2007b), have also proved to be sensitive measures of treatment response in treatments for disorders as varied as right temporal lobectomy (Glosser, Leis, Tracy, & Sperling, 2005), trichotillomania (Woods, Wetterneck, & Flessner, 2006), and irritable bowel syndrome (Bush, Pretorius, & Stuart, 2002). The STR and NON scales provide indicators of the respondent's perception of their environment with respect to the extent of environmental stressors and the availability of social supports to buffer this stress. These scales have been found to be related to the degree to which respondents feel connected to their community (Mashek, Cannaday, & Tangney, 2007) and to participation in support groups (Stevens & Duttlinger, 1998). With respect to the assessment of suicide risk, the SUI scale has been found to predict the number of subsequent suicide risk assessments in incarcerated men, and differentiate individuals who did or did not make subsequent suicidal gestures (Wang et al., 1997). Breshears, Brenner, Harwood, and Gutierrez (2010) found

that SUI predicted suicidal behavior in veterans who had experience traumatic brain injuries. SUI also has been found to be sensitive to the effects of treatments targeting self-damaging behaviors, such as dialectical behavior therapy (Harley et al., 2007).

Although unremarkable in the mean profile presented in Fig. 5.1, the two interpersonal scales of the PAI are often informative in the assessment of borderline personality phenomena. These two scales were designed to provide an assessment of the interpersonal style of subjects along two dimensions: (a) a warm and affiliative vs. a cold and rejecting axis, and (b) an axis characterized by a dominating, controlling vs. a meekly submissive style. These axes correspond to the two main vectors of the "interpersonal circumplex" that has provided a theoretical foundation for a number of different assessments and treatments (Horowitz & Strack, 2011). The PAI DOM and WRM scales have been found to be related to these other assessments in expected ways (e.g., Ansell, Kurtz, Demoor, & Markey, 2011). A number of studies have demonstrated that the combination of high DOM and low WRM scores, reflecting "hostile control," is particularly problematic. For example, this pattern has been associated with noncompliance, aggressive acts, and overall poor treatment progress in offender populations (Magyar et al., 2012). Inconsistent results in placing borderline personality along these dimensions across various studies have been interpreted by some as representing internal conflict or ambivalence on these dimensions, as opposed to a lack of relevance of these dimensions. Hopwood and Morey (2007) tested this hypothesis in a large clinical sample using inconsistency in self-report item responding on DOM and WRM to operationalize psychological conflict. This study found that individuals with borderline personality features (and not individuals with antisocial personality as a control group) were more inconsistent in item responding to both scales than were individuals without borderline features. Such findings may suggest that variability, as well as mean scores, on the interpersonal dimensions may be important for the conceptualizing borderline personality, and that such an approach offers a novel means of assessing interpersonal ambivalence.

Correlates of the BOR Scale in Youth Samples

The PAI instruments, and in particular the BOR scale, have been applied in studying a variety of problems in children, adolescents, and young adults. This research has taken a number of different forms. There are a host of studies that have sought to establish concurrent correlates of BOR elevations. Other studies have attempted to identify developmental precursors of individuals who demonstrate elevations on the BOR scale. Finally, a smaller literature exists attempting to identify developmental or longitudinal outcomes of individuals with BOR elevations. While such research provides important information surrounding the construct of borderline personality, it also provides evidence for the concurrent, antecedent, and predictive validity (respectively) of borderline personality characteristics as assessed by the PAI instruments. The following sections provide a brief overview of some of this research.

Antecedent validity studies. As noted above, one of the common research themes has involved an attempt to identify developmental precursors to PAI-BOR elevations observed in adolescents or young adults. Perhaps the most common issue has involved the investigation of childhood abuse experiences. In one of the earliest studies of this issue, Cherepon and Prinzhorn (1994) examined the PAI profiles of women abused (broadly defined) during childhood or adolescence, as compared to profiles of non-abused female patients. These investigators found that the BOR scale provided the greatest differentiation of these groups among all PAI scales. The women with a history of abuse tended to report significantly higher levels of BOR and most BOR subscales (particularly BOR-N) than the non-abused group; only the BOR-S subscale did not differentiate between the groups. This finding has been replicated and refined in a number of subsequent investigations. For example, Durrett et al. (2004) assessed college freshmen and found

that those with significant BOR elevations were more likely to report sexual abuse (total, by relative, and by nonrelative) on both interview and self-reported measures of abuse; however, they found no significant differences in reported physical abuse by borderline features status. In a similar study, Nickell, Waudby, & Trull (2002) showed that BOR scores were significantly associated with sexual abuse, and to a lesser extent, physical abuse history. Bailey, Moran, and Pederson (2007) studied a group of adolescent mothers, and found that BOR full scale and all BOR subscales, particularly BOR-A, were related to childhood sexual abuse of these women. Of note, the BOR-N subscale was specifically related to mental disorientation in these women during discussion of their abuse experiences.

Although relationships to childhood sexual abuse tend to be most prominent, BOR associations with other forms of childhood maltreatment have also been observed. For example, Allen (2008) correlated BOR with the *Comprehensive Child Maltreatment Scale*, measuring perception of experiences of six different forms of maltreating acts during childhood (Higgins & McCabe, 2001). Allen (2008) found that BOR was related to experiences of parental degradation, terrorizing, ignoring, and physical abuse, but not to experiences of isolation or of witnessing family violence. Similarly, Leary, Kelley, Morrow, and Mikulka (2008) studied the Revised Conflict Tactics Scales (CTS; Straus et al., 1995) and found that BOR-N demonstrated the highest association with use of parental corporal punishment of myriad psychosocial variables examined in the study; the BOR-I subscale was also associated with such punishment, while the remaining BOR subscales were not investigated in that study.

Another line of investigation has focused upon the type of child-rearing experiences of those who go on to demonstrate elevations on the BOR scale. For example, Nickell, Waudby, & Trull (2002) identified a variety of bonding or attachment variables to be related to elevated BOR scores in young adults, including reduced perceptions of care expressed by the respondent's mother, overprotectiveness and a discouragement of autonomy from the mother, an increased likelihood of an anxious or ambivalent attachment pattern, and a reduced likelihood of a secure interpersonal attachment pattern. In addition to attachment styles, the concept of childhood *invalidation* as described by Linehan (1993) has also been a focus of recent PAI-BOR research. Sauer and Baer (2010) examined the recollection of college students and their parents concerning the children's emotional style in childhood and the parenting they received, and scored these recollections for both validation and invalidation. As anticipated, the total (summed across raters) invalidation score was positively related to BOR scores, while total validation was inversely related to BOR. The BOR-A subscale was most related of the subscales to emotional vulnerability, total invalidation, and total validation, while BOR-S tended to be minimally related. One finding of particular interest in this study is that there was some evidence that there was better agreement between parents and their children on the invalidation measures when the child had lower BOR scores; young adults with above average scores on BOR rated their parents as more invalidating then their parents rated themselves, although there was still significant agreement on the ratings. In another interesting study examining the interaction between childhood sexual abuse and experiences of invalidation, Hong, Ilardi, and Lishner (2011) found that a real or perceived invalidating reaction to a child disclosing sexual abuse to a caregiver or other attachment figure may place children at increased risk for higher BOR scores as a young adult. Among those who had experienced such abuse, the BOR scale was positively correlated with both general and abuse-specific invalidation, as well as maternal lack of support, and maternal accusation and blaming of the child.

Concurrent validity studies. A number of investigations have examined the concurrent correlates of BOR scale elevations among children and adolescents. As noted previously, the PAI profiles of individuals diagnosed with borderline personality tend to be characterized by multiple other elevations, consistent with the general observation of high diagnostic comorbidity for

BPD. In the PAI-A adolescent clinical standardization sample, a host of other PAI scales demonstrated substantial correlations (i.e., at least 0.65) with the BOR scale, including NIM, PIM (negative), ANX, DEP, PAR, SCZ, and RXR (negative), reflecting the polymorphous symptom presentation typical of an individual with borderline personality functioning. Although BOR correlations with SUI were somewhat lower than for these other scales, subsequent research has confirmed the heightened risk for suicidality among high BOR scorers. Venta, Ross, Schatte, and Sharp (2012) studied the relationship of the BPFSC adaptation of the BOR (Crick et al., 2005) to suicidal ideation and behavior in a sample of 106 adolescents (average age 14.6 years) admitted to an inpatient psychiatric unit in a county hospital. These investigators found that the BPFSC was significantly related to the frequency and intensity of suicidal ideation, as well as to the seriousness of intent to die demonstrated in suicide attempts. They also found that high scorers tended to experience suicidal ideation earlier in life than individuals with other psychiatric disorders. BOR elevations have also been found to be related to efforts at nonlethal but deliberate self-harm in adolescents; Gratz et al. (2012) studied a large sample of middle- and high-school students using the BPFS-C and found that scores significantly predicted the presence of deliberate self-harming behaviors beyond demographic characteristics and their interactions.

Another diagnostic area that demonstrates associations with the BOR scale is attention-deficit disorder. Morey (2007a, 2007b) noted in the validation studies described in the PAI-A manual that BOR correlated significantly with various scores from the Connors ADHD scale (CAARS; Conners et al., 1999). PAI-A BOR correlated 0.61 with the CAARS ADHD Index, and was particularly related ($r = 0.73$) to the CAARS Impulsivity/Emotional Lability subscale. Distel et al. (2011) also administered the CAARS as part of their twin study examining the heritability of BOR scores, and estimated a phenotypic correlation between BOR scores and ADHD symptoms of $r = 0.59$, which could be explained 49 % by genetic factors and 51 % by environmental factors and measurement error. These investigators thus concluded that shared etiology between BOR scores and ADHD symptoms is likely responsible for the observed comorbidity of the two disorders.

Aside from diagnostic and symptomatic comorbidity, a host of other correlates of BOR elevations in youth have been explored. For example, attachment style has been a common area of investigation given the relational difficulties that characterize this disorder. One early study (Kurtz, Morey, & Tomarken, 1993) investigated the relationship of PAI-BOR to the Bell Object Relations Inventory (BORI: Bell, Billington, & Becker, 1986) and found BOR elevations to be particularly related to an insecure attachment scale. Similar findings were reported by Bailey et al. (2007), who examined an at-risk sample of adolescent mothers using the Adult Attachment Interview (AAI; George et al., 1985). Another problematic interpersonal area related to BOR scores involves relational aggression; for example, Werner and Crick (1999) found that older adolescents described by their peers as relationally aggressive tended to obtain higher scores on all four subscales of BOR—but that these associations were observed only in girls and not in boys. In considering this gender-linked finding, it is important to note that BOR did not demonstrate mean gender differences in either the PAI or the PAI-A normative community samples. The authors noted that relational aggression would tend to increase peer rejection, thus perpetuating the impact of such traits on long-term personality development. Along these lines, Ayduk et al. (2007) examined the association of BOR and rejection sensitivity, with the expected results that high BOR scorers were more sensitive to rejection. However, they also found that this relationship appears to be moderated by executive control, such that executive control might act as a buffer against rejection sensitivity heightening vulnerability to borderline personality features.

Certain cognitive styles have also been examined as correlates of high BOR scores. Hawes, Helyer, Herlianto, and Willing (2013) used an

implicit association test to measure automatic schemas that might reflect a self-concept that is prone to the experience of shame. These investigators found that such a shame-prone self-concept was associated with higher scores on the BOR-I subscale (using the BPFS-C version), but that this association was only found among girls. Sharp et al. (2011) found that adolescents with greater levels of borderline personality features as measured by the BPFSC tended to engage in hypermentalization, where they make elaborate and complex interpretations of social cues which are often inaccurate. As noted earlier, Kurtz and Morey (1999) found that high BOR scorers demonstrated a tendency to make negative evaluative judgments, relative to low BOR scores, when confronted with neutral or ambiguous affective information, while not differing in judgments when affective information was unambiguously positive or negative.

A final important area of study for correlates of high BOR scores have been investigations of the caretaking style of parents demonstrating such elevations. As might be expected, higher BOR scores tend to be associated with greater parenting problems. For example, Perepletchikova, Ansell, and Axelrod (2012) assessed mothers of children removed from the home due to abuse as compared to a community control group of mothers without CPS, and found that 50 % of CPS-involved mothers were elevated on BOR, compared with 15 % of control mothers. Other studies have focused upon the specific nature of the interaction between high BOR mothers and their children. For example, Barends (2002) conducted a study examining mothers with high scores on BOR and their relationships with their 2- and 3-year old children, which included observational data of the parent–child interaction as well as self-report parenting measures. Mothers with higher BOR scores were more flat and less positive with their children, felt less effective as parents, and had children with poorer adjustment. One interesting finding was that mothers with high BOR scores reported higher ambivalence towards their children as compared to mothers with lower scores when measured after the children participated in a free play session. After a problem-solving session with their child, however, high BOR mothers were *less* ambivalent toward their children than mothers with lower borderline traits. In general, mothers with lower levels of borderline characteristics tended not to vary in degree of ambivalence towards their children according to differences in the child-rearing situation, while high BOR mothers demonstrated much more fluctuation in their reported ambivalence.

Macfie and Swan (2009) examined a slightly older cohort of children aged 4–7, again comparing children's representations of the caregiver–child relationship (using a story-stem completing method) as a function of maternal scores on the BOR scale. Children whose mothers had high scores on BOR told stories with more parent–child role reversal, more fear of abandonment, and more negative mother–child and father–child relationship expectations than those with mothers scoring low on BOR; there were also marginally more intrusion of traumatic themes in the stories provided by children with high BOR mothers. Of the subscales, generally maladaptive relationship issues were most strongly associated with BOR-I and BOR-S elevations. Extending this area of investigation into adolescence, Grassetti (2011) studied adolescents aged 14–18 years and compared perceptions of parenting quality as well as borderline symptoms among those with mothers scoring high and low on BOR. Adolescents of high BOR mothers provided lower ratings of *parents as sources of support* and lower ratings for the *affective quality* of parental attachment relationships than did comparison adolescents, although there was no difference for ratings of autonomy facilitation. The high BOR mothers themselves demonstrated significantly lower ratings on various parental attachment quality subscales measuring the affective quality of attachment, parental fostering autonomy, and parental role in providing emotional support. Every maternal BOR subscale was positively correlated with adolescent scores on BOR-A, while maternal BOR-A was also related to adolescent BOR-N. Such results suggest that the presence of parental borderline personality features indicates that special care may need to be paid to the child of such a parent, given that the parenting

style of a BPD parent appears to have a negative impact on a child or adolescent and may places them at a higher risk for the development of borderline personality features themselves. The identification of parental borderline personality features can also be useful for the purposes of integrating parent-focused interventions for children identified to be at risk for the development of BPD.

Predictive validity studies. Because borderline personality often results in serious functional impairment and it is a difficult disorder to treat, the potential benefits of early identification and intervention could have dramatically significant effects on the quality of life and outcomes of a client. Unfortunately, long-term longitudinal outcome data on youth presenting with BOR elevations are very limited. In one of the first examinations of the BPFS-C, Crick et al. (2005) studied a sample of fourth and fifth graders and found that features reflecting hypothesized childhood expression of borderline phenomena (Geiger & Crick, 2001), such as cognitive sensitivity, emotional sensitivity, friend exclusivity, and aggression, tracked together with children's BPFS-C scores over the course of a year. Further, each of the hypothesized features uniquely predicted borderline personality features over time, above and beyond the longitudinal association between BPFS-C scores and the other three features. The authors concluded that failure to master important developmental tasks in childhood, for example the ability to inhibit aggressive outbursts, places children at risk for subsequent development of borderline pathology.

The few remaining longitudinal investigations have primarily focused upon intermediate outcomes in mid-to-late adolescence. For example, Cashel et al. (2006) found that the BOR scale was the best predictor of subsequent psychiatric referrals in a sample of female adolescents in a juvenile detention facility. Trull (1995) and Trull et al. (1997) examined borderline personality features in adolescents during their transition to college using the BOR scale, completing a 2-year follow-up on participants. During the 2-year follow-up, Trull et al. (1997) found that those individuals in the significant borderline personality features group displayed poorer overall outcomes than those individuals in the nonsignificant borderline personality features group, including an increased likelihood for experiencing serious academic difficulties, as well as an increased risk for the development of mood disorders and interpersonal dysfunction. Subsequent follow-ups determined that the high BOR students were more likely to use treatment, medications, and medical health visits (Bagge, Steppe, & Trull, 2005) as well as more likely to develop alcohol problems, particularly if BOR-S was elevated (Stepp, Trull, & Sher, 2005). These findings suggest that borderline personality traits, even when measured at a subclinical level, can have a significantly negative impact on the development of an adolescent transitioning into adulthood.

Future Directions

The material presented in this chapter makes it apparent that the PAI instruments can provide a wealth of information useful in the assessment of borderline features in youth. As with the borderline construct itself, there continues to be much to learn. Of particular value would be long-term outcome studies of individuals with borderline features as determined with PAI assessments. Comparable research on adults demonstrates that there is considerable change among such individuals over periods as long as 10 years (Gunderson et al., 2011), also in periods as short as 6 months (Gunderson et al., 2003). Conducting such research would provide greater insight into the longer term stability of elevations on, for example, the BOR scale. However, with the richness of the additional information available from the full PAI instrument, it might be possible to determine other factors that might serve to moderate this course, serving to potentially identify elements reflecting resiliency, or perhaps those representing particular targets for relapse prevention. Even lacking such studies, preliminary evidence on the antecedent, concurrent, and predictive validity of the PAI for assessing borderline features in young respondents highlights its utility and applicability in both clinical and research settings.

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Conceptualizing Youth BPD Within an MMPI-A Framework

6

Martin Sellbom and Matthew A. Jarrett

Introduction

While borderline personality disorder (BPD) has long been researched in adults, only recently have scholars begun to study BPD in adolescence. In some ways, this delay in adolescent research is surprising, given that the initial clinical description of BPD stemmed from the developmental literature (Kernberg, 1967; Masterson & Rinsley, 1975). Part of the reason for the delay in research on adolescent BPD has been the long held belief that personality patterns do not become stable until adulthood. More recently, though, this assumption has been challenged. For example, a change was made in the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revised* (*DSM-IV-TR*; American Psychiatric Association [APA], 2000) to indicate how BPD should be diagnosed in adolescence (i.e., a stable behavior pattern of 1 year prior to age 18 rather than the 2 year requirement for adults). Aside from alterations to *DSM-IV* criteria, there has also been a resurgence of interest in the developmental perspective as it relates to BPD. For example, this interest was reflected in a special issue of *Development and Psychopathology* in 2005, which was devoted exclusively to the development of BPD. Although research has supported the presence of borderline traits in adolescence, debate still exists regarding the diagnosis of BPD in adolescence, and many clinicians are reluctant to diagnosis BPD during this age period. Clearly, evidence-based assessment of BPD will be a critical issue for future research and practice.

In order to advance assessment practices for youth BPD, the current chapter will focus on assessment strategies for BPD utilizing a dimensional personality trait perspective. Prior to discussing these strategies, we will review research on personality traits in relation to personality disorders with a specific focus on BPD. We will also draw upon the temperament literature to explore how early temperament characteristics might relate to developmental pathways for BPD. More specifically, we will consider negative affectivity and inhibitory control as predominant traits underlying the disorder. Due to the intense trauma and stress-induced pseudo-psychotic symptoms experienced by many individuals with BPD, we also considered the domain of psychoticism. Finally, we devote a significant portion of this chapter to the assessment of BPD using the *Minnesota Multiphasic Personality Inventory—Adolescent* (MMPI-A; Butcher et al., 1992) and offer suggestions for future clinical assessment practices.

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Temperament, Personality, and Psychopathology

Personality disorders have been researched extensively over the years, but one of the debates in the field has been whether personality disorders should be defined as categorical entities or combinations of extreme personality dimensions (Widiger & Clark, 2000). For example, in relation to BPD, there has been concern about the empirical support for a categorical model of BPD as well as issues surrounding the heterogeneity of this disorder given its broad criterion set. The dimensional view was highly influential in the recent *DSM-5* personality disorders workgroup proposal for PDs, which led to an alternative model for PDs being included in Section III (Emerging Models and Measures) for further research (APA, 2013). This approach will utilize dimensional trait domains, including antagonism, detachment, disinhibition, negative affectivity, and psychoticism, to define personality disorders (e.g., combinations of these traits reflecting BPD). Clearly, more research is needed on this dimensional approach, as this it would have far-reaching implications for the evidence-based assessment of personality disorders if adopted in a future version of the DSM.

A similar perspective has also emerged in the child and adolescent literature (although not yet directly integrated into *DSM-5*), as research has sought to better link temperament and psychopathology. Based on a review of temperament and psychopathology linkages, Nigg (2006) emphasized two temperament dimensions, one relating to regulation (e.g., constraint, effortful control, inhibition) and another dimension relating to reactivity (e.g., approach vs. withdrawal). Although these dimensions have long been recognized in the temperament research literature (Derryberry & Rothbart, 1997), they have only recently been applied to understanding the nature of psychopathology (Nigg, Goldsmith, & Sachek, 2004). Importantly, and as will be discussed later, these domains also map well onto our developing understanding of BPD from a developmental psychopathology perspective. In addition, this research on temperament and psychopathology may have important implications for better understanding children and adolescents who will be at risk for the development of BPD.

Conceptualizing BPD from a Developmental Trait Perspective

One of the challenges in understanding BPD from a personality perspective is the fact that BPD represents broad dysfunction across domains such as affect, cognition, behavior, and interpersonal relations. At the same time, three dimensions have more clearly emerged as critical in our understanding of BPD from a personality perspective: disinhibition, negative affectivity, and psychoticism. In addition, these domains will also characterize a dimensional perspective of BPD as included in *DSM-5* Section III. First, the domain of disinhibition seems critical in our understanding of BPD and is manifested in the risk-taking and impulsive behaviors seen in patients with BPD (e.g., self-harm, participation in risky activities). While disinhibition has been thought of as a core dysfunction in adults with BPD (Depue & Lenzenweger, 2001), it has also emerged as an important factor in the development of BPD (Nigg, Silk, Stavro, & Miller, 2005). It is important to note, however, that disinhibition is thought to be a core feature in Axis I childhood disorders, including attention-deficit/hyperactivity disorder (ADHD) and conduct disorder (CD; Nigg, 2001); thus, disinhibition does not uniquely reflect BPD.[1] A second temperament domain, which has been deemed to be critical in our understanding of BPD, is negative affectivity. This domain is typically manifested as emotional lability, anxiousness, separation insecurity, and depressivity in patients with BPD (APA, 2013). Moreover, negative affectivity also plays a key role in Axis I anxiety and mood disorders (e.g., Tackett, Quilty, Sellbom, Rector, & Bagby, 2008; Watson, 2005); so once again, negative affectivity by itself is not unique

[1] In adulthood, it is also the defining temperament domain of antisocial PD.

to BPD. Specific negative affects, such as intense anger and hostility likely differentiate BPD from these disorders, but these traits might also be observed in children with disorders such as oppositional defiant disorder (ODD) and conduct disorder (CD).[2]

So how might one think about the intersection of disinhibition and negative affectivity, particularly in relation to the development of BPD? In relation to temperament and psychopathology, Nigg (2006) argues that children and adolescents high on both disinhibition and negative affectivity might present as children with ADHD and comorbid internalizing disorders such as anxiety and depression. Interestingly, this comorbidity is rather common in children with the Combined Type of ADHD or ADHD-C (i.e., children with inattention and hyperactivity/impulsivity), as clinical samples of children with ADHD typically show a rate of comorbidity in the range of 30–40 % with a somewhat lower rate of 25 % in the population (Jarrett & Ollendick, 2008; Tannock, 2009). While this diagnostic configuration might capture disinhibition and negative affectivity, it does not entirely capture anger and hostility, emotional lability, or the intense separation insecurity of BPD. Interestingly, ADHD also puts children at much greater risk for disorders associated with oppositionality and aggressive and antisocial behaviors. For example, in the Multimodal Treatment of ADHD Study (MTA Study), the largest clinical trial ever conducted for ADHD, approximately 50 % of children met criteria for ODD or CD (MTA Cooperative Group, 1999). In addition, 50 % of children with ADHD + ODD/CD met criteria for an anxiety disorder. Overall, in thinking about children and adolescents at risk for BPD, we might consider children with ADHD + ODD/CD + anxiety/depression as at greater risk for the development of BPD; yet, this constellation of symptoms does not entirely represent the severe disruptions in self-functioning and interpersonal functioning seen in BPD.

What might be some pathways to the disrupted interpersonal and self-functioning seen in BPD? As noted by Nigg et al. (2005), there may be different etiological pathways (or developmental routes) to BPD. For example, such pathways or developmental routes might involve different childhood risk factors and socialization experiences that contribute to the development of BPD in adolescence. Overall, the authors noted two primary pathways. One pathway to BPD may involve early impulsivity and disinhibition deficits that evolve into the clinical picture described above (i.e., ADHD and ODD or CD in the context of negative affectivity) with the negative socialization experiences associated with this dysfunction leading to personality disturbance. Importantly, such a pathway might involve the absence of a trauma history. Alternatively, Nigg et al. (2005) propose an alternate pathway to BPD that is primarily trauma-driven in that an environment involving significant maltreatment disrupts normal affective functioning, which secondarily affects the development of inhibitory control. See Table 6.1 for more detail regarding these possible pathways.

Although a more detailed discussion of etiology is outside of the scope of the current chapter, these pathways are emphasized since they may be important to consider from an assessment standpoint. For example, pathways have been recognized as important in the assessment of conduct disorder (e.g., childhood vs. adolescent onset), since childhood onset is associated with a poorer prognosis. In relation to assessment of BPD in adolescence, a more trauma-relevant pathway may result in unique elevations on temperament/personality traits in comparison to a pathway not involving trauma (e.g., greater elevations on factors related to dissociation) and may have implications for treatment response.

Finally, a third personality factor frequently recognized and highlighted in the adult literature, psychoticism, might be useful in the identification of BPD. This domain reflects a propensity towards experiencing unusual sensory states, odd and eccentric beliefs, paranoid ideation, and

[2] In the DSM-5 Section III, Antagonism is included as a domain for BPD, primarily due to the hostility facet; however, hostility also loads on negative affectivity in the DSM-5 Section III trait model. As such, we contend that negative affectivity and disinhibition should be the primary temperament domains considered for this disorder.

Table 6.1 Pathways to BPD based on Nigg et al. (2005)

Pathway	Infancy	Preschool	School age	Adolescence	Adulthood
Non-trauma	Genetic predisposition to ADHD; early deficits in reactivity that may be genetically driven or affected by prenatal influences; emerging difficulties with inhibitory control	Parent–child conflict stemming from behavioral deficits leading to ODD	Academic and social impairment; growing emotional and behavioral problems	Increased academic and social impairment leading to identity issues; increasing antisocial behavior leading to CD	BPD
Trauma	Early maltreatment (in combination with biological influences) leads to affective instability	Inhibitory control deficits secondary to affective disruption; parent–child relationship difficulties contributing to behavioral deficits leading to ODD	Academic and social impairment; growing emotional and behavioral problems	Increased academic and social impairment leading to identity issues; increasing antisocial behavior leading to CD	BPD

poor reality testing (Harkness & McNulty, 1994; Tackett, Silberschmidt, Krueger, & Sponheim, 2008; Watson, Clark, & Chmielewski, 2008). Disinhibition and negative affectivity do not seem to completely capture the BPD deficits seen in identity or self-direction. In relation to the trauma-relevant pathway, it is likely that children and adolescents who exhibit significant disinhibition and negative affectivity also display paranoid and/or dissociative states when under stress, as emphasized in the current *DSM-5* criteria. In turn, psychoticism might serve to capture these symptoms. Given adult studies showing that up to 85 % of patients with BPD have a trauma history (Venta, Kenkel-Mikelonis & Sharp, 2012), a factor such as psychoticism is likely to be important to consider for many BPD cases, since trauma is often associated with dissociative states that may be reflected in personality domains such as psychoticism.

Conceptualizing BPD from a MMPI-A Perspective

The main goal of the current chapter is to describe how to best use the MMPI-A to generate information that is directly relevant to the conceptual formulation of BPD in adolescence that was just presented. This will be accomplished by first considering research with the original MMPI in both adult and adolescent samples and then moving on to more contemporary findings that can best inform BPD assessment using the MMPI-A. All of this information will subsequently be synthesized into a coherent set of recommendations for BPD assessment practices via the MMPI-A as well as providing future directions for research in this area.

The MMPI-A is a 478-item true/false self-report inventory designed to assess the social, emotional, and behavioral functioning of adolescents between the ages of 14 and 18 (Butcher et al., 1992). The normative sample of the MMPI-A is a large, nationally representative sample, consisting of 805 boys and 815 girls between the ages of 14 and 18 who were randomly recruited from schools in the United States. The adequacy of MMPI-A scores based on the normative sample in various demographic groups has been supported in previous research (e.g., Schinka, Elkins, & Archer, 1998). The MMPI-A contains ten, basic *Clinical* scales that were retained during the revision process from the original MMPI (Butcher et al., 1992). Overall, these scales are intended to provide a broad

overview of the problems and difficulties being reported by the adolescent, with clarification of high scores achieved using the 31 *Harris-Lingoes and Si subscales* (Ben-Porath, Hostetler, Butcher, & Graham, 1995; Harris & Lingoes, 1955). Following the methods used to develop the MMPI-2 Content scales, Williams, Butcher, Ben-Porath, and Graham (1992) developed 15 *Content* scales to provide a method of assessing the basic content domains of the MMPI-A item pool. The Content scales were intended to provide an additional method of clarifying the adolescent's self-presentation and identifying which interpretative statements from the Clinical scales should be emphasized (Butcher et al., 1992). More explicit clarification of high scores on the content scales can be obtained through examining scores on the *Content Component* scales (Sherwood, Ben-Porath, & Williams, 1997). The MMPI-A also contains a set of six *Supplementary* scales that were either carried over from the MMPI or added to the test during its development (Butcher et al., 1992). These scales are intended to enhance the clinical picture of the adolescent provided by the Clinical and Content scales by assessing important areas not covered by other MMPI-A scales. Lastly, the MMPI-A contains adolescent versions of the *Personality Psychopathology Five* (PSY-5; McNulty, Harkness, Ben-Porath, & Williams, 1997) scales that were originally developed for the MMPI-2 to index broad dimensions of personality pathology. All of the MMPI-A scales discussed throughout this chapter relevant to the assessment of BPD are presented in Table 6.2.

Clinical Scales

The MMPI (and MMPI-A) Clinical scales represent the initial sources of MMPI-related information for any type of psychopathology, including BPD. A plethora of research studies (mostly in adults), particularly with the original MMPI, have been conducted with these scales with respect to differential diagnosis and generating profile configuration for BPD patients (see e.g., Gartner, Hurt, & Gartner, 1989; Morey & Smith, 1988; Resnick, Goldberg, Schulz, & Schulz, 1988; Zalewski & Archer, 1991; for reviews).

In terms of profile configurations, we identified 16 studies that reported mean Clinical scale profiles for unique groups of patients with BPD (Abramowitz, Carroll, & Schaffer, 1984; Archer, Ball, & Hunter, 1985; Bell-Pringle, Pate, & Brown, 1997; Evans, Ruff, Braff, & Ainsworth, 1984; Evans, Ruff, Braff, & Cox, 1986; Gustin, 1983; Hurt, Clarkin, Frances, Abrams, & Hunt, 1985; Jonsdottir-Baldursson & Horvath, 1987; Kroll et al., 1981; Morey, Blashfield, Web,b & Jewell, 1988; Newmark, Chassin, Evans, & Gentry, 1984; Patrick, 1984; Resnick et al., 1983; Skinstad, 1994; Snyder, Pitts, Goodpaster, Sajadi, & Gustin, 1982; Widiger, Sanderson, & Warner, 1986).[3] Fifteen of these studies revealed that Scale 8 (Schizophrenia), which is a heterogeneous measure of thought disturbance, dissociation, odd and eccentric characteristics, cognitive complaints, and negative emotionality (Archer, 2005), had the highest elevation. In the lone study in which this was not the case (Resnick et al., 1983), Scale 8 was the second most elevated. Furthermore, in almost all of these studies, Scales, 4, 2, 7, and 6 have also been elevated (i.e., >70 T) in varying order, and 10 of 16 studies showed elevations on at least 6 Clinical scales. With respect to code types, which typically involve the two or three most elevated scales in the profile, the majority of studies have found support for various iterations that include Scale 8 (e.g., 8-2, 8-4, 8-6, 8-7, 8-4-2, 8-7-2), but also others (e.g., 2-7, 4-2) in some patients (Morey & Smith, 1988; Resnick et al., 1988; Zalewski & Archer, 1991). In the lone study that examined BPD in adolescence (Archer et al., 1985), the following scales were elevated, in order, 8, 1, 2, 4, and 6. Overall, these findings led some scholars to discuss the so-called "floating" profile in the assessment of BPD in which most of the Clinical scales are elevated just over 70 T (Gartner et al., 1989; Hurt et al., 1985; Newmark et al., 1984; Newmark & Sines, 1972). Again, Table 6.2

[3] One of these studies (Bell-Pringle et al., 1997) used MMPI-2 data; the rest used the original MMPI. None of these studies used the MMPI-A.

Table 6.2 MMPI-A scales discussed in this chapter

Scale	Abbreviation[a]	Description
Clinical scales		
Hypochondriasis	1/Hs	Preoccupation with bodily functioning; illness anxiety
Depression	2/D	Emotional distress, dysphoria; anhedonia; hopelessness; life dissatisfaction
Hysteria	3/Hy	Somatic preoccupation, especially concerning neurological symptoms; self-centered and dramatic interpersonal style
Psychopathic deviate	4/Pd	Nonconformity to social norms and standards; authority problems; impulsivity; antisocial and reckless behaviors; emotional distress; alienation
Paranoia	6/Pa	Persecutory ideation; interpersonal mistrust and sensitivity; cynicism
Psychasthenia	7/Pt	Negative emotionality, including anxiety, obsessive-compulsivity, sadness; Feelings of inferiority and inadequacy
Schizophrenia	8/Sc	Bizarre thoughts processes; overt psychotic symptoms; social isolation; emotional and behavioral dysregulation; cognitive complaints
Hypomania	9/Ma	Elevated mood; grandiosity; irritability; cognitive and behavioral hyperactivity, including impulsivity
Personality psychopathology five (PSY-5) scales		
Psychoticism	PSYC	Propensity towards experiencing unusual sensory states; odd and eccentric beliefs; paranoid ideation; poor reality testing
Disconstraint	DISC	Proclivity towards disinhibition; impulsivity; sensation seeking; thrill seeking; nonconforming behavior
Neuroticism/Negative emotionality	NEGE	Propensity toward a wide range of negative emotions, including depression, anxiety, anger, fear, guilt; emotional dyscontrol; sensitivity to criticism; low self-esteem; interpersonal dependency
Content/content component scales		
Depression	A-dep	Clinical depression; sadness; apathy/anhedonia; demoralization
Dysphoria	A-dep1	Sadness; depression; despondency
Self-deprecation	A-dep2	Poor self-esteem
Suicidal ideation	A-dep4	Suicidal ideation, gestures, behaviors
Low self-esteem	A-lse	Poor self-esteem or self-confidence; feelings of inadequacy; interpersonal passivity
Self-doubt	A-lse1	Poor self-esteem; unstable self-image
Interpersonal Submissiveness	A-lse2	Interpersonal passivity; insecurity/vulnerability in relationships
Anger	A-ang	Anger; low frustration tolerance; impatience; hostility; grouchiness
Explosive behavior	A-ang1	Poor anger control; reactive aggression; physical aggressiveness
Irritability	A-ang2	Low frustration tolerance; irritable mood; impatience
Conduct problems	A-con	Externalizing behaviors; poor impulse control; antisocial attitudes
Acting out behavior	A-con1	Poor impulse control; risk-taking behavior; antisocial behavior
Anxiety	A-anx	Generalized anxiety; stress reactivity; worry; rumination
Alienation	A-aln	Interpersonal isolation; alienation; frustration about social interactions; blame externalization
Bizarre Mentation	A-biz	Psychotic thought processes; unusual thoughts and ideas; poor reality testing; poor emotional control; paranoid ideation
Supplementary scales		
Immaturity	IMM	Psychological maturation; ego/identity development; egocentricity; poor anger control
MacAndrew Alcoholism Scale-Revised	MAC-R	Risk for alcohol and drug abuse; propensity towards impulsivity, risk-taking, and sensation seeking

[a]Clinical scales are typically referred to by their scale number (e.g., Scale 1) to avoid the diagnostic connotations associated with their labels

provides descriptions associated with each Clinical scale and BPD-relevant information.

In addition to identifying mean Clinical profiles, researchers have also attempted to determine how such profiles relate to the differential diagnosis of BPD. In general, research studies have shown only marginal support for using specific profile configurations in accurately diagnosing BPD (Morey & Smith, 1988; Widiger et al., 1986). For instance, Newmark et al. (1984) found that only 30 % of individuals with floating profiles met criteria for BPD; however, they provided no data on the proportion of BPD patients who had floating profiles, which would have been important with respect to sensitivity. Widiger et al. (1986) found the best support for an 8-2-4 codetype, with 57 % of BPD patients showing this configuration as opposed to only 15 % of patients with other personality disorders. These positive findings were confounded by a 93 % base rate of BPD. In addition, specificity rates decreased when accounting for overlap with antisocial and schizotypal PDs. Bell-Pringle and colleagues (1997) found that using four specific code types (8-4-2, 8-2-4, 8-4-7, or 8-2-7) was associated with poor sensitivity in their patient sample (9 %), but excellent specificity in their student comparison group (95 %); unfortunately, these results are limited by the very low base rate of these specific code types in this particular patient sample, likely because Scales 8, 4, 2, 6, and 7 were all highly elevated. Thus, research has clearly shown that a specific Clinical scale configuration or code type will likely not be useful in accurately identifying BPD; however, it is possible that individual elevations on scales, such as Scale 8, 4, 2, and 7 might be suggestive of the possible presence of the disorder.

Morey and Smith (1988) recommended that the MMPI Clinical scales might be more useful in differential diagnosis, with which we concur, and patterns of these mean scale differences across diagnostic groups also speak to the conceptual similarities between BPD and other psychiatric conditions. With respect to unipolar depressive disorders, adult BPD patients were clearly higher on 4, 6, 8, and 9, which is reflective of disinhibition and psychoticism, than patients with major depressive disorder (Gandolfo, Templer, Cappelletty, & Cannon, 1991; Snyder, Pitts et al., 1982; Snyder, Sajadi, Pitts, & Goodpaster, 1982). Archer et al. (1985) found that adolescents with BPD were higher on 1, 2, 4, 6, 7, 8, and 9 than a group of dysthymic patients. In terms of Schizophrenia, Evans and colleagues (Evans et al., 1984) showed that BPD patients had higher scores on 2, 3, 4, and 7 relative to patients with Schizophrenia, with the largest difference being associated with Scale 4. Thus, greater emotional and disinhibitory proclivities separated patients with BPD from those with schizophrenia. Furthermore, in adolescent inpatients, those with BPD scored significantly higher on Scales 1, 2, and 8 than any other patients (dysthymia, conduct disorder, other PDs), primarily because the other patients included those with conduct disorder; BPD was higher on scale 4 than all other groups.

Taken together, these findings with the MMPI Clinical scales reveal some general patterns that are relevant to out conceptual formulation of BPD in adolescence. Patients with BPD tend to have mean profiles that involve Scales 8, 4, 2, 7, and 6, which are linked to underlying proclivities towards negative affectivity, including emotional lability (Scales 7 and 8), depressive affect (Scales 2 and 7), alienation (Scales 4 and 8), intense anger and hostility (Scales 4 and 8), as well as disinhibition (Scale 4) and traits reflecting fleeting dissociation and paranoia (i.e., psychoticism; Scales 6 and 8).

In conclusion, although numerous studies exist to provide a Clinical scale formulation of BPD, there are a number of problems associated with outright reliance on these scales. First, they are only loosely grounded in the conceptual formulation of adolescent BPD presented earlier. Second, most of the research clearly indicates relatively poor specificity with respect to profile configurations measuring BPD, likely owing to the many different manifestations of the disorder (e.g., Tyrer & Johnson, 1996). Finally, the Clinical scales themselves are quite heterogeneous and show limited discriminant validity with respect to linking them to *DSM-5* and other temperament domain and facet traits. We therefore

prefer a more conceptually rooted MMPI-A approach that, subject to empirical validation, can be better linked to contemporary BPD formulations in youth.

Personality Psychopathology Five (PSY-5) scales

The MMPI-2 PSY-5 scales (Harkness, McNulty, & Ben-Porath, 1995) provide a measurement model for five dimensional personality constructs that describe normal to abnormal range personality traits. Harkness and McNulty (1994) identified these major dimensions of personality pathology in a series of latent root analyses using a mixture of pathological descriptors (per *DSM-III-R* personality disorder criteria) embedded within a pool of normal-range descriptors. The PSY-5 Scales were developed to assist in assessing personality pathology from a dimensional perspective. The scales, which McNulty et al. (1997) adapted for the MMPI-A, include Aggressiveness (AGGR), Psychoticism (PSYC), Disconstraint (DISC), Neuroticism/Negative/Emotionality (NEGE), and Introversion/Low Positive Emotionality (INTR). Furthermore, it should be highlighted that the five construct domains mirror those five domain traits currently included in *DSM-5* Section III (APA, 2013)—Antagonism, Psychoticism, Disinhibition, Negative Affectivity, and Detachment. Indeed, the *DSM-5* Personality and Personality Disorders workgroup explicitly stated that the proposed *DSM-5* personality trait model "at the domain-level, bears a strong resemblance to Dr. Allan Harkness' Personality Psychopathology Five (PSY-5) model of clinically relevant personality variants" (APA, 2011). Recent research has also demonstrated that the PSY-5 scales converge with PID-5 (official measurement of *DSM-5* trait system; Krueger, Derringer, Markon, Watson, & Skodol, 2012) domain scales in a young adult sample (Anderson et al., 2013).

We propose that three MMPI-A PSY-5 scales in particular are related to the measurement of BPD (see Table 6.3). First and foremost, Neuroticism/Negative Emotionality (NEGE) reflects "a broad affective disposition to experience a wide range of unpleasant emotions, particularly anxiety, nervousness, and guilt, leading to internal suffering" (McNulty et al., 1997, p. 250; see also Tellegen & Waller, 2008). This construct is directly linked to the negative affectivity temperament domain observed in childhood/adolescence (Nigg, 2006) as well as the domain included in *DSM-5* Section III. Second, the PSY-5 domain Disconstraint (DISC) reflects a propensity towards impulsivity, sensation seeking, risk-taking, and nonconforming behavior (McNulty et al., 1997; Tellegen & Waller, 2008) and parallels (in the opposite direction) the inhibitory control temperament domain described earlier (Nigg, 2006; Nigg et al., 2005), as well as the Disinhibition domain included in *DSM-5* Section III. Finally, although less emphasized in the *DSM-5* personality structure but clearly present in historical and empirical accounts of BPD in general, PSY-5 Psychoticism (PSYC) maps onto the stress-induced, fleeting paranoid and dissociative experiences sometimes experienced in individuals with BPD (Barnow, Arens, Dinu-Biringer, Spitzer, & Lang, 2010).

Research on the MMPI-A PSY-5 scales has been sparse relative to their adult (i.e., MMPI-2 and MMPI-2-RF) counterparts, but initial research has been quite promising in that the nomological networks associated with these scale scores are quite similar to those on the MMPI-2/MMPI-2-RF (McNulty et al., 1997; Veltri et al., 2009; see Harkness, Finn, McNulty, & Shields, 2012 for a review). Research with MMPI-2 PSY-5 scales directly relevant to BPD has been quite promising. For example, Trull, Useda, Costa, and McCrae (1995) estimated regression models that showed that the MMPI-2 PSY-5 scales as a set predicted 51 and 64 % of variance in SIDP-R and PDQ-R BPD symptom counts, respectively, in a clinical sample. Unfortunately, they did not report individual beta weights in their regression equations to indicate which PSY-5 scales were uniquely associated with BPD. Wygant, Sellbom, Graham, and Schenk (2006) showed that NEGE and PSYC, but not DISC, were moderately to strongly correlated with number of self-reported BPD criteria in a large clinical sample. Furthermore, Bagby, Sellbom, Costa, and Widiger (2008)

Table 6.3 DSM-IV and DSM-5 BPD criteria and corresponding MMPI-A scales

BPD criteria		MMPI-A scales		
DSM-IV/DSM-5 Section II	DSM-5 Section III trait facets	PSY-5	Content scales (and CC scale)	Supplementary scales
1. Frantic efforts to avoid real or imagined abandonment	NA—separation insecurity; impairment in intimacy functioning	NEGE	A-lse2	
2. Unstable and intense interpersonal relationships alternating between idealization and devaluation	Impairment in intimacy functioning; impairment in empathy functioning	NEGE	A-dep2; A-aln	
3. Identity disturbance: markedly and persistently unstable self-image or sense of self	Impairment in identity functioning	NEGE	A-lse1	IMM
4. Impulsivity in at least two areas that are self-damaging	DIS—impulsivity; DIS—risk taking; impairment in self-direction	DISC	A-con	MAC-R; IMM
5. Recurrent suicidal behavior, gestures, threats, or self-mutilation	NA—depressivity; DIS—impulsivity	NEGE; DISC	A-dep4; A-con1	
6. Affective instability due to marked reactivity of mood (e.g., intense dysphoria, irritability, or anxiety)	NA—emotional liability; NA—anxiousness	NEGE	A-anx; A-dep1; A-ang2	
7. Chronic feelings of emptiness	NA—depressivity; Impairment in Identity	NEGE	A-dep	
8. Inappropriate or intense anger or difficulty controlling anger	NA—hostility; NA—emotional liability	NEGE	A-ang1	IMM
9. Transient, stress-related paranoid ideation or severe dissociative symptoms	Impairment in identity functioning	PSYC	A-biz	

NA negative affectivity, *DIS* disinhibition, *NEGE* Neuroticism/Negative emotionality, *DISC* disconstraint, *PSYC* psychoticism, *A-lse2* interpersonal submissiveness, *A-dep2* self-deprecation, *A-aln* alienation, *A-lse1* self-doubt, *A-con* conduct problems, *A-dep4* suicidal ideation, *A-con1* acting out behavior, *A-anx* anxiety, *A dep1* dysphoria, *A-ang2* irritability, *A-dep* depression, *A-ang1* explosive anger, *A-biz* Bizarre Mentation, *IMM* immaturity, *MAC-R* MacAndrew Alcoholism Scale-revised

examined associations between SCID-II-PQ BPD counts and PSY-5 scales in a large mental health sample. They found that NEGE and PSYC, but not DISC, were associated with BPD symptom counts. Regression models showed that the PSY-5 scales, collectively, accounted for 38 % of variance in self-reported BPD symptoms. Most recently, Sellbom, Smid, De Saeger, Smit, and Kamphuis (2014) examined the associations between the PSY-5 scales and interview-based BPD symptoms counts in a forensic psychiatric hospital for male violent offenders ($n = 162$) and a clinical inpatient sample ($n = 190$). The SIPD-IV and SCID-II modules for BPD were used in these respective samples. Sellbom et al. found that NEGE, DISC, and PSYC were all significantly correlated with BPD counts. In a negative binomial regression model, all three scales contributed uniquely to the prediction of BPD symptoms in the clinical sample, but only NEGE and DISC contributed uniquely to this prediction in the male forensic psychiatric sample. The inconsistency of DISC across these studies is surprising and could perhaps be attributed to the use of self-report measurement of BPD in the non-supportive studies.

In conclusion, we believe that elevations on NEGE, DISC, and PSYC can reflect an important profile that is highly related to the personality

psychopathology associated with adolescent BPD. These domains are conceptually linked to Nigg's (2006) developmental temperament domains of disinhibition and negative affectivity, which also coincide with those of *DSM-5* Section III BPD. Psychoticism maps onto the consistently observed features of BPD and recognized *DSM-IV*/*DSM-5* criterion. However, the PSY-5 domains nonetheless have limitations in that they are broad, and elevations on NEGE, DISC, and/or PSYC would not render high specificity with respect to adolescent BPD. Therefore, it would be worthwhile to consider additional, more specific scales that might better map onto the facets of these broader temperament domains.

Content and Supplementary Scales

There are several MMPI-A content and supplementary scales that could fill this function. Table 6.3 shows how these scales map onto the specific *DSM-IV*/*DSM-5* Section II criteria as well as *DSM-5* Section III facets. The Depression (A-dep) Content scale, and in particular, its Content Component scales reflecting dysphoria (A-dep1), self-deprecation (A-dep3), and suicidal ideation (A-dep4) are associated with a number of BPD criteria. Research has shown that the A-dep Content scale index a general sense of depression, sadness, pervasive hopelessness, self-mutilation, suicidality, and anhedonia (Arita & Baer, 1998; Handel, Archer, Elkins, Mason, & Simonds-Bisbee, 2011; Veltri et al., 2009; Williams et al., 1992), with specific measurement of intense dysphoria (A-dep1), low self-esteem (A-dep2), and suicidality (A-dep4) (Sherwood et al., 1997). Thus, these scales would map onto several BPD criteria quite well, including unstable and intense relationships (#2; A-dep2), recurrent suicidal behavior or gestures (#4; A-dep4), affective instability (#6; A-dep1), and chronic feelings of emptiness (#7; A-dep). The Anxiety Content scale (A-anx) measures generalized anxiety, including stress reactivity, intense anxiety, and affective instability, which has been supported by several empirical studies (Arita & Baer, 1998; Handel et al., 2011; Kopper, Osman, Osman, & Hoffman, 1998; Veltri et al., 2009). This measurement also maps onto the affective instability criterion (#6).

The Low Self-Esteem Content scale facets Self-Doubt (A-lse1) and Interpersonal Submissiveness (A-lse2) reflect feelings of low self-worth and poor self-confidence (A-lse1) as well as passivity and fear of rejection (A-lse2)—all these descriptors have been empirically validated (Forbey & Ben-Porath, 2003; Sherwood et al., 1997; Veltri et al., 2009; Williams et al., 1992). Thus, these scales are conceptually relevant to the identity disturbance/unstable self-image (#3; A-lse1) and frantic efforts to avoid abandonment (#1; A-lse2) criteria. The Anger Content scale facets Explosive Behavior (A-ang1) and Irritability (A-ang2) reflect characteristics that correspond to intense anger/poor anger control (BPD #8) and affective instability (BPD#6), respectively. Indeed, research evidence supports large correlations with trait anger (Arita & Baer, 1998), aggressive behavior and irritability (Arita & Baer, 1998; Cashel, Rogers, Sewell, & Holliman, 1998; Veltri et al., 2009), and moodiness (Veltri et al., 2009). Moreover, the Alienation Content scale (A-aln) indexes feelings of interpersonal alienation, frustration, withdrawal, and isolation (Archer, 2005). Such individuals are unlikely to be trusting of others and will question the loyalty of those close to them (Tellegen & Waller, 2008). This underlying construct is related to the BPD criterion of intense and/or unstable relationships (#2).

The Conduct Problems Content scale (A-con) is associated with poor impulse control, risk-taking, and proclivity towards an assortment of antisocial and otherwise self-harming behavior (Archer, 2005; Williams et al., 1992). This scale seems directly relevant to the impulsivity/self-harm BPD criterion (#4). The Conduct Problems Content Component scale Acting Out Behavior (A-con1) is more specifically linked to poor impulse control, which also has relevance to the BPD suicidality criterion (#5). Research with the A-con scale has supported its association with impulsive and externalizing behavior, antisociality, interpersonal defiance, and drug and alcohol abuse (Arita & Baer, 1998; Veltri et al., 2009).

Two Supplementary scales, Immaturity (IMM; Archer, Pancoast, & Gordon, 1994) and MacAndrew Alcoholism Scale-Revised (MAC-R), also reflect constructs relevant to BPD assessment. IMM is a broader scale that indexes psychological maturation and ego/identity development (Archer, 2005). It maps onto two BPD criteria, including identity disturbance (#3) and inappropriate anger/aggression (#8). Research on this scale has revealed that youth who score high tend to be egocentric, have poor identity development, be quick to anger, engage in bullying behavior, and have significant interpersonal and academic difficulties (Archer et al., 1994; Imhof & Archer, 1997; Zinn, McCumber, & Dahlstrom, 1999). The MAC-R scale is a broad-based measure of personality characteristics that are relevant to risk for alcohol and drug abuse, and reflects a propensity towards impulsivity, risk-taking, and sensation seeking (Archer, 2005), which has been confirmed by a number of research studies (e.g., Archer et al., 1989; Archer & Klinefelter, 1992; Veltri et al., 2009). This scale maps onto the impulsivity BPD criterion (#4).

The Bizarre Mentation Content scale (A-biz), at least at moderate levels, reflects unusual thought processes, strange experiences (including dissociation), and paranoid ideation. Like PSYC, this scale would be directly relevant to the BPD criterion of transient, stress-related paranoid or dissociative symptoms (#9). Archer and Gordon (1991) found that this scale was related to poor reality testing and emotional dyscontrol; in some adolescents, it has also been linked to poor impulse control (Veltri et al., 2009; Williams et al., 1992).

Finally, in *DSM-5* Section III, a new alternative dimensional model for BPD and other personality disorders will be defined by maladaptive constellations of dimensional traits facets (rather than trait domains) and impairment criteria (Skodol, 2012) that at least superficially also appear to resemble personality traits. The mapping of *DSM-IV*/*DSM-5* Section II and *DSM-5* Section III criteria onto one another, as well as MMPI-A measurement is depicted in Table 6.3. As can be seen from this table, the MMPI-A scales just described do not only map onto *DSM-IV* criteria but many of them also map onto trait facets proposed for *DSM-5* Section III PDs. For instance, the A-dep scale is reflective of depressivity, A-anx of anxiousness, and A-con1 of impulsivity. This information can be particularly useful as the DSM eventually transitions between these approaches to defining BPD.

In conclusion, there is considerable reason to believe that the content (and in particular, content component) and supplementary scales just discussed can provide for a more nuanced measurement of BPD. They map onto both *DSM-IV*/*DSM-5* Section II criteria as well as *DSM-5* Section III facets. Moreover, the constructs underlying these specific scales can all be viewed as facets of negative affectivity and disinhibition of the most commonly used personality/temperament personality models proposed and validated in the literature.

Clinical Implications and Future Directions

The goal of the current chapter was to provide a conceptualization of youth BPD from the perspective of the MMPI-A. BPD is one of the most controversial and challenging disorders to diagnose in adolescent mental health settings (Sharp & Romero, 2007). Since the MMPI-A is one of the most frequently used adolescent personality inventories (Archer & Krishnamurthy, 2002), it may be a critical measure for future assessment of emerging BPD. As noted earlier, there may be particular clinical profiles in which the inclusion of the MMPI-A may be valuable in assessing emerging BPD symptoms. Given the length of the MMPI-A, it may be difficult to include the measure in all assessment batteries, but as noted earlier, adolescents who may be at risk for BPD are likely to show a constellation of disinhibition, negative affectivity, and psychoticism. Such children are likely to present with disorders such as ADHD and/or ODD/CD along with comorbid internalizing disorders such as anxiety and depression. Early detection of emerging BDP symptoms may be particularly important in order to target these symptoms before they become more stable and treatment resistant.

In relation to the MMPI-A more specifically, clinicians can use the MMPI-A to gather evidence about BPD symptoms and criteria at varying levels of specificity. The MMPI-A Clinical scales have the most extensive validation, with respect to differential diagnosis of BPD, but are also the least specific. A combination of scale elevations on Scales 2, 4, 6, 7, and 8 in particular should alert the clinician to the potential presence of BPD symptoms. Adhering to a more direct conceptualization of temperament traits underlying youth BPD, the MMPI-A PSY-5 scales provide measurement that directly maps onto the domains of negative affectivity (i.e., NEGE) and (lack of) inhibitory control (i.e., DISC). We also contend, based on classic definitions of BPD, clinical presentations, and empirical evidence presented in this chapter, that psychoticism is an important domain that might indicate BPD symptoms in at least some youth with the disorder. Thus, a combination of NEGE, DISC, and/or PSYC should alert the clinician to the potential maladaptive personality pattern that underlies youth BPD.

Although directly relevant to personality psychopathology, the PSY-5 scales nonetheless do not provide sufficient specificity for the assessment of youth BPD. Therefore, clinicians should also consult the specific Content, Content Component, and Supplementary scales outlined earlier. These scales represent more specific measurement of symptoms of BPD. For instance, an adolescent who produces elevations on NEGE and DISC, but also A-lse1, A-lse2, A-con, A-dep (and particular A-dep1, A-dep4), A-ang1, A-ang2, and IMM is going to present a profile reflecting intense self-doubt, sensitivity to criticism, poor ego development (A-lse1, IMM; BPD#3), poor impulse control and risk-taking behavior (A-con1; BPD#4), suicidality and possible self-mutilation (A-dep, A-dep4; BPD#5), emotional instability and mood reactivity (including intense dysphoria, irritability) (A-dep1, A-ang2; BPD#6), and intense anger control problems and aggression (A-ang1, IMM; BPD#8).

Although this conceptual analysis of MMPI-A scales can be linked to extant literature relevant to youth BPD, most of the specific links indicated here have not received extensive empirical validation, especially in adolescent samples. As such, future research needs to directly validate the proposed conceptualization in youth carefully diagnosed with BPD using MMPI-A scale scores. It would be important to incorporate extant measurement of the temperament domains outlined in our conceptual formulation of youth BPD (i.e., negative affectivity and inhibitory control) in addition to measurement of the personality trait system included in *DSM-5* Section III. Furthermore, sophisticated statistical methods, such as latent class analysis (or preferably, factor mixture modeling; see Hallquist & Pilkonis, 2012) can be utilized to examine whether patterns of MMPI-A profiles reflect differential manifestations of BPD in youth samples. Finally, it will be important for scholars to evaluate the utility of MMPI-A assessment of youth BPD in a range of samples. In addition to outpatient and inpatient mental health samples, the behaviors manifested by many youth per Nigg et al.'s (2005) developmental trajectories for BPD strongly suggest varying levels of antisociality; thus, forensic samples would be important to examine as well.

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Developmental Manifestations of Borderline Personality Pathology from an Age-Specific Dimensional Personality Disorder Trait Framework

7

B. De Clercq, M. Decuyper, and E. De Caluwé

There are many ways to conceptualize adult borderline personality pathology, either along established dimensions of various personality pathology taxonomies (Schedule for Nonadaptive and Adaptive Personality; SNAP, Clark, 1993; Dimensional Assessment of Personality Pathology-Basic Questionnaire; DAPP-BQ, Livesley, 1990) or along the more traditional DSM-criteria (DSM-IV-TR, American Psychiatric Association [APA], 2000) that are strongly embedded within adult diagnostic procedures. The clinical manifestation of adult borderline personality disorder (BPD) is complex and includes impulsive behaviors, marked affective instability, unstable intimate relationships, and neuropsychological abnormalities, such as dissociative experiences (Paris, 2005a). This clinical richness and the associated high level of impairment have challenged many research groups to understand more firmly the etiology of borderline pathology (Cartwright, 2008; Paris, 2009).

The path towards the etiological roots of BPD is slippery, however, because there is no one-to-one relationship between the child and adult borderline construct (Paris, 2005b). Although there are shared elements between the child and adult BPD (Sharp & Romero, 2007), the assumption that all adult patients with BPD have a clear history of childhood BPD symptoms stands in contrast to the reality of the development of personality disorders, and reduces the etiological complexity of psychopathology into a linear one-dimensional framework. In addition, the construct, as such, implies that adult BPD symptoms cannot be defined from a number of clear-cut risk factors, but rather develop from multiple adversities (Bradley, Jenei, & Westen, 2005; Cartwright, 2008; Paris, 2009) that tend to cumulate with age into a complex clinical picture that was historically framed at the border of neurosis and psychosis (Kernberg, 1984). Beyond these challenges, unraveling the content and significance of BPD precursors is complicated by the fact that (1) a childhood diagnosis of BPD is seen as controversial because of its stigmatizing effect (Cartwright, 2008; Chanen & McCutcheon, 2008), that further impeded longitudinal research and resulted in a lack of empirical evidence (Carlson, Egeland, & Sroufe, 2009; Cohen, Crawford, Johnson, & Kasen, 2005) and (2) because childhood taxonomies of personality (e.g., the Hierarchical Personality Inventory for Children; HiPIC, Mervielde & De Fruyt, 1999, 2002; Mervielde, De Fruyt, & De Clercq, 2005; ICID, Halverson et al., 2003) and personality pathology (e.g., the Shedler-Westen Assessment Procedure-200; SWAP-200-A, Westen, Shedler, Durrett, Glass, & Martens, 2003 or the Dimensional Personality Symptom Itempool; DIPSI, De Clercq, De Fruyt, Van Leeuwen, & Mervielde, 2006) have only recently gained their status next to their adult

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counterparts, which hindered the conceptualization of trait vulnerabilities in younger age groups for a long time (Widiger & Clark, 2000).

Given the above, it is tempting to conceptualize the childhood BPD construct into workable and trait-oriented clusters of symptoms. In addition, the question of normality or abnormality of the childhood BPD construct creates a tension that can be understood from the specific developmental context of childhood that tends to normalize early BPD-related symptoms on the one hand and the importance of describing potential at-risk traits in an early stage of development on the other (Chanen & Kaess, 2012). An empirically based and age-specific proposal of potential BPD-related traits in younger age groups may therefore be the safest strategy for developing a well-balanced and valid childhood BPD profile. From this perspective, the current chapter presents data on early trait correlates of adult-related BPD pathology. More specifically, the childhood BPD trait profile will be framed from the DIPSI (De Clercq et al., 2006), an established taxonomy of early maladaptive traits that offers an age-specific and comprehensive description of early maladaptive personality tendencies. This specific dimensional approach for delineating a childhood BPD profile can be advocated from recent evidence suggesting that childhood BPD dimensions are measurable and relatively stable across time (Chanen & Kaess, 2012).

The Controversial Character of Childhood BPD Pathology

Personality disorder precursors in childhood and adolescents have received little systematic attention in the literature (Widiger, De Clercq, & De Fruyt, 2009) for reasons that have been extensively reviewed in previous publications (De Fruyt & De Clercq, 2012; Tackett, 2010). Although more recent evidence on the stability and predictive validity of childhood trait dimensions (Caspi, Roberts, & Shiner, 2005; De Clercq, Van Leeuwen, Van den Noortgate, De Bolle, & De Fruyt, 2009; De Fruyt et al., 2006) has encouraged the field to invest in longitudinal research programs on the course of personality disorders, the developmental trajectory of BPD still remains largely unclear (Crowell, Beauchaine, & Linehan, 2009). Much of the existing knowledge on environmental childhood risk factors for BPD pathology results from retrospective studies in adults with personality pathology (e.g., Elzy, 2011; Klonsky, Oltmanns, Turkheimer, & Fiedler, 2000), indicating that family adversity, including frequent conflict, low social support, parental neglect, as well as sexual and physical abuse, are closely associated with borderline personality features in adulthood.

From a child perspective, the term *borderline personality of childhood* (*BPC*) historically describes a complex and severe form of behavioral pathology, with a mixed clinical picture of externalizing, internalizing, and cognitive symptoms (Ad-Dab'bagh & Greenfield, 2001). Geiger and Crick (2001) more specifically identified cognitive and emotional sensitivity, the desire to have exclusive friendships, and relational aggression as indicators of BPD pathology in childhood, and Crick, Murray-Close, and Woods (2005) showed that these childhood characteristics empirically tracked together with the scores on an independent BPD measure over the course of 1 year. The childhood BPD syndrome has also been labeled as *multiple complex developmental disorder* (Lincoln, Bloom, Katz, & Boksenbaum, 1998), reflecting the complex nature of the displayed abnormalities in BPC. A sparse number of prospective studies have shown that these BPD features are rather stable over time (Cohen et al., 2005; Crick et al., 2005), and that BPC children continue to exhibit severe problems over time (Paris, 2003). Zelkowitz et al. (2007) for instance showed that children diagnosed with BPC were more likely to exhibit a combination of both internalizing and externalizing problem behaviors 5–7 years later and also showed a more impaired functional status compared with a general group of referred children without BPD features. Likewise, Belsky et al. (2012) demonstrated from a longitudinal twin-study that 5-year-old children with poor cognitive functioning, impulsivity, and behavioral–emotional problems more commonly

developed into early adolescents with a BPD-related profile.

Across a wider age-span, Carlson et al. (2009) found that childhood measures of activity and emotionality, and adolescent measures of attention, emotion regulation, and relational functioning maintained to be predictors of BPD symptomatology in adulthood. Burke and Stepp (2012), Stepp, Burke, Hipwell, and Loeber (2012) suggested from an Axis I perspective that childhood attention deficit hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) predicted BPD symptoms at age 14 and 24, respectively, indicating that ADHD shares the clinical features of impulsivity, poor self-regulation and executive functioning with BPD, whereas ODD reflects a shared element with BPD in terms of anger and interpersonal turmoil.

Although these results indicate that BPD can be traced back to childhood, it seems rather difficult to delineate the core aspects of BPD precursors in terms of well-defined constructs. It must also be stressed that early maladaptive traits are subject to maturational influences (Lenzenweger & Castro, 2005) and interact with environmental influences (Hooley, Cole, & Gironde, 2012), with many children with BPD characteristics turning into adaptive individuals. For the minority of children with a rather stable profile of symptomatology throughout childhood and adolescence, it is in addition important to note that they are not exclusively at risk for later BPD pathology, but are in general more prone to display various patterns of personality disorders (Lofgren, Bemporad, King, Lindem, & Odriscoll, 1991) when they grow older. This heterogeneity in developmental trajectories of BPD pathology can mainly be understood from a number of developmental principles or methodological issues (De Fruyt & De Clercq, 2012). From a conceptual viewpoint, however, an additional reason why evidence on the childhood BPD profile is so divergent and difficult to grasp in terms of its underlying features is because the operationalization of the childhood BPD construct varies substantially across studies and is in addition often solely based upon conceptual criteria (Geiger & Crick, 2001) or derived from adult measures (i.e., the Children in the Community Study; Cohen et al., 2005). Until recently, no studies relied on a comprehensive and age-specific personality pathology trait measure that was able to propose trait-based risk factors for later BPD pathology from an inclusive and child-oriented perspective. In an attempt to address this issue, the identification of BPD-related traits in the present chapter will rely on a taxonomy of maladaptive traits that was specifically developed to describe childhood manifestations of personality pathology within a dimensional framework.

Dimensional Assessment of Personality Pathology Traits in Childhood: The DIPSI

Combining a bottom-up and a top-down approach, De Clercq et al. (2006) constructed a broad and comprehensive taxonomy of trait-related symptoms in childhood. This Dimensional Personality Symptom Item Pool (DIPSI) was primarily developed from the more extreme item content of the personality items of a childhood FFM measure (i.e., the HiPIC; Mervielde & De Fruyt, 1999). The initial pool of maladaptive HiPIC descriptors was extended with a top-down strategy, by adding items of adult measures for personality pathology (i.e., the Structured Clinical Interview for DSM-IV Axis-II Personality Disorders (SCID-II); First, Gibbon, Spitzer, Williams, & Benjamin, 1997 and the Assessment of DSM-IV Personality Disorders (ADP-IV); Schotte, de Doncker, Vankerckhoven, Vertommen, & Cosyns, 1998), that were assumed to be relevant in younger age groups. The resulting DIPSI item set comprises 172 items that are structured in 27 maladaptive personality facets, hierarchically organized in four broad personality dimensions. This four-dimensional structure is consistent with O'Connor (2005), Saulsman and Page (2004), and Widiger and Simonsen (2005), and is further referred to as disagreeableness (including extreme low-end variants of benevolence and conscientiousness and high-end variants of

extraversion), emotional instability (referring to both anxious and depressive traits and also including a dependency component), introversion (describing extreme low-end variants of extraversion such as withdrawn traits and shyness), and compulsivity (including the extremes of conscientiousness traits such as perfectionism and extreme order).

The DIPSI's four higher-order maladaptive trait factors represent unique trait constructs that are associated with, but distinct from the established higher-order dimensions of internalizing and externalizing problem behavior (De Clercq, Van Leeuwen, De Fruyt, Van Hiel, & Mervielde, 2008) as described in the Child Behavior Checklist (CBCL; Achenbach, 1991). Their nature is further reflected in the correlation with normal-range higher-order personality traits (De Clercq et al., 2006), and in their conceptual fit within the common higher-order framework of the FFM (Markon, Krueger, & Watson, 2005). The lower-order traits are to be interpreted along the extremes of general lower-order trait facets (Widiger et al., 2009), but additionally provide a more detailed description of pathological trait features that are not fully accounted for by general trait or temperament models (De Clercq et al., 2009). From a conceptual point of view, it has also been demonstrated that the DIPSI lower-order traits show a notable coverage of potential personality disorder precursors (De Clercq et al., 2009) as delineated by Geiger and Crick (2001). Recent studies have indicated that DIPSI facets can be used to reliably and validly describe psychopathic traits (Decuyper, De Bolle, De Fruyt, & De Clercq, 2011), obsessive-compulsive symptoms (Aelterman, De Clercq, De Bolle, & De Fruyt, 2011), and autism spectrum symptoms (De Clercq et al., 2010) in childhood and adolescence. Given these different sources of evidence that support the validity of the DIPSI for describing various disorder-based aspects of maladaptation, it seems plausible that specific DIPSI facets may also be relevant correlates of BPD-related pathology.

DIPSI Trait Correlates of BPD Pathology from a Cross-Sectional and Longitudinal Perspective

In an attempt to empirically delineate which age-specific facets of personality pathology are associated with BPD pathology, the current chapter presents data from two different perspectives. In a first approach, we will explore how DIPSI facets are cross-sectionally associated with two established operationalizations of BPD that are embedded within either the categorical or the dimensional tradition for describing personality disorders. A second approach takes a longitudinal perspective and focuses on the longitudinal associations of DIPSI traits with a dimensional operationalization of BPD, as advised by the most recent DSM-5 proposal of the APA Board of Trustees (Arlington, December 1, 2012).

Both perspectives rely on different samples and will be separately described in the next section. Based upon these results, a comprehensive empirically based childhood BPD profile of DIPSI traits will be proposed.

A Cross-Sectional Perspective on Borderline PD and DIPSI Trait Precursors

Cross-sectional associations between childhood maladaptive traits and BPD symptomatology were examined in a community sample of 293 boys ($N = 144$, 49.10 %) and girls ($N = 149$, 50.90 %) from a multi-informant perspective. Age ranged from 12 to 18 years, with a mean age of 15.93 (SD = 1.35). These adolescents and their mothers were recruited via secondary schools in Flanders. Researchers and undergraduate students of Ghent University visited secondary schools, distributed inventories, and provided detailed oral and written instructions on how to complete the questionnaires. Written informed consent was obtained from all participants at

the moment of assessment. This sample was previously used for other research purposes by Decuyper, De Clercq, De Bolle, and De Fruyt (2009).

In a first analysis, the BPD-related construct was assessed using the maternal-rated borderline scale of the Flemish ADP-IV (Schotte et al., 2004) measure that includes 94 items reflecting the content of the 10 DSM-IV personality disorder criteria. The second analysis relied on the self- and maternal-rated FFM BPD count (Miller, Bagby, Pilkonis, Reynolds, & Lynam, 2005), a count technique that conceptualizes PDs from a general trait perspective and uses FFM facet score counts to index the degree of similarity between an individual's FFM scores and those expected from BPD patients. Decuyper et al. (2009) recently showed that the FFM PD counts, including the FFM borderline count, are applicable in adolescent samples. They also provided normative data and PD count benchmarks enabling the use of FFM scores for PD screening purposes in adolescence.

Pearson correlations between maternal-rated DIPSI facets and the categorical ADP-IV BPD scale (see Table 7.1) show that BPD pathology is significantly ($p < 0.002$ according to the Bonferroni correction) and positively associated with all DIPSI facets, except for the Compulsivity facet Extreme Order. A similar picture is obtained for the maternal-rated FFM BPD count, which is positively correlated with most of the DIPSI facets, except for inflexibility, perfectionism, extreme achievement striving, extreme order, and withdrawn traits. This overall maladaptive trait profile of BPD pathology across the childhood trait domains of disagreeableness, emotional instability, and introversion may be understood from the idea that BPD-related pathology in childhood and adolescence is characterized by a complex clinical picture of internalizing, externalizing, and cognitive problems (Ad-Dab'bagh & Greenfield, 2001). These observed correlations may however also be inflated because of the same-informant design, or may reflect that the DIPSI traits capture a more general component of dysfunction. Beyond the overall association of the adult BPD construct with childhood maladaptive traits, there is however a specific set of DIPSI traits that strongly correlates ($r \geq 0.30$; Cohen, 1988) with the adult BPD construct across informants (using self-ratings for the FFM BDL count and maternal DIPSI ratings). These are affective lability, impulsivity, ineffective coping, hyperexpressive traits, irritable–aggressive traits, and risk behavior. The blending of these traits results in a trait profile that conceptually aligns with the definition and previous findings on the childhood BPD construct (Paris, 2003, 2005c) hence underscoring the validity of each of these childhood maladaptive traits as relevant age-specific descriptors of BPD-related pathology.

A Longitudinal Perspective on Borderline PD and DIPSI Trait Precursors

The longitudinal analyses rely on three different samples, including one same-informant and two cross-informant designs. The first sample ($N = 501$; 65.5 % girls; 8–18 years old, $M = 12.93$, $SD = 2.26$), comprises data from children of the general population as well as referred children, that were combined in order to maximize the variability in scores. Mothers provided information on their children's maladaptive personality traits (DIPSI) at Time 1 and also assessed BPD prototype-related traits 4 years later, as measured with the proposed trait measure for describing personality pathology in DSM-5 (Personality Inventory for DSM-5 (PID-5); Krueger et al., 2011). The PID-5 measure consists of 25 facets of personality pathology that can be empirically structured in five broadband domains of maladaptive personality variation (i.e., negative affect, detachment, antagonism, disinhibition, and psychoticism). The use of this dimensional trait measure in the current study can be understood from the recent suggestion of the APA (Arlington, December 1, 2012), stating that the implementation of a dimensional trait model in future editions of the DSM requires more research in order to fully underpin this "categorical to dimensional" shift.

Table 7.1 Bivariate correlations between DIPSI scales and the ADP-IV borderline scale and FFM borderline counts

	BDL ADP-IV maternal	BDL PD count maternal	BDL PD count self
Maternal-rated DIPSI facets			
DIS: Hyperexpressive traits	0.54*	0.57*	0.31*
DIS: Hyperactive traits	0.53*	0.47*	0.24*
DIS: Dominance—Egocentrism	0.53*	0.49*	0.29*
DIS: Impulsivity	0.65*	0.63*	0.34*
DIS: Irritable—Aggressive traits	0.70*	0.62*	0.30*
DIS: Disorderliness	0.50*	0.48*	0.29*
DIS: Distraction	0.56*	0.48*	0.20*
DIS: Risk behavior	0.55*	0.48*	0.30*
DIS: Narcissistic traits	0.49*	0.43*	0.23*
DIS: Affective lability	0.73*	0.67*	0.38*
DIS: Resistance	0.59*	0.45*	0.19*
DIS: Lack of empathy	0.47*	0.32*	0.11
INS: Dependency	0.55*	0.46*	0.16
INS: Anxious traits	0.61*	0.58*	0.25*
INS: Lack of self-confidence	0.57*	0.50*	0.27*
INS: Insecure attachment	0.58*	0.42*	0.15
INS: Submissiveness	0.43*	0.32*	0.12
INS: Ineffective coping	0.68*	0.67*	0.33*
INS: Separation anxiety	0.58*	0.46*	0.24*
INS: Depressive traits	0.70*	0.59*	0.28*
INS: Inflexibility	0.61*	0.40*	0.14
ITR: Shyness	0.47*	0.29*	0.08
ITR: Paranoid traits	0.53*	0.37*	0.13
ITR: Withdrawn traits	0.36*	0.12	−0.03
COM: Perfectionism	0.34*	0.17	0.03
COM: Extreme achievement striving	0.23*	0.14	0.05
COM: Extreme order	0.15	−0.04	−0.06

DIS Disagreeableness, *INS* Emotional Instability, *COM* Compulsivity, *ITR* Introversion
*$p < 0.002$ according to the Bonferroni adjustment

In order to define the BPD construct relying on PID-5 scores, we summed the scores on the DSM-5 traits that have been appointed as indicative of BPD pathology (www.DSM-5.org): Emotional Lability, Anxiousness, Separation Insecurity, Depressivity, Impulsivity, Risk Taking, and Hostility.

The cross-informant perspective was implemented in two different samples, including a mixed sample of community and referred children ($N = 307$; 61.7 % girls; 8–16 years old, $M = 11.67$, $SD = 1.77$), with maternal ratings on the DIPSI at Time 1 and PID-5 self-ratings 4 years later, and a population sample of children ($N = 370$; 65.4 % girls; 7–18 years old, $M = 12.84$, $SD = 2.47$), with self-ratings on the DIPSI at Time 1 and maternal PID-5 ratings 4 years later (see Table 7.2). All samples were part of the ongoing Personality and Longitudinal Affect Study of Ghent University, as already described in De Bolle, Beyers, De Clercq, and De Fruyt (2012) and in De Clercq et al. (2009).

Hierarchical regression analyses were conducted to empirically explore which DIPSI facets are able to predict the childhood BPD prototype, either from a same- or a cross-informant perspective and each time controlling for gender and age (in Step 1).

All DIPSI facets that correlated substantially (≥0.30; Cohen, 1988) across the categorical

Table 7.2 Cross-informant hierarchical regression analysis predicting the borderline prototype (T2) from DIPSI traits (T1)

Ratings	Mother-ratings (T1) → Self-ratings (T2) ($N = 307$)						Self-ratings (T1) → Mother-ratings (T2) ($N = 370$)					
Step and variable	B	S.E.	β	R^2	F	F_{change}	B	S.E.	β	R^2	F	F_{change}
DV: Borderline Prototype												
Step 1				0.00	0.31	0.31				0.03	4.65*	4.65*
Age	−0.05	0.08	−0.04				0.04	0.05	0.04			
Sex	0.16	0.30	0.03				0.71	0.25	0.15**			
Step 2				0.13	5.63***	7.39***				0.10	5.22***	5.30***
DIS: Hyperexpressive traits	0.12	0.25	0.04				−0.01	0.25	−0.00			
DIS: Impulsivity	0.04	0.26	0.01				−0.22	0.20	−0.07			
DIS: Irritable–aggressive traits	−0.15	0.36	−0.05				0.61	0.25	0.19*			
DIS: Risk behavior	0.22	0.30	0.06				0.18	0.20	0.06			
DIS: Affective lability	1.04	0.32	0.37**				0.03	0.20	0.01			
INS: Ineffective coping	−0.08	0.27	−0.03				0.36	0.21	0.13			

DIS Disagreeableness, *INS* Emotional Instability, *ITR* Introversion
*$p < 0.05$. **$p < 0.01$. ***$p < 0.001$

Table 7.3 Same-informant hierarchical regression analysis predicting the borderline prototype (T2) from DIPSI traits (T1) (both mother-ratings) ($N = 501$)

Step and variable	B	S.E.	β	R^2	F	F_{change}
DV: Borderline prototype						
Step 1				0.03	7.72**	7.72**
Age	−0.18	0.05	−0.15**			
Sex	0.58	0.25	0.11*			
Step 2				0.31	27.07***	32.52***
DIS: Hyperexpressive traits	0.01	0.19	0.00			
DIS: Impulsivity	0.03	0.19	0.01			
DIS: Irritable–aggressive traits	0.90	0.27	0.26**			
DIS: Risk behavior	0.21	0.22	0.05			
DIS: Affective lability	0.54	0.24	0.18*			
INS: Ineffective coping	0.30	0.20	0.10			

DIS Disagreeableness, *INS* Emotional Instability, *ITR* Introversion
*$p < 0.05$. **$p < 0.01$. ***$p < 0.001$

(ADP-IV BDL scale) and dimensional (FFM BDL count) BPD-related constructs as reflected in Table 7.1 were entered as a block of predictors (i.e., hyperexpressive traits, impulsivity, irritable–aggressive traits, risk behavior, affective lability, and ineffective coping) with the DSM-5 BPD prototype as dependent variable (see Table 7.3).

Same-informant ratings. In the prediction of the BPD prototype, Table 7.3 shows that Step 1 explained 3 % of the variance, with age as a significant negative predictor and sex as a significant positive predictor. Boys had lower BPD prototype scores compared to girls. In Step 2, 31 % of the BPD prototype variance was explained with irritable–aggressive traits as well as affective lability as significant predictors.

Cross-informant ratings. Predicting the self-rated BPD prototype based upon maternal DIPSI ratings (see Table 7.2), Step 1 explained no variance. Childhood maladaptive traits appeared to explain 13 % of the variance of the BPD type 4 years later, with affective lability as the only significant predictor. Predicting the maternal-rated BPD type from self-ratings on the DIPSI, gender showed to be a significant predictor in a first step of the regression analysis, with boys scoring lower on the BPD prototype compared to girls. In Step 2, self-rated maladaptive traits explained 10 % of the variance of the BPD prototype, with irritable–aggressive Traits as significant predictor. To summarize, this longitudinal same- and cross-informant perspective clearly underscores that BPD pathology can be predicted from childhood personality difficulties, suggesting that irritable–aggressive traits and affective lability are two core predictive components for later BPD-related difficulties.

An Age-Specific Dimensional Proposal for Childhood BPD Pathology

The present results indicate that adult operationalizations of BPD can be predicted from childhood maladaptive traits, with irritable–aggressive traits and affective lability as the most significant precursors, and in a broader sense also hyperexpressive traits, impulsivity, risk behavior, and ineffective stress coping. This empirically based childhood maladaptive trait description of BPD disorder captures a significant part of the established clinical picture of BPD disorder, but is not inclusive. More specifically, the emotional instability component may be somewhat underrepresented, since only ineffective coping showed to be a significant correlate across raters. BPD is however described as a disorder with a considerable internalizing component, such as manifest anxious and depressive traits (Belsky et al., 2012;

Table 7.4 An age-specific dimensional childhood borderline profile from the DIPSI framework

DIPSI facet	Score	Description
Hyperexpressive traits	High[a]	The child behaves in a way to stand out. He or she constantly expresses feelings and thoughts in an inappropriate way. The environment often interprets these expressions as disturbing
Impulsivity	High	The child acts and reacts impulsively, both in social situations and cognitive tasks
Irritable–aggressive traits	High	The child is easily frustrated and expresses his anger in an uncontrolled way
Risk behavior	High	The child is fearless and experiences a hunger for adventure and excitement
Affective lability	High	The child displays extreme mood swings
Ineffective stress coping	High	The child cannot cope with stress and is easily overwhelmed by emotions
Anxious traits	High	The child often worries and experiences an unrealistic fear. The child reacts overanxious in a variety of situations
Lack of self-confidence	High	The child considers himself/herself as less important than others. The child has a low self-esteem and doesn't believe in his/her own capacities
Insecure attachment	High	The child shows clinging behavior that is age-inappropriate
Depressive traits	High	The child is easily discouraged and often pessimistic
Paranoid traits	High	The child is suspicious and has no confidence in other people

[a]A decile of eight or more is considered to represent a high score. Normative data are available upon request

Hooley et al., 2012) and characteristic insecure attachment patterns (Scott, Levy, & Pincus, 2009). Although the content of the DIPSI facet ineffective coping is broad, representing both dysfunctional stress coping, a sensitivity to stress, and the tendency to be easily overwhelmed by emotions, including emotions of anxiety and panic, it can be assumed that the DIPSI facets anxious traits, lack of self-confidence, insecure attachment, and depressive traits may be significant additional indicators in order to describe the internalizing aspect of the BPD profile in the most comprehensive way. Also, the DIPSI scale paranoid traits can be expected to be a prominent correlate from a conceptual point of view, because of the brief episodes of psychotic or paranoid thinking that are often observed in children (Paris, 2003; Zelkowitz et al., 2007) with BPD features. Moreover, the symptom "transient, stress-related, paranoid ideation, or severe dissociative symptoms" is also included in the DSM-IV (APA, 1994) diagnostic BPD criteria for adults, and research has shown that approximately 75 % of the BPD patients had paranoid ideas and/or experienced episodes of dissociation (Hooley et al., 2012).

From these empirical findings and additional conceptual arguments, we propose a comprehensive childhood BPD profile from the DIPSI framework (see Table 7.4 for further description) that may provide the most inclusive age-specific description of the traits that are relevant at a young age for understanding later personality difficulties in terms of BPD pathology. We assume that children who are overly expressive, impulsive, and irritable, and who experience mood swings, anxiety, little self-confidence, and display an insecure attachment pattern, together with a behavioral profile of risk behavior and ineffective coping strategies, are most at risk for developing a maladaptive personality that aligns with the adult concept of BPD.

Conclusions

Although the burden of BPD is broadly documented (Crowell et al., 2009; Skodol et al., 2002), research focusing on its developmental precursors has been lacking for a long time. This absence of a life-span perspective is shared by almost all personality disorders, except for antisocial personality disorder that has been systematically linked to childhood conduct disorder since DSM-III (APA, 1980). The tremendous individual, family and public cost of BPD can however at least be considered noteworthy, with many of the BPD patients showing a lasting unstable and

destructive pattern of psychosocial functioning, chronic relapse, and societal isolation. This impaired outcome of adult BPD signifies the importance of identifying and treating those individuals that are at risk for developing BPD at a much younger age than early adulthood, especially because there is evidence that developmental BPD manifestations show a higher level of plasticity in younger age groups (Lenzenweger & Castro, 2005). Recently, Sharp, Ha, Michonski, Venta, and Carbone (2012) indicated, however, that early intervention is hindered by the fact that there are few reliable and valid measures with which to identify youth with BPD traits. Corroborating this statement, we argue that studies on BPD precursors that include child measures developed to describe trait pathology in a broad sense, may contribute in a unique way to this research field compared to specific childhood BPD measures. Although a recent measure of childhood BPD traits (i.e., the Borderline Personality Features Scale for Children (BPFS-C); Crick et al., 2005) appears to be promising in terms of psychometric properties and validity (Chang, Sharp, & Ha, 2011), only a comprehensive trait measure takes into account that the borderline outcome may develop from other traits than the "typical" childhood equivalents of borderline pathology (i.e., equifinality, Cicchetti & Rogosch, 1996). Moreover, a dimensional trait measure meets a number of age-specific problems associated with the traditional categorical assessment, such as the ability to detect subtle variations in scores over time that have naturally been observed in younger age groups (Lenzenweger & Castro, 2005: Miller, Muehlenkamp, & Jacobson, 2008).

From such a comprehensive child-specific maladaptive trait perspective, the current chapter presents data on early dimensional trait correlates of various borderline operationalizations embedded within both the categorical and dimensional research tradition, across three independent samples of Flemish children. The data are characterized by a multi-informant design and frame borderline-related precursors from both cross-sectional and longitudinal associations of the DIPSI maladaptive traits with an adult borderline measure. Cross-sectional analyses suggest that six specific DIPSI trait facets are replicable correlates across both categorical and dimensionally oriented operationalizations of BPD, suggesting that these traits may capture a substantial part of core variance of the borderline construct. The majority of these traits represent an age-specific reflection of the typical borderline profile, such as impulsivity, irritable–aggressive traits, affective lability, and ineffective stress coping. Two other traits, hyperexpressive traits and risk behavior, represent a rather new perspective on potential relevant developmental manifestations of borderline disorder. Although both traits are subsumed under the disagreeableness trait domain and can hence be considered as indicators of the established externalizing trait component of borderline disorder, they may intuitively not be the most obvious externalizing traits that one would select as a borderline precursor. On the other hand, they can both be assumed to share some etiological factors with the DIPSI trait impulsivity (De Clercq et al., 2006), and can hence be considered as two traits that may broaden the core impulsivity domain towards associated features of impulsivity-related maladaptation (Cooper, Wood, & Orcutt, 2003). From the principle of heterotypic continuity (Cicchetti & Crick, 2009), stating that the phenotypic manifestation of a single underlying trait may vary with age, hyperexpressive traits and risk behavior may also signify an age-specific expression of an established adult borderline trait, and hence represent a more evident aspect of borderline disorder than initially assumed.

Out of these six DIPSI trait correlates, two facets remain significant predictors of the adult BPD construct in an independent longitudinal and multi-informant design, with irritable–aggressive traits and affective lability representing developmental expressions of

later BPD symptomatology. Both of these DIPSI facets share their disagreeable and emotional instability component (see De Clercq et al., 2006 for significant loadings on both dimensions), and may provide a viable developmental explanation for the extensive internalizing–externalizing comorbidity in BPD that has been repeatedly illustrated in empirical studies (Gunderson, 2001; Skodol et al., 2002). Conceptually, these results connect with the biosocial developmental model of borderline personality of Linehan (1993) at the descriptive level, in which various aspects of emotional dysregulation (such as irritable–-aggressive traits and affective lability) are proposed as core child-related vulnerabilities for developing BPD. Future research should however examine to what extent these two traits represent a comprehensive representation of those child factors that contribute to the ontogenesis of BPD.

From this essential issue of the need for comprehensiveness, we therefore propose additional conceptually based indicators of childhood borderline pathology, that broaden the assessment of the childhood BPD-related symptoms towards the aspect of anxiousness, depressive traits, insecure attachment, and paranoid traits. This DIPSI childhood borderline profile affords the opportunity to adopt a broad but age-specific BPD operationalization in future studies on the development of borderline disorder, and may further be valuable in studies on heritability estimates of BPD at the specific age of childhood, given that this research area is still rather unexplored (Bornovalova, Hicks, Iacono, & McGue, 2009).

From a critical point of view, the present results should be interpreted as tentative, given the short follow-up time span as well as the restriction of only two assessment points. Also, the childhood BPD profile was exclusively delineated from a descriptive level of associations, and does not guarantee that biologically based precursors of adult BPD are covered. Future research should in addition explore to what extent the proposed DIPSI BPD traits interact with the multiple contextual factors that have been identified as environmental risk factors for BPD (e.g., Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Widom, Czaja, & Paris, 2009), and should further verify the various developmental trajectories of those children that present high scores on the proposed DIPSI BPD traits in relation to the specific developmental context of the child. Only large prospective studies will be able to identify how these early traits can be considered as direct vulnerability factors for a BPD outcome, and to what extent the transactional processes of these traits with the environment lead to BPD maladaptation. In addition, future research that incorporates multiple maladaptive traits as predictors of later BPD pathology should empirically delineate the relative contribution of each of these traits in terms of their impact on the developmental course of the child and the eventual outcome.

In sum, the current data suggest that the DIPSI framework generates a number of facets that are significantly associated with adult operationalizations of BPD across multiple informants, and suggest that the DIPSI measure may contribute to the field of childhood assessment of BPD pathology from a behaviorally oriented and non-stigmatizing perspective.

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Conceptualizing Youth Borderline Personality Disorder Within a Normative Personality Framework

8

Jennifer L. Tackett and Shauna C. Kushner

Broadly conceived, personality scientists emphasize an enhanced understanding of individual differences in thoughts, feelings, and behavior (e.g., John, Naumann, & Soto, 2008). A long-standing area of research, personality psychologists have typically focused on adult populations, with extensions to children and adolescents increasing in recent decades (e.g., Caspi, Roberts, & Shiner, 2005; Shiner & Caspi, 2003). A richer history exists, however, on temperament research in children and adolescents (Rothbart & Bates, 2006; Zentner & Shiner, 2012). In this chapter, we begin by describing individual differences approaches in younger age groups and briefly define predominant models used in current child personality research, next turning to the relevance of normal personality perspectives for understanding the development of youth borderline personality disorder (BPD).

Individual differences in children have historically been investigated under the domain of temperamental traits (Rothbart & Bates, 2006). Temperament and personality are clearly intersecting domains of study, although they have largely proceeded in parallel, with distinct researchers and measures employed (De Pauw & Mervielde, 2010; Tackett, 2006). The study of temperament emphasizes traits that are biologically based and present from very early in life, even in infancy. Temperament measures typically emphasize constructs such as physiological regulation and reactivity that are not usually seen in personality measures, although higher-order trait models of temperament and personality show substantial agreement (Tackett, Slobodskaya et al., 2012).

In recent years, researchers have substantially accelerated our understanding of child personality by elucidating our theoretical understanding of child personality constructs (Shiner, 1998; Shiner & Caspi, 2003) and developing comprehensive questionnaire methods to assess child personality traits (Halverson et al., 2003; Mervielde & De Fruyt, 1999). Although still in its infancy, the field of child personality research generally agrees on a five-factor higher-order structure of child personality that maps on to the familiar Five Factor Model (FFM) of adult personality (John et al., 2008). Specifically, higher-order traits in most major models of child personality include neuroticism (N), extraversion (E), agreeableness (A), conscientiousness (C), and openness to experience (O).

Higher-order trait models in temperament research overlap substantially with the child FFM, with traits typically including surgency (analogous to E), negative emotionality (analogous to N), and effortful control (analogous to C and, to some extent, A; Rothbart & Bates, 2006; Tackett, Slobodskaya et al., 2012). In addition, child personality traits are organized hierarchically

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in a manner that facilitates direct connection with both temperament and adult personality models (Tackett, Slobodskaya et al., 2012). Specifically, two-factor models of child individual differences reflect broad approach- versus avoidance-motivation factors and three-factor models reflect the classic higher-order temperament structure (Tackett, Slobodskaya et al., 2012). In children, specific deviations from standard adult models of personality include issues such as the presence of activity-related traits in child personality measures that are not typically found in adult measures, higher covariance between A and N in childhood, substantial compliance-related content defining childhood A, and higher covariance between C and O in childhood (Tackett, Slobodskaya et al., 2012). In the context of this brief history of research on normative dispositional differences in youth, we rely on the child-based FFM to form the framework in this chapter, drawing liberally on relevant traits from temperament and adult personality models as well. Our goal in this chapter is to illustrate the utility of a normative personality perspective in furthering our understanding of youth BPD.

Defining Youth BPD

DSM-IV conceptualizes BPD as a pervasive and inflexible pattern of instability and impulsivity that causes distress or impairment (American Psychiatric Association [APA], 2000). Although standard clinical practice often discourages diagnosis of BPD in children and adolescents, ample evidence has converged on the early emergence of BPD characteristics (Paris, 2005). Existing reluctance toward identifying BPD in childhood and adolescence has limited our understanding of the etiological roots and early manifestations of the disorder (Crick, Murray-Close, & Woods, 2005; Miller, Muehlenkamp, & Jacobson, 2008). Although DSM-IV permits BPD diagnoses in childhood and adolescence, there remain no explicit criteria for early manifestations of BPD traits. Indeed, there is evidence for differences in the manifestation of BPD features across development (see also Chaps. 3 and 18, current volume). For example, within a sample of children and adults who met DSM-IV criteria for BPD diagnosis, preadolescent youth more often reported being angry and moody and less frequently reported paranoia/dissociation, serious identity disturbance, impulsivity, or frantic efforts to avoid abandonment than adults (Zanarini et al., 2011). One recent proposal for conceptualizing BPD in youth summarized four core dysfunctional areas, corresponding broadly to the DSM formulation: Identity disturbance, affective instability, relationship difficulties, and impulsivity (Miller et al., 2008).

In this chapter, we maintain a developmentally sensitive approach to considering BPD manifestations early in the lifespan with a focus on stable underlying dimensions, while also making efforts to connect with DSM approaches to the disorder. Table 8.1 displays the overlap between the four core dysfunctional areas defined by Miller et al. (2008) and the lower-order facets of normative measures of temperament and personality in youth. Specifically, we hoped to illustrate the extent to which existing measures of temperament and child personality already tap into core components of youth BPD (and the extent to which they do not). As you see from Table 8.1, impulsivity is the core dysfunctional area in youth BPD that is best captured by existing models of temperament and child personality. Affective instability is somewhat captured across emotional domains, although within-person variability (i.e., specifically capturing the "instability" aspect of this construct) may not be as directly assessed by existing temperament/personality measures. Relational factors are captured in both temperament and child personality measures as well, although the implied impairment associated with personality pathology is not typically directly assessed. Identity disturbance is the core dysfunctional area in youth BPD that is the most poorly assessed by existing temperament and child personality measures. Attention to this domain, in particular, may require independent measures of identity functioning and development. Overall, though, existing measures of temperament and child personality do tap into relevant constructs for core

Table 8.1 Table with overlap between traits and relevance for BPD

Core dysfunctional areas[a]	Temperament[b]	Child personality[c]
Relationship difficulties	*Affiliation*	*Altruism*$_{(HiPIC)}$
	Aggression$_{(EATQ\text{-}R)}$	Antagonism$_{(ICID)}$
	Assertiveness/Dominance$_{(TMCQ)}$	*Compliance*
		Considerate$_{(ICID)}$
		Dominance$_{(HiPIC)}$
		Sociability$_{(ICID)}$
		Strong willed$_{(ICID)}$
Identity disturbance	–	Egocentrism$_{(HiPIC)}$
		Self-confidence$_{(HiPIC)}$
Impulsivity	*Activation control*	Activity level$_{(ICID)}$
	Activity level	*Concentration*$_{(HiPIC)}$
	Approach$_{(CBQ)}$	Distractible$_{(ICID)}$
	Attention/Attentional control	Energy$_{(HiPIC)}$
	Impulsivity	*Orderliness/Organized*
	Inhibitory control	*Persistence*$_{(HiPIC)}$
Affective Instability	Anger/Frustration	Anxiety
	Depressive mood$_{(EATQ\text{-}R)}$	Expressiveness$_{(HiPIC)}$
	Discomfort	Fearful/Insecure$_{(ICID)}$
	Fear	Irritability$_{(HiPIC)}$
	Sadness	Negative affect$_{(ICID)}$

Traits in italics denote dimensions that would be negatively associated with features of core dysfunctional areas. Subscripts indicate questionnaires on which specific traits are measured. Traits without subscripts are common across measures

[a]Core dysfunctional areas identified by Miller et al. (2008)

[b]Traits measured using the Rothbart family of instruments (Children's Behavior Questionnaire [CBQ]; Temperament in Middle Childhood Questionnaire [TMCQ]; Revised Early Adolescent Temperament Questionnaire [EATQ-R])

[c]Traits measured using the child Five Factor Model of personality instruments (Hierarchical Personality Inventory for Children [HiPIC]; Inventory of Children's Individual Differences [ICID])

aspects of youth BPD. Against this framework, we next examine three theoretical approaches that illustrate the potential relevance and utility of normative personality perspectives for youth BPD: the developmental psychopathology approach, theoretical models of personality–psychopathology relationships, and hierarchical investigations of personality structure.

Theoretical Perspectives

Developmental Psychopathology

Developmental psychopathology is an integrative framework for examining adaptive and maladaptive behavioral development (Achenbach, 1974; Cicchetti, 2000; Masten, 2004, 2006; Sroufe, 1989; Sroufe & Rutter, 1984). This framework was advanced through the merger of two historically parallel lines of research—developmental psychology and psychopathology—with the purpose of promoting adaptive behavior and preventing or mitigating the impact of behavioral problems. To this end, developmental psychopathologists utilize multidisciplinary techniques, often within longitudinal designs, to examine individual and familial differences in vulnerability for behavioral disorders.

Of particular interest to developmental psychopathology are the principles of equifinality and multifinality, which describe the heterogeneity in the development of maladaptive behavior across individuals (Cicchetti & Rogosch, 1996). Specifically, equifinality refers to diverse pathways that result in the development of a

particular disorder, whereas multifinality refers to the myriad effects that a particular factor may have on a developing organism. Within this framework, psychopathology is generally considered in relation to normative functioning (e.g., whether individuals successfully master age-specific behaviors; Masten, Burt, & Coatsworth, 1998). Further, the development of adaptive and maladaptive behaviors is conceptualized as embedded within broader environmental contexts and resulting through continuous social interactions with individuals and groups (Masten & Coatsworth, 1995). Normative and pathological behaviors are therefore examined in relation to the transactional influence of biological, psychological, and social factors on individuals within a developmental context (Cicchetti & Toth, 2009). In the context of the current chapter, the developmental psychopathology perspective argues for the importance of examining normal personality development alongside the development of maladaptive personality factors involved in youth BPD and offers key guiding principles (e.g., equifinality/multifinality) that can further our understanding of the relationship between normal personality development and youth BPD.

Relevance for youth BPD: Within the developmental psychopathology framework, BPD can be conceptualized with respect to the diverse pathways and influences that contribute to its development. This perspective may help identify early etiological precursors and improve prevention and treatment efforts, which are crucial given the serious negative outcomes associated with the disorder (e.g., interpersonal problems, risk for mortality through suicide; Paris & Zweig-Frank, 2001; Whisman & Schonbrun, 2009). The extant literature suggests that specific biological and psychosocial factors may influence the development of both BPD and normative personality traits. We will now summarize evidence for etiological precursors of BPD, drawing connections to related findings for normative personality traits.

Research has begun to identify the possible genetic and biological underpinnings of BPD. Behavioral genetics research has yielded support for a heritable component of BPD, although estimates vary across studies (0.35–0.69; Torgersen et al., 2000, 2008). Importantly, the heritability estimates for youth BPD are similar in magnitude to those typically found for normative personality traits (Bergeman et al., 1993; Bouchard, 1994; Jang, Livesley, & Vernon, 1996; Loehlin, 1992). Symptoms related to aggression and impulsivity have been associated with possible functional impairments in the serotonin, dopamine, monoamine oxidase, and vasopressin systems, whereas emotional lability has been associated with functional impairments in cholinergic and nonadrenergic systems and the hypothalamic–pituitary–adrenal axis (for a review, see Crowell, Beauchaine, & Linehan, 2009), suggesting unique biological vulnerabilities for different core features of BPD. Normative personality traits have also been linked to neurobiological function. For example, multiple studies have linked dopamine to high E, low C, and novelty-seeking (Benjamin et al., 1996; Ebstein et al., 1996; Noble et al., 1998). Behaviors related to disinhibition (e.g., risk-taking) have also been linked to monoamine oxidase function (Zuckerman & Kuhlman, 2000). Other research has linked serotonin function, particularly the 5-HTTLPR S allele, to a variety for phenotypic behaviors, such as negative affect and social disaffiliation (Greenberg et al., 2000; Hamer, Greenberg, Shabol, & Murphy, 1999; Lesch et al., 1996). These results suggest that features of BPD and normative personality traits share common biological factors. Nevertheless, it is important to acknowledge that genetic and biological contributions always function within a broader social–environmental context; as such, we now turn to consider the possible contributions from psychosocial risk factors.

Most research on the etiology of BPD has investigated potential psychosocial risk factors. For example, BPD has been linked with early experiences of maltreatment, including sexual abuse (Bandelow et al., 2005; Herman, Perry, & Van Der Kolk, 1989; Ludolph et al., 1990; Ogata et al., 1990; Paris, Zweig-Frank, & Guzder, 1994; Timmerman & Emmelkamp, 2001; Yen et al., 2002; see also Chap. 16, current volume), physical abuse, emotional abuse, and neglect (Bandelow

et al., 2005; Carlson, Egeland, & Sroufe, 2009; Gratz, Latzman, Tull, Reynolds, & Lejuez, 2011; Helgeland & Torgersen, 2004; Joyce et al., 2003). Influential life events contributing to environmental instability (e.g., divorce, moving, changing schools, bullying) have also been associated with BPD (Bandelow et al., 2005; Carlson et al., 2010; Golomb et al., 1997; Helgeland & Torgersen, 2004; Ludolph et al., 1990; Zanarini, 2000). In addition, BPD has been associated with early relational experiences of insecure attachment (Barone, 2003; Carlson et al., 2010) and negative parenting attitudes and styles (Bandelow et al., 2005; Joyce et al., 2003). Together, these early experiences create a stressful environment that poses a challenge for optimal development. This is consistent with Linehan's (1993) biosocial theory of BPD development, which proposes that BPD features, specifically emotional dysregulation, develops within the context of an "invalidating environment" wherein the child's expression of emotion is discouraged or prohibited. When coupled with biological predispositions toward emotionality, such experiences impede the acquisition of adequate emotion regulation abilities (Putnam & Silk, 2005).

Examinations of normative personality traits provide complementary support for the role of early adverse experiences in the development of BPD. Research suggests that parents who are emotionally unavailable or who deny negative emotions may interfere with the development of effective emotion regulation (Fabes, Poulin, Eisenberg, & Madden-Derdich, 2002; Jones, Eisenberg, Fabes, & MacKinnon, 2002). Similarly, maltreated youth report higher levels of negative affectivity, unstable emotional control, and heightened sensitivity to stress compared to non-maltreated youth (Rogosch & Cicchetti, 2004), which corresponds with the affective instability feature of BPD. Maltreated youth also tend to be rated less favorably by their peers (Rogosch & Cicchetti, 2004) and encounter more interpersonal difficulties than their non-maltreated counterparts (Bolger, Patterson, & Kupersmidt, 1998; Mueller & Silverman, 1989), corresponding with the relationship difficulties feature of BPD. Consistent with the principle of multifinality, however, early experiences of abuse and maltreatment are neither necessary nor sufficient for developing BPD. For example, not all individuals with BPD have experienced abuse, nor do all instances of abuse result in a BPD diagnosis (Joyce et al., 2003). This research suggests that early adverse events and negative relational experiences shape personality traits relevant for self-regulation and social capacities, thereby increasing risk for developing BPD. However, it is also possible that adaptive personality traits in early life help explain why some children who experience early abuse do not develop aspects of youth BPD. That is, while early environmental experiences may shape personality development, early personality may also moderate the impact of specific environmental experiences. These findings demonstrate how consideration of normative traits may shed light on the mechanisms underlying the development of borderline pathology.

Models of Personality/Psychopathology

Another useful approach in examining the explanatory power of normal personality for youth BPD comes from prominent theoretical models of personality–psychopathology relationships. Several such explanatory models have been proposed to account for relations between personality traits and psychopathology in childhood and adolescence (see Tackett, 2006, for a review). The *vulnerability/predisposition model* suggests that personality traits serve as risk or resiliency factors for later psychopathology. The *complication/scar model*, in contrast, suggests that the experience of a psychological disorder may change the individual's underlying personality traits. Both models assume a causal relationship among personality and psychopathology, albeit in different directions. In contrast, the *pathoplasty/exacerbation model* describes the non-etiological influence of personality traits on the manifestation of a particular disorder, which may account for inter-individual heterogeneity in course and symptomology. In contrast to these models,

which conceptualize personality and psychopathology as non-overlapping phenomena, the *spectrum model* places personality and psychopathology along the same quantitative continuum. According to a spectrum model perspective, personality pathology may represent the maladaptive expression of extreme variations in normal personality traits, with shared causal factors and related manifestations.

It is important to note that these models of personality–psychopathology associations are not mutually exclusive (De Bolle, Beyers, De Clercq, & De Fruyt, 2012; Widiger, Verheul, & Van Den Brink, 1999). For example, much evidence in support of the vulnerability/predisposition model can also be used as evidence for a potential spectrum explanation (i.e., early traits and later disorders may reflect different manifestations of the same underlying phenomenon; Tackett, 2006). Further research is needed to help disentangle the complex relations among personality and psychopathology, particularly with regard to the less-studied pathoplasty/exacerbation and complication/scar models. Research on these theoretical models for personality–psychopathology relationships is sparse, especially at the level of individual diagnoses such as BPD. We next summarize overarching cross-sectional findings linking normal personality traits to youth BPD, then discuss the limited existing evidence speaking to the explanatory models described here.

Relevance for youth BPD: Research on cross-sectional associations between normative personality dimensions and BPD symptoms has primarily been conducted on adults, with some notable extensions to youth. In relation to higher-order FFM traits, BPD features have been most consistently linked with high N and low A, and to a lesser extent low C and E in adulthood (e.g., De Clercq, De Fruyt, & Van Leeuwen, 2004; Saulsman & Page, 2004). Several of these associations map on to the core dysfunctional domains of youth BPD identified by Miller et al. (2008)—that is, high N is linked to affective instability, low A is linked to relationship difficulties, and low C is linked to impulsivity (see also Table 8.1). The possibility of differential associations at the lower-order trait level may help explain why research has yielded discrepant associations between BPD symptoms and E across studies (i.e., Saulsman & Page, 2004). For example, child and adolescent BPD has been associated with higher novelty seeking (Joyce et al., 2003). These robust associations have led some researchers to speculate that PDs may be best conceptualized as maladaptive and extreme variants of normative personality traits (Trull & McCrae, 1994), thus invoking the spectrum hypothesis of personality–psychopathology associations.

This work has established robust associations among BPD features and personality traits across development, but it has largely focused on static associations and therefore cannot address their intraindividual stability over time (Samuel & Widiger, 2008). Extensive literature suggests that there is considerable continuity among early temperamental dispositions and personality traits in adulthood (Halverson, Kohnstamm, & Martin, 1994; Roberts & DelVecchio, 2000). Nevertheless, it has also been suggested that adults with BPD exhibit considerable change and variability in N and C over time (Hopwood et al., 2009). In a study examining personality trait and PD stability among adults, Warner et al. (2004) observed that changes in BPD followed changes in traits, over and above the stability of traits and symptoms. Further, BPD was the only PD studied to evidence an association among baseline symptoms and changes in traits, which may be due to the influence of affective lability on the measurement of traits. This evidence suggests that bidirectional influences may lead to change in both normal-range personality and BPD symptoms. This research lends support to a vulnerability and/or spectrum model explanation (the two models were not directly tested in this study) as well as a complication/scar model and should be extended to younger age groups.

Recently, Crowell et al. (2009) formulated a biosocial developmental model that also helps to frame the existing evidence for associations among normative traits and BPD features (see also Chap. 9, this volume). This extension of Linehan's (1993) biosocial theory proposes that

impulsivity emerges—independent of emotion dysregulation—as the result of biological vulnerabilities, whereas emotional dysregulation is primarily influenced by psychosocial factors (e.g., the caregiving environment) and baseline characteristics of the child (e.g., emotional sensitivity). Difficulties with impulsivity and emotional dysregulation are further exacerbated through reciprocally reinforcing interactions between biological vulnerability and environmental risk. To illustrate, youth who are more impulsive and prone to experience negative affect may evoke negative responses from peers, thereby increasing the likelihood of relationship difficulties and intensifying negative affect. This theoretical description of BPD development potentially supports both a vulnerability and a complication/scar model. Specifically, it maintains that individual characteristics represent preexisting risk for BPD, but also that traits may be further intensified (scarred) through experience. Greater understanding of these transactional influences presents a promising avenue for future research investigations.

The spectrum approach can also be useful in understanding comorbidity among different disorders (e.g., comorbidity between youth BPD and general externalizing problems) via common personality characteristics. One recent study examined the hypothesis that BPD-relevant traits (specifically traits in the domains of Antagonism and Emotional Instability measured with the Dimensional Personality Symptom Item Pool [DIPSI]; see Chap. 7, current volume) should be incorporated into a broader spectrum of youth externalizing problems (Tackett, Herzhoff, & De Clercq, 2012). These findings highlighted the centrality of traits in these domains in conceptualizing general youth externalizing problems (e.g., physical aggression, rule-breaking behaviors) but also revealed a complex pattern across development. Specifically, BPD trait-externalizing behavior associations were strongest at periods of greatest prevalence for the behavior in question, suggesting that the role played by personality pathology in influencing behavior may be particularly illuminated during normative periods across development. That is, to best understand how youth BPD traits influence other forms of psychopathology, it may be important to focus on periods of high prevalence ("normative" periods) for the behaviors of interest. These findings support a spectrum conceptualization between youth BPD traits and other forms of externalizing behavior, and further underline the need to attend to the changing nature of these relationships across development.

Hierarchical Models of Personality

Hierarchical models have long been used to characterize personality psychology and have been conceptualized as endemic to the study of personality trait structure (Digman, 1997; Markon, 2009). Specifically, personality trait structure is often characterized as consisting of higher-order traits, which characterize broad levels of abstraction (e.g., extraversion/surgency) and which subsume lower-order traits, which characterize more narrowly defined domains (e.g., gregariousness). We focus here on these two aspects of hierarchical models (higher- and lower-order structure) and emphasize that both are relevant for better understanding youth BPD.

Personality hierarchy has proven a useful tool for capturing aspects of higher-level personality traits. Specifically, researchers have identified a higher-order personality hierarchy which integrates diverse trait models of adult personality, increasing communication across researchers and integration of research findings (DeYoung, 2006; Digman, 1997; Markon, Krueger, & Watson, 2005). This hierarchy includes broad traits reflecting approach- and avoidance-motivated tendencies at the two-factor level, differentiates self-regulatory characteristics at the three-factor level, distinguishes between intrapersonal and interpersonal self-regulation at the four-factor level, and differentiates approach-motivated tendencies into E and O traits at the five-factor level. Importantly, this higher-order personality hierarchy has also emerged in research with children and adolescents (Goldberg, 2001; Martel, Nigg, & Lucas, 2008; Tackett, Krueger, Iacono, &

Mcgue, 2008), albeit with some differences from the established hierarchy in adults. One recent study investigated continuity of this higher-order hierarchy across childhood (ages 3–14) and multiple countries, and largely found support for continuity of this structure across both age and country (Tackett, Slobodskaya et al., 2012). Specific relevance for the higher-order personality hierarchy and youth BPD is described below.

Another important aspect of personality hierarchy is the distinction between higher-order and lower-order traits, or domains and facets. Specifically, some researchers have suggested that personality facets may hold better utility for prediction of specific behaviors (Paunonen & Ashton, 2001), including for personality disorder, in particular (Reynolds & Clark, 2001). There may be aspects of pathological behavior in the domain of youth BPD that are not well-captured by higher-order personality domains and require a more specific level of personality assessment. Recent investigations on the higher-order structure of psychopathology are consistent with this idea, such that BPD in adults appears to represent a more complex combination of broader dimensions than other common disorders (Eaton et al., 2011), and these connections have been found in samples of children and adolescents as well (Tackett, Herzhoff, & De Clercq, 2012). Thus, the personality hierarchy may serve another useful purpose in understanding youth BPD by offering more nuanced and differentiated information at the lower-order trait, or facet, level.

Relevance for youth BPD: Researchers have begun to extend earlier work on the joint structure of abnormal and normal personality traits (Markon et al., 2005) to hierarchical models of personality pathology in adult samples. For example, two recent empirical investigations examined the higher-order hierarchy of personality disorder (Kushner, Quilty, Tackett, & Bagby, 2011; Wright et al., 2012). Prominent four-factor models of personality pathology emerge from these hierarchies at Level 4 (e.g., Livesley & Jackson, 2009; Livesley, Jang, & Vernon, 1998; Widiger & Simonsen, 2005). At the five-factor level, the components derived by Wright et al. (2012) are consistent with investigations observing a fifth-factor labeled psychoticism (e.g., Harkness & McNulty, 1994; Tackett, Silberschmidt, Krueger, & Sponheim, 2008). It is notable that Kushner et al. (2011) did not have adequate content coverage to observe a fifth-factor reflecting psychotic or peculiarity items, but identified a fifth-factor which was labeled Need for Approval which showed particular relevance for adult BPD.

More recently, Kushner, Tackett, and De Clercq (2013) applied the structural examination of personality trait hierarchies to an adolescent community sample. Specifically, Kushner et al. examined the joint structure of two measures of youth personality pathology: the DIPSI (De Clercq, De Fruyt, & Mervielde, 2003) and the youth-version of the Schedule for Nonadaptive and Adaptive Personality (SNAP-Y; Linde, Stringer, Simms, & Clark, 2012). The DIPSI is a bottom-up instrument that was developed specifically with youth, whereas the SNAP-Y was adapted from an adult assessment instrument and is therefore a top-down measure; accordingly, this investigation permitted a novel opportunity to bridge previously disparate areas of research on child personality and pathological traits in adulthood. The results of this investigation revealed important differences from the structure observed in adults (see Fig. 8.1). In particular, notable differences included evidence for higher covariation between disagreeableness and emotional instability than has been found in adult samples (as seen in the disagreeableness component at Level 3 breaking off from emotional instability at Level 2). In addition, a distinct higher-order trait reflecting detachment (i.e., pathological introversion) typically emerged at Level 4 in the adolescent sample, whereas it typically emerges at Level 3 in adult samples.

This previous work has shown robust patterns of the higher-order hierarchy of major personality and personality pathology traits, but such findings leave open questions regarding which level of the higher-order hierarchy researchers and clinicians should use. Perhaps the ideal way to approach this question is to consider increasingly complex trait models by balancing rational

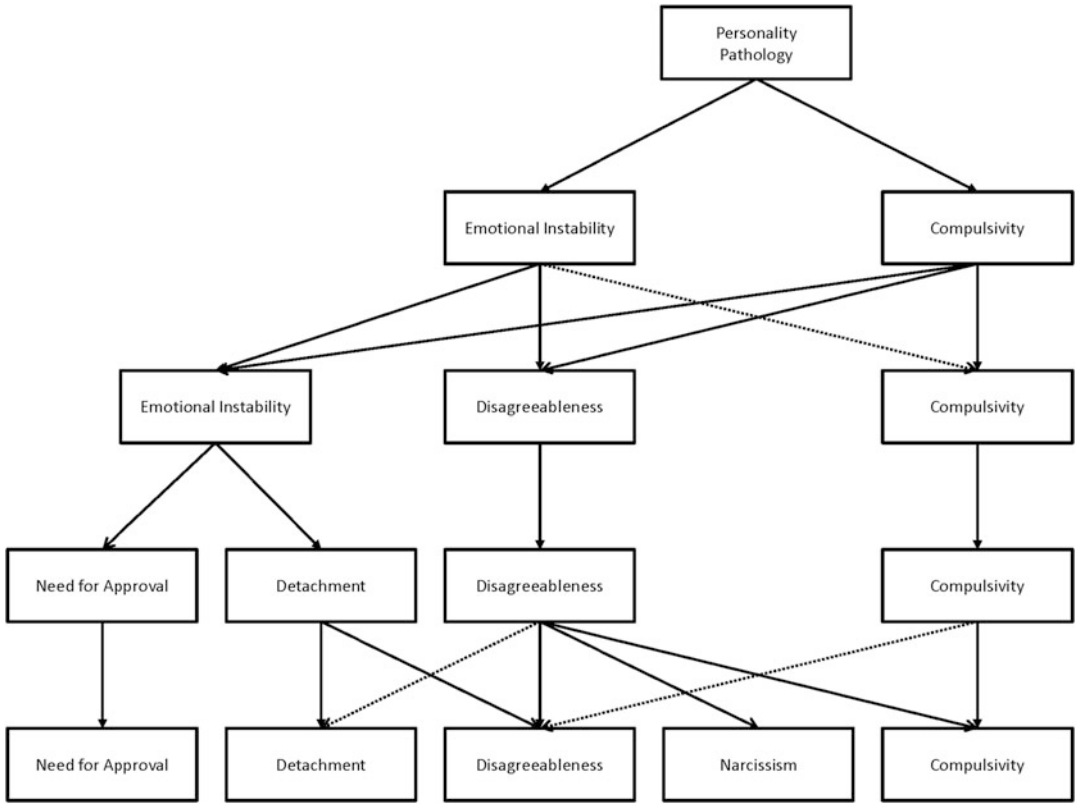

Fig. 8.1 The joint hierarchical structure of two measures of adolescent personality pathology (adapted from Kushner et al., 2013). *Note*: Negative path estimates across levels of the hierarchy are denoted with *broken lines*

(e.g., are the distinct traits conceptually meaningful) and empirical (e.g., do the distinct traits provide incremental explanatory power) criteria in specific disorder contexts. For example, Tackett, Quilty, Sellbom, Rector, and Bagby (2008) used empirical criteria to demonstrate that Level 5 factors were maximally useful for differentiating DSM-IV internalizing disorders. Similarly, Kushner et al. (2011) also used empirical criteria and concluded that Level 5 of the hierarchy enhanced the capacity for predicting symptoms of DSM-IV PDs. In particular, 39 % of the variance in BPD symptoms were predicted by Level 5 emotional dysregulation (affective instability, self-harming behavior, identity problems, anxiousness, and cognitive distortion), dissocial behavior (callousness, conduct problems, stimulus seeking, and rejection), and need for approval (insecure attachment, narcissism, and submissiveness). This constituted a 6 % increase from the Level 4 components, which lacked a specific factor related to interpersonal dysfunction (i.e., need for approval), thus highlighting the importance of relationship difficulties as a core component of borderline pathology. Together, these data provide compelling evidence for the clinical utility of Level 5 factors in understanding youth BPD. It is also notable that a need for approval subfactor was observed by Kushner et al. (2013), suggesting that this trait is both salient and measurable in adolescence, and may have added utility in measuring features of BPD in youth.

Trait structure may differ across development, however (e.g., Digman & Shmelyov, 1996; Rothbart, Ahadi, & Evans, 2000; Rothbart & Bates, 2006; Shiner & Caspi, 2003), which calls for focused research on personality hierarchy across different ages in childhood and adolescence (Tackett, Slobodskaya et al., 2012).

Of particular relevance for youth BPD, Tackett, Slobodskaya et al. (2012) demonstrated that factors related to negative emotionality and self-regulation deviated somewhat compared to those observed in adulthood. Specifically, extraction of a typically defined N component proved particularly difficult in younger ages, consistent with other studies (e.g., Tackett, Krueger et al., 2008). Along these lines, antagonism played a prominent role in N among 12–14-year-olds (Tackett, Slobodskaya et al., 2012). This is consistent with the recent Kushner et al. (2013) study, wherein aspects of aggression loaded more highly on emotional instability than disagreeableness, suggesting that aggressive and nonaggressive negative affect are more strongly linked among youth than adults. In addition, aspects of self-regulation (A and C traits) became more clearly distinguished across childhood, potentially reflecting the broad self-regulatory trait (effortful control) measured in early temperament models (Rothbart & Bates, 2006). In both normal and abnormal personality trait hierarchies in youth (Kushner et al., 2013; Tackett, Slobodskaya et al., 2012), aspects of antagonism/disinhibition covaried more highly with N than is typically seen in adult samples. These findings raise questions about the extent to which such age-related differences reflect measurement challenges in younger age groups versus true developmental phenomena, and raise important caution for examining the role of these traits in youth BPD.

It is also important to consider contributions of lower-order facets to conceptualizing youth BPD (Paunonen & Ashton, 2001; Reynolds & Clark, 2001), as facets may have particular utility for differentiating related diagnoses. Research has demonstrated significant correlations among BPD symptoms and lower-order facets from each of the Big Five/FFM domains among children and adolescents (De Clercq et al., 2004; De Clercq & De Fruyt, 2003). After partialling out the influence of general pathology (i.e., comorbidity), lower-order facets of N (depression, impulsiveness, vulnerability, anxiety, and low self-confidence), A (trust, irritability, and low compliance), E (low shyness), and C (persistence) account for unique variance in youth BPD symptoms (De Clercq et al., 2003, 2004). These results support the suggestion that personality facets hold better utility for prediction of specific behaviors (Paunonen & Ashton, 2001; Reynolds & Clark, 2001). In sum, the assessment of higher-order factors may be especially useful for detecting common variance in disorders characterized by emotional dysregulation and impulsivity (e.g., externalizing disorders), whereas lower-order facets may offer more nuanced information about the particular syndrome (e.g., youth BPD).

Summary and Conclusions

In sum, we have discussed the relevance of normal-range personality traits for a better understanding of youth BPD in three theoretical contexts: the developmental psychopathology perspective, theoretical models of personality–psychopathology relationships, and hierarchical models of personality trait structure. Although general reluctance toward identifying BPD in youth has limited research on its early manifestations (Crick et al., 2005; Miller et al., 2008), we reviewed the limited but rapidly growing empirical evidence speaking to each domain.

Personality traits related to self-control and the regulation of emotions reflect early dispositional tendencies that may become more or less adaptive when coupled with early adversity, such as maltreatment, environmental instability, and negative relational experiences, depending on a "goodness of fit" with the demands and expectations of the environment (i.e., Thomas & Chess, 1977). Early findings are consistent with a developmental psychopathology account of youth BPD development, such that biological predispositions and environmental risk factors interact to shape the development of normal personality traits as well as youth BPD symptomatology (e.g., Crowell et al., 2009; Linehan, 1993). Thus, an understanding of how early personality traits such as N, A, and C emerge and grow across development is highly relevant for an understanding of youth BPD emergence.

Empirical research examining theoretical models of personality–psychopathology relationships with respect to youth BPD is quite limited. Initial evidence supports both a vulnerability and spectrum account of the relationship between youth BPD traits and normal-range personality (Crowell et al., 2009; Tackett, Herzhoff, & De Clercq, 2012). Importantly, disentangling vulnerability and spectrum accounts can be difficult and requires focused research designs that examine evidence for continuity (e.g., De Bolle et al., 2012; Tackett, Herzhoff, & De Clercq, 2012) or underlying common causal factors resulting in both normal-range personality traits and psychopathology (e.g., Tackett, Waldman, Van Hulle, & Lahey, 2011). Finally, extensions of personality hierarchical structure have only recently been extended to adult PD traits (Kushner et al., 2011; Wright et al., 2012), normal-range child personality traits (Tackett, Slobodskaya et al., 2012), and youth PD traits (Kushner et al., 2013). This work has initially suggested convergence across normal/abnormal trait models and across adolescents/adults, but differences have emerged as well. Future research should begin tackling the difficult question regarding the extent to which such differences reflect true developmental phenomena versus measurement limitations in younger age groups (Tackett, Slobodskaya et al., 2012).

The findings reviewed here have relevance for both assessment and treatment of youth in clinical settings. Despite its longstanding reputation as a chronic and enduring condition, recent empirical advances suggest that BPD features in youth have shown considerable plasticity, underscoring the need for early intervention (Lenzenweger & Castro, 2005). Extant data also imply that adolescents with elevated BPD traits compose the large majority of adults with BPD (Crawford et al., 2005), suggesting that traits represent precursor signs that are ideal candidates for identifying those most in need of selective prevention (Chanen, McCutcheon, Jovev, Jackson, & McGorry, 2007). Personality assessments may therefore help to identify at-risk youth and thereby improve the delivery of preventive efforts. Most promisingly, Chanen et al. (2008) observed that screening for BPD in youth outpatients is indeed feasible and that targeting youth with these early precursors has yielded positive treatment outcomes (Chanen et al., 2009).

In addition to traits as potential targets for prevention/intervention efforts, identifying discrepancies in personality assessment may be useful for youth BPD. Informant discrepancies have proven to have clinical utility for children and adolescents (De Los Reyes, Alfano, & Beidel, 2010; De Los Reyes, Goodman, Kliewer, & Reid-Quinones, 2008; De Los Reyes & Kazdin, 2005). For example, mother–father discrepancies in child personality assessment significantly predict internalizing problems (Tackett, 2012). Importantly, informant discrepancies have also shown utility in the context of adult PD and may have practical and theoretical implications for clinical assessment (Klonsky, Oltmanns, & Turkheimer, 2002). Informant ratings of FFM personality prototypes account for significant additional variance in BPD symptoms, as well as other personality disorders (Lawton, Shields, & Oltmanns, 2011). This suggests an important area of future investigation and highlights the clinical applicability of personality assessment for youth BPD.

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Part III

Etiology and Core Components

The Neurobiology of Adolescent-Onset Borderline Personality Disorder

9

Marianne Goodman, M. Mercedes Perez-Rodriguez, and Larry Siever

Borderline personality disorder (BPD) is a disabling disorder characterized by poor affect regulation and poor impulse control. This often results in impaired interpersonal relationships and maladaptive behavioral patterns, including anger dyscontrol, aggression towards others, and self-destructive behaviors. The disorder remains notoriously difficult to treat effectively, with many patients responding poorly or partially even to the most widely accepted treatment strategies (Paris, 2005). Evidence suggests that early detection of BPD can attenuate the severity of symptoms (Chanen, Jackson et al., 2008); however, little is known about early predictors of this disorder. Using diagnostic instruments similar to those used in adults, researchers are diagnosing adolescent presentation of BPD to describe the incidence, phenomenology, and negative prognosis for the development of Axis I/II disorders in adulthood (Chanen, Velakoulis et al., 2008; Crawford et al., 2008). Prevalence estimates of adolescent onset BPD in the community range from 0.9 to 3.0 % (Bernstein et al., 1993; Lewinsohn, Rohde, Seeley, & Klein, 1997), while clinical populations are considerably higher; 11 % in outpatient populations (Chanen et al., 2004), and 32–49 % in adolescent inpatient units (Burket & Myers, 1995; Grilo et al., 1996).

Clarifying the underlying biology of BPD, including the etiological mechanisms, genetic factors, and pathological processes is essential for a full understanding of the disorder and provides a basis for the development of more effective treatment intervention and preventive strategies (Beauchaine, Hong, & Marsh, 2008; Chanen & Kaess, 2012). Over the past two decades, neurobiological studies in adult BPD have made important strides, but inquiry into adolescent-onset BPD is still in its infancy.

Genotypes and Endophenotypes in BPD

Because of the complex genetics of psychiatric disorders (Plomin, Owen, & McGuffin, 1994), any hopes of uncovering Mendelian inheritance patterns for the most part been abandoned in favor of models incorporating genetics, epigenetics (Wong, Gottesman, & Petronis, 2005), environmental factors (Kendler, 1995), and gene–environment interactions (Caspi et al., 2003). It has further become apparent that, rather than conforming to a "one gene-one illness" model, the risk of developing psychopathology is conveyed by multiple genes of small effect (Collins, Brooks, & Chakravarti, 1998). In order

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to derive meaningful information about the genotypes underlying mental illnesses, researchers have increasingly focused their attention on "endophenotypes," defined in one recent review as "measurable components unseen by the unaided eye along the pathway between disease and distal genotype" (Gottesman & Gould, 2003). To be considered an endophenotype, a feature should be measurable, reproducible, and state-independent, and it should occur at a greater rate in affected probands than in unaffected family members or in the general population and at a greater rate in unaffected family members than in the general population (Balanzá-Martínez et al., 2008).

The quest for biological endophenotypes of BPD is gaining momentum (Goodman, New, Triebwasser, Collins, & Siever, 2010; New, Goodman, Triebwasser, & Siever, 2008). Recent advances include a meta-analysis highlighting reduced amygdala and hippocampal volumes (Ruocco, Amirthavasagam, Choi-Kain, & McMain, 2012); however, few proposed candidates currently exist. This chapter will review findings with a neurobiological focus including genetic, neuropeptides, neuroendocrine, and neuroimaging studies relevant to BPD and highlight data pertaining to adolescents.

Genetic Studies

Adult BPD

Genetic Vulnerability

As with many other illnesses, the etiology of BPD is likely to be an interaction of heritable vulnerability with environmental factors that combine to bring about the full presentation of disease (Distel et al., 2011).

Family/Twin Studies

Family studies can indirectly reflect heritability; however, only twin studies provide definitive evidence for genetic heritability. Unfortunately, only limited data from twin studies is available for BPD. Recent data examining genetic and environmental risk of endorsed DSM personality disorder criteria in 2,894 members of the Norwegian Institute of Public Health Twin Panel suggests a broad heritability to personality pathology suggestive of negative emotionality and a specific heritability of the BPD intermediate phenotype impulsive aggression (Kendler et al., 2008). Another twin study of BPD examining 92 monozygotic twins and 129 dizygotic twins showed that BPD was substantially heritable, with a heritability score of 0.69, i.e., 69 % of the variance in BPD was accounted for by genetic factors (Torgersen, 2000). A more recent web-based cohort ($n = 44{,}112$) including 542 twin pairs (Kendler, Myers, & Reichborn-Kjennerud, 2011) found the four dimensions of BPD load as one highly heritable factors (heritability = 60 %).

Genetic Studies

Research into the specific genes involved in BPD is at a very early stage and highlights the role of the serotonin system. A case–control study showed a significant association between the serotonin transporter (5-HTT) gene and BPD, with higher frequencies of the 10 repeat of the VNTR marker and the S-10 haplotype, and fewer 12 repeat and LA-12 haplotype, in BPD patients compared with healthy controls (HCs) (Ni, Chan et al., 2006). This result is consistent with findings of a genetic association between the low-expressing S allele and aggressive behavior (Cadoret et al., 2003) as well as with NEO ratings of neuroticism, which is characterized by negative emotionality including anxiety, depression, vulnerability, and hostility (Sen et al., 2004). A simultaneous case–control study of the same gene in BPD, however, was unable to replicate the association found by Ni and colleagues (Pascual et al., 2008).

Another gene that has been implicated in impulsive aggression and suicidal behavior, common features of BPD, is that for tryptophan hydroxylase (TPH), the rate-limiting enzyme in 5-HT biosynthesis. Two isoforms, TPH-1 and TPH-2, are known. TPH-1 has been correlated with various psychiatric and behavioral disorders by gene polymorphism association studies. A recent case–control study of 95 women with

BPD and 98 healthy controls showed that one six-SNP haplotype was absent from the control group, while representing about one-quarter of all haplotypes in the BPD group. A "sliding window" analysis attributed the strongest disease association to haplotype configurations located between the gene promoter and intron 3 (Zaboli et al., 2006). More recent studies (Perez-Rodriguez et al., 2010) found an association between the previously identified "risk" haplotype at the TPH-2 locus and BPD diagnosis, impulsive aggression, affective lability, and suicidal/parasuicidal behaviors. Wilson et al. (2009) reported an association between the tryptophan hydroxylase-1 A218C polymorphism is associated and BPD diagnosis, but not suicidal behavior.

Single reports were published for 5HT2a (Ni, Bismil et al., 2006), 5HT2c (Ni et al., 2009), and monoamine oxidase A (Ni et al., 2007). While these preliminary studies of specific genes implicated in BPD are suggestive and support the presence of a serotonergic abnormality in this disorder, they will require replication for any clear conclusions to be drawn.

adolescent BPD

There exists only one candidate gene association study of BPD traits in youth.

Hankin et al. (2011) report on an association between the short allele of 5-HTTLPR and BPD traits in two independent studies of community youth that persisted after controlling for depression. While preliminary, this promising data suggests links between the serotonin transporter and developmental aspects of BPD.

Neuropeptides

Oxytocin and Social Cognition

Adult BPD

Social cognition or mentalization, the ability to assign mental states (emotions, thoughts, and purpose) to oneself and others (i.e., the ability to read social cues), is crucial to maintaining social relationships (Eisenberg & Miller, 1987). Abnormal social cognition characterized by hypermentalizing (Dziobek et al., 2006, 2011; Sharp et al., 2011) is a core feature of BPD, severely impairs functioning, and is a key target of psychotherapeutic interventions that have shown efficacy in treating borderline patients (Bateman & Fonagy, 2010; Leichsenring, Leibing, Kruse, New, & Leweke, 2011; Lis & Bohus, 2013; Mak & Lam, 2013; New et al., 2008; Stoffers et al., 2012). Oxytocin is a key regulator of social cognition and mentalization likely through frontolimbic modulation (Bartz, Zaki, Bolger, & Ochsner, 2011; Bos, Panksepp, Bluthe, & van Honk, 2012; Guastella & MacLeod, 2012; Meyer-Lindenberg, Domes, Kirsch, & Heinrichs, 2011). It has been suggested that deficits in the attachment and affiliative systems modulated by oxytocin may underlie the impulsive aggressive reactions to perceived rejection and loss that are common in BPD (Stanley & Siever, 2010). Because of its anxiolytic and prosocial effects (Macdonald & Macdonald, 2009; Meyer-Lindenberg et al., 2011; Zink & Meyer-Lindenberg, 2012), oxytocin has been suggested as a promising novel treatment for the social cognition abnormalities found in BPD (Meyer-Lindenberg et al., 2011).

Few studies have examined the effects of oxytocin administration in BPD patients, with conflicting results (Bartz, Simeon et al., 2011; Bartz, Zaki et al., 2011; Simeon et al., 2011). The only two published studies of oxytocin administration in BPD have shown that oxytocin modestly decreased the subjective anxiety resulting from the Trier Social Stress Test (Simeon et al., 2011), but it also decreased the level of cooperative behavior in a trust game in BPD (Bartz, Simeon et al., 2011).

Although research examining the genetics of the oxytocin system in BPD populations is lacking, some evidence suggests that it may play a role in the pathophysiology of BPD. Data from genetic studies suggest that the oxytocin system is involved in some of the core dimensions underlying BPD, such as interpersonal dysfunction and impulsive aggression

(Stanley & Siever, 2010). For example, oxytocin receptor gene (OXTR) polymorphisms have been linked to empathy (Wu, Li, & Su, 2012), aggressive behavior (Johansson et al., 2012), and prosocial temperament (Tost et al., 2010). Oxytocin gene polymorphisms have been associated with aggressive antisocial behaviors in children (Malik, Zai, Abu, Nowrouzi, & Beitchman, 2012) and with dopaminergic response to stress (Love et al., 2012), and oxytocin gene-knockout mice are highly aggressive (Ragnauth et al., 2005). We have shown that oxytocin genotypes and haplotypes are significantly associated with the core BPD dimensions of impulsivity, aggression, and anxious attachment (Perez-Rodriguez et al., unpublished data).

adolescent BPD

Although no studies to date have examined the effects of oxytocin administration in children or adolescents with BPD, there are two published clinical trials of intranasal oxytocin in children and adolescents with autism spectrum disorders (Guastella et al., 2010; Tachibana et al., 2013). Guastella et al. found that intranasal oxytocin administration improved performance on the Reading the Mind in the Eyes Task, a measure of social cognition, in youth (12–19 years of age) with Autistic or Asperger's Disorder (Guastella et al., 2010). Tachibana et al. reported improved communication and social interaction in youth (10–14 years of age) with autism spectrum disorders after treatment with intranasal oxytocin for about 7 months (Tachibana et al., 2013).

Opioids

Adult BPD

Because of their involvement in social attachment, endogenous opioids are a recent area of interest in the biology of BPD. In fact, a deficit in endogenous opiates might underlie the interpersonal difficulties that are central to BPD pathology (New & Stanley, 2010; Stanley & Siever, 2010). Moreover, one theory about non-suicidal self-injury (NSSI), a behavior common in BPD, is that it represents a method of releasing endogenous opioids, to compensate for an opioid deficit (New & Stanley, 2010; Stanley & Siever, 2010). One recent imaging study measured μ-opioid receptor binding, with the μ-opiate ligand [^{11}C] carfentanil, in BPD patients during induction of neutral and sad emotions (Prossin et al., 2010). They found greater baseline μ-opioid receptor availability in BPD, indicating a deficit in endogenous opioids. They also observed that BPD patients increased endogenous opiate availability more than controls during sad mood induction, which might reflect a compensatory response and is consistent with lower levels of endogenous opioids in self-injurers (Sher & Stanley, 2009; Stanley & Siever, 2010). We have found an association between genetic variants of the μ-opioid receptor and affective instability and aggression (Perez et al., unpublished data).

Adolescent BPD

There are no studies to date examining the opioid system in adolescent or children with BPD.

Neuroendocrine: HPA Axis

Adult BPD

Several investigators (Grossman et al., 2003; Lange et al., 2005; Rinne et al., 2002) have found enhanced cortisol suppression in individuals with BPD and comorbid posttraumatic stress disorder (PTSD), though they have concluded that the response was due to the comorbid PTSD and not the BPD diagnosis itself. However, a brief report (Carrasco et al., 2007), using a 0.25 mg dexamethasone suppression test dose, found enhanced cortisol suppression in individuals with BPD without PTSD, suggesting that increased feedback inhibition of the HPA axis may exist in BPD that is not accounted for by PTSD. Walter et al. (2008), in a small pilot study, noted a delayed cortisol response after psychosocial stress in BPD compared to healthy control (HC) subjects.

Adolescent BPD

Pituitary volume in adolescent BPD was found not to differ from HC (Garner et al., 2007); however, pituitary volume was associated with number of parasuicidal events (Jovev et al., 2008). In a small sample of adolescents with NSSI, a potential precursor to BPD, reductions in cortisol secretion to acute stress was found suggesting that HPA axis is hyporesponsive in these adolescents (Kaess et al., 2012). The authors speculate on the role of hyposecretion of cortisol and vulnerability to maladaptive stress responses and the development of BPD.

Evoked Potentials

Adult BPD

Psychiatric research on P300, also known as P3b, a component of event-related potentials (ERP), has revealed abnormal P3b amplitudes in adult subjects with BPD suggesting deficits in novelty detection and orienting as well as the inhibitory aspect of the attentional process (Meares, Schore, & Melkonian, 2011). Schuermann, Kathmann, Stiglmayr, Renneberg, and Endrass (2011) found that BPD patients' P3b amplitudes were increased following negative feedback, relative to the control subjects, while engaging in the Iowa Gambling Task.

adolescent BPD

There exist several studies using P300 in Adolescent BPD. Using a visual oddball task, Houston, Ceballos, Hesselbrock, and Bauer (2005) found that adolescent female BPD subjects did not exhibit the expected age-related reduction in P3b amplitudes suggesting abnormal brain maturation in adolescents with BPD features (Houston et al., 2005). This conflicts with another study using the same paradigm, but differentiating conduct disorder (CD) and adolescents with BPD features. Only the adolescents with CD demonstrated the P300 abnormalities (Ceballos, Houston, Hesselbrock, & Bauer, 2006). However, other research in adolescents with BPD, using a Stroop color-word task demonstrated neurophysiological abnormalities even after controlling for depression and conduct disorder (Houston, Bauer, & Hesselbrock, 2004). These results highlight the complexity of unraveling the contribution of Axis I comorbidities from BPD in the pathological processes seen in these individuals. Additional studies are needed to understand the trajectory of the BPD illness during adolescent brain development and its interaction with co-occurring Axis I disorders.

Neuroimaging

Over the past decade, much of the literature concerning the biological basis of BPD has shifted from endocrine parameters to direct visualization of brain structure and function through neuroimaging. This chapter will review the brain regions implicated in both adult and adolescent BPD. Brain regions of interest in BPD are diagrammed in Fig. 9.1.

Anterior Cingulate Cortex (ACC) and ACC/Orbital Frontal Cortex (OFC) Coupling

Adult BPD

Evidence suggests decreased gray matter volume and increased white matter volume in rostral (Hazlett et al., 2007) and subgenual (Minzenberg, Fan, New, Tang, & Siever, 2008) cingulate in individuals with BPD compared to healthy controls.

Functional imaging studies in BPD have tended to show decreased activation of ACC in response to provocation. Schmahl et al. (2006) noted in 12 BPD subjects (1 with current major depressive disorder (MDD) and 11 with history of MDD) diminished activation of perigenual ACC with induction of pain. Several other

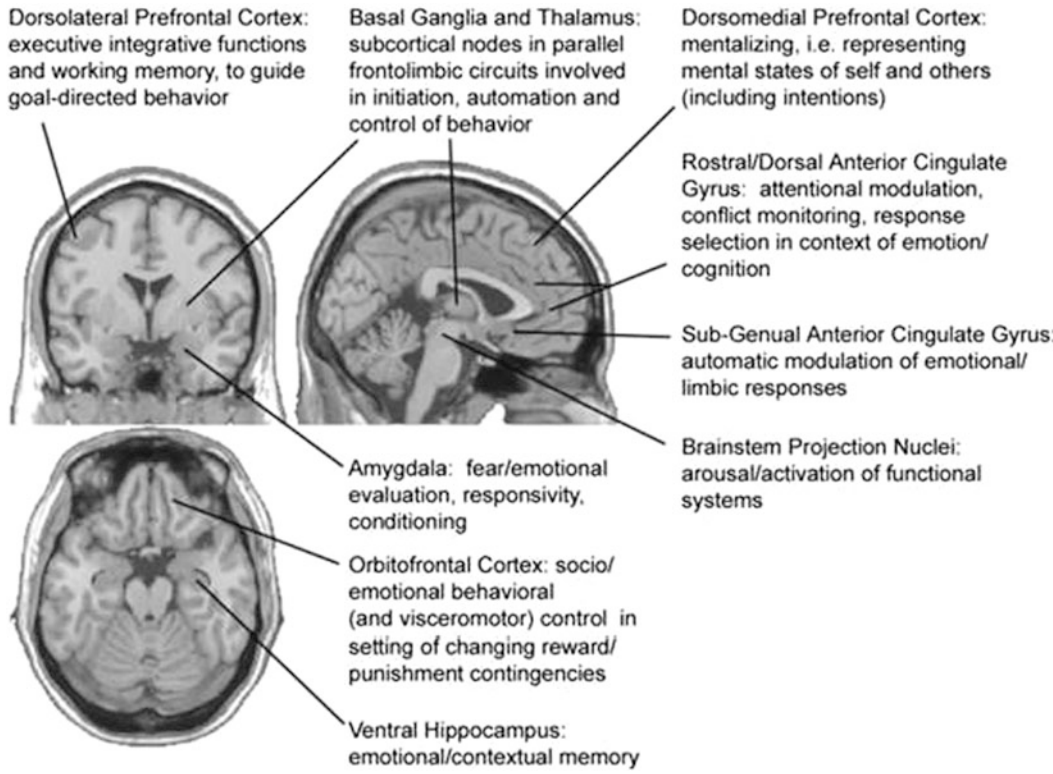

Fig. 9.1 Brain regions implicated in BPD. With permission from Dr. Gary Brendel, MD

functional resonance imaging (fMRI) studies in BPD also show decreased activation of ACC in response to provocation (e.g., Hazlett et al., 2005; Minzenberg, Fan, New, Tang, & Siever, 2007; Schnell, Dietrich, Schnitker, Daumann, & Herpertz, 2007). Silbersweig et al. (2007), using a behavioral inhibition task during the induction of negative emotion with fMRI, demonstrated decreased activation in subgenual ACC and OFC, with increases in amygdala activity, prompting his group and another (Siegle, 2007) to propose that BPD sits at the "intersection of cognition and emotion" and ponder whether this constellation of impaired regions is specific to BPD.

Pharmacologic probes have also shown decreased metabolic activity in ACC and OFC in response to serotonergic challenge in impulsive aggressive patients with BPD (New et al., 2002; Siever et al., 1999) and BPD patients with affective instability (Soloff et al., 2003) compared to HCs, and decreased coupling of resting metabolism between OFC and ventral ACC has been reported by our group (New et al., 2007). A recent case study of a patient with schizencephaly (da Rocha et al., 2008) resulting in a primary ACC and secondary OFC lesion, who prominently manifested symptoms of BPD, supports the notion of important interconnections between these two brain regions in the development of BPD.

Adolescent BPD

A summary of neuroimaging studies conducted in adolescents is provided in Table 9.1.

In adolescent onset BPD, there exist only three morphometric studies examining ACC volume in adolescents with BPD (Brunner et al., 2010; Goodman et al., 2011; Whittle et al., 2009) and the results are conflicting. Using

Table 9.1 Neuroimaging studies in adolescent BPD

Study name	Region of interest	Number of subjects	Methodological considerations	Findings
Brunner et al. (2010)	Fronto-limbic structures	20 BPD, 20 HC, 20 clinical comparison (CC); all right handed females only	VBM	↓ DLPFC gray bilaterally and ↓ left OFC in BPD compared to controls, no difference between BPD and CC, no difference in limbic structures
Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008)	Fronto-limbic structures	20 BPD, 20 HC mixed gender	ROI	↓ OFC gray matter BPD, no difference hippocampus or amygdala
Goodman et al. (2011)	Anterior cingulate, OFC, DLPFC	13 BPD-MDD mixed gender, 13 HC	ROI	↓ BA 24 gray but not white matter. Smaller BA 24 volume associated with BPD but not MDD indices. No differences in OFC, DLPFC
Garner et al. (2007)	Pituitary volume	Same subjects as Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008)	ROI	No difference in pituitary volume in BPD and HC + childhood trauma was associated with ↓ size
Jovev et al. (2008)	Pituitary volume	Same subjects as Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008)	ROI	↑ Pituitary volume was associated with ↑ # lifetime parasuicidal events
Takahashi, Chanen, Wood, Walterfang et al. (2009), Takahashi, Chanen, Wood, Yücel et al. (2009)	Insular cortex	Same subjects as Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008)	ROI	No differences in the gray matter volume of the insula in BPD vs. HC. Violent BPD had smaller insula bilaterally compared to nonviolent BPD
Takahashi, Chanen, Wood, Walterfang et al. (2009), Takahashi, Chanen, Wood, Yücel et al. (2009)	Adhesio interthalamica (AI), cavum septum pellucidum (CSP), third ventricle	Same subjects as Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008)	ROI	Shorter AI in BPD, no differences in CSP between BPD and HC, larger third ventricle size in BPD; but no clinical correlations with findings
Takahashi et al. (2010)	Superior temporal gyrus (STG)	Same subjects as Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008)	ROI	No difference in STG volume between BPD and HC, more violent BPD patients had smaller volumes
Walterfang et al. (2010)	Corpus callosum, ventricular volume	Same subjects as Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008)	ROI	No difference in total callosal area, length, curvature, shape, or ventricular volume in BPD and HC
Whittle et al. (2009)	Anterior cingulate	Subset of Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008); 15 females	ROI	↓ Left ACC volume in BPD; correlate with measures of impulsivity and parasuicidal behavior

region-of-interest methodology, Whittle et al. (2009) reported decreased left ACC volume in 15 female BPD adolescents with a wide range of Axis I comorbidities (Whittle et al., 2009). More recently, Brunner et al. (2010) compared 20 female adolescents with BPD, 20 psychiatric ill adolescents without BPD and 20 healthy control (HC) subjects using voxel-based morphology (VBM) and reported no ACC abnormalities. The primary finding of Goodman et al. (2011) is that adolescents with BPD and comorbid MDD have reduced Brodmann Area (BA) 24 gray matter volume than HCs but no differences in prefrontal cortex (PFC). This finding raises the possibility of a neurodevelopmental abnormality in BPD as the group has previously reported similar gray matter volume reduction in BA 24 in adults with BPD (Hazlett et al., 2005) using the identical methodology to the present study.

Conflicting results for the three adolescent BPD studies may pertain to small sample size and differences in: subject selection, psychiatric comorbidities, and imaging methodology. The Goodman et al. (2011) adolescents were all inpatients with comorbid MDD and may represent a more severely ill group with greater treatment duration and comorbid psychopathology. Alternatively, Brunner et al. (2010) employed a whole brain VBM approach that may be less sensitive to group differences than the cytoarchitecturally derived approach used by Goodman and colleagues.

To date there are no fMRI studies of any brain region in adolescent BPD.

Volume studies examining adolescent BPD OFC volumes have been reported including the Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008) study that assessed 20 BPD adolescent outpatients referred to their at-risk clinic and compared them to 20 healthy controls, and the Brunner et al. (2010) and Goodman et al. (2011) studies described above. Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008) and Brunner et al. (2010) both found that BPD patient groups exhibited gray volume reductions in the OFC gray matter compared with HC, which was not noted in the Goodman et al. (2011) study.

Amygdala

Adult BPD

Structural imaging of amygdala volume in adult BPD, has yielded discrepant results, with reports of volume reduction (Driessen et al., 2000), perhaps reflecting excitotoxicity with volume loss, alongside studies citing no volume differences (Brambilla et al., 2004; New et al., 2007; Zetzsche et al., 2006). The amygdala has been viewed as the subcortical structure from which fear and perhaps anger may emerge. Amygdala activity is typically studied *after* exposure to a fear-inducing stimulus. fMRI studies in BPD show increased amygdala activity to specific types of stimulus, e.g., "unresolved" life events (Schmahl et al., 2006), emotional faces (Donegan et al., 2003), positive and negative emotional pictures (Hazlett et al., 2012), but not at rest as in MDD. Similar amygdala hyperactivity is seen in impulsive aggressive personality disordered subjects to emotional faces (Coccaro, McCloskey, Fitzgerald, & Phan, 2007). In addition, BPD patients seem to show particularly robust responses to other emotions, including anger (Minzenberg et al., 2007).

Adolescent BPD

Only one study has investigated amygdalar volumes in adolescent BPD and no fMRI data exists for amygdalar activity in BPD adolescents. Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008) did not find any differences in amygdalar volume between BPD and HCs.

Hippocampus

Adult BPD

In adult BPD, hippocampal volume loss has been reported in some (Chanen, Jackson et al., 2008; Chanen, Velakoulis et al., 2008; Schmahl,

Vermetten, Elzinga, & Douglas Bremner, 2003; Zetzsche et al., 2007) studies, but appears to be associated with the extent of trauma (Irle, Lange, & Sachsse, 2005) and abuse history (Brambilla et al., 2004), reflecting comorbidities with PTSD rather than specificity to BPD itself. An exception to this, however, is a recent study (Zetzsche et al., 2007) that found hippocampal volume reductions in BPD to be inversely correlated with aggressive but not impulsive symptomatology.

Adolescent BPD

Chanen, Jackson et al. (2008) and Chanen, Velakoulis et al. (2008) report hippocampal volumes on their cohort of 20 adolescent BPD subjects and found no differences in volume between BPD and HCs.

Other Brain Regions

Adult BPD

In BPD, findings of posterior cingulate activation were noted by New et al. (2002) in their 5-HT challenge study; however, other findings include volume loss (Hazlett et al., 2005) in the region and diminished uptake with positron emission tomography (PET) scanning in BPD females with dissociation and history of childhood sexual trauma, phenomena which complicate the clinical picture and obscure the direct contribution of BPD symptomatology to the posterior cingulate findings (Lange, Kracht, Herholz, Sachsse, & Irle, 2005). A recent meta-analysis of functional MRI studies of negative emotionality in BPD (Ruocco et al., 2012) identifies abnormal processing of negative emotion with heightened activity in the posterior cingulate cortex and insula and less activation in the subgenual ACC and DLPFC. Corpus callosum abnormalities have been reported in adult BPD with comorbid attention deficit disorder (Rüsch et al., 2007) but no differences were found in another pilot study (Zanetti et al., 2007).

Adolescent BPD

Using the same BPD adolescents from Chanen, Jackson et al. (2008), Chanen, Velakoulis et al. (2008), Takahashi, Chanen, Wood, Yücel et al. (2009) assessed the volume of midline brain structures and found a shorter adhesio interthalamica with no clinical correlations and no differences in the cavum septum pellucidum. In addition, in three separate analyses, with the original 20 Chanen teenage BPD subjects (Chanen, Jackson et al., 2008; Chanen, Velakoulis et al., 2008), corpus callosum volume, shape, curvature and third ventricle volume (Walterfang et al., 2010) insular cortex volume (Takahashi, Chanen, Wood, Walterfang et al., 2009), and superior temporal gyrus volume were assessed and compared to HC. All three studies had negative findings; however, insular volume reductions were found in impulsive adolescent BPD compared to non-impulsive adolescent BPD (Takahashi, Chanen, Wood, Walterfang et al., 2009) and superior temporal gyrus volume reductions were found in violent adolescent BPD compared to nonviolent BPD (Takahashi et al., 2010).

Diminished Serotonin Function

Adult BPD

There exists considerable evidence from multiple perspectives, including peripheral, postmortem, imaging, and antidepressant treatment studies, of diminished 5-HT function in BPD. The mechanism of the serotonergic abnormality in BPD has recently been examined with molecular neuroimaging studies. A PET study of 5-HT synthesis showed lower synthesis in men with BPD compared to controls in medial frontal gyrus, ACC, superior temporal gyrus, and corpus striatum; women with BPD had lower 5-HT synthesis compared to controls in right ACC and superior temporal gyrus (Leyton et al., 2001). Increased 5-HT2a binding in neocortical regions, including OFC and temporal cortex has been noted in BPD with impulsive aggression (Siever et al.,

unpublished data) as was increased binding in the hippocampus found in impulsive BPD females independent of mood (Soloff et al., 2007). More recently, we employed the 5-HTT PET radiotracer [11C]McN 5652 to show reduced availability of 5-HTT in ACC of personality disordered individuals with impulsive aggression compared to healthy controls, suggesting reduced serotonergic innervation in this brain region (Frankle et al., 2005). Interestingly, evidence shows an association between a particular haplotype in the 5-HTT gene (10 repeat of the VNTR intronic marker and the short form of a promoter polymorphism) and BPD, which lends further support to the notion that genetic differences in 5-HTT may play a role in the etiology of the disorder (Ni, Chan et al., 2006). Impulsive aggressive subjects with BPD are being studied in our lab with PET to determine whether reduced numbers of 5-HTT as indexed by [^{11}C] DASB-specific binding exists in the cingulate cortex.

Taken together, the published evidence regarding 5-HT suggests that a serotonergic abnormality that may underlie the impulsive aggressive symptoms of BPD and may be related to specific genetic risk factors, but the precise molecular nature of this abnormality is not yet clear.

adolescent BPD

We are not aware of any molecular studies in Adolescent BPD to date.

Conclusion

This chapter reviewed current neurobiological findings in BPD; concentrating on the work in adolescent-onset BPD. Genetic, neuroendocrine, neuropeptide, and neuroimaging data was highlighted.

The understanding of the neurobiology of adolescent BPD is in its infancy. While preliminary studies have focused mainly on volumetric studies of various brain regions and measurements of HPA axis components, this research is just beginning to gain momentum.

Findings so far await replication and discrepant results are not surprising given the small sample sizes, methodological differences and range of adolescent psychopathology including both comorbid Axis I disorders and severity of symptoms. Future work examining larger samples, using other methodologies such as fMRI, paying careful attention to comorbidity, and performing in a longitudinal capacity will enhance our understanding of adolescent-onset presentation of BPD.

The field is in an early stage, raising many more questions than have been answered. Important questions include: (1) Is adolescent-onset BPD a more severe biological disturbance than adult-onset BPD, similar to early-onset schizophrenia? (2) What can the study of adolescent-onset BPD inform about the early biological manifestations of BPD? (3) Are there potential targets for treatment interventions in adolescent BPD? (4) Can we identify biomarkers to identify those at greatest risk for more severe illness, adverse outcome, and positive or negative treatment effect? (5) What are the ways in which environmental influences interact with biological vulnerabilities? (6) How best to disentangle maladaptive from normative patterns of neurobiological function given the developing adolescent brain? (7) How does Axis II psychopathology interact with Axis I comorbidities?

Biological studies in both adult and adolescent-onset BPD will continue to shed light on the etiological mechanisms, genetic factors and pathological processes of the disorder, information essential to developing more effective screening, treatment, and preventive strategies.

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Behavior Genetic Approaches to BPD 10

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Definition and Core Constructs

As described by the fourth edition of the Diagnostic Statistical Manual of Mental Disorders (DSM-IV TR; American Psychiatric Association, 2000), borderline personality disorder (BPD) is a psychological disturbance typified by a pervasive pattern of emotional reactivity, a labile sense of identity, impulsivity, and instability in interpersonal relationships. More specifically, DSM-IV-TR diagnostic criteria for BPD include the combination of five out of nine total symptoms such as desperate efforts to avoid abandonment, tumultuous relationships due to alternating extremes of idealization and devaluation, an unstable sense of self, self-damaging impulsivity evidenced in at least two areas, recurrent suicidal behavior, intense mood reactivity lasting for short intervals of time, chronic feelings of emptiness, inappropriate and intense anger, and temporary stress-related paranoid ideation or related severe dissociative symptoms. Further, the severity of BPD lies in findings suggesting that in both clinical and community samples, the disorder frequently co-occurs with multiple Axis I disorders, including major depression, posttraumatic stress disorder, eating disorders, and substance use disorders (Grant et al., 2008; Oldham, Skodol, Kellman, & Hyler, 1995; Skodol et al., 2002; Trull, Sher, Minks-Brown, Durbin & Burr, 2000; Zanarini et al., 1998). Moreover, BPD has been found to co-occur with other personality disorder diagnoses (Barrachina et al., 2011), specifically with antisocial personality disorder and avoidant and dependent personality disorders (Zanarini et al., 1998).

Although currently characterized by scarcity, the literature on BPD heritability is expanding and thus far has implicated the role of both genetic and environmental influences. However, results surrounding the disorder's genetic transmission have not been consistent across studies. In the current chapter, we review the available evidence supporting the familial transmission of BPD, evidence for genetic and environmental effects on BPD across time, and disorders found to frequently co-occur with BPD. We will describe how the available evidence may inform the basic processes that underlie the etiology and trajectory of BPD. A final purpose of the chapter will be to cover special issues related to BPD regarding the information that genetic approaches can yield when attempting to understand the disorder's etiology and overlap with comorbid pathology.

Family Studies of BPD

A classic family study uses two sets of participants: probands (individuals with or

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without the target disorder) and their first-degree relatives such as siblings or parents. Next, rates of target psychopathology are compared between relatives of probands with the form of psychopathology in question to relatives of probands without target psychopathology. This type of study design allows for the examination of the familial transmission of the disorder.

Over the past two decades, more than 20 family studies have been conducted on BPD. Overall, the preponderance of research has shown that BPD is familial; probands with BPD are more likely to have first-degree relatives with BPD than probands without BPD (e.g., Links, Steiner, & Huxley, 1988; Loranger, Oldham, & Tulis, 1982) with few exceptions (e.g., Pope, Jonas, Hudson, Cohen, & Gunderson, 1983; Reich, Yates, & Nduaguba, 1989; Stone, Kahn, & Fley, 1981). Among the potential explanations for these differences, White, Gunderson, Zanarini, and Hudson (2003) suggested lack of assessment reliability and low methodological rigor as study limitations. In general however, the prevalence for BPD in relatives of BPD probands has been shown to range between 9.1 and 24.9 % (Bandelow et al., 2005; Links et al., 1988; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1988), suggesting the "familiality" of BPD.

However, in a recent review, White and colleagues highlighted several methodological problems in the extant familial studies on BPD. First, none of the studies using direct assessment of BPD had adequate sample sizes of BPD probands (Ns ranging from 17 to 80, White et al., 2003). In addition, the same investigation concluded that only Zanarini et al. (2004) used a larger sample of probands (with a sample size of 341). However, this latter study was still limited by using information about relatives of probands obtained from the BPD probands themselves rather than self-report from their first-degree relatives (White et al., 2003). This reliance on information from probands to determine a diagnosis in relatives rather than direct interviews of these relatives presents challenges in the conclusions that may be drawn from these designs in that they may be influenced by distortions in probands' self-report, including inaccurate description of the affected family member memory recall failure, among others. A more recent study (Gunderson et al., 2011) was able to address some of these methodological limitations by recruiting a large sample of probands with and without BPD ($N = 368$), siblings, and parents ($N = 885$). Study results indicated that if the proband met DSM-IV criteria for BPD, the family member exhibited almost a threefold risk of familial aggregation relative to those with proband lacking the diagnosis (Gunderson et al., 2011).

An additional methodological consideration that may limit the conclusions drawn from family studies is that these are unable to parse genetic and environmental influences on the disorder's transmission. For example, first-degree relatives typically share both genes and environment. And, a number of environmental risk factors for BPD have been identified as potential mechanisms through which individuals are placed at an increased risk of developing BPD such as parenting behaviors and abuse (see Trull, 2001; Zanarini et al., 1997). Yet, the presence or absence of these risk factors is believed to nonrandom, and such experiences may be genetically influenced in their own right (Jaffee, Caspi, Moffitt, & Taylor, 2004; Jaffee & Price, 2007; Lyons et al., 1993; Schulz-Heik et al., 2010; Stein, Jang, Taylor, Vernon, & Livesley, 2002). In other words, children born to a parent with BPD may be at an increased genetic risk for maladaptive behavior, but may also be likely to experience environmental risk factors such as maladaptive parenting or a history of abuse (Distel, Rebollo-Mesa, Willemsen, Derom, & Trull, 2009; Gunderson & Lyons-Ruth, 2008). Therefore, results from family studies may reflect genetic transmission, environmental transmission, or the combination or even the interplay of the two. In addressing some of these limitations, quantitative genetic approaches (described in the section below) have been utilized to parse genetic and environmental transmission. These designs also allow the parsing of "purely" genetic and environmental effects from gene–environment interplay.

Quantitative Genetic Studies of BPD

Historically, a way in which the interplay between the genetic and environmental influences involved in the expression of BPD has been examined is within classic twin studies. Twin studies are based on the premise that monozygotic (MZ) twins share 100 % of their genes, whereas dizygotic (DZ) twins share on average half their genes—of those genes that vary between people. These designs allow estimation of genetic influence by comparing the extent to which MZ twins are more similar on a construct than are DZ twins or regular non-twin siblings. The excess of observed twin resemblance after accounting for genetic influence is indicative of shared environmental influence (nongenetic influences that contribute to similarity between twins such as common familial experiences, neighborhood influences, or socioeconomic status). Environmental influences contributing to differences between twin pairs represent non-shared environmental influences and also capture measurement error (Plomin, DeFries, & McClearn, 1990).

Heritability in children. Currently, only a modest body of research exists examining the heritability of BPD. A small study comprises 112 twin pairs between the ages of 4 and 15 (70 monozygotic and 42 dyzygotic pairs) (Coolidge, Thede, & Jang, 2001) assessed dimensionally via the DSM-IV diagnostic criteria reported a BPD heritability estimate of 76 %. The authors indicated that an unstable self-concept that was present in children from a young age was suggestive of a possible inherited neurobiological dysfunction. As part of the study's limitations, the authors indicated that the investigation comprised a small sample size, and did not address the effects of age and gender.

Although less directly relevant, there are studies investigating variables that have long been considered putative vulnerabilities or endophenotypes for BPD, including negative affect, aggression (Ligthart, Bartels, Hoekstra, Hudziak, & Boomsma, 2005)—especially relational aggression (Crick, Murray-Closse, & Woods, 2005; Gottesman & Gould, 2003; Werner & Crick, 1999), behavioral disinhibition, and affective dysregulation. A recent study examining negative affect in children 7–13 years old estimated the contribution of genetic influence to be 48 % and shared environmental factors as 38 %, and a low contribution of non-shared environmental factors at 13 % (Mikolajewski, Allan, Hart, Lonigan, & Taylor, 2012). Another study of girls ages 10–12 reported the genetic contribution to negative affect at 63 % with the shared environmental influence of 53 % and a non-shared environmental influence of 57 % (Neiss, Stevenson, Legrand, Iacono, & Sedikides, 2009). The heritability of direct aggression in children at 7 years old has been demonstrated to be 53 % for males and 60 % (Ligthart et al., 2005). The same study also reported the heritability of relational aggression to be 66 %, with no gender differences (Ligthart et al., 2005). In a meta-analysis of impulsivity/behavioral disinhibition on studies from infants to adolescents, the additive genetic effect was estimated at 34 % with a nonadditive genetic effect of 16 % and a non-shared environmental effect of 50 % (Bezdjian, Baker, & Tuvblad, 2011).

Heritability in adolescents. Although limited, investigations surrounding the heritability of BPD are burgeoning. A study conducted in the Netherlands ($n = 3{,}918$), Belgium ($n = 904$), and Australia ($n = 674$) with adolescent pair twins found that across these countries, estimates of genetic influences of BPD features were approximately 42 % (Distel et al., 2008). Of note was that the remaining 58 % of the variance was accounted for by non-shared environmental factors and that shared environmental factors did not contribute to the variance. As will be discussed in the next subsection, the pattern surrounding the negligible contribution of shared environment has also been reported in adults. A more recent investigation (Bornovalova, Hicks, Iacono, & McGue, 2009), explored the longitudinal course and heritability of BPD traits in adolescence over a period of 10 years. Participants in this sample ranged from ages 14 to 24. Participants in this sample were followed longitudinally from ages 14 to 24. Results from this study suggested that the influences of BPD

heritability were contingent upon age. For example, at age 14, there was a modest contribution of genetic (31 %) and shared environmental (20 %) factors, and a large contribution of non-shared environmental factors (50 %). At age 17, there was a larger contribution of genetic (38 %) and smaller contribution of shared environmental (12 %) factors, whereas the non-shared environmental influences stayed relatively stable (51 %) (see also Bornovalova, Hicks, Iacono, & McGue, 2012 for similar results).

With regard to heritability of putative vulnerabilities for BPD in adolescents, in general, twin and family studies have shown aggression in general (Hines and Saudino, 2004) and relational aggression in particular (Tackett, Waldman, & Lahey, 2009) to highly heritable. In a sample of 1,981 6- to 18-year-old twin pairs (36 % female, 34 % male), relational aggression was assessed via maternal and self-report using a structured interview (Tackett et al., 2009). Results indicated that that the common variance between maternal and self-report of relational aggression was substantially influenced by additive genetic (accounting for approximately 63 % of the variance in the construct) and shared environmental influences (accounting for approximately 37 %). In a large study of adolescent twins at age 17, negative affect (as indicated by internalizing symptoms), was estimated to have a heritability of 36 % (Blonigen, Hicks, Krueger, Patrick, & Iacono, 2005). The same study also reported behavioral inhibition to have a heritability of 53 %.

Heritability in adults. Studies focusing on the heritability of adult BPD have generally provided inconsistent results, with heritability estimates ranging between 42 and 70 %. For example, findings from a study examining BPD heritability in an adult sample of 92 MZ and 129 DZ pairs suggested that the additive genetic effect on the disorder was 70 %, with 30 % of the remaining effect was a result of non-shared environmental factors; the effect of the shared environment was virtually zero (Torgersen et al., 2000). The lack of shared environmental influences was also reported in a later study also led by Torgersen et al. (2008) involving 1,386 Norwegian twin pairs interviewed using the Structured Interview for DSM-IV Personality Disorders. In this investigation, heritability was estimated at 35 % for BPD traits (Torgersen et al., 2008). Further, recent large-scale studies heritability estimates have shown to be moderate, ranging from 35 to 40 % (Bornovalova et al., 2012; Distel et al., 2008; Kendler et al., 2011; Kendler, Myers, & Reichborn-Kjennerud, 2011). Likewise, in the above-described longitudinal study, Bornovalova et al. reported that, at age 20, heritability estimates were ~35 %, shared environmental influences were 8 %, and non-shared environment was 57 %. By age 24, most of the influences were additive genetic (~46 %) and non-shared environmental (54 %).

Similar findings have been obtained when examining putative temperamental vulnerabilities contributing to BPD, including negative affect, affective dysregulation and behavioral undercontrol. For these vulnerabilities, moderate heritability estimates of 40–60 % have been reported (Jang, Livesley, Vernon, & Jackson, 1996; Livesley, Jang, Jackson, & Vernon, 1993; Livesley, Jang, & Vernon, 1998). Research examining different BPD factors, including self-harm, affective instability, negative relationships, and identity problems, found that overall the heritability of the latent BPD factor was 51 % and that unique environmental influences accounted for 49 % in a sample of 5,533 adult twins (age 18–90) and 1,202 siblings from the Netherlands, Belgium, and Australia (Distel et al., 2010). For each BPD factor, around 50 % of its variance was explained by the latent BPD factor, except for self-harm, for which the latent factor accounted for 33 % (Distel et al., 2010).

Considerations in Interpreting Disparate Findings

Altogether, investigations to date demonstrate a few inconsistencies. First, BPD heritability rates have been reported in different studies ranging between 35 and 76 %. Further, the contribution of genetic and shared environmental influences on BPD has been shown to be a function of age.

For example, BPD heritability studies in adolescents have shown that genes contribute to 31 % of the variance for 14-year-old adolescents, increase to 35–38 % at ages 17 and 20, until increasing to 46 % at age 24. Likewise, shared environment contributes to approximately 20 % of the variance for 14-year-old adolescents, but declines steadily to 12 % at age 17, 8 % at age 20, until decreasing to 0 % at age 24 (Bornovalova et al., 2009). It should be noted that these shifting heritability and shared environmental influences are estimated using the *same adolescents followed longitudinally*. There are a few possible explanations for these inconsistencies. However, before providing possible explanations, there are two points that need to be made—regarding both the stability of BPD estimates themselves, as well as the stability (and meaning) of heritability and shared environmental estimates over time.

Foremost, it is important to note that, despite the classic "categorical" conceptualization of BPD implying lifelong stability, level of BPD features actually varies a great deal over a lifespan. In a recent study of the trajectory of BPD in adolescent females, Bornovalova et al. (2009, 2012) reported that BPD feature levels are relatively high and stable at age 14 and 17; begin to decline by age 20; and decline even further by age 24. Similar findings have been reported in two other studies of adolescents, with low rates of diagnostic continuity over 2–4 years (Biskin, Paris, Renaud, Raz, & Zelkowitz, 2011; Mattanah, Becker, Levy, Edell, & McGlashan, 1995). Further, in adult samples, over a 1-year follow-up, Shea et al. (2002) found that only 41 % of individuals diagnosed with BPD at baseline continued to meet criteria for the disorder. Further supporting these results, Zanarini et al. (2007) later examined the time to remission of BPD symptomatology over a 10-year prospective follow-up in a sample of adults and found that 50 % of the symptoms examined declined across individuals and that only 15 % of those who endorsed these symptoms during baseline, continued to do so at the follow-up. Altogether, these findings reflect the high degree of variability in BPD symptomatology, and as a result, the low rates of BPD diagnostic continuity. Likewise, two other studies demonstrated that BPD features are elevated in mid-adolescence and then demonstrate a declining trajectory (Bernstein, Cohen, Velez, & Schwab-Stone, 1993; Bornovalova et al., 2009; Johnson et al., 2000).

Second, it is important to know that *heritability estimates themselves* are not stable over time, and this is true across multiple types of psychopathology. In a meta-analysis of age-related changes in heritability of behavioral phenotypes over adolescence and young adulthood, Bergen and colleagues found a statistically significant increase in heritability of externalizing behaviors, anxiety symptoms, depressive symptoms, IQ, and social attitudes from adolescence to young adulthood (Bergen, Gardner, & Kendler, 2007). That is, many disorders shift from being relatively equally influenced by genetic, shared environmental, and non-shared environmental influences in adolescence to being influenced by genetic and non-shared environmental influences solely in adulthood. The above-mentioned study of Bornovalova et al. (2009, see also Bornovalova et al., 2012) followed this pattern.

Possible Explanations for Shifting Estimates of Heritability and Shared Environment

This shift in heritability may represent one of the four possible causes. The first, and perhaps simplest, explanation for this shift in heritability is that it may be an artifact of measurement error. More specifically, we struggle with measuring these constructs in youth populations, and the shift from ACE to AE models may be accounted for by the fact that kids become better reporters over time as they mature and/or gain insight. Additionally, this shift may be driven by changes in reporter, from parent report in young children to self-report in older children.

If the measurement considerations are ruled out, there is a second possibility that if the shift in heritability could represent an increase or change in gene expression. In response to environmental or developmental cues (e.g., puberty, Whitelaw

& Whitelaw, 2006), genes can turn "on" and "off." Indeed, adolescence coincides with puberty, a time where hormonal changes may influence the expression of genetic vulnerabilities (Cicchetti & Rogosch, 2002; Walker, Sabuwalla, & Huot, 2004). The stress-response system may be particularly important in understanding the intersection of behavioral and biological interactions influencing the developmental discontinuities observed in the heritability of psychopathology (Walker et al., 2004). Additionally, the environmental context itself can affect the influence of a gene and its product (e.g., Bergen et al., 2007; Gottlieb, 2000, 1998; Zhang & Meaney, 2010), thus influencing the phenotypic expression of the vulnerability.

Third, shifting heritability may represent reduced common environmental influences—the fact that environmental constraints on genetic expression become looser with age. That is, in adolescence, parents exert a strong degree of control over the behavior of their offspring. This influence manifests as shared environment in classic heritability estimates. As parents exert less control and influence on development, offspring have more freedom to express their genes. This, in turn, leads to a reduction in common environmental effects as well as an increase in heritability estimates.

Finally, it could be due to a rising importance of active gene–environment correlations (Plomin, DeFries, & Loehlin, 1977). Active gene–environment correlations arise when a genetically influenced characteristic of an individual leads that individual to seek out certain environments. Additionally, active gene–environment correlations predict that the genetically identical MZ twins will be more likely to select similar environments than the less genetically similar DZ twins. This may be reflected by consistently high (or even increasing) MZ twin correlations and progressively lower DZ twin correlations and what may account for increasing heritability.

This might be reflected by, for instance, individuals with BPD seeking out (although hardly intentionally) damaging events like volatile, violent relationships, or drug ridden neighborhoods. In support of this notion, in a study of adults, Distel et al. examined the genetic correlation between BPD and several environmental stressors (divorce, violent assault, sexual assault, and job loss). Results indicated that BPD features had a genetic correlation of 0.27 with divorce, 0.28 with violent assault, 0.39 with sexual assault, and 0.28 with job loss. These findings suggest that once individuals are on their own with a reduced influence of familial factors, genetic factors that are responsible for BPD characteristics may lead to a selection of "fitting" (frequently maladaptive) environments. Likewise, Bornovalova et al. (2013) reported a genetic correlation of 0.78 between BPD features and childhood trauma, indicating that there is a common genetic influence on the propensity to (self-reported) childhood emotional, physical, and sexual abuse and adult BPD features. Altogether, these findings suggest that BPD-related genes control exposure to selective environments (see Kendler & Eaves, 1986; Kendler & Baker, 2007; Kendler, 1995 for a discussion of this topic). Interestingly, this notion is consistent with the seminal conceptualization of the development of BPD (Linehan, 1993), suggesting that BPD traits form from the transaction of genetic vulnerability and environmental risk. Indeed, Linehan (1993) and Crowell, Beauchaine, and Linehan (2009) suggested that individuals at risk for BPD evoke maladaptive responses from their caregivers—and later on, may evoke or select maladaptive responses from the environment "at large."

Unfortunately, classic twin studies are unable to disentangle genetic and environmental effects from the notion of genetic control of environmental exposure, or to distinguish between these three possible processes. Future work using more complicated methodology is needed to tell which of these three processes is integral to understanding what is going on in BPD, and we review possibilities for this type of future work in the Future Directions section.

Shared Liability and the Etiological Models of BPD

An additional benefit of genetically informative designs is that they can also detect shared liability between BPD and other forms of psychopathology. For instance, in the case of family studies, researchers are able to examine what type of psychopathology (other than BPD) a family member of a BPD proband has—or vice versa. Likewise, in twin studies, researchers are able to examine the genetic and environmental influence common to both BPD and other forms of psychopathology.

The ability to examine shared liability between BPD and other forms of psychopathology is important for several reasons. First, as noted above, BPD is highly comorbid with multiple forms of both internalizing (e.g., depression and anxiety disorders) and externalizing (e.g., substance abuse and antisocial personality disorder) psychopathology (Grant et al., 2008; Kendler, Aggen et al., 2011; Kendler, Myers, & Reichborn-Kjennerud, 2011; Oldham et al., 1995; Skodol et al., 2002; Trull, Sher, Minks-Brown, Durbin & Burr, 2000; Zanarini et al., 1998). An internalizing dimension indicates a propensity to express and direct distress inwards and is typical of mood and anxiety disorders. The externalizing dimension, on the other hand, characterizes individuals' tendency to express distress outwards, and includes substance use disorders (James & Taylor, 2008). Further breaking these dimensions down, the internalizing factor contains two subfactors: anxious-misery (also known as "distress") and fear (Kendler, Prescott, Myers, & Neale, 2003; Krueger & Markon, 2006), which is relatively stable over time (Vollebergh et al., 2001) and shown high rates of heritability (Hicks, Krueger, Iacono, McGue, & Patrick, 2004). Given the observation that BPD individuals tend to endorse both of these dimensions (e.g., anger outbursts, feelings of abandonment), researchers have attempted to identify the best fitting models for BPD examining both externalizing and internalizing factors.

Examining shared liability through genetically informed designs is also important because more recent models of psychopathology in general place BPD as cross-loading on both internalizing and externalizing psychopathology. James and Taylor (2008) for example, found that in a population sample that comprises 1,197 individuals between the ages of 19 and 22, BPD served as a multidimensional indicator of the externalizing factor and the anxious-misery subfactor for both men and women. An additional nationally representative sample of 34,653 adult participants yielded similar results, in which BPD functioned as an indicator of both the distress component of INT and EXT across gender (Eaton et al., 2011). The corroborating results suggest that BPD is related to both INT and EXT with minimal gender differences. Examining the common genetic and environmental contributions to internalizing and externalizing dimensions and BPD may be beneficial in understanding the latter's structure.

Family Studies of Shared Liability

Several studies have documented family members of probands with BPD with both INT and EXT (White et al., 2003). For instance, two studies report that family members of BPD are significantly more likely to have a mood disorder (Major Depressive Disorder, Bipolar I Disorder) than family members of probands without BPD (Pope et al., 1983; Riso, Klein, Anderson, & Ouimette, 2000). With regard to the EXT dimension, three studies report that family members of BPD are significantly more likely to have an alcohol or drug use disorder and antisocial personality disorder than family members of probands without BPD (Loranger & Tulis, 1985; Reich, 1989; Schulz et al., 1989). A more recent study (Zanarini, Barison, Frankenburg, Reich, & Hudson, 2009) reported that, compared to relatives of probands with "other" personality disorder, relatives of probands with BPD were 1.4–4.9 more times more likely to have a mood (Bipolar I, Major Depressive, and

Dysthymic) disorder; 2.9–2.1 times more likely to have an alcohol or drug use disorder; and 2.5–12 times more likely to have an anxiety (Obsessive Compulsive, Generalized Anxiety, and Panic) disorder. The highest familiality was shown for Generalized Anxiety Disorder, with families of BPD probands showing a 12-fold increase in the probability of reporting Generalized Anxiety Disorder. Thus, overall, seminal and recent family studies indicate that BPD shares "familiality" with both INT and EXT psychopathology.

Genetic Studies of Shared Liability

Several studies have documented a shared genetic and, to a lesser degree, environmental overlap between BPD with INT and EXT psychopathology (Kendler et al., 2008; Kendler, Aggen et al., 2011; Kendler, Myers, & Reichborn-Kjennerud, 2011). For instance, in a recent elegant study, Kendler, Aggen et al. (2011), Kendler, Myers, and Reichborn-Kjennerud (2011) reported that BPD shares unique environmental effects with all personality disorders and with Axis I INT disorders. Genetically, BPD is more closely tied to Axis I and II EXT disorders. Following this work and attempting to integrate the genetic and environmental overlap in BPD of both INT and EXT dimensions, Bornovalova et al. (2011) found BPD and EXT showed moderate common genetic and small common non-shared environmental influences at both ages 17 and 24, and both cross-sectionally and longitudinally. Likewise, BPD showed common genetic—and to a lesser degree, non-shared environmental effects with INT psychopathology at both ages, and for both cross-sectional and longitudinal analyses. And, as this study examined BPD traits in both males and females, it is important to note that there were very few gender differences in common influences at either age. Altogether, findings from these studies suggest that BPD loads on and overlaps with INT and EXT disorders, which seemingly indicates that BPD is a "transitional" disorder (Kendler, Aggen et al., 2011; Kendler, Myers, & Reichborn-Kjennerud, 2011). The research on BPD seems to implicate that the underlying mechanism driving the etiology of BPD is related to both INT and EXT symptomatology. Finally, Bornovalova et al. (2012) examined the genetic and environmental influences to BPD features and substance use at ages 14 and 17. Results indicated that shared environmental effects primarily accounted for the comorbidity between BPD traits and substance use at age 14, whereas genetic effects primarily accounted for their co-occurrence at age 18. These developmental trends may be due to a shift from passive gene–environment correlation (where parents provide both genetic and environmental influences on child psychopathology) in childhood and early adolescence to active gene–environment correlation processes in late adolescence and adulthood where offspring select their own environments that may predispose one to substance use (Scarr & McCartney, 1983). This study suggests that not only individual disorders show shifting genetic and environment influences; *common* influences on multiple forms of psychopathology also undergo changes and may inform research on the etiology and maintenance of BPD.

Future Directions in Genetics of BPD Research

Much remains unknown regarding the role of genetic and environmental factors in the development and maintenance of BPD. This chapter has highlighted recent research that indicates the shifting heritability and shared environmental influences on BPD across development, but the mechanisms underlying this shift remain relatively speculative. The first possibility underlying this shift is, of course, the simplest: the problem of measurement. Our difficulty measuring BPD in youth populations may influence the changes observed in these constructs over time. As we noted above, developmental changes and improved insight or changes in rater may significantly contribute to the changes observed in BPD constructs. This possibility is noteworthy and

must be addressed or ruled out before we consider any other "more interesting" explanations.

If measurement issues are not driving changes in heritability, then, as we noted above, there are at least three possible mechanisms driving these shifts: the activation of previously inactive genes; the diminishing influence of shared environment; and the emerging importance of BPD-related genes leading to a selection of specific (mainly maladaptive) environments. To date, only one study has tested the latter possibility (Distel et al., 2011), with findings suggesting that individuals genetically predisposed to BPD traits are likely to select environments including divorce, exposure to violent and sexual assault, and job loss. However, this report was cross-sectional. More studies are needed to test, for instance, if BPD traits in adolescence show a genetic correlation with future maladaptive environmental events. Moreover, it would be informative to expand the range of environmental events which show a genetic overlap a genetic predisposition to BPD. This, in turn, will test the possibility that genetic influences on BPD increase with age due to the genetic control of exposure to environmental events. This type of work requires methodology beyond classic twin and family studies. Multivariate biometric models in adult twin studies that collect information across time on both BPD features and environmental events (and then correlate the genetic and environmental influences on these events across time) allow researchers to examine how individuals select their own environment.

There are several other methods that remain unexplored in BPD research. Research that follows shared etiology between BPD features and other (INT and EXT) psychopathology over development is also needed to see whether findings found in adults are consistent across kids, adolescents, adults, and aging individuals. Using this methodology would enable researchers to determine common liability between BPD and other forms of psychopathology—and in turn, understand the etiological influences on BPD across time.

The field would also benefit from research conducted using adoption studies. In this chapter, we did not review adoption studies in this chapter (due to the complete lack of these on the topic of BPD). In an adoptive family (where caregivers do not share genes with offspring), only environmental influences can be the "cause" of an environmental effect (e.g., maladaptive parenting) on offspring BPD traits. Of course, biological families provide both genes and environmental influences on offspring pathology. By comparing the effect of a target environmental event on offspring, pathology between adoptive and biological families allows one to disentangle the genetic and environmental influences on BPD psychopathology—processes that are impossible to disentangle in classic family and twin studies.

Continued research in the field of molecular genetics would also benefit the study of BPD. The area is in its infancy, and, to our knowledge, almost no studies have specifically examined the genetics of BPD per se (for an exception, see Wilson et al., 2009, documenting the association of BPD diagnosis with the tryptophan hydroxylase-1 A218C polymorphism). Moreover, traits associated with BPD (i.e., suicidality, negative emotionality, and impulsivity) have been linked with the s-allele of 5HTTR (Amstadter et al., 2012; Hayden et al., 2008) and DRD4 (Keltikangas-Järvinen, Räikkönen, Ekelund, & Peltonen, 2003; Sheese, Voelker, Rothbart, & Posner, 2007). Additional studies have examined the interaction of these—as well as other—alleles with environmental stressors such as child emotional or physical abuse on traits associated with BPD (e.g., Perroud et al., 2008; Simons et al., 2011; Perroud et al., 2010; for a detailed review, see Carpenter, Tomko, Trull, & Boomsma, 2013). However, these studies are scarce and difficult to interpret for several reasons. First, it is well known that effect sizes for individual polymorphisms predicting complex phenotypes tend to range from small to moderate (Plomin, Haworth, & Davis, 2009). These limitations are compounded when considering the typically limited sample sizes used in molecular genetic research. Thus, there has been difficulty in replicating the findings linking specific polymorphisms with behavioral phenotypes (e.g., Hawi, Millar, Daly, Fitzgerald, &

Gill, 2000). Additionally, it is unlikely that complex syndromes such as BPD are due to variants in any single gene; rather, BPD is more likely to be polygenic in nature (Goldman, Oroszi, & Ducci, 2005). In other words, multiple genes influence phenotypes, and each gene has a small effect on phenotypic expression. To address this issue, researchers have recently begun to develop methods by which risk indices for specific behaviors are assigned based on an individual's genetic makeup. Specifically, these indices take into account a *set* of genes across a given system (e.g., dopamine or serotonin systems). In such a way, one is able to describe a complex phenotype using a more complete account of the underlying factors while at the same time bypassing difficulty in replicating genetic effects. To date, this method has been applied to phenotypes such as sensation seeking and cocaine dependence with success (Derringer et al., 2010; Derringer et al., 2012). However this methodology has yet to be applied to the study of BPD. Molecular genetic approaches such as those that take into account a set of genes across a given system may be fruitful in BPD research and should be considered. Ultimately, information gleaned from such research could inform the design of preventative and treatment interventions, which could reduce the suffering associated with this form of mental illness.

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A Biosocial Model of BPD: Theory and Empirical Evidence

11

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Introduction

In this chapter, we describe our biosocial developmental model of borderline personality disorder (BPD) with particular attention to findings that have emerged since our initial review (Crowell, Beauchaine, & Linehan, 2009). We outline a complex, heterotypic trajectory from childhood vulnerabilities to adult BPD. Specifically, we hypothesize that trait impulsivity interacts with family-level risk factors to increase risk for BPD. We also theorize that self-inflicted injury and other clinical features emerge by adolescence and are reliable predictors of later BPD for some adolescents. Finally, we highlight future directions for research, intervention, and prevention of adolescent BPD traits.

For the past 30 years, many mental health professionals have operated under the assumption that BPD is a persistent, pervasive, and relatively intransigent condition that emerges in late adolescence or early adulthood (American Psychiatric Association [APA], 2000). There are several beliefs about BPD and other personality disorders (PDs) that follow from these core assumptions (Crick, Murray-Close, & Woods, 2005). First, PDs are thought to be as stable and enduring as other personality traits, such as conscientiousness and neuroticism. Second, PDs are presumably more difficult to treat than many other disorders, especially those which are episodic and less characterological in nature. Third, diagnosing PDs at a young age is viewed as inappropriate because personality is assumed to be coalescing in late adolescence. Consequently, many are reluctant to diagnose PD until adulthood given that stigma associated with longstanding disorders of character.

Over the past decade, evidence has mounted that challenges these assumptions about PDs, especially BPD (Lenzenweger, Clarkin, Levy, Yeomans, & Kernberg, 2012; Skodol et al., 2002; Zanarini, Frankenburg, Hennen, & Silk, 2003). Our research, and that of others, has introduced a different set of hypotheses (see Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Crowell et al., 2009 for reviews). This work is guided by evidence that BPD is no more stable or intractable than other psychiatric syndromes, and more importantly, the diagnosis does not emerge de novo in young adulthood (e.g., Burke & Stepp, 2012; Stepp, Burke, Hipwell, & Loeber, 2012). Rather, there are reliable precursors to BPD that can be identified and treated earlier in development. This conceptual shift follows from the developmental psychopathology (DP) perspective. The DP paradigm views both normal and atypical outcomes as emerging from complex transactions between individual-level vulnerabilities (e.g.,

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trait impulsivity) and environmental risks (e.g., child maltreatment). Importantly, these outcomes are fluid across time (see Beauchaine & McNulty, 2013) and are not viewed as static endpoints.

In this chapter we review our biosocial developmental model of BPD, with particular attention to findings that have emerged since our Crowell et al. (2009) review. One promising area of growth is longitudinal research. There are now several studies that have followed vulnerable/at risk children into adolescence or adulthood when BPD and related clinical problems could be assessed. Unfortunately, none of these studies were designed prospectively[1] and, as a consequence, there is often poor measurement of early risk for BPD. However, when this work is dovetailed with (a) the growing cross-sectional literature on adolescent borderline pathology (BP) and (b) retrospective accounts collected from adults with BPD, a more comprehensive representation begins to emerge. As with any psychiatric disorder, better understanding of the developmental process leading to BPD is critical for early identification and prevention (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008).

Definitions and Core Constructs

There are diverse approaches to conceptualizing borderline pathology in childhood and adolescence. Some researchers apply criteria listed in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; APA, 2000) to pediatric populations (see Sharp & Romero, 2007 for a review). However, with the exception of older adolescents, few youth reach the diagnostic threshold for a BPD diagnosis. Moreover, children who meet criteria for BPD rarely maintain the diagnosis by late adolescence or adulthood. In fact, very early borderline pathology is a better predictor of other disorders than of BPD, such as conduct disorder (CD), attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), depression, psychotic symptoms, and antisocial personality disorder (ASPD; Lofgren, Bemporad, King, & Lindem, 1991; Vela, Gottlieb, & Gottlieb, 1983). Thus, early manifestations of BPD may not take the same form as the adult diagnosis, which precludes simple downward extension of the DSM criteria.

Given the complexity of longitudinal pathways to psychiatric disorders, developmental psychopathologists seek to identify vulnerabilities and risk factors associated *probabilistically* with later BPD. Adherents to the DP perspective view normal and abnormal development as equally important to understanding the emergence of psychopathology. Indeed, many psychiatric disorders reflect extreme expressions of normative ontogenic processes, or mark failure to navigate one or more typical developmental tasks (Cicchetti & Rogosch, 2002; Macfie, 2009; Sroufe & Rutter, 1984). Furthermore, children may make a transition between adaptive and maladaptive trajectories at any age and, consequently, each developmental stage represents a potential point for intervention. There are two concepts from the DP literature that are especially relevant: (1) equifinality and multifinality and (2) homotypic and heterotypic continuity.

Developmental psychopathologists seek to account for both equifinality and multifinality across development (Cicchetti, 1984). *Equifinality* refers to multiple developmental trajectories converging on an apparently single behavioral outcome. For example, several distinct pathways (e.g., early adversity, peer rejection, acute stressors) have been identified that lead to depression (Gotlib & Joormann, 2010). In contrast, *multifinality* occurs when one vulnerability or risk factor prompts many diverse outcomes. For example, child abuse may potentiate depression,

[1] The ideal prospective longitudinal study would assess relevant genetic risk markers and temperamental characteristics in infancy, neurological functioning, parental psychopathology and health behaviors across development, contextual risk factors at multiple levels of analysis, and internalizing, externalizing, and BPD-specific problems at every time point. By early adolescence, adult BPD criteria could be assessed along with developmentally normed measures. Genotyping would allow for tests of gene–environment interactions (G × E) and correlations (*r*GE). Such studies would continue into young adulthood, at which time most cases of BPD could be identified. Current longitudinal studies either neglected to assess BPD-relevant problems across development, have small sample sizes, or did not follow participants into young adulthood.

ODD, no diagnosis, or numerous other consequences (Cicchetti & Toth, 1995). Multifinality and equifinality are important when considering the emergence of BPD across development (Beauchaine et al., 2009). Many youth with early borderline features do not develop BPD (multifinality), whereas adults with BPD traverse different developmental pathways to the disorder (equifinality).

Heterotypic continuity refers to changes in the manifest form of psychopathology, despite a common underlying vulnerability (see Beauchaine & McNulty, 2013). For example, many adult males with ASPD followed a developmental trajectory that began with early hyperactivity in preschool, and progressed to ODD, CD, substance use, and ASPD across development (Beauchaine, Hinshaw, & Pang, 2010). In this case, expression of heritable trait impulsivity changes across development such that different diagnoses are met at different ages.

In part, heterotypic continuity is an artifact of a diagnostic system that specifies different diagnoses based upon developmental shifts in behavior—even when each disorder can be explained by a common underlying vulnerability. Substance use and ADHD appear to arise from a common stable inter-individual difference (Chang, Lichtenstein, & Larsson, 2012; Kousha, Shahrivar, & Alaghband-Rad, 2012; Wilson & Levin, 2005). However, the current diagnostic manual marks topographical changes in behavior with distinct categorical diagnoses. Developmental psychopathologists believe that psychiatric syndromes are better understood by focusing on the underlying vulnerability traits that give rise to broad classes of behavior. This perspective has been supported across factor analytic twin studies of both children and adults, which find that disorders along the externalizing spectrum are highly comorbid and derive from a common etiology (e.g., Baker, Jacobson, Raine, Lozano, & Bezdjian, 2007; Krueger, 1999; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Krueger, Markon, Patrick, & Iacono, 2005; Siewert, Stallings, & Hewitt, 2003).

Homotypic continuity occurs when a person maintains a single diagnosis across development. Social phobia, for example, is fairly continuous across developmental stages (Bittner et al., 2007; Pine, Cohen, Gurley, Brook, & Ma, 1998). In many cases, depression is also homotypically continuous, especially from late adolescence into adulthood (Rutter, Kim-Cohen, & Maughan, 2006). Research on BPD, however, reveals a developmental pathway characterized by both heterotypic and homotypic continuity. There is emerging evidence that many adults with BPD exhibited ADHD symptoms as children, then followed an externalizing trajectory characterized by conduct problems, substance use, and adolescent self-injury (Beauchaine et al., 2009; Hinshaw et al., 2012; Stepp et al., 2012). However, by late adolescence, features specific to BPD emerge and show a high degree of homotypic continuity with the adult diagnosis (Bornovalova, Hicks, Iacono, & McGue, 2009; Lenzenweger, 1999; Winograd, Cohen, & Chen, 2008).

Following from the DP perspective, we propose a complex, heterotypic trajectory from childhood vulnerabilities to adult BPD. We hypothesize that precursors to BPD appear early in development, and include heritable biological vulnerabilities that are potentiated by contextual risk. According to our model, development of BPD is most likely to occur among individuals who inherit trait impulsivity from their parents, and subsequently acquire poor emotion regulation skills, primarily through socialization mechanisms. From this perspective, BPD results from complex interactions between individual-level vulnerabilities and environmental risk factors, as is the case for many if not most psychiatric disorders (see e.g., Cicchetti & Dawson, 2002). Below we review evidence that invalidating family environments, especially those characterized by intermittent reinforcement of negative affect, heighten risk for BPD among vulnerable individuals.

Finally, we hypothesize that self-inflicted injury (SII) and other BPD features emerge by adolescence and are reliable predictors of later BPD (Crowell et al., 2009, 2012; Lamph, 2011). Indeed, BPD and SII appear to derive from a common etiology as evidenced by shared biological vulnerabilities, environmental risk factors, and maladaptive coping strategies

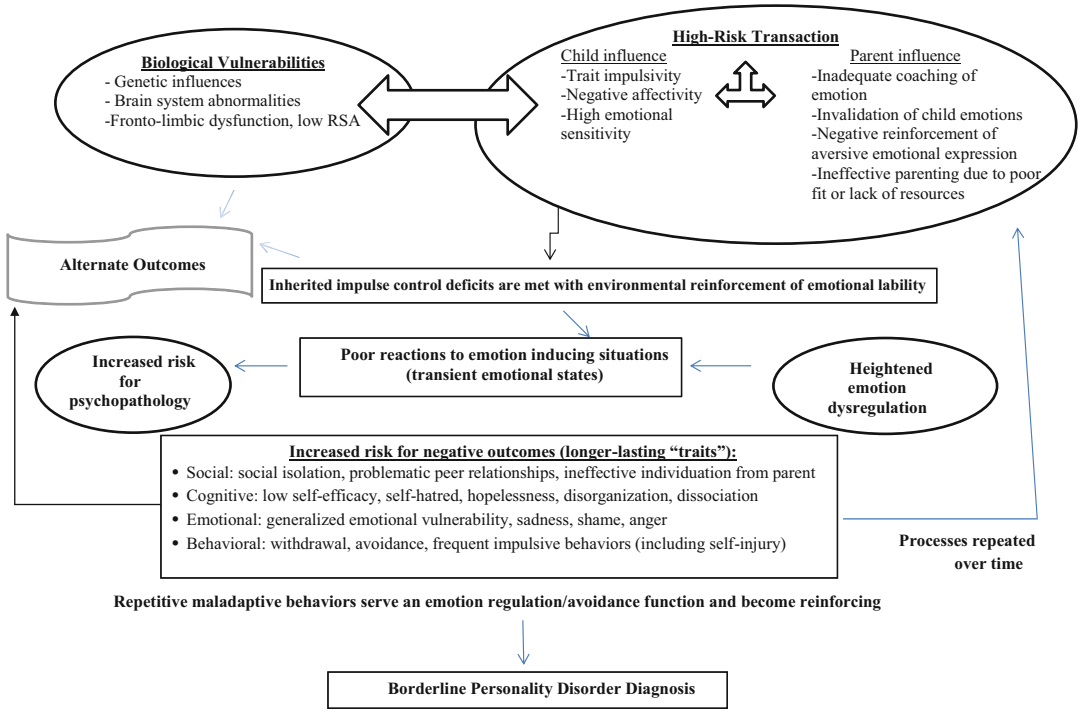

Fig. 11.1 A biosocial developmental model of borderline pathology

(Crowell, Kaufman, & Lenzenweger, 2013). Both conditions are also costly and extremely debilitating mental health problems. For our purposes, we define SII as any act of self-injury that is done with the intent of causing bodily harm or death, including non-suicidal self-injury, suicide attempts, and suicide (see Nock et al., 2008 for an expanded discussion of SII).

Theoretical Perspective

Literature reviewed below follows from our biosocial developmental model of borderline personality development (Crowell et al., 2009) in which we propose that:

1. Trait impulsivity—a highly heritable biological vulnerability—confers risk for BPD and other disorders of behavioral dyscontrol.
2. Emotional lability is shaped and maintained within high-risk familial contexts, which are characterized by intermittent reinforcement of aversive behavior and chronic invalidation of emotional expression.
3. Heritable impulsivity interacts across development with additional environmental risk factors to potentiate more extreme behavior and emotion dysregulation, disrupt peer relationships, and affect academic performance.
4. By adolescence, these Biology × Environment interactions promote a constellation of identifiable problems and maladaptive coping strategies—including early borderline features and repetitive self-injury—which fall along a heterotypically continuous trajectory to BPD.
5. Adolescent borderline pathology may further exacerbate risk for BPD in adulthood by interfering with normative development, the ability to form appropriate interpersonal relationships, and development of adaptive coping strategies.

The following review is guided by our etiological theory (for other models see Fonagy, Target, & Gergely, 2000; Judd & McGlashan, 2003; Kernberg, 1967, 1975, 1976). Core features of our model are represented in Fig. 11.1.

Empirical Research

Relative to most disorders, there is still limited research on the childhood precursors of BPD (however, see Sharp & Romero, 2007 for a review of this emerging literature). Moreover, because early manifestations of BPD are often not consistent with adult criteria (see above), researchers must examine the broader developmental literature with attention to clinical problems that may fall along the heterotypically continuous BPD trajectory. Consistent with the above discussion outlining both impulsivity and mood lability components, this literature suggests that BPD involves both externalizing and internalizing features—although considerable heterogeneity exists within the diagnosis (Eaton et al., 2011; Lenzenweger, Clarkin, Yeomans, Kernberg, & Levy, 2008). For this reason, research on the development of comorbid psychopathology (e.g., conduct disorder and depression) may offer unique insight into developmental processes that give rise to BPD. Within this literature, there are well-characterized patterns describing the emergence of emotional lability among impulsive youth. This population is of particular interest to our theory of borderline personality development given their problems with both behavioral and emotional dyscontrol.

Hypothesis 1: Trait Impulsivity Confers Risk for Borderline Pathology

Impulsivity has been defined in various ways over time and across studies. We are interested in two interrelated forms of impulsivity, behavioral impulsivity and trait impulsivity. Behavioral impulsivity refers to actions that are emitted quickly and with limited forethought (e.g., Oas, 1985). In contrast, trait impulsivity can be conceptualized as a highly heritable biological vulnerability that gives rise to behavioral impulsivity, but more importantly, confers risk for diverse forms of psychopathology that fall along the externalizing spectrum (e.g., Beauchaine & Neuhaus, 2008; Krueger et al., 2002). Although behavioral impulsivity is often an expression of underlying trait vulnerability, those with impulsive personality traits may or may not engage in pathologically impulsive behaviors. This disconnect occurs for a variety of reasons, including high levels of trait anxiety, which serve to dampen propensities toward behavioral impulsivity (see, e.g., Beauchaine, 2001; Sauder, Beauchaine, Gatzke-Kopp, Shannon, & Aylward, 2012), emergence of normative increases in top-down control over behavior across development (see Beauchaine & McNulty, 2013), strong environmental contingencies, and/or selection into environments that require less behavioral control (see Zanarini et al., 2003). Although many people with BPD engage in impulsive behaviors (e.g., shoplifting, substance use, repetitive self-injury), trait impulsivity is more relevant to understanding the etiology of BPD.

That said, there are many etiological pathways to impulsivity (see Neuhaus & Beauchaine, 2013). Some well-documented influences include brain injuries (e.g., head trauma, hypoxia), teratogens exposure (e.g., alcohol, lead), trauma exposure (e.g., child abuse), and genetically conferred vulnerabilities, among other factors (Neuhaus & Beauchaine, 2013). Oftentimes, such etiological agents are not independent. For example, genetically vulnerable parents are more likely than controls to expose their children to teratogens both pre- and postnatally, and impulsive children are more likely to acquire head injuries (see Shannon Bowen & Gatzke-Kopp, 2013). Many of these risk factors, including child abuse and neglect, compromise neurodevelopment in prefrontal brain regions (Hanson et al., 2010), amplifying impulsivity further, and potentiating development of impulse control disorders including BPD (Lobbestael & Arntz, 2010).

According to our theory (Beauchaine et al., 2009; Crowell et al., 2009; Derbidge & Beauchaine, 2014), trait impulsivity— primarily heritable but perhaps acquired through other mechanisms—is a principal predisposing vulnerability to BPD. As noted above, trait impulsivity is highly heritable (~0.8), confers risk for all disorders along the externalizing

spectrum (see Beauchaine & McNulty, 2013; Krueger et al., 2002; Tackett, Daoud, De Bolle, & Burt, 2013), and arises from individual differences in both dopaminergic and serotonergic function (Beauchaine et al., 2009; Krueger et al., 2002). A thorough review of neurotransmitter function in BPD is beyond the scope of this chapter (see Crowell et al., 2013 for a review). Rather, in sections below we focus on recent longitudinal studies, many of which support the hypothesis that BPD is a disorder characterized first by trait impulsivity, with emotion dysregulation emerging later through complex interactions between impulsive individuals and high-risk environments. This trajectory is similar to that observed for antisocial personality development (Beauchaine & McNulty, 2013), which is highly comorbid with BPD, and is often diagnosed among males from the same families as females who are diagnosed with BPD (for a review see Beauchaine et al., 2009).

Several studies now indicate that borderline traits are associated with early life impulsivity. For example, Belsky et al. (2012) examined borderline personality features in a cohort of 12-year-old children followed since birth. Borderline personality-related characteristics (BPRCs) were (1) highly heritable; (2) more common among children who displayed early behavioral/emotional problems, poor cognitive function, and impulsivity; and (3) co-occurred with conduct disorder, depression, anxiety, and psychotic symptoms. Moreover, harsh treatment in the family and familial history of psychiatric illness interacted to predict later BPRCs, consistent with transactional models of borderline personality development.

Studies following children into adolescence yield similar results. Hinshaw et al. (2012), in a 10-year prospective follow-up study, found that girls with ADHD continued to evidence high rates of hyperactivity/impulsivity, comorbid psychopathology, and impairment. More importantly for the impulsivity hypothesis, however, girls with the combined-subtype showed higher rates of SII than controls and girls with the inattentive-subtype. These effects remained following control of several covariates including age, IQ, demographics, and comorbidities. Similarly, data from the Pittsburgh Girls Study revealed that ADHD and ODD scores measured in middle childhood predicted BPD symptoms at age 14 (Stepp et al., 2012). Moreover, growth in ADHD scores from ages 10–13, and growth in ODD scores from ages 8–10, predicted BPD symptoms better than depression or conduct disorder. These and other studies reveal that BPD likely falls along a heterotypically continuous externalizing trajectory (see also Bornovalova, Hicks, Iacono, & McGue, 2013).

Hypothesis 2: Emotion Dysregulation Is Shaped Within Invalidating Family Contexts

Even though impulsivity is highly heritable (see above), its specific expression is shaped considerably by environmental risk exposure (Beauchaine et al., 2010). Parental psychopathology, for example, may affect the developmental context, increasing risk for personality disorders (Wilson & Durbin, 2012). Child abuse and neglect are also reliable predictors of later BPD, especially among those with genetic vulnerabilities (Cox et al., 2012; Gratz, Latzman, Tull, Reynolds, & Lejuez, 2011; Soloff, Lynch, & Kelly, 2002; Widom, Czaja, & Paris, 2009; Zanarini, Laudate, Frankenburg, Reich, & Fitzmaurice, 2011). However, many adults with BPD report no history of abuse. This suggests that potentially more subtle contextual factors may also contribute to borderline personality development. Indeed, Linehan's (1993) highly influential theory suggests that BPD emerges when biologically vulnerable individuals are raised within invalidating family contexts (Beauchaine et al., 2009; Crowell et al., 2009). Child abuse falls at the utmost extreme of invalidation. However, emerging evidence suggests that less extreme forms of invalidation reinforce emotional liability and canalize emotion dysregulation over time (see Crowell et al., 2013), increasing risk for self-injury and borderline personality development

among those who are predisposed by virtue of heritable vulnerability (i.e., trait impulsivity).

There are three core components of the invalidating environment theory (Crowell et al., 2013). First, invalidating environments reject and/or dismiss children's emotional expressions, which are often intense and overwhelming. The child's emotional needs may exceed the family's capacity to provide support and validation. Second, invalidating environments intermittently reinforce extreme emotional expressions. For the child, such negative outbursts may function to elicit emotional support, or allow him/her to delay or avoid parental demands (i.e., escape conditioning). Third, over time, such parent–child interactions lead to more severe emotion dysregulation, defined as intense responding to emotion evocation and a slow return to emotional baseline (see e.g., Kuo & Linehan, 2009).

The first component of the invalidating environment theory is that children at risk for BPD experience chronic invalidation, which, importantly, may be elicited partially by their trait impulsivity (evocative *r*GE). It is well established that temperamental difficulty and impulsivity are stable characteristics that predict a range of adverse outcomes (Caspi, 2000) some of which emerge from evocative effects (Scaramella & Leve, 2004). To our knowledge, there are no prospective studies linking early temperamental difficulties/impulsivity to SII or other BPD traits. We know, however, that (1) temperamentally difficult and impulsive males who are also reared in coercive family contexts are at high risk for antisocial outcomes (Beauchaine et al., 2009, 2010), and (2) females with BPD often emerge from the same families as males with ASPD (see above).

Self-injuring adolescents differ from depressed teens on measures of externalizing psychopathology, borderline personality traits, and psychophysiological biomarkers of trait impulsivity (e.g., reduced electrodermal activity; Crowell et al., 2012). Self-injuring adolescents also exhibit lower peripheral serotonin levels—another biomarker of trait impulsivity—than typically developing adolescents (Crowell et al., 2005). Moreover, peripheral serotonin interacts with mother–daughter dyadic negativity and conflict to predict SII. Specifically, we found that adolescents with low serotonin levels scored higher on SII regardless of dyadic negativity, whereas those with higher serotonin showed a linear relation between negativity and lifetime SII (Crowell et al., 2008). Although these are cross-sectional findings, they provide support for the notion that self-injuring adolescents show biological vulnerabilities similar to those observed in other externalizing samples, and that Biology × Environment interactions account for more variance in SII than either variable alone.

The second component of the invalidating environment theory is that parents and other caregivers intermittently reinforce extreme emotional expressions. One method of evaluating such interaction patterns follows from coercion theory, which characterizes developmental trajectories leading to aggression and delinquency (Patterson, DeBaryshe, & Ramsey, 1989; Snyder, Schrepferman, & St. Peter, 1997). Coercion theory describes the development of emotion dysregulation in a manner that is similar to Linehan's (1993) invalidating environment theory. Coercive processes are defined as patterns of interaction in which aversive tactics increase over time due to their effectiveness at reducing similarly aversive behaviors of the interaction partner (again, escape conditioning). In other words, the most successful way to end a disagreement is to escalate conflict. Unfortunately, these negative operant reinforcement processes ultimately result in more extreme emotional behaviors from both children and their parents (Patterson, Reid, & Dishion, 1998). The intermittent success of conflict escalation makes it difficult for families to alter these patterns once they become habitual.

Recently, we demonstrated that self-injuring adolescents and their mothers differ from control dyads in their moment-to-moment conversational tactics (Crowell et al., 2013). Using microanalytic scoring of conflict, we found that adolescents and mothers in SII dyads were more aversive with one another, and more likely to match and escalate conflict than controls.

Control mothers matched their adolescent's verbal behavior only at the lowest level of aversiveness, and de-escalated conflict at higher levels of aversiveness. In contrast, the only significant de-escalations by mothers in SII dyads occurred in response to extreme adolescent behaviors. This supports the hypothesis that dysregulated teen behaviors function to reduce conflict and, consequently, are inadvertently reinforced by the parent.

Finally, emotion dysregulation is predicted to increase over time, taking on trait-like qualities that are reflected in physiological functioning. Following from this supposition, we have tested whether parent and child aversiveness interact to predict resting respiratory sinus arrhythmia (RSA)—a peripheral biomarker of emotion regulatory capacity (see Beauchaine, 2001; Beauchaine, Gatzke-Kopp, & Mead, 2007), among adolescents who self-injure (Crowell et al., 2013). Consistent with the invalidating environment theory, we found that low-aversive teens with highly aversive mothers had the highest RSA, indicating strong self-regulatory capacity, whereas teens in high-high dyads showed the lowest RSA, indicating poor self-regulatory capacity. Taken together, these findings indicate that conflict escalation shapes emotional lability and emotion dysregulation, promoting both aggression and self-injury, and increasing risk for development of later BPD and ASPD in adolescence and adulthood (Beauchaine et al., 2009).

Hypothesis 3: Biology–Environment Interactions Shape Early Borderline Traits

Cross-sectional research does not allow us to determine whether contextual risk factors precede, follow, or correlate with borderline personality traits. However, most scholars agree that Biology × Environment interactions shape borderline traits across development, and that the etiology of BPD is complex and nonlinear (see Belsky et al., 2012 for a review). Nowhere is this complexity more apparent than in the growing literature on Gene × Environment interactions (G × E). At present, small sample sizes, inconsistent or unreplicated findings, poor measurement of environment, and failure to consider *r*GE correlations plague psychiatric genetics (see Beauchaine & Gatzke-Kopp, 2013). Furthermore, research on G × E in BPD is limited. However, there is a growing literature examining genetic and environmental effects on BPD-relevant traits, such as impulsivity, emotion regulation, anger, and SII (see Carpenter, Tomko, Trull, & Boomsma, 2013 for a review).

In several studies, genes associated with trait impulsivity interact with environmental risk factors to predict BPD traits. For example, Wilson et al. (2012) reported that a polymorphism in the tryptophan hydroxylase gene (TPH1) interacted with childhood abuse to predict BPD and SII in adulthood. TPH is a rate-limiting enzyme involved in the biosynthesis of 5HT. Variations in the TPH1 gene may confer risk for a number of disorders associated with higher suicide risk (Saetre et al., 2010). Similarly, Brezo et al. (2010) followed over 1,200 individuals for 22 years and found that three variants of the 5HTR2A gene interacted with sexual and/or physical abuse histories to predict later suicidal behavior. Moreover, different genes interacted with stress to predict depression, suggesting that depression and suicidality may have distinct etiologies (note, however, that replication of such findings will be important).

Molecular genetics research on the serotonin transporter (5HTT) polymorphism has yielded conflicting though interesting results (Zalsman, 2010). Several studies indicate that individuals with the short allele (i.e., s/s or s/l genotypes) are more likely than l/l individuals to engage in suicidal behavior (Anguelova, Benkelfat, & Turecki, 2003) or to have committed suicide by violent means (Lin & Tsai, 2004). Although further work is needed, evidence also suggests that adolescents and adults with BPD have fewer platelet serotonin transporter binding sites, and that this variability is likely due to the less efficient polymorphisms (s/s, s/l) of the 5HTT gene (Greenberg et al., 1999; Hankin et al., 2011).

Unfortunately, much of the research on G × E in BPD has neglected to assess *r*GE

correlations. However, recent work has begun to examine *r*GE correlations in more detail. For example, one study examined the causal association between abuse in childhood and BPD at age 24 (Bornovalova et al., 2013). As expected, they found that abuse, BPD traits, and internalizing and externalizing symptoms were correlated. However, there was no evidence of a causal effect of childhood abuse on BPD traits. Rather, the association between abuse and BPD stemmed from common genetic influences that are better accounted for by heritable vulnerability to internalizing and externalizing psychopathology. These findings highlight the need for further research on gene–environment relations. Clearly, biological vulnerabilities, environmental risk factors, acute and chronic stressors, and ongoing parent–child dynamics are not independent but correlate and interact across time to shape development (Fruzzetti, Shenk, & Hoffman, 2005).

Hypothesis 4: Self-Injury Is a Precursor to Adult BPD

We hypothesize that by adolescence, identifiable precursors to BPD begin to emerge for many—if not most—people who are on a BPD trajectory. As can be gleaned in part from above, these include risky and impulsive behaviors, emotional lability, a poor sense of self or lack of identity, interpersonal turbulence, extreme anger, feelings of emptiness, and SII. For many reasons, this constellation of emotions and behaviors has not been labeled reliably as borderline pathology. Strong negative emotions, risky behaviors, and identity struggles all show normative increases during adolescence (Arnett, 1999). Thus, many practitioners are understandably reluctant to label these problems as pathological (Miller, Rathus, & Linehan, 2007). Furthermore, many borderline symptoms overlap with Axis I disorders, especially conduct disorder and depression. When coupled with the *DSM* proscription against early diagnosis of PDs, it is not surprising that most cases of early BPD are missed.

Contrary to the assumptions listed above, a growing body of empirical evidence indicates that BPD features—and many other PD features—are not normative in adolescence and can be differentiated from other clinical disorders (see Beauchaine et al., 2009). For example, we recently examined whether self-injuring adolescents differ from depressed adolescents and healthy controls on BPD traits (Crowell et al., 2012). We chose to examine risk for BPD in this sample because SII overlaps with borderline pathology on several key dimensions. As we have articulated elsewhere (see Crowell et al., 2009), SII and BPD are both characterized by extreme emotion dysregulation. In fact, up to 90 % of those with BPD meet this diagnostic criterion (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2004), and a primary function of self-injury is to regulate overwhelming negative affect (Crowell et al., 2005). SII is also one of nine criteria used to diagnose BPD, and approximately 70 % of those with the diagnosis report a history of suicidal or non-suicidal SII (Gerson & Stanley, 2002; Paris, 2002); of those, approximately two thirds initiated SII prior to age 18 (Zanarini et al., 2006). For these and other reasons articulated herein, we believe that self-injuring adolescents are at elevated risk for later BPD.

To assess this possibility further, we assessed differences between SII participants and controls on continuous measures of BPD criteria. In such comparisons, adolescents who self-injure differ from typical controls on every feature of BPD (e.g., Crowell et al., 2012). Compared with the depressed group, self-injuring adolescents were also more likely to report self-damaging impulsivity and frantic efforts to avoid abandonment. There were no differences between the depressed and SII participants on self-reported anger, affective instability, identity disturbance, or dissociation. However, only 7 % of the depressed participants met the full criteria for BPD whereas 37 % of the SII group met the diagnostic threshold.

Longitudinal studies find that (1) BPD can be assessed reliably during adolescence, (2) SII is a common precursor to adult BPD, and (3) many

adolescents with BPD continue to meet diagnostic criteria in adulthood (Biskin, Paris, Renaud, Raz, & Zelkowitz, 2011). At present, however, it is impossible to determine whether (1) SII and BPD are correlated but best conceptualized as distinct conditions, (2) SII plays a causal role in the development of BPD, or (3) BPD and SII derive from a common etiology and result from similar vulnerabilities and risk factors. Future research should disentangle these possible associations between BPD and SII in order to determine the optimal timing and content of intervention strategies.

Hypothesis 5: Adolescent BPD Increases Risk for Adult BPD

We theorize that SII and BPD share a common etiology *and* that SII increases risk for adult BPD directly, via evocative effects (see Crowell et al., 2009; Hughes et al., 2012). There are many potential mechanisms through which SII could influence an adolescent's developmental context: hospitalizations could disrupt normative adolescent experiences (e.g., high school graduation) or family and friends may become rigid, controlling, or frightened. Similar to delinquent males, self-injuring adolescents may also be more likely to select deviant peer groups (active *r*GE; see Beauchaine et al., 2009; Hankin et al., 2011). Regardless of the exact mechanisms, it is important for intervention to start early and target SII and BPD features specifically (Miller, Rathus, Linehan, Wetzler, & Leigh, 1997).

Most self-injuring adolescents receive treatment for depression. This is not surprising given that attempted suicide is a diagnostic criterion for major depressive disorder (MDD), and given that BPD and MDD are highly comorbid. However, the common view that SII is a symptom of depression may be increasing heterogeneity in clinical trials and decreasing treatment efficacy. For example, 58 of 163 adolescents enrolled in the Adolescent Depression Antidepressants and Psychotherapy Trial (ADAPT) had a recent history of SII (Wilkinson, Kelvin, Roberts, Dubicka, & Goodyer, 2011). Most of these adolescents responded poorly to the intervention, leading the authors to conclude that there may be "a subtype of depression characterized by self-injury that leads to a poor response to treatment" (p. 499). We suspect that depression with and without self-injury have different etiologies and require distinct intervention strategies (Crowell et al., 2012).

Implications and Future Directions

In the time since we proposed our developmental model (Beauchaine et al., 2009; Crowell et al., 2009), several studies have provided evidence that is consistent with our initial hypotheses. For example, many individuals with BPD appear to follow a developmental trajectory similar to other externalizing disorders. We have also found that SII overlaps with other externalizing disorders on the contextual mechanisms shaping emotional lability. Indeed, negative reinforcement of conflict escalation appears to potentiate risk for emotion dysregulation, later SII, and borderline personality development in much the same way as it potentiates risk for emotional lability and aggression among those with conduct problems. There is also emerging evidence that G × E interactions exacerbate these problems across development. Future research should follow adolescents with SII and/or BPD into adulthood. This could enhance our understanding of key risk and protective factors for the development of BPD during this critical life stage.

It is also important for researchers and clinicians to view BPD as a cluster of emotions and behaviors rather than as a reified diagnostic entity. Future longitudinal studies should begin assessing BPD-relevant behavior problems early in development, especially extreme mood lability, identity problems, SII, and self-damaging impulsivity. Researchers should also assess parent–child interaction patterns, given findings that conflict escalation is one mechanism leading to emotion dysregulation among self-injuring adolescents (Crowell et al., 2013). Clinically, preventative interventions could begin earlier in development. There are well-established

interventions for ADHD that target self-control and parent–child dynamics (e.g., Webster-Stratton & Reid, 2003). There are also several promising interventions for depression that could be enhanced or adapted to target BPD features (e.g., Stice, Shaw, Bohon, Marti, & Rohde, 2009). Finally, dialectical behavior therapy has been adapted for use with self-injuring adolescents (Miller et al., 2007). This intervention is promising because it targets adolescent BPD features and family dysfunction directly.

The past decade has witnessed an important shift in the study of early borderline pathology. Until recently, the field was polarized—either BPD exists in children and adolescents or it does not. There are now several theories of borderline personality development that neither require nor assume that children and adolescents will meet the adult diagnostic criteria (Tackett, Balsis, Oltmanns, & Krueger, 2009). It is also understood that some adolescents who engage in self-injury and/or meet criteria for BPD will not maintain the diagnosis by adulthood (Biskin et al., 2011). This could occur for many reasons (e.g., effective intervention, positive peer influences) but does not imply that a BPD diagnosis was inappropriate. Although a few adolescents will desist naturally from a borderline trajectory, early identification and prevention hold promise for reducing the burden of BPD.

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Development of Emotional Cascades in Borderline Personality Disorder

12

Edward A. Selby, Amy Kranzler, and Emily Panza

Introduction

Historically, problems with emotional experience (Trull et al., 2008) and dysregulated behaviors (Glenn & Klonsky, 2009) have been examined individually in those struggling with BPD, but little research has explored whether these two major facets of BPD are connected. A recent theory, the *Emotional Cascade Model* (Selby & Joiner, 2009), builds off of previous theories of BPD (Linehan, 1993) and aims to better understand how emotional reactivity is connected to the dysregulated behavioral experiences of those with BPD. The purpose of this chapter is to provide a brief overview of the developmental factors that may contribute to the progression of interconnected problems with emotional reactivity and dysregulation over the course of childhood and adolescence.

Definitions and Core Constructs

Rumination: Rumination refers to repetitive thinking about an upsetting situation or an emotional experience, and about how the present situation relates to past problems or potential future problems (Nolen-Hoeksema, 1991).

Emotional Cascade: An emotional cascade occurs when an individual experiences an upsetting event, and responds to that event with intense, broadly focused, and rapid rumination. This rumination results in a progressively increasing, reciprocal cycle of negative emotion and rumination that ultimately creates a highly aversive cognitive-emotional state, elevating motivation to reduce the aversive experience (Selby & Joiner, 2009).

Dysregulated Behavior: A dysregulated, or disihibited, behavior is engaged in by an individual that results in harmful consequences to that individual. Common behaviors exhibited by those with BPD include self-injury, bulimic behaviors, substance use, social behaviors, aggressive behaviors, and suicidal behavior (Selby, Anestis, & Joiner, 2008).

Invalidation: Invalidation is the perception and actual experience of pervasive criticizing or trivializing of one's communication of internal experiences by others, as well as repeatedly being punished for appropriate emotional expression (Linehan, 1993).

Emotional Cascade Model and the Emergence of Borderline Personality Disorder

The Emotional Cascade Model (Selby & Joiner, 2009) proposes that people with BPD may experience frequent and intense elevations in

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negative emotion, which may lead to subsequent behavioral dysregulation, as a result of emotional cascades. An emotional cascade may begin when an individual experiences an upsetting event, which leads him or her to ruminate on the upsetting event. However, as he or she ruminates, elevations in negative emotion result, which then reciprocally lead to increased rumination, with negative emotion and rumination reciprocally influencing each other (Moberly & Watkins, 2008). The result of this process may be a self-amplifying positive feedback loop of intense rumination and negative emotion that causes a progressively more intense and aversive emotional state, termed an "emotional cascade" by Selby and Joiner (2009), which is painful and difficult to tolerate.

In this model, it is during emotional cascades that dysregulated behaviors are more likely to occur. These behaviors are used to provide distraction from the rumination component of an emotional cascade, by providing intense physical sensations to focus on instead of ruminating on the problem. Potential examples of physical sensations arising from these behaviors include feelings of pain or the sight of blood in non-suicidal self-injury (NSSI) (Nock et al., 2006), physical sensations induced by substance use, or the taste of food or feeling of fullness in binge-eating (Mitchell et al., 1999). Due to the salience of these strong physical sensations, these behaviors may short-circuit the emotional cascade by decreasing rumination, resulting in subsequent decreases in negative emotion and increases in feelings of relief (Hilt, Cha, & Nolen-Hoeksema, 2008). Importantly, once an emotional cascade is in a full self-generating cycle, it may be difficult to stop the cascade and less potent distractions may not pull attention away from the problem enough to bring emotional relief. This may explain why other seemingly more adaptive behaviors (e.g., reading, going for a walk) are not used for distraction.

Although emotional cascades may be implicated in multiple disorders, BPD may be the prototypical psychological disorder characterized by emotional cascades. Selby and Joiner (2009) posit that BPD is a disorder in which the core psychopathology results from emotional cascades, which occur more frequently and perhaps more intensely than in other disorders exhibiting behavioral dysregulation[1] (e.g., substance use disorders, eating disorders). In this sense, the extreme behavioral dysregulation associated with BPD may be viewed as extreme on the continuum of emotional cascades, whereas other disorders may be closer toward the center of this spectrum.

Emergence of BPD

The emotional cascade model provides important insight into the development of BPD, positing that the experience of multiple emotional cascades contributes to the emergence and progression of BPD over time. By integrating a time/developmental factor into their model, Selby and Joiner (2009) note that many factors may contribute to the eventual development of emotional cascades. Furthermore, the experience of emotional cascades alters the developmental trajectory so that BPD arises or "emerges" from the complex interactions between multiple factors: time, emotional cascades, and dysregulated behaviors. This emergence model of BPD is displayed in Fig. 12.1 and it demonstrates many important factors that research has identified as relevant for BPD emergence that may contribute to emotional cascades, which are the heart of this interacting network. In this diagram, as factors such as cognitive distortions and perceived invalidation contribute to the development of emotional cascades, repeated experience with emotional cascades and dysregulated behaviors

[1] Of note, we use the term dysregulated behavior interchangeably with the concept of impulsive behavioral experiences. However, it should be noted that there is not complete overlap between the concepts of dysregulated behaviors and impulsive behavior. Impulsive behavior can be more broadly conceptualized as engaging in harmful behaviors, failure to plan ahead, acting without thinking, and lack of perseverance (Whiteside et al., 2005). We conceptualize dysregulated behaviors a specific facet of impulsive behavior involving overt behaviors that ultimately result in harm to the individual (e.g., self-injury, substance use).

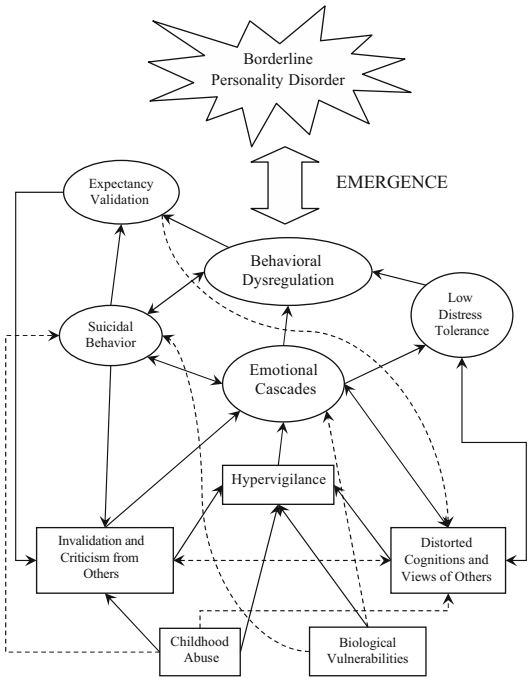

Fig. 12.1 Emotional cascade model and emergence of BPD. Note: Reprinted from Selby and Joiner (2009)

may subsequently increase these original factors, such that new cognitive distortions develop and they receive increased invalidation from others (e.g., others saying, "Why do you act so crazy?"). Over time, additional problems may arise in response to emotional cascades, such as increases in invalidation, low distress tolerance, and suicidal behavior. It is the synthesis of these various interactions between constructs that may result in the "emergence" of BPD. Similar to an economy, which is comprised of many simultaneously interacting individuals, corporations, and governments all trading, buying and selling, BPD may be comprised of many biological, cognitive, social, and environmental factors that interact with each other through emotional cascades. For example, an individual with a biological predisposition to BPD and a highly critical family may have distorted cognitions, and these biological, social, and cognitive factors feed into intense rumination and emotional cascades, which lead to dysregulated behavior that maintains and worsens the disorder. However, an important extension of the emergence viewpoint of this model (as illustrated in Fig. 12.1) is that the presence of BPD also feeds back into the underlying network that contributes to it, maintaining the disorder by worsening the factors that contribute to it.

Support for the Emotional Cascade Model

To date there are a number of studies that have linked rumination to BPD above and beyond what is attributable to comorbid depressive symptoms (Abela, Payne, & Moussaly, 2003; Baer & Sauer, 2011; Selby, Anestis, Bender, & Joiner, 2009), and evidence has been found to support the association between emotional cascades and dysregulated behaviors such as alcohol use, binge-eating and purging, NSSI, suicide attempts, seeking reassurance excessively, and nightmares (Selby, Anestis, & Joiner, 2008; Selby et al., 2009; Selby, Connell, & Joiner, 2010; Selby, Ribeiro, & Joiner, 2013). The majority of this research has been conducted using undergraduate samples. The experience of emotional cascades in BPD has been further examined using an experimental rumination induction (Selby et al., 2009), where undergraduate participants were instructed to ruminate for 5 min about an upsetting event in their life. Results indicated that those with BPD diagnoses demonstrated greater reactivity and intensity of negative emotion following the rumination induction than control participants. Again, these findings held after controlling for depressive symptoms.

In a more recent study, Selby and Joiner (2013) examined emotional cascades in BPD using experience sampling methodology, where participants are assessed multiple times daily over a number of days using personal digital assistants (PDAs). Participants exhibiting dysregulated behaviors, including many with BPD, were followed over a period of 2 weeks, during which they completed frequent assessments of negative emotion, rumination, and dysregulated behaviors. Using these data, Selby and Joiner (2013) demonstrated that there was an interaction between momentary rumination and level of negative emotion, a

relationship that prospectively predicted an increased probability of a participant engaging in a dysregulated behavior such as binge-eating over the next 2–3 h. Furthermore, there is also evidence that emotional cascades may be characterized by instability of rumination and negative emotion, where rapid fluctuations in these levels can occur throughout the day, in contrast to average levels of rumination and negative emotion (Selby, Franklin, Carson-Wong, & Rizvi, 2013). Thus, both absolute levels and instability of both rumination and negative emotion may independently predict dysregulated behaviors such as self-injury. Overall, these and other findings suggest that emotional cascades may be distinct from rumination alone, and are consistently associated with BPD psychopathology. However, given that the Emotional Cascade Model is relatively new, additional research is needed to further support the potential role of emotional cascades in the development of BPD.

Developmental Perspectives and the Emotional Cascade Model

Although the symptoms of BPD may arise when emotional cascades interact with a number of environmental factors, there are likely a number of specific developmental factors that occur during childhood and adolescence, which may contribute to the eventual development of emotional cascades and BPD. It is also likely that biological factors simultaneously influence the development of rumination and tendency toward dysregulated behavior, and through childhood and adolescence many cognitive and social factors may further enhance these biological factors, increasing risk for the development of emotional cascades and dysregulated behaviors. For clarity of presentation and discussion, we will discuss these factors as they individually contribute to the potential development of (1) emotional cascades and (2) dysregulated behaviors. Within each section we have grouped these different factors into biological factors, cognitive factors, and social factors.

Development of Emotional Cascades

Biological Factors

The Emotional Cascade Model considers biological factors as important underlying contributors to the development of emotional cascades and BPD, primarily by increasing one's tendency to ruminate and to experience negative emotion. There is preliminary evidence to suggest that biological factors contribute to both of these tendencies, although the direct mechanisms linking the two are not yet clear. Starting at the most basic level, a few genetic factors may be important contributors. For example, brain-derived neurotrophic factor (BDNF), a protein that is regulated by the presence of the BDNF gene, may have a role in the development of rumination. The BDNF gene has been linked to elevated rates of affective disorders and substance abuse, and genetic research has indicated that individuals with BDNF polymorphisms express significantly higher levels of rumination (Beevers, Wells, Ellis, & McGeary, 2009; Clasen, Wells, Knopik, McGeary, & Beevers, 2011; Liu et al., 2005). It is thought that problems with BDNF may contribute to poor executive control (Beevers et al., 2009), and poor executive control may contribute to the development of an inflexible and ruminative style for approaching problems (Davis & Nolen-Hoeksema, 2000). Similarly, polymorphisms in the serotonin transporter gene (5-HTTLPR) interact with life stress to predict elevated levels of rumination in healthy adults (Clasen et al., 2011). Importantly, both BDNF (Tadić et al., 2009) and serotonin transporter gene polymorphisms (Ni et al., 2006) have also been linked to BPD symptoms.

In addition to genetics, the development of emotional cascades in BPD may have other biological contributions. For example, a biologically grounded temperament toward negative emotion has been linked to rumination in adolescents (Verstraeten, Vasey, Raes, & Bijttebier, 2009). Similarly, adults high in

neuroticism have also been found to exhibit elevated rates of rumination (Muris, Roelofs, Rassin, Franken, & Mayer, 2005). From a developmental perspective, early emotions may play an organizational role in the development of personality, exerting a pervasive influence across domains of behavior (Malatesta & Wilson, 1988). Some children may be particularly predisposed to negative emotions, and over time these emotions may be consolidated into more stable patterns of thought and behavior (Malatesta & Wilson, 1988). Other factors may include biologically based difficulties with selective attention and memory for negative emotion (Baer, Peters, Eisenlohr-Moul, Geiger, & Sauer, 2012).

Another biological factor potentially involved in the development of emotional cascades in BPD may be changes in hormone levels during adolescence. One study demonstrated that fluctuations in estrogen are associated with severity of BPD symptoms, and that use of birth control can increase BPD symptoms (Desoto, Geary, Hoard, Sheldon, & Cooper, 2003). Adolescence is a time characterized by many changes, especially biological, and changes in hormone levels may directly contribute to ruminative tendencies or indirectly contribute by increasing stressors to ruminate about (Hilt & Nolen-Hoeksema, 2009). Changes in estrogen are relevant to changes in female body shape, and these changes (or dissatisfaction with these changes) may provide additional problems, such as self-objectification and body shame, that foster rumination and contribute to the high comorbidity between BPD and eating disorders (Grabe, Hyde, & Lindberg, 2007; Selby et al., 2012; Selby, Bulik, et al., 2010). Furthermore, there is evidence that rumination increases in girls during adolescence (Hyde, Mezulis, & Abramson, 2008). Although no studies have yet found an association between rumination and estrogen levels, rumination has been linked to other stress hormones such as cortisol (Zoccola & Dickerson, 2012), suggesting that hormones might be involved in eventual development of emotional cascades. Thus a number of genetic, temperamental, and hormonal factors may contribute to the eventual development of emotional cascades, and these factors are likely influenced by the development of cognitive features during childhood and adolescence. Investigation into the role of biological and cognitive factors in the development of emotional cascades is a critical area for future research.

Cognitive Factors

Childhood and adolescence are important periods in the development of cognitive approaches to problem solving and emotion regulation, and it is likely that maladaptive responses such as rumination are learned during these times (Silk, Steinberg, & Morris, 2003). The gradual development of rumination may then contribute to the later development of emotional cascades. The generation of both rumination and emotional reactivity is likely also impacted by a variety of cognitive factors, including developments in basic emotional understanding and the formation of adaptive and maladaptive cognitive strategies to cope with the increasing stressors of adolescence.

In order to learn adaptive emotion regulation (as opposed to maladaptive strategies such as rumination), youths must first develop a fundamental understanding of their emotions, including both emotional awareness and emotional clarity. Emotional awareness refers to the ability to notice, attend to, and differentiate emotional cues and experiences, and emotional clarity refers to the ability to correctly label and differentiate between distinct emotional states (Barrett, Gross, Christensen, & Benvenuto, 2001). This internal awareness requires both abstract and meta-cognitive abilities that develop during early adolescence (Gottman, Katz, & Hooven, 1997). When the development of this awareness is impaired, individuals exhibit fewer constructive coping strategies and higher levels of maladaptive strategies such as rumination (Gohm & Clore, 2000; Rieffe, Oosterveld, Miers, Terwogt, & Ly, 2008). As one might expect, youths that are less informed about their own emotional experiences are less capable of developing appropriate repertoires of coping responses, and instead engage in ineffective approaches such as rumination. It is therefore significant that by adulthood, many with BPD

have been found to express decreased emotional awareness and clarity (Leible & Snell, 2004; Levine, Marziali, & Hood, 1997).

Instead of recognizing their internal experiences, youth (as well as adults) with BPD often seek to avoid them, a maladaptive emotion regulation strategy that can contribute to rumination and emotional cascades. In fact, there is extensive evidence that adults and adolescents with BPD tend to avoid aversive emotions, rather than tolerate the experience of them (Schramm, Venta, & Sharp, 2013). One such method of avoidance is through thought suppression, which refers to deliberate attempts to push unpleasant or unwanted cognitions out of awareness (Wegner, Schneider, Carter, & White, 1987). It has been consistently demonstrated that thought suppression has a paradoxical rebound effect wherein the thoughts being suppressed return with greater frequency and intensity (Abramowitz, Tolin, & Street, 2001). In this way, thought suppression may be another factor that contributes to the development of emotional cascades, by elongating and exacerbating the presence of the negative thoughts that constitute rumination. Adolescents may be particularly vulnerable to thought suppression due to the sudden increase in unwanted thoughts that may arise in response to the increased stressors of adolescence (Larson & Richards, 1991; Steinberg, 1987). In addition, adolescents are just beginning to develop the ability to independently regulate their emotions (Steinberg et al., 2006), and these newly developed adaptive strategies may not yet be readily available for use in the face of increased stressors. In support of this notion, thought suppression is associated with increased self-harm in women with BPD (Chapman, Specht, & Cellucci, 2005), and has been found to mediate the relationship between emotional reactivity and self-injurious behavior in adolescents (Najmi, Wegner, & Nock, 2007). This link between thought suppression and dysregulated behavior in individuals with BPD suggests that emotional cascades may be influenced or worsened by thought suppression, and it further highlights the role of upsetting thoughts in promoting dysregulated behaviors in those with BPD.

In addition to emotional awareness and suppression, other cognitive factors in development may contribute to emotional cascades by further triggering excessive rumination. In particular, adolescence is a time characterized by lack of stability and uncertainty about one's identity. For many, identity does not solidify until adulthood, and for most adolescents their identity is changing on a regular basis, as are their peer groups, general interests, and methods of coping (Westen, Betan, & Defife, 2011). These fluctuations are consistent with findings that BPD symptoms themselves may also exhibit some instability in adolescence (Bernstein et al., 1993; Bondurant, Greenfield, & Tse, 2004). The pursuit of identity formulation and meaning during adolescence likely serves as an important process in the development of ruminative tendencies (Segerstrom, Stanton, Alden, & Shortridge, 2003) and may contribute to the finding that there is a general rise in rumination levels during adolescence (Hampecetermann, 2005). While identity formation is critical to healthy development, the explorative rumination that often occurs as part of the process is not critical, and has been linked to less adaptive identity development (Luyckx et al., 2008). In the process of identity formation and its accompanying explorative rumination, many adolescents may develop maladaptive self-views or cognitions revolving around frustrations with status among peers, body image concerns, and/or beliefs about competence, each of which may contribute to ruminative tendencies (Flynn, Kecmanovic, & Alloy, 2010). In this way, uncertainty over normal adolescent concerns as well as issues with fluctuating identity may contribute to the development of rumination and eventually emotional cascades.

Thus, during childhood and adolescence multiple factors may result in the development of a ruminative response to problems. Youth that are lacking emotional understanding and awareness may be less capable of developing effective coping strategies, rendering them more prone to maladaptive attempts to regulate emotion through methods such as thought suppression and rumination. The presence of increased

stressors and uncertain self-views may exacerbate these processes by placing further demand for regulation, and together these factors may all fuel the eventual development of emotional cascades. However, development of cognitive factors during childhood and adolescence does not occur in isolation, but is deeply connected to the child's social environment.

Social and Interpersonal Factors

According to the Emotional Cascade Model, interpersonal factors play a major role in the initial development of emotional cascades and BPD in a manner consistent with other theories of BPD psychopathology (Linehan, 1993). Although rumination is often conceptualized as a cognitive factor, the experience of rumination (and by extension emotional cascades) occurs within an interpersonal environment that frequently includes many triggers for rumination. The child's family and peer environments each play a role in this process by serving both as contexts in which they learn emotion regulation strategies, as well as sources of potential conflict and stress that require the use of these strategies.

The ability to regulate emotion is significantly impacted by the family environment (Crowell, Beauchaine, & Linehan, 2009; Shipman & Zeman, 2001). Parents teach their young children much of the fundamentals of basic emotional coping skills through social modeling and explicit instruction. As such, when parents lack effective emotion regulation skills themselves, their children often exhibit the same difficulties (Goodman & Gotlib, 1999). Youth who grow up in such environments without effective emotional support and modeling from their parents often exhibit deficits in the basic building blocks of emotional regulation, such as emotional awareness, and coping skills simply because they were never taught them. Over time, these deficits in basic skills contribute to more significant deficits in adaptive regulation strategies, leaving these youth more prone to turn to maladaptive strategies such as rumination. In line with this notion, maternal encouragement of emotional expression has been found to mediate the relationship between female sex and rumination levels in youth, linking more maternal encouragement with greater levels of rumination (Cox, Mezulis, & Hyde, 2010).

In addition, conflict in the family environment may contribute to significant stressors in the lives of children and adolescents. This is particularly important given research suggesting that feelings of conflict with parents and reduced support from one's family during childhood was associated with BPD symptoms as an adult (Klonsky, Oltmanns, Turkheimer, & Fiedler, 2000). Also, problems in attachment to parents during early childhood are linked to later development of BPD, with an insecure attachment having the strongest relationship with later BPD (Agrawal, Gunderson, Holmes, & Lyons-Ruth, 2004). These difficulties in attachment may cause a child to feel neglected or angry with one or both parents on a chronic basis, and as a result he or she may have to learn to cope with upsetting experiences without any parental assistance. Research on children and adolescents has found that over-controlling parenting styles and family interaction styles characterized by negative-submissive expressivity serve as risk factors for later development of rumination, with worse rumination developing when both negative family style and negative affective temperament of child were present (Hilt, Armstrong, & Essex, 2012).

Parents may also be direct contributors to upsetting events in the child or adolescent's life through experiences such as invalidation. Invalidation, which is a major facet of Linehan's (1993) theory of BPD, is defined as the experience of pervasive criticizing or trivializing of the communication of one's internal experiences by others, as well as repeated punishment of appropriate emotional expression. Perceived invalidation during childhood has been found to predict increased romantic relationship problems during adulthood (Selby, Braithwaite, Joiner, & Fincham, 2008), demonstrating the potential impact of invalidation on later development. Furthermore, the experiences of negative emotional intensity and sensitivity have been linked to experiences such as family invalidation (Selby, Yen & Sprito, 2013). It is thought that

invalidation interferes with a child's development of appropriate ways to handle difficult problems and decreases later problem-solving skills (Bray, Barrowclough, & Lobban, 2007), potentially making them more likely to turn to maladaptive strategies such as rumination.

One of the earliest factors that may contribute to later development of emotional cascades may be the experience of childhood abuse, an extreme form of invalidation, which has been linked to later development of BPD (Ball & Links, 2009; Carlson, Egeland, & Sroufe, 2009). Abuse may result in major damage to perceptions of what should be trusted relationships, leading to lifelong scars that further interfere with the development of new and appropriate trusting relationships. Abuse during childhood may lead children and adolescents to begin wondering who can be trusted or may be seeking to harm them, and this may set the stage for the development of ruminative tendencies. This notion is supported by findings that youth who have been maltreated exhibit increased difficulty with emotion regulation and elevated rumination levels later in life (Conway, Mendelson, Giannopoulos, Csank, & Holm, 2004; Rieder, & Cicchetti, 1989). However, despite the many studies linking childhood abuse to development of BPD symptoms and the potential role of abuse in developing rumination, abuse is likely to be a contributing factor for BPD rather than a causal factor. For example, it was recently demonstrated that the effects of abuse on later development of BPD might be due to shared familial genetic effects for psychopathology as opposed to a direct causal influence of the abuse (Bornovalova et al., 2013; see also Chapter ##, this volume).

Although some of the most relevant social experiences involved in the development of emotional cascades involve the family environment, another equally important social influence may involve the peer environment. Selby et al. (2013) found that perceptions of peer invalidation and negative emotional intensity were moderately correlated on a weekly basis over 6 months, indicating the importance of peer environment on negative emotional reactivity. Other forms of peer invalidation, such as hostility or aggression, may also play a role, as being the victim of bullying has been associated with increased odds of a subsequent suicide attempt, particularly in girls (Klomeck et al., 2009).

Beyond peer invalidation, peer friendships may ironically also contribute to the development of ruminative tendencies. In many friendships, even healthy ones, there may be processes that facilitate individual rumination through the common process of co-rumination, where the communication style between peers or friends is characterized by extensive problem talk, including rehashing problems, speculating about problems, and focusing on negative emotion (Rose, 2002). Unfortunately, such discussion with peers may only be somewhat productive, as co-rumination may focus on the unfairness of a situation or the role others play in the problem, rather than focusing on active problem-solving. Thus co-rumination, like individual rumination, may further increase negative emotion, and research indicates that elevated co-rumination in children and adolescents is associated with elevated internalizing symptoms (Calmes & Roberts, 2008; Schwartz-Mette & Rose, 2012).

Other, less pernicious, social factors may be involved in the development of emotional cascades during youth. For example, some research suggests that rumination is facilitated by feminine gender roles (Simonson, Mezulis, & Davis, 2011), and given that many individuals with BPD are female, this may also contribute to the development of rumination in BPD. Thus, a number of family and peer environment factors, as well as some larger societal factors, may contribute to the development of rumination and eventual emotional cascades for some adolescents, particularly girls.

Development of Dysregulated Behaviors to Cope with Emotional Cascades

Although we have covered many factors that may contribute to the development of emotional cascades and eventually BPD, it is also important

to understand that overt behavior may reciprocally interact with internal emotional experiences. Some who may be on the path to developing emotional cascades may learn effective and adaptive behaviors to cope, and if so they may diverge from a course of developing BPD. However, others may have internal and social experiences that promote engagement in dysregulated behaviors in response to developing emotional cascades, which may strengthen the trajectory toward a BPD diagnosis.

Biological Factors

Biological factors have been well established in the development of impulsivity and dysregulated behaviors. Just as genetic factors have been implicated in the onset of rumination (Beevers et al., 2009; Clasen et al., 2011), they have also been implicated in contributing to behavioral dysregulation. For example, polymorphisms with the dopamine transporter gene have been linked to BPD symptoms (Joyce et al., 2006), and dopamine dysregulation in the brain has been linked to trait impulsivity (Buckholtz et al., 2010). Similar findings have been found for the role of serotonin in impulsive behavior (Nomura et al., 2006). Although it can be difficult to determine if these genetic factors contribute distinctly to behavioral dysregulation or to both emotional and behavioral dysregulation, they nonetheless appear to be involved in a way that is consistent with the emotional cascade model. If one has a biological vulnerability toward dysregulated behavior, then when experiencing emotional cascades such vulnerability may be activated and promote the use of nonadaptive behavior to cope.

Another critical factor in the development of dysregulated behaviors is the biologically influenced temperament trait of disinhibition, which has been defined as the inability or unwillingness to inhibit behavioral impulses (Iacono, Carlson, Taylor, Elkins, & McGue, 1999) and is highly related to the development of BPD (Clark, 2005). Presence of a disinhibited temperament as a child is highly associated with later development of externalizing behaviors (Shaw, Owens, Giovannelli, & Winslow, 2001). However, it is important to note that a disinhibited temperament could lead to impulsive behavior that is not generally within the context of BPD, such as antisocial behavior, so it is important for future research to distinguish how such a temperament can lead to phenotypically distinct behavioral outcomes. Thus, genetic and biological influences on temperament may set the stage for the later development of dysregulated behaviors in response to emotional cascades. During childhood and adolescence, those who have these biological vulnerabilities may also be more susceptible to the cognitive and social factors that facilitate the use of dysregulated behaviors.

Cognitive Factors

The impact of cognitive factors on the development of dysregulated behaviors develops throughout childhood and adolescence. At the most basic level, some youth may not possess knowledge of adaptive behavioral responses. Early in the process of development, children learn how to develop behavioral skills for coping with upsetting emotions, and with healthy parental involvement many children develop healthy coping skills, such as learning how to seek out interpersonal assistance with problems and how to maintain goal-directed behavior in the face of upsetting emotions. However, due to a variety of circumstances, such as lack of appropriate teaching or invalidation, those who go on to develop BPD may fail to learn appropriate or effective behavioral coping skills.

Other youth may have learned adaptive behavioral strategies, but lack self-efficacy in their ability to effectively execute them. In these cases, youths' beliefs about their ability to cope with difficult emotions and situations play an important role in their choice of behavioral responses. For example, when faced with difficult emotions, children that feel less confident in their ability to problem solving may instead seek comfort in impulsive and avoidant behaviors,

such as alcohol or substance use. In line with this notion, beliefs about a lack of access to effective emotion regulation strategies in adulthood have been found to be strongly associated with BPD symptoms, particularly impulsivity (Glenn & Klonsky, 2009). Some of these beliefs may also vary depending on the perceived controllability of the situation. One meta-analysis found that active coping efforts were helpful when youth were experiencing controllable stressors, but not when experiencing uncontrollable stressors. In fact, the study found that using active coping strategies in response to uncontrollable stressors led to more externalizing behavior (Clarke, 2006). As they develop, children who learn to focus on the controllable aspects of a situation, rather than on the uncontrollable parts, may therefore be better equipped to engage in adaptive behavioral coping.

Another maladaptive pattern of cognitions that has been associated with BPD, and may contribute to the development of emotional cascades, is low distress tolerance (Gratz, Rosethal, Tull, Lejuez, & Gunderson, 2006). Distress tolerance is comprised of beliefs that one can continue pursuing goal-directed activity despite the presence of emotional and physical feelings of distress or discomfort. Development of maladaptive beliefs about distress or discomfort (e.g., "I can't handle this!") may render an adolescent more prone to impulsively turn to dysregulated behaviors as quick and effective methods of avoidance when an emotional cascade occurs. This inability to tolerate the negative emotions that begin to arise for even a short time makes it more difficult to employ strategies such as support seeking, which may be slower but more adaptive in the long run. Importantly, Selby and Joiner (2009) posit that with the experience of multiple emotional cascades and dysregulated behaviors over time, low distress tolerance may be further reinforced as dysregulated behaviors are used sooner and more frequently to cope with emotional cascades.

Alternatively, the emotional experiences of those who go on to develop BPD may be so intense (possibly due to developing emotional cascades) that many traditional coping behaviors may not be helpful to them. In therapy, patients with BPD are frequently encouraged to engage in behaviors in such as taking a walk, talking to a friend, or taking a shower or bath in response to negative emotion (Linehan, 1993). However, it is possible that these behaviors do not cause sensations that are potent enough to effectively distract from emotional cascades. Along these lines, Selby, Connell, and Joiner (2010) have posited that one of the reasons for the development of self-injury may be because other behavioral methods (including some dysregulated behaviors) may not be potent enough to stop emotional cascades, so those who are less afraid of pain may be willing to engage in self-injury as a distraction from emotional cascades. Although further research is needed on the distraction qualities of healthy and dysregulated behaviors, the emotional experiences of those who go on to develop BPD may be so extreme that traditional coping methods taught in childhood may not be strong enough to help cope with those experiences.

Another cognitive dysfunction that may be associated with dysregulated behaviors is poor executive functioning (Baskin-Sommers et al., 2012). Deficits in cognitive control and working memory capacity may reduce a child's ability to inhibit responses to their intense emotional reactions and remain focused on less salient goal-directed and adaptive behaviors. Significantly, deficits in these aspects of executive functioning, such as attention and working memory, have been associated with BPD (Gvirts et al., 2012; Legris & van Reekum, 2006). In fact, executive functioning deficits in the planning domain are most significant in people with BPD (Ruocco, 2005). These deficits may truly make it more difficult for youth to employ adaptive coping responses, reinforcing their beliefs about their inability to cope adaptively. Over the course of development, the combination of emotional intensity, maladaptive cognitions about the ability to cope effectively and tolerate distress, as well as deficits in executive functioning, may compound and result in an increasingly rapid trajectory toward dysregulated behaviors and BPD.

Social Factors

As mentioned previously, biological and cognitive factors that promote the development of dysregulated behaviors in BPD do not occur in isolation, and they are highly impacted by the social environment of a child or adolescent. As with rumination, the child's family and peer environments play critical roles by serving as both contexts in which they learn adaptive and maladaptive behavioral responses, as well as sources of potential conflict and stress that trigger these responses.

In order to consider engaging in dysregulated behaviors, youth must first be exposed to these behaviors as possibilities. This often occurs through witnessing friends and family members and modeling their behaviors. As noted earlier, having a mother with BPD symptoms may contribute to the later cognitive and emotional difficulties of a child (Herr, Hammen, & Brennan, 2008), and along these same lines having a parent who exhibits dysregulated behaviors may contribute to later use of similar behaviors, potentially due to modeling effects for coping. Fathers may also have the potential to model dysregulated behaviors such as substance abuse and aggressive behavior in response to negative emotion.

Similarly, peer socialization during adolescence may also provide a source of information for youth regarding potential dysregulated behaviors. In a process termed peer contagion (Dishion & Tipsord, 2011), peers learn dysregulated behaviors from one another. Youth witness their friends engaging in dysregulated behaviors such as substance abuse, self-injury, and bingeing and later may experiment with these behaviors as forms of emotion regulation to cope with increasing negative emotion. Furthermore, peers with common interests often select each other as friends, even when the common interests are problematic such as with psychiatric symptoms (Gilbert & Meyer, 2004). This may result in adolescents with BPD symptoms forming friendships with each other and learning maladaptive behaviors from each other.

Youth may be further exposed to dysregulated behaviors through the media, which conveys strong messages to adolescents about how they should be behaving. For example, the media-violent behavior link is well established (Johnson, Cohen, Smailes, Kasen, & Brook, 2002), exposing youth to aggressive behaviors they may later model as a method of coping with upsetting emotions that arise. Media may promote involvement in many dysregulated behaviors, including reckless driving, binging and purging, substance use, or risky sexual behaviors. Research has found that one of the most significant social influences on NSSI involves the internet, where adolescents may search for websites about self-injury, which normalize and encourage such behavior (Whitlock, Powers, & Eckenrode, 2006). Similarly, websites promote other behaviors such as drug use and eating-disordered behaviors (Juarascio, Shoaib, & Timko, 2010). An adolescent who is struggling to identify coping strategies for their emerging emotional cascades may stumble upon these websites, and end up trying one of these dysregulated behaviors to cope the next time an emotional cascade occurs.

In addition, social conflict with both family and peers may play a particularly salient role in developing dysregulated behaviors, as conflict with others may be a major motivator for dysregulated behaviors. Many who go on to develop dysregulated behaviors and BPD may have poor social problem-solving skills, increasing their likelihood of experiencing social stressors. For example, adolescent self-injurers have also been found to exhibit deficits in social problem-solving abilities (Nock & Mendes, 2008). Deficits in the ability to solve social problems through traditional means may leave a feeling of a "behavioral void" for the individual. Without knowledge of effective means to cope with social conflicts, and in response to rising emotional cascades, he or she may use NSSI (or another dysregulated behavior) as a strategy that helps momentarily resolve upsetting emotions.

The social factors that contribute to dysregulated behaviors are numerous, and only a few have been discussed. This is an important area for future research, and understanding the social determinants of dysregulated behaviors

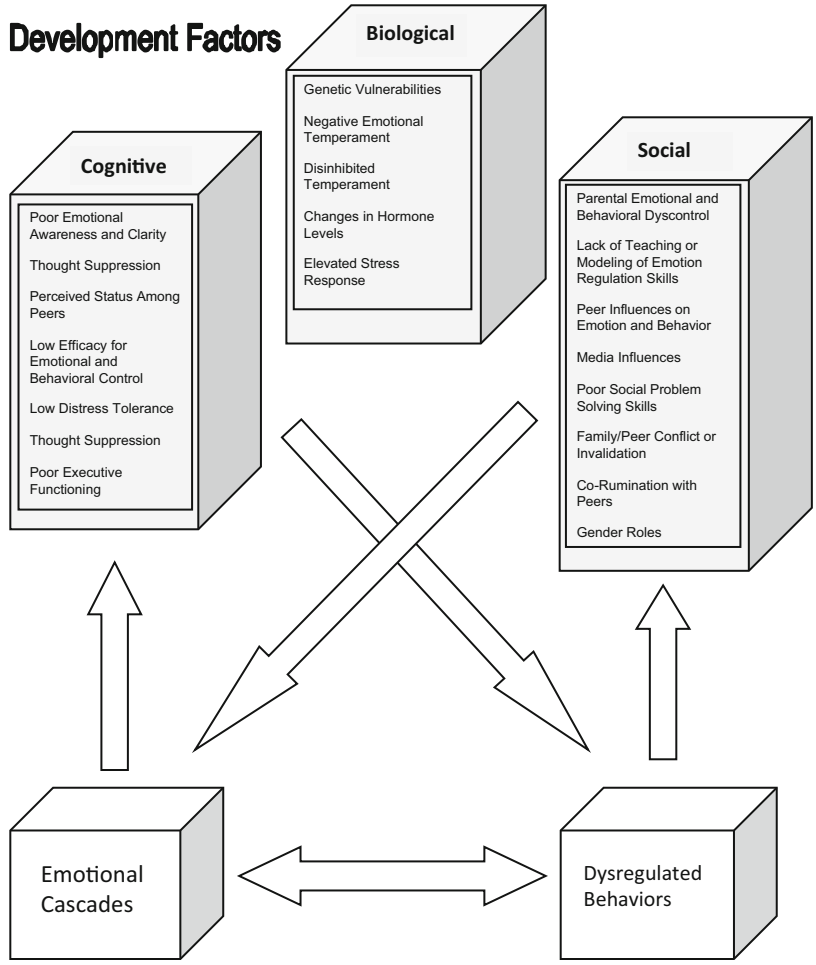

Fig. 12.2 Developmental contributions to the development of emotional cascades and dysregulated behaviors

may also help us understand why those with BPD may use certain behaviors to cope versus others. Furthermore, a better understanding of the social factors that contribute to the development of dysregulated behaviors in BPD may also provide insight in how to alter such behaviors.

A Developmental Emotional Cascade Model of BPD

Throughout this chapter a variety of biological, cognitive, and social factors that contribute to the development of both emotional cascades and dysregulated behaviors have been discussed. However, one of the strengths of the Emotional Cascade Model is that it views the simultaneous interaction between all of these factors to be critical in the development of BPD, connected through emotional cascades, rather than focusing on one factor as the primary cause of BPD. In this view, BPD may truly develop throughout childhood and adolescence, finally emerging when many of these problems have been firmly established.

Figure 12.2 shows the various factors discussed in this chapter that may contribute to the development of emotional cascades and dysregulated behaviors. In this model, a child who is born with inherent genetic and temperamental risk factors for BPD may be at elevated risk for developing emotional cascades and associated dysregulated behaviors. Over the

course of childhood and adolescence, these biological risk factors are likely impacted by the development of additional cognitive and social risk factors that promote a ruminative tendency, leading to the potential development of emotional cascades. Simultaneously, this child may also experience additional social and cognitive factors that make them more inclined toward behavioral dysregulation as a method of coping. As the child experiences emotional cascades, he or she may search for any effective behavior that produces sensations that are potent enough to help stop the emotional cascade. Through trial and error, dysregulated behaviors may emerge as the most effective (but least adaptive) immediate means of stopping emotional cascades.

However, throughout the course of development, the experience of emotional cascades and dysregulated behaviors may exacerbate the social and cognitive factors that originally contributed to them. Emotional cascades may result in increased parental and/or peer invalidation or the generation of maladaptive beliefs about the ability to regulate emotions, resulting in a developmental positive feedback loop increasing the occurrence of emotional cascades. Through the Emotional Cascade Model, the development of BPD is not seen as a static outcome, but rather a dynamic and fluctuating experience that emerges over time. We have delineated a number of biological, cognitive, and social factors that interact together in various ways during childhood and adolescence to promote the development of rumination and impulsive behavior, which may eventually develop into the experience of emotional cascades and dysregulated behaviors, followed ultimately by the emergence of BPD.

Future Research and Clinical Implications

Research on the Emotional Cascade Model has indicated that it may be useful in understanding the nature and development of BPD. However, it is important to note that the model is still relatively new and is in need of additional research to clarify and support it. One important direction for future research of the model includes further investigation of the emotional cascade process and its distinction it from rumination, which occurs in other disorders like depression. Additionally, more research is needed on the emotion regulation and distraction effects of dysregulated behaviors relative to other behaviors; at present minimal experimental research has examined if dysregulated behaviors actually result in decreased negative emotion and rumination. By understanding the reinforcing properties of dysregulated behaviors, we may be better able to understand why adolescents and adults with BPD do not use healthy or adaptive coping behaviors. More research is also needed to understand interpersonal dynamics in the role of emotional cascades, and to determine if different people (i.e. family, peers) are more prone to eliciting emotional cascades and why. One way to do this may involve the investigation of social cognitive functioning (often referred to as mentalization; Fonagy & Bateman, 2006) interacts with emotional processing deficits to predict the experience of emotional cascades. Such findings could highlight the potential role of social concerns in emotional cascades and help determine if emotional cascades arise primarily in response to problematic social interactions, or if they can arise across many situations. Another final direction for future research involves the examination of how longitudinal designs could investigate factors experienced during childhood and adolescence and their contribution to the development of emotional cascades. Such a study could involve repeated longitudinal assessment of rumination, emotional processing, and invalidation experiences and determine if progressively increasing difficulties in each of these areas over time interact to predict subsequent diagnosis of BPD. Such findings would support the notion that emotional cascades emerge in adolescence, and over time they contribute to the eventual development of a BPD diagnosis, as would be expected based on the emotional cascade model.

One potentially important clinical implication of the Emotional Cascade Model and the factors discussed in this chapter may be using this understanding to improve social-emotional education

for children, particularly those that may be at higher risk (Greenberg et al., 2003). An understanding of the cascading nature of these processes has important implications for the promotion of early prevention and intervention strategies that target the many contributing factors before they are consolidated into stable patterns of emotional and behavioral responses. Primary interventions may teach those who are at risk for later development of emotional cascades crucial skills for handling upsetting problems and emotional states adaptively. Early implementation of these skills may help to diminish emotional cascades before they start. Given the discussion throughout this chapter of the damaging effects that experiencing emotional cascades can have, preventing or reducing them may in turn prevent aggravation of the factors that contribute to them.

For adolescents who are already struggling with the experience of emotional cascades, the Emotional Cascade Model may also have important implications. By understanding the specific function of dysregulated behaviors in providing potent distractions, therapists are better suited to help clients find more effective replacements for these maladaptive strategies. Therapists should work in collaboration with their clients to identify adaptive coping strategies that could be effective in distracting them from rumination. If the therapist suggests a behavior that the patient feels would not help to divert attention away from upsetting thoughts and emotions, then alternative behaviors should be identified. Additionally, identifying adolescent-relevant social factors that contribute to emotional cascades, such as the use of social media, should be recognized as a contributing factor in the adolescent negative emotional experience.

Finally, from an emotional cascade standpoint, there may be additional and potentially more efficient treatment options that might supplement traditional therapy. Recent epidemiologic research suggests that only 7 % of adults with BPD visited a psychologist in the last year, while 36 % sought nontraditional treatments from online and community resources (Selby & McHugh, 2013)—suggesting that development of novel treatment methodologies may help improve treatment accessibility for those with BPD. Smartphone apps may provide one such treatment model, and they may be particularly good for helping patients identify adaptive ways of coping with emotional cascades, and they may be particularly appealing to adolescents. Integration of smartphones into treatment for daily assessment of emotional, cognitive, behavioral, and interpersonal experience may therefore be both helpful and well received. Such apps could help alert the patient when his or her rumination levels are high, and it could provide lists of alternative activities to engage in when ruminating (including other smartphone apps or games). By helping the patient identify and distract from rumination early in an emotional cascade process, he or she may be able to avert a potential dysregulated behavior, resulting in increased beliefs about one's ability to cope and manage emotion appropriately and a potentially more adaptive cascade of experiences.

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Emotion Dysregulation Among Adolescents with Borderline Personality Disorder

13

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Emotion dysregulation encompasses a broad range of difficulties related to emotional experience and modulation. The term emotion dysregulation has been applied to problems with the intensity, frequency, and duration of emotional responses, as well as difficulties modulating emotional experiences in effective and adaptive ways (Bloch, Moran, & Kring, 2010). Emotion dysregulation appears in many theoretical accounts of the pathogenesis and phenomenology of borderline personality disorder (BPD). It has been suggested that facets of emotion dysregulation are observable during childhood and adolescence, prior to the emergence of BPD, and that adolescents with BPD symptomatology experience greater emotion dysregulation than their peers (Crowell et al., 2009). Despite recent empirical efforts to characterize the relationship between emotion dysregulation and borderline symptomatology among adolescents, many questions remain unanswered about the role of emotion dysregulation in the development of BPD, as well as the nature and extent of emotion dysregulation among adolescents who have BPD. The goal of this chapter is to review current research that addresses the relationship between emotion dysregulation and BPD among adolescents and young adults between the ages of 10 and 24.

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Emotion Dysregulation: Definition and Core Constructs

Emotions are functional and adaptive. Negative affect signals that something is wrong, that a discrepancy exists between one's current situation and one's desired state of being, which motivates behavior to reduce the discrepancy (Carver & Scheier, 2010). Fear suggests the presence of threat and motivates escape behavior, while sadness signals loss and triggers attempts to replace the lost person or object, and anger signals that one has been harmed and motivates approach behavior aimed at fighting, harming, or defeating the source of injury. Emotional responses involve the subjective experience of emotion, behavioral tendencies, and physiological reactions (e.g., autonomic, neuroendocrine; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005).

The adaptive aspects of emotions are undeniable, but at the same time the frequent and intense experience of these emotions combined with an inability to cope with their occurrence can severely disrupt one's functioning. The term *emotion dysregulation* has been used to capture this profound impairment and encompasses two dimensions of emotional difficulties (Cicchetti, Ackerman, & Izard, 1995; see Table 13.1). The first dimension is *affective dysfunction*, which refers to problems with the frequency, intensity,

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Table 13.1 Facets of emotion dysregulation

Type 1: affective dysfunction	Type 2: emotion regulation problems
a. Emotional sensitivity/reactivity	a. Absence of adaptive, effective emotion regulation strategies
b. Emotional intensity	b. Inability to tolerate distress
c. Prolonged emotional responses	c. Poor control over problematic mood-dependent behavior

Table 13.2 Prototypical strategies to modify emotional experiences, adapted from Gross and Thompson (2007)

Situation selection	Approach/avoid certain people, places, or activities to regulate emotion
Situation modification	Act on the situation itself to modify its emotional impact
Attentional deployment	Choose what aspect of the situation to attend to
Cognitive change	Change the way one construes the meaning of a situation
Response modulation	Influence one's emotional response tendencies once they have been elicited

and duration of emotional experience and expression. The second is the related but distinct dimension we refer to here as *emotion regulation problems* which refers to difficulties tolerating and modulating emotional responses, as well as difficulties controlling mood-dependent behavior.

Before discussing each dimension, a few points of clarification are worth mentioning with regard to our terminology. In some conceptualizations and strategies for assessment (e.g., Gratz & Roemer, 2004), the overarching term emotion dysregulation does not include affective dysfunction and only consists of the self-regulatory aspects of the construct. To retain consistency with biosocial and developmental models of BPD (Bloch et al., 2010; Crowell, Beauchaine, & Linehan, 2009; Katz & Gottman, 1991; Linehan, 1993), we retain both dimensions in our definition of emotion regulation. Second, the term emotion dysregulation can at times be used to refer to the overarching construct as well as the self-regulatory aspect of the construct which can produce some confusion as to which is being referenced. To address this issue, we chose to retain the term emotion dysregulation for the overarching construct and create the term "emotion regulation problems" to characterize the self-regulatory dimension.

Affective Dysfunction. The dimension of affective dysfunction includes marked emotional reactions to apparently minor provocations, and they may be affected by events that don't seem to disturb others. These emotional reactions tend to be extreme (e.g., rage instead of anger, or panic instead of anxiety), frequent, and long-lasting. These intense and enduring emotional responses have enduring effects on subjective mood, arousal, thoughts, and behavior. Extreme emotion distorts information processing, leading to attention, memory, and attribution biases for mood-congruent information, which may further amplify negative emotions. In addition, extreme emotions trigger goal reprioritization: reducing the source of distress becomes the focal goal, interrupting effort toward longer-term and higher-priority goals, often leading to impulsive mood-dependent behavior.

Emotion Regulation Problems. The dimension of emotion regulation problems is characterized by lack of access to strategies to regulate emotional experiences, as well as loss of control over behavior when distressed. In general, emotion regulation involves the use of strategies (both automatic and effortful) to modulate the intensity of emotional experience and expression in the service of one's goals (Werner & Gross, 2010). Prototypical emotion regulation strategies appear in Table 13.2.

Emotion regulation efforts may target the subjective, physiological, or behavioral components of an emotional response (Mauss et al., 2005), and may vary as a function of the specific emotional experience. Successful emotion regulation involves the flexible application of strategies that are both effective and consistent with one's other goals given the context. This definition

acknowledges that a single strategy is unlikely to be effective for every emotion, and that the optimal strategy may vary depending on the resources that are available and the demands of the current situation. For example, seeking emotional support from a close friend or family member is widely regarded as an effective and adaptive way to reduce distress (Coan, Schaefer, & Davidson, 2006; Shaver & Mikulincer, 2007), yet *exclusive* reliance on this strategy may fray relationships and eventually could lead to estrangement and isolation (Coyne, 1976; Pettit & Joiner, 2006; Potthoff, Holahan, & Joiner, 1995). Further, use of interpersonal emotion regulation strategies may not be appropriate or effective if people in the immediate environment are unsupportive, unreliable, or dangerous (cite something about disorganized attachment).

Emotion regulation problems arise when one does not have access to effective strategies to reduce the intensity or duration of emotional response. In some cases, emotion regulation strategies simply have no impact, and in other cases, efforts to regulate emotions may actually backfire and result in amplification of negative emotional experiences (Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Selby & Joiner, 2009). Individuals who lack emotion regulation strategies may struggle to control mood-dependent behavior (e.g., temper outbursts, avoidance), or may turn to more extreme and self-damaging forms of behavior (e.g., substance use, overeating, non-suicidal self-injury) in an effort to reduce negative emotions (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Nock & Prinstein, 2005). Although these behaviors may be effective at reducing negative affect in the short term, they often conflict with other goals and have serious negative consequences.

Emotion Dysregulation Summary. To summarize, emotion dysregulation encompasses two dimensions of emotional difficulties: affective dysfunction and emotion regulation problems. Affective dysfunction refers to the characteristic frequency, intensity, and duration of emotional responses, whereas emotion regulation problems refer to problematic responses to emotions, including the use of ineffective or maladaptive emotion regulation strategies, or dyscontrol over mood-dependent behavior. Both dimensions of emotion dysregulation are characteristic of people with BPD and appear in theoretical accounts of the disorder.

Theoretical Perspectives on Emotion Dysregulation and the Development of BPD

Most models of BPD acknowledge emotion dysregulation, in some form, as a prominent clinical characteristic of this disorder (Gunderson, Kolb, & Austin, 1981; Kernberg, 1984). Indeed, three of the nine DSM-IV-TR diagnostic criteria for BPD directly address aspects of the construct affective dysfunction: instability of affect due to emotional reactivity of mood; intense and inappropriate anger, or difficulty controlling anger; and chronic feelings of emptiness (APA, 2000). However, psychoanalytic and psychodynamic perspectives consider emotional difficulties as secondary to other features of the disorder, such as disrupted attachment (Masterson & Rinsley, 1975; Paris, Zweig-Frank, & Guzder, 1993), impaired reflective capacities (Fonagy, Luyten, & Strathearn, 2011), pathological ego structure (Gunderson & Singer, 1975; Kernberg, 1984), or defensive reactions to prolonged psychological trauma (Herman & van der Kolk, 1987). In contrast, behavioral (biosocial) models propose that emotion dysregulation is the defining characteristic of BPD, and suggest that emotion dysregulation plays a key role in the development of the disorder (Linehan, 1993; Linehan & Koerner, 1993). Accordingly, much of the research on emotion dysregulation in BPD is informed by and interpreted within a behavioral framework, which is the basis of this chapter.

The biosocial model of BPD was initially proposed by Linehan (1993) and was later elaborated by Crowell et al. (2009; see also this volume). Within this framework, emotion

dysregulation emerges from the interaction of biologically based temperamental vulnerabilities to affective dysfunction and a caregiving environment that is characterized by maltreatment or emotional invalidation (e.g., punishing or minimizing the child's emotional experiences and expressions). According to the biosocial model, children at greatest risk for extreme emotion dysregulation (and eventually BPD) are biologically predisposed to experience heightened negative affectivity, emotional sensitivity, and emotional intensity, as well as elevated trait impulsivity.

These biological vulnerabilities are magnified through reciprocal transactions with invalidating, insensitive and inconsistent caregiving environments. In invalidating environments, caregivers treat the child's emotional experiences as unwarranted and intolerable, and respond inconsistently to emotional distress. In particular, caregivers may ignore or punish low-intensity expressions of emotion, but may be more responsive to extreme expressions, effectively teaching children that hyperbolic displays of emotion are the only way to elicit a nurturing response. Children with a temperamental predisposition to experience intense emotions, who are raised in invalidating environments, do not learn to identify, modulate, or tolerate their emotional responses, leading to vacillation between emotional inhibition and emotional liability (Crowell et al., 2009). Furthermore, because these children do not learn adaptive strategies to regulate or tolerate distress, they may adopt maladaptive emotion regulation strategies (e.g., emotional suppression) or use extreme forms of behavior to modulate negative affect (e.g., non-suicidal self-injury, substance use). These maladaptive forms of emotion regulation are maintained because they are intermittently effective in reducing negative affect, and they may result in a narrowed range of functional alternatives.

The interaction of affective dysfunction and emotion regulation problems leads to a pervasive pattern of dysfunction across social, cognitive, emotional, and behavioral domains. Over time, these maladaptive patterns are repeated and reinforced, leading to a diagnosis of BPD. Those at greatest risk for developing BPD are individuals who experience affective dysfunction *and* who have limited access to strategies to modulate the intensity and duration of emotional experiences and/or are unable to remain engaged goal-directed, value-consistent behavior when they are upset. With this framework in mind, the goal of the current chapter is to summarize what is known about emotion dysregulation among adolescents and young adults with BPD, considering relevant empirical work that addresses affective dysfunction and emotion regulation problems.

Existing Empirical Support for Emotion-Based Developmental Models

Based on the depth, reach, and influence of emotion-focused developmental theories of BPD, one would expect a large body of research directly testing the existing models. However, there is very little prospective, longitudinal research that would allow us to draw conclusions about the interactive and causal effects of affective dysfunction and emotion regulation problems on the development of BPD. However, there is a growing body of work that seeks to characterize the nature and extent of emotion dysregulation among adolescents with BPD. While the latter does not provide a test of the causal relations inherent in developmental models, it does provide an important starting point that can guide future work. Table 13.3 outlines the key information from the studies reviewed below.

Existing Empirical Support for Affective Dysfunction and Emotion Regulation Problems in Youth with BPD

Affective Dysfunction

Affective dysfunction refers to a constellation of temperamental vulnerabilities that confer risk for

Table 13.3 Summary of findings from studies of emotion dysregulation among adolescents with BPD symptomatology

References	BPD assessment	*N*	Female (%)	Mean age	Participants	Stimulus or stressor	Findings
Affective dysfunction							
Robin et al. (2012)	DSM-IV BPD	44	100	17	Clinical vs. healthy	Facial expressions of varying intensity	1. BPD required more intense facial expression to correctly identify anger, happiness
							2. No group differences in identification accuracy for 100 % expression
Jovev et al. (2011)	3+ DSM-IV BPD	41	85	19	Community vs. healthy	Facial expressions of varying intensity	No differences in intensity of facial expression to correctly identify emotion
Snowden et al. (2013)	BPQ	150	50	21	College students	Facial expressions at 40 % intensity	+ Correlation between borderline traits and heightened recognition of fearful faces in females
Jovev et al. (2012)	3+ DSM-IV BPD	41	85	19	Community vs. healthy	Pairs of emotional faces	Attentional bias for fearful faces
Tragesser et al. (2008)	PAI-BOR	121	69	19	College students	Teasing scenario	+ Correlation between borderline traits and sadness, anger, retaliatory and withdrawing behavior
Lawrence et al. (2011)	4+ DSM-IV BPD	52	90	19	Clinical vs. healthy	Exclusion (Cyberball)	1. BPD group rated emotions as more intense at all time points;
							2. No difference in intensity or duration of response to exclusion
Ruocco et al. (2010)	DSM-IV BPD	20	100	22	Community and clinical vs. healthy	Exclusion (confederates)	1. BPD group showed hyperactivation of the LMPFC, associated with perspective taking, following exclusion
							2. No differences in subjective emotional response to exclusion
Woodberry et al. (2008)	OMNI BDS	41	100	23	Community vs. healthy	Invalidating comment during frustrating task	1. BPD group reported less comfort with emotion at all points;
							2. No group differences in physiological or subjective arousal in response to invalidation
Weinberg et al. (2009)	MSI-BPD	40	73	20	Community vs. healthy (all students)	Trier Social Stress Test	1. BPD showed greater overall parasympathetic arousal, lower overall sympathetic arousal
							2. No group differences in physiological response to stress, but greater subjective frustration in BPD group
Gratz et al. (2009)	CPNI (parent report)	263	45	11	Community youth and their caregivers	–	1. Affective dysfunction predicted BPD for boys and girls
							2. Interaction of affective dysfunction and disinhibition predicted BPD features for girls only

(continued)

Table 13.3 (continued)

References	BPD assessment	*N*	Female (%)	Mean age	Participants	Stimulus or stressor	Findings
Ayduk et al. (2008)	PAI-BOR	379	65	21	College students	–	1. + Correlation of BPD features and rejection sensitivity
							2. Interaction of rejection sensitivity and executive control predicted BPD features
Emotion regulation problems							
Glenn and Klonsky (2009)	MSI-BPD and SIDP-IV	243 and 30	48 and 50	–	College students	–	1. + Correlation of BPD symptoms and various emotion regulation problems
							2. After controlling for affective dysfunction, + correlation of BPD symptoms with lack of emotion regulation strategies and impulse control problems
Sharp et al. (2012)	BPFSC; CI-BPD	190	59	15	Clinical	–	BPD symptomatology + correlated with overall emotion regulation problems
Cheavens et al. (2005)	IIP-A; IIP-S	202	61	19	College students	–	1. Negative affect intensity/reactivity, parental criticism and thought suppression related to + BPD symptomatology
							2. Thought suppression fully mediated effect for negative affect intensity/reactivity and parental criticism
Sauer and Baer (2009)	PAI-BOR	104	77	19	College students	–	1. Affective dysfunction, thought suppression associated with BPD severity
							2. Thought suppression-mediated relationship between affective dysfunction and BPD severity

BPD borderline personality disorder, *CPNI* Coolidge personality and neuropsychological inventory for children, *DSM-IV* diagnostic and statistical manual of mental disorders, 4th edition, text revision, *BPQ* borderline personality questionnaire, *PAI-BOR* personality assessment inventory—borderline features, *OMNI BDS* borderline disorder scale, *MDD* major depressive disorder, *MSI-BPD* McLean screening instrument for borderline personality disorder, *SIDP-IV* structured interview for DSM-IV personality, *BPFSC* borderline personality features scale for children, *CI-BPD* childhood interview for borderline personality disorder, *IIP-A* inventory of interpersonal problems—aggression, *IIP-S* inventory of interpersonal problems—interpersonal sensitivity

various forms of psychopathology, including BPD. Affective dysfunction encompasses emotional sensitivity or reactivity, emotional intensity, and prolonged emotional responses (Cole, Martin, & Dennis, 2004; Linehan, 1993; Mullin & Hinshaw, 2007). Affective dysfunction is most commonly assessed using self-report measures, which query trait-like tendencies, as well as state-dependent emotional responses to discrete events. Although self-report measures are well suited to assess subjective emotional experiences, they may fail to capture other dimensions (e.g., cognitive, behavioral, physiological) of emotional responding (Mauss et al., 2005). The need for comprehensive assessment is especially important for youth with BPD, given findings that suggest a discrepancy between subjective accounts and objective indicators of emotional responding among adults with BPD (Rosenthal et al., 2008). A number of novel experimental methods have been applied to answer critical questions about affective dysfunction among youth with BPD.

Do Adolescents with BPD Respond Differently to Social–Emotional Cues?

One facet of affective dysfunction is emotional reactivity which refers to the tendency to have emotional responses to low-intensity stimuli. Among youth with BPD, emotional reactivity may be revealed in heightened sensitivity to emotional cues, including attentional bias for threatening emotional stimuli, the ability to detect subtle interpersonal cues that others do not (Jovev et al., 2011, 2012; Snowden, Craig, & Gray, 2013). A number of studies have been conducted to examine whether youth with BPD exhibit greater reactivity to emotional cues.

Attentional bias refers to the tendency to disproportionately allocate attention to cues that are consistent with one's goals. As examples, a person who is extremely hungry may exhibit attentional bias for food-related cues, whereas a person who fears abandonment may exhibit attentional bias for signs of rejection. Jovev et al. (2012) conducted a study of attentional bias for emotional cues in a sample of adolescents. The BPD group consisted of 21 adolescents (18 female) between 15 and 24 years old (mean age of 19), and 20 healthy control participants (13 female) recruited from the community (mean age of 20). BPD was assessed using a symptom questionnaire, and participants were assigned to the BPD group if they endorsed three or more symptoms of BPD.

This study employed a modified dot probe task to examine whether subliminal exposure to emotional cues interfered with performance on a simple discrimination task. In the dot probe task, participants were presented with pairs of emotional faces (happy, angry, fearful, or neutral) that were either identical to one another (fearful–fearful, neutral–neutral, etc.) or different from one another (a neutral face paired a happy, angry, or fearful face). Each pair of faces was presented for 30 ms, below the threshold for conscious detection. After the offset of the faces, two dots appeared in the same location as one of the faces. The dots were oriented either vertically (:) or horizontally (..), and participants were asked to use a keyboard to indicate their orientation.

Results indicated that when the neutral and fearful faces were paired, the BPD group was faster to respond when the probe appeared where the fearful face had been, than when the probe appeared where the neutral face had been. Follow-up analyses revealed that the effect was due to slower responses when the probe occupied the position of the neutral face, rather than faster responses when the probe occupied the position of the fearful face, which the authors interpreted as evidence that participants in the BPD group had difficulty disengaging their attention from subliminal fear cues. Notably, within the BPD group, attentional bias for emotional stimuli was observed only for fear, and not for happy or angry expressions. No attentional bias for any type of emotion was observed in the control group. The results of this study support the presence of attentional bias for subliminal fear-related cues, which may be interpreted as evidence of heightened emotional reactivity in youth BPD.

Because BPD is characterized by reactivity to emotional cues, some researchers have

hypothesized that youth with BPD may be able to detect facial expressions at lower levels of expressiveness (Frank & Hoffman, 1986). With regard to accuracy of facial recognition, some have suggested that people with BPD may be more accurate in recognizing and labeling low-intensity emotional expressions, while others have suggested that they may be impaired in this domain because they tend to make negative attributions of neutral or blended emotional expressions (Domes et al., 2008; Meyer, Pilkonis, & Beevers, 2004; Wagner & Linehan, 1999). To date, a small number of studies have examined these domains of information processing.

Facial emotion morphing tasks have been used to evaluate facial emotion discrimination (the ability to detect facial emotions of varying degrees of expressiveness), as well as emotion recognition (the ability to recognize and label facial emotions), both of which may suggest greater emotional reactivity or sensitivity. Facial morphing studies use digitally altered images to create a series of pictures of varying degrees of facial expressiveness. To achieve this effect, a composite picture is created by morphing a picture of a neutral expression with a picture of a strong expression of emotion (typically happiness, sadness, anger, fear, disgust, and surprise). The intensity of expressed facial emotion can be manipulated by incrementally adjusting the balance between the neutral and intense facial expressions. During the experiment, participants are shown increasingly more expressive faces, and are instructed to respond as soon as they detect the emotion being shown, and then identify the emotion by selecting the appropriate label from a list of possible answers. Facial emotion detection is defined as the earliest correct response for each emotion, and emotion recognition accuracy is defined as the percentage of correct identifications at the highest degree of expressiveness.

Robin et al. (2012) examined facial emotion detection and recognition accuracy in a sample of 22 female adolescents diagnosed with BPD, recruited from outpatient psychiatric clinics in Europe, and 22 healthy controls, matched on age, gender, and socioeconomic status. Participants' ranged in age from 15 to 19 years old (mean age of 16.9 in the BPD group and 16.2 in the control group). Participants completed diagnostic interviews for Axis I and II disorders, and completed the facial morphing procedure. With regard to facial emotion detection, results indicated that participants with BPD required a significantly greater level of facial expressiveness to correctly identify facial emotions. However, this difference was slight: overall, participants with BPD required 30 % intensity to correctly identify emotions, compared to 28 % for participants in the control group. This overall effect was driven by significant group differences in the detection of anger (32 % intensity in the BPD group vs. 30 % intensity in the control group) and happiness (25 % in the BPD group vs. 20 % in the control group). With regard to facial recognition accuracy at 100 % expression, the BPD group did not differ from the control group (84 % vs. 85 % correct, respectively).

Jovev et al. (2011) used the same procedure in a sample of adolescent outpatients recruited from a clinic in Australia (this sample is summarized under Jovev et al., 2012, above). As before, the BPD group consisted of youth who endorsed three or more symptoms of BPD on a self-report questionnaire. Across all emotions, there was no effect of diagnostic status on detection of facial emotion, suggesting that the BPD and control groups required a comparable level of expressiveness to correctly detect facial emotions. The BPD group required moderately (though nonsignificantly) greater facial expressiveness to correctly detect disgust (29 % intensity in the BPD group vs. 26 % in the control group) and fear (30 % vs. 27 %).

Snowden et al. (2013) examined the relationship between gender, BPD features, and emotion detection and recognition accuracy in a sample of college students (51 % female) with a mean age of 21. BPD was assessed using a self-report questionnaire of BPD-related traits. During the emotion recognition accuracy procedure, emotion faces (happiness, sadness, fear, or anger, all at 40 % expressiveness) were displayed one at a time for .5 s. After the offset of each image, participants had to select the appropriate label

for the emotion from among four choices. Recognition accuracy was indexed as the percentage of correct responses. Males and females performed similarly on the recognition accuracy task (55 % correct), and endorsed comparable levels of BPD features. However, among females, higher levels of BPD features were associated with improved recognition accuracy for fear ($r = .20$), while for males, higher levels of BPD features were associated with poorer recognition accuracy for fear ($r = -.19$).

Summary. In summary, there is limited evidence of generalized emotional reactivity as indexed by sensitivity to facial emotion cues. However, an interesting pattern of findings emerged related to processing of fear cues among young females with BPD. In a clinical sample (86 % female), adolescents with elevated BPD symptoms showed attentional bias for subliminal fear-related cues, yet they required a slightly greater degree of expressiveness to correctly identify very subtle fearful facial expressions (Jovev et al., 2012). In a sample of college students, BPD features were modestly associated with more accurate recognition of fearful faces among females, but not among males (Snowden et al., 2013). This pattern of findings suggests that females with BPD are not necessarily more sensitive to the most subtle expressions of fear, but they accurately recognize and preferentially attend to full expressions of fear, even when those cues are presented below the threshold of conscious awareness. Findings seem to suggest that youth with BPD exhibit a specific, rather than generalized, type of reactivity, particularly to fear-related cues.

Do Adolescents with BPD Show Heightened Emotional Reactions to Social Stressors?

Another way to examine affective dysfunction in BPD is to examine subjective and physiological emotional responding to stressful situations. Because people with BPD are thought to be especially reactive to interpersonal stressors, a number of studies have been conducted to examine the effect of aversive social experiences on subjective, behavioral, and physiological responses to stress.

Two studies have examined self-reported emotional responses to aversive interpersonal experiences as a function of BPD symptomatology. In a study by Tragesser, Lippman, Trull, and Barrett (2008), participants ($N = 121$, mean age of 19, 69 % female) completed self-report measures of BPD symptoms (PAI-BOR) and rejection sensitivity, then read a teasing scenario in which the relationship to the teaser (friend vs. stranger) and the content of the teasing (sensitive vs. nonsensitive material) were manipulated. After reading the scenario, participants rated the intensity of their emotions and the likelihood that they would engage in a range of behaviors (e.g., glare, make a mean comment, apologize, look away). Results indicated that participants with higher levels of BPD features reported greater anger and sadness in response to teasing, regardless of the relationship to the teaser or the content of the teasing. Higher BPD scores also were associated with greater self-reported likelihood of engaging in retaliatory behavior (e.g., glaring, making a mean comment) as well as withdrawal (e.g., looking away), however, after controlling for anger scores, these effects were no longer significant. Findings suggest that individuals with BPD features may be more likely to respond to teasing by retaliating or withdrawing because of the intensity of their emotional responses to teasing.

Lawrence, Chanen, and Allen (2011) investigated the effect of ostracism on mood in a sample of outpatient youth with four or more symptoms of BPD ($n = 30$, mean age of 19, 90 % female) and healthy control participants ($n = 22$, mean age of 19, 86 % female). Ostracism was induced using Cyberball, a virtual ball-toss game in which the participant is systematically excluded from a game of catch with three other players. Participants completed ratings of 13 mood states before, immediately following and 15 min after the ostracism experience. Results showed that the BPD group rated their emotions as more intense at every time point; however, there were no group differences in the degree of emotional responding to the rejection experience: all participants reported increased feelings of anger, rejection, surprise, suspicion,

and decreased joy. Timing of emotional recovery did not differ between the two groups. Findings suggest that, compared to healthy individuals, those youth with BPD experience negative emotions as more intense, overall, but that their emotional responses to mild rejection are similar to their healthy peers.

Ruocco et al. (2010) observed neural responses to exclusion among participants with and without BPD. This study focused specifically on the medial prefrontal cortex (mPFC), a region of the brain that mediates various aspects of social information processing, including self-referential judgments (i.e., evaluating oneself to relative to another) and perspective-taking (D'Argembeau et al., 2007). Participants were ten young females with BPD (mean age of 22) recruited from the community and university counseling centers, and ten females without psychiatric illness (mean age of 19) who were recruited from the community. BPD was diagnosed based on a structured clinical interview. Evoked neural activation was measured using functional near infrared spectroscopy (fNIRS), which estimates cerebral blood flow based on the differential absorption of infrared light in oxygenated and deoxygenated blood. The fNIRS has superior temporal resolution to fMRI, and does not require the participant to be isolated in a scanner; however, the fNIRS has slightly poorer spatial resolution. Each participant experienced an inclusion condition, in which she was included in a simple card game with two confederates, and an exclusion condition, in which the confederates completely ignored her after the first round of the game.

Results from the study indicated that the exclusion condition successfully induced feelings of rejection, and participants with and without BPD experienced comparable increases in rejection. During the inclusion condition, the BPD and control groups showed similar levels of activation in the left mPFC, however, in the exclusion condition, the BPD group showed significantly greater activation of the left mPFC than controls. Moreover, severity of rejection and abandonment fears was positively associated with activation in the mPFC during the exclusion condition ($r = .49$). Results indicate that when they experience rejection, females with BPD experience greater activity in a brain region associated with self-referential evaluations and perspective taking. This finding may appear to conflict with previous research that has identified distress-induced *hypo*activation in regions of the prefrontal cortex associated with behavioral inhibition among participants with BPD. However, it is consistent with findings that suggest that BPD symptom severity is associated with the tendency to make excessively elaborate attributions about others' mental states (Sharp et al., 2011).

Woodberry, Gallo, and Nock (2008) conducted a study to examine the effect of validation and invalidation on various measures of emotional responding in young females with features of BPD. Twenty-three females who endorsed high levels of BPD symptoms (mean age of 23) and 18 healthy controls (mean age of 22) were asked to complete a set of relatively easy anagrams, followed by a set of unsolvable anagrams. During the unsolvable anagrams, a research assistant made either a validating comment ("Most people find this set of anagrams really frustrating") or an invalidating comment ("There's no need to get really frustrated. They're just anagrams."). Throughout the session, participants provided self-reports of emotional valence, arousal and comfort with their current feelings. Skin conductance, a measure of activity in the sympathetic nervous system was assessed as a physiological index of emotional reactivity. At baseline and throughout the experimental session, the BPD group reported less happiness and greater discomfort with their current emotional state, but did not differ from controls in either subjective or physiological levels of sympathetic arousal. Regardless of diagnostic status, participants showed a greater increase in skin conductance following invalidation than validation; however, the BPD group did not show a stronger emotional response to invalidation on any dimension of emotional responding. Unexpectedly, participants with BPD showed a significant increase in positive emotional valence after experiencing validation. Findings suggest that,

relative to healthy controls, individuals with BPD were less happy and more uncomfortable with their emotions; however, they did not differ from controls in subjective or objective emotional arousal. Moreover, participants with BPD did not show evidence of increased subjective or objective reactivity to an interpersonal stressor.

Weinberg, Klonsky, and Hajcak (2009) examined sympathetic and parasympathetic responding to social stress in a sample of college students (mean age of 20, 73 % female). Individuals with BPD ($n = 12$) and healthy controls ($n = 28$) provided frustration ratings, as well as measures of heart rate variability (an index of sympathetic activity) and respiratory sinus arrhythmia (an index of parasympathetic activation) before, during, and after exposure to the Trier Social Stress Test. Overall, the BPD group showed increased sympathetic activity and decreased parasympathetic activity; however, there were no group differences in physiological responses to the stressor. Despite the absence of group differences in physiological responses to the social stressor, the BPD group reported greater subjective frustration following the stressor.

Taken together, there does not seem to be consistent evidence that youth with BPD exhibit elevated emotional reactivity to social stressors. This unexpected finding may suggest that the relationship between BPD and affective dysfunction is somewhat complex. For example, it is plausible that only certain subgroups of youth with BPD show increased subjective and physiological reactivity to stressors; however, the studies reviewed here do not consider clinical subgroups or the presence of moderating variables.

Related, it is likely that affective dysfunction alone does not lead to BPD, but rather the disorder may be more likely to develop among individuals with both affective dysfunction and other temperamental vulnerabilities. Recall that the elaborated biosocial model of BPD (Crowell et al., 2009) includes affective dysfunction *and* impulsivity as temperamental risk factors for BPD. In other words, the relationship between affective dysfunction and BPD may emerge only when an individual also is highly impulsive. Indeed, two studies have found that the greatest BPD symptom severity was observed among people who reported both affective dysfunction and disinhibition, while lower levels of BPD traits were observed among people who reported affective vulnerability but not disinhibition.

For example, Gratz et al. (2009) examined the relationship between affective dysfunction, disinhibition, and childhood BPD symptoms in a community sample of children ($N = 263$, mean age of 11, 45 % female) and their caregivers (87 % mothers). Affective dysfunction (e.g., emotional sensitivity, moodiness) and BPD symptoms were assessed using caregiver's report of the child's typical behavior. Each child completed a self-report measure of sensation seeking (one aspect of disinhibition). For girls, both affective dysfunction and sensation seeking were independently associated with BPD symptom severity, that is, girls with greater affective dysfunction and greater sensation seeking also exhibited higher levels of BPD traits. In addition, there was an interaction of affective dysfunction and sensation seeking, such that the greatest BPD symptom severity was observed among girls who had high levels of both vulnerability factors. Among boys, affective dysfunction was associated with greater BPD symptom severity, however, sensation was not associated with BPD symptomatology, nor was there an interaction of affective dysfunction and sensation seeking. Thus, for boys, affective vulnerability alone confers greater risk for BPD-related difficulties, whereas for girls, the combination of affective vulnerability and disinhibition (sensation seeking) is associated with higher levels of BPD features.

A similar pattern of findings emerged in a study conducted by Ayduk et al. (2008). In this study, college students ($N = 379$, mean age of 21, 65 % female) completed self-report measures of rejection sensitivity (a construct similar to emotional reactivity to interpersonal stress), executive control (a construct inversely related to disinhibition), and BPD symptomatology. Results revealed a significant association between rejection sensitivity and borderline personality features ($r = .29$). However, this effect

was moderated by executive control: rejection sensitivity was significantly related to BPD symptom severity among youth with low executive control, but not among youth with high executive control.

Summary. Taken together, these studies provide equivocal evidence that youth with BPD exhibit heightened emotional reactivity or greater emotional intensity in response to interpersonal stressors. Of the studies reviewed here, only two found significant effects of interpersonal stressors on subjective reports of negative emotion (frustration, anger, sadness). Although Ruocco et al. (2010) did not find differences in subjective emotional responses to exclusion, this study did identify differential patterns of brain activity in the BPD and control groups, such that the participants with BPD showed greater activation in the mPFC following exclusion. Of the two studies that assessed physiological responses to interpersonal stressors, neither found evidence of increased sympathetic arousal (skin conductance, heart rate variability) in response to the stressors, nor was there evidence of increased parasympathetic activity (respiratory sinus arrhythmia). Notably, baseline group differences emerged in three studies: relative to control participants, youth with BPD reported greater intensity of emotions, less happiness and less emotional comfort, and exhibited greater overall sympathetic arousal.

One explanation for these modest findings is the possibility that not all individuals with BPD show exaggerated responses to interpersonal cues and stress. The presence of a moderating variable, such as disinhibition, may help to explain the surprising absence of group differences in emotional reactivity. Two studies found evidence that the relationship between emotional variables (affective dysfunction, rejection sensitivity) and BPD was strongest among individuals who also reported high levels of disinhibition. Although it is difficult to draw conclusions given methodological and statistical discrepancies in self-report and laboratory studies, findings do provide some support for the moderating role of impulsivity in the relationship between affective dysfunction and BPD.

Emotion Regulation Problems

Whereas affective dysfunction refers to extreme or excessive emotional responding, emotion regulation problems have to do with maladaptive responses to emotions, including the use of emotion regulation strategies that are ineffective or have high probability of negative consequences, as well as involvement in mood-dependent behavior that interferes with one's goals.

Is There a Relationship Between BPD and Emotion Regulation Problems Among Adolescents?

One of the most widely used measures of emotion regulation problems is the Difficulties in Emotion Regulation Scale (DERS: Gratz & Roemer, 2004). The DERS consists of six subscales, proposed to measure distinct emotion regulation problems: Clarity (the degree to which a person knows, and feels clear, about what they are feeling); Awareness (the extent to which a person attends to and acknowledges emotional experiences); Nonacceptance (the extent to which a person experiences negative secondary emotional responses, such as guilt or embarrassment, when they are upset); Goals (the extent to which a person is able to persist in goal-directed behavior when upset); Impulse (the extent to which a person perceives a loss of control over behavior when upset); and Strategies (the degree to which a person believes that there is little that can be done to modulate negative emotions). Questions are worded such that higher scores indicate greater emotion regulation problems. Both the overall subscale and the individual subscale scores are typically reported. Although the relationship between BPD and emotion dysregulation has been extensively documented in adult samples (Bornovalova et al., 2008; Chapman, Leung, & Lynch, 2008; Gratz & Roemer, 2004), comparatively less is known about the nature of emotion regulation problems in adolescents with BPD.

Sharp, Ha, Michonski, Venta, and Carbone (2012) examined the relationship between BPD and emotion regulation difficulties in a sample of youth recruited from an inpatient psychiatric

clinic ($N = 190$, mean age of 15.4, 59 % female). Adolescents completed self-report measures of emotion dysregulation, BPD (Borderline Personality Features Scale for Children, Personality Assessment Inventory for Adolescents) and internalizing and externalizing disorders, as well as a semi-structured diagnostic interview (Childhood Interview for Borderline Personality Disorder). Overall emotion regulation difficulties, as assessed by the DERS, was significantly correlated with adolescents' paper-and-pencil self-reports of BPD symptomatology ($r = .70$) and interviewer-rated symptom severity ($r = .55$). When evaluated categorically, youth diagnosed with BPD reported significantly more emotion regulation problems than clinical youth without BPD.

Glenn and Klonsky (2009) also explored the relationship between BPD symptoms and emotion dysregulation in two separate samples of undergraduates (sample 1: $n = 243$, 48 % female; sample 2: $n = 30$, 50 % female). In sample 1, participants completed a self-report measure of BPD, and in sample 2, participants completed a semi-structured diagnostic interview for BPD, and all participants completed self-report measures of emotion dysregulation, negative affect, depression, and anxiety. Glenn and Klonsky (2009) reported moderate relationships between overall emotion dysregulation and BPD assessed by self-report ($r = .54$) and diagnostic interview ($r = .64$). In both samples, BPD symptoms were positively and significantly correlated with most facets of emotion dysregulation, including lack of emotional clarity ($r = .35$ [sample 1], .34 [sample 2]), emotional nonacceptance ($r = .38$, .45), difficulty engaging in goal directed behavior when upset ($r = .34$, .49), impulse control problems ($r = .47$, .76), and lack of access to emotion regulation strategies ($r = .55$, .67). No significant association of BPD and emotional awareness was found in either sample ($r = .03$, .18). To test the hypothesis that emotion dysregulation would be associated with BPD beyond the effects of negative emotionality, Glenn and Klonsky examined partial correlations of BPD and aspects of emotion dysregulation, controlling for negative emotionality. In sample 1, after controlling for negative emotionality, the associations between BPD symptoms and emotion dysregulation were diminished, but remained significant. In sample 2, many of the associations between BPD and aspects of emotion dysregulation were no longer significant. However, in both samples, BPD symptoms remained robustly associated with the strategies subscale ($r = .24$, .37) and the impulse subscale ($r = .40$, .55) after controlling for negative emotionality.

Summary. These findings suggest that there is a robust, positive relationship between overall emotion regulation problems and BPD symptomatology. This pattern was identified in a high-severity clinical population (i.e., adolescents in inpatient treatment) and among nonclinical young adults. Within the nonclinical sample, BPD symptom severity was associated with greater emotion regulation problems in all domains except emotional awareness. However, for some emotion regulation problems (e.g., emotional nonacceptance, emotional clarity, difficulty engaging in goal-directed behavior when upset), the relationship with BPD was explained, in part, by the characteristic intensity of negative affect that the participant experienced. In other words, youth with BPD may experience more emotion regulation problems in these particular domains because they tend to experience more intense negative emotions, in general (i.e., affective dysfunction). However, the relationship between BPD two specific facets of emotion regulation (lack of access to emotion regulation strategies, and engagement in mood-dependent behavior) was strong even after controlling for negative emotionality. That is, young adults with BPD reported fewer strategies to modulate their emotions and greater engagement in problematic mood-dependent behavior, but this was not due simply to the fact that they experienced stronger negative affect. Young adults with BPD symptoms may perceive their emotions as unmanageable, and may struggle to control mood-dependent behavior, even if their emotions are not particularly intense.

Is There a Relationship Between BPD and Use of Problematic Emotion Regulation Strategies Among Adolescents?

Young adults who endorse higher levels of BPD symptomatology also report difficulties implementing effective appropriate emotion regulation strategies (Glenn & Klonsky, 2009). A possible explanation for this finding is that youth with BPD may try to use emotion regulation strategies that backfire and actually increase negative emotions. One such strategy is suppression, which involves responding to negative thoughts and emotions with self-judgment and self-criticism, and attempting to control and avoid negative thoughts and emotions. People use suppression to inhibit unwanted thoughts and emotional experiences; however, this strategy may be counterproductive. Suppressive emotion regulation strategies often fail to decrease subjective distress, and actually increase the frequency and intensity of unwanted thoughts, increase attentional bias for cues related to the suppressed thought or emotion, and increase physiological arousal (Clark, Ball, & Pape, 1991; Gross, 1998; Lavy & van den Hout, 1994; Wegner, Schneider, Carter, & White, 1987; Wenzlaff & Wegner, 2000). Use of thought suppression is associated with anxiety disorders, depression, and eating disorders (Aldao & Nolen-Hoeksema, 2010), and there have been several efforts to characterize the relationship between thought suppression and BPD among young adults.

Cheavens et al. (2005) examined the effect of negative affect intensity/reactivity and parental criticism on BPD features in a sample of college students ($N = 202$, mean age of 19). Participants completed a self-report measure of BPD features, suppression, negative emotion intensity/reactivity, and parental criticism. Results indicated that negative emotion intensity and reactivity, parental criticism, and thought suppression were each independently associated with greater BPD symptomatology. Further, they found that suppression fully mediated the effects of negative affect intensity/reactivity and parental criticism on BPD symptomatology. Findings suggest that it is not the effect of either negative affectivity or parental criticism, per se, that predict difficulties with BPD-related symptoms, but rather, the potentially maladaptive way in which people respond to negative affect and parental criticism, specifically through avoidance and attempts to control their thoughts and emotions.

In a replication and extension of this study, Sauer and Baer (2009) examined the relationship between childhood affective dysfunction (e.g., emotional sensitivity and reactivity), parental invalidation of emotional experiences, and the presence of BPD symptoms, in a sample of college students ($N = 104$, mean age of 19, 77 % female). Participants completed a self-report measure of BPD symptoms (PAI-BOR), as well as measures of childhood emotional intensity and parental invalidation (e.g., the extent to which parents reacted to the child's emotional expressions by becoming distressed, punishing, or minimizing). In addition, participants completed measures of thought suppression and fear of negative emotions. Consistent with the findings from Cheavens et al. (2005), they found that self-reported affective dysfunction, parental invalidation, and suppression were each independently associated with BPD symptomatology. Further, they found evidence that the relationship between affective dysfunction and BPD symptoms was partially mediated by suppression, and that the relationship between parental invalidation and BPD symptoms was fully mediated by suppression. Subsequent analyses revealed that the relationship between affective dysfunction and thought suppression was partially mediated by fear of emotions, and likewise, the relationship between parental emotional invalidation and thought suppression was fully mediated by fear of emotions. In other words, results indicate how early temperamental and environmental vulnerabilities may contribute to the development of BPD. Specifically, both affective dysfunction and parental invalidation increased fear of emotions, which was associated with greater use of thought suppression as an emotion regulation strategy, which, in turn, predicted BPD symptom severity in late adolescence.

Summary. Consistent with Linehan's biosocial model, findings support a robust relationship between BPD features and emotion regulation problems. A particularly strong relationship emerged between BPD and two domains of emotion regulation difficulties: inadequate emotion regulation strategies and lack of control over mood-dependent behavior. Indeed, research on thought suppression, a specific form of problematic emotion regulation, identified a relationship between the use of thought suppression and BPD symptom severity. Further, two studies found that thought suppression mediated (or partially mediated) the relationship between BPD and thought suppression. In other words, it is not affective dysfunction, per se, that is associated with BPD symptom severity, but rather, ineffective or maladaptive attempts to manage frequent and intense negative emotions.

Current Status and Future Directions

Studies of affective dysfunction in adolescents with BPD (or BPD features) did not reveal a consistent pattern of emotional responses to social cues or interpersonal stress. With regard to sensitivity and reactivity to facial cues, female adolescents with BPD were more accurate than controls in identifying fearful faces, and showed attentional bias for fearful faces, whereas no consistent effects were found for other types of emotional expressions. Findings may provide preliminary evidence of emotional reactivity to fear cues among youth with BPD, but continued research is warranted to replicate this effect and ascertain its implications for real-world emotional and interpersonal functioning.

Several studies examined subjective and physiological responses to interpersonal stressors, including teasing, exclusion, negative evaluation, and invalidation. Findings were striking for their inconsistency; however, several patterns warrant comment. First, although the laboratory stressors successfully increased negative emotions for participants with and without BPD, youth with BPD did not exhibit more intense reactions than controls. Second, of the two studies that assessed emotional recovery following the stressor, neither found evidence of prolonged return to emotional baseline. Finally, although youth with BPD did not respond more strongly to the stressors, they tended to report greater overall negative affectivity relative to controls.

There are both methodological and conceptual explanations for these findings. Unexpected results may be attributed to the use of subtle interpersonal stressors that lacked ecological validity (e.g., an invalidating comment from an unknown research confederate; a computerized ball toss game). Consistent with this perspective, group differences in subjective emotional response to the stressor were observed in the study that used the most elaborate stressor (5 min of speeded mental arithmetic, interrupted by frequent negative evaluative statements from the experimenter; Weinberg et al., 2009). The use of more extreme and aversive manipulations poses obvious ethical challenges, but may be necessary to approximate the profound affective dysfunction that is documented in the clinical literature. Another possibility is that real, albeit modest, group differences in emotional responding were obscured by lack of statistical power. Although larger samples will partially address this problem, an important (and feasible) next step is for future research to include descriptive statistics and/or effect sizes for group differences in emotional responses, which will facilitate future meta-analysis.

Another interpretation of these unexpected findings is that the relationship between affective dysfunction and BPD may be moderated by other variables, with evidence currently pointing to disinhibitory traits (e.g., poor executive control, low ego strength). Poor impulse control limits one's ability to modulate unwanted emotional responses and inhibit problematic behavior. Youth who experience frequent and extreme emotional reactions, and who have a limited capacity to inhibit unwanted emotions and behavior, may be especially vulnerable to develop BPD (Crowell et al., 2009). Moreover, youth who experience affective dysfunction but have superior inhibitory control may be better

able to manage their emotions and, as a result, less likely to develop BPD. Continued research is needed to explore the interactive effects of affective dysfunction and impulsivity on the development of BPD.

Of course, it is also possible that youth with and without BPD do not differ systematically in their emotional reactions. Youth with and without BPD may have similar frequency, intensity, and duration of emotional responses, but may respond differently to their emotional experiences. Indeed, findings suggest that youth with BPD report more emotion regulation problems than youth without BPD (Sharp et al., 2012). In addition, BPD symptom severity was positively associated with perceived lack of access to effective emotion regulation strategies, as well as the use of thought suppression, a potentially problematic emotion regulation strategy. A number of studies found that thought suppression accounted for the relationship between affective dysfunction and BPD symptom severity in youth with BPD. These findings provide empirical support for the notion that the relationship between affective dysfunction and BPD symptom severity can be explained, at least in part, by the use of problematic strategies to manage negative emotions. Presently, only one specific form of maladaptive emotion regulation (thought suppression) has been studied. Additional research is warranted to identify additional adaptive and maladaptive emotion regulation strategies used by youth with BPD, and to understand *why* youth with BPD perceive a lack of access to emotion regulation strategies (e.g., lack of knowledge, failure to implement strategies).

There have been great advances in our understanding of the nature of emotion dysregulation in youth with BPD, yet many questions remain unanswered. A clear limitation of current research is the reliance on cross-sectional studies. Although extant research provides important information about affective dysfunction and emotion regulation problems in youth with BPD, it does not allow us to draw conclusions about the *development* of BPD. There is a clear need for prospective, longitudinal research that will allow us to test more sophisticated etiological models of the disorder that incorporate biological, psychological, and social levels of analysis. Ultimately, this research will have important implications for prevention and intervention for vulnerable youth.

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Self-Injurious Behaviors in Adolescents with Borderline Personality Disorder

14

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Introduction

This chapter focuses on the associations between self-injurious behaviors (SIB) and borderline personality disorder (BPD) pathology in adolescents. Adolescence marks a period of high risk for the onset of SIB (including both deliberate self-harm (DSH) and suicidal behaviors), with adolescents with BPD constituting a particularly vulnerable population for SIB. After providing a brief overview of the definitions of and distinctions between DSH and suicidal behaviors, we review Crowell and colleagues' (Crowell, Beauchaine, & Lenzenweger, 2008; Crowell, Beauchaine, & Linehan, 2009) developmental model of SIB and BPD, which proposes that SIB may be an early indicator of BPD in adolescents. According to this model, trait impulsivity and emotion dysregulation are the key features underlying the development of BPD and SIB. Thus, the final sections of this chapter review the research on the associations between SIB and both emotion dysregulation and trait impulsivity among adolescents in general (as well as adolescents with BPD in particular). Finally, clinical implications of this research are discussed, as are needed future directions in this line of inquiry.

Definition and Significance of Self-Injurious Behaviors in Borderline Personality Disorder

SIB may be defined broadly as the deliberate, direct destruction of body tissue with or without suicidal intent (comparable to the definition of *parasuicidal behaviors*; Kreitman, 1977; Linehan, 1993). As such, SIB may involve a clear or ambivalent intent to die (i.e., suicidal behaviors; see Crowell et al., 2008; O'Carroll et al., 1996) or no intent to die (i.e., DSH; Chapman, Gratz, & Brown, 2006; Fliege, Lee, Grimm, & Klapp, 2009; Gratz, 2001; Pattison & Kahan, 1983). Although often examined as a broad class of behaviors (due to their phenotypic similarities and common co-occurrence; Joiner et al., 2005; Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006), researchers have increasingly argued for the importance of distinguishing between SIB on the basis of the intent to die (e.g., Chapman et al., 2006; Gratz, 2003; Gunderson, 2001; Jacobson & Gould, 2007; Nock & Kessler, 2006; Pattison & Kahan, 1983; Walsh & Rosen, 1988), given theoretical and empirical literature emphasizing differences in the functions of these behaviors. For example, DSH (also referred to as nonsuicidal self-injury) has often been conceptualized as antithetical to suicidal behaviors

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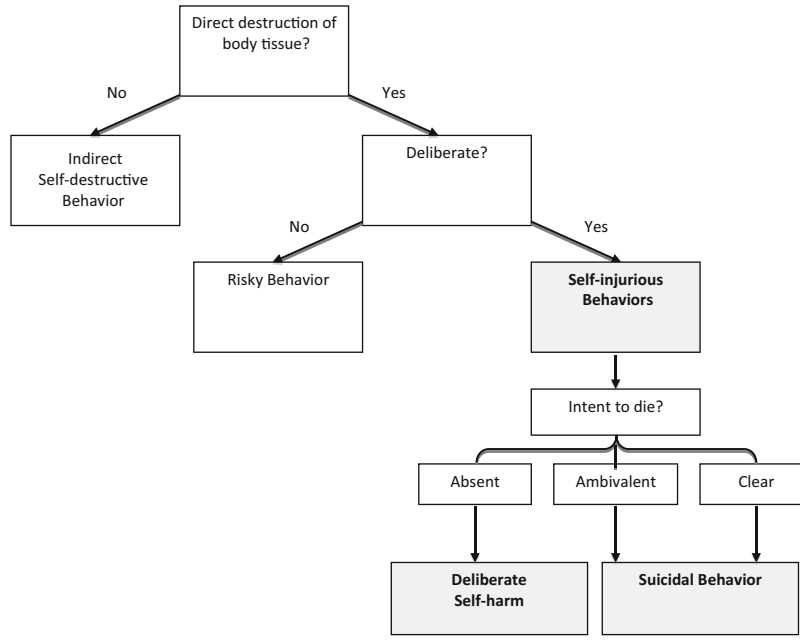

Fig. 14.1 Definition and classification of self-destructive and self-injurious behaviors

(e.g., Gratz, 2003; Pattison & Kahan, 1983; Sabo, Gunderson, Najavits, Chauncey, & Kisiel, 1995), and a growing body of empirical research provides evidence for different functions and correlates of these behaviors (see Brown, Comtois, & Linehan, 2002; Muehlenkamp & Gutierrez, 2004; Nock & Kessler, 2006). Thus, whenever possible, we will differentiate between DSH and suicidal behaviors throughout this chapter, using the term SIB only when discussing theories of these behaviors as a whole or reviewing research that does not differentiate between SIB with and without suicidal intent. Figure 14.1 depicts the distinctions between various self-destructive behaviors and SIB, including the defining characteristics of both DSH and suicidal behaviors.

Public Health Relevance

The public health consequences of SIB are staggering, particularly among adolescents. For example, SIB in general are associated with a number of intrapersonal and interpersonal difficulties among adolescents (e.g., internalizing and externalizing psychopathology and social problem solving deficits; Hawton, Kingsbury, Steinhardt, James, & Fagg, 1999; Hawton, Rodham, Evans, & Weatherall, 2002), as well as heightened risk for suicide (Hawton, Houston, & Shepperd, 1999; O'Connor & Sheehy, 2000; Skegg, 2005). Likewise, suicidal behaviors in adolescents have been found to be associated with a range of negative consequences, including psychopathology (Gould et al., 1998), school problems (Lewinsohn, Rohde, & Seeley, 1993), and both future suicide attempts (Sapyta et al., 2012) and completed suicide (Shafii, Carrigan, Whittinghill, & Derrick, 1985). DSH has also been found to be associated with a wide range of negative outcomes among adolescents, including interpersonal problems, low self-esteem, emotional and behavioral difficulties, and substantial psychological suffering and distress (Bjarehed & Lundh, 2008; Hilt, Cha, & Nolen-Hoeksema, 2008; Jacobson & Gould, 2007). Furthermore, although DSH and suicidal behaviors represent functionally distinct behaviors, DSH poses increased risk for later suicidal behaviors among adolescents (Guan, Fox, & Prinstein, 2012; Jacobson & Gould, 2007), emerging as the strongest predictor of suicide attempts within adolescent samples (Asarnow et al., 2011). Moreover, research indicates that adolescents

with a history of multiple suicide attempts (versus a single attempt) are more likely to report engaging in DSH (Esposito, Spirito, Boergers, & Donaldson, 2003).

Of note, research suggests that adolescents who engage in both suicidal behaviors and DSH may be a particularly high-risk group, evidencing greater impairment than adolescents who engage in either DSH or suicidal behaviors. For example, studies have found that adolescents with both DSH and suicidal behaviors evidence (a) higher levels of depression, loneliness, anger, and risk-taking than adolescents with only suicidal behaviors (Guertin, Lloyd-Richardson, Spirito, Donaldson, & Boergers, 2001), and (b) both higher levels of depression and suicidal ideation and higher rates of major depression and PTSD diagnoses than adolescents with DSH only (Jacobson, Muehlenkamp, Miller, & Turner, 2008). Likewise, among adolescent psychiatric inpatients with a history of DSH, frequency of suicidal behaviors was positively associated with the duration of DSH, as well as the number of DSH methods used (Nock et al., 2006), providing some evidence that the co-occurrence of suicidal behaviors with DSH may be associated with more chronic and severe DSH.

Developmental Model of Self-Injurious Behaviors and BPD in Adolescents

Suicidal behaviors and DSH are considered core features of BPD, and constitute one criterion for the disorder (American Psychological Association, 2000). The relevance of these behaviors to BPD among adolescents is particularly great, as SIB have been theorized to be a developmental precursor to BPD in some youth (Crowell et al., 2008, 2009; Paris, 2005). Specifically, one of the most well-articulated and comprehensive developmental models of BPD, the Biosocial Developmental Model (Crowell et al., 2008, 2009; see also Chap. 9 in the present volume), proposes that SIB may be an early indicator of BPD in adolescents, reflecting a key step for some youth in the developmental trajectory toward BPD (Crowell et al., 2008, 2009). Drawing upon Linehan's (1993) biosocial theory of the development of BPD (a model that has been applied extensively to the pathogenesis of DSH in adults; see Gratz, 2003, 2006; Gratz & Roemer, 2008), Crowell et al.'s (2009) model incorporates a developmental psychopathology perspective. As such, its stronger developmental emphasis makes it particularly well-suited to understand BPD-related pathology in adolescents.

According to this model, trait impulsivity and emotion dysregulation are the key features underlying the development of both BPD and SIB, with SIB emerging earlier in development as a maladaptive coping strategy that marks increased risk for BPD among a subset of youth (Crowell et al., 2009). Specifically, these features are proposed to have both independent and transactional influences on the development of BPD and SIB, with heightened risk for these phenomena thought to stem from the combination of impulsivity and emotion dysregulation (Crowell et al., 2009, 2012). Indeed, it is the confluence of these factors and their transactions over time that is believed to increase the risk for maladaptive emotion regulation strategies, such as SIB, in adolescence. As such, emotion dysregulation is considered the proximal risk factor for SIB, and is thought to become particularly salient during adolescence.

Development of Emotion Regulation in Childhood and Adolescence

The emphasis on emotion regulation within the Biosocial Developmental Model of BPD is consistent with developmental literature emphasizing the relevance of emotion regulation to adaptive functioning across multiple domains (Calkins, 1994; Cole, Michel, & Teti, 1994; Kopp, 1989). Indeed, researchers in the area of developmental psychology have long suggested that adaptive emotion regulation is integral to normative development (Cole et al., 1994; Thompson, 1994). Although emotion regulation capacities begin to develop in infancy in the context of the attachment relationship (Calkins, 2004; Eisenberg, Cumberland, & Spinrad, 1998;

Eisenberg, Spinrad, & Eggum, 2010; Kopp, 1989; Rothbart, Ziaie, & O'Boyle, 1992), the development of more sophisticated emotion regulation capacities continues throughout childhood (Saarni, 1979; Southam-Gerow & Kendall, 2002). Given that most children develop the capacity for emotion regulation by late childhood (Gnepp & Hess, 1986), early adolescence represents a time when children may begin to utilize these capacities or, conversely, when deficits in these capacities may emerge or become apparent. Thus, adolescence may represent a particularly crucial developmental period for the emergence of behaviors thought to stem from emotion dysregulation, such as SIB and BPD more broadly.

Consistent with this theory, not only is adolescence characterized by increasing emotional demands associated with a variety of biological, relational, and psychological changes linked to puberty and emerging social responsibilities (Graber & Brooks-Gunn, 1996; Larson, Moneta, Richards, & Wilson, 2002; Larson & Richards, 1991), the development of independent emotion regulation (in the absence of external regulation by caregivers) is considered an important developmental milestone of this period (Steinberg et al., 2006). Thus, as a result of this simultaneous increase in emotional demands and decrease in the external regulation of emotions by caregivers, adolescence is marked by increased demands on emotion regulatory capacity. Consequently, it is during this developmental period when maladaptive emotion regulation strategies, such as SIB, may be most likely to emerge among youth at risk for BPD (i.e., those with heightened levels of impulsivity and/or emotion dysregulation).

Emergence of SIB in Adolescence

Consistent with these theories, there is extensive evidence for the emergence of SIB in early adolescence (Dougherty et al., 2009; Jacobson & Gould, 2007; Young, van Beinum, Sweeting, & West, 2007). For example, in a study of adolescent psychiatric inpatients (aged 12–19) with a history of at least one episode of DSH in the year prior to hospital admission, the average age of onset of DSH was 12 years (Ferrara, Terrinoni, & Williams, 2012). These findings are comparable to those obtained in a large community sample of adolescents (average age = 16.5), which also found an average age of onset of DSH of 12 years (Cerutti, Manca, Presaghi, & Gratz, 2011). Likewise, research suggests that the risk for first onset of suicidal behavior increases in early adolescence and peaks at age 16 (Nock et al., 2008), with suicide emerging as one of the leading causes of death among both early and late adolescents (Anderson & Smith, 2003; Centers for Disease Control and Prevention, 2007; Evans, Hawton, Rodham, & Deeks, 2005).

Moreover, consistent with Crowell et al.'s (2009) theory that SIB may be a developmental precursor to BPD, preliminary evidence suggests that the presence of BPD in late adolescents and adults is associated with an earlier age of onset of DSH. For example, in a study examining DSH subgroups within a sample of self-harming late adolescents and young adults, BPD symptoms were elevated among the subgroup of self-harming participants with the earliest age of onset of DSH (i.e., 11.5 years; Klonsky & Olino, 2008). Furthermore, among a large sample of adult patients with BPD and DSH, 33 % reported first engaging in DSH during childhood (prior to the age of 13; Zanarini et al., 2006). Given findings from nonclinical community samples of adolescents indicating very low rates of DSH prior to the age of 11 (i.e., under 5 %; see Young, Sweeting, & Ellaway, 2011), evidence for such a high rate of DSH during childhood among individuals with BPD is notable. Thus, these studies provide suggestive evidence that the age of onset of SIB may be earlier among those at risk for BPD.

Research on Self-Injurious Behaviors and BPD in Adolescents

Co-occurrence of SIB and BPD in Adolescents

Adolescents with BPD are a population at high risk for SIB (Links, Gould, & Ratnayake, 2003;

Paris, 2005). For example, a recent review of the literature on suicidal behaviors and cluster B personality disorders in youth concluded that youth with BPD are at increased risk for suicidal behavior and suicide completion (Links et al., 2003). Likewise, a recent study by Sharp, Ha, Michonski, Venta, and Carbone (2012) found that adolescent psychiatric inpatients with BPD reported significantly more frequent DSH than those without BPD.

Furthermore, youth with BPD are overrepresented among adolescent self-injuring samples (see Table 14.1). For example, rates of BPD among adolescent psychiatric inpatients with a history of DSH range from 52 to 64 % (Ferrara et al., 2012; Nock et al., 2006). Likewise, within a sample of adolescent patients (aged 12–19) receiving psychiatric treatment following a suicide attempt ($N = 40$), 55 % met criteria for BPD (Crumley, 1979). Moreover, rates of BPD have been found to be significantly higher among adolescent inpatients with (versus without) a history of suicidal behaviors (i.e., 30 % versus 9 %, respectively; Halfon, Laget, & Barrie, 1995). Finally, with regard to deaths by suicide among adolescents and young adults, a retrospective file review conducted in Sweden found that 33 % of those who died by suicide met criteria for BPD (Runeson & Beskow, 1991).

Associations Between SIB and BPD Pathology in Adolescents

Studies consistently demonstrate robust associations between SIB and BPD pathology in adolescents. For example, adolescents with a history of repeated SIB have been found to report higher levels of BPD features than both healthy and depressed adolescents without a history of SIB (Crowell et al., 2012). Likewise, adolescent outpatients (between the ages of 12 and 19) who reported any SIB (including DSH and/or suicidal behaviors) were more likely to endorse clinically significant BPD features than outpatients without a history of SIB (Jacobson et al., 2008). Moreover, BPD symptoms have been found to predict repeated (versus single-episode) SIB among adolescent outpatients ($N = 441$; average age $= 14.9$) with a history of any SIB (Muehlenkamp, Ertelt, Miller, & Claes, 2011).

With regard to suicidal and DSH behaviors specifically, a chart review study of adolescent male offenders (aged 12–20, average age $= 16$) in a residential facility found that those with high levels of BPD traits ($n = 239$) reported more suicidal behavior than those with low levels of BPD traits ($n = 1{,}197$; Taylor, James, Reeves, & Kistner, 2009). Furthermore, several studies have provided evidence for a robust association between DSH and BPD pathology among adolescents. For example, in a large community sample of middle- and high-school students ($N = 1{,}931$), Gratz et al. (2012) found that BPD features were reliably associated with both the presence of DSH and frequent DSH, above and beyond relevant demographic characteristics. Similarly, BPD symptoms were associated with both the presence and frequency of DSH within a large community sample of Italian adolescents (Cerutti et al., 2011). Of note, in both of these studies, the association between BPD pathology and DSH remained significant when items pertaining to SIB were excluded from the calculation of the BPD variables. Moreover, in data drawn from a large-scale longitudinal examination of Chinese adolescents (Year 1: $N = 6{,}212$; Year 2: $N = 6{,}393$), four core features of BPD (affective instability, disturbed interpersonal relationships, unstable sense of self, and behavioral impulsivity) measured at Years 1 and 2 reliably distinguished between adolescents with and without a history of DSH (You, Leung, Lai, & Fu, 2012). Finally, the aforementioned study by Sharp et al. (2012) extends research on the association between DSH and BPD pathology to a clinical sample, providing evidence for a significant positive association between BPD symptoms and DSH frequency among adolescent psychiatric inpatients.

Co-occurrence of DSH and Suicidal Behaviors in BPD in Adolescents

Although preliminary, there is some evidence to suggest that adolescents with (versus without)

Table 14.1 Rates of BPD (and elevated BPD features) in adolescent self-injuring samples

Citation	Sample	*N*	Age M (SD)	Measure to assess BPD	% BPD
Self-injurious behaviors					
Crowell et al. (2012)	Adolescents with ≥3 SIB in past 6 months, or ≥5 SIB in lifetime with ≥1 in past 6 months	27	16.3 (1.0)	SCID-II for DSM-IV (First et al., 1997)	37
Deliberate self-harm					
Cerutti et al. (2011)	Secondary school students with ≥1 DSH behavior	98	16.5 (1.7)[a]	Structured clinical interview for DSM-III-R personality questionnaire (Spitzer et al., 1990); ≥5 BPD symptoms	68
Ferrara et al. (2012)	Adolescent inpatients with ≥1 DSH behavior in the past 12 months	52	15.5 (1.7)	SCID-II for DSM-IV (First et al., 1997)	64
Jacobson et al. (2008)	Adolescent outpatients in specialty depression and suicide clinic	30	15.1 (1.7)[b]	SCID-II for DSM-IV (First et al., 1997); ≥4 BPD symptoms (excluding SIB item)	27
Kaess et al. (2012)	Female adolescents with ≥5 DSH behaviors in the past 12 months from a psychiatry department	14	16.6 (1.7)	Clinical diagnoses according to ICD-10 criteria	43
Nock et al. (2006)	Adolescent psychiatric inpatients with DSH behavior in the past 12 months	89	14.7 (1.4)	Diagnostic interview for DSM-IV personality disorders (Zanarini et al., 1996)	52
Suicidal behaviors					
Crumley (1979)	Adolescents seen in psychiatric practice after a suicide attempt	40	15.8 (no SD available)	Diagnoses according to DSM-III	55
Halfon et al. (1995)	Adolescent psychiatric inpatients with history of suicide attempt	61	13 (13.4)	Diagnoses according to DSM-III-R	30
Jacobson et al. (2008)	Adolescent outpatients in specialty depression and suicide clinic	38	15.1 (1.7)[b]	SCID-II for DSM-IV (First et al., 1997); ≥4 BPD symptoms (excluding SIB item)	38
Both deliberate self-harm and suicidal behaviors					
Jacobson et al. (2008)	Adolescent outpatients in specialty depression and suicide clinic	40	15.1 (1.7)[b]	SCID-II for DSM-IV (First et al., 1997); ≥4 BPD symptoms (excluding SIB item)	44

BPD borderline personality disorder, *SIB* self-injurious behaviors, *SCID-II* structured clinical interview for DSM-IV axis II disorders, *DSM* Diagnostic and Statistical Manual of Mental Disorders, *DSH* deliberate self-harm, *ICD-10* International Statistical Classification of Diseases and Related Health Problems, 10th revision

[a]Average age of full sample of adolescents

[b]Age averaged across all self-injuring subgroups

BPD are more likely to engage in both types of SIB (DSH and suicidal behaviors), rather than either DSH or suicidal behaviors alone (Muehlenkamp et al., 2011). For example, among adolescent outpatients (average age = 14.9) with a history of recent SIB (in the past 16 weeks), both levels of BPD symptoms and rates of BPD per se were significantly higher among the group of participants with a history of both DSH and suicidal behaviors, compared to those with a history of only DSH or suicidal behaviors alone (Muehlenkamp et al., 2011). Moreover, in a study examining DSH subgroups within a sample of self-harming late adolescents and young adults, the highest levels of BPD symptoms were found among the subgroup of self-harming participants with the greatest rates of suicidal behaviors (Klonsky & Olino, 2008). Given the aforementioned research suggesting greater impairment, worse outcomes, and more

chronic SIB among individuals with co-occurring DSH and suicidal behaviors (Guertin et al., 2001; Jacobson et al., 2008; Nock et al., 2006), these studies suggest that adolescents with BPD may be at risk for a more severe course of SIB.

Research on the Relation Between Emotion Dysregulation and Self-Injurious Behaviors in Adolescents

Most of the research examining the association between emotion dysregulation and SIB within adolescents has focused on SIB outside the context of BPD. These studies have provided support for a robust association between multiple dimensions of emotion dysregulation and SIB among adolescents. For example, in a recent study of DSH in adolescent inpatients, Perez, Venta, Garnaat, and Sharp (2012) found that adolescents with a history of DSH (versus those without a history of DSH) reported higher levels of overall emotion dysregulation, as well as the specific dimensions of lack of emotional awareness and clarity, emotional nonacceptance, difficulties engaging in goal-directed behaviors when distressed, difficulties controlling impulsive behaviors when distressed, and limited access to effective emotion regulation strategies. Findings of this study also highlighted the particular relevance of difficulties accessing effective emotion regulation strategies to DSH within this population, as only this dimension of emotion dysregulation evidenced a unique association with DSH (above and beyond the other dimensions of emotion dysregulation; Perez et al., 2012). Consistent with these findings, a recent study of late adolescents (aged 18–19) found that those with a history of DSH reported greater use of maladaptive coping strategies than those without a history of DSH (Cawood & Huprich, 2011). Finally, there is some evidence that emotion dysregulation mediates the association between adverse environmental experiences and DSH in adolescence. Specifically, in a study of female adolescent psychiatric inpatients aged 13–18 (Adrian, Zeman, Erdley, Lisa, & Sim, 2011), emotion dysregulation was found to mediate the association between adverse family and peer experiences and DSH frequency (consistent with findings among young adults that emotion dysregulation partially mediates the association between environmental stressors and DSH; Gratz & Roemer, 2008).

Likewise, adolescents with a history of SIB in general have been found to report higher levels of overall emotion dysregulation and the specific dimension of difficulties controlling impulsive behaviors when distressed than both healthy and depressed adolescents without a history of SIB, and higher levels of the specific emotion dysregulation dimensions of lack of emotional awareness and clarity, emotional nonacceptance, difficulties engaging in goal-directed behaviors when distressed, and limited access to effective emotion regulation strategies than healthy adolescents (Crowell et al., 2012). Furthermore, within a large sample of Catalonian high-school students ($N = 1{,}171$, aged 12–16), adolescents with past-year SIB (compared to those without past-year SIB) reported greater use of avoidant coping strategies (Kirchner, Ferrer, Forns, & Zanini, 2011). Finally, there is some evidence to suggest that certain dimensions of emotion dysregulation may be relevant to suicidal behaviors in particular among youth. For example, the ability to understand and manage emotions has been found to moderate the association between childhood sexual abuse and suicidal behavior among adolescents, serving as a protective factor for suicidal behaviors within this population (Cha & Nock, 2009). Moreover, in a large study of depressed youth, the presence of suicidal behaviors was associated with greater maladaptive (and fewer adaptive) emotion regulation strategies, even when controlling for depression severity (Tamás et al., 2007).

Importantly, evidence for an association between emotion dysregulation and SIB in adolescence has also been provided by studies using physiological and neurophysiological measures of emotion regulation. For example, in one laboratory study, adolescent females with a history of repeated SIB ($n = 23$) were found to evidence deficits in emotion regulation across a number of

indices, compared to control participants ($n = 23$; Crowell et al., 2005). Specifically, adolescents with a history of SIB evidenced lower peripheral serotonin levels and lower respiratory sinus arrhythmia (both in general and in response to emotion inductions), all of which are considered biological markers of poor emotion regulation capacity (Crowell et al., 2005). Likewise, abnormalities in the anterior cingulate gyrus (a region of the brain involved in the regulation of emotional responses) have been linked to suicidal behavior in adolescents (Goodman et al., 2011). Specifically, in a structural imaging study of healthy adolescents and adolescents with BPD and co-occurring major depression, Goodman et al. (2011) found that smaller volume of the Brodmann area of the anterior cingulate gyrus was associated with greater number of suicide attempts.

The Relation Between Emotion Dysregulation and SIB Among Adolescents with BPD

Although preliminary, similar findings of an association between emotion dysregulation and SIB have also been reported among adolescents with BPD pathology. For example, in the aforementioned study of coping strategies among late adolescents with and without DSH (Cawood & Huprich, 2011), not only did adolescents with DSH report higher levels of BPD symptoms than those without DSH, also the association between BPD symptoms and DSH in this sample was partially mediated by the greater use of maladaptive coping strategies and lower use of adaptive coping strategies. Moreover, in a sample of late adolescents and young adults with a history of DSH ($N = 205$, average age = 18.5), elevated levels of BPD symptoms were found among the group of self-harming individuals who endorsed emotion regulatory functions of DSH almost exclusively (Klonsky & Olino, 2008). Finally, although not a direct index of emotion dysregulation per se, findings of a positive association between pituitary gland volume (an indicator of hyperactivity and dysfunction of the hypothalamic–pituitary–adrenal (HPA) axis) and lifetime frequency of SIB among adolescents with BPD (Jovev et al., 2008) provide evidence for dysregulated stress responding more broadly in SIB among adolescents with BPD.

Research on the Relation Between Impulsivity and Self-Injurious Behaviors in Adolescents

Consistent with the theorized relevance of trait impulsivity to SIB in adolescents (Crowell et al., 2009), empirical research within both clinical and community settings provides some support for an association between impulsivity and SIB among adolescents. Several studies using self-report measures of impulsivity have found an association between SIB and impulsivity among adolescents. For example, in a community sample of youth aged 10–13, those with suicidal ideation and/or SIB reported greater impulsivity on two self-report measures than an age-, gender-, and race/ethnicity-matched comparison group of youth without suicidal ideation or SIB (Giannetta et al., 2012). Moreover, in a large community sample of Chinese adolescents aged 11–19 ($N = 6{,}374$), a measure of behavioral impulsivity (operationalized as the frequency of impulsive behaviors in general, excluding any form of SIB) was associated with both the presence and severity of DSH, distinguishing between adolescents with and without a history of DSH, as well as between self-harming adolescents with mild and severe DSH (You, Leung, Fu, & Lai, 2011). In addition, although three core features of BPD (i.e., affective instability, disturbed relationships, and behavioral impulsivity) were prospective predictors of the presence of DSH 1 year later, only behavioral impulsivity emerged as a unique prospective predictor of DSH frequency, accounting for unique variance in DSH frequency in the 1-year follow-up (above and beyond the other core BPD features; You et al., 2012).

Finally, evidence has been provided for an association between self-reported impulsivity

and suicidal behaviors, with Kingsbury, Hawton, Steinhardt, and James (1999) finding higher impulsivity on the Impulsivity Control Scale (ICS; Plutchik & van Praag, 1989) among adolescents with suicidal behaviors, compared to psychiatric controls (even when controlling for depression symptoms). Notably, however, other studies using the ICS within adolescent psychiatric patient samples have failed to provide a strong support for an association between impulsivity and suicidal behaviors among adolescents. For example, Zaitsoff and Grilo (2010) found that differences on the ICS between adolescent inpatients with and without a history of suicidal behavior did not remain significant when controlling for depression, and Wetzler et al. (1996) found no significant differences on the ICS between adolescent outpatients with and without a history of suicidal behavior.

Evidence for an association between SIB in general and impulsivity has also been provided by studies using behavioral and physiological measures of impulsivity. For example, adolescents with a history of repeated SIB have been found to demonstrate (a) lower resting electrodermal responding (a reliable biomarker of trait impulsivity) than both healthy and depressed adolescents without a history of SIB (Crowell et al., 2012), and (b) lower levels of peripheral serotonin (a biological measure of impulsivity) than age-matched healthy controls (Crowell et al., 2005). Moreover, and consistent with other research suggesting greater difficulties among adolescents with both DSH and suicidal behavior (Guertin et al., 2001; Jacobson et al., 2008; Nock et al., 2006), Dougherty et al. (2009) found that adolescents with both DSH and suicidal behaviors evidenced greater impulsivity on both self-report and behavioral measures than adolescents with DSH only.

Finally, emerging research suggests that impulsivity may be associated with SIB particularly among adolescents with BPD. Specifically, in a study comparing the factors associated with SIB among adolescent inpatients with BPD versus major depressive disorder (MDD), Horesh, Orbach, Gothelf, Efrati, and Apter (2003) found that the association between impulsivity and SIB differed as a function of diagnostic status, with ICS scores positively associated with the presence and severity of SIB among adolescents with BPD but not those with MDD. Although not conclusive, these findings suggest that impulsivity may be a particularly important vulnerability factor for SIB among adolescents with BPD, with other vulnerability factors emerging as more relevant to SIB among adolescents with other forms of psychopathology.

Future Directions and Clinical Implications

The advancement of developmental models of the pathogenesis of SIB in BPD (Crowell et al., 2009) in recent years has paved the way for research on the mechanisms underlying SIB in adolescents with and without BPD, improving our understanding of the factors that increase risk for these behaviors. Yet, despite increasing evidence that emotion dysregulation and impulsivity are associated with both SIB and BPD features in adolescents (Crowell et al., 2012; Gratz et al., 2009; Gratz, Latzman, Tull, Reynolds, & Lejuez, 2011; Sharp et al., 2012), little is known about the factors that distinguish youth with BPD who engage in SIB from those who do not. Given that the presence of SIB is associated with a range of negative consequences (e.g., Hawton, Kingsbury et al., 1999; Hawton et al., 2002; Jacobson & Gould, 2007), including heightened risk for suicide (Asarnow et al., 2011; O'Connor & Sheehy, 2000; Sapyta et al., 2012; Shafii et al., 1985), knowing the factors that distinguish between BPD youth with and without co-occurring SIB has important public health and clinical implications. Future research examining the factors that predict co-occurring SIB among adolescents with BPD is needed, as are longitudinal studies examining the risk factors for the development of SIB in particular, versus other maladaptive behaviors that serve a similar emotion-regulating function (e.g., substance abuse, risky sexual behaviors, and disordered eating behaviors; see Gratz & Tull, 2010; Safer, Telch, & Chen, 2009; Tull, Weiss, Adams, & Gratz, 2012). Two factors that may be important to consider in this regard are negative beliefs

about the self and body disregard (see Bjarehed & Lundh, 2008; Chapman et al., 2006; Muehlenkamp & Brausch, 2012), both of which may explain why some adolescents with BPD choose to regulate their emotions through SIB.

Likewise, further research is needed to elucidate differences between self-injuring youth with and without BPD. Although evidence for the moderating role of BPD pathology in the associations between DSH and its putative underlying mechanisms (including aspects of emotion dysregulation and inexpressivity) has been provided within a young adult sample (Gratz, Breetz, & Tull, 2010), the extent to which BPD moderates the associations between SIB and other factors among adolescents remains relatively unexplored. Research is also needed to examine the moderating role of gender in the interrelations of SIB, BPD, emotion dysregulation, and impulsivity among youth. Gender-based multifinality is a central component of developmental models of BPD (Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, 2009; Crowell et al., 2009), and has been proposed to explain the greater risk for BPD observed in adolescent girls versus boys. Likewise, there is some support for gender-based equifinality in the development of SIB in adolescents and young adults, as the emotion regulation-related factors associated with both SIB in general and DSH in particular have been found to differ across gender (Gratz et al., 2011; Kirchner et al., 2011). Research examining gender-based pathways to SIB may elucidate gender-specific risk factors for SIB, as well as identify additional risk factors for DSH and suicidal behaviors that have been overlooked in models developed on the basis of theoretical and empirical literature on females.

Finally, research is needed to examine the different correlates of suicidal versus DSH behaviors among youth with BPD. Little research has examined differences in the functions of and risk factors for these behaviors within adolescent populations, or the factors that predict their co-occurrence. Such research will be crucial in elucidating how best to intervene in these behaviors, as well as identifying youth with BPD who may be most at risk for completed suicide.

As research continues to examine the mechanisms underlying SIB in youth with BPD, the inclusion of multimodal assessments of the constructs of interest will be imperative. With a few notable exceptions (Crowell et al., 2005, 2012; Dougherty et al., 2009; Goodman et al., 2011), the majority of the research in this area has relied exclusively on self-report measures of emotion and behavioral (impulse) regulation, responses to which may be influenced by an individual's willingness and/or ability to report accurately on his or her experiences. Indeed, given that youth with BPD have likely encountered numerous obstacles to the development of adaptive emotion regulation (e.g., invalidating or abusive environments, physical and/or emotional neglect, high rates of family psychopathology; Beauchaine et al., 2009; Goldman, D'Angelo, & DeMaso, 1993; Horesh, Ratner, Laor, & Toren, 2008; Johnson, Smailes, Cohen, Brown, & Bernstein, 2000; Venta, Kenkel-Mikelonis, & Sharp, 2012), they are likely to experience deficits in emotional awareness and clarity (limiting their ability to accurately report on their internal states). Consequently, future studies would benefit from a more expanded use of behavioral, biological, and/or psychophysiological measures of emotion dysregulation and impulsivity. For example, the Behavioral Indicator of Resilience to Distress (BIRD; Daughters et al., 2009) is a behavioral measure of one aspect of emotion dysregulation (i.e., distress intolerance) that has been found to be associated with internalizing and externalizing behaviors among male and female adolescents (Daughters et al., 2009). Likewise, the Youth Version of the Balloon Analogue Risk Task (BART-Y; Lejuez et al., 2007), a behavioral measure of risk-taking propensity, has demonstrated associations with a wide range of impulsive and risky behaviors (e.g., risky sexual behavior, substance abuse, involvement in physical fights) among adolescents.

Theoretical and empirical literature emphasizing the role of emotion dysregulation and impulsivity in SIB among youth with BPD

have important implications for the selection, refinement, and development of effective interventions for these behaviors. Preliminary research provides support for the utility of several treatment approaches for SIB among youth in general (for reviews, see Robinson, Hetrick, & Martin, 2011 and Washburn et al., 2012)—most of which target emotion dysregulation and/or impulsivity in some manner (e.g., manualized cognitive-behavioral therapy package for adolescent self-harm (Taylor et al., 2011), cognitive-behavioral therapy intervention (Slee, Garnefski, van der Leeden, Arensman, & Spinhoven, 2008), and developmental group psychotherapy (Wood, Trainor, Rothwell, Moore, & Harrington, 2001; although see Green et al., 2011 and Hazell et al., 2009 for conflicting results)). Yet, despite evidence suggesting the potential utility of these treatments for adolescent SIB in general, there is currently no evidence for the utility of these treatments among youth with BPD in particular. However, research on the treatment of SIB in adults has found that short-term treatments for SIB in general (not specific to BPD) are not effective for patients with BPD, and may lead to an increase in the repetition of SIB among individuals with BPD (Tyrer et al., 2004). Thus, given that youth with BPD are at heightened risk for SIB, it is crucial to pinpoint treatments for SIB that are effective specifically within this population.

One promising treatment in this regard is mentalization-based treatment (MBT), a psychoanalytic treatment that focuses on the enhancement of mentalization (i.e., the ability to understand and reflect upon one's own and other's internal states and their relationship to behaviors; Bateman & Fonagy, 2004). Found to be efficacious in the treatment of SIB among adults with BPD (Bateman & Fonagy, 1999), a modified version of this treatment for adolescents (MBT-A) has recently been examined within a sample of adolescents with recent SIB and depression (many of whom also met criteria for BPD). Results of a randomized controlled trial revealed positive effects of MBT-A on SIB and depression symptoms (Rossouw & Fonagy, 2012). Notably, although MBT does not target emotion dysregulation or impulsivity directly, the enhancement of mentalization is theorized to increase emotion regulation and behavioral control and, as such, may indirectly target both of these mechanisms.

Finally, one treatment that may be particularly useful in the treatment of SIB among youth with BPD is dialectical behavior therapy for adolescents (DBT-A; Rathus & Miller, 2002), a 16-week behavioral treatment that involves weekly individual therapy and multifamily skills groups, as well as family therapy as needed. Consistent with DBT for adult women with SIB (Linehan, 1993), DBT-A teaches adolescents a number of skills focused on improving emotion regulation and reducing impulsive behaviors. Further, research provides support for the utility of DBT-A in the treatment of both SIB and BPD pathology, as well as their proposed underlying mechanisms (i.e., emotion dysregulation and impulsivity), among adolescent outpatients with subthreshold or threshold BPD (Flesichhaker et al., 2011; Rathus & Miller, 2002; for a review of research on DBT-A, see Klein & Miller, 2011). Although the results of these studies are promising, further research examining the efficacy of DBT-A in the treatment of both SIB and BPD among larger and more diverse groups of youth is needed. Likewise, research is needed to examine the utility of DBT-A or some of its skills training modules (e.g., emotion regulation and distress tolerance skills) in the prevention of SIB among at-risk youth (e.g., see Perepletchikova et al., 2011).

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The Social–Cognitive Basis of BPD: A Theory of Hypermentalizing

15

Carla Sharp

It is broadly acknowledged that BPD is characterized by dysregulation in four domains: emotion (e.g., anger, affective instability), interpersonal (e.g., unstable relationships and abandonment fears), cognitive (e.g., dissociation), and behavioral (impulsivity, self-harm). While conceptualizations of BPD vary in terms of the weight placed on the interpersonal aspects of borderline psychopathology, most approaches acknowledge the interpersonal context, nature, or sequelae of BPD. To explain the interpersonal nature of BPD, researchers have examined its social–cognitive basis. While this research historically lagged behind research investigating dysregulation of mood and impulse control, there has been an explosion of research examining the social–cognitive basis of BPD over the last decade as exemplified by recent special issues of personality disorder journals dedicated to this topic (e.g., Sharp & Sieswerda, 2013).

The aim of the chapter is to review and discuss this literature with the ultimate goal of providing an integrated framework for theory and research. I begin with a description of the behavioral phenotype of disrupted interpersonal relationships in BPD, especially in the context of adolescence, which justifies a social–cognitive approach to BPD. Next, the ever-expanding empirical support for the social–cognitive basis of interpersonal disruptions in BPD in adults and adolescents is discussed. Acknowledging the multicomponent nature of the construct of social cognition and reflecting the three major developmental theories of BPD (Linehan's biosocial theory, Fonagy's mentalization-based theory, and attachment theory), this literature is organized by reference to the three social–cognitive constructs most often studied in relation to BPD: emotion recognition, mentalizing (or theory of mind), and trust. After reviewing empirical evidence in support of the relation of these constructs to BPD features, I present a possible resolution to understand and explain inconsistencies among findings by suggesting a recursive social information processing model culminating in hypermentalizing in BPD. As such, it is hoped that a hypermentalizing theory of BPD will provide a framework for future research in the social cognition of BPD by integrating the biosocial, mentalizing, and attachment approaches to BPD.

The Behavioral Phenotype of Disrupted Interpersonal Relationships in BPD

Popular psychology trade books with titles such as "Stop walking on eggshells: Taking your life back when someone you care about has borderline personality disorder" and "I hate you—don't leave me: Understanding borderline personality disorder" captures the stereotypical view of the

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interpersonal nature of BPD. It is true that a major feature of BPD is difficulties with interpersonal relationships. Research has shown that adults with BPD experience a greater number of breakups and conflicts in romantic relationships (Labonte & Paris, 1993). Data from the Collaborative Longitudinal Personality Disorders Study have also shown that patients with BPD (compared to other personality disorders) have significantly more impairment in social relationships as indicated by increased frequencies of conflicts with parents, friends, and siblings (Skodol et al., 2002). Research has also shown that couples in which one partner meets criteria for BPD show lower marital satisfaction, higher attachment insecurity, more demand/withdraw communication problems, and higher levels of violence (Bouchard & Sabourin, 2009; Bouchard, Sabourin, Lussier, & Villeneuve, 2009).

This pattern of results has also been found for children and adolescents with borderline features. For instance, Daley, Burge, and Hammen (2000) have shown that adolescents with BPD experience a greater number of breakups and conflicts in romantic relationships. The Children in the Community Study also showed that adolescent BPD assessed at mean age 16 was associated with elevated partner conflict during the transition to adulthood (i.e., age 17–27) (Chen et al., 2004) and lower levels of intimacy (Crawford, Cohen, Johnson, Sneed, & Brook, 2004). Recently, we have also demonstrated an association between teen dating violence and rates of BPD features in adolescence (Reuter, Sharp, & Temple, 2014). In children, Crick, Murray-Close, and Woods (2005) demonstrated a relation between borderline features and tendencies for hostile attributional biases and intense emotional reactions during ambiguous peer scenarios, in addition to enmeshed relationships with best friends, and relational and physical aggression.

In summary, research shows that disrupted interpersonal relationships are a hallmark feature of BPD in adults, children, and adolescents. This behavioral phenotype is represented in seven of the nine criteria of the DSM-IV-TR (American Psychiatric Association, 2000), which requires that five of nine criteria are met in order for a diagnosis of BPD to be made. Two criteria explicitly cover problems in interpersonal relationships: criteria #1 (frantic efforts to avoid real or imagined abandonment) and #2 (a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluations). However, in the discussion of diagnostic features associated with each DSM criterion (American Psychiatric Association, 2000, p. 707) the interpersonal nature of most other criteria is clearly evident. For example, criterion #3 describes how identity disturbance manifests itself most often in situations in which an individual feels a lack of meaningful relationships, nurturing, and support. The impulsivity criterion (#4) includes unsafe sex and anger outbursts in the context of relationships. History of self-harm/suicide attempts (criterion #5) are described to be often precipitated by threats of separation or rejections. Reactivity of mood or affective instability (criterion #6) is said to often reflect the individual's extreme reactivity to interpersonal stresses and criterion #8 (anger) is described as often elicited when a caregiver or lover is seen as neglectful, withholding, uncaring, or abandoning.

A recent interview with a 14-year-old girl admitted as an inpatient to a psychiatric hospital illustrates how these symptoms manifest in the lives of adolescents with BPD. In explaining how and why she had been admitted to the hospital, the girl shared that she had been in an argument on the telephone with her boyfriend because he had chosen to go out with his friends to a party rather than visit her. The couple argued about this and her boyfriend refused to leave the party. The boyfriend ended the conversation and hung up the phone. The girl was so upset about this that she called and texted him dozens of times immediately after that. He did not respond to any of these attempts at communication which only upset the girl more. In a moment of overwhelming emotion, she stole her mother's car and decided she would drive to the party to find her boyfriend. She called many times from the car as

she pulled onto the highway and, in one of these calls, left a message saying that she would kill herself if he wasn't at the party when she arrived. The girl, too young to be driving her mother's car, got into an accident on the highway. She was unable to control her emotions when the police arrived. She was convinced that her boyfriend intended to break up with her and said that she would kill herself if she wasn't able to talk with him right away. The police arrested the girl and brought her to an inpatient unit where she was interviewed by our staff.

Given the centrality of interpersonal disturbance in BPD, it naturally follows that theoreticians and researchers have looked to social cognition to explain these disruptions. Social cognition refers to the mental processes involved in perceiving, attending to, remembering, thinking about, and making sense of the people in our social world (Moskowitz, 2005), or the ability to understand ourselves and others as individuals with beliefs, feelings, and personality (Mitchell, Macrae, & Banaji, 2004). Over the last decade, research examining the biases, impairments, and deficits associated with BPD have dramatically increased. This literature is reviewed below by selectively focusing on the emotion recognition, mentalizing (theory of mind), and trust. Other social–cognitive constructs examined in the context of BPD include emotional intelligence (Gardner & Qualter, 2009; Leible & Snell, 2004), alexithymia (Lemche, Klann-Delius, Koch, & Joraschky, 2004), teasing (Tragesser, Lippman, Trull, & Barrett, 2008), metacognitive capacity (Semerari et al., 2005), social exclusion (Ruocco et al., 2010; Staebler et al., 2011), and a range of cognitive biases such as dichotomous thinking that are applied to social stimuli, but are not in themselves social variables (Arntz, Appels, & Sieswerda, 2000; Arntz & Veen, 2001; Baer, Peters, Eisenlohr-Moul, Geiger, & Sauer, 2012; Veen & Arntz, 2000) These are not discussed here, but readers are referred to the recent special issue on social cognition and personality disorder (Sharp & Sieswerda, 2013) for coverage of these constructs.

Emotion Recognition in BPD

Linehan's Biosocial Theory

In Linehan's (1993) biosocial theory, she argues that the interpersonal problems associated with BPD mainly arise from impaired emotion regulation. Specifically, borderline patients have been described as highly vigilant for social stimuli, social rejection, and social threat. The accurate inference of the mental states of others from external cues such as the face (emotion recognition) is essential for guiding and regulating behavior in social situations (Domes, Schulze, & Herpertz, 2009a). It is therefore not surprising that, of all social–cognitive constructs, emotion recognition in BPD has the most mature literature base.

Alterations in Emotion Recognition

Several studies have demonstrated alterations in emotion recognition in BPD especially for expressions of intense negative emotions such as anger, disgust, and fear in forced-choice studies (Bland, Williams, Scharer, & Manning, 2004; Levine, Marziali, & Hood, 1997; Meyer, Pilkonis, & Beevers, 2004), studies eliciting verbal descriptions of others' emotional states (Wagner & Linehan, 1999), studies investigating error patterns in addition to success in facial recognition (Unoka, Fogd, Fuzy, & Csukly, 2011), paradigms using technology to electronically morph facial affect from a neutral expression to basic emotional expressions with increasing intensity (Domes et al., 2008), studies with timed paradigms (Dyck et al., 2009), and multimodal studies that require integration of visual and auditory information (Minzenberg, Poole, & Vinogradov, 2006b).

Alterations in emotion recognition have also been demonstrated for borderline traits in adolescent samples. von Ceumern-Lindenstjerna et al. (2010) demonstrated a correlation between current mood and attentional bias to negative faces,

suggesting an inability to disengage attention from negative facial expressions during attentional maintenance when in negative mood. Using a face-morphing task, Robin et al. (2012) demonstrated no impairment in BPD adolescents in fully expressed emotions. However, borderline adolescents were slower at identifying change for both anger and happiness compared to healthy controls, suggesting that the impairment in BPD is associated with subtle impairments at lower levels of intensity of facial expression.

Enhanced Emotion Recognition

Several studies have found enhanced emotion recognition in BPD. Lynch et al. (2006) used morphing technology and demonstrated an enhanced capacity in BPD patients to correctly classify facial emotions at a lower level of intensity. Domes et al. (2008) demonstrated enhanced learning over the course of their morphing experiment in BPD patients so that borderline patients showed a reduction in detection threshold over the course of the experiment whereas the control group did not. Two earlier studies also found increased accuracy in identifying the emotional content of videotaped vignettes as either positive or negative (Frank & Hoffman, 1986) and increased levels of empathy (Ladisich & Feil, 1988) in borderline patients. In a much cited study, Fertuck et al. (2009) showed that mental state discrimination based on the eye region of the face (emotion recognition) was enhanced in BPD. Similarly, Franzen et al. (2011) showed that borderline patients were as good as non-patients in using facial expression to guide decision making in the context of a trust task. Several other studies have demonstrated no differences for emotion recognition capacities between BPD patients and healthy controls, both in adults (Frick et al., 2012; Minzenberg, Poole & Vinogradov, 2006a) and in adolescents (Jovev et al., 2011).

Conclusions

Two main conclusions can be drawn from these studies. First, authors have suggested that borderline patients do not show a general deficit in emotion recognition, but rather a "negativity bias" manifested as hyper-responsiveness (hypersensitivity) to negative emotions like anger and fear. This bias may not be specific to social–emotional stimuli as several studies (see von Ceumern-Lindenstjerna et al., 2010) have demonstrated negative biases in borderline patients for non-social stimuli. Therefore, it may be that the negative bias for social stimuli discussed here is part of this general bias toward negative emotion. However, these biases may not be specific to BPD (not all studies control for depression and other comorbidities), and not all studies have been able to show a negativity bias in emotion recognition (e.g., Arntz et al., 2000; Frick et al., 2012). Nevertheless, the proposed hypervigilence for negative emotion (or emotion in general according to Frick et al., 2012) is thought to associate with reduced amygdala volume and enhanced amygdala responding to emotional stimuli such as negative facial expressions, coupled with regulatory deficits of the orbital and prefrontal cortices (Domes et al., 2009a; Frick et al., 2012). Indeed, three neuroimaging studies that explicitly investigated neural responses to emotion recognition in BPD have confirmed this hypothesis. Donegan et al. (2003) showed that borderline patients demonstrated significantly greater left amygdala activation to the facial expressions of emotion (vs. a fixation point) compared with normal control subjects. Minzenberg, Fan, New, Tang, and Siever (2007) found that borderline patients exhibited changes in fronto-limbic activity in the processing of fear stimuli, with exaggerated amygdala response and impaired emotion-modulation of ACC activity. Similarly, Frick et al. (2012) demonstrated stronger activation of the amygdala in response to affective pictures regardless of valence, compared to healthy controls.

Second, it appears that more complex emotion recognition tasks more consistently distinguish BPD from non-BPD groups. For instance, in the Minzenberg et al. (2006b)

study, where facial, prosodic (the aspect of speech that communicates meaning by variation in stress and pitch independent of lexical and syntactic content), and integrated facial/prosodic stimuli were used, borderline patients showed no problems with isolated facial of prosodic emotion, but instead demonstrated deficits in higher order integration of social information. Similarly, Dyck et al. (2009) investigated the ability of individuals with BPD to recognize negative and neutral emotions, in both timed and untimed trials. They found that individuals with BPD were significantly impaired in their recognition when the task was timed. However, no such difficulty was noted when the participants were not timed. Thus, the participants with BPD were significantly impaired when under time pressure and were less able to correctly judge negative or neutral affect in a hasty manner. It is possible therefore that borderline patients have emotion recognition deficits when tasks require the integration of different modes of processing (emotion recognition and speed of response), or when tasks are presented in the context of heightened emotional arousal (Dixon-Gordon, Chapman, Lovasz, & Walters, 2011).

Theory of Mind/Mentalizing in BPD

Fonagy's Mentalization-Based Theory

Another prerequisite for optimal interpersonal functioning is the capacity to take the intentions, emotions, and beliefs of others into account during social interactions. This capacity is referred to as theory of mind (ToM) (Premack & Woodruff, 1978) or mentalizing (Fonagy, 1991; Frith, 1989). Often, the term mentalizing is used interchangeably with social cognition (Sharp, Fonagy & Allen, 2012) and therefore serves as an umbrella term for other social–cognitive constructs including emotion recognition or trust. For the purposes of this section, however, I will define mentalizing strictly as ToM. Accordingly, only studies that explicitly made use of ToM paradigms will be reviewed in this section.

The mentalization-based theory of BPD was proposed by Fonagy and colleagues (Fonagy, 1989, 1991; Fonagy, Gergely, Jurist, & Target, 2002; Fonagy & Luyten, 2009b; Sharp & Fonagy, 2008a, 2008b) and posits that a vulnerability to failures or misinterpretations of actions in terms of underpinning mental states may account for core features of BPD. In particular, Fonagy and colleagues have argued that as the child's attachment relationships have an important role to play in the acquisition of social–cognitive capacities, disruptions of early attachment experiences can derail social–cognitive (metalizing) development (see Fonagy & Luyten, 2009a for a comprehensive description of this developmental framework for the development of BPD). As with emotion recognition studies of BPD, the evidence is mixed regarding the presence of impairments or deficits in mentalizing.

Evidence for Mentalizing Deficits in BPD

Harari, Shamay-Tsoory, Ravid, and Levkovitz (2010) assessed cognitive and affective ToM in patients with BPD and healthy controls. Using the Faux Pas task (Baron Cohen, Jolliffe, Mortimore, & Robertson, 1997) alongside an assessment of empathy, they demonstrated impairment in cognitive ToM and empathy, but not affective ToM and empathy in BPD patients. Impairment in ToM was also demonstrated by Preissler, Dziobek, Ritter, Heekeren, and Roepke (2010) who used the Movie Assessment of Social Cognition (MASC) (Dziobek et al., 2006), which is a more complex and ecologically valid ToM task. They showed that borderline female adults with BPD, compared with healthy controls, showed impaired abilities on items assessing emotions, thoughts, and intentions of movie characters.

Evidence Against Mentalizing Deficits in BPD

In contrast to studies showing a mentalizing deficit in BPD, other studies have failed to demonstrate a deficit per se. For instance, Arntz, Bernstein, Oorschot, and Schobre (2009), using Happé's (Happé, 1994) Advanced Test of ToM (inferring other participants' thoughts, feelings, and intentions in complex social situations that involve double bluff, mistakes, persuasion, and white lie), found no evidence for deficits in ToM capacities. In fact, borderline patients performed better than non-patients.

Using Baron-Cohen's Eyes Test (Baron Cohen, Wheelwright, Hill, Raste, & Plumb, 2001), a measure originally described as a ToM task, Schilling et al. (2012) also found no impairment in ToM for borderline patients, similar to findings of (Fertuck et al., 2009). Interestingly, borderline patients did report higher confidence in their decisions during the task compared to healthy controls, reflecting a potential rigidity (instead of deficit) in the social–cognitive style of borderline patients.

Ghiassi, Dimaggio, and Brune (2010) used a test of cognitive mentalizing skills in which scenes of cartoon picture stories about social interactions had to be sorted and questions about mental state reasoning answered and found comparable performance in BPD and healthy controls. The so-called "superiority" in ToM was suggested by Franzen et al. (2011) who made use of a simulated interaction of a multi-round trust task with several virtual partners to compare ToM in borderline patients with healthy controls. The fairness of the interaction partners as well as the emotional facial expression that allowed subjects to infer the partner's intention within an individual exchange round was manipulated. Results showed that both borderline patients and non-patients made use of emotional expressions of partners to guide their decision-making to invest in their partners. However, borderline patients were able to ignore a behavior-incongruent facial expression when offers were low. In some ways then, borderline patients were better at reading the true intentions of their partners in the games while the non-patients were "fooled" by offers that did not correspond to partners' facial expressions.

Conclusions

While the Franzen study did show some social–cognitive impairment in borderline patients (see next section on Trust), BPD superiority in ToM echoes some of the enhanced emotion recognition capacities discussed in the previous section. Several possible reasons have been offered for these theory-incongruent findings (Sharp et al., 2013). First, it is possible that deficient mentalizing is only apparent under conditions of high arousal (Dixon-Gordon et al., 2011; Fonagy & Luyten, 2009a). As complexity of tasks increase (for example in the Preissler et al., 2010) study, and more emotional demands are placed on processing, mentalizing therefore may begin to fall apart in borderline patients. In an innovative study Dixon-Gordon et al. (2011) demonstrated this notion clearly. Negative emotion (through social rejection) was induced in college students after which a social problem-solving task was administered. While this study requires replication in clinical samples, results demonstrated that those with high BPD traits had trouble generating relevant solutions to social problems, and increases in negative emotions during the mood induction mediated the relation between borderline features and reductions in social problem-solving performance.

Second, as proposed by Fonagy and Luyten (2009a), it is possible that deficient mentalizing is a consequence of lack of integration between social–cognitive systems that subserve implicit, unreflective mentalizing (lower level automatic processing) vs. systems that subserve more reflective thought (higher order cognitive processes) (Lieberman, 2007). This notion would explain the negative findings using Happé's task as pointing to an inability of Happé's task to

distinguish between these subsystems, and is consistent with Harari et al. (2010)'s findings that borderline subjects had no trouble with affective automatic responses, but struggled with cognitive empathy and ToM that requires higher-order processing. Franzen et al. (2011) also interpreted the ToM "superiority" in their trust task to point to the possibility that individuals with BPD make use of explicit-controlled processing when mindreading while healthy controls use automatic processing to guide their decision making. This may reflect a cognitive processing issue as suggested by Dyck et al. (2009) and discussed earlier, or may reflect the result of a learning history that taught these individuals to evaluate social interaction partners more carefully without relying on the first automatic judgment. Interpreted in this way, superiority is not really superiority, but enhanced mentalizing used inappropriately that deviates from normative behavior.

A third possibility is that many of the tasks that are associated with negative findings (like Happé's task as well as the Eyes Test), are simply too far removed from real-life social interactions. When tasks are used that more closely approximate real-life interaction, a clearer deficit or impairment related to BPD emerges. The fact that Franzen study did not elicit social–cognitive deficits suggests that it cannot be the mere ecological validity of a task, but points to a fourth possibility—perhaps the most parsimonious of all.

It is also possible that BPD is in fact not associated with deficits (i.e., lack of) in mentalizing at all, but represents *altered mentalizing*. Consistent with the latter view, Sharp et al. (2011) recently used the Movie Assessment of Social Cognition (Dziobek et al., 2006) in adolescents with borderline traits to demonstrate that hypermentalizing (excessive theory of mind) uniquely associated with borderline traits as opposed to the "no mentalizing" or" "less mentalizing" subscales of the MASC. Hypermentalizing, also referred to as excessive ToM (Dziobek et al., 2006), can be defined as a social–cognitive process that involves making assumptions about other people's mental states that go so far beyond observable data that the average observer will struggle to see how they are justified (Sharp, Ha, et al., 2012), due to confusion between self- and other mental states. As such, it involves overattribution of mental states to others and their likely misinterpretation. For example (Sharp, Ha, et al., 2012), person A invites person B to dinner, but B replies hurriedly that she is unavailable because she has a prior engagement. A then assumes that B does not want to spend time with her because of a minor incident of misunderstanding that she recalls from several years ago, where A did not turn up for B's birthday party. A then generates a complex narrative about B's "overreaction" and her apparent "inability to forgive." This is referred to as hypermentalizing because A was using mental states to explain B's actions, but overattributed mental states that were unlikely to be real, and more reflective of A's own mental states at the time of the original misunderstanding. The fact that the hypermentalizing subscale is *the only* type of mentalizing to be associated with BPD features when considered alongside undermentalizing and no mentalizing in the same sample makes a strong case for hypermentalizing as the most likely social–cognitive correlate of BPD. I return to these findings in the final part of the chapter where I present an integrated model of social cognition for BPD with hypermentalizing as the final output in a series of recursive social information processing steps.

Trust

The Attachment Theory of BPD

Insecure attachment has long been described as an important etiological factor for the development of borderline pathology (Gunderson, 1984; Gunderson, 1996; Gunderson & Singer, 1975; Kernberg, 1967). Empirical evidence has supported the link between insecure attachment and BPD cross-sectionally and retrospectively in

adults (see Levy, 2005; Levy, Meehan, Weber, Reynoso, & Clarkin, 2005; Sharp & Fonagy, 2008a, 2008b for a review). In addition, three prospective longitudinal studies have shown that attachment disturbance in infancy and adolescence predicted BPD symptoms in adulthood (Bezirganian, Cohen & Brook, 1993; Carlson, Egeland & Sroufe, 2009; Lyons-Ruth, 2008). Attachment, as defined by Bowlby (1973, 1980), refers to the preparedness of the infant to seek protection from attachment figures, coupled with the attachment figures' natural disposition to provide care. This reciprocity creates an enduring bond between caregiver and infant and lays the foundation for the experience of trust in relationships.

Anomalies in Trust in BPD

In recent years, trust has been innovatively operationalized within a behavioral or neuroeconomics framework to study disruptions in interpersonal relationships associated with psychopathology (see Sharp, 2012; Sharp, Monterosso, & Montague, 2012 for reviews). In this context, trust is defined as an exchange between two players in which cooperation and defection can be parametrically encoded as the amount of money designated for the partner. The basic one-shot trust task was initially proposed by (Camerer & Weigelt, 1988) and further developed by (Berg, Dickhaut, & McCabe, 1995). One player (the Investor) is endowed with a certain amount of money (or points as proxies for money). The Investor can keep all the money or decide to "'invest"' some amount with the partner (the Trustee). The amount invested is tripled in value as it is sent to the Trustee, who then decides what portion to return to the Investor.

King-Casas et al. (2008) used the iterated version of the trust task to examine trust in adults with BPD. The game was played ten times over, with total points earned displayed to both parties at the end of the game. Results showed that when cooperation began to falter in the iterated exchange, normal controls responded with increased hemodynamic activity in the anterior insular cortex, and this neural response preceded an attempt to coax back cooperation from their partner by signaling increased trust. In contrast, a relative insensitivity of the insula was observed in patients with BPD which was associated with a failure to coax back partners into the game. Similarly, Unoka, Seres, Aspan, Bodi, and Keri (2009) showed that decreased trust was specific to borderline (compared to depressed) patients and in a follow-up study, demonstrated mistrust to be specific to situations where *social* risk-taking is relevant (as opposed to risk-taking in general).

Franzen et al. (2011) found no evidence of deviations in perception of social norms in their study using the trust task. BPD patients assessed the trustees' fairness similar to non-patients. They were able to integrate these evaluations of actual behavior into a generalized image of the social partner. However, they did show alterations in the assessment of *their own* interaction behavior in that unfair behavior of the social partner influenced borderline patients, but not healthy controls. In particular, with the lack of emotional cues, borderline subjects judged their own behavior as more unfair than non-patients. The authors used Young's schema mode of punitive parent to interpret this finding. However, this may also be interpreted as a sign of merging of self and other so that when presented with an ambiguous other, borderline patients assume the identity of the other (in this case being unfair). A similar finding was demonstrated by Frick et al. (2012) in the context of an emotion recognition paradigm while using fMRI. Patients with BPD showed superiority in recognition of facial expressions, but this was associated with increased amygdala and medial frontal activation while healthy controls showed greater activation in the insula and superior temporal gyri, suggesting overactive and exaggerated resonance with the other's' mental states in BPD with weaker top-down modulation.

Putting it all Together: A Theory of Hypermentalizing

How then should the mixed findings for emotion recognition, mentalizing, and trust be integrated? In the preceding sections, I have reviewed several explanations for the mixed findings—yet, to be integrated into one framework. To recap, four main explanations have been offered: (1) That social–cognitive deficits are apparent only under conditions of high arousal. Therefore, patients with BPD should not demonstrate across the board social–cognitive deficits, but only during tasks that are emotionally loaded. (2) That social–cognitive deficits are apparent only under conditions that require integration across different cognitive modalities. Here, we would expect individuals with BPD to do well in tasks that accesses only one modality (like emotion recognition), but poorly on tasks that require integration of multiple sources of information (like cartoon jokes). (3) That the social–cognitive deficits in BPD reflect a lack of integration between social–cognitive systems that subserve implicit, unreflective mentalizing (lower level automatic processing) vs. systems that subserve more reflective thought (higher order cognitive processes). Therefore, borderline patients might use explicit-controlled process when more automatic processing is required and vice versa. (4) That individuals with BPD do not suffer from deficits per se, but that their social–cognitive style is characterized by overattribution of mental states to other, and confusion or conflation of own mental states with those of the other—thus hypermentalizing.

Here, I put forward the notion that the concept of hypermentalizing incorporates explanations 1–3 by first defining hypermentalizing as the type of mentalizing that occurs under conditions of high arousal associated with enhanced amygdala activation coupled with regulatory deficits of the orbital and prefrontal cortices. By defining hypermentalizing as such, explanation 1 is dealt with. If we then take a *social information processing approach* to how hypermentalizing may come about, explanations 2 and 3 become precursors to the ultimate endpoint of hypermentalizing in a recursive model where this process becomes iterative with escalating emotion dysregulation. This model would explain why, if measures representing only one processing step in the model are included in a study, positive findings for deficits in social–cognitive capacity may ensue. The model would also explain why in some cases enhanced social–cognitive function have been demonstrated: these would be studies where there is an over-reliance on controlled-explicit social–cognitive reasoning, which in isolation would seem superior, but in the context of the full processing sequence are precursors to an outcome of hypermentalizing. Figure 15.1 represents the hypermentalizing theory of BPD linking the empirical findings discussed in preceding sections of the chapter to each processing step.

Turning the above model on its head enables a description of an important treatment target in approaches wishing to incorporate rectification of a hypermentalizing social–cognitive style. An *optimal mentalizer* is someone who maintains executive control over integrated cognitive processing during emotionally intense interpersonal interactions. This allows the individual to move fluidly between automatic-implicit and controlled-explicit social–cognitive processing as demanded by the situation. The optimal mentalizer is therefore able to adaptively modify social–cognitive processing in a contextually appropriate manner that maximizes fitness with environmental demands, thereby reducing errors in interpretation.

The hypermentalizing theory of BPD is in line with recent work in the field of cognitive vulnerability which has focused on integrating different cognitive vulnerability factors into one design, given that it is unlikely that each cognitive vulnerability theory is presenting a distinct etiological pathway leading to the development of psychopathology (Abela & Hankin, 2008). Applied here, a *multiplicative approach* to social–cognitive vulnerabilities suggest that vulnerability factors interact synergistically to

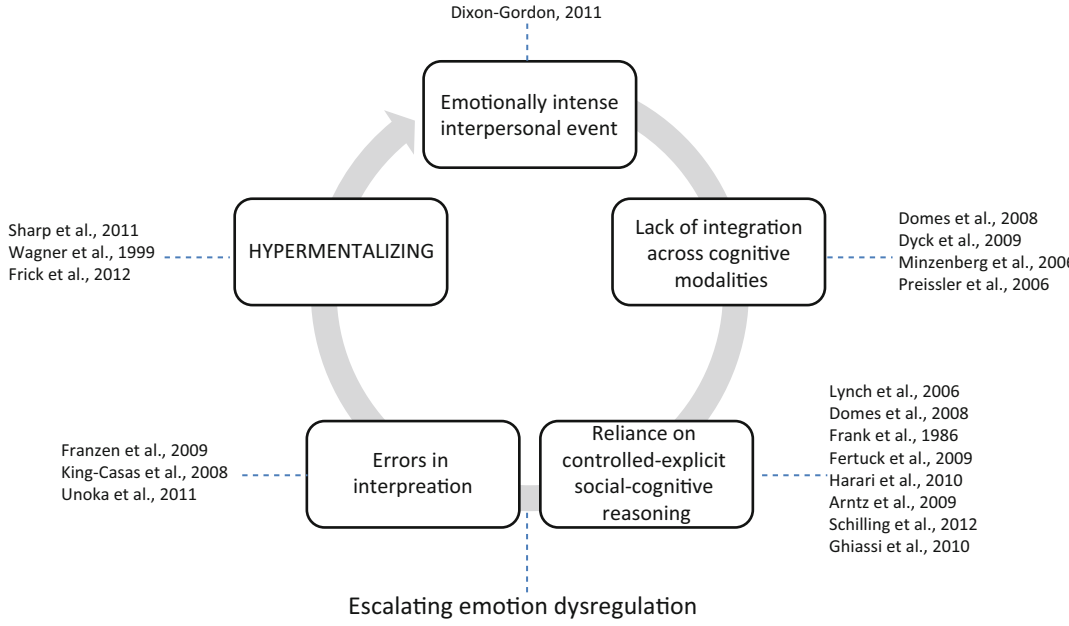

Fig. 15.1 The hypermentalizing theory of BPD

potentiate the interpersonal event-borderline reaction relationship, such that the greatest increases in borderline symptoms following an emotionally intense interpersonal situation will be observed in individuals with multiple social–cognitive vulnerability tendencies. The hypermentalizing theory of BPD also constitutes an explicit attempt to integrate Linehan's biosocial theory of BPD which emphasis emotional arousal and the inability to regulate emotions, with Fonagy's mentalizing theory which emphasizes the social–cognitive basis of BPD.

Future Research: Downward Extension to Adolescence

Because of most of the research linking social–cognitive impairment to BPD has been carried out in adults, little is known about when this relationship emerges, whether and how it changes over developmental time, or whether social–cognitive variables interact with developmental transitions to increase or decrease the risk for BPD. In childhood, early social–cognitive processes are still developing with mentalizing capacity only coming fully on line at age 4. Therefore individual differences in social cognition may be only weakly (if at all) predictive of BPD (although it might be predictive of what might develop into core components of BPD, for instance, studies have linked ToM with executive functioning capacity, which in turn plays an important role in the development of emotion regulation). In adolescence, and early adulthood, when most individuals would have acquired mature social–cognitive capacity, individual differences in these strategies may be more strongly associated with BPD. Those who lag behind in the maturation process may be at particular risk for developing BPD. Here, the interaction with environmental factors like stressful life events, difficult relationships with parents or stressful developmental transitions will increase the risk for BPD.

In incorporating a developmental framework, it will be important to demonstrate continuity across the lifespan in social–cognitive processes. If impaired social cognition represents a vulnerability (or diathesis) for BPD, it would show

some degree of temporal stability. Currently no data exist on the stability of social–cognitive processes, its developmental specificity or the mean level changes across development. It is expected that some social–cognitive processes (e.g., social referencing) would show homotypic continuity across the lifespan, but that others, like ToM may demonstrate heterotypic continuity. It is, for instance, possible that a preschooler in adverse circumstances characterized by insecure attachment relations to primary caregivers may show delayed passing of the false-belief task (as shown by Fonagy, Steele, Moran, Steele, & Higgitt, 1991; Fonagy, Redfern, & Charman, 1997), but by the time she reaches adolescence "undermentalizing" has transformed into hypermentalizing (as shown by Sharp et al., 2011). In this regard, basic research on the developmental course of social–cognitive development is essential.

A final consideration for future research on the social–cognitive basis of BPD especially during development is gender differences. Gender differences in social cognition have been observed in adults (Baron-Cohen, 2003) which raises the obvious question as to when these differs first emerge and whether they can account for the gender differences in the prevalence reported in some studies of BPD. These gender differences may of course be attributable to differential treatment of boys and girls, but more compatible with a hypermentalizing theory of BPD is the biological differences in stress sensitivity.

In all, much progress has been made in elucidating the social–cognitive basis of BPD across development. The next generation of research in this area is likely to be characterized by a strong developmental psychopathology approach that makes use of methods across multiple units of analyses within a developmental design. Also, given that biological systems are unlikely to map onto single areas of dysfunction, the next generation of research will also be characterized by methodologies and constructs that cuts across traditional theoretical divisions in the field.

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Recommended Reading

Domes, G., Schulze, L., & Herpertz, S. C. (2009). Emotion Recognition in Borderline Personality Disorder - a Review of the Literature. *Journal of Personality Disorders, 23*(1), 6–19 [Excellent review of emotion recognition deficits in BPD].

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Part IV

Developmental Course and Psychosocial Correlates

The Longitudinal Course of Borderline Personality Disorder in Youth

16

Amanda Venta, Kathrin Herzhoff, Patricia Cohen, and Carla Sharp

Borderline Personality Disorder Grows Down

Understanding the developmental course of borderline personality disorder (BPD) has only recently become a reasonable goal due, in large part, to the fact that the theoretical evolution and empirical investigation of BPD first focused on adults, with much later and less discussion of the disorder in youth. Indeed, the term "borderline" first appeared in the psychoanalytic literature in the 1930s (Stern, 1938) and it was not until nearly four decades later that discussions of the disorder's childhood roots began to emerge (Adler & Buie, 1979; Masterson, 1972). With regard to assessment, the diagnostic interview for borderline patients (Gunderson & Kolb, 1978; Gunderson, Kolb, & Austin, 1981 as cited by Bradley & Westen, 2005) was first created for adults in the late 1970s and it was not until 25 years later that the first diagnostic interview for BPD in children (Childhood Interview for DSM-IV BPD; Zanarini, 2003) emerged. Diagnosis of BPD followed the same pattern, with the disorder first appearing in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III, American Psychiatric Association, 1980), but not including a provision for diagnosis in youth until the fourth edition in 1994 (DSM-IV, American Psychiatric Association, 1994). In much the same way, empirical research on the course of BPD has been dominated by adult samples, with research supporting the diagnosis in youth and exploring its developmental course published only in the last decade.

This chapter will review much of this work with a focus on studies exploring the developmental course of borderline personality traits across youth and early adulthood. A great deal of this work has come from the Children in the Community Study (CIC; e.g., Cohen, Crawford, Johnson, & Kasen, 2005) and as such, these studies will occupy the majority of the review. Then we will summarize what has been learned from other, more recent, prospective longitudinal studies of BPD with children and adolescents. A summary of findings, limitations, and directions for future research is provided at the end of each review section. The final section will include reflections regarding the clinical implications of this research as well as a summary table of key studies and suggested readings (Table 16.1).

Understanding the developmental course of psychopathology in general, and borderline traits in particular, is important because demonstrating a common course to symptom patterns across individuals is a fundamental criterion for establishing the validity of a disorder (Robins & Guze, 1970). In Chap. 1 (and elsewhere) in this volume, authors have referred to the controversy around the validity of BPD in children and

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C. Sharp and J.L. Tackett (eds.), *Handbook of Borderline Personality Disorder in Children and Adolescents*,
DOI 10.1007/978-1-4939-0591-1_16,

Table 16.1 Methods of key studies and suggestions for further reading

	Sample				Assessment				
Citation	Age at T1	Size	Sex	Type	PD measure	Time points	Years	Report	Findings
Children in the Community Studies									
Kasen et al. (1999)	9–16	551	Both	Community	Items adapted from Personality Diagnostic Questionnaire or Structured Clinical Interview for DSM-III-R PD Items written by Bernstein et al. (1993)	3	10	Self mother	• Odds of young adult PD increased with adolescent PD, disruptive behavior disorder, anxiety, depression, and comorbidity
Johnson et al. (2000)	14	816	Both	Community	Items taken from the Diagnostic Interview Schedule for Children, Personality Diagnostic Questionnaire, Disorganizing Poverty Interview and modified for DSM-IV	3	9	Self mother	• PD traits declined during adolescence and early adulthood • Adolescents with PD had elevated PD traits as young adults • Stability of PD traits was similar during adolescence and early adulthood
Crawford et al. (2001a)	10–14	407	Both	Community	Items from Disorganizing Poverty Interview, Quality of Life Interview, Personality Diagnostic Questionnaire, and Structured Clinical Interview for DSM-III-R Personality Disorder, DISC	3	8	Self mother	• Cluster B traits were highly stable and more stable than Axis I symptoms
Crawford et al. (2001b)	10–14	407	Both	Community	Items from Disorganizing Poverty Interview, Quality of Life Interview, Personality Diagnostic Questionnaire, and Structured Clinical Interview for DSM-III-R Personality Disorder, DISC	3	8	Self mother	• Internalizing and externalizing symptoms predicted subsequent Cluster B in girls • Cluster B traits in boys 10–14 predicted externalizing symptoms 2 years later
Crawford et al. (2004)	13.8 and 18.6	714	Both	Community	Categorical diagnoses and dimensional symptom scales based on DSM-IV (Johnson et al., 1999, 2000)	2	6	Self	• PD inversely related to well being • Cluster B traits inversely related to intimacy, strengthened during transition to adulthood
Crawford et al. (2005)	22	644	Both	Community	Children in the Community Self-Report Scales (Crawford et al., 2005) SCID-II SCID-II-Personality Questionnaire	2	11	Self	• Children in the Community Self-Report Scales at 22 predicted self-report and SCID-II PQ at age 33 • PD traits decline with age and positive skew increases

Winograd et al. (2008)	14	636	Both	Community	Children in the Community Self-Report Scales (Crawford et al., 2005)	4	20	Self	• Borderline traits in adolescence predicted borderline traits, diagnosis, need for services, and impairment in adulthood
Other Empirical Work									
Garnet et al. (1994)	15–19	21	Both	Inpatient	Personality Disorder Examination (DSM-III-R)	2	2	Self	• In stable subgroup ($N = 7$), most stable traits: emptiness and boredom (100 %), inappropriate and intense anger (86 %), • Least stable symptoms: unstable, intense relationships (50 %), impulsiveness (57 %) • Presence of any one trait did not predict stability
Mattanah et al. (1995)	12–18	65	Both	Inpatient	Personality Disorder Examination (DSM-III-R)	2	2	Self	• Unstable diagnosis (23 % diagnosed at both times)
Meijer et al. (1998)	15.2	36	Both	Inpatient	Diagnostic Interview for Borderline patients (DIB)	2	3	Self	• Unstable diagnosis (14 % diagnosed at both times)
Grilo et al. (2001)	15.6	60	Both	Inpatient	Personality Disorder Examination (DSM-III-R)	2	2	Self	• Decreasing trait level • Unstable diagnosis
Chanen et al. (2004)	15–18	101	Both	Outpatient	Structured Clinical Interview for DSM Axis II Disorders	2	2	Self	• Moderate differential stability • Moderate absolute stability
Crick et al. (2005)	4th–6th grade	400	Both	Community	Borderline Personality Features	3	1	Self	• Moderate differential stability • Higher trait levels in girls than boys
Bornovalova et al. (2009)	14	617–1,492	Female	Community	Multidimensional Personality Questionnaire—Borderline Personality Disorder Scale	3	10	Self	• High differential stability • Decreasing trait levels • Stability influenced highly by genetic factors and modestly by nonshared environmental factors
Stepp et al. (2010)	6–8	2,451	Female	Community	BPD-related items of impulsivity, negative affectivity, and interpersonal aggression	6	6	Parent teacher	• High year-to-year stability of parent report • Moderate year-to-year stability of teacher report

adolescence. Exploring the ways in which borderline traits evolve and/or remain stable across development is an important step toward fully establishing the validity of this disorder in youth.

Children in the Community Study

The CIC Study is based upon a sample of New York children assessed for the first time in 1975 (at ages 1–10 years) in order to determine indicators of need for social services (Kogan, Smith, & Jenkins, 1977). This sample was followed-up for the first time in 1983 and a number of subsequent times. The aims of this study have shifted to include greater understanding of the development of psychopathology in youth. The influence of CIC studies on the fields of developmental psychopathology and clinical psychology has been vast; however, the chapter will focus exclusively on the conclusions of CIC studies with direct relevance for the developmental course of borderline traits. Studies have been organized into sections (below) based on their main conclusions.

Homotypic Continuity: BPD Now and Later

Homotypic continuity is defined as stability in the same or similar behavioral responses over time—that is, a disorder predicting itself over time (e.g., earlier BPD predicting later BPD). Homotypic continuity supports the notion that a single disease process expresses itself robustly across developmental contexts (Costello, Copeland, & Angold, 2011). In this case, homotypic continuity can be discussed at two levels: first, the degree to which borderline traits present in youth persist into adulthood and second, the degree to which a BPD diagnosis in youth is present in adulthood.

With regard to trait stability, CIC studies have generally concluded that borderline traits that exist in youth are quite stable into adulthood. Generally, stability coefficients for Cluster B traits (traits of BPD, histrionic personality disorder, and narcissistic personality disorder, excluding antisocial traits) were significant for both males and females across three time points spanning from 10 to 24 years of age (Crawford, Cohen, & Brook, 2001a). More specifically, ipsative stability coefficients (indicating the extent to which an individual's absolute borderline trait levels remain stable over time) of borderline traits were significant over the course of 9 years and three time points ($M_1 = 13.8$ years, $M_2 = 16.1$ years, $M_3 = 22.0$ years; $ICC_{1,2} = .37$, $ICC_{2,3} = .36$, $ICC_{1,3} = .28$ all $p < .0001$), indicating borderline trait stability from adolescence to young adulthood (Johnson et al., 2000). Differential stability coefficients (indicating stability in relative position of individuals within a group over time) were also significant for borderline traits across time points ($r_{1,2} = .44$, $r_{2,3} = .36$, $r_{1,3} = .38$ all $p < .001$; Johnson et al., 2000), showing that those that present with greater borderline traits in youth will represent the same trait level (relative to their peers) in young adulthood. Importantly, one CIC report conducted by Winograd, Cohen, and Chen (2008) revealed that borderline traits in adolescence are significantly associated with borderline traits even at a 20 years follow-up ($r = .39$) and that early traits predict substantial impairment in a variety of areas during adulthood. Specifically, higher borderline traits at baseline ($M = 14$ years) predicted borderline traits ($\beta = .27$) and BPD diagnosis (OR = 9.36) at follow-up ($M = 33$ years), as well as poorer role, social, academic, and occupational functioning and lower partner involvement and life satisfaction. In addition to highlighting the very long-term stability of borderline traits from adolescence to adulthood, this study emphasizes the importance of identifying traits early in an effort to avoid poor outcomes in adulthood.

In sum, CIC studies find that the presence of Cluster B traits, generally, and borderline traits, specifically, is stable during early, middle, and late adolescence and adulthood. This conclusion is particularly important in addressing controversy over labeling borderline traits in youth arising from concerns that personality is unstable during adolescence (Meijer, Goedhart, & Treffers, 1998).

Findings of the CIC "suggest that personality disorder trait stability during early and middle adolescence may not be lower than the stability of personality disorder traits during early and middle adulthood" (p. 272, Johnson et al., 2000). This suggests that resistance to identifying borderline traits in youth for fear of personality instability is unjustified if the same concern is not associated with identifying borderline traits in early and middle adulthood. Results of the CIC studies also speak to controversy in assessing and treating borderline traits in youth based on the concern that the traits will resolve completely on their own. CIC studies provide clear evidence of trait stability into adulthood which is, in some instances, greater than the stability associated with internalizing or externalizing symptoms (Crawford et al., 2001a), which are widely assessed and treated in youth.

In two reports from CIC, results showed stability of diagnosis as well; however, these results should be interpreted with caution given relatively low base rates of those meeting criteria for BPD in this sample. Specifically, individuals with BPD diagnoses during adolescence had a significantly greater number of borderline traits in early adulthood than those without a BPD diagnosis in adolescence (Johnson et al., 2000). More generally, the odds of having a Cluster B (excluding antisocial) diagnosis during young adulthood are up to 7.44 times greater ($p < .001$) if a Cluster B disorder (without other psychopathology) is present during adolescence (Kasen, Cohen, Skodol, Johnson, & Brook, 1999). This risk is even greater when a Cluster B disorder co-occurs with an Axis I disorder in youth (Kasen et al., 1999). Although no CIC report to date has explored the stability of the BPD diagnosis specifically, data from the CIC study by and large provide preliminary evidence that it too would remain stable from adolescence to adulthood.

Ebbs and Flows: Within-Group Variation in Borderline Symptoms

Despite the evidence of stability in borderline traits, data from the CIC study have shown that neither the consequences nor the number of Cluster B and borderline symptoms are identical across developmental stages. One of the clearest conclusions about the course of symptoms is that they fluctuate in prevalence (defined as the number of symptoms endorsed) with age. Generally, all personality disorder symptoms seem to be most prevalent during early adolescence and decline until the mid to late 20s (Johnson et al., 2000). With regard to Cluster B symptoms specifically (excluding antisocial symptoms), prevalence of symptoms increases during adolescence (from 13.8 years to between 18.6 and 19.8 years) and then decreases until early adulthood (24.3 years; Crawford, Cohen, Johnson, Sneed, & Brook, 2004). Although both subjects who did and did not meet criteria for a Cluster B disorder demonstrated this same pattern of increasing, peaking, and then decreasing symptoms, the group who met diagnostic criteria for a Cluster B disorder endorsed more symptoms than individuals without a Cluster B disorder throughout.

With regard to borderline traits specifically, data from the CIC study have revealed a general pattern of decreasing prevalence from early adolescence through early adulthood. In one report, the number of borderline traits endorsed decreased 59 % from ages 9–12 to ages 25–28 and, taken together, the correlation between borderline traits and age was significant and negative (Johnson et al., 2000). This pattern maps more clearly onto the pattern associated with personality disorder in general, rather than with the pattern identified for Cluster B symptoms. That is, borderline trait prevalence follows the trajectory of general personality disorder traits, which is a general decline with age, rather than the pattern of Cluster B traits. Perhaps, then, collapsing borderline traits into a general heading of Cluster B traits for data analyses obscures uniqueness associated with the trajectory of borderline traits. In another report, however, it was determined that despite a general trend of decreasing borderline symptoms from ages 12 to 23, people with a history of sexual abuse reported consistently elevated borderline symptoms (Cohen, Brown, & Smailes, 2001),

indicating that even within borderline symptoms, developmental trajectories are varied. Moreover, Cohen et al. (2008) found that although borderline symptoms generally decrease, low parental socioeconomic status and IQ and high stressful life events, trauma, and problematic parenting are associated with increased prevalence of borderline symptoms from early adolescence through adulthood. These findings speak to the vast within-group variance in BPD, an issue central to criticisms of the many different ways in which the criteria for BPD can be met, and point to the role of moderators and mediators of symptom trajectory in evaluating the longitudinal course of BPD in the future.

The CIC study has also had access to a large number of youth without psychopathology and can therefore speak to how the differences between diagnostic groups and community controls change across development. With regard to borderline symptoms, Crawford et al. (2005) noted that though symptoms decreased with age, positive skew increased. In other words, though prevalence of borderline traits drops from adolescence to adulthood, it does so more for young adults who had lower borderline traits at the start. Therefore, as the normative level of borderline traits drop, the gap between those with high and low traits at the start widens and adolescents with high borderline traits become increasingly different from their peers (Crawford et al., 2005). Indeed, CIC reports have contributed to a general view of late adolescence as a critical period for personality disorder development during which maladaptive personality traits become significantly more deviant from the norm (Clark, 2005; Tackett, Balsis, Oltmanns, & Krueger, 2009).

Developmental changes also seem to produce changes in the way that borderline traits affect the lives of adolescents and young adults. For instance, borderline traits become associated with elevated partner conflict during the transition from adolescence to adulthood though prior to adolescence, personality disorder is not associated with partner conflict (Chen et al., 2004). Cluster B traits also become increasingly associated with lower self-reported intimacy moving from adolescence to early adulthood, manifested in a negative relationship between Cluster B traits and intimacy that is twice as strong for older adolescent females and young adults as for early adolescents (Crawford et al., 2004). Possible explanations for these changes include the growing importance of partners and intimacy during the transition from adolescence to adulthood and the developmental task of identity consolidation posed by Erikson being inhibited by Cluster B traits (Crawford et al., 2004)—though all explanations for these changes are speculative at this time. These findings line up with the growing view of adolescence and the transition to adulthood as a critical period in the development of personality disorder (as alluded to previously; Clark, 2005; Tackett et al., 2009), because they indicate that disordered personality traits become most impairing during this developmental phase by becoming more distinct from normative personality and beginning to interfere with interpersonal functioning. Given the large longitudinal database associated with the CIC study, evaluation of critical periods in the development of personality disorder may be a promising area of future research.

Heterotypic Continuity: Changing Faces

Despite a number of CIC studies indicating homotypic continuity, there is no question that several reports generated from CIC studies have come to alternate conclusions about the course of BPD, stressing instead heterotypic continuity. Heterotypic continuity is defined as stability of an underlying construct that is manifested differently across time as a result of changing developmental capacities (Sroufe & Rutter, 1984)—that is, that different disorders predict one another over time (e.g., depression predicting later BPD). Heterotypic continuity supports the notion that different disorders reflect a general disease process that has specific phenotypic expressions in different developmental contexts, an idea that has been supported in a number of CIC reports. Interestingly, even the heterotypic

continuities of BPD appear to be quite diverse such that borderline traits do not necessarily remain borderline traits or become one other disorder, but rather appear tied to a variety of other disorders across development including both internalizing and externalizing and Axis I and II disorders. Specifically, early Cluster B traits (excluding antisocial traits) predict externalizing diagnoses (Crawford, Cohen, & Brook, 2001b), disruptive behavior (Johnson et al., 1999), and aggressive/criminal behavior (Johnson et al., 2000) in adolescence. Early borderline traits, specifically, are associated with later narcissistic traits (Hamigami, McArdle, & Cohen, 2000) and substance abuse disorders (Cohen et al., 2005). Finally, heterotypic continuity has also been documented in the opposite direction, with internalizing symptoms in early adolescence predicting Cluster B traits during middle adolescence among girls (Crawford et al., 2001b) and externalizing problems predicting personality disorder in adolescence (Bernstein, Cohen, Skodol, & Bezirganian, 1996).

Comorbidity: Complicating the Continuity Picture

Though the CIC results presented thus far have been categorized into those that illustrate either homotypic or heterotypic continuity, the reality is complicated by the fact that heterotypic and homotypic continuities coexist, reflecting differing trajectories over different developmental stages, in different subgroups, etc. as well as the very high rate of comorbidity among individuals (at every developmental stage) with BPD. For example, a child psychologist might receive parent reports indicating symptoms like oppositional behavior, tantrums, and peer problems that are diagnosed as an externalizing disorder. Later in the child's life, a clinician might speak directly with the child who exhibits the same symptoms but is now able to articulate that her behavior is the result of fears of abandonment, sensitivity to rejection, and an unstable mood, leading the psychologist in the direction of BPD and therefore representing heterotypic continuity (strictly speaking). In this case, the overlap in symptoms between BPD and externalizing disorders has led to what appears to be heterotypic continuity but might actually represent homotypic continuity with a change in diagnosis attributable to overlap in diagnostic criteria and reflective of the fact that both disorders may lie on a continuum of general externalizing behavior.

Another example illustrates the opposite scenario. An adolescent is affectively unstable, frequently angry, and self-injuring and her psychologist suspects the presence of borderline traits. As the adolescent ages, she finds that her affective instability and anger have isolated her from her peers, prevented her from forming intimate relationships, and led her to spend much time alone. She presents to a psychologist as an adult. Her affective instability and anger have subsided and she is currently bothered only by social isolation, low mood, and self-injurious behavior. Still, the psychologist diagnoses her with BPD based upon her present symptoms and her continued endorsement of "evil," "bad," and "angry" perceptions of herself. Though, strictly speaking this seems to represent homotypic continuity, it might actually represent how borderline traits in youth, despite decreasing to subclinical levels across development, can influence self- and other perceptions in ways that have lasting effects. Perhaps the individual is really in the midst of a Major Depressive Episode subsequent to isolation and rejection associated with borderline traits in her youth. Though she describes herself in a manner consistent with BPD, it may be that she is reflecting negative global and stable attributions about herself associated with a depressive process, making this an example of heterotypic continuity instead. Though both examples are clearly contrived for the purposes of illustration, they do just that—they illustrate how comorbidity in BPD muddies the waters with regard to homotypic and heterotypic continuity.

While complex and multifactorial, comorbidity in BPD is an essential focus of research because, in addition to complicating assessment and treatment, it is predictive of a more severe

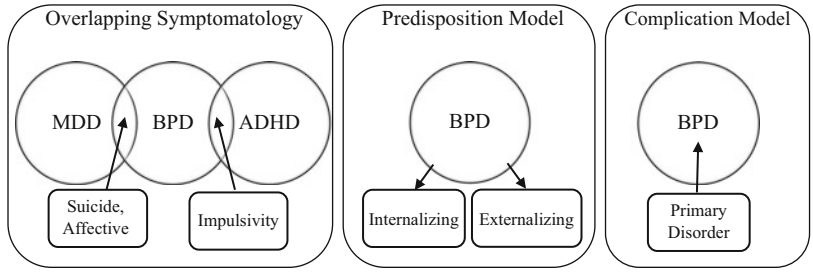

Fig. 16.1 Hypothetical models of comorbidity between Axis I and II

trajectory (Kasen et al., 1999). Three models of comorbidity between Axis I and II disorders have been proposed by CIC authors Crawford et al. (2001b) which help to organize the effect that comorbidity has on the longitudinal course of the disorder. These three models are portrayed in Fig. 16.1. The *Overlapping Symptomatology Model* suggests that comorbidity may simply be an artifact of overlapping symptoms between BPD and internalizing and externalizing disorders (Crawford et al., 2001b). Therefore, what appears, initially, to be comorbidity or heterotypic continuity is instead explained by problems with a categorical diagnostic system and disorders with some overlapping symptoms. The *Predisposition Model* suggests that underlying character disturbance such as a personality disorder gives rise to internalizing and externalizing symptoms (as in the second example; Gunderson & Elliott, 1985 as cited by Crawford et al., 2001b). That is, the problems associated with borderline traits may lead to circumstances in which an individual begins to experience symptoms of and behaviors associated with internalizing and externalizing Axis I disorders. Finally, the *Complication Model* suggests that personality change is a complication of a primary disorder (Akiskal, Hirschfeld, & Yerevanian, 1983 as cited by Crawford et al., 2001b) where the experience of having an Axis I disorder affects personality development in favor of an Axis II pattern.

All three of these models have found some support in CIC data on the course of BPD and there is no consensus as to which of them best captures the longitudinal relation between BPD and Axis I disorders. It is also important to note that many of the CIC conclusions regarding comorbidity and continuities are age- and gender specific, suggesting that examination of these moderating and mediating factors is essential in better understanding the course of BPD (Crawford et al., 2001b).

A great deal of support for the Overlapping Symptomatology Model has come solely from the diagnostic criteria of BPD and other disorders. As briefly noted earlier, the DSM-IV-TR (American Psychiatric Association, 2000) criteria for BPD require five (or more) of nine diagnostic criteria. Among these, *seven* of them are closely mirrored in other DSM-IV-TR disorders as follows: frantic efforts to avoid real or imagined abandonment (e.g., separation anxiety disorder; possibly social phobia); identity disturbance; impulsivity (e.g., attention deficit hyperactivity disorder; antisocial personality disorder); recurrent suicidal behavior or self-mutilating (e.g., major depressive disorder); affective instability (e.g., bipolar disorder); inappropriate, intense anger or difficulty controlling anger (e.g., bipolar disorder; oppositional defiant disorder; antisocial personality disorder); and transient, stress-related paranoid ideation or dissociation (e.g., posttraumatic stress disorder; substance-induced delirium; delusional disorder; paranoid personality disorder). Given this tremendous overlap in diagnostic criteria, and the DSM-IV-TR's current model of diagnosis, it is very difficult to determine whether comorbidity in BPD represents co-occurring, but pathologically distinct, syndromes or a current failure in the diagnostic system. As previously discussed, this overlap may also account for conflicting evidence in favor of both heterotypic and homotypic continuities. For example, in one CIC report (Kasen et al., 1999), 38 % of adolescents with a Cluster B disorder

were also diagnosed with anxiety, 28 % with a depressive disorder, and 47 % with a disruptive behavior disorder. Therefore, depending upon the diagnostic inclinations of a particular clinician or parameters of a given research study, nearly half of individuals with a Cluster B disorder could be categorized in one of several ways or assigned multiple diagnoses, complicating conclusions that can be drawn about the continuity of BPD.

In support of the Predisposition Model, the CIC has found evidence of Cluster B traits in youth predicting later externalizing symptoms. Specifically, Cluster B traits in children ages 10–14 predicted externalizing symptoms during mid-adolescence for both genders (Crawford et al., 2001b). The same report did not find that Cluster B traits in youth were associated with later internalizing disorder. One explanation for the increased risk of an externalizing trajectory for youth with Cluster B traits is that Cluster B personality disorders are "organized around a novelty-seeking temperament associated with disorderly, exploratory, and impulsive behavior" (p. 346, Crawford et al., 2001b citing Cloninger, 1987). Therefore, with time, externalizing behavior emerges as a manifestation of Cluster B personality traits. An important caveat to the Predisposition Model is that, of course, not all children who display Cluster B traits go on to exhibit externalizing disorders, illustrating the principle of multifinality (the concept that one factor can lead to several psychopathologic outcomes, dependent upon the individual context) fundamental to developmental psychopathology research.

The Complication Model is well described in CIC findings that demonstrate that Cluster B traits, and borderline traits more specifically, are associated with earlier psychopathological symptoms of various other kinds, illustrating the principle of equifinality instead (the concept that the same psychopathological outcome can result from several different prior factors). For example, Crawford et al. (2001b) found evidence that, for girls, internalizing symptoms at ages 10–14 predicted Cluster B traits during mid-adolescence while internalizing symptoms at ages 12–17 did not pose the same risk for Cluster B traits in later adolescence or adulthood. The authors (Crawford et al., 2001b) suggest that this is perhaps indicative of a critical period for personality development that occurs during the transition to adolescence. If disrupted by internalizing symptoms during that time, personality development may be more likely to include Cluster B traits. A similar pattern is evident in Crawford et al.'s (2001b) work with regard to externalizing symptoms for girls—externalizing symptoms during mid-adolescence (12–17 years) are predictive of Cluster B traits during early adulthood (excluding antisocial traits). This relation is attributed by the authors to interference in maturation or identity integration (occurring during mid-adolescence) by externalizing symptoms or unlabeled homotypic continuity wherein externalizing symptoms during mid-adolescence are a manifestation of Cluster B traits.

Another CIC report by Kasen et al. (1999) found similar evidence for the Complication Model and, further, identified the diagnostic importance of comorbid Axis I and II disorders for the personality trajectory of youth. Specifically, Kasen et al. (1999) found that risk of Cluster B personality disorder (excluding antisocial) in young adulthood was increased by the prior presence of a disruptive behavior disorder, depression, and what they refer to as "other Axis I disorder." The risk for Cluster B personality disorder was even higher when there was previous comorbidity occurring between Cluster B personality disorder and a disruptive behavior disorder, anxiety disorder, and depression, suggesting that the view of personality disorder as a Complication of a primary Axis I disorder is further exacerbated when Cluster B traits are already present in youth.

Summary, Limitations, and Future Directions of the CIC Studies

In summary, the work of the CIC on the developmental course of Cluster B personality disorder generally, and BPD specifically, has led to varied

conclusions ranging from disorder stability from youth through early adulthood to heterotypic continuities in which personality pathology changes into Axis I symptoms or vice versa. Indeed, the conclusions of the CIC are too varied to come to a consensus about the longitudinal course of BPD, rather, they illustrate the complexity of longitudinal psychopathology research and the need for further investigation into the moderators and mediators of developmental course hinted at in the CIC findings. Not considered thus far as an explanation for this complex picture is the heterogeneity of BPD—that is, the possibility that heterotypic continuity may simply reflect the presence of subgroups of BPD.

Additionally, CIC authors have used three models of comorbidity, the Overlapping Symptomatology, Predisposition, and Complication Models exclusively. While this framework has proved organizationally useful, these three models are not representative of the highly varied models of comorbidity used within personality and psychopathology research more generally. Indeed, a literature review conducted by Krueger and Markon (2006) presents several bivariate and multivariate comorbidity models that have not been evaluated in previous CIC reports. Of these, the Liability-Spectrum Model is presented as a viable model for making sense of personality traits in psychopathological comorbidity, and therefore, likely represents a valuable extension to current understandings of comorbidity in the CIC study.

The conclusions of CIC study findings are also tempered by a number of limitations often cited by the authors themselves. Perhaps the greatest limitation of this work is that measurement of personality disorder traits has changed across developmental stages and time (Cohen et al., 2005; Johnson et al., 2000). This limitation is, in many ways, a consequence of the changing conceptualization of personality disorders in various iterations of the DSM as well as the paucity of developmentally appropriate and sensitive measures of personality disorder in youth. Although measures within CIC studies have demonstrated high correspondence, measurement changes certainly affect stability estimates as well as conclusions that can be drawn about continuity of traits. Further, CIC authors themselves cite reliance on DSM criteria as a limitation insofar as it requires "arbitrary criterion-based cutoffs" (p. 468, Cohen et al., 2005), ignores subthreshold but clinically relevant personality disorder (Cohen et al., 2005), and was developed specifically for adult diagnosis. Finally, a limitation highly relevant for this chapter is that in many CIC reports personality disorder traits and diagnoses are grouped into clusters (e.g., Cluster B) in order to address problems of low power resulting from few diagnoses of each personality disorder (Cohen et al., 2005). Therefore, the findings of many CIC studies obscure information about the developmental course of BPD by combining participants who display borderline traits with those who display similar, but distinct, Cluster B traits.

The limitations of CIC studies also serve as useful indicators of valuable future research since the CIC studies currently make up the majority of the literature on the developmental course of BPD. One of the most often cited directions for future research among CIC authors is the desire to extend the age ranges considered in studies assessing the course of personality disorder. While most studies to date have focused on late childhood or early adolescence through early adulthood, there is a need to explore early childhood factors as well as extend current theories about the course of BPD into midlife and older adults (Cohen et al., 2005). Examining traits in early childhood may also avoid the complexity that comorbidity adds to existing longitudinal work by identifying how traits are manifested when they first emerge. There is also a great need to differentiate among risk factors known to be associated with adult personality disorder into those that "create an ongoing vulnerability" and "'kindling' risks that lead to variation in symptom expression over time" (p. 480, Cohen et al., 2005) if we are to use knowledge of risk factors effectively in clinical practice.

Beyond the Children in the Community Study

This section focuses on the developmental course of BPD and BPD characteristics in children and adolescents based on findings from prospective longitudinal studies other than the CIC (Bornovalova, Hicks, Iacono, & McGue, 2009; Chanen et al., 2004; Crick, Murray-Close, & Woods, 2005; Garnet, Levy, Mattanah, Edell, & Mcglashan, 1994; Grilo, Becker, Edell, & McGlashan, 2001; Lofgren, Bemporad, King, Lindem, & O'Driscoll, 1991; Mattanah, Becker, Levy, Edell, & Mcglashan, 1995; Meijer et al., 1998; Stepp, Pilkonis, Hipwell, Loeber, & Stouthamer-Loeber, 2010), some of which address limitations in the CIC studies. These studies tend to approach longitudinal research with two general statistical techniques and can therefore be grouped into those that comment on differential stability and those that focus on absolute stability. Together participants in these studies span ages 6–24 and findings suggest, like many of the CIC reports have, that (1) BPD traits exhibit at least moderate and sometimes strong order stability from childhood throughout adolescence and into adulthood, (2) borderline trait levels decrease from childhood to adulthood, and (3) the developmental course of BPD traits in children and adolescents is influenced by factors such as the youth's gender, genes, and environment.

Differential Stability of BPD Traits: Relative Severity in Youth Predicts Relative Severity in Adulthood

Studies that have examined the differential stability of BPD traits in children and adolescents suggest that BPD traits are at least moderately, and sometimes strongly, stable from as early as 6 years of age. Although studies have used different statistics, such as Pearson correlation coefficients (Bornovalova et al., 2009; Chanen et al., 2004; Crick et al., 2005) and intraclass correlation coefficients (ICC; Grilo et al., 2001; Stepp et al., 2010), to analyze the stability of BPD traits, complicating direct comparison across them, they generally suggest that parent-reported borderline traits (Stepp et al., 2010) show higher differential stability (ICC ranged from .71 to .85) than do self- (Bornovalova et al., 2009; Chanen et al., 2004; Crick et al., 2005; Grilo et al., 2001; r ranged from .47 to .73; ICC = .16) or teacher-reported (Stepp et al., 2010; ICC ranged from .49 to .77) borderline traits. One study further found that differential stability increases for parent-reported borderline traits (at least from 6 to 12 years) whereas it increases until age 8 years, levels off, and then decreases at age 11 years for teacher-reported borderline traits (Stepp et al., 2010). Stepp et al. (2010), however, point out that even the moderately high stability of teacher reports is impressive given that this stability was analyzed across different teachers as the girls in their study moved from one grade to the next. Similarly, Crick et al. (2005) point out that the moderately high stability of self-reported borderline traits in their study of fourth through sixth graders is impressive given that children generally moved from one school grade to the next during study periods and such grade transitions are usually associated with many changes (e.g., new teachers and peers) which can affect children's functioning (e.g., their relationships with their teachers and peers).

Furthermore, studies that have examined the stability of specific aspects of BPD in children and adolescents suggest that depending on the reporter of borderline traits, certain traits show higher stability than do others. For example, teacher-reported relational aggression and depression were found to be less stable than were teacher-reported cognitive dyscontrol, hyperactivity, and (lack of) self-control (Stepp et al., 2010). Stepp et al. (2010) suggest that the difference in stability across these aspects might be due to teachers being poorer reporters of internalizing aspects such as depression than they are of externalizing aspects such as hyperactivity; however, these findings need to be interpreted carefully given that they were not adjusted for reliability. Self-reported borderline traits, albeit

in a very small sample ($N = 7$) of adolescents with persistent BPD diagnoses over a 2-year study period, were not found to show such differences along internalizing and externalizing dimensions (Garnet et al., 1994), which further supports Stepp et al.' (2010) argument that the reporter type might influence the stability of borderline traits. However, Garnet et al's findings need to be interpreted even more carefully given that they likely had insufficient power to support a null hypothesis of no differences along internalizing and externalizing dimensions. Specifically, in this small sample of adolescents with persistent BPD diagnoses, both internalizing borderline traits such as emptiness and boredom and externalizing aspects such as inappropriate and intense anger were found to be the most stable traits; however, the presence of any one trait did not predict stability of a BPD diagnosis. In another small inpatient sample ($N = 36$), borderline traits such as "conflict about receiving care," "dependency and masochism," and "areas or periods of special achievement," were found to be more stable than aspects such as "automutilation," "manipulative suicide or suicide threat," "derealization," "depersonalization," and "brief paranoid experiences" (Meijer et al., 1998). Meijer and colleagues argue that a decrease of the less persistent traits, and especially self-harm and suicide (threat), was expected as adolescents were released from the psychiatric inpatient unit. Taken together, these findings suggest that the stability of BPD traits might differ based on their nature (e.g., internalizing versus externalizing), their reporter (e.g., parent versus teacher), as well as their severity (e.g., self-harm versus less severe symptoms). Given the previous studies' limitations in terms of their small sample size and their failure to adjust for reliability, however, it remains necessary to replicate these findings on self-reported borderline traits in larger community samples.

Finally, studies also generally suggest that borderline traits in community (Bornovalova et al., 2009; Crick et al., 2005; Stepp et al., 2010) or outpatient (Chanen et al., 2004) samples are more stable than they are in inpatient samples (Grilo et al., 2001; Meijer et al., 1998). Grilo et al. (2001) posit that the low stability of borderline traits in their severely ill inpatient sample might not generalize to community and outpatient samples possibly because their sample was undergoing intense treatment expected to change (i.e., improve) certain borderline traits. In summary, studies on the differential stability of BPD traits suggest that borderline trait levels decrease from childhood to adulthood; however, what moderates the magnitude as well as the course of that stability is still an open question. Promising moderators seem to be the reporters of BPD traits (e.g., parent versus self versus teacher), the nature of BPD traits (e.g., internalizing versus externalizing), and the nature of the sample (e.g., clinical versus community). In addition to leaving questions regarding potential moderators open, studies on the differential stability of BPD traits also leave questions regarding changes in mean levels of BPD traits open. These questions can be answered by studies on the absolute stability of BPD traits.

Absolute Stability of BPD Traits: Absolute Severity Decreases from Youth to Adulthood

Studies that have examined the absolute stability of BPD traits in children and adolescents suggest that borderline trait levels decrease from childhood to adulthood. This decrease in borderline trait levels was found in community (Bornovalova et al., 2009; Crick et al., 2005) and inpatient samples (Grilo et al., 2001) but not in an outpatient sample (Chanen et al., 2004). Specifically, prevalence of borderline trait levels was found to decrease slightly from fourth to sixth grade (Crick et al., 2005), highly from 14 to 24 years (but not from 14 to 17 years and only moderately from 14 to 20 years; Bornovalova et al., 2009), and slightly from 15 to 17 years (Grilo et al., 2001). In light of these findings of change and the small size of youth with BPD in the outpatient sample ($N = 12$), Chanen et al.'s (2004) findings of no change should be interpreted with caution. Together with CIC findings (Bernstein et al., 1993; Johnson et al., 2000), these studies suggest that borderline traits peak during puberty and

decrease thereafter (as well as possibly just before puberty; Crick et al., 2005), highlighting adolescence as an especially critical developmental period in the course of BPD.

Finally, studies that have examined the persistence of BPD diagnoses suggest that these diagnoses are not very persistent (Chanen et al., 2004; Lofgren, Bemporad, King, & Lindem, 1991; Mattanah et al., 1995; Meijer et al., 1998). Although the sizes of these inpatient samples were small ($N < 65$), it is worth pointing out that symptom improvement, especially in such areas as self-harm and suicide (threat) as well as dissociation and paranoia, contributed to the improvement in BPD. Other traits such as "inappropriate and intense anger" (Garnet et al., 1994), on the other hand, were found to be more persistent. Furthermore, although youth diagnosed with BPD at baseline often did not maintain their diagnosis, at least one study found that they were more likely to be diagnosed with other personality disorders (especially antisocial personality disorder) at a follow-up (Lofgren et al., 1991). In summary, studies on the absolute stability of BPD traits suggest that borderline trait levels decrease from childhood to adulthood. Furthermore, they identify early adolescence as a critical developmental period where borderline trait levels peak. Similar to studies on the differential stability of BPD traits, studies on the absolute stability of BPD traits also leave questions regarding what moderates the course of that stability open. These questions have been tackled by only a few studies so far.

Factors Influencing the Developmental Course of BPD

Of the many factors that might influence the developmental course of BPD traits in children and adolescents, a few studies have examined two: (1) gender and (2) relative contributions from genetic and environmental factors, going far beyond those explored in the majority of CIC studies. One study that examined gender differences in borderline traits found that levels of these traits were greater for girls than for boys at the beginning of the study, and over the 1-year period of the study these scores decreased for girls but not for boys (Crick et al., 2005). This finding was contrary to Crick et al.'s hypothesis based on women having more borderline symptoms than men but might be explained by the developmental period examined in their study. Crick et al. (2005) suggest that expanding the developmental period under investigation to include adolescence, when rates of psychopathology increase especially for girls, might show a similar increase in BPD scores for girls. For example, in an older sample of 15-to-18-year-old adolescents, no interaction between gender and time was found (Chanen et al., 2004).

Secondly, one study that examined genetic and environmental factors on the developmental course of borderline traits in children and adolescents found that the stability of borderline traits was strongly influenced by additive genetic factors and moderately influenced by nonshared environmental factors (Bornovalova et al., 2009). This finding suggests that although a person's borderline trait score at a single time point was only moderately heritable (a^2 ranged from .31 to .46), a person's (change in) borderline trait scores over time was strongly heritable ($a^2 = .76$). Bornovalova et al., (2009) argue that this difference in heritability might be due to reduced measurement error when borderline trait scores were accounted for across time. Furthermore, they hypothesized that the increasing heritability in BPD traits over time might have its origin in declining shared environmental influence, changes in gene expression, or active gene–environment correlations whereby individuals actively choose environments that turn on their genetic expression. Finally, they hypothesized that the nonshared environmental variance might have its origin in childhood abuse and other traumatic events, differential treatment by parents, and unpredictable events like accidents. In summary, studies on factors influencing the developmental course of BPD traits suggest that the course in children and adolescents is influenced by the youth's gender, genes, and environment. Furthermore, they also underline the importance of attending to developmental periods that might differ in their criticality for different influential factors. For example, Bornovalova's interesting hypotheses

form important future directions that could inform our understanding of when and where interventions are the most useful.

Summary, Limitations, and Future Directions

In sum, findings from the reviewed studies after CIC suggest that BPD traits can be reliably measured as early as 6 years of age. Although no single study spanned the complete developmental period from 6 to 24 years, together they suggest that these early borderline traits are at least moderately stable. These studies in many ways built upon a foundation provided by the CIC study and, as a result, were able to improve upon some of the former's limitations. Specifically, later studies have made use of empirically derived measures and a variety of samples (including inpatients) allowing them to evaluate a true BPD diagnosis. However, some important limitations in this area of research remain. Even longitudinal studies continue to have a relatively limited age range (relative to the actual development of personality) and therefore little is known about the very early and later development of BPD traits. In addition, the range of mediators and moderators affecting the course of BPD traits has only begun to be examined leaving many possibilities for innovation in the future. Finally, the mechanisms associated with emerging, changing, and resolving BPD traits are still relatively unknown and research has focused on a highly important but relatively superficial level of analyses thus far. As knowledge of the developmental pattern of BPD traits continues to emerge, mechanistic studies will become essential in making this research useful for theoretical and clinical purposes.

Clinical Implications

Emerging neuroscience findings indicate that adolescence is an important transitional period from childhood to early adulthood, involving the maturation of emotional regulation, social, and cognitive-processing abilities and related brain areas (see Giedd et al., 2012 for a review). Because of this important transition, it is both a time of increased vulnerability and an opportunity to affect change in developmental trajectories that may become entrenched over time. In addressing problems in adolescence that may represent borderline pathology, clinicians (and parents) ask several important questions. In this section, we examine how the empirical literature discussed above may elucidate those questions.

How do I know that what I see is not normal "storm-and-stress" of adolescence? Data on the longitudinal course of BPD suggest within-subject continuity and variability in prevalence of traits between subjects. In addition, the characterization of adolescence as a ubiquitous period of storm and stress has been mostly dispelled by research in normative development (Arnett, 1999). Not all adolescents experience storm and stress, but storm and stress is more likely during adolescence than at other ages. Even so, severity, intensity, and frequency of traits can be used to distinguish normative variation in conflict with parents, mood disruptions, and risk behavior from pathological extremes.

How do I know it's not conduct disorder or depression or even bipolar disorder? The measures used in the studies discussed in this chapter and elsewhere in this volume are validated to reliably identify adolescents with borderline traits. These measures show discriminant validity (distinguishing BPD from other disorders) and should be considered in routine assessment of clinical cases of adolescents where borderline features are suspected.

How do I know that personality disorder traits won't just blow over? As discussed in this chapter, on average, there seems to be significant continuity in borderline traits over time—either through homotypic continuity (the phenomenology of features remain the same), or through heterotypic continuity (the underlying disorder expresses itself in different behavioral phenotypes over time). Either way, this suggests that traits should be taken seriously and intervention around specific borderline traits should be planned.

Adult and adolescent studies show that many individuals do not meet criteria for BPD over time—why should I then take a diagnosis of BPD in an adolescent seriously? As discussed earlier, PD traits (rather than diagnosis) are more stable over time. These findings are consistent with a dimensional view of BPD (also suggested for the DSM-V overhaul). We would therefore argue that although an individual may move categories over time, their trajectory over time would be stable in terms of overall high severity level of traits. In addition, research reviewed in this chapter suggests important developmental processes for BPD. Consistent with research in adults, there are more or less stable aspects of BPD. For example, impulsive and acute symptomatic behaviors like self-harm may remit more quickly, in contrast to underlying aspects of personality, like social interaction, that may remain more stable.

Conclusion

While great strides have been made in examining the developmental course of BPD, with the CIC study laying the foundation and several new studies building on CIC, to our knowledge there are no explicitly funded studies exploring the course and outcomes of BPD. The studies reviewed here have been studies focusing on other issues, with BPD measurement as a by-product. For the field to move forward significantly, we call for dedicated funding to examine the longitudinal course of BPD, similar to landmark studies in depression and anxiety. Only through large scale, prospective studies that include measures explicitly designed to capture BPD and distinguishing pathology can the longitudinal course of BPD be truly determined. Another important goal for future work is the use of a framework, as suggested by the NIMH Research Domain Criteria, where underlying biological systems are tracked through development with associated psychopathology at the behavioral phenotypic level.

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The Externalizing Pathway to Borderline Personality Disorder in Youth

17

Stephanie D. Stepp, Diana J. Whalen, and Sarah L. Pedersen

Definitions and Core Constructs

Borderline personality disorder (BPD) is a debilitating psychiatric disorder characterized by the presence of any five of nine symptoms from the Diagnostic and Statistical Manual of Mental Disorders (DSM-V; American Psychiatric Association, 2013). Symptoms include emotional instability and anger, impulsive and suicidal behaviors, interpersonal turmoil, as well as cognitive and identity disturbances. The severe nature of BPD, including a high rate of concurrent comorbid psychiatric disorders and physical health problems, and with suicide rates almost 50 times higher in those with BPD than in the general population, BPD is a major public health concern (Frankenburg & Zanarini, 2006; Grant et al., 2008; Holm & Severinsson, 2011). In light of the serious costs associated with this disorder, elucidating pathways that lead to BPD is of the utmost importance in order to ultimately prevent such suffering.

Several developmental theories of BPD place disruptions in the emotional system as key in the subsequent manifestation of other BPD features (Cole, Llera, & Pemberton, 2009; Linehan, 1993; Putnam & Silk, 2005). Linehan's *biosocial theory of BPD* (1993) is one of the most thoroughly delineated etiological models of BPD (for other models, see Fonagy, Target, & Gergely, 2000; Judd & McGlashan, 2003; Kernberg, 1967, 1975, 1976). According to the biosocial theory, BPD is primarily a disorder of the emotion regulation system and emerges from transactions between individuals with biological vulnerabilities to emotional instability and specific environmental influences, specifically an invalidating family environment. Emotional instability subsequently leads to maladaptive behavior patterns as either a direct consequence or to cope with the emotional response. Specifically, emotional instability results in impairment in communicating about emotions and preferences, which leads to vacillations in interpersonal behavior, from passivity to angry demands. Additionally, individuals with BPD engage in impulsive behaviors, such as substance use, and suicide behaviors to regulate intense and sustained negative affective states (Brown, Comtois, & Linehan, 2002; Yen et al., 2004). Lastly, individuals with emotional instability are likely to have identity disturbances. Without the ability to identify feelings, understand affective triggers, and learn how to regulate affect in pursuit of goal-directed behavior, one's self-image is constantly in flux (Linehan, 1993; Putnam & Silk, 2005). Crowell, Beauchaine, and Linehan (2009) revised the biosocial model to include impulsivity as an early temperamental factor that also sets the stage for the development of BPD.

Extending this model, the present chapter puts forth an externalizing path to BPD. We will shed

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light on the development of BPD through this externalizing pathway by reviewing the developmental antecedents that give rise to the impulsive action component of the emotional instability piece of the disorder in the context of deleterious family environments. Specifically, we propose that individuals who engage in impulsive behaviors while experiencing a strong mood may be most at risk for the development of BPD. Understanding the reciprocal and cascading effects of child characteristics and environmental conditions under which BPD is likely to manifest will ultimately provide an empirical base for prevention and intervention programs. We will begin by providing definitions of core constructs including the internalizing-externalizing model of psychopathology, externalizing disorders, developmental antecedents, precursors, and risk factors.

The *internalizing-externalizing model of psychopathology* is an empirically derived set of latent variables that accounts for underlying factors common to many psychiatric disorders from childhood through adulthood and can explain common patterns of comorbidity (Achenbach, 1978, 1995; Krueger, Caspi, Moffitt, & Silva, 1998; Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011). In a large twin sample of children, Lahey et al. (2011) found evidence for a general psychopathology factor and two lower-order factors of internalizing and externalizing dimensions. The internalizing dimension was associated with social phobia, agoraphobia, specific phobia, separation anxiety disorder, and obsessive-compulsive disorder. The externalizing dimension was associated with *conduct disorder* (CD), *oppositional defiant disorder* (ODD), and *attention-deficit/hyperactivity disorder* (ADHD). In contrast with finding using adult samples, depression and generalized anxiety disorder only loaded on the general psychopathology factor and not the internalizing dimension. In adult samples, the internalizing dimension is often divided into two sub-factors: fear and distress. Fear is associated with panic disorder and social and simple phobias, while distress is associated with major depression, dysthymia, and generalized anxiety disorder (Eaton, South, & Krueger, 2010). Although BPD has not been included in studies of the structure of psychopathology in children, in adult samples BPD loads on both internalizing and externalizing dimensions, suggesting a possible common developmental pathway for this disorder and other externalizing disorders (Eaton et al., 2011; James & Taylor, 2008).

Childhood externalizing disorders include ADHD, ODD, and CD. While ADHD is listed as a neurodevelopmental disorder in the DSM-5, this disorder is linked to the externalizing spectrum of psychopathology and is commonly comorbid with other childhood disruptive behavior disorders like ODD and CD (Lahey et al., 2011). ADHD is the most common psychiatric disorder occurring in children and is hallmarked by symptoms of inattention, disorganization, hyperactivity, and/or impulsivity (American Psychiatric Association, 2013). These symptoms can significantly impact a child's quality of life and frequently interfere with academic and social functioning. Thirty to 60 % of children with ADHD will go on to exhibit clinically significant symptoms into adulthood (Chamberlain et al., 2011).

ODD refers to a recurrent, persistent pattern of negativistic, hostile, defiant, and disobedient behaviors toward others, particularly authority figures (American Psychiatric Association, 2013). ODD is often considered a milder form of CD and may represent an early stage in the development of CD. In addition, studies have shown that as many as 65 % of youth with ADHD also have ODD (Biederman et al., 1996). CD is characterized by a repetitive, persistent pattern of behavior that involves significant violations of the rights of others and/or major societal norms (American Psychiatric Association, 2013). Rates of CD rise steeply in adolescence and approximately 60 % of youth meeting criteria for CD also meet criteria for ODD (Maughan, Rowe, Messer, Goodman, & Meltzer, 2004).

Developmental antecedents is a broad term used herein to refer to both precursors and risk factors (see Stepp, Olino, Klein, Seeley, & Lewinsohn, 2013). *Precursors* are early signs and symptoms in childhood or adolescence that

precede onset of a disorder (Eaton, Badawi, & Melton, 1995; Keenan, Loeber, & Green, 1999) and may bear resemblance to symptoms as manifested in adult disorders, such as chronic irritability in childhood predicting later mood disorders (Stringaris, Cohen, Pine, & Leibenluft, 2009). However, precursors are often developmentally appropriate manifestations, and as such they are not necessarily identical to the features of the disorder in later developmental stages, such as the interpersonal turmoil that characterizes BPD manifesting as preferences for friend exclusivity in childhood and unstable romantic relationships in adulthood (Crick, Murray-Close, & Woods, 2005). In contrast, *risk factors* are experiential or environmental factors (e.g., poverty, parental psychopathology, trauma) that increase the probability of developing psychopathology (Cicchetti & Rogosch, 1999). For the purposes of this review, we will use the term precursors to refer to individual-level, internal factors while reserving the term risk factors for external factors.

Theoretical Perspective

BPD is a complex and debilitating psychological disorder that typically emerges by adolescence or early adulthood and is characterized by dysfunction across four domains: emotions, behaviors, relationships, and identity (American Psychiatric Association, 2013). BPD is challenging for clinicians to treat and is associated with considerable impairment, including social stigma, elevated risk for suicide, and poor social, occupational, and academic outcomes (Bagge et al., 2004; Bagge, Stepp, & Trull, 2005; Bender et al., 2001; Skodol et al., 2005; Soloff, Lynch, & Kelly, 2002; Trull, Useda, Conforti, & Doan, 1997; Zweig-Frank, & Paris, 2002). Impulsive behavior is a hallmark feature of this disorder and manifests as suicide and self-injury, substance abuse, bingeing and purging, and other risky behaviors. The identification of developmental pathways to BPD is crucial for the early identification of children most at risk for BPD and the associated impulsive behaviors.

In order to identify children who might be vulnerable to this externalizing pathway, it is important to consider individual-level precursors, such as temperament, as well as family-level risk factors, including parenting, and the transactions between these individual and family characteristics. Two developmental precursors, emotional instability and impulsivity, in the context of a deleterious family/social environment, likely sets the stage for the traits and symptoms that eventually manifest as BPD by adolescence or young adulthood. While much has been written about the role of the invalidating environment in shaping the development of BPD features among children and youth who have high levels of emotional instability (e.g., Fruzzetti, Shenk, & Hoffman, 2005; Linehan, 1993), less work has focused on the externalizing pathway to BPD. Further, little research has explicated the role of the family environment in shaping the development of BPD features among children and youth who have high levels of impulsivity. This chapter reviews literature that integrates individual- and family-level characteristics with a focus on the externalizing pathway to BPD. Specifically, in a closer examination of the childhood characteristics in the biosocial model (Crowell et al., 2009; Linehan, 1993), we posit that a subset of impulsive youth will go on to develop BPD; specifically those characterized by *emotional impulsivity*, that is those who engage in impulsive behaviors or actions when in a strong positive or negative mood state. We propose that these individual characteristics within the context of a highly conflictual and invalidating family relationships will transact over time ultimately resulting in BPD (see Fig. 17.1).

Internalizing-Externalizing Dimensions of Psychopathology and BPD

Although BPD is a unitary construct, BPD symptoms are a mix of emotional instability (e.g., frequent displays of inappropriate anger) and impulsivity (e.g., impulsiveness with money, substance use, promiscuous sexual

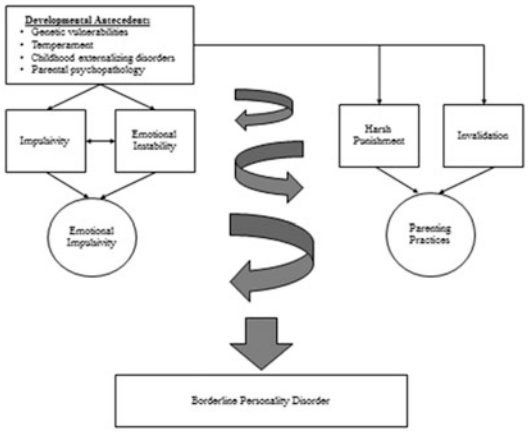

Fig. 17.1 Children with high levels of emotional impulsivity in the context of harsh punishment and invalidating parenting practices are at increased likelihood of developing borderline personality disorder. The bidirectional arrows highlight the transactional, reciprocal nature of children's impulsivity and parenting practices in the developmental course of this disorder

behavior, binge eating, or other risky behavior). These core features have also been implicated in the development of BPD. For example, Trull (2001) found that personality traits assessing impulse control and a component of emotional instability, negative affectivity, predicted BPD features in young adults over a 2-year time period. In an extension of the biosocial model of BPD development, trait impulsivity and emotional instability are two developmental precursors that increase vulnerability for the development of BPD (Crowell et al., 2009). These factors are likely to be recognized early in childhood and, in the context of deleterious family and social transactions, may lead to the development of BPD. Once the disorder emerges, individuals are likely to engage in impulsive behavior in the face of high levels of emotion instability, thus, these features are inextricably linked by this point in development.

When compared to other psychiatric disorders, BPD in adults is distinct in the nosological landscape by its position on both the internalizing and externalizing dimensions of psychopathology (Eaton et al., 2011; James & Taylor, 2008). Interestingly, suicide behavior has been specifically linked to the externalizing dimension of psychopathology even after controlling for internalizing symptoms as well as comorbid internalizing-externalizing symptoms (Verona, Sachs-Ericsson, & Joiner, 2004). For women compared to men, suicide behavior was more strongly linked to comorbid internalizing-externalizing symptoms, suggesting that this feature in BPD may emerge from the combined internalizing-externalizing features of the disorder, especially for women. Using a large general US population survey of adults, Eaton et al. (2011) found that BPD fit best on both the distress and externalizing factors for both men and women. Additionally, James and Taylor (2008) reported similar findings in a large cohort sample of young adults aged 19–22. Specifically, they found that BPD was situated on both the distress and externalizing factors for men. However, for women they found that two models were equally satisfactory, one with BPD located on both the distress and externalizing factors and the other with BPD located on the distress factor only.

These findings have the potential to shed light on the developmental course and etiology of BPD in addition to helping to explain the pattern of comorbidity that is often observed among individuals with BPD. That is, BPD shares liability with both internalizing and externalizing forms of psychopathology, which can help explain why it is likely to be comorbid with many forms of internalizing psychopathology, such as major depressive disorder, and is also likely to be comorbid with externalizing psychopathology, such as substance use disorders (Eaton et al., 2011).

These findings suggest that what distinguishes individuals with BPD from those with internalizing disorders alone, such as mood and anxiety disorders, is the propensity to engage in impulsive and risky behavior; likewise, what distinguishes individuals with BPD from those with externalizing disorders alone is the propensity for extreme mood lability and emotional reactivity. As a result, we understand that emotional instability and impulsivity may be the two independent drivers of the ultimate manifestation

of the signs and symptoms hallmark of a BPD diagnosis. Alternatively, it may be that emotional instability and impulsivity overlap to result in the hallmark behaviors of BPD, such as self-harm, suicide behaviors, and extreme reactions to fears of abandonment. For those with BPD, extreme emotion can often be thought of as the fuel required to ignite impulsive behavior. These individuals may not necessarily engage in such rash behaviors when not distressed. Specifically, individuals with BPD may have higher levels of positive and negative urgency facets of impulsivity that increase the likelihood of acting rashly in response to a strong positive or negative mood (Cyders & Smith, 2007). For example, when feeling shame and anger in response to an argument with a significant other, the emotionally impulsive individual may hastily pack up their belongings to move. Thus, within the context of experiencing strong emotions, individuals with high levels of negative and/or positive urgency will make impulsive decisions and engage in impulsive behavior and may be most at risk for BPD.

The Externalizing Pathway to BPD During Adolescence

Recognizable symptoms and features of BPD are likely to first manifest during adolescence (Bradley, Conklin, & Westen, 2005; Westen & Chang, 2000). Normative adolescent development consists of engagement in more independent emotion regulation and self-control strategies. Research has shown that while impulsivity declines on average across adolescence (Steinberg et al., 2008), there are notable differences seen in this "maturation" and this is related to risk-taking behavior (e.g., Johnson, Brent, Bridge, & Connolly, 2007; Monahan, Steinberg, Cauffman, & Mulvey, 2009). Thus, deficits in self-regulatory skills and steady or increasing levels of impulsivity become more apparent during this developmental period as the majority of adolescents normatively experience increased self-control. The group of adolescents with continued self-control deficits may be particularly at risk for developing BPD during this period. Specifically, self-injury, a hallmark feature of this disorder, has been reported to onset by adolescence in 2/3 of BPD patients (Zanarini et al., 2006). Additionally, other risk-taking behaviors, such as substance use and risky sexual behavior, also increase during adolescence (Steinberg et al., 2008). For adolescents with BPD symptoms, this may result in associating with deviant peer groups, increased parental conflict and environmental shifts that continue the escalation of these symptoms to meeting full BPD diagnosis.

High levels of impulsivity and emotional instability increase the likelihood of experiencing a chaotic environment as well as interfere with normative developmental processes of increasing autonomy and self-control. This likely results in youth with these symptoms being at increased risk for poor outcomes into and during adulthood. For example, BPD symptoms during the college years have been found to be associated with poor social and academic outcomes (Bagge et al., 2004; Trull et al., 1997). BPD symptoms during adolescence have also been shown to predict worse functioning over several decades. In a large community sample, higher levels of BPD symptoms during adolescence predicted less productive adult role functioning over 20 years, including poor academic and occupational achievements (Winograd, Cohen, & Chen, 2008). BPD symptoms also predicted poor social and relationship functioning, including lack of social support and less involvement in romantic relationships well into adulthood. Thus, it does not appear that BPD symptoms in adolescence and early adulthood reflect a transitory problem in functioning, but may indicate a poor prognosis for some youth. We will shed light on the development of BPD through the externalizing pathway by reviewing the developmental antecedents that give rise to the impulsivity component of the disorder.

Antecedents of the Externalizing Pathway to BPD

Research has only just begun to identify developmental precursors and risk factors of BPD (Cohen, Crawford, Johnson, & Kasen, 2005; Crawford, Cohen, Chen, Anglin, & Ehrensaft, 2009; Johnson, Smailes, Cohen, Brown, & Bernstein, 2000; Lenzenweger & Cicchetti, 2005; Winograd et al., 2008; Yen et al., 2004). So far, studies suggest several putative antecedents to the later development of BPD, including difficult child temperament (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2006), childhood externalizing disorders (Burke & Stepp, 2012; Stepp, Burke, Hipwell, & Loeber, 2012), parental psychiatric disorders (White, Gunderson, Zanarini, & Hudson, 2003), poor parenting practices (Johnson, Cohen, Chen, Kasen, & Brook, 2006, Johnson, Cohen, Kasen, & Brook, 2006), and dysfunctional family environments (Affifi et al., 2011; Fruzzetti et al., 2005; Zanarini et al., 1997). In subsequent sections we will review empirical research on individual- and family-level characteristics, as well as their interaction that lead to BPD via an externalizing pathway.

Individual Characteristics

Genetic Vulnerabilities

There is good evidence for the transgenerational transmission of this disorder (for a review, see White et al., 2003), which highlights the genetic vulnerability in those with BPD. For instance, family studies assessing the rates of BPD diagnoses and related traits in first-degree relatives have found a 4- to 20-fold increase in prevalence or morbidity risk for BPD compared to the general population (e.g., Barnow, Spitzer, Grabe, Kessleer, & Freyberger, 2006; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1988). Research supports an even stronger familial aggregation of core features of BPD, namely emotional instability and impulsivity, compared to the fully diagnosed disorder (Silverman et al., 1991). These features have been found to aggregate separately, suggesting that they may be inherited independently. Given the familial aggregation of these traits, it is not surprising that relatives of probands with BPD are also at increased risk for related psychiatric disorders, including major depressive disorder, substance use disorders, and antisocial personality disorder (Riso, Klein, Anderson, & Ouimette, 2000; Schulz, Soloff, Morganstern, Di Franco, & Schulz, 1989; Zanarini et al., 1988).

Given the high rate of family transmission with the disorder and associated features, offspring of parents with BPD may inherit genes predisposing them to emotional instability and/or impulsivity. Twin studies offer evidence for the genetic transmission of BPD. In a large, multinational community-based adult twin sample, Distel et al. (2008) reported a heritability estimate of 42 % for BPD features. Torgersen et al. (2000) reported a much higher heritability estimate of 69 % for the diagnosis of BPD in a relatively small twin sample of clinic-referred adult participants. The discrepancy in heritability estimates is likely due to differences in sample size and sample ascertainment across the two studies. In addition, genetic influences may be stronger for individuals with more extreme forms of the disorder (i.e., those that are clinically referred and carry the diagnosis). Thus, findings from the population-based study suggest a relatively strong influence for both genetic and unique environmental experiences in accounting for variation in BPD features. More work is needed to understand the genetic underpinnings of the specific impulsivity and emotional instability features of the disorder.

Child Temperament

Temperamental characteristics such as negative emotionality and poor impulse control are widely recognized in virtually all etiological theories as early markers or precursors of BPD traits. Temperament is defined as the physiological basis for

individual differences in reactivity and self-regulation and is heritable and relatively stable (Rothbart & Bates, 2006). In one study, infant activity and emotionality at 30 months were positively correlated with BPD symptoms at age 28, but infant impulsivity was not related to later BPD (Carlson, Egeland, & Sroufe, 2009). However, studies with adolescent samples have demonstrated associations between BPD features, such as non-suicidal self-injury, and physiological correlates of emotional instability and impulsivity (Crowell et al., 2005, 2009, 2012). A recent prospective study with a large twin sample showed that impulsivity and behavioral and affective dysregulation at age 5 predicted BPD features at age 12 (Belsky et al., 2012). These findings highlight the importance of both impulsivity and emotional instability early in development in increasing risk for BPD and point to the possibility that emotionally impulsive children may be most at risk.

Childhood Externalizing Disorders and Comorbidity with BPD

Furthermore, evidence suggests that externalizing problems, such as CD and ODD, during childhood and early adolescence are prospectively associated with BPD symptoms in adolescent girls (Stepp et al., 2012) and young men (Burke & Stepp, 2012). Externalizing problems may be precursors of BPD due to the fact that these problems share underlying features of impulsivity and some aspects of emotional instability, such as negative affectivity. Common factors underlie childhood externalizing disorders (ADHD, CD, and ODD) and BPD. These shared factors include behavioral and neurocognitive impairments, as well as emotional and interpersonal disturbances, particularly anger and hostility, which point to possible developmental links between these childhood disorders and BPD. Burke and Stepp (2012) used prospective data from the Developmental Trends Study (Loeber, Green, Lahey, Frick, & McBurnett, 2000), a clinic-referred sample of 177 boys, to test relationships among childhood psychiatric disorders and BPD in young adulthood. They found that childhood and adolescent symptoms of ODD and ADHD as well as marijuana use predicted BPD symptoms at age 24. Upon further examination regarding which factors of ODD predicted BPD, the oppositional behavioral symptoms (e.g., argumentativeness), but not the affective symptoms (e.g., anger), were uniquely related to BPD even after accounting for symptoms of other personality disorders. The findings regarding the lack of prospective association between certain childhood psychiatric disorders and BPD are also of interest. Specifically, conduct disorder (CD), depression, and anxiety were not related to BPD symptoms in young adulthood. Additionally, when other personality disorder symptoms were included in analyses, ADHD and marijuana use were no longer related. These findings suggest a unique prospective association between the behavioral dimension of ODD and BPD in young adult men.

Stepp et al. (2012) provided another examination of this topic using data from the Pittsburgh Girls Study (Keenan et al., 2010), an accelerated longitudinal cohort design which oversampled families living in poverty. This study used data from girls in the two oldest cohorts ($N = 1{,}233$) and included data spanning ages 8–14 years. These authors examined the childhood and adolescent developmental trajectories of ADHD and ODD symptoms in predicting BPD symptoms during adolescence. Given the overlap among symptoms of childhood psychiatric disorders and BPD symptoms, this study rigorously controlled for childhood symptoms of CD and depression. Additionally, to examine whether ADHD and ODD trajectories would specifically predict BPD symptoms during adolescence, adolescent symptoms of CD and depression were included as outcomes. The authors found that ADHD and ODD symptoms at age 8 predicted BPD symptoms at age 14. Moreover, the rate of growth in ADHD symptoms from ages 8–10 years and the rate of growth in ODD symptoms from age 10–13 predicted BPD symptoms at age 14. These patterns of prospective associations were not found for CD and depression at age 14. These results highlight a potential developmental pathway from childhood

ADHD and ODD to BPD in adolescence. Increasing ADHD symptoms from ages 8–10 years and increasing ODD symptoms from 10 to 13 years may indicate risk for girls who will develop BPD symptoms in adolescence. These two papers highlight specific childhood externalizing disorders that may place youth at risk for BPD. These precursors appear to impact girls and boys similarly and highlight the shared features among children with ADHD and ODD who may later develop BPD.

Family Characteristics

Parental Psychopathology

Several studies suggest that parental psychopathology serves as a risk factor for BPD in offspring (e.g., Belsky et al., 2012; Bradley, Jenei, & Westen, 2005; Helgeland & Torgersen, 2004; White et al., 2003). In particular, there is evidence that retrospectively reported parental antisocial problems and mood disorders are associated with BPD features in young adult offspring (Trull, 2001). Antisocial features and mood disorders in parents have also been shown to predict children's internalizing and externalizing problems at various stages of development (Zahn-Waxler, Druggal, & Gruber, 2002). Stepp et al. (2013) found that maternal BPD and paternal substance use were predictive of BPD symptoms in adolescence, even after controlling for other adolescent factors, such as suicide behaviors and childhood psychiatric disorders. Hence, parental psychopathology, especially antisocial behaviors, substance use, or depressed mood, likely increases the risk for BPD features during adolescence via increasing liability for offspring internalizing and externalizing disorders. One of the mechanisms of this transmission is likely parenting.

Parenting Practices

Developmental models of BPD posit that invalidating parenting experiences transact with a child's genetic vulnerabilities to put them at risk for the emergence of BPD (Fruzzetti et al., 2005; Linehan, 1993). The specific characteristics that constitute "invalidating parenting" have yet to be reliably defined and measured. Using Linehan's (1993) original definition as a guide, empirical work suggests that invalidating parenting relationships are characterized by emotional and/or physical neglect, dysfunctional parenting practices (including parents' emotional and behavioral responses to their children), and poor parent–child relationship quality (Eisenberg, Cumberland, & Spinrad, 1998; Eisenberg et al., 1999; Gottman, Katz, & Hooven, 1997). Specific parenting practices or parenting behaviors defined by both content and socialization goals (Morris, Silk, Steinberg, Myers, & Robinson, 2007) can contribute to invalidation in the family environments in those at risk for the development of BPD. In normative samples, negative, invalidating parenting behaviors have been associated with social and emotional difficulties throughout childhood (Eisenberg et al., 1999; Kiff, Lengua, & Zalewski, 2011; Silk et al., 2009) and psychological distress in adulthood (Krause, Mendelson, & Lynch, 2003).

Parental control or discipline is one of the primary ways in which parents socialize their children (Bates & Pettit, 2007; Morris et al., 2007). In general, harsh discipline has been associated with worse child outcomes, including delinquent and problem behaviors (Bender et al., 2007; Koenig, Ialongo, Wagner, Poduska, & Kellam, 2002). For example, Bender et al. (2007) found that reports of maternal and paternal harsh discipline were associated with concurrent adolescent externalizing behaviors. In a large meta-analysis of the effects of corporal punishment on later child behavior outcomes, Gershoff (2002) found that higher levels of physical punishment were associated with significant increases in child delinquent and aggressive behaviors.

The Children in the Community Study (CIC; Cohen et al., 2005) has prospectively assessed the development of personality disorders in a community sample of youth studied into adulthood. This study has shown that the combination

of maternal inconsistency in parenting practices and maternal emotional over-involvement predicted the persistence or emergence of BPD (and not other personality disorders) 2.5 years later during adolescence (Bezirganian, Cohen, & Brook, 1993). Parenting behaviors during childhood, specifically low warmth and harsh punishment, were also shown to predict BPD during adulthood (Johnson, Cohen, Kasen, & Brook, 2006). Winsper, Zanarini, and Wolke (2012) also found a prospective relationship between harsh punishment and risk for BPD in children. Another more recent prospective study with a low-income community sample showed that BPD symptoms in offspring at age 28 were related to early relational experiences, including maternal hostility, boundary dissolution, and family life stress (Carlson et al., 2009). However, these studies did not examine the reciprocal influences of parenting and BPD symptoms.

Most research has asked patients with BPD to retrospectively report on the parental care that they received as children. In such studies, BPD patients portray the parenting that they received as children quite unfavorably (Sansone & Sansone, 2009), often describing their parents as neglectful, invalidating, over-involved, and indifferent (Gunderson & Lyoo, 1997; Weaver & Clum, 1993; Zweig-Frank & Paris, 2002). These individuals also describe relationships with caregivers and the ambience in their households as conflictual and inconsistent (Zanarini et al., 2000). A series of studies by Gratz and colleagues (Gratz, 2006; Gratz, Conrad, & Roemer, 2002; Gratz et al., 2011; Gratz, Latzman, Tull, Reynolds, & Lejuez, 2011) have retrospectively linked both BPD features and self-injury to caregiver emotional neglect and lack of behavioral control. Several independent lines of research have also confirmed an association between parental emotional unavailability and neglect and BPD features, such as self-injury (Bureau, Easterbrooks, & Lyons-Ruth, 2009; Helgeland & Torgersen, 2004; Lyons-Ruth, Choi-Kain, Pechtel, Bertha, & Gunderson, 2011).

Individual and Family-Level Transactions

Gene X Environment Risk

Gene–environment interaction models demonstrate the importance of an individual's unique social environment in moderating the effects of genes on the development of psychopathology and other maladaptive outcomes (Cacioppo, Bernston, Sheridan, & McClintock, 2000). Parenting serves as an important environmental context for offspring of mothers with BPD. Theoretical models (Fruzzetti et al., 2005; Linehan, 1993) posit that invalidating parenting experiences transact with a child's genetic vulnerabilities to put them at risk for poor psychosocial outcomes, including BPD and related psychopathology. For instance, negative parent–child relationships can exacerbate both the internalizing and externalizing symptoms in youth who have high levels of emotional instability (Feinberg, Kan, & Hetherington, 2007; Huh, Tristan, Wade, & Stice, 2006). Other evidence also suggests that warm and accepting parenting can shield a child from negative outcomes associated with genetic and physiological vulnerabilities (Eley et al., 2004). Although it is impossible to modify a child's genetic vulnerabilities, parenting practices may be modified and thus offer an environmental context ripe for prevention and intervention efforts.

Bidirectional Parent–Child Influences

Unfortunately, research on developmental antecedents reviewed above has not assessed the impact that the child's early BPD features and symptoms may have on parenting behaviors. BPD symptoms are often viewed as a consequence, rather than as a driving force of these associations. However, children with high levels of impulsivity and emotional instability may evoke more negative parenting behaviors over

time, further increasing risk for BPD. Accordingly, bidirectional relations have been found between child temperament and parenting in healthy samples, such that inconsistent discipline increased negative emotionality in middle childhood, and child irritability led to more inconsistent discipline by parents (Lengua & Kovacs, 2005).

Both parent and child-driven effects create change in behaviors and symptoms across development (Sameroff, 1975). In healthy samples, there is evidence for reciprocal influences when both the adolescent and parent are engaged in negative behaviors during problem-solving interactions (Rueter & Conger, 1998). Using a large, longitudinal sample, Rueter and Conger (1998) found evidence for the bidirectional nature of parent–adolescent interactions, particularly when behavior was negative. For example, if both the parent and adolescent displayed ineffective or coercive behavior, the interactions between them grew more negative over time. Interestingly, the authors also found evidence for declines in nurturing parenting over time when the adolescent alone was disruptive and inflexible. Finally, harsh/inconsistent parenting was related to lower levels of flexible, involved adolescent behavior, indicating that this parenting strategy discouraged the development of effective adolescent problem-solving skills. In addition, supportive parenting behaviors declined over time when the adolescent behaved consistently negative and inflexible.

There is ample evidence for bidirectional influences between parenting practices and child internalizing and externalizing problems (Pardini, 2008). In a study of 496 adolescent girls, Huh et al. (2006) found that externalizing problems predicted a decreased perception of parental support and parental control 1 year later, but did not find support for the converse. However, the authors noted reciprocal relations between adolescent substance use and decreased perceived parental control. Bidirectional influences have also been shown between parenting practices and children's behavior problems and callous-unemotional traits (Hawes, Dadds, Frost, & Hasking, 2011; Larsson, Viding, Rijsdijk, & Plomin, 2008. Hawes et al. (2011) found that callous-unemotional (CU) traits uniquely accounted for change in three domains of parenting (inconsistent discipline, punishment, and parental involvement). Likewise, multiple domains of parenting (positive parenting, parental involvement, and poor monitoring/supervision) uniquely predicted change in CU traits. In addition, Larsson et al. (2008) found that antisocial behavior at age 4 independently explained 3.1 % of the heritability in parental negativity at age 7; thus, genetically influenced child antisocial behavior evokes parental negativity. Related research has shown that parental hostility is both a response to adolescent antisocial behaviors and a contributing factor to the development of these behaviors (Scaramella, Conger, Spoth, & Simons, 2003). Depressive symptoms in adolescence have also been shown to negatively predict the quality of the parent–adolescent relationship (Branje, Hale, Frijns, & Meeus, 2010) and to elicit more hostile parenting responses and decreases in warm parenting techniques, which may relate to further adolescent withdrawal and depression (Kim, Conger, Lorenz, & Elder, 2001; Slesnick & Waldron, 1997).

Having a child with BPD symptoms may be perceived by parents as a burden (Goodman et al., 2011), and this may negatively impact the way parents respond to their child. In turn, these negative effects on parenting responsiveness may further increase the child's maladaptive behaviors. Additionally, many parents report difficulties adjusting to their adolescent's individuation and autonomy-striving (Silverberg & Steinberg, 1990). Parents of adolescents with BPD symptoms may have an even harder time coping with this developmental change, especially if the adolescent engages in risky and problematic behaviors that parents perceive as worthy of discipline. In response to such behaviors, parents may increase the amount of control and discipline they exercise. In addition, if the adolescent is engaging in risky and dangerous behaviors, these are not likely to evoke parental warmth. Thus, adolescents who develop BPD are likely to be in a family environment characterized by low emotional warmth and

high expectations regarding behavioral control. These parenting behaviors are likely to have been shaped over time by the child's extreme emotional reactions and displays of dangerous behaviors.

Future Research and Clinical Implications

Taken together, the findings reviewed above suggest that: (1) child characteristics, particularly temperament and personality traits related to impulsivity and emotional instability, may be biologically influenced precursors to BPD and (2) facets of harsh and invalidating parenting and parental psychopathology may also contribute to the development of BPD, exacerbating impulsive behaviors over time. However, most previous research on the development of BPD has relied on the retrospective reports of adults, and few longitudinal studies are able to prospectively examine links between childhood impulsivity, environmental experiences, and BPD. Furthermore, previous work is limited by the use of univariate models of risk factors to predict BPD, which fail to account for the bidirectional and transactional nature of the relations between these interdependent developmental processes. BPD symptoms and parenting may influence each other via two processes: First, the trait-like, stable components of BPD symptoms and parenting behaviors may be associated over time. Second, state-like, year-to-year variations in BPD symptoms and parenting behaviors may also be linked. Linehan's biosocial theory (1993) and other prominent theoretical models (Crowell et al., 2009; Fruzzetti et al., 2005) emphasize the transactional nature of the development of BPD, with child characteristics (e.g., temperament) and characteristics of the environment (e.g., caregivers' responses) interacting with each other over time, with both the trait- and state-components of these processes influencing one another. Although none of the identified developmental antecedents are, taken in isolation, unique to the development of BPD, the use of multivariate longitudinal models incorporating several putative risk factors creates a more robust model of prospective risk that may include unique pathways to BPD.

More research is urgently needed that utilizes multimodal assessment batteries of behaviors and traits in impulsivity, parenting strategies, and parent–youth transactions. These processes should be examined at multiple different levels of resolution: in real-time (e.g., ecological momentary assessment protocols, observational ratings, autonomic reactivity monitoring, experimental manipulations) charting the course of their development over months and years. Better understanding these transdiagnostic features (emotional instability, impulsivity, and emotional impulsivity) will also inform how parent–youth transactions are related to near-neighbor internalizing and externalizing problems (e.g., depression, conduct problems). This type of research agenda will likely have the most translatable impact to work with vulnerable youth and families.

Supporting families who have these vulnerable children should be a top clinical priority and may decrease risk for developing more serious conditions in adolescence and young adulthood. Practitioners are encouraged to discuss these issues with patients and their families. It is also important to highlight that BPD can be reliably and meaningfully diagnosed during adolescence (Miller, Muehlenkamp, & Jacobson, 2008). However, there is resistance to diagnose adolescents with this disorder, especially in clinical practice (Griffiths, 2011). The fear surrounding the BPD diagnosis in many mental health treatment facilities coupled with the lack of access to treatment for youth with BPD may contribute to the unwillingness to diagnose this condition prior to adulthood. However, a proper diagnosis is necessary for appropriate treatment. Without awareness of the number of youth in need, it is unlikely that more services will be made available for this population. Furthermore, the reluctance to diagnose or study BPD in youth results from the notion that personality traits are not stable until adulthood. However, the long-term stability of personality disorders has recently been called into question for both adolescent

and adult clinical samples. It appears that BPD remits within 3 years for most individuals regardless of age (Mattanah, Becker, Levy, Edell, & McGlashan, 1995; Meijer, Goedhart, & Treffers, 1998; Shea et al., 2002; Zanarini, Frankenburg, Hennen, & Silk, 2003). It is important to note that when studies have considered a more dimensional approach toward classification, the stability and reliability of symptoms and features are higher (Clark, 2009). Specifically, evidence suggests that although there is a decline in the mean level of BPD traits from adolescence into young adulthood, the rank-order stability of these traits is high during this developmental period and parallels the stability of BPD traits found during adulthood (Bornovalova, Hicks, Iacono, & McGue, 2009; Lenzenweger, 1999).

Given the impairment associated with BPD symptoms, it is not surprising that individuals with BPD are likely to first seek treatment during adolescence. There is hope for improvements in the quality of lives of youth who present for treatment with these problems. Several clinical trials have been conducted with adolescents who have BPD and have been shown to be effective in reducing symptoms (Chanen et al., 2008; Klein & Miller, 2011). In sum, there is an urgent need for assessment and treatment services for impulsive youth who also have difficulty with emotion regulation during childhood and adolescence as BPD can develop during adolescence. Early detection and treatment may lead to improved outcomes for these youth.

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Childhood Adversity and the Development of Borderline Personality Disorder

18

Mary C. Zanarini and Michelle M. Wedig

Introduction

Borderline personality disorder (BPD) is both a common and serious psychiatric disorder. It affects about 2 % of American adults (Swartz, Blazer, George, & Winfield, 1990) and is associated with high levels of psychiatric care as well as high levels of social service utilization (Skodol, Buckley, & Charles, 1983; Swartz et al., 1990). The etiology of BPD has been a source of intense clinical interest for almost 40 years. The first attempt to explain the development of BPD came from the psychoanalytic community and over the years, three major psychodynamic theories of the pathogenesis of the disorder have been proposed.

In the first of these theories, Kernberg (1975) suggests that excessive early aggression has led the young child to split his or her positive and negative images of him or herself and his or her mother. This excess aggression may have been inborn or it may have been caused by real frustrations. In either case, the preborderline child is unable to merge his or her positive and negative images and attendant affects to achieve a more realistic and ambivalent view of him or herself and others.

In the second of these theories, Adler and Buie (1979) suggest that failures in early mothering have led to a failure to develop stable object constancy. Because the preborderline child's mothering was inconsistent and oftentimes insensitive and nonempathic, the child fails to develop a consistent view of him or herself or others that he or she can use in times of stress to comfort and sustain him or herself.

In the third of these theories, Masterson (1972) suggests that fear of abandonment is the central factor in borderline psychopathology. He believes that the mother of the future borderline patient interfered with her child's natural autonomous strivings by withdrawing emotionally when the child acted in an independent manner during the phase of development that Mahler (1971) has termed "separation-individuation." Later experiences that require independent behavior lead to a recrudescence of the dysphoria and abandonment panic that the borderline patient felt as a child when faced with a seemingly insoluble dilemma (either continue to behave dependently or lose needed emotional support).

First-Generation Studies of the Pathogenesis of BPD

While these theories contain much clinical wisdom, it was difficult for many observers to believe that such a serious psychiatric disorder would typically be the result of such relatively subtle childhood difficulties. Nonetheless, the first

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generation of studies of the environmental factors that might be of etiological significance for BPD focused on issues raised in these psychodynamic theories. Two topics were studied with particular care: parental separation or loss and disturbed parental involvement.

Studies of Parental Separation or Loss

Grinker was the first to study BPD, finding in a sample of hospitalized nonpsychotic patients that there were four subtypes of borderline patients (1968). In a follow-up study of this sample, Walsh (1977) found that a majority of families of borderline patients (57 %) had histories of parental loss through divorce or death; a significantly higher percentage than was found in a group of matched schizophrenic comparison subjects. In addition, half the borderline patients had experienced a serious chronic parental illness which often required extensive hospitalization. Only 21 % of the patients with BPD came from families that had not experienced the loss of a parent through death, divorce, or serious illness.

Soloff and Millward (1983) compared separation experiences in the background of 45 patients with BPD to those of depressed and schizophrenic comparison groups. They found that patients with BPD were significantly more likely to come from broken families than those in either control group. They also found that patients with BPD had a significantly higher incidence of loss of their fathers by divorce or death (47 %). Akiskal et al. (1985) found that 37 % of borderline patients had experienced a developmentally important loss, a percentage that was significantly higher than that of affective comparison subjects and significantly lower than that of personality-disordered comparison subjects.

Taken together, the results of these early studies suggest that loss was common in the childhood experiences of borderline patients. These results also suggest that such losses were significantly more common among those with BPD than among comparison subjects.

Studies of Disturbed Parental Involvement

The original study to characterize the families of borderline patients (Grinker, Werble, & Drye, 1968) found that a minority (12.8 %) were characterized by relationships in which the parents were overinvolved and overprotective. Another nine families were characterized by a pervasive denial of problems, which was evidenced by the absence of marital discord and the lack of strong parental affect of either a positive or negative nature. However, the most common pattern observed in these families (about a third) was a high degree of discord between the mother and her children and between the two parents.

Subsequently, Walsh (1977) found that a greater percentage (57 %) of patients with BPD felt that they were overinvolved with one parent with whom they had a special relationship. These relationships were judged as supportive of the parent's need to be needed, but destructive to the patient's need to have a life of his or her own. Walsh also found that most (87 %) of the borderline patients characterized their relationship with one or both parents as remote or lacking in feelings of attachment. In addition, she found that 64 % of the borderline sample reported strongly negative, highly conflictual relationships with their parents, which were characterized by parental hostility, devaluation, or frank abuse.

Gunderson, Kerr, and Englund (1980) studied three groups of patients who had intact families: those with borderline personality, paranoid schizophrenia, and neurosis/other axis II disorders. The borderline patients had parents who were found to be less likely than those of neurotics, or patients who function reasonably well but suffer from inner pain about which others may not know, but more likely than those of schizophrenics to invest in their children at the expense of their marriage. More generally, results failed to show a high level of overinvolved families for the borderline patients but rather that their parents were involved with one another to the exclusion of their children.

Frank and Paris (1981) compared the accounts of parental attitudes of three patient samples: those with BPD, those with neuroses/other personality disorders, and healthy comparison subjects. All three groups reported disturbed attitudes in their mothers. In addition, the borderline group remembered their fathers as significantly less interested in and less approving of them in general than did the other two groups. Their fathers, more specifically, were reported to be less interested in and less approving of dependent behaviors than fathers of comparison subjects with neuroses/other personality disorders. In a subsequent small study, Frank and Hoffman (1986) found that females with BPD remembered both their mothers and fathers as significantly less nurturant and less affectionate than did neurotic comparison subjects. In a third study by this group, Paris and Frank (1989) found that borderline women perceived their parents as significantly less caring than comparison women. In a fourth study by this group, Zweig-Frank and Paris (1991) studied the childhood recollections of a mixed gender sample of 62 borderline patients and compared these recollections to those reported by 99 nonpsychotic psychiatric comparison subjects. They found that the borderline patients remembered both their fathers and their mothers as having been significantly less caring and more protective/controlling than did the nonborderline patients.

Soloff and Millward (1983) found that inpatients with BPD, as well as comparison subjects with depression and schizophrenia, saw their mothers as being overinvolved with them. The patients with BPD were, however, significantly more likely to report their fathers as being underinvolved than patients from either control group. Soloff and Millward also reported that the borderline patients in their study saw their relationships with their mothers and fathers as being significantly more negative and conflictual than did the two comparison groups.

Goldberg, Mann, Wise, and Segall (1985) retrospectively assessed the parental attitudes of 24 patients with BPD, 22 general psychiatric comparison subjects, and ten normal comparison subjects using a self-report questionnaire. They found that patients with BPD remembered both their parents as significantly less caring than did those in either comparison group. They also found that patients with BPD remembered their parents as significantly more overprotective than did the healthy comparison subjects.

Torgersen and Alnaes (1992) studied care and protection in the childhood histories of 36 borderline patients, 19 schizotypal patients (five of whom also met criteria for BPD), 165 patients with other types of personality disorders, and 52 patients without substantial axis II pathology. Borderline patients reported lower maternal and paternal care than patients with other personality disorders and no personality disorders. They also reported more maternal protection than schizotypal patients.

Four conclusions emerged from these studies: (1) prolonged childhood separations are both common and discriminating for borderline patients; (2) patients with BPD usually see their relationships to their mothers as highly conflictual, distant, and/or overprotective; (3) the father's failure to be present and involved is an even more discriminating aspect of these families than the mother's problems; and (4) disturbed relationships with both parents may be both more specific for BPD and pathogenic than that with either one alone.

Second-Generation Studies of the Pathogenesis of BPD

The second generation of studies of the environmental factors that may be pathogenic for BPD built upon the methodological limitations of the studies reviewed above. Most of the second-generation studies that will be reviewed below (see Table 18.1 for details) have incorporated the three following methodological advances: (1) diagnoses were determined using semistructured interviews, (2) childhood experiences were assessed using semistructured interviews, and (3) diagnostic and childhood information was obtained blind to information pertaining to the other domain. Second-generation studies have also tended to focus more on childhood experiences of abuse (and

Table 18.1 Prevalence of childhood physical and sexual abuse in criteria-defined adult borderline patients

Study	*N*	BPD comparison subjects	Tx status	Gender	Physical abuse	Caretaker sexual abuse	Overall sexual abuse
Links et al.	88	42 BPD trait	Inpatients	Mixed	29[a]	26[a]	–
Zanarini et al.	50	55 Axis II	Outpatients	Mixed	46	26[a]	–
Herman et al.	21	34 Mixed	Outpatients/ symptomatic volunteers	Mixed	71	–	67[a]
Ogata et al.	24	18 Depressed	Inpatients	Mixed	42	25	71[a]
Shearer et al.	40	–	Inpatients	All female	25	28	40
Salzman et al.	31	–	Symptomatic volunteers	Mixed	10	0	16
Paris et al.	78	72 Axis II	Outpatients	All female	73[a]	29	71[a]
Paris et al.	61	60 Axis II	Outpatients/ symptomatic volunteers	All male	66	12[a]	48[a]
Zanarini et al.	358	109 Axis II	Inpatients	Mixed	59[a]	27[a]	62[a]

[a]Reported by a significantly higher percentage of borderline patients/subjects than comparison subjects

neglect) than on childhood experiences of parental loss and disturbed parental involvement.

Links, Steiner, Offord, and Eppel (1988) and Zanarini, Gunderson, Marino, Schwartz, and Frankenburg (1989) published their results almost simultaneously. Links et al. compared the childhood experiences of 88 borderline inpatients to 42 inpatients with borderline traits. They found that borderline patients were significantly more likely than comparison subjects to have reported being sexually abused by a caretaker, being physically abused by a caretaker, and being separated from their primary caretaker for a period of 3 months or more.

Zanarini et al. compared the childhood experiences of 50 borderline outpatients to those of 55 outpatient axis II subjects. They found that a significantly higher percentage of borderline patients than axis II comparison subjects reported being verbally abused and sexually abused by a caretaker before the age of 18. They also found that rates of physical abuse by caretakers and three forms of caretaker neglect (i.e., physical neglect, emotional withdrawal, and inconsistent treatment) did not significantly distinguish borderline patients from axis II comparison subjects. In terms of early separations that lasted a month or more, about equal percentages of borderline patients and axis II comparison subjects reported such a separation from a caretaker before the age of 6. However, when the comparison subjects were broken down into those who met DSM-III criteria for antisocial personality disorder (who were mostly men) and those who met DSM-III criteria for dysthymic disorder plus some other form of personality disorder (who were mostly women), a significantly higher percentage of borderline than antisocial patients reported at least one such early childhood separation. A significantly higher percentage of borderline than antisocial patients also reported having a caretaker withdraw from them emotionally.

Herman, Perry, and van der Kolk (1989) compared the childhood histories of physical and sexual abuse of 21 borderline outpatients/symptomatic volunteers to those of 11 with borderline

traits and 23 with other axis II disorders or bipolar II disorder. When compared ordinally (by summing across age periods), both physical and sexual abuse before the age of 19 were significantly more common among borderline patients than comparison subjects. However, when compared nominally, sexual abuse continued to significantly distinguish borderline patients from comparison subjects but physical abuse no longer successfully discriminated the groups.

Ogata et al. (1990) compared the childhood experiences of 24 borderline inpatients with those of 18 depressed inpatients. They found that a significantly higher percentage of borderline patients than comparison subjects reported being sexually abused during childhood and/or adolescence. They also found that borderline patients reported a high rate of physical abuse and a low rate of physical neglect. However, neither type of pathological experience was significantly more common among borderline patients than comparison subjects. In terms of the parameters of abuse, 21 % of the borderline patients reported being abused by their father, 4 % by their mother, 29 % by a sibling, 25 % by another relative, and 50 % by a nonrelative. Fifty-three percent reported being abused by multiple perpetrators and 41 % reported penetration. The average age of onset was between 7 and 10 years (depending on the relationship to the abuser).

Both Shearer, Peters, Quaytman, and Ogden (1990) and Salzman et al. (1993) conducted uncontrolled studies of the childhood experiences of borderline patients. Shearer et al. studied 40 female inpatients and Salzman et al. studied 31 symptomatic volunteers representing the mild end of the outpatient borderline continuum. Shearer et al. found that 40 % of their borderline cohort reported being sexually abused by a nonpeer before the age of 15, 27.5 % reported some type of incest, 17.5 % reported particularly severe sexual abuse, and 25 % reported being physically abused to the point of injury. Salzman et al. found that 16.1 % of their borderline patients reported being sexually abused during childhood and 9.7 % reported a childhood history of physical abuse. No patients reported being sexually abused by a caretaker and only one reported being sexually abused by a relative.

Three main findings have emerged from these studies. First, both physical and sexual abuse are relatively common in the childhood histories of criteria-defined borderline patients. Second, physical abuse is generally not reported significantly more often by borderline patients than comparison subjects. Third, sexual abuse is consistently reported significantly more often by borderline patients than depressed or personality-disordered comparison subjects.

Third Generation of Studies of the Pathogenesis of BPD

Third-generation studies are described below (see Table 18.1 for details). These studies share a number of conceptual and methodological features. Most important among these features are a tendency to assess a range of pathological childhood experiences rather than focusing solely on the prevalence of sexual abuse, a tendency to more explicitly explore the important parameters of sexual abuse, and a tendency to use multivariate analyses in determining significant findings.

Paris, Zweig-Frank, and Guzder (1994a, 1994b) published the first two studies of this kind; the first detailed the pathological childhood experiences reported by borderline women and the second detailed the pathological childhood experiences reported by borderline men. In the first of these studies, Paris et al. (1994a) compared the childhood histories of 78 borderline women and 72 female axis II comparison subjects. They found that a significantly higher percentage of the borderline patients had a history of sexual abuse (71 % vs. 46 %), sexual abuse involving penetration (33 % vs. 6 %), sexual abuse by relatives other than caretakers or siblings (24 % vs. 8 %), and sexual abuse by multiple perpetrators (37 % vs. 14 %). Somewhat surprisingly, most of the sexual abuse experiences reported by borderline patients (and comparison subjects) were one-time occurrences (80 % vs. 67 %) and comparison

subjects were more likely than borderline women to report having been sexually abused by a caretaker (36 % vs. 29 %). Paris and colleagues also found that physical abuse was significantly more common among borderline women than female comparison subjects (73 % vs. 53 %) and that borderline patients reported less maternal affection than comparison subjects. Only sexual abuse was found to be a significant multivariate predictor of the borderline diagnosis.

Paris and his colleagues (1994b) also studied the childhood experiences of male borderline patients ($N = 61$) and male axis II comparison subjects ($N = 60$). As with the female borderline patients, overall sexual abuse (48 % vs. 25 %) and penetration (18 % vs. 2 %) were reported by a significantly higher percentage of borderline patients than axis II comparison subjects. Also as with the female borderline patients, single-incident abuse was very common (78 % vs. 87 %). However, the perpetrators were somewhat different for men and women. Male borderline patients were significantly more likely than axis II comparison subjects to report having been sexually abused by a caretaker (12 % vs. 0 %) and by a stranger (23 % vs. 8 %). Male borderline patients were also significantly more likely to have reported having had a prolonged childhood separation or loss as well as to report more paternal control. In multivariate analyses, the significant predictors were found to be sexual abuse and childhood separation or loss.

Zanarini et al. (1997) published a second study of the childhood experiences of borderline patients. This study compared the reported pathological experiences of 358 borderline inpatients with the reported pathological experiences of 109 hospitalized axis II comparison subjects. These investigators found that 91 % of these borderline patients reported some type of childhood abuse and 92 % reported some type of childhood neglect. In terms of reported childhood abuse, borderline patients were significantly more likely than axis II comparison subjects to report having been verbally, emotionally, physically, and sexually abused by a caretaker (27 %) as well as sexually abused by a noncaretaker (56 %). All told, 62 % of borderline patients reported a childhood history of sexual abuse compared with 32 % of comparison subjects; a highly significant difference. In terms of neglect, borderline patients were significantly more likely than axis II comparison subjects to report having a caretaker neglect their physical care, withdraw from them emotionally, treat them inconsistently, deny their thoughts and feelings, fail to establish a real emotional relationship with them, place them in the role of a parent, and fail to provide them with needed protection. Zanarini and her colleagues also found that borderline patients with a reported childhood history of sexual abuse were significantly more likely than those without such a history to report having experienced all of the types of abuse and neglect studied. In addition, when all bivariate predictors were considered together, only four were found to be significant predictors of a borderline diagnosis: female gender, sexual abuse by a male noncaretaker, emotional denial by a male caretaker, and inconsistent treatment by a female caretaker.

Zanarini and colleagues also published two other studies on childhood adversity and its relationship to the development of BPD. The first (Zanarini et al., 2000) used the same sample as the study above and focused on reports of biparental abuse and neglect. Eighty-four percent of borderline patients reported having experienced some type of biparental abuse and/or neglect before the age of 18; 55 % reported a childhood history of biparental abuse and 77 % reported a childhood history of biparental neglect. These experiences were also reported by a substantial percentage of axis II comparison subjects (biparental abuse and/or neglect = 61 %, biparental abuse = 31 %, and biparental neglect = 55 %). However, borderline patients were significantly more likely than axis II comparison subjects to report having been verbally, emotionally, and physically but not sexually abused by caretakers of both genders. They were also significantly more likely than comparison subjects to report having caretakers of both genders deny the validity of their thoughts and feelings, fail to provide them with needed protection, neglect their physical care, withdraw from them emotionally, and treat them inconsistently.

The second study (Zanarini et al., 2002) focused on 362 inpatients (290 borderline patients and 72 axis II comparison subjects) who have been followed prospectively for 20 years in the NIMH-funded study—the McLean Study of Adult Development (MSAD) (Zanarini, Frankenburg, Hennen, & Silk, 2003). Over 50 % of sexually abused borderline patients reported being abused both in childhood and adolescence, on at least a weekly basis, for a minimum of 1 year, by a parent or other person well known to the patient, and by two or more perpetrators. Over 50 % also reported that their abuse involved at least one form of penetration and the use of force or violence. Using multiple regression modeling and controlling for age, gender, and race, it was found that the severity of reported childhood sexual abuse was significantly related to the severity of symptoms in all four core sectors of borderline psychopathology (affects, cognitions, impulsivity, and disturbed interpersonal relationships), the overall severity of BPD, and the overall severity of psychosocial impairment. It was also found that the severity of childhood neglect was significantly related to five of the ten outcome factors studied, including the overall severity of BPD, and that the severity of other forms of childhood abuse was significantly related to two of these factors, including the severity of psychosocial impairment. Taken together, the results of this study suggest that the majority of sexually abused borderline inpatients may have been severely abused. They also suggest that the severity of childhood sexual abuse, other forms of childhood abuse, and childhood neglect may all play a role in the symptomatic severity and psychosocial impairment characteristic of BPD.

Three main findings have emerged from this generation of studies. First, sexual abuse by noncaretakers is more discriminating for BPD than caretaker sexual abuse. Second, borderline patients are more likely than comparison subjects to report severe forms of sexual abuse, particularly those involving penetration. Third, sexual abuse typically takes place in an environment of biparental abuse and neglect.

Childhood Adversity Reported by Adolescents with BPD

Despite the clinical interest in BPD in adolescents, there has been little research on early childhood experiences and other etiological factors in these girls and boys. Three early studies pertain to the childhood experiences of adolescents with BPD. Bradley (1979) found that the majority (64 %) of children or adolescents with BPD had prolonged separations, which were defined as separations of 3–4 weeks from the primary caretaker, most commonly the mother of the child in the first 5 years of life, and that they were significantly more likely to have had such separations than either psychotic or personality-disordered comparison subjects.

Westen, Ludolph, Misle, Ruffins, and Block (1990) studied the childhood experiences of 50 female adolescent inpatients. They found that a significantly higher percentage of the 27 borderline girls than the 23 mixed psychiatric comparison subjects reported a childhood history of sexual abuse and physical neglect. Physical abuse, while common, did not significantly distinguish the two groups. In terms of the parameters of abuse, 29.6 % of the borderline patients reported being sexually abused by their fathers, 7.4 % by their mothers, and 40.7 % by others. In terms of these other types of abusers, 28 % of the borderline patients reported being abused by neighbors and friends, 8 % by siblings, and 8 % by extended family members. And most of the sexual abuse started during the latency years.

Westen and colleagues (Ludolph et al., 1990) also conducted another study using these same subjects. Variables most likely to predict BPD included history of disrupted attachments, maternal neglect, maternal rejection, grossly inappropriate parental behavior, number of mother and father surrogates, physical abuse, and sexual abuse. Families of borderline adolescents were found to be chronically disrupted, particularly during the early childhood years.

More recently, Venta, Kenkel-Mikelonis, and Sharp (2012) studied the childhood experiences

of 82 adolescent inpatients: 19 meeting DSM-IV criteria for BPD and 63 met DSM-IV criteria for other nonpsychotic psychiatric disorders. It was found that borderline patients were significantly more likely to report a history of childhood sexual abuse than the comparison subjects (53 % vs. 17 %). Or looked at another way, borderline patients were three times as likely to report being sexually abused before adulthood than comparison subjects—who were also hospitalized due to the severity of their symptom presentation.

Toward a Multifactorial Model of the Etiology of BPD

Unfortunately, the results of the studies of the childhood experiences of adults with BPD described above led many clinicians in the 1990s to believe that sexual abuse per se is both a necessary and sufficient precondition for the development of BPD. While appealing in its simplicity, this view is simply not consonant with the relevant research findings. No study has found that all borderline patients report having been sexually abused and not all sexually abused patients in these studies meet criteria for BPD. Rather, the majority of studies of adults with BPD have found that 40–71 % of borderline patients and 19–46 % of comparison subjects report some type of sexual abuse during childhood and/or adolescence. It is particularly important to remember that most of these studies were conducted on severely ill inpatients. It may well be that outpatients who have never been hospitalized would report lower levels of adversity. It is also important to remember that the majority of abuse survivors in the community do not seem to develop any type of serious adult psychopathology (Browne & Finkelhor, 1986; Herman, Russell, & Trocki, 1986).

A number of authors have suggested that a multifactorial model of the etiology of BPD in adults best captures the complexity of borderline psychopathology (Paris, 1994; Zanarini & Frankenburg, 1994). This model suggests that borderline symptomatology and its comorbid manifestations are the final end product of a complex admixture of innate temperament, difficult childhood experiences (including family history of psychiatric disorder), and relatively subtle forms of neurobiological dysfunction (which may be sequelae of these childhood experiences and/or innate vulnerabilities that underlie temperamental aspects of BPD).

Consistent with this multifactorial model, we have suggested a tripartite model of the etiology of BPD (Zanarini & Frankenburg, 1994). We believe that three factors—one environmental in nature, one constitutional in nature, and one representing the interaction of the other two or a triggering factor—are necessary (but perhaps not sufficient) for the development of BPD.

The first of these factors is a home environment that is traumatic in a broadly defined sense. In some cases, the trauma might be confined to the types of childhood experiences that can be categorized as unfortunate but not totally unexpected. These experiences, which we call Type I Trauma, would include prolonged early separations, chronic insensitivity to the preborderline child's feelings and needs, and serious emotional discord in the family, perhaps leading to separation or divorce. Type II Trauma would include frequent experiences of verbal and emotional abuse, neglect of age-appropriate physical needs, and circumscribed episodes of parental psychiatric illness. Type III Trauma would include experiences of frank physical and sexual abuse, chronic types of caretaker psychiatric illness, particularly axis II psychopathology and substance abuse, and a generally chaotic and dysfunctional home environment (e.g., parents repeatedly engage in shouting matches, no one abides by family rules or honors other family members' personal boundaries).

While for heuristic purposes we have defined three different types of environmental trauma that we have found are common in the histories of borderline patients, these types of trauma often occur sequentially or co-occur in the childhoods of many adult borderline patients.

The second factor necessary for the development of BPD is a vulnerable temperament. Studies by other groups have found that BPD is

associated with a temperament characterized by a high degree of neuroticism (i.e., emotional pain) as well as a low degree of agreeableness (i.e., strong individuality) (Clarkin, Hull, Cantor, & Sanderson, 1993; Soldz, Budman, Demby, & Merry, 1993; Trull, 1992; Zweig-Frank & Paris, 1995). BPD has also been found to be the only axis II disorder that is associated with a high degree of both harm avoidance (i.e., compulsivity) and novelty seeking (i.e., impulsivity) (Svrakic, Whitehead, Przybeck, & Cloninger, 1993).

Previously, our group has described emotional hypochondriasis as the primary defense of borderline patients and a hyperbolic stance as the behavioral manifestation of this defense (Zanarini & Frankenburg, 1994). Recent research by our group has confirmed the primacy of emotional hypochondriasis in the defensive hierarchy of borderline patients both cross-sectionally (Zanarini, Weingeroff, & Frankenburg, 2009) and longitudinally (Zanarini, Frankenburg, & Fitzmaurice, 2013). Recent research by our group has also confirmed the centrality of a hyperbolic temperament to the borderline diagnosis (Hopwood, Thomas, & Zanarini, 2012; Hopwood & Zanarini, 2012).

In this view, emotional hypochondriasis is defined as the transformation of unbearable feelings of rage, sorrow, shame, and/or terror into unremitting attempts to get others to pay attention to the enormity of the emotional pain that one feels. These attempts are usually indirect and involve a covert reproach of what is perceived as the listener's "insensitivity," "stupidity," or "malevolence."

The outward manifestation of this defense is the hyperbolic stance of the borderline patient. To put it most succinctly, nothing that can be stated dramatically is said simply and nothing that can be stated once goes unrepeated. In other words, much as Willie Loman's wife in Arthur Miller's *The Death of a Salesman* (Miller, 1986) believed that "attention must be paid" to his deteriorating situation, borderline patients insist that attention be paid to the enormity of their subjective pain—pain that is often consciously perceived and openly discussed as "the worst pain anyone has felt since the history of the world began." Perhaps most prototypic of this behavior are the deliberately, physically self-mutilative acts and manipulative (help-seeking) suicide efforts engaged in by borderline patients when under interpersonal stress and feeling alone.

While many would be in pain given the difficult childhood environment typical of borderline patients, the manner in which borderline patients handle their pain is both characteristic and distinguishing. They both insist that their pain be recognized and present it in a difficult-to-identify manner due to a combination of habituation and shame.

The third factor necessary for the development of BPD is a triggering event or series of events. The experiences that we would describe as triggering can be normative in nature, such as moving away from home to attend college or starting an intimate relationship. They also can be traumatic in nature, such as being seriously injured in a car crash or date raped. In either case, such an event seems necessary to propel a borderline person toward the full expression of his or her psychopathology and/or to enter treatment and, thus, have his or her condition noticed by mental health professionals for the first time.

The role of this triggering event seems to be that of a bridge between the intense dysphoria and frustration resulting from difficult childhood experiences and the preborderline person's innate vulnerability, which we have conceptualized as a hyperbolic temperament. Without such an event or series of events, which remind the preborderline person of old feelings of rageful despair and may represent the final degree of frustration that he or she can bear, such a person might be viewed as intense and demanding, but not clearly ill or impaired. Or he or she might be viewed as having a mild outpatient case of BPD. In our experience, these borderline patients display the same types of affective, cognitive, and interpersonal symptomatology characteristic of inpatients with BPD. However, they lack the impulsivity, particularly the lack of repeated episodes of self-harm and repeated suicide attempts, of these near-neighbor patients and display a capacity for collaborative

therapeutic work that is initially lacking in this more severely disturbed BPD group.

Thus, the model of the development of BPD that we have proposed is tripartite in nature. Traumatic childhood experiences occur which engender intense feelings of rage, sorrow, shame, and/or terror. These experiences, which tend to occur within the family and out of public view, interact with a preborderline patient's innate hyperbolic temperament to create an inner sense of emotional misery and almost total frustration. A normative or traumatic triggering event then occurs which reminds the preborderline person of earlier stress or trauma. This event or series of events acts as a catalyst for the fruition of a full-blown borderline condition with its characteristic symptom pattern (i.e., chronic, intense dysphoria; transient paranoid or dissociative experiences; impulsivity in a number of self-destructive areas; troubled interpersonal relationships marred by problems such as demandingness, manipulation, and extreme dependency).

The nature of the relationship between these etiologic factors is as yet unknown. It is also unclear if they are the same for adolescents with full-blown BPD.

The list of etiological factors will probably grow over time. For example, four well-designed family history or family studies (Gunderson et al., 2011; Links, Steiner, & Huxley, 1988; Zanarini, Barison, Frankenburg, Reich, & Hudson, 2009; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1988) have found that BPD and its symptomatic constituents "run" in families. As yet, it is not clear whether this familial association represents a true biological vulnerability, the effects of living with a mentally ill relative, or some combination of the two. In addition, twin studies have found a moderate degree of heritability of the sectors of borderline psychopathology (Distel et al., 2008; Torgersen et al., 2008).

In time, we will understand the etiology of BPD more fully. While enormous strides have been made, research into the multifactorial basis of BPD is still in its infancy. In particular, studies of adolescents with BPD are needed. For now, we suggest that one can admire borderline patients for the integrity with which they have dealt with their pain. After all, not many people remain so loyal to and so respectful of both disheartening childhood experiences and a temperament marked by significant vulnerability.

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Suggested Reading

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Parenting and the Development of Borderline Personality Disorder

19

Jenny Macfie and Jennifer M. Strimpfel

There is an interesting conceptual similarity between domains of dysfunction in borderline personality disorder (BPD) and developmental tasks of early childhood (Macfie, 2009; Sroufe, Egeland, Carlson, & Collins, 2005). BPD has been described as a disorder of *attachment*, with symptoms of fear of abandonment and unstable and intense relationships (Fonagy, Target, & Gergely, 2000; Gunderson, 1996; Liotti & Pasquini, 2000). BPD has been described as a disorder of *self*, with symptoms of identity disturbance, feelings of emptiness, and dissociative symptoms (Westen & Cohen, 1993). BPD has also been described as a disorder of *self-regulation*, with symptoms of impulsivity, suicidal behaviors, self-mutilation, affective instability, and difficulty controlling anger (Posner et al., 2003). Children address the developmental tasks of *attachment* to caregivers in the first year, *self-development* with the beginnings of autonomy in toddlerhood, early *self-regulation* of emotion and behavior in the preschool period, and rework each in adolescence (Macfie, 2009; Sroufe et al., 2005; Sroufe & Rutter, 1984). It is an empirical question whether or not BPD has its origins in part in failure to negotiate early childhood tasks. In the current chapter we assess the existing evidence for such a model, focusing particularly, but not exclusively, on the role of parenting. We then propose future directions for research and implications for interventions.

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Definitions and Scope

BPD may be assessed with a categorical diagnosis from a clinical interview (American Psychiatric Association, 1994, 2000; Gunderson, Kolb, & Austin, 1981). In addition, BPD may be assessed along a continuum by counting number of symptoms. Borderline features, derived from factor analysis, may also be assessed along a continuum with a self-report questionnaire (Morey, 1991, 2007). They include: affective instability, negative relationships, identity disturbance and self-harm, all of which correlate highly with a diagnosis of BPD (Morey, 1991, 2007). However, recent studies have suggested a unidimensional factor structure for BPD (see Chap. 4). The advantage of a diagnosis is that it brings to mind a particular clinical presentation (one that may vary, however, depending on the constellation of symptoms) and places individuals in groups. The advantage of a continuous measure is that everyone appears somewhere along the continuum. In this chapter we conceptualize BPD as first appearing in adolescence (Ludolph et al., 1990) or early adulthood (American Psychiatric Association, 2000), but argue that precursors may appear in childhood

"Parents" in the literature usually refers to mothers rather than fathers or both parents (Seifer

C. Sharp and J.L. Tackett (eds.), *Handbook of Borderline Personality Disorder in Children and Adolescents*,
DOI 10.1007/978-1-4939-0591-1_19,

& Dickstein, 2000). The term "caregivers" often replaces "parents" in the child development literature in order to acknowledge that people other than parents are likely and qualified to bring up children. However, in this chapter, research reviewed is limited to biological parents, and so the term "parents" is retained, with mothers referred to when appropriate.

Blaming parents, specifically mothers, for the development of psychopathology has a long and ignominious history including "schizophrenogenic" mothers for schizophrenia (Fromm-Reichmann, 1948) and "refrigerator" mothers for autism (Bettelheim, 1967; Kanner, 1949). A "mother-bashing" quality has also been noted in literature on depressed mothers (Downey & Coyne, 1990). It is important to emphasize that it not possible to disentangle the effects of genetics and environment in most studies (Sameroff & Chandler, 1975; Seifer & Dickstein, 2000), that BPD has a large genetic component (Torgersen et al., 2000), and that problems with parenting may best be conceptualized as the result of individual factors such as parental psychopathology in interaction with stressful contexts.

Indeed, the determinants of parenting include the psychological resources of the parent as well as qualities of the child and the balance between stress and support in the environment (Belsky, 1984). Parenting can be viewed at the level of the individual parent with constructs such as sensitivity, hostility, intrusiveness, and supportiveness (Biringen, Robinson, & Emde, 1998). Parenting can also be viewed at the level of the family system (Cox & Paley, 1997) including dyadic assessments of infant–parent attachment (Ainsworth, Blehar, Waters, & Wall, 1978) and of parent–child role reversal (Macfie, McElwain, Houts, & Cox, 2005). Parents who have psychopathology put their children at high risk of developing the same disorder (Downey & Coyne, 1990; Mednick & McNeil, 1968), which we discuss in terms of BPD.

Theoretical Background

We review the literature on parenting and the development of BPD from a developmental psychopathology perspective (Cicchetti, 1984; Cicchetti & Toth, 2006; Sroufe & Rutter, 1984). Developmental psychopathology takes a life-span approach to studying pathways to disorder versus resilience. Success or failure at stage-salient tasks, including attachment, self-development, and self-regulation in early childhood, may make the development of psychopathology more or less likely. Study of children who are at high risk of developing the disorder (such as offspring of mothers who have the disorder) along with normative comparisons is useful. Not only does normative development inform atypical development, but atypical development informs normative development, and both may inform interventions to bring development back on track and prevent the development of psychopathology.

Parenting changes as a child develops (Sroufe et al., 2005). For a secure attachment relationship to develop between the infant and the parent, parenting needs to be consistently sensitive and responsive. For self-development to develop in the toddler period, parenting becomes more challenging. While still being consistently sensitive and responsive, a parent also needs to balance support for a child's autonomy with setting limits to keep him or her safe, teaching social mores, and helping to resolve tantrums. Building on a secure attachment, the beginnings of autonomy, and dyadic regulation, a child in the preschool period develops the beginnings of self-regulation and is able to meet the expectations of a preschool setting. In adolescence, attachment is revisited in romantic relationships, self-development in establishing an identity, and self-regulation in the context of hormonal changes affecting mood, potential availability of drugs and alcohol, less parental oversight, and the gap between physical maturation and taking adult social roles. Indeed, adolescent development in the USA may look a little like BPD (Macfie, 2009).

A child's early experience with parents may be studied at the level of behavior and also at the level of mental representation. In order for an early experience to influence later development it has to be internalized (Carlson, Sroufe, & Egeland,

2004). Mental representations, also termed internal working models or schemas, are thought to develop from a child's early attachment relationships and provide templates to predict others' behavior, guide the child's own behavior, and shape the child's view of him or herself (Ainsworth et al., 1978; Bowlby, 1969/1982, 1973, 1980; Bretherton & Munholland, 2008; Young, 1990). Thus a child with a secure attachment in infancy is thought to develop representations of others as trustworthy, the self as worthy of care, and be well able to regulate emotions and behavior as he or she embarks on relationships with teachers and with peers. On the other hand, a child with an insecure attachment may develop representations of others as rejecting or ambivalent, the self as not worthy of care, and tend either to under or overregulate emotions in future relationships.

A child who is unable to form an organized attachment in infancy (secure or insecure) is classified as disorganized (Hesse & Main, 2006; Main & Solomon, 1990). Disorganized attachment develops in the context of the parents being seen as either frightening (e.g., maltreating) or frightened (e.g., grieving a recent loss). When distressed, the infant in a disorganized attachment appears to be caught between approach and avoidance, wanting comfort but afraid to be close to the parent. This results in bizarre behavior such as approaching the parent but backwards, stereotypies such as finger-flicking, or standing still staring as if in a trance (Main & Hesse, 1990). Thus the infant–parent attachment system, designed to buffer the infant from stress, is disorganized and the infant remains hypervigilant and fearful (Solomon & George, 2011). A child with a disorganized attachment may develop confused and contradictory internal working models of others and of self, and may have difficulty regulating emotions resulting in atypical responses such as dissociation and self-harm.

Atypical representations formed in early relationships with parents and carried forward to adolescence or early adulthood are theorized to make the development of psychopathology more likely (Bowlby, 1977). To gain a window on these representations with preschool children we can ask children to complete the beginnings of stories about challenging family situations presented to them with household props and family dolls (Bretherton, Oppenheim, Buchsbaum, Emde, & the MacArthur Narrative Group, 1990). The resulting videotaped narratives can then be coded for themes of interest.

Parents' representations of their own childhood experience, assessed from transcripts of semi-structured Adult Attachment Interviews, AAI (George, Kaplan, & Main, 1984; Main & Goldwyn, 1991; Main, Goldwyn, & Hesse, 2002), may also be coded and their effect on their parenting and on their children's representations examined. AAIs are coded as secure (free to discuss childhood experiences coherently), insecure (incoherently preoccupied with, or dismissive of, difficult childhood experiences), and unresolved with respect to the experience of loss or abuse. Adults with BPD are mostly classified as preoccupied and unresolved (Bakermans-Kranenburg & van IJzendoorn, 2009). Furthermore, adults with BPD are characterized as displaying more hostile/helpless representations on the AAI (Lyons-Ruth, Melnick, Patrick, & Hobson, 2007). In turn, mothers' unresolved (Main & Hesse, 1990) and hostile/helpless (Lyons-Ruth, Bronfman, & Parsons, 1999) AAI representations predict disorganized attachment with their infants.

Empirical Background

There are no definitive studies which elucidate the relationship between parenting and the development of BPD in adolescence or adulthood over time. Retrospective reports of childhood experience from adults who have BPD provide a rich source of data, although reports of problematic parenting may reflect current salience more than etiological significance. Retrospective reports also rarely differentiate between experiences in different developmental periods, and so are unable to identify disruptions in particular developmental tasks implicated in the etiology of BPD. Prospective longitudinal studies that assess

development between infancy and adulthood avoid retrospective bias, include many different factors, but, depending on the sample, may not result in a high percentage of BPD diagnoses. Studies of children at high risk of developing BPD in adolescence or adulthood, including maltreated children and offspring of mothers with BPD, may also help identify putative precursors to BPD, but need to be followed longitudinally.

Retrospective studies. There is a large literature on the relationship between retrospective reports of childhood maltreatment and BPD in adults, which is addressed in full elsewhere (see Chap. 16). Here we review studies of parental factors that may be conceptualized as falling in the domain of attachment (separation from parents), self-development (overprotection, inappropriate punishment, inconsistency, and role reversal), and self-regulation (emotional withdrawal and invalidation of thoughts and feelings).

Separation from parents during childhood, thus disrupting the attachment relationship, is frequently reported by adults who have BPD. Individuals with BPD are more likely to report having been placed in foster care or being raised by a non-parent as children than are normative comparisons (Bandelow et al., 2005) or individuals with other psychiatric diagnoses (Ludolph et al., 1990). Moreover, separations from parents before the age of 5 are more commonly reported by adults with BPD (and by those with antisocial personality disorder), than by adults with dysthymia together with any other personality disorder (Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1989). Furthermore, adults with BPD are more likely to report their fathers having been absent during their childhood than are individuals with depression or schizophrenia (Soloff & Millward, 1983), or with diagnoses other than BPD (Frank & Hoffman, 1986). Adults with BPD also more frequently report divorce of their parents than do those with depression or schizophrenia (Soloff & Millward, 1983) or normative comparisons (Bandelow et al., 2005).

In addition to attachment, retrospective reports of adults with BPD also include factors that may be related to children's self-development: overprotection, inappropriate punishment, inconsistency, and role reversal. Adults with BPD report more overprotection than do those with schizotypal personality disorder (Torgersen & Alnæs, 1992), and reports of parental overprotection are correlated with borderline features in college students (Nickell, Waudby, & Trull, 2002). In addition, more adults with BPD report that their parents used inappropriate punishment than do individuals with other diagnoses (Frank & Hoffman, 1986), and normative comparisons (Bandelow et al., 2005). Moreover, adults with BPD report having experienced more inconsistent treatment by their parents than do those with other personality disorders (Zanarini et al., 1989, 1997, 2000). Finally, adults with BPD report more role reversal with their parents, with the child placed in the role of parent, than do those with other personality disorders (Zanarini et al., 1997).

Retrospective reports by individuals with BPD also include factors that may be related to self-regulation: emotional withdrawal and invalidation of thoughts and feelings. Adults with BPD are more likely to report that their parents withdrew emotionally from them during childhood than are those with other disorders (Zanarini et al., 1989, 1997, 2000). Adults with BPD are also more likely to report that their parents denied the validity of their thoughts and feelings than do individuals with other personality disorder (Zanarini et al., 1997, 2000).

Concurrent studies. Young women with BPD report less current protection in their relationship with their mothers than did those without BPD. Moreover, on arriving at the lab with their mothers for a problem-solving discussion, the young women with BPD demonstrated a higher cortisol response than did comparisons (Lyons-Ruth, Choi-Kain, Pechtel, Bertha, & Gunderson, 2011)

Prospective studies. There have been several studies that assess the development of BPD longitudinally. In the first reviewed here, children at risk due to being born into poverty were followed from birth to age 28 (Carlson, Egeland, & Sroufe, 2009). A wide range of endogenous factors

(including activity level at 6 months and emotionality at 30 months) and environmental factors (including life stress between 3 and 42 months) were significantly correlated with the number of BPD symptoms in adulthood. In the domain of attachment, maltreatment assessed in infancy and between age 4 ½ and 18, disorganized attachment age 12–18 months, maternal hostility at 42 months, and family/father disruption from 12 months to 18 years, were associated with BPD symptoms age 28. In the domain of self-development, role reversal at 42 months, and at 13 years, were associated with BPD in adulthood. In the domain of self-regulation, children's attentional disturbance and emotional and behavioral instability at age 12 were correlated with BPD symptoms age 28 (Carlson et al., 2009).

In addition to examining development at the level of behavior, this longitudinal study also assessed children's representations. Self-representation disturbance age 8–12 was associated with BPD symptoms in adulthood, and mediated the relationship between disorganized attachment with mothers in infancy and BPD symptoms in adulthood (Carlson et al., 2009). This study has made a very important contribution to our understanding of the relationship between child development and BPD. However, by age 28, only 2 % ($N = 4$) had actually developed BPD (E. Carlson, personal communication, August 8, 2010).

In a prospective study in a community sample, family adversity, suboptimal parenting, and conflict between parents predicted BPD symptoms age 11 (Winsper, Zanarini, & Wolke, 2012). In the same sample, extended separations from mother prior to age 5 predicted BPD symptoms from early adolescence to middle adulthood (Crawford, Cohen, Chen, Anglin, & Ehrensaft, 2009). When these mothers were assessed with their adolescents average age 14 years at Time 1 and 16 ½ years at Time 2 (Bezirganian, Cohen, & Brook, 1993), 10 % of the adolescents at Time 1 were diagnosed with BPD using a DSM structured clinical interview, and 7 % at Time 2. Mothers' parenting was assessed with self-report questionnaires, thus avoiding biases both of retrospective data and of children's reporting on their mothers' parenting, mothers being more likely to under- rather than overestimate problems. Maternal inconsistency in the context of high maternal over-involvement (defined as a role reversal in which the mother depends on the child to meet her needs) at Time 1, predicted the persistence or emergence of BPD and no other personality disorder at Time 2. Furthermore, in another study using the same longitudinal data, low parental affection and aversive parental behavior when the children were average age 6 were associated with BPD at average age 22 and 23. However, these factors were not specific to BPD: they were also associated with the development of other personality disorders (Johnson, Cohen, Chen, Kasen, & Brook, 2006).

A separate longitudinal study of adolescents in a community sample (Arens, Grabe, Spitzer, & Barnow, 2011), tested Marsha Linehan's biosocial theory that invalidating parenting interacts with biological vulnerabilities to cause BPD (Linehan, 1993). Indeed, an interaction between the adolescents' temperamental trait of harm avoidance with perceived maternal overprotective parenting at age 15 predicted BPD at age 20. The authors conclude that overprotective parenting may inhibit the development of adaptive emotion regulation. When a child displays negative emotions, the mother may respond by being overprotective, which hampers the child from trying out emotion regulation strategies on his/her own. However, findings were not specific to BPD: they did not differ for those who were diagnosed as depressed (Arens et al., 2011).

Putative precursors to BPD in at-risk groups. Groups at risk for developing BPD include maltreated children and offspring of women with BPD (Lenzenweger & Cicchetti, 2005). Study of these groups may therefore inform the relationship between parenting and the development of BPD. A cross-sectional study of maltreated school-age children (assessed for having experienced sexual abuse, physical abuse, emotional abuse, and neglect), examined a composite of putative precursors to BPD in the domain of self-regulation: self-reports of affective lability, lack of conscientiousness, conflicted relationships, self-harm, and peer reports of

"upsets others," relational aggression, and "is disliked" (Rogosch & Cicchetti, 2005). In terms of the relevance to parenting, although the perpetrators of maltreatment were not identified, parents are known to be the most common perpetrators (U. S. Department of Health and Human Services, 2010), and parental neglect may facilitate maltreatment by others. Maltreated children scored higher than did nonmaltreated comparisons on the composite. There were no significant differences between physically abused, sexually abused, and neglected children, and emotionally abused children did not differ from nonmaltreated children. Lending credence to the likelihood of these being precursors to BPD, children high on the composite were less efficient in their attentional processing, a deficit characteristic of adults with BPD, than were other children (Rogosch & Cicchetti, 2005).

Offspring of women with BPD are also at high risk of developing BPD. Although there are no studies of the prevalence of BPD in offspring specifically, as noted above, BPD has a large hereditary component (Torgersen et al., 2000), and first degree relatives of those with BPD are more likely to have the disorder than are those in the general population (Links, Steiner, & Huxley, 1988; Loranger, Oldham, & Tulis, 1982; Zanarini, Frankenburg, et al., 2004). Parenting of mothers who have BPD may therefore inform the relationship between parenting and precursors to BPD, with a higher percentage of children actually developing BPD than is found in normative and poverty at-risk samples.

In the domain of attachment, mothers with BPD demonstrate more intrusive insensitivity when their infants are 2 months (Crandell, Patrick, & Hobson, 2003) and 13 months (Hobson, Patrick, Crandell, Garcia-Perez, & Lee, 2005), and more frightened/disoriented behavior with their 1-year-old infants (Hobson et al., 2009), than do normative comparisons. Moreover, at 13 months, 80 % of these infant offspring are classified as disorganized in their attachment to their mothers (Hobson et al., 2005), the same percentage found in maltreated children (Carlson, 1998). Furthermore, mothers with BPD are less sensitive and more hostile than are normative comparisons (Macfie et al., 2007). Additionally, offspring of women with BPD age 4–7 are more likely to have been maltreated than comparisons (Reid, Campion, Watkins, & Macfie, 2007).

In the domain of self-development, infant offspring of women with BPD, age 3–26 months are less responsive to, and interactive with, their mothers than are normative comparisons (Newman, Stevenson, Bergman, & Boyce, 2007). Similarly, children age 4–7 of mothers with BPD are less responsive to, and involving of, their mothers than are normative comparisons (Macfie et al., 2007). In the domain of self-regulation, compared with normative comparisons, offspring of women with BPD age 4–7 are more emotionally reactive and withdrawn, with symptoms associated with affective disorders, anxiety disorders, and attention deficit hyperactivity disorder (Campion et al., 2011). Moreover, offspring of women with BPD display more behavior problems age 4–18 than do offspring of women with other personality disorders (Weiss et al., 1996), and more behavior problems at age 11–18 than do offspring of depressed mothers, mothers with Cluster C personality disorders, and normative comparisons (Barnow, Spitzer, Grabe, Kessler, & Freyberger, 2006).

Mothers with BPD are less close to, and less supportive of autonomy for their adolescents, and their adolescents are more likely to change their opinions to placate their mothers, than are normative comparisons (Frankel, McCullum, Trupe, Jones, & Macfie, 2009). These adolescents are also more likely to demonstrate more general, verbal and relational aggression, and self-harm than are normative comparisons (Swan, Campion, Watkins, Price, & Macfie, 2009), and are more preoccupied in self-report measures of romantic attachment (Watkins et al., 2009). Importantly, offspring age 14–17 also report more borderline features themselves than do normative comparisons (Watkins et al., 2011).

At the level of representation, offspring of women with BPD age 4–7 display more putative precursors to BPD than do normative comparisons. In the domain of attachment, offspring of women

with BPD, compared with normative comparisons, tell stories with more negative mother–child and father–child relationship expectations and fear of abandonment; in the domain of self-development, they tell stories with more role reversal, incongruent, and shameful representations of the self; and in the domain of self-regulation they display more narrative incoherence, confusion between self and reality, confusion between self and fantasy, and fantasy proneness, the latter three being associated with dissociation (Macfie & Swan, 2009). Furthermore, narrative representations thought to be related to BPD symptoms (fear of abandonment, role reversal, incongruent child, confusion between self and fantasy, and destruction of objects) are associated with their mothers' preoccupied/unresolved representations of their own childhood assessed with the AAI (Macfie, Swan, Fitzpatrick, Watkins, & Rivas, in press). These representations may be transmitted from one generation to the next with implications for the development of BPD. Indeed mothers' parenting mediated the relationship between mothers' preoccupied/unresolved representations of their own childhood and their children's representations of a fear of abandonment (Macfie et al., in press).

A Proposed Model

It is clear from the empirical literature that prospective studies validate retrospective reports of adults with BPD. Maltreatment, separation from parents, parental overprotection and inconsistency, role reversal, and invalidation reported in retrospective studies also predict BPD symptoms or a BPD diagnosis in adolescence or early adulthood. Prospective studies, however, add many important etiological factors including disorganized attachment in infancy and representational development in childhood, which validate the choice of maltreated children and offspring of women with BPD as groups at high risk of developing BPD. Both are characterized by 80 % being disorganized in their attachment with their mothers in infancy and by developing atypical representations in the preschool period. Maltreated children's representations contain less parent empathy for children, but more child empathy for parents in a role reversal (Macfie et al., 1999), and demonstrate an increase in dissociation across the preschool period compared with nonmaltreated children (Macfie, Cicchetti, & Toth, 2001). The narratives of offspring of women with BPD include negative portrayals in the domains of attachment, self-development, and self-regulation, reviewed above (Macfie & Swan, 2009). Although not all maltreated children nor all offspring of women with BPD will develop BPD, study of their development may inform risk factors for the disorder.

We propose a model of parenting and the development of BPD in Fig. 19.1. Parenting may be affected not only by parent temperament, child temperament, and environmental context, but also by representations of the parent's own childhood experiences. Problematic parenting then predicts infant–parent disorganized attachment. It is theorized that for a disorganized attachment to develop, the infant's distress triggers the parent's own unresolved childhood memories of not being soothed (Fraiberg, Adelson, & Shapiro, 1975). The parent may then feel helpless to care for the infant, and may become angry at demands made by the infant, and may abdicate in part from the role of the parent (George & Solomon, 2008). Because the parent's first priority becomes to soothe him or herself, the parent may display contradictory hostile/helpless responses to the infant's need for comfort and closeness. This may in turn frighten the infant who remains unsoothed (Lyons-Ruth, Bronfman, & Atwood, 1999; Lyons-Ruth & Jacobwitz, 2008; Main & Hesse, 1990). This hostile/helpless stance toward the infant is associated with atypical affective communication (Bowlby, 1988; Main, Kaplan, & Cassidy, 1985), which includes frightening, hostile-intrusive, and role-reversed behaviors (Lyons-Ruth, Bronfman, & Atwood, 1999).

Disorganized attachment in infancy in turn predicts parent–toddler role reversal (Macfie, Fitzpatrick, Rivas, & Cox, 2008). When parents of toddlers encourage their children to focus on their (the parents') needs rather than on their

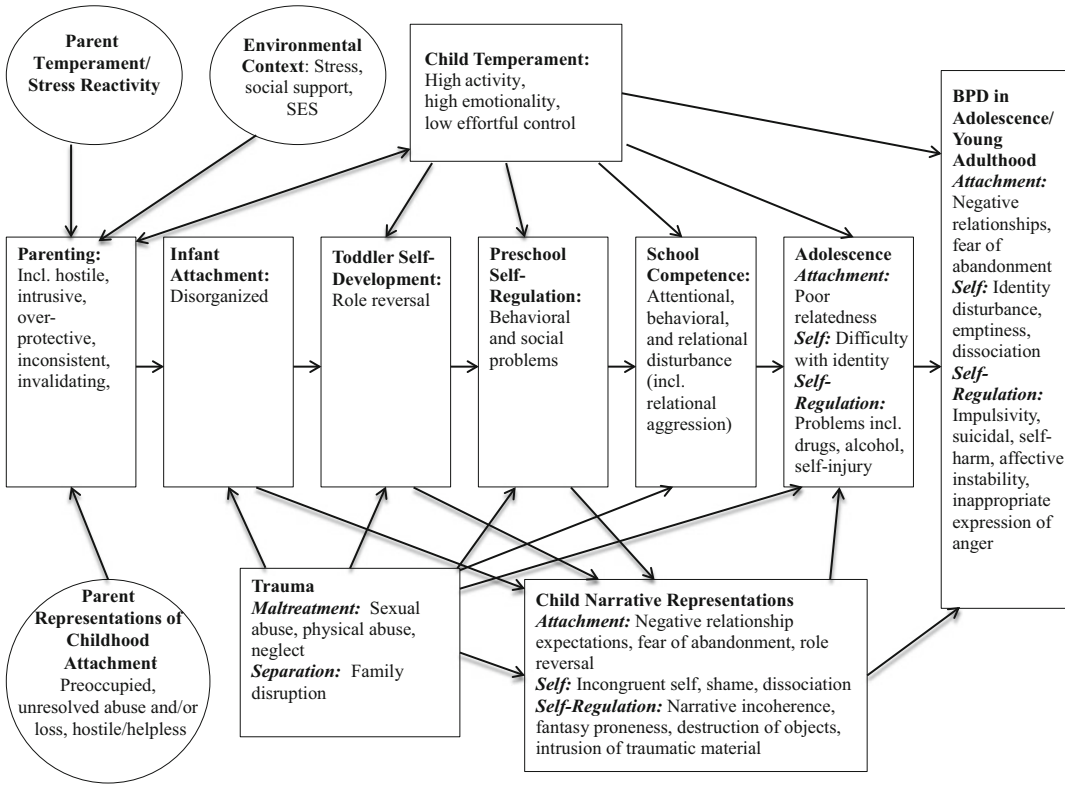

Fig. 19.1 Parenting and the development of BPD—a proposed model

own, the development of autonomy and self-regulation suffer, the need for care remains unfulfilled. In a study of mother–child role reversal in a normative sample, role reversal is repeated in the next generation as children seek to meet the need for care with their own children: a girl grows up to look to her daughter for care, and a boy grows up to marry a woman who looks to their son for care (Macfie, McElwain, et al., 2005). Role reversal in turn predicts problems with self-regulation in kindergarten (Macfie, Houts, McElwain, & Cox, 2005), which may then affect school functioning and peer relationships, which lead to problems in adolescence. Difficulty in adolescence in the domain of attachment extends to romantic relationships, difficulty with self-development extends to identity, and difficulty with self-regulation extends to impulsive, self-damaging behaviors including the use of drugs, alcohol, sexual activity, and self-injury. However, failure to negotiate each developmental task between infancy and adolescence successfully may not lead to BPD specifically. The experience of trauma including maltreatment and separation, and the moderating effects of child temperament, may each play a role. Children with emotionally reactive temperaments who are low in effortful control may be more likely to develop BPD. Moreover, representational development may provide the process by which early developmental failure is carried forward to make the development of BPD more likely.

Future Research

The main goal of future research on parenting and the development of BPD is to inform preventive interventions. BPD is a severe and chronic disorder that involves self-destructive behavior, inappropriate displays of anger, and frantic help

seeking. We know that this combination makes the disorder extremely challenging for health care providers (Gunderson, 2001), a challenge that is costly in terms of individual suffering and in terms of burden on the health care system. For example, in one study 47 % of chronic pain patients were diagnosed with BPD (Sansone, Whitecar, Meier, & Murry, 2001), and individuals with BPD utilize mental health services at higher rates than does any diagnostic group other than schizophrenia (Swartz, Blazer, George, & Winfield, 1990). Seventy to 90 % of individuals with BPD repeatedly attempt suicide or make suicidal gestures (Gunderson & Ridolfi, 2001; Linehan & Heard, 1999), which involve intensive utilization of mental health services (Roy, 2001), and completed suicide occurs in 8–10 % (American Psychiatric Association, 1994; Paris, 1993; Stone, 1990).

In order to design preventive interventions, we ideally need longitudinal studies in high risk samples from infancy to early adulthood. We focus here on offspring of mothers with BPD because a higher proportion than in other risk groups may be expected to develop BPD. In this way, processes underlying success versus failure at developmental tasks in interaction with temperament/stress reactivity, maltreatment/separation, and parenting can be identified. With a better understanding of how BPD develops, both the timing and the target of developmentally informed interventions can be specified. In order to get development back onto an adaptive pathway, we need information on factors both common to developmental failure in general and specific to BPD in particular. What is currently absent from the literature on offspring of women with BPD is the study of stress reactivity, which, in interaction with failure at developmental tasks might potentiate the development of BPD. Disruptions in the HPA axis, including atypical cortisol patterns, which impair the ability of children to manage current stress may, in interaction with environmental variables, make the development of BPD more likely.

We need further validation for two promising measures that assess putative BPD symptoms in school-aged children. For the first, a normative sample of children in fourth to sixth grade was assessed for putative borderline features three times during a 1-year period (Crick, Murray-Close, & Woods, 2005). The authors adapted the borderline features scale from the Personality Assessment Inventory, PAI (Morey, 1991) for children. They validated it against assessment of a hostile, paranoid world view, intense and unstable emotion, overly close relationships, and relational aggression, and found considerable construct validity and stability (Crick et al., 2005). For the second, an adult DSM-IV interview has been scaled down for use with children: simpler language, omission of age-inappropriate behaviors, and a more structured format (Zanarini, Horwood, Waylen, & Wolke, 2004). As noted previously family adversity and problematic parenting predicted BPD symptoms aged 11 using this scale (Winsper et al., 2012). Both scales might be compared with each other, and profitably be used with children at high risk for developing BPD, including offspring of mothers with the disorder.

Research on the relationships between parenting, child temperament, and the development of BPD is needed. We know that child temperament (angry tantrums, frequent crying, demands for attention, and reactive mood) predicts BPD symptoms in adolescence and early adulthood (Crawford et al., 2009). However part of the genetic component to BPD may result from an interaction between child temperament and parenting. There is a large body of research indicating that child temperament and parenting influence each other in a bidirectional manner, and that certain temperaments may make a child more susceptible to the effects of negative parenting (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Kiff, Lengua, & Zalewski, 2011). Children with temperaments considered "difficult" (irritable or high in negative emotionality) may elicit parenting behaviors associated with later development of BPD, such as low warmth (Kendler, Sham, & MacLean, 1997), low responsiveness (Koenig, Barry, & Kochanska, 2010), inconsistency (Lengua & Kovacs, 2005), and overprotection/overcontrol (Bridgett et al., 2009).

Finally, longitudinal study of offspring of women with BPD is needed. There has only been one study of offspring of women with BPD and normative comparisons who were followed up over a 11-month period in infancy (Crandell et al., 2003; Hobson et al., 2005). Offspring of women with BPD ideally need to be followed from infancy to adolescence/early adulthood. Not only would this inform the development of BPD and preventive interventions, but, because BPD affects mainly women in their childbearing years, it would also inform the course of BPD over time.

Clinical Implications

In the context of parenting issues, a promising target for intervention to prevent BPD is reflective functioning. Reflective functioning is the ability to understand one's own and others' behavior in terms of beliefs and feelings, also termed mentalization (Fonagy, Target, Steele, & Steele, 1998). Individuals with BPD have the lowest scores on reflective functioning compared with those with other disorders (Fonagy et al., 1996). They may therefore react angrily to what they fear is a threat but is not, often harming their relationships and themselves. In terms of parenting, reflective functioning predicts security of attachment (Fonagy, Steele, & Steele, 1991). Higher reflective functioning would help prevent the development of disorganized attachment, which would in turn help prevent the development of BPD. Mentalization-based therapy (see Chap. 22) is designed to improve reflective functioning by focusing on the relationship between the therapist and the person with BPD. Mentalization-based therapy led to a reduction in BPD symptoms and subjective distress at the end of the intervention and at follow-ups as long as 8 years (Bateman & Fonagy, 1999, 2001, 2008). Rather than wait until BPD has fully developed, preventive interventions targeted at reflective functioning may be instituted with parents of children at risk.

Child–Parent Psychotherapy (CPP) is an attachment-based intervention, which includes a focus on improving reflective functioning in both the parent and the child (Lieberman, 1992). For example, a mother and her young child (infant, toddler, or preschooler) meet with the therapist. The mother feels understood by the therapist, and learns more about her own and her child's feelings, beliefs, and needs, so that the mother–child relationship becomes a greater source of security to the child. Indeed, CPP leads to an increase in attachment security in depressed mother–toddler pairs (Cicchetti, Toth, & Rogosch, 1999) and an increase in positive, and decrease in negative representations in maltreated children's stories (Toth, Maughan, Manly, Spagnola, & Cicchetti, 2002). If the intervention is instituted with mothers with BPD and their children, both mothers' symptoms might improve, and their children's development set onto a more adaptive pathway, away from the future development of BPD. In addition, interventions that aim to prevent a disorder may, inform the etiology of the disorder, including the role of parenting (Cowan & Cowan, 2002).

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A Contemporary Interpersonal Model of Borderline Personality Development

20

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The concept of borderline personality is, like the pattern of behavior it describes, complicated, diffuse, and messy. Although it is widely researched and its clinical importance is extensively demonstrated (Skodol et al., 2011), it is also inconsistently defined and commonly misunderstood. Even giving a name to the construct requires careful consideration. Referring to "Borderline Personality Disorder" (BPD) risks being mistaken to mean a strict reference to people who have five or more symptoms in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5, American Psychiatric Association, 2013), without concern for anyone below that threshold. This would be an overly narrow conception from most perspectives. Referring to "borderline personality traits" can be confusing because that term is used in the literature to suggest different things, such as the component traits that relate to BPD (e.g., neuroticism, antagonism, and disinhibition; Kendler, Myers, & Reichborn-Kjennerud, 2011) or individuals with subclinical symptoms of BPD (e.g., Seres, Unoka, & Keri, 2009). The issue is further complicated by the popularity of the psychodynamic concept of "borderline personality organization" (Kernberg, 1975), which refers to a level of personality functioning rather than a specific diagnostic category (i.e., at the border between psychotic and neurotic). Although we do use BPD to indicate people who meet diagnostic criteria when appropriate, throughout this chapter we primarily use the term *borderline personality*. We intend for this term to imply a concept whose definition is sufficiently broad that it can accommodate the elements of different conceptions of the borderline personality construct in the literature, including the diagnosis of BPD, borderline personality organization, subclinical borderline symptoms, and associated traits and dynamics.

Historically personality disorder theorists and researchers have not tended to focus on childhood or adolescence, limiting the scope and depth of research on the developmental trajectories of borderline personality (Shiner, 2009). It is intuitive that understanding the processes by which borderline personality develops represents a critical step toward understanding its construct validity, as well as how to best assess and treat it in adulthood. Given the large gaps that currently exist in the research literature on borderline personality development, contemporary developmental research is poised to play a critical role in clarifying the concept.

Interpersonal theory (Pincus & Ansell, 2013) also has considerable potential for clarifying borderline personality, particularly when coupled with a developmental perspective. First, as described below, social factors seem to play a particularly important role in the expression of borderline

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features. Second, interpersonal theory has a long and clinically rich tradition of describing personality pathology (Benjamin, 1996; Carson, 1969; Horowitz, 2004; Kiesler, 1986; Leary, 1957; Pincus & Wiggins, 1990; Sullivan, 1953). Interpersonal researchers have also articulated and evaluated systematic hypotheses about personality development (Critchfield & Benjamin, 2008; Wright, Pincus, & Lenzenweger, 2012), symptom expression (Cain et al., 2012; Pincus & Wright, 2011), and psychosocial intervention (Anchin & Pincus, 2010; Benjamin, 2003; Cain & Pincus, in press; Hopwood, 2010; Pincus & Cain, 2008; Tracey, 2004). Third, these hypotheses are linked to a robust suite of assessment tools based on a well-validated model of interpersonal functioning that can be used to operationalize both dispositional (Locke, 2011) and dynamic (Moskowitz & Zuroff, 2004; Pincus et al., in press) interpersonal constructs and test-specific hypotheses from different theoretical perspectives (Hopwood, 2010). Overall, the interpersonal paradigm in clinical psychology is well suited to accommodate and test hypotheses from different theories regarding interpersonal aspects of borderline personality development.

Our aim in this chapter is to identify ways in which contemporary integrative interpersonal theory (Pincus, 2005) can be useful for conceptualizing borderline personality development. We first review empirical research on interpersonal aspects of borderline development and expression. We then outline the fundamental principles and define the central constructs of interpersonal theory. We conclude with an interpersonal formulation of developmental patterns in an individual with borderline personality.

Research on Interpersonal Aspects of Borderline Personality Development

The broad literature examining correlates of borderline personality provides information about interpersonal functioning that can be useful for generating developmental hypotheses. In this section we review the empirical literature on borderline personality with particular attention to its interpersonal aspects. We begin with research on the interpersonal context of borderline symptoms, which point to the importance of developmental factors. We then review research on borderline personality development that further suggests the importance of interpersonal contexts.

Interpersonal Context of Borderline Personality Symptoms

Symptoms involving efforts to avoid abandonment, stress-induced paranoia, stormy or unstable relationships, and alternations between extremes of idealization and devaluation of others allude to a core conflict about closeness to others. On the one hand, the borderline individual desires attachment and fears intensely the possibility that others might abandon them (Bornstein, Becker-Matero, Winarick, & Reichman, 2010). On the other, borderline individuals are intensely mistrustful of others and can be hostile and aggressive (King-Casas et al., 2008).

Clinical theorists suggest that the unstable interpersonal behavior characteristic of borderline personality reflects a chaotic inner experience involving identity diffusion, insecure/ambivalent attachment, need for validation of inner pain, and difficulties with mentalization (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). Consistent with these characterizations, research identifies social cognitive factors that reinforce and interact with this interpersonal ambivalence (see also Chap. 12, Sharp). Borderline individuals are unusually effective at detecting changes in others' emotions (Lynch et al., 2006) and are significantly more sensitive than non-borderline controls to nonverbal cues (Frank & Hoffman, 1986) including facial expressions of emotion (Wagner & Linehan, 1999). However, borderline individuals have a negative bias in the interpretation of emotionally neutral faces (Daros, Kazkanis, & Ruocco, 2013), as well as a tendency to interpret interpersonal situations in extremes (Veen & Arntz,

2000) and to attribute extreme traits to others (Barnow et al., 2009). This research suggests that the borderline individual is acutely sensitive to affective aspects of the social environment, but tends to process others' affects and infer others' intentions with significant negative bias. Thus, while they may desire closeness and fear abandonment, borderline individuals are prone to see danger and mal-intent in others, and therefore vacillate between fear of distance and fear of closeness.

Negative affect is a predictable result of this pattern; however, many forms of psychopathology involve proneness to negative emotions (e.g., Lahey, 2009). The negative affective patterns of borderline personality can be distinguished by two factors. First, individuals with borderline personality report significantly more variable negative emotions than those reported by individuals with depression and other disorders over time (Hopwood et al., 2009; Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007; Trull et al., 2008). Second, research suggests that negative moods are more specifically linked to interpersonal precipitants for borderline than non-borderline individuals. For instance, using an experience sampling methodology, Sadikaj, Russell, Moskowitz, and Paris (2010) found that individuals with borderline personality report a greater increase in negative affect relative to controls when they perceive others as cold, but a lesser increase in positive affect relative to controls when they perceive others as warm. Physiological correlates of negative mood such as cortisol response to stressors are also associated with more interpersonal precipitants in BPD (Lyons-Ruth, Choi-Kain, Pechtel, Bertha, & Gunderson, 2011), particularly when there is a history of interpersonal trauma (Limberg, Barnow, Freyberger, & Hamm, 2011). Moreover, in experiments in which participants are led to believe they are being ostracized, individuals with BPD have more intense negative emotional responses than controls (Lawrence, Chanen, & Allen, 2011), and during interpersonal interactions in general, borderline individuals feel more anxious, more ashamed (Drapeau, Perry, & Koerner, 2009), and more aggressive (Zanarini et al., 1998) than those without borderline features.

There are also differences in the ways that borderline and more purely internalizing (e.g., depressed, anxious) individuals characteristically respond to negative emotions. Borderline personality is associated with coping strategies that lead to a broad array of significant self-harming behaviors, including substance abuse, impulsive spending, self-mutilation, high risk sexual behavior, and suicidality. Relationship quality predicts these various forms of self-harm behavior more strongly among BPD patients than in patients with comorbid depression or other personality disorder diagnoses (Yeomans, Hull, & Clarkin, 1994; Whipple & Fowler, 2011). Borderline personality is also more strongly associated with suicide precipitants that are more interpersonal in nature (e.g., arguments with significant others; feeling disappointed by, angry with, or abandoned by significant others) than precipitants that are not interpersonal in nature (physical illness, work stressors, etc.; Brodsky, Groves, Oquendo, Mann, & Stanley, 2006). Such observations suggest the value in prioritizing the interpersonal context clinically to reduce self-harm in individuals with BPD (Yeomans et al., 1994).

Finally, basic cognitive deficits involving dissociation and paranoid distortion under stress can also be understood in an interpersonal context. Paranoid ideations are interpersonal by definition in that the objects of paranoid ideations are other people (or representations of other people). This link is supported further by research suggesting that paranoid ideations are more common in borderline individuals with greater social anxiety (Martin & Penn, 2001) and that interpersonal stress has been implicated in the development of acute psychotic symptoms in individuals with BPD (Barnow et al., 2010). Dissociative processes also tend to occur in an interpersonal context among individuals with borderline personality. Major theories of dissociation imply that it is influenced, in part, by interpersonal trauma (Zlotnick et al., 2010); and Westen, Betan, and DeFife (2011) found that fearing one would "no longer exist" after a relationship ends

was associated with BPD even after controlling for other personality pathology.

Interpersonal Correlates of Borderline Personality Development

Research suggests that heritable and environmental influences on borderline features interact dynamically over the course development (Carlson, Egeland, & Sroufe, 2009). Trauma and neglect are often featured among environmental influences. Specifically, verbal, physical, and sexual abuse each predicts adult borderline symptoms (Zanarini et al., 2000). However, some research suggests that sexual abuse is more strongly associated with adult borderline personality than other forms of childhood abuse (Zanarini et al., 1997; see also Chap. 17 by Zanarini & Wedig, 2014). Other childhood risk factors for borderline personality include parental neglect (Zweig-Frank & Paris, 1991), invalidation, (Linehan, 1993; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1989), lack of parental protection (Lyons-Ruth et al., 2011), and early separation or loss (Soloff & Millward, 1983).

In terms of heritable dispositions, borderline personality among adults is correlated with relatively stable traits related to neuroticism (negative affect), disinhibition (low effortful control or conscientiousness), and antagonism (low agreeableness or low affiliativeness) (Samuel & Widiger, 2008). These traits as well as BPD are typically about 50 % heritable throughout adolescence and adulthood (Bornovalova, Hicks, Iacono, & McGue, 2009; Donnellan, Burt, Levendosky, & Klump, 2008), and some evidence suggests that the genetic influences on personality traits and BPD are mostly shared (Distel et al., 2008). It is also possible that children's temperaments and environments are correlated. Given the heritability of borderline features, it would not be surprising if parents of individuals with borderline personality had borderline characteristics themselves (Selby, Braithwaite, Joiner, & Fincham, 2008), and that these characteristics could increase risk for inconsistent, neglectful, and even abusive parenting. Likewise, pre-borderline behavior of the child involving an intense need for validation and impulsive behavior may evoke maladaptive parenting in the caretaking environment.

Principles and Constructs of Contemporary Interpersonal Theory

Given that existing research consistently indicates the importance of interpersonal contexts for understanding the development and expression of borderline personality, it seems natural to draw upon a model of personality and psychopathology that focuses on interpersonal functioning in order to make developmental hypotheses. In this section, we describe basic principles and constructs of interpersonal theory (e.g., Sullivan, 1953; Leary, 1957; Horowitz & Strack, 2010; Wiggins, 1991; Pincus, 2005; Pincus & Ansell, 2013; Pincus, Lukowitsky, & Wright, 2010; see Table 20.1), with an emphasis on their relevance for understanding borderline personality development.

The Interpersonal Situation

From an interpersonal point of view, the expression of borderline personality and most other psychopathology is fundamentally interpersonal, in the sense that people are known to have psychopathology primarily through their interpersonal behavior (e.g., communications of distress, dysfunctional social behavior). The most effective treatments for borderline personality are also interpersonal (i.e., involve a therapist as opposed to a medical procedure or pill) and borderline personality is the only disorder to date for which the American Psychiatric Association's treatment guidelines specify psychotherapy as the treatment of choice (Yeomans, Levy, & Meehan, 2012).

The value of interpersonal theory for understanding borderline personality lies in its ability to connect the nature of dysfunction with therapeutic and developmental principles by focusing on the *interpersonal situation* (Sullivan, 1953).

Table 20.1 Key interpersonal constructs

Interpersonal construct	Definition
Interpersonal situation	An event consisting of a self, other, and an associated affect. The self and other may be real or imagined
Agency	A meta-construct involving self-definition, achievement, and power which manifests in interpersonal situations as dominance (vs. submission)
Communion	A meta-construct involving intimacy, nurturance, and affiliation which manifests in interpersonal situations as warmth (vs. coldness)
Complementarity	The probabilistic tendency for warmth to be met with warmth and dominance to be met with submission in interpersonal situations
Identification	The tendency to do unto others in interpersonal situations what was done to the self during development
Recapitulation	The tendency to perceive and reenact past interpersonal roles in present interpersonal situations
Introjection	The tendency to treat one's self the way important others treated the self
Dysregulation	The experience and manifestation of affective, self, and field regulatory difficulties in interpersonal situations
Parataxic distortion	The distortion of aspects of an interpersonal situation due to the influence of past interpersonal situations on perception and behavior
Interpersonal signature	A consistent pattern of interpersonal situations that characterizes an individual's personality and pathology

Interpersonal situations involve the dynamic relations between a *self* seeking the satisfaction of motives for security and self-esteem, an *other* with whom the self interacts in an interpersonal field, and an associated *affect* that signals motive satisfaction (Hopwood, Wright, Ansell, & Pincus, 2013; Pincus & Hopwood, 2012). It is important to note that interpersonal situations can be composed of actual, proximal interactants, or mental representations of self and/or others (Blatt, Auerbach, & Levy, 1997; Lukowitsky & Pincus, 2011; Pincus & Ansell, 2013; Sullivan, 1953).

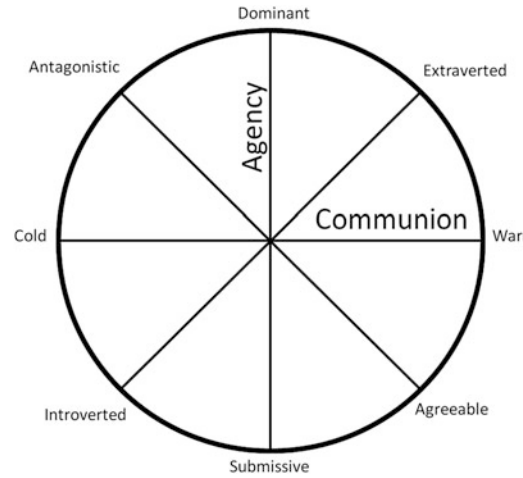

Fig. 20.1 The interpersonal circumplex

The Interpersonal Circumplex and Interpersonal Assessment

Patterns of interpersonal situations can be described using the *Interpersonal Circumplex* (IPC; Leary, 1957; Wiggins, 1979) (Fig. 20.1). The origins of the IPC lie in Sullivan's proposition that security and self-esteem reflect the fundamental motives of human behavior, as mediated through interpersonal situations (Fournier, Moskowitz, & Zuroff, 2011; Pincus & Wright, 2011; Wiggins, 1996). The model crystallized through research on group psychotherapy by Timothy Leary and his colleagues at Kaiser Permanente in the 1950s (Leary, 1957), who noted that the variety of behaviors observed in patients' interactions could be conceptualized using a circular model structured by dominance and warmth. Wiggins (1991) established connections between the IPC dimensions of dominance and warmth and Sullivan's security and self-esteem, both of which refer to the broader metaconstructs of *Agency* (dominance, self-esteem) and *Communion* (warmth, security), respectively (Bakan, 1966; McAdams, 1985). The appearance of constructs that similarly

correspond to agency and communion in a number of other literatures in the social sciences (e.g., Bem's [1974] masculine and feminine; Freud's love and work [Erikson, 1950]; Hogan's [Hogan et al., 1985] getting ahead and getting along) speaks to the integrative potential of the interpersonal model (Wiggins, 1991, 2003).

The IPC provides a flexible system for describing individual differences in clinically relevant interpersonal behavior. Individuals can vary in the *intensity* of interpersonal behavior, which would be signified by the distance of a behavioral rating from the center of the circle. For instance, an empathic expression by the therapist to a client's disclosure of a painful memory would be warmer than supportive silence, and the difference between these behaviors would be signified by their relative distance to the "east" (i.e., towards warmth) on the IPC. The *nature* of behavior can also be indicted by the IPC. For example, a directive to describe the event further is more dominant than a supportive acceptance of whatever the client wished to disclose in that situation. However, the directive will be "north of the equator" (i.e., towards dominance) whereas the expression of non-directive support will be "south of the equator," even if both interventions are similarly warm and thus similarly distant from the center of the circle.

IPC assessments have been developed for a number of domains including behaviors, traits, problems, capabilities, strengths, sensitivities, and values (Hopwood et al., 2011; Locke, 2011). Discrepancies across these "interpersonal surfaces" in an individual's profile can be clinically informative about *personality dynamics* (Pincus et al., in press). For instance, it may be more distressing for a person to experience cold interactions when they value interpersonal warmth than when they value interpersonal distance in relationships. Finally, individuals can vary in terms of *temporal dynamics* (Pincus et al., in press), which have been investigated both within (Sadler, Ethier, Gunn, Duong, & Woody, 2009) and across (Moskowitz & Zuroff, 2004) interactions. The development of dynamic models that include an assessment of others is a particularly exciting development in interpersonal assessment, as it enables the investigation of hypotheses that are increasingly closer to the more innovative and clinically important aspects of interpersonal theory (Pincus & Wright, 2011).

Overall, research suggests that borderline personality is difficult to capture in terms of stable individual differences in interpersonal behavior (Hopwood & Morey, 2007; Wiggins & Pincus, 1989). For instance, although individuals with borderline personality generally report warm-submissive problems as being most typical (Hilsenroth, Menaker, Peters, & Pincus, 2007), they are nearly equally likely to display behaviors and experience difficulties related to other segments of the IPC (Hopwood & Morey, 2007; Leihener et al., 2003; Ryan & Shean, 2007). Furthermore, experience-sampling research suggests that borderline personality is associated with interpersonal variability across interactions (Russell et al., 2007; Sadikaj et al., 2010). The ability to capture both dispositional tendencies and temporal dynamics allows the interpersonal system to provide a comprehensive, evidence-based assessment model for borderline personality features.

Complementarity

Interpersonal research consistently suggests that interactions tend to follow a dynamic pattern known as *complementarity* (Carson, 1969; Kiesler, 1983). Complementary interactions occur when there is oppositeness on dominance (the more dominant one person is, the more submissive is the other) and similarity on warmth (the warmer one person is, the warmer is the other). In general, it is presumed that interpersonal situations characterized by complementarity are associated with positive affects, relationship stability, and motive satisfaction, whereas deviations from complementarity are associated with anxiety, relationship instability, and frustrated motives (Horowitz et al., 2006; Kiesler, 1996). Empirical research supports complementarity as a probabilistic pattern of interpersonal behavior (Sadler et al., 2009), and also suggests that deviations from complementarity are related to distress and

dysfunction (Markey, Lowmaster, & Eichler, 2010) and that individuals with borderline personality are particularly prone to deviate from normative patterns of complementarity (Hopwood, 2008; Russell et al., 2007).

Copy Processes

Social learning is considered a major influence on behavior from an interpersonal perspective. Benjamin (1993, 2003) proposed three copy processes, or forms of social learning. The first is *identification*, or the tendency to act toward others as important others treated the self. According to this principle, for example, an individual whose parents were aggressive will tend to be aggressive toward others. The second is *recapitulation*, or the tendency to bring past developmental roles to new interpersonal situations. The individual who learned to fear emotional storms from parents will anticipate unpredictability from others, such as employers or therapists, and may tend to "walk on eggshells" and interpret any affective or neutral states in others as indications of anger and upset. The third is *introjection*, or the tendency to feel about and treat oneself the way one was treated. For example, an individual with critical parents may tend to suffer problems with self-criticism, negatively impacting their identity and self-esteem in adulthood.

Benjamin (1996) has provided a detailed formulation of the copy processes associated with borderline personality involving four main features. The first is a chaotic family environment which created a need for the dramatic and a discomfort with consistency. The second is chronic trauma and abandonment which created the characteristic conflict between idealization and devaluation. The third is having been attacked or criticized for healthy self-definition and self-love, which leads to conflicts about mature development and psychosocial adaptation. The fourth is having been reinforced and nurtured for being sick and miserable. Although some empirical research supports the influence of copy processes on interpersonal behavior and the association of maladaptive copy processes to health (Benjamin, 1994; Conroy & Pincus, 2006; Critchfield & Benjamin, 2008, 2010), hypotheses specific to borderline personality have not been tested directly.

Parataxic Distortion

One of the consequences of social learning is the development of certain templates, or internal working models (Bretherton & Munholland, 2008), that affect the attributions and expectations people bring to interpersonal situations. Sullivan (1953) termed misperceptions of interpersonal behavior associated with these templates *parataxic distortion*s. Distortions related to borderline personality are likely to be organized around specific social learning patterns such as those emphasized by Benjamin (1996), as well, perhaps, as more basic perceptual deficits associated with psychopathology. For example, one possible explanation for why a borderline patient is always on the lookout for others to abandon him is that he experienced chronic abandonment during development and tends to expect it (i.e., recapitulate) in new situations. This might be facilitated by his acute sensitivity to others' affective cues and tendency to interpret those cues with a negative bias. The mildest gesture of disinterest on the part of the clinician, whatever the intentions (if any) behind it, might be construed as total rejection by the borderline patient. The perception of rejection can lead to maladaptive efforts to seek reassurance or stop what is expected to happen, which can result in actual rejection in the interpersonal field, reinforcing the pattern.

Dysregulation

From a contemporary interpersonal perspective, dysregulation can occur in any of the three elements of the interpersonal situation (i.e., self, affect, interpersonal field; Horowitz, 2004; Pincus, 2005; Pincus & Hopwood, 2012). *Self regulation* involves how one thinks about himself or herself, and the degree to which one has the capacity to

regulate self-esteem and maintain a stable, goal-directed self-concept. *Affect regulation* involves hypersensitivity to interpersonal stressors, difficulties tolerating negative emotions, and deficits in constraining behavioral responses to affective experiences. *Field regulation* involves behaving in such a manner that satisfies one's interpersonal goals. In healthy interpersonal situations, field regulation tends to be mutually satisfying, whereas the efforts to regulate the interpersonal field by individuals with personality pathology characteristically lead to dissatisfaction, relationship ruptures, and dysregulation.

From an interpersonal perspective, dysregulation in borderline personality can be characterized by a dysfunctional level of affective and self-concept instability that is highly contingent upon communal aspects of the interpersonal field. Research on self-dysregulation in borderline personality involves vacillations between primarily negative self states involving insecurity, neediness, self-hate, emptiness, and identity confusion (Bender & Skodol, 2007). These vacillations, which can become so severe as to involve disruptions in reality testing and dissociation (Barnow et al., 2012), are influenced in large part by affectively charged aspects of others' communal behavior (Donegan et al., 2003; Sadikaj et al., 2010). Research on affect dysregulation suggests that borderline individuals tend to have difficulties tolerating negative affect (Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006) and constraining impulses in the face of negative emotions (Soloff, Metzler, et al., 2003). The vacillation and unpredictability characteristic of borderline interpersonal fields (Russell et al., 2007) undoubtedly contributes to the notable social dysfunction associated with BPD (Skodol et al., 2005).

Interpersonal Signatures

Interpersonal situations cascade over time in "interpersonal signatures" (Cain & Pincus, in press; Fournier, Moskowitz, & Zuroff, 2009; Pincus et al., 2010; Pincus & Hopwood, 2012), or series of "if-then" contingencies that reflect a characteristic sequence of interpersonal situations within or across interactions. For instance Roche, Pincus, Hyde, Ram, and Conroy (2013) found between-person variability in the within-person covariation of perceptions of others' dominance and warmth. This finding implies that some patients will tend to interpret their therapist's assigning homework as an expression of indifference or even hostility, whereas others will tend to experience homework assignments as reflecting therapist concern.

Such patterns provide important insights about personality and personality pathology. From an interpersonal perspective, identifying and altering these patterns is the core therapeutic task. This therapeutic task may be accomplished in different ways at different stages of a particular signature (Cain & Pincus, in press; Pincus & Hopwood, 2012), and is facilitated by interpersonal assessment and treatment principles, which we will briefly outline here. The first step involves conducting a comprehensive interpersonal assessment. This would ideally include self- and informant-perspectives on multiple IPC surfaces using standardized instruments, as well as an assessment of interpersonal dynamics in and out of the consulting room using multiple measurement methods (Pincus, 2010; Pincus et al., in press). Such an assessment would provide critical information about personality conflicts, interpersonal style, the degree and nature of interpersonal difficulties, specific contextual information about the kinds of situations that are most troubling, and the factors likely to influence those situations. Thus the assessment would alert the clinician to how the patient will relate to them in general, provide data with which to develop hypotheses about the impact of the therapist's behavior, and indicate when and where interventions are most likely to have a therapeutic (or counter-therapeutic) effect (Hopwood et al. 2013).

Interpersonal psychotherapy can be broadly characterized as involving the purposeful manipulation of therapeutic behavior to support or alter certain interpersonal signatures (Anchin & Pincus, 2010; Pincus & Cain, 2008). In general, behaving in a manner that is warm and complementary to the patient's behavior with respect to

dominance will result in smooth and stable interactions, whereas being cold and engaging in power-struggles or unproductive exchanges of passive submissiveness will result in uncomfortable, unsatisfying, and unstable interactions. This occurs both within and across sessions. A number of studies suggest that a between-session pattern involving high complementarity in early stages that facilitates the alliance, followed by non-complementarity in the middle stage which facilitates change, followed by a resolution stage again characterized by new patterns of complementarity, is associated with better outcomes than otherwise (Tracey, 2004). Thus far, research applying these principles to the treatment of borderline personality has been limited.

Summary

In this section we have laid out the basic constructs of interpersonal theory (Table 20.1) as they pertain to borderline personality development. The foundation of the interpersonal tradition is the assumption that personality is expressed and experienced in relationships, and thus the appropriate focus for understanding personality and psychopathology is the interpersonal situation. Interpersonal situations occur between a self and other and are associated with an affective experience that signals the satisfaction or frustration of motives for self-esteem and security. Agency and communion are metaconcepts that align with self-esteem and security as well as the major axes of interpersonal behavior as conceptualized by the IPC, dominance and warmth. Agency involves motives and behavior related to self-definition, achievement, and power, whereas communion involves motives and behavior related to intimacy, nurturance, and affiliation. Behavior between people in interpersonal situations is probabilistically complementarity, meaning that individuals' warmth tends to beget warmth whereas dominance begets submission. Complementarity provides a framework for understanding social dysfunction in psychopathology as well as the developmental principles of identification, recapitulation, and introjection. These principles refer to social learning processes that lead individuals to internalize working models that influence how they perceive and relate to others as adults. They can also be useful for understanding dysfunctional interpersonal patterns. Such patterns can be characterized by dysregulation in affective experience, self-concept, and behavioral output, as well as parataxic distortions of interpersonal input.

Borderline Personality Development from an Interpersonal Perspective

In considering borderline personality development from an interpersonal perspective, it is important to reemphasize that the term "interpersonal" should not be misinterpreted as implying that everything that is important for personality pathology happens between people (Pincus & Hopwood, 2012). Although research and theory incorporating the role of temperamental dispositions into the interpersonal framework are underdeveloped, it is certainly not the case that the interpersonal view would assume that borderline personality is caused solely by events that occurred between people during development *per se*, or that all the symptoms manifest in a way that is directly interpersonal. Significant nuance and flexibility in both contemporary interpersonal theory and temperament models provides ample opportunity for integration (Gratz, Latzman, Tull, Reynolds, & Lejuez, 2011; Hopwood, 2010; Pincus & Hopwood, 2012; Pincus et al., 2010).

For example, a heightened tendency to experience negative emotions predicts both later BPD and adverse life events (Gleason, Powers, & Oltmanns, 2012), implying that the temperament factors which lead to intrapersonal borderline symptoms may also contribute to the likelihood of traumatic experiences. Conversely, environmental experiences during certain periods of development may alter the expression of certain dispositions (Nisenbaum, Links, Eynan, & Heisel, 2010) and potentiate or antagonize epigenetic effects (Zhang & Meaney, 2010). Much remains unknown about the interplay between nature and

nurture in borderline development, as is the case with most other psychological phenotypes.

The value of contemporary interpersonal theory for understanding the etiology of borderline personality development is that it can provide a bridge between the stable dispositions emphasized by temperament and trait models and the dynamics emphasized by psychodynamic and social cognitive models. This bridge is provided by the IPC, which is used to conceptualize interpersonal traits (e.g., Wiggins, 1979), interpersonal dynamics (e.g., Sadler, Ethier, & Woody, 2011), and relations between traits and dynamics (e.g., Roche, Pincus, Conroy, Hyde, & Ram, in press) using the same measurement model. Interpersonal theory also provides a bridge between developmental and adult patterns of interpersonal behavior that can be informative about the nature of personality development (e.g., Benjamin, 1993; Pincus & Ansell, 2013). From an interpersonal perspective, although contexts may change over time, these patterns endure, particularly in the case of personality pathology (Benjamin, 1996). The primary focus of interpersonal diagnosis is on these patterns (Pincus & Wright, 2011).

Interpersonal Diagnosis

There are three junctures between interpersonal diagnosis (Leary, 1957) and the diagnosis of borderline personality. The first involves patterns related to personality pathology in general. From an interpersonal perspective, personality disorders have in common a propensity for parataxic distortion and associated dysregulation that leads to profound interpersonal dysfunction (Hopwood et al., 2013). These features provide an evidence-based and theoretically anchored model for distinguishing individuals with and without personality pathology, and among individuals with personality pathology, distinguishing those with greater and lesser levels of severity.

The second juncture involves what distinguishes borderline personality from other disorders. Interpersonal researchers have historically characterized personality disorders as exhibiting rigid interpersonal styles. The consistent projection of certain personality disorders (e.g., avoidant, dependent, histrionic) onto specific segments IPC measures supports the notion that they can be described in terms of inflexible interpersonal styles (e.g., Wiggins & Pincus, 1989). However, borderline personality does not consistently project onto the IPC (Hopwood & Morey, 2007), suggesting that it cannot be effectively described by a persistent interpersonal pattern. The typical borderline pattern involves temporal instability on the communal axis of the IPC (Kiesler, 1996; Russell et al., 2007). This pattern implies developmental features involving temperamental and environmental instability that are also supported by existing research. At the broadest level, it seems that borderline personality develops when a temperamental predisposition for emotional lability and disinhibition coupled with invalidating, uncertain, and chaotic environments leads to patterns of dysregulation and parataxic distortions characterized by instability in emotions, self-concept, perceptions of others, and communal motives and behaviors.

To fully conceptualize the developmental dynamics of an individual with borderline personality, the interpersonal diagnosis must go beyond characterizing average diagnostic prototypes to accommodate the idiographic patterns of that individual's interpersonal behavior and difficulties. The third level of an interpersonal diagnosis involves developing a formulation of the dynamic signatures that recur across development. In contrast to molar diagnostic labels that typically do not provide enough nuance for specific hypotheses, these patterns reflect the level at which an interpersonal diagnosis becomes most clinically and developmentally informative (Cain & Pincus, in press). In what follows, we describe a model for the development of interpersonal patterns in an individual with borderline personality.

An Interpersonal Formulation of Borderline Personality Development

Pincus and Hopwood (2012) described the following sequence for a patient diagnosed with BPD named Jennifer and her therapist (Fig. 20.2). Sessions would often start with a baseline expectation of a positive working

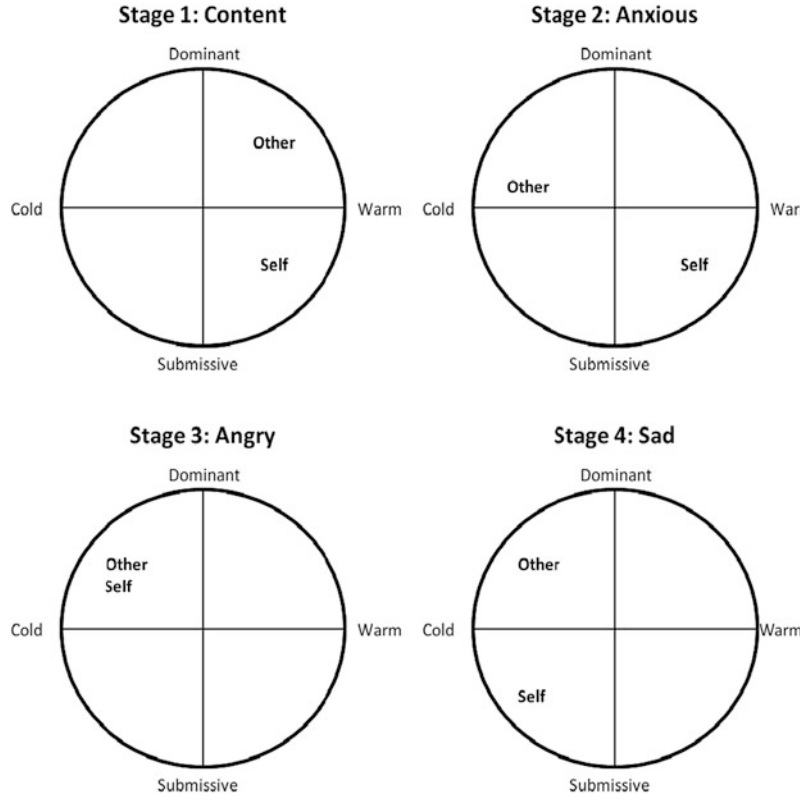

Fig. 20.2 Jennifer's pathological interpersonal signature. *Note*. Reprinted by permission of Oxford University Press, USA

relationship and hopefulness (Stage 1: therapist and patient are warm, patient experiences positive affects). At some point, Jennifer would experience the therapist as cold (whether or not this was objectively true or due to parataxic distortion) and would take this behavior as a sign of disinterest (Stage 2). She would become anxious and increasingly dysregulated, leading to angry and accusatory outbursts toward the therapist. When this behavior caught the therapist off-guard, the therapist was at risk to react countertherapeutically. For instance, he might become defensive and argue with the client. Even if he were able to rationalize his behavior as a therapeutic intervention, the effect would be an unhelpful power struggle between the therapist and patient (Stage 3: therapist and patient are both cold-dominant, Jennifer becomes overwhelmed with negative affect, is unable to mentalize the interpersonal situation or consider the therapist's perspective or observations). If unresolved, such an interaction could lead to an unfortunately familiar negative outcome (Stage 4: therapist is cold-dominant, Jennifer is cold-submissive and feels helpless, ashamed, and abandoned).

As predicted by interpersonal theory, Jennifer and her therapist observed this pattern in standardized assessments, the therapy process, and in Jennifer's current and past relationships. Her relationship with her father was particularly salient, and traumatic episodes from that relationship were a recurring theme in the treatment. At times her father showed genuine affection, but at other times he showed pseudo-affection, such as when he "paraded" her academic achievements as a way of boosting his own reputation. He was often unavailable, being too engrossed in his work or his regular, heated arguments with Jennifer's mother. He was emotionally and physically abusive. Jennifer recalled that it was very important for her to be close to her father, but she also felt as though she always needed to "walk on eggshells," feeling vulnerable to attachment loss

or abuse. She felt unable to predict or control his behavior. Her mother, who was mostly concerned with "protecting the family," focused on putting on a happy public face and refused to openly discuss the matter. In the absence of a consistent caretaking relationship to regulate her needs for validation and emotional support, Jennifer characteristically engaged in impulsive regulation efforts throughout her life. As a child, this would involve minor externalizing behaviors (e.g., "talking back"). As an adolescent and adult, these behaviors became more risky (e.g., substance abuse, impulsive sexual behavior).

Jennifer related a story from her childhood (around age 7) in which her mother was going away for the day, and her father had promised to take her to the zoo. Jennifer was pleased that he had dedicated time to spend with her (Fig. 20.2, Stage 1). As she excitedly prepared to go to the zoo, she tried on different outfits, each time presenting herself to him with pride and glee. He humored her initially, but eventually became annoyed that she was taking so long to get ready, and said "just pick something already so that we can go!" The daughter became anxious (Stage 2). Jennifer felt as though she needed to do something to correct things, in order to avoid her father's bad mood. She ran to his chair, jumped on his lap, and gave him a hug. Her father pushed her off of his lap aggressively and said "Alright this is enough! If you don't get ready in 2 min, you can forget about going to the zoo." Jennifer recalled experiencing intense anger (Stage 3) as she fell to the floor. She recalled having witnessed her mother lash out in similar moments with her father. She screamed "you never wanted to take me to the zoo in the first place!" The father said "Well, you obviously aren't a big enough girl to spend a day at the zoo with your dad. Forget it. Go to your room and stay there until you can learn to behave." She spent that day feeling helpless, unloved, and sad (Stage 4). She and her father never spoke about that episode or many other similar events. Jennifer was left alone to mentalize these experiences, which she did in the form of a pathological schema and corresponding interpersonal signature. This signature would be recapitulated in future relationships, and thus episodes like this appear to be of significant consequence for her interpersonal behavior during adulthood.

Research suggests that the shame, powerlessness, and intense fragility experienced by the borderline child in caretaking dyads is replicated in the form of intense attachments and rejections during adolescence (Bouchard, Sabourin, Lussier, & Villeneuve, 2009; Chen et al., 2004; Clifton, Pilkonis, & McCarty, 2007; Hill et al., 2008). During treatment Jennifer recalled chronic feelings of shame and emptiness during adolescence. She was able to relate these experiences to how her father treated her as though she was only lovable on his terms, and her mother acting as though validating her feelings was less important than maintaining the appearance of a happy family. She expressed the view that it was freeing for her to realize that she did not have to accept (i.e., introject) her parents' implicit feelings about her, but this provided only a short-term relief. The feelings were powerful, and she continued to unwittingly bring them into new relationships via parataxic distortion. The pattern depicted in Fig. 20.2 contributed to difficulties developing lasting attachments that could have provided sufficient support, validation, and concern. The lack of such relationships exacerbated distress and dysfunction, impeding adaptive interpersonal learning.

One salient example involved an episode with her first boyfriend. She described how she had "fallen head over heels" for her classmate, John, when she was 15 years old. As John was 1 year older than her, had a car, and was very popular, Jennifer was insecure about their relationship and had deep questions about why he would be interested in her. Their relationship developed quickly, and she felt very close to him. However, she was also very jealous, particularly because her parents were quite strict and did not permit her to spend as much time with John as she wanted. She recalled ruminating about John being with other girls while she was stuck at home. One evening she and John had plans to meet at a party at her friend's house. She was excited to see him (Fig. 20.2, Stage 1). When she

arrived, he was talking to a girl who was 2 years older than Jennifer. She assumed that John was interested in this other girl and immediately felt insecure and anxious (Stage 2). Notably, Jennifer acknowledged to her therapist that John did not end up dating the woman he was speaking to at the party, who in fact was dating one of John's friends at the time. Thus it is very likely that her attribution reflected a parataxic distortion, similar to her tendency to infer that her therapist did not care about her. Jennifer initially tried to get his attention without the girl noticing, and her lack of success confirmed for her that John had chosen this other girl instead. She approached John angrily, interrupted the conversation, and said "I thought you came here to hang out with me" (Stage 3). John then became angry, and told her to "grow up." She asked him to go outside to talk with her, and he refused. Jennifer recalled with shame having made quite a scene, and leaving the party shortly thereafter. John broke up with her the following day (Stage 4).

It is important to reemphasize two assumptions about the role of interpersonal formulation in understanding the development of behavioral patterns associated with borderline personality. First, we are not suggesting that interpersonal patterns cause borderline personality. An assumption of interpersonal theory is that focusing on the interpersonal patterns that characterize pathology maximizes clinical utility (Pincus & Hopwood, 2012; Pincus & Wright, 2011). This is particularly the case given that research is not sufficient to link causal mechanisms to treatments at this point, but we also believe that even as casual mechanisms are better established, maladaptive interpersonal patterns (i.e., disturbed interpersonal relations) will continue to be the main pathway through which borderline personality is expressed and that therapeutic relationships will continue to be a powerful means of facilitating the development of more adaptive patterns.

We are also not suggesting that the model in Fig. 20.2 reflects *the* borderline personality signature. The maladaptive characteristics of interpersonal patterns extend beyond specific themes, and include the qualities of moderation vs. intensity, oscillation vs. stability, rigidity vs. flexibility, and distortion vs. accuracy (Pincus & Wright, 2011). Some elements might be characteristic of borderline personality (e.g., instability along the communal axis of interpersonal behavior), others a function of personality pathology in general (e.g., the preponderance of negative affect), and still others might characterize Jennifer in particular (e.g., the angry response to anxiety over perceived disinterest, the resolution in sad withdrawal). From a contemporary interpersonal perspective, there is considerable potential in using interpersonal assessment to distinguish between (a) features of personality pathology in general, (b) specific interpersonal patterns related to borderline personality in particular, and (c) aspects of interpersonal functioning that can be used to describe individual differences among individuals with borderline personality.

Applying Interpersonal Theory to Research on Borderline Personality Development

While a large body of basic research has demonstrated the validity of interpersonal principles such as complementarity, copy processes, and the structure of the IPC, it would be useful for research to establish the validity of some of the core features of the model with respect to borderline personality specifically. As described above, initial research is consistent with basic elements of the model—such as the observation that individuals with borderline personality tend to be more variable across interactions, tend to couple negative interactions with negative affect more tightly than others, and tend to experience cognitive dysregulation in the face of interpersonal stressors to a greater degree than others (e.g., Sadikaj et al., 2010). However, there has been relatively less research on how these features develop during childhood and adolescence. It would be fruitful for future researchers to apply interpersonal theory to study borderline personality development.

Although most IPC assessment measures have been developed and validated for use with adults, instruments are currently validated for the assessment of IPC traits (Sodano & Tracey, 2006), goals (Ojanen, Gronroos, & Salmivalli, 2005) and behaviors (Di Blas, Grassi, Luccio, &

Momenté, 2012; Markey, Markey, & Tinsley, 2005; Klahr, Thomas, Hopwood, Klump, & Burt, 2013) in children and adolescents. However, many of the more clinically oriented instruments, such as the Inventory of Interpersonal Problems, have limited validity evidence in developing samples. Constructing assessment measures sensitive to developmental changes in interpersonal functioning that relate to borderline personality would thus be an important first step toward more fully testing hypotheses offered in this chapter.

Conclusion

We have reviewed research on the interpersonal contexts and correlates of borderline personality development and expression, outlined the major principles and constructs of interpersonal theory as they pertain to borderline personality, and presented an interpersonal approach to clinical formulation as a method for generating and testing developmental and clinical hypotheses in the treatment of a patient with borderline personality. Contemporary interpersonal theory "asserts that when we look at a domain of personality or its substrates, our best bet may be to look at it in relation to interpersonal functioning" (Pincus, 2005, p. 294). Thus we believe that contemporary interpersonal theory provides a nomological net within which to formulate, test, and ultimately integrate more theoretically narrow hypotheses about the development of borderline personality. The availability of a large body of research, evidence-based interpersonal principles, and an extensively validated measurement model can facilitate this integration. We believe such an integration will be most likely with an increased focus on interpersonal situations, which bring together all of the major features of interpersonal diagnosis into tight, testable, developmentally sensitive, and clinically rich formulations and provide powerful clues about the developmental environment and phenomenology of individuals with borderline personality.

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Part V

Treatment

Mentalization-Based Treatment for Adolescents with Borderline Traits

21

Peter Fonagy, Trudie Rossouw, Carla Sharp, Anthony Bateman, Liz Allison, and Clare Farrar

In this chapter, we apply the mentalization construct to provide a framework for understanding not only emerging personality disorder in adolescence but also adolescent breakdown more broadly (see also Chap. 12, current volume). We summarize the neurodevelopmental changes that occur in adolescence and how these temporarily compromise different facets of mentalization. We describe the principles and structure of mentalization-based treatment for adolescents (MBT-A), which incorporates monthly sessions of mentalization-based treatment for families (MBT-F). We then discuss the particular relevance of the mentalization construct for understanding self-harm in adolescence and describe the results of a recent pragmatic small-scale randomized superiority trial comparing MBT-A with treatment as usual (TAU) for adolescents with self-harm.

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Theoretical Foundations

Using the Mentalization Construct to Understand Emerging Personality Disorder in Adolescence

The developmental phase of adolescence is marked by psychological turmoil, impulsivity, dramatic and rapidly fluctuating mood, and heightened vulnerability to adaptive breakdown. Identity seems elusive, and bouts of despair alternate with feelings of invincibility. About one-third of adolescents experience a particularly stormy adolescence, marked by pervasive misery and maladjustment, impaired relations, emotional storms, regression in coping and adaptive competence, limited capacity to meet adaptive demands, struggles with identity, conflicts with parents and parental values, impulsive and self-harmful behavior, and painful questions about self-esteem and self-worth (Offer & Offer, 1975).

Seeking to identify the factors that enable a successful transition from the turmoil of adolescence to a more stable adulthood, Hauser, Allen, and Golden (2006) followed a sample of 150 teenagers, half of whom had been psychiatrically hospitalized in their early adolescence. Initially they were seen in annual interviews conducted over 4 years. Ten years later, they underwent in-depth interviews with interviewers blind to their past. A "surprising" group of former patients were functioning in the top half of all the young adults, both former patients and never

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hospitalized, in measures of social and emotional functioning, quality of relationships, antisocial behavior, and psychiatric symptoms. These individuals liked their lives and talked about them openly in a lively and fluent manner. They had lasting and satisfying relationships and were involved in work or education that they found meaningful. They were interested in psychological experience and thought about themselves and about others' experience, and they felt hopeful and optimistic about the future. Hauser et al. (2006) identified three key protective factors: (a) reflection, that is, the capacity and willingness to recognize, experience, and reflect on one's own thoughts, feelings and motivations; (b) agency, that is, a sense of oneself as effective and responsible for one's actions; and (c) relatedness, that is, a valuing of relationships that takes the form of openness to the other's perspective and of efforts to engage with others.

Adolescence appears to be a critical point for preventive and therapeutic intervention because of the increased prevalence of severe psychiatric problems and adaptive breakdown in general (Merikangas et al., 2010), and BPD symptomatology in particular (Chanen, Jovev, & Jackson, 2007). As adolescent turmoil impacts upon society, peers, school environments, family functioning and ultimately, the adolescents' own capacity to meet developmental tasks, it shapes lifespan trajectories, leading to the persistence of psychopathology.

A central hypothesis of our dynamic, adaptive system framework of developmental psychopathology is that psychiatric disorders reflect dysfunctions in core processes and mechanisms involved in social-emotional adaptation that are developmental in nature and emerge in the context of interactive systems (Masten, 2006). Thus, our mentalization-based approach to the development of BPD in adolescence and its treatment is based on the assumption that a phase-specific compromise in the capacity to mentalize occurs during adolescence.

Mentalizing is the capacity to understand and interpret other people's and one's own behavior in terms of mental states, such as desires, feelings, and beliefs (Fonagy, 1991). The mentalization-based treatment (MBT) approach assumes that the acquisition of this capacity is influenced by the quality of early relationships with caregivers, that it is vulnerable to disruption under interpersonal stress, and that core symptoms of BPD can be understood in terms of impaired mentalizing capacity in the context of attachment relationships (see Fonagy & Luyten, 2009 for a full review of the model). MBT aims to facilitate mentalizing in the context of interpersonally challenging situations.

The ability to mentalize develops in the context of an attachment relationship. At birth, infants are unable to regulate their own emotions (Fonagy, Gergely, & Target, 2007). The infant's acquisition of this capacity is facilitated by the caregiver's ability to accurately understand and respond to the moment-to-moment changes in the infant's emotional state (Fonagy et al., 2007). The caregiver mirrors back the baby's emotional experience in a "marked" way, which labels it and communicates that it is controllable. The markedness of the mirroring signals that it is symbolic of the baby's emotion, and not the mother's own emotional state, and forms what has been termed a "secondary representation" of the experience in the baby's mind (Bateman & Fonagy, 2010). These representations form the foundations for the development of a self-representation and for a sense of agency.

However, constitutional vulnerabilities (Koenigsberg et al., 2002; New, Goodman, Triebwasser, & Siever, 2008; Ni et al., 2007; Ni, Chan, Chan, McMain, & Kennedy, 2009; Siever, Torgersen, Gunderson, Livesley, & Kendler, 2002; Skodol et al., 2002) and/or exposure to neglect and invalidation in early attachment relationships (Battle et al., 2004) may result in enfeebled mentalizing capacities in attachment contexts. When the caregiver fails to respond to the infant's affective displays or the caregiver's responses are mismatched, rather than internalizing a representation of his/her own experience, the infant will internalize an image of the caregiver as part of his/her self-representation. Hence, the secondary representation in the infant's mind will be foreign to his/her actual mental state and intentionality.

Nevertheless, this alien representation becomes part of the inner self concept; we have referred to this discontinuity within the self as the "alien self." This leaves the infant vulnerable to affect dysregulation, with a weakened capacity to represent internal states of emotional arousal in attachment relationships. Experiences of incoherence within the self and affect dysregulation are subsequently dealt with through externalization, which can lead to an intense need for the attachment figure as a vehicle for the alien self. Before externalization, the inner experience of the alien self can be akin to the experience of an inner tormentor—a constant experience of inner criticism, self-hatred, lack of internal validation, and expectation of failure. Once the alien self has been externalized or projected, the external world may come to be perceived as potentially hostile, humiliating, and attacking. One way of conceptualizing self-harm and suicidality in adolescents with emerging BPD is as a result of the projection of the alien self into the individual's own body, where the fantasy is that it can be attacked and destroyed.

When the capacity to mentalize is compromised or absent, prementalistic modes of representing subjectivity that are normal in very young children tend to reemerge. The clearest of these is the tendency to assume that mental states are direct representations of psychical reality, which is normal in a 20-month-old child (Gopnik & Meltzoff, 1997). Mentalization gives way to a kind of "psychic equivalence" (Target & Fonagy, 1996) which clinicians often consider under the heading of "concreteness of thought." What is thought is experienced as real and true. The young child (and at times, for example, the individual with BPD) has an overriding sense of certainty in relation to his/her subjective experience. The hypothesis that a situation is dangerous ("there is a tiger under the bed" or "these drugs are harming me") demands extreme measures of avoidance because it is experienced in the mode of psychic equivalence, so that even a passing thought feels real. No alternative perspectives are possible; there is a suspension of the experience of doubt. This can add drama as well as risk to interpersonal experience. The sometimes exaggerated reactions of patients are justified by the seriousness with which they suddenly experience their own and others' thoughts and feelings. The vividness and bizarreness of subjective experience can appear as "quasipsychotic" symptoms of BPD patients (Zanarini, Gunderson, & Frankenburg, 1990) and is also manifest in the physically compelling memories associated with posttraumatic stress disorder (Morrison, Frame, & Larkin, 2003).

Disturbances of subjective experience linked to a failure of mentalization can also take other forms. Thoughts and feelings can come to be almost dissociated to the point of near meaninglessness. The young child creates mental models and pretend worlds, but can maintain these only for as long as they achieve complete separateness from the world of physical reality (Gopnik, 1993). In an analogous manner, patients can discuss experiences without contextualizing these in any kind of physical or material reality, as if they were creating a pretend world. Attempting psychotherapy with patients who are in this pretend mode can lead the therapist to lengthy but inconsequential discussions of internal experience which have no link to genuine experience.

Developmentally early modes of conceptualizing action solely in terms of that which is apparent can come to dominate motivation. Within this "teleological" outcomes-orientated mode there is a primacy of the physical and observable. Experience is felt to be valid only when its consequences are apparent to all. Affection, for example, is true only when accompanied by a physical expression (e.g., a touch or caress). A teleological mode of functioning may be apparent in acts of self-harm, the aim of which is to bring about actions on the part of others that represent proofs of concern. This can be mistaken for manipulativeness, resulting in patients being subtly reprimanded. Yet when the capacity to mentalize is impaired or absent, the individual may be compelled to provoke visible evidence of concern from others because of their limited capacity to experience concern in circumstances where an individual whose mentalizing capacity was intact would not find any reason to doubt it.

It is important to note that the loss of mentalizing is rarely total. More recently, a number of different dimensions of mentalizing capacity have been identified, which may be absent from patients' experience of self and other in different patterns (Fonagy & Luyten, 2009).

Children reaching adolescence with an enfeebled capacity to mentalize in the context of attachment will be less able to cope with the developmental challenges of adolescence. That is, they are less able to integrate a vastly changed body, to manage increased sexuality and affective intensity, and to deal with a greater capacity for abstraction and symbolization in a reorganized sense of self, while also coping with the increased focus on peer-directed norms and interactions and the psychosocial demands of achieving autonomy, separation, and the assumption of distinct adult roles. All this takes place in the neurodevelopmental context of synaptic "pruning" (see below), which temporarily reduces the individual's ability to modulate affect and arousal, and a limbic system generating a hunger for novelty and stimulation. All these factors converge to precipitate the adaptive collapse we identify as emerging BPD in adolescence.

In summary, we propose that adolescence is the point at which vulnerabilities resulting from early developmental difficulties are exacerbated by neurodevelopmental changes, weakening mentalizing and mentalizing-mediated affect regulation, and by intense psychosocial and developmental pressures that place greater demands on the capacity to represent the self and regulate affect. This combination of factors creates the conditions for the symptomatic expression of BPD. While no empirical study has yet demonstrated a link between mentalizing skills and protection from adult BPD in adolescents meeting criteria for BPD, the decline in BPD rates in adulthood permits us to hypothesize that the recruitment of mentalizing skills may open a path to resilience. This hypothesis, together with the conceptual soundness and empirical support of the mentalization-based approach to BPD and its treatment in adults, inspired us to develop and test an adolescent model of MBT, described later in the chapter. This model is designed to address the specific developmental issues facing young people with BPD, and adaptive breakdown in general. By harnessing natural protective, adaptation-promoting processes, we aim to create a framework to organize preventive and therapeutic interventions for young people and families in difficulty.

Mentalizing Problems in Adolescence

Findings from neuroscience suggest that adolescents' increased vulnerability to breakdown and psychiatric disorders is associated with the neurodevelopmental changes that occur in adolescence. These changes temporarily compromise different facets of mentalization, resulting in poor integration of cognitive, explicit, controlled, internally focused mentalizing with affective, implicit, automatic, externally focused mentalizing (Fonagy & Luyten, 2009). This phase-specific compromise has a particular impact on individuals with preexisting impairments, including a low threshold for the very intense and rapid activation of the attachment system and a corresponding deactivation of controlled mentalizing. These individuals are already likely to have difficulties with differentiating self and others and to suffer affect dysregulation in attachment and emotional contexts, and the impact of the neurodevelopmental changes of adolescence may leave them particularly vulnerable to developing BPD.

The adolescent brain appears to undergo two distinct neurodevelopmental processes, particularly involving the prefrontal cortex: (1) synaptic formation, followed by synaptic pruning and (2) axonal myelination, which increases the efficiency of neural transmission in the prefrontal cortex, the superior temporal cortex/superior temporal sulcus and other cortical areas. Nelson, Leibenluft, McClure, and Pine (2005) have proposed that these changes can be understood as part of a three-stage model of social information processing, involving (1) a node for detecting socially relevant cues, which matures in infancy and early childhood, (2) a node to ascribe emotional significance to the social cues, which

matures in adolescence, and (3) a cognitive-regulatory node, which matures in late adolescence/early adulthood, and serves to inhibit responses and direct behavior. While the findings on the changes in brain structures and connectivity during adolescence are complex, it appears likely that the capacities subserved by these regions also undergo developmental changes associated with less efficient connections and more diffuse activity (Leichsenring, Leibing, Kruse, New, & Leweke, 2011; New et al., 2007; Siever & Weinstein, 2009). This is consistent with findings of increasing frontal and prefrontal activity on social cognitive/mentalizing tasks between childhood and adolescence, when synaptic formation is occurring (Yurgelun-Todd & Killgore, 2006), and decreasing activity between adolescence and adulthood, when synaptic pruning takes place (Wang, Lee, Sigman, & Dapretto, 2006). These changes suggest that a developmental window might exist particularly early in adolescence, when focusing on mentalizing in the self and others might provide the kind of developmental assistance that children with adverse attachment histories may most acutely need (Fonagy & Luyten, 2011).

Evidence thus points to neurodevelopmental changes in adolescence impacting and probably disrupting the regulation of mood/affect and impulse/action by cognitive, controlled mentalizing. Developmentally, the capacity for top-down control lags behind. These changes suggest the possible neurodevelopmental context that makes adolescence a time of increased vulnerability to adaptive breakdown and, in the case of predisposed and at-risk individuals, to psychiatric disorders, particularly to the core struggles of affect dysregulation and impulsive dyscontrol that characterize BPD.

Principles of Mentalization-Based Treatment for Adolescents (MBT-A)

Mentalization-based treatment for adolescents (MBT-A) is a modification of a program for adults developed by Bateman and Fonagy (2004), usually delivered as a combination of individual and family therapy. MBT-A is a psychodynamic psychotherapy with roots in attachment theory. The primary aim of MBT-A is to help young people and their families improve their awareness of their own mental states and the mental states of others by enhancing their capacity to mentalize. The emphasis is on improving their understanding of the mental states and processes that drive behavior and relational patterns.

The fundamental assumption underlying MBT is that certain maladaptive behavior patterns and/or escalating family conflicts commonly result from a failure in mentalization, which impacts on the family members' individual and collective capacity to regulate affect. Failure of mentalizing results in affect dysregulation in an individual; as affect storms further derail thinking capacity, this in turn produces further mentalization failure. Such emotional dysregulation rarely remains confined within the individual; typically, the dysregulation and failure of mentalization migrates into the individual's interpersonal world. Here, escalating interpersonal misunderstanding and conflict resulting from mentalization failure may lead to difficulties in and even breakdown of many relationships. These transactional processes are presented diagramatically in Figure 21.1.

Furthermore, the spreading mentalization failure often culminates in some form of concrete act or "acting-out" behavior by one or more of the individuals involved; in adolescents, this is commonly self-harm, physical violence, slamming doors, breaking objects, taking alcohol/drugs, or running away.

The emphasis of MBT is not on managing the symptomatic, overt behaviors, but on understanding the specific way in which mentalization has broken down and the impact of this on the individual's social system. To this end, an important and recurring task in treatment is to track back to the moment before the breakdown in mentalization, in order to explore and understand the emotional and interpersonal context in which the mentalization failure originated.

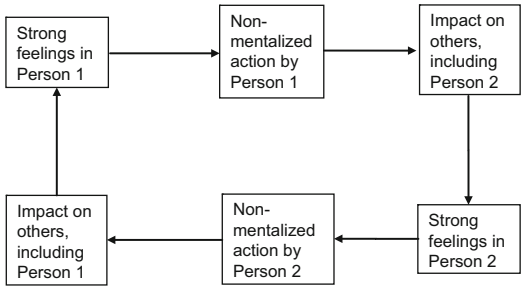

Fig. 21.1 The role of the alien self in non-mentalizing cycles in interpersonal interactions

General Principles

The MBT therapist adheres to a number of principles in order to help the young person and his/her family restore and consolidate their capacity to mentalize. The first principle is for the therapist to maintain a *mentalizing stance*, in which the primary concern of the therapist is the state of mind of the adolescent or family in the therapeutic session. The therapist continually constructs and reconstructs an image of the patient in his/her mind to help the patient understand what he/she feels and why. In this way the patient and therapist develop a mentalizing process together (Bateman & Fonagy, 2006). In the first instance the therapist aims to establish a therapeutic alliance with the patient, often underpinned by an empathic attitude towards the struggles of the adolescent and the family. The mentalizing stance requires the therapist to adopt an attitude of "not-knowing" combined with curiosity, modeling an awareness of the opaqueness of mental states and the consequent need for reflection on what the other might be feeling in order to account for their behavior (rather than assuming knowledge and reacting impulsively). In order to understand what the patient is experiencing from moment to moment, the therapist asks for detailed descriptions of the experience (by using "what" questions), rather than requesting explanations (using "why" questions); this helps to create reflection and the opportunity to explore the relational context. The therapist also actively highlights alternative perspectives, to model to the patient the legitimacy of differences in perspective. As part of the mentalizing stance, the therapist should be sensitive to his/her own errors, take responsibility for these and, where appropriate, use them as opportunities to revisit and mentalize what happened, exploring the feelings engendered in both patient and therapist as the result of the error. This also involves self-reflection and self-questioning about the therapist's own contribution to a patient's mental state.

A second basic principle of MBT necessitates a *mentalizing structure around the therapist* in order for the MBT therapist to maintain the mentalizing stance. The key elements of this structure include a mentalizing clinical team, a theoretically coherent treatment approach, and consistent application of the approach over time. In dealing with young people and their families who communicate and manage their emotions through action and acting-out behavior, the therapist may be invited, and expected, to take part in some form of action in order to intervene. As depicted in Figure 21.1, highly aroused states in an individual easily induce aroused states and mentalization failure in those around them. The therapist is not immune to this experience, and at times when the therapist is confronted with immediate anxiety or the provocation of particular types of acting-out behavior, his/her own ability to mentalize will be challenged. For this reason a thinking, and mentalizing, team, who are not exposed to the heat of the arousal, is an essential resource to help the therapist restore his/her own mentalizing abilities before he/she acts impulsively.

Third, the therapist's interventions are guided by the principle that *affective states are usually aroused in interpersonal contexts*. Therapists often encounter young people who state that they feel depressed and wish to die, and who report that their feelings are not related to anything particular—it is "just the way they feel." In such instances this assumption can help the therapist to identify a way through this senseless state in order to mentalize the adolescent's experience of meaningless distress by identifying links with recent interpersonal encounters. Thus, this work is intrinsically relationship focused.

The final principle, given the proneness to mentalization failures amongst young people, is the therapist's role in *creating a mentalizing scaffold* to protect against the young person's vulnerabilities. This involves tailoring interventions in line with the patient's level of arousal to ensure that the fragile capacity to mentalize is not compromised further. Thus, it is important for the therapist to be empathically attuned to the patient, as this guides mentalizing dialogue. When the heat of arousal is high, interpretations should be short, empathic, supportive, and affect-focused; when the patient is in a calmer state, further exploration may be possible, although deep interpretations about unconscious drives or unfamiliar aspects of a person's psyche rarely feature within this model.

These guiding principles underline the importance of keeping in mind that patients are not static—in certain states of mind their ability to listen and interact is likely to be very limited, while at other times they will be more readily able to engage. MBT therapists should be "light on their feet" and alter their interventions according to the patient's mental state, which can change from moment to moment. The approach also calls for the therapist to be sensitive to their own contribution to the patient's state of mind. It is important that the therapist and clinical team notice their own contributions to the process and take responsibility for mistakes in order to prevent the escalation of mentalizing breakdowns. Misunderstandings will inevitably happen; the challenge is to be aware of, take responsibility for, and try to repair them; in other words, to understand misunderstanding. Consistent re-experiencing of efforts to rewind and mentalize the moments when mentalization breaks down in the therapeutic process ultimately strengthens the patient's own mentalizing ability.

The Role of the Alien Self in Mentalization Failure

Figure 21.1 illustrates the role of the alien self in non-mentalizing cycles in interpersonal interactions, as might occur within a family. In situations where the alien self is activated and expressed, mentalization failure can lead to escalating mutual misunderstandings between the parties, ultimately leaving both parties feeling attacked by the other, while at the same time feeling terribly bad feelings about themselves (e.g., they are bad, incompetent, unlovable).

It is important for clinicians to be aware of the potential presence of the alien self and its ability to elicit non-mentalizing cycles of interaction. It is particularly important to be aware of the likelihood in these circumstances for adolescents and their families to feel blamed or attacked. The golden rule is to form an empathic alliance with individuals in this state, and to acknowledge how hard it must be to feel as if whatever they try fails. In this way the therapist makes empathic contact with the authentic self, which creates an experience of safety in the therapeutic alliance and a reprieve from the onslaught of the alien self. This, in turn, creates the opportunity for reflection, setting the stage to try to mentalize where things went wrong. Once mentalization is restored, the influence of the alien self will be reduced.

Remoralization, Remediation, and Rehabilitation of Mentalizing

When family life is dominated by non-mentalizing interactions, people fail to make sense to one another. As described above, when under the influence of the alien self, all parties often end up feeling bad or blamed and attacked. Thus, at the start of family-based MBT, the therapist provides the family with a formulation (described in the next section of this chapter), which explains the therapist's understanding of the family's difficulties in a mentalizing and empathic framework. The formulation aims to help the family members to see themselves and the others in the family from a different perspective—one that describes behaviors and interactions as understandable responses to mental states, and mental states as understandable responses to others' behaviors. This "remoralization" is

supported by interventions that aim to remedy the adolescent's specific neuropsychiatric and addictive disorders that exacerbate his/her mentalizing problems and which, in turn, are made worse by breakdowns in mentalizing. These two steps serve as the launch pad for the longer-term process of restoration of the mentalizing capacities that generate agency, reflection, and connections with others, and promote more effective means of managing stress, adversity and vulnerability (Bateman & Fonagy, 1999).

The MBT-A Outpatient Intervention

The MBT-A program for adolescent outpatients is a year-long manualized psychotherapy program involving weekly individual sessions, often but not exclusively in concert with monthly sessions of mentalization-based family therapy (MBT-F; see below for a brief description and Asen & Fonagy, 2012 for further details). Treatment is divided up into four phases, all of which are derived from the original MBT model for adults (Bateman & Fonagy, 2004). A treatment manual can be obtained from the second author at request.

Assessment

The assessment phase normally lasts approximately 2 weeks and often involves one or two sessions with the MBT-A therapist, as well as sessions with the family therapist. Psychiatric disorders are assessed for, using a combination of clinical evaluations (observations and interview) and standardized measures. The diagnostic evaluation aims to identify conditions that may require adjunctive treatments such as medication, to highlight any comorbidities, and to make the therapist aware of any psychiatric conditions that can impair the ability to mentalize. Cognitive and executive functioning and emotional regulation are assessed. Knowledge of any cognitive impairment that may interfere with the adolescent's mentalizing capacity will help the therapist to mentalize the experience of the adolescent and family in a way that incorporates these difficulties.

A key part of this phase is assessment of the adolescent's ability to mentalize. Mentalization failure may be persistent (as may be the case in adolescents with certain neurodevelopmental disorders) or intermittent; in the latter case, an individual may mentalize well in general, but in emotionally highly charged situations may show a temporary inability to recognize the feelings and experiences of others. Mentalization ability is therefore assessed in the context of attachment relationships in the adolescent's life, focusing on relationships with the family and with peers. Mentalization within the family may be assessed by observing interpersonal interactions during a family session and by the use of mentalizing questions that aim to elicit information about the young person's ability to understand their own and others' feelings (e.g., "What do you think you felt inside when you did that?"; "What do you think your Mom felt when she said that?"). In addition, while the focus of MBT-A is on current events, questions about the adolescent's relationships over a longer time frame, and about how they think their current life has been affected by the past, are used to help assess the adolescent's ability to self-reflect and mentalize about self and other.

A range of assessment tools may also be considered to complement clinical observation of mentalizing capacity. For instance, the Reflective Functioning Questionnaire for Youth (Ha, Sharp, Ensink, Fonagy, & Cirino, 2013) has recently been validated and provides a good measure of self-report mentalizing capacity in adolescents. The Movie Task for the Assessment of Social Cognition (Dziobek et al., 2006) has also been used in inpatient adolescent settings (Sharp et al., 2009) and provides valuable information for mentalization-based case formulation.

Initial Phase

The assessment phase is followed by a session with the adolescent, during which they receive a copy of their formulation, which is discussed

between the patient and therapist. A similar process occurs in MBT-F with a formulation session discussing the difficulties within the family. An example of an MBT-A formulation is provided in the Appendix. The formulation explains the diagnosis in mentalizing and relational terms. It aims to demonstrate understanding of the adolescent (and family, in MBT-F) in a way that makes them feel understood and to help them to see themselves both objectively and in terms of the subjective experience of the other family members. The formulation provides a launch pad for outlining a treatment plan for the adolescent and family. In MBT-F, it also provides a basis for discussion within the family; this plays the important role of enlisting the family members as partners in the therapy, and helps to move the focus from the adolescent's problems as behaviors that need to be controlled or eliminated to a mentalizing conversation that enables the family members to understand each other's point of view and express their own perspectives.

The formulation includes a crisis plan. This aims to highlight factors that may trigger an emotional storm or impulsive behavior in an adolescent (for example, times when the adolescent may feel rejected, humiliated or bad about themselves). It suggests actions that the adolescent can take to try to restart mentalization (e.g., to stop and reflect on what happened in the moments before the feelings arose) or, failing that, to engage in activities to try to divert the adolescent away from impulsive, destructive or self-harming behaviors (such as strenuous physical exercise, talking to a trusted person, or doing something to occupy themselves).

A treatment contract is also agreed. This sets out the duration of treatment and commitment required from all those participating; it explains the importance of everyone's engagement and the process of working together in the therapy.

The family formulation session is followed by a psychoeducation session, which may be delivered to the individual family or in a group format. This aims to help the family understand the principles that behavior has meaning, that feelings arise in a relational context, and that people have a powerful emotional impact on one another. Psychoeducation may involve informal discussion with the family, using examples from everyday life, or in multifamily groups it may make use of group discussion, role-play and videos.

Middle Phase

The middle phase of MBT can be seen as the remediation and rehabilitation phase of therapy, and lasts 9–10 months. It aims to enhance mentalization in the adolescent and family through developing their ability to become more aware of mental states in themselves and others. Sessions aim to enhance the patient's capacity to represent his/her own and others' feelings more accurately in situations that entail intense emotions (activation of attachment feelings by rejection, interpersonal conflict, etc.). The ultimate aim is to transform non-mentalizing, and the coercive interactions that result from non-mentalizing states, into a more mentalized way of interacting within the family, which enables more mutual understanding and trust, and clearer communication. This phase also aims to help the adolescent and family gain better impulse control (as impulsivity undermines the development and use of mentalizing ability). In the course of this phase, specific interventions are introduced to manage harmful or impulsive behaviors such as self-harm, substance abuse, or threatening or violent behaviors.

MBT-A sessions are on the whole unstructured, focused on the young person's current and recent interpersonal experiences, and maintain a constant focus on the mental states likely to have been evoked by these experiences. The aim of the family sessions is to improve the family's ability to mentalize, particularly in the context of family conflict (both conflicts concerning the patient and those mainly involving other family members). Throughout the sessions, the therapist maintains a mentalizing stance (as described above), which acts as a constant orientation. A number of specific techniques are also used

throughout the therapeutic work described below. In the Appendix, we also provide a short excerpt from a session with a family to demonstrate some of the principles below:

Supportive and empathic interventions are used to establish emotional contact and a therapeutic alliance. At the start of therapy, this technique is used almost exclusively; only once the alliance is established will other techniques be useful. At any stage in the therapy when the adolescent is in a state of emotional arousal and mentalization failure, the therapist returns to this empathic stance to attempt to mentalize the aroused affect with the young person. This intervention is an active process, making use of active questioning and where necessary checking that the therapist has understood what the adolescent has said.

Clarification and elaboration techniques are frequently used to try to make sense of the adolescent's behavior. Clarification is an active technique, in which the therapist asks many questions with the aim of reconstructing the events that led to a mentalization breakdown, so that they are more clearly understood. Clarification is followed by, or used in conjunction with, affect elaboration, in which the therapist attempts to elicit feeling states. Here, the therapist may often help the young person by reflecting on how it must feel to be in that situation, but without telling the patient what he/she is feeling. Careful exploration can uncover deeper feelings that may not be apparent—for example, the adolescent may appear angry, but underlying this there may be a sense of guilt, humiliation, or failure. Clarification and elaboration act as mechanisms to slow the action down and help to identify feelings while trying to identify the interpersonal context in which these feelings were triggered. In this way the therapist helps the young person to mentalize what they feel and what happened.

Basic mentalizing techniques can be summarized as "Stop, listen, look" and "Stop, rewind, explore" (Bateman & Fonagy, 2006). At a point in the session where it appears that non-mentalization interactions are taking place, the therapist encourages the adolescent to "rewind" to the point where mentalization was lost and then explore what happened at that point. Challenges in the session (such as acting-out behavior in an MBT-F session) are also used to try to restart mentalizing, in the hope that the difficult feelings underlying the behavior can be expressed and mentalized.

In the context of MBT, "transference" refers to all relationships in the patient's life, not just the therapeutic relationship. It is restricted to the here-and-now; the emphasis is on understanding the current interpersonal interactions and the feeling states that result from these interactions. If it becomes clear that a patient has particular ways of expressing or experiencing specific feelings, the therapist may comment on this to draw the young person's attention to a characteristic non-mentalizing distortion that seems to occur in several interactions. We call these comments *transference tracers*.

Having used clarification and elaboration to explore and understand the adolescent's perspective, if the heat of the therapeutic interaction is not too high the therapist may proceed to present alternative perspectives for the young person to consider. This technique, which we refer to as *interpretive mentalizing*, must be used with caution, adopting an inquisitive, "wondering" approach so that the patient does not feel that his/her perspective is being dismissed by the therapist.

Mentalizing the transference involves the therapist reflecting with the patient on the here-and-now interaction between them. The therapist supports the patient to think about how each impacts on the other, how their different perspectives are affected by their interaction and by their thoughts about the other's mind, feelings, or thoughts. The same techniques that are used to mentalize other relationships (clarification and affect elaboration) are used in mentalizing the transference. This also involves the therapist accepting responsibility for any enactment, that is, situations where the therapist may have been drawn into the transference and acted in a way that is consistent with the patient's expectations.

In general, interventions are simple "soundbite" interventions that do not require excessive processing competencies on the part

of the young person. They are affect-focused (e.g., love, desire, hurt, catastrophe, excitement) because in that domain the young person finds it easier to generate constructions of subjective states. Interventions aim to focus consistently on the patient's mind (and not, as might be tempting in the case of self-harm, for example, on the young person's behavior). They relate to current events or activity—that is, to the adolescent's mental reality (either evidence-based or in working memory)—where subjective states are more likely to be accessible. To facilitate accessibility, the therapist often uses his/her own mind as a model, not in the sense of self-disclosure, but as a normalizing influence suggesting to the young person how the therapist may feel or may think in the context the young person presents. In general, the therapist aims at identifying non-mentalizing and recovering mentalizing on the many occasions when apparently it appears to be lost in the course of the discourse. The therapist focuses on a break in mentalizing—which may be apparent because of psychic equivalence, pretend mode, or teleological thinking—and, as discussed above, "rewinds" to the moment before the break occurred. The therapist encourages the young person to explore the current emotional context in the session by identifying the momentary affective state between patient and therapist, including identifying the therapist's contribution to the break in mentalizing (which, importantly, demonstrates humility to the young person). The therapist may gradually seek to mentalize the therapeutic relationship, but does so only very slowly and carefully because the activation of intense attachment feelings in the adolescent will undermine his/her capacity to mentalize.

Final Phase

As in other psychodynamic psychotherapies based on ideas from attachment theory, the final phase addresses separation issues along with managing anticipated challenges in a mentalizing manner. It aims to increase the adolescent's independence and responsibility, and consolidate relational stability and a sense of mastery (as opposed to helplessness or passivity) in the adolescent and his/her family. In addition, a coping plan is created for the family, setting out what to do in the future if difficulties return. The final phase of MBT-A lasts for approximately 2 months and commonly includes a tapering-off of sessions at the end. Some families also find it helpful to return for one final family session a few months afterwards.

Mentalization-Based Treatment for Families (MBT-F)

MBT-F, which can also be used as a stand-alone intervention (Asen & Fonagy, 2012), integrates attachment theory with systemic practice, making links between external relationships and inner worlds and connecting behavior and interaction patterns with meaning-making. The aim of MBT-F specifically is to enhance mentalization in family relations and to reduce impulsive enactments, coercion, non-mentalizing interactions, and escalating affective storms. MBT-F focuses on emotions as cues to what goes on within individual family members and pays specific attention to emotional regulation. A key goal of the approach is to increase the empathic understanding that parents and caregivers have for their children and, depending on the developmental stages of the children, vice versa. Asen and Fonagy (2012) describe the treatment intervention as an MBT-F "loop," which involves working with the family as collaborators to identify a moment of non-mentalization. The moment is then highlighted and given a name (which externalizes it). Then, great care is taken to mentalize the moment and to see what everyone experienced in the moment. This may highlight the aspects of the interplay between the inner and external representations of the family in each member of the family. This moment can often be generalized to other moments. In this work the emphasis is on thinking about feelings before planning actions or thinking about how to do things differently next time.

Adolescent Self-Injury and Mentalization

The percentage prevalence of deliberate self-harm among adolescents in Europe has been increasing and has reached double figures in most countries (Madge et al., 2008). The phenomenology for non-suicidal self-injury (NSSI) is fairly well known and includes diverse factors including: (1) to relieve intense distressing affect by the use of sharp physical pain, which can distract the sufferer from his/her unbearable feelings; (2) self-punishment, which the adolescent sees as "deserved"; (3) to gain attention so that other people can see the young person's distress; (4) to make others feel guilty and change their behavior; (5) to fit in socially with peers who self-injure (Vrouva & Fonagy, 2009; Vrouva, Fonagy, Fearon, & Roussow, 2010). NSSI predicts the persistence of suicidal ideation (Asarnow et al., 2011; Wilkinson & Goodyer, 2011). NSSI and suicidal behavior are on the same spectrum of self-destructive behavior and have similar correlates. Wilkinson, Kelvin, Roberts, Dubicka, and Goodyer (2011) found that 55 % percent of adolescents who had self-injured previously made suicide attempts in the 28 weeks postassessment, compared with less than 20 % of those who had not self-harmed at baseline.

NSSI has been shown to precede other suicidal behavior, probably due to similar diatheses (Ougrin, Tranah, Leigh, Taylor, & Asarnow, 2012). These include: (1) poor social problem-solving ability; (2) high levels of arousal in response to frustration; (3) difficulties with emotion regulation and distress tolerance; (4) frequent persecuting, self-critical cognitions; (5) poor family functioning; and (6) history of trauma, which perhaps desensitizes to pain.

However, mentalizing theory might also allow us to account for the link between NSSI and suicidal behavior in terms of a compromised mentalizing capacity. Risk factors common to NSSI, suicidal behavior, and vulnerability to loss of mentalization include trauma, age, family functioning, and problems of verbal expression. Furthermore, emotion dysregulation which has loss of mentalizing at its root is clearly linked with NSSI. The subjective experiences that "make sense" of both NSSI and suicidal ideation are pre-mentalizing subjective states, for example, psychic equivalence, and pretend mode. The "manipulativeness" that may be evident in adolescents with NSSI or suicidal ideation is an indication of the teleological stance in action ("I have to *do* something to make you think something"). Projective mechanisms (experiencing pain/pleasure through taking possession of the subjective world of the object) are also common to the two.

Treatments specifically designed to manage NSSI have not been particularly helpful in adolescents. For example, a meta-analysis of engagement in the randomized controlled trials (RCTs) that have reported the effect of specific psychological treatment versus TAU in adolescents with self-harm yielded no differences favoring specialist treatment (Ougrin & Latif, 2011). A developmental group therapy for adolescents with a history of repeated self-harm (Wood, Trainor, Rothwell, Moore, & Harrington, 2001) has initially yielded positive results; however, a replication single-blind study with parallel randomized groups undertaken at three sites in Australia was unsuccessful (Hazell et al., 2009). Similarly, a replication in the United Kingdom using an analogous design to the original study, with 183 adolescents in each group, also found no treatment effects relative to TAU (Green et al., 2011). More recently, 77 adolescents (aged 12–18 years) with a history of deliberate self-harm who met two DSM-IV criteria for BPD were randomly assigned to 16 weeks of DBT or enhanced usual care (EUC). Results from posttreatment assessments indicated that DBT appeared to be more effective than EUC in reducing deliberate self-harm, suicidal ideation, depression, and hopelessness (Cooney, Davis, Thompson, Wharewera-Mika, Stewart, & Miller, 2010). Twenty-nine adolescents with a history of a suicide attempt or NSSI in the past 3 months were randomly assigned to DBT or TAU. A third randomized trial evaluating DBT for adolescents diagnosed

with bipolar disorder was recently conducted by Tina Goldstein, Ph.D. and colleagues at the University of Pittsburgh (Goldstein et al., 2007).

Multisystemic therapy has also been assessed as a treatment for suicidal behavior in youths, compared with emergency psychiatric hospitalization (Huey et al., 2004). Multisystemic therapy was significantly more effective than emergency hospitalization at decreasing rates of attempted suicide at 1-year follow-up, but this was based on a single item from the Child Behavior Checklist 92-item self-report questionnaire, which is not a valid measure of suicidality. Treatment effects were not evident in parental reports. In a pilot trial involving 31 patients, Donaldson, Spirito, and Esposito-Smythers (2005) also reported no difference between supportive relational and skills-based treatments for adolescents following a suicide attempt. A study of early intervention for adolescents with BPD compared cognitive analytic therapy and good clinical care (Chanen, Jackson, et al., 2008; Chanen, Jovev, McCutcheon, Jackson, & McGorry, 2008) and found no difference between the two.

The negative results of these early trials of treatments for NSSI suggest that engaging adolescents who self-injure is difficult. Therapies focused on problem-solving, specific cognitive distortions, or enhancing parenting supervision (rather than mentalization of the young person by the family) assume that the problem is egodystonic, something that the young person would like to tackle. The treaters appear not to recognize the developmental challenge with mentalization for *all* adolescents and the fertile context this creates for action-oriented expressions of self states. Further, in ignoring attachment theory and the relationship between mentalization and attachment, the interventions overlook the likely failure of mentalization in an attachment context. The unevenness of adolescents' capacity to cope with their own subjective experiences, and those of their attachment figures, may be a clue to why young people who are perfectly capable of solving problems and who appear to lack few skills when working with a therapist show substantial deficits when confronting interpersonal situations that activate their attachment system. Related to this, we should also consider the significant impact of trauma on the young person's willingness to engage in mentalizing in social situations.

Evidence for the MBT-A Intervention

Self-harm is a common feature of BPD in adults and among adolescents who show borderline features. On the basis of the evidence from RCTs showing MBT to be effective in reducing self-harm in adult patients (Bateman & Fonagy, 1999, 2008), Rossouw and Fonagy (2012) carried out an RCT of the outpatient MBT-A intervention described above in a sample of adolescents with self-harm.

The study was a pragmatic small-scale randomized superiority trial comparing MBT-A with TAU for adolescents with self-harm (defined as any intentionally self-inflicted injury). Eighty participants (mean age 14.7 years, 85 % female) were recruited from consecutive cases presenting with self-harm to community mental health services or acute hospital emergency rooms in north-east London; all cases who did not require inpatient treatment were invited to participate. Individuals with a comorbid diagnosis of psychosis, severe learning disability (IQ < 65), pervasive developmental disorder, or eating disorder in the absence of self-harm were excluded. At intake, there was a high level of mental disorder: 97 % of adolescents met criteria for depression and 73 % BPD. Participants presented with a variety of methods of self-harm either in the index episode or in the past (95 % cutting, 64 % overdose, and 80 % attempted suicide).

Participants were randomly allocated to either MBT-A (intended to be delivered with MBT-F sessions as described above) or TAU. TAU was not manualized but was delivered based on UK National Institute for Health and Clinical Excellence guidance for self-harm (National Institute for Health and Clinical Excellence, 2004). In the MBT-A arm there was a special focus on self-harm behavior viewed by the young person

and therapist from a mentalizing standpoint. MBT sees the function of NSSI as reestablishing the "self-structure" (sense of self-coherence) impaired following the loss of mentalizing. The intervention therefore involves exploring the reasons for destabilization of the self-structure (e.g., "Tell me when you first began to feel anxious that you might do something") leading to a "mentalizing functional analysis" tracing the feelings and thoughts that the young person was struggling to fully experience and which were "short-circuited" in the act of self-injury. Affect forms the focus of the therapy, as it does in MBT-A in general. Mostly, the contents of subjective experience concern feelings of badness, experienced in psychic equivalence as "I am bad." The therapy explores a range of affects concerning rejection, loss, hurt, abandonment, and panic. The difference is not in the specific content of the feelings, but rather in the experience they create in the young person, which goes considerably beyond that which would normally be associated with such affects. Thus, for example, emptiness and experience of a void or a sense of "being in a black hole" may be the sequelae of intense emotional experiences because of the loss of sense of a mentalizing agentive self underpinning the individual's subjectivity. In linking the experience to the interpersonal context that triggered the feelings, by rewinding to the moments before mentalizing was lost, the therapist rebuilds the adolescent's capacity to tackle these situations in a mentalizing way wherever possible.

The primary outcome was self-harm assessed by self-report at baseline and 3-monthly until 12 months following randomization, using the self-harm scale of the Risk-Taking and Self-Harm Inventory (RTSHI). Self-reported self-harm was confirmed with interview at baseline and 12 months, using the Childhood Interview for DSM-IV Borderline Personality Disorder (CI-BPD). Secondary outcomes included depression, risk-taking, and emerging BPD. Two measures related to hypothesized mechanisms of change were also administered pre- and posttreatment: mentalization was assessed using the How I Feel (HIF) questionnaire, and attachment status was assessed using the Experience of Close Relationships Inventory (ECR).

Overall, the number of hours of clinical attention received by the two groups did not differ significantly, and there was no difference between the two treatments in the percentage of patients completing 12 months of treatment (50 % MBT-A, 43 % TAU). Significantly fewer participants in the TAU group (33 %) than in the MBT-A group (63 %) received a family-based intervention ($p = 0.003$). In one-third of cases in the MBT-A group, no family sessions were attended; this was mostly linked to the family's refusal to participate in the adolescent's treatment, and in a few cases the young person did not wish the family to be involved. The number of psychiatric review sessions did not differ significantly between groups ($p = 0.10$).

Both groups showed significant reductions in both self-harm and risk-taking behavior, but the decrease in RTSHI scores was significantly greater for the MBT-A group on both variables. At 12 months, self-harm scores were significantly lower for the MBT-A group. The odds of reporting at least one incident of self-harm in the past 3 months was reduced only for the MBT-A group; the between-group difference was significant at 12 months (56 % vs. 83 %, $p = 0.01$). Interview data on self-harm confirmed the self-report result. In the TAU group, 68 % of participants were rated as definitely self-harming by the blind assessor, compared with only 43 % of the MBT-A group ($p < 0.05$).

The authors concluded that, in general, both groups benefitted from treatment in terms of both self and observer reports of self-harm. Neither intervention achieved a complete recovery, and 69 % of the overall sample were still self-harming at the end of the 12-month intervention. However, those receiving MBT-A had a better recovery rate than those receiving TAU (44 % vs. 17 %). Interview-based assessments blind to group assignment confirmed the differential effectiveness of the treatments and estimated the rate of recovery in both groups somewhat higher (57 % vs. 32 %, respectively).

The standardized mean difference between baseline and posttreatment depression scores for the MBT-A group was 1.12 ($d = 0.49$), indicating moderate improvement. The authors note that these findings in relation to depression are clinically relevant, as 97 % of the sample had comorbid depression and depression is thought to play a central role in triggering self-harm (Wilkinson et al., 2011).

Although the study did not focus on BPD and did not aim to recruit individuals with BPD, nearly 75 % of participants met DSM criteria for BPD. A reduction was seen in both BPD diagnosis and BPD traits in the MBT-A group at the end of treatment, in line with previous reports of MBT in moderating BPD symptoms (Bateman & Fonagy 2001, 2009), suggesting that the outpatient MBT-A intervention may be useful for adolescents with BPD.

Conclusion

MBT-A works by helping the young person learn about the complexities of his/her thoughts and feelings about him/herself and others, how this relates to his/her responses, and how "errors" in understanding him/herself and others lead to actions. It is not for the MBT-A therapist to "tell" the young person how he/she feels, what he/she thinks, how he/she should behave, or what the underlying reasons are—conscious or unconscious—for his/her actions or difficulties. By adopting an inquisitive, "not-knowing" stance, the therapist conveys a sense that mental states are opaque but worthy of interest and potentially extremely helpful in interpreting one's own and others' actions. At the heart of MBT-A is the idea that the adolescent will regain his/her developmentally appropriate competencies if the therapist makes their mind available for the young person to find his/her own capacity to think therein. In this way the therapy replicates the developmental process, where the infant's seeking of his/her subjectivity in the caregiver enables him/her to develop a sense of agency and competency in relation to his/her subjective experience.

Appendix

Example of an MBT-A formulation

Background Information

When you were referred to this service you reported a 2-year history of feeling depressed and harming yourself. At times you have felt so depressed that life did not feel worth living. You thought your parents' divorce 3 years ago, your mother's subsequent depression, your father's drinking and his recent violent relationship with his girlfriend all played a role in making you depressed. You spoke about feeling guilty as if it was all your fault. Before you came to us for help you entered into a relationship in which you allowed someone to treat you in a disrespectful manner, almost as if you were being punished. All of this made you feel terrible about yourself.

Personality Style

From what you told us and based on the tests you completed, it seems as if you tend to be an introverted person and that you value time on your own, as it helps you to feel calm. When you are with friends you can feel very worried that you will be hurt or that you will not be liked. When you feel like that, you hold yourself back, but in doing so you do not give people a chance to like you, which in turn reinforces for you that they do not like you.

It also seems that at times you are able to form passionate attachments to others, but then you can become suspicious and anxious that you may be rejected. It seems that relationships can at times make you feel a rollercoaster of different feelings, from love to anger. Sometimes your mood can also swing from sad to happy. Sometimes you can feel so overwhelmed emotionally that your mind goes blank and then you can feel numb. The problem with this coping strategy is that it then makes you feel disconnected from what you or other people feel, and then it is sometimes difficult to understand what is going on and so action feels as if it is the only thing

available to you—it is at these times that you have a tendency to harm yourself.

You often relate to others in a self-sacrificing manner and at times even allow others to take advantage of you. You also at times tend to present yourself in a negative light to others. You can feel deep pain as your mind often dwells on past pain and misfortunes. This is very sad, because then you are not able to see your own good qualities.

Treatment Plan

We propose to offer you a treatment in which we suggest a combination of individual therapy once a week and family therapy once or twice a month delivered by the community team.

Crisis Plan

Trigger factors that you and I identified are times when you feel rejected, humiliated, or bad about yourself. As we have discussed, these feelings do not just arrive out of the blue: they are likely to have been triggered in a close relationship. When you have those feelings you tend to rush into an action to take the feelings away. When you feel like that again, I would like you to try to stop the action by trying to delay it for 10 min. Then, use the 10 min to try to reflect on what was happening a few moments before you had the bad feeling. That might help you to understand more clearly what it is that you feel, as well as what might have happened in a close relationship that may have contributed to the feeling. Once you have this understanding more clearly it may be easier to think about a solution or to see things from a different perspective. Once that has happened you may not feel as if you need to rush into action any more. If that fails and you still feel as if you might harm yourself, try to explore alternatives to self-harm:

Do something physical and strenuous like going for a run, try to distract yourself, talk to a friend or someone you trust, or try to think about a person you know who loves you and imagine what that person would feel and say to you if you were to talk to them.

Sometimes you harm yourself when you feel emotionally numb. When you get into such a state of mind, try to remember that it is not a good state of mind for you to be in and it is harmful to you. Try to bring yourself back to reality—do something to occupy yourself, like talking to someone, playing a game, writing a poem, painting, or watching something on TV that can hold your attention. Don't just sit and stare into space with your mind full of negative thoughts about yourself.

If all else fails, call the clinic and ask to speak to me and I will call you back when I can.

Vignette from MBT-A

This is an example of a session with a 15-year-old male who was referred to our service with a history of cutting himself, taking overdoses and having great difficulty in managing relations at school. He also has a strong history of violent outbursts and impulsive behavior, including one incident in which he was reprimanded by the police for attacking another youth. He grew up with his mother and two half siblings from different fathers. His mother had a past history of drug abuse. The young man experienced life as unpredictable; he grew up surrounded by volatile relationships and experienced consistently inconsistent boundaries. This upbringing meant he had very little ability to manage his own feelings and hence frequently fell back on concrete ways of trying to reassure himself of his safety and manage his feelings. This vignette from one of his sessions illustrates both his concrete mentalizing style and the therapist's attempts to mentalize his feelings.

Patient: I broke up with Michelle. You remember I wanted to see her last Friday and she said she was busy. Later I found out that she was only busy for an hour and I could have seen her. So Saturday I thought "I am not having it, I may as well end it with her rather than wait around for her." I sent her a text and said, "If you do not call by 5 o'clock it is over." She texted straight back saying "I am sorry but I am a happy person and you are always moaning and it brings me down." So I thought, OK whatever, and just left it.

Therapist: Gosh, what did that make you feel?

P: I felt nothing. I just don't understand, I was always happy when I was with her. I don't see how she could say I am always moaning. The only thing I moaned about was that she just never answered her phone. Any boyfriend would want that, isn't it?

T: So when she did not answer her phone, what did you feel?

P: It felt as if she did not care. Jenny always answered her phone and that is how I knew she cared.

T: And when you felt she did not care, what did you do?

P: I would phone her non-stop and I would text and leave messages. It is not right to ignore me like this. I sometimes called her 20 times and she would ignore me. I then think she's met someone else. And I sort of saw it coming, so Friday evening when I went dancing I flirted with people and then I met this new girl. So I thought I'd like to take her out, so I pretended to be drunk and then said to her that I would like to take her out. I thought if I pretend to be drunk and if she says no, then I will just say the next day that I was drunk and that I do not remember anything. Then I won't have to feel embarrassed. So she did not do that, but said she'd like to go out with me. So Saturday when I dumped Michelle I already had the other one lined up, so I did not really care about Michelle any more. So now life has moved on and this weekend I will go out with her for the first time. And this week I felt really happy. This girl is really special. We have so much in common, she is pretty. . .

T: Can I just slow things down a bit to try and catch up?

P: Yes it is a bit fast isn't it? I always do that, I always have one in reserve. The minute I see trouble coming I get one in reserve.

T: It seems to me all of this action about phoning her so many times and getting another girl in reserve are all ways in which you try and manage terribly anxious feelings inside you.

P: Yes but now I don't feel it because the new girl answers her phone all the time, just like Jenny did, so it helps me.

T: So when Michelle did not answer her phone, what did you feel?

P: I felt anxious that she was seeing another guy and then I phoned again and again.

T: If I thought someone I like is seeing someone else, it would make me feel angry.

P: Yes, I felt like I could smash my phone up. I wanted to break her door down.

T: So part of phoning her so many times was also an angry thing?

P: Yes I suppose it is a bit smothering, maybe that is why she said I was moaning. But any guy will be upset if he is ignored. . .

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Transference-Focused Psychotherapy for Personality Disorders in Adolescence

22

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This chapter will focus on the adaptation of the adult transference-focused psychotherapy to personality disorders in adolescents (TFP-A). The model of personality disorders and their treatment is based on contemporary psychoanalytic object relations theory as developed by Kernberg (1984, 1992) and supported by findings from current evidence-based and neurobiological research (Clarkin, Levy, Lenzenweger, & Kernberg, 2004; Clarkin & Posner, 2005; Doering et al., 2010; Levy et al., 2006). In the first section we will examine the challenges of adolescents for the consolidation of personality and identity. This will be done using a perspective that integrates neurobiology with research and theory of affect, affect regulation and aggression, as well as sexuality. We will then present a contemporary object relations theory for understanding the development of personality disorders. This is followed with a section on "Assessment" and finally we will present our approach to the treatment with main tactics, strategies, and techniques.

Introduction

The prevalence of borderline personality disorder (BPD) in adolescents seems to be at least as high, if not higher than in adulthood (Chabrol, Montovany, Chouicha, Callahan, & Mullet, 2001; Chabrol et al., 2004; Cohen, Crawford, Johnson, & Kasen, 2005; Johnson, Bromley, Bornstein, & Sneed, 2006; Lewinsohn, Rohde, Seeley, & Klein, 1997) due in part to the increased independence, exploration of new environments, and social responsibility at a time when neurobiological systems involved in constraint and planning to regulate impulsiveness are relatively immature. In spite of the high prevalence of BPD in adolescents, until recently child and adolescent clinicians have been cautioned not to apply the adult criteria to children and adolescents. This was partly due to concerns that behaviors that might be normative in children and adolescents might be misdiagnosed as signs of BPD. It was also because of important nosological concerns regarding whether the BPD diagnosis in childhood was in fact a precursor of adult BPD specifically, or a precursor of adult psychiatric disorder more generally (Kestenbaum, 2012). Due to the advocacy work of pioneers like Paulina Kernberg (Kernberg, Hajal, & Normandin, 1998; Kernberg, Weiner, & Bardenstein, 2000; Kernberg & Wiener, 2004), we know that a constellation of BPD type symptoms can be observed in childhood and are unlikely to resolve without intervention specifically targeting personality issues. Much further work is needed to shed light on BPD symptoms in children, to differentiate children at risk for developing BPD and other serious psychiatric disorders in adulthood, in order for them to receive appropriate treatment

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and medication especially during adolescence (Pettit, 1997), although work in this regard, as exemplified in the current volume, is underway.

Recent practice guidelines regarding diagnosis and treatment of children and adolescents with BPD recommend that the diagnosis using the adult criteria can be made reliably from age 13 (NICE Clinical Guidelines, 2009; Noorloos & Huijgen, 2011). Few clinicians who work with adolescents currently use the BPD diagnosis, although the majority agree that BPD can be diagnosed in adolescence (Laurenssen, Hutsebaut, Feenstra, Van Busschbach, & Luyten, 2013). Today there is increasing recognition of the positive implications of early diagnosis as a way of recognizing the suffering of these young patients and their families, helping them to understand the problem and access appropriate treatment as early as possible. Moreover, a leading research group, Chanen, Jovev, McCutcheon, Jackson, and McGorry (2008), promotes immediate action for first presentation of BPD traits or disorder, similar to early intervention for first-episode psychosis.

The development of BPD is a lengthy multidetermined process with its roots in early childhood (Carlson, Egeland, & Sroufe, 2009; Lenzenweger & Cicchetti, 2005). There is a genetic contribution (Distel, Hottenga, Trull, & Boomsma, 2008; see also Chap. 11), but what is inherited is biological vulnerabilities involving a combination of genes (Skodol et al., 2002). In early infancy and childhood temperamental traits like negative affectivity, stress reactivity, and impulsivity (Goodman, New, & Siever, 2004; Posner et al., 2003) are linked to emotional vulnerability and an increase in risk of developing personality disorders (PDs), in interaction with environmental factors that influence gene expression. Both genetic and environmental factors are thought to have the most profound impact during the early postnatal period, a time when the forebrain is undergoing rapid growth (Depue, 2009). Themes of neglect and abuse associated with family dysfunction and parental psychopathology are common, especially when family interactions are invalidating, conflictual, negative, critical (Fruzzetti, Shenk, & Hoffman, 2005), and nonempathic (Guttman & Laporte, 2000). Temperamental traits of extreme emotional intensity (i.e., the tendency to extreme reactions) and reactivity (i.e., high sensitivity to emotional stimuli) may also be an important, but as yet relatively less researched, vulnerability factor, and may help to understand the development BPD symptoms such as self-harm in the apparent absence of the usual risk factors. Findings from a rare longitudinal study by Carlson et al. (2009) on the development of BPD from infancy to early adulthood confirm that temperament and early histories of attachment disorganization, parental hostility and abuse, and family life stress are important risk factors. These factors predicted disturbances in many domains of functioning in middle childhood and early adolescence including attentional, emotional, and behavioral regulation as well as relationships and self-representation. Moreover the study findings underscore the important role of *self-representation* in early adolescence, showing that it mediated the relationship between attachment disorganization and BPD symptoms in early adulthood. We consider that disturbances in the organization of the self are central to the development of BPD and these disturbances will manifest in the core dimensions of the self involving negative affect, self-regulation, motivation, and reward, as well as control systems involving attention and reflectiveness, interpersonal interaction, and affiliation.

Disturbances in Organization of the Self

Our view is consistent with a developmental psychopathology perspective that emphasizes the organization of experience and patterns of adaptation, with borderline personality dysfunction reflecting "a disturbance in core dimensions of self-competence that interact and transact to

form the foundation of subsequent functioning" (Carlson et al., 2009). In this perspective the self is defined as an organization of attitudes, feelings, expectations and meanings, and manifestations in attention, behavior, and relationships. Positive adaptation is facilitated by the integration of biological, emotional, cognitive, and representational capacities that enable the child and adolescent to respond flexibly to current and future developmental challenges. Maladaptation or psychopathology reflects rigid patterns of responding that compromise development. Development is seen as a series of qualitative reorganizations within the framework set by early experience. In the context of interactions with responsive caregivers, children develop adaptive and flexible patterns of attentional and emotional regulation, and develop positive representations of self and others, and individual and interpersonal skills that help them maintain their self-organization and facilitate the maintenance of close supportive relationships with others. This provides the framework for further integration of developing cognitive and social capacities in interaction with the social world that is reinforced and structured by the parents.

Disorganized attachment and overwhelming emotional experience, such as trauma, disrupt the normal processes of organization and integration of self. The absence or breakdown of early dyadic regulation systems for establishing an affect regulation pattern that forms the basis of self-regulation is considered to be an important source of later adaptational vulnerabilities and long-standing difficulties in self-regulation (Liotti, 1999). Malevolent caregiving or caregiving characterized by contradictory cues or the frightened or frightening maternal affects described by Lyons-Ruth (2003) are considered to be disorganizing as they evoke intense emotional reactions and conflicting needs that overwhelm the immature regulatory capacities, and result in a collapse in regulatory strategies (Hesse & Main, 2000).

To summarize, children who have experienced overwhelming affect in the absence of attachment relationships, where their needs for security are responded to and where they are helped to regulate and develop the basis for self-regulatory skills, are likely to develop long-standing difficulties in emotion and self-regulation. Unless they are in relationships where emotions are acknowledged and discussed they are unlikely to develop the capacity to know their strengths and weaknesses and develop balanced representations of others. These representational capacities are considered important for processing and top down regulation when emotions are evoked. Poorly developed emotional understanding and communication skills also place them at a further disadvantage when confronted by traumatic experiences, as they do not have the capacities needed to identify their feelings, and discuss their experiences and reactions.

Challenges of Adolescence for Personality

Adolescence is widely considered to be a critical period when key developmental tasks of attachment, self-development in strivings for autonomy, and emotional and behavioral self-regulation are reworked in the context of intimate relationships, identity formation, and management of risky behavior (Macfie, 2009; Sroufe, Egeland, Carlson, & Collins, 2005; Sroufe & Rutter, 1984). Despite the common portrayal of adolescence as a period of crisis with the adolescent weathering a storm of overwhelming affects and impulses, the majority of adolescents manage to engage with the challenges and opportunities of this period without any marked difficulties. Longitudinal research from the perspective of self-esteem, for example, shows that as many as 87 % of adolescents show consistently high and increasing self-esteem from early adolescence to early adulthood (Birkeland, Melkevik, Holsen, & Wold, 2012). They have an overall sense of continuity where they are able to integrate new roles and experiences on the way and arrive at the end of adolescence with a coherent sense of identity reflecting their own values and ideals. Most adolescents are able to resolve the inevitable confusion as their social and life experiences widen and confront them with new

ideas and opportunities that are challenging to integrate this into their existing identity. While their identity might become more nuanced in so far as they separate from their parents, develop social networks, and enter into their first romantic relationships, there is a strong sense of continuity of their personalities and the sense that they build on their childhood selves, rather than reinvent themselves, and that even as they become more independent and develop views and values that are more differentiated from their parents, they maintain their relationships with parents and family members. In addition, even where they develop views and values which may differ and in some cases be in conflict with that of their parents, they maintain a certain capacity to see past these differences and appreciate positive aspects of the parents and are able to turn to their parents for help in times of need.

In contrast, and based on clinical observation, the vast majority of adolescents who are diagnosed with BPD have childhood histories of long-standing and marked difficulties in affect and behavior regulation. Adolescents with relational disturbances, involving inappropriate aggression directed toward others such as oppositional deviant disorder (ODD) and conduct disorder (CD) during childhood, frequently meet criteria for BPD during adolescence. Some other adolescents with BPD may not have the same overt difficulties in behavior or affect regulation and aggression of ODD or CD, but may have a stable pattern of inflexible and maladaptive reactions. These maladaptive qualities might be difficult to observe in situations that are structured, non-challenging, or predictable; they are more likely to appear in periods of change and stress. For example, how the child handles the transitions between junior and high school, activities that make greater interpersonal demands such as making new friends and establishing a level of intimacy in a relationship or situations involving challenges, competition and the risk of failure and humiliation such as taking tests, team sports, or performing publicly at school, or that make new demands for autonomy such as a sleepover, and finding a job and performing a job in the absence of supervision. Consistent maladaptive reactions to these situations may be indicative of disturbances in characteristic defenses and coping mechanisms and these underlying difficulties will become more and more evident at each developmental period so that there may be definite but less flamboyant evolution toward a PD when faced with the inevitable challenges of adolescence to separate, become more independent, and establish social and intimate relationships outside of the security of the family.

Included in this group are adolescents who present with BPD who have childhood histories that seem at first glance unremarkable and without any apparent psychiatric difficulties, and who seemed at most to have been somewhat sensitive, dependent, submissive, and obsessional as children. Adolescents who develop eating disorders or engage in self-harm frequently have such apparently unremarkable childhood histories. We can conclude that these adolescents, like those with ODD and CD, have long-standing difficulties that have become over time entrenched into their personalities and increasingly evident in the context of demands to become autonomous, take on increasing responsibilities, and make decisions while at the same time separating from parents and developing new intimate relationships.

We consider that there is also another small group of adolescents without childhood psychological problems or personality difficulties who may develop identity crisis that is difficult to distinguish from BPD, at adolescence when they are confronted with overwhelming challenges and discontinuities that surpass their capacity to absorb and integrate this in a way that preserves a sense of continuity and coherence. In the longitudinal trajectory study of self-esteem by Birkeland et al. (2012) this group, representing approximately 7 % of adolescents, presents with an u-shaped trajectory where their initially good self-esteem decreases markedly between ages 14 and 18, reaching its lowest level at late adolescence, before improving during the next 5 years. This group of adolescents may either have pre-existing fragilities that compromise their capacities to adapt to change or their difficulties

during adolescence may lead to some kind of scarring, as their global self-esteem at age 30 is significantly lower than that of individuals with consistently high self-esteem during adolescence, and they present with significantly higher levels of depression. This would suggest that adolescents who experience identity crisis or sharp decreases in self-esteem may also warrant therapeutic interventions and potentially derive significant long-term benefits from therapy. Sexual abuse, including sexual abuse just before or at the beginning of adolescence, may be particularly destabilizing and this may be the final straw that breaks the camel's back for girls who may have shown resilient personality characteristics and who were able to continue function well at school and invest in friendships in the context of parental neglect, substance abuse, psychological problems, and immaturity. In addition to the range of PTSD symptoms that may be expected to resolve in due course, sexual abuse may interfere with the capacity of adolescent girls to form intimate relationships and to develop trust in partners.

Adolescents who engage in increased risk taking, especially where drugs, alcohol, and sex are involved, may also be more at higher risk of presenting with identity crisis and lowered self-esteem when they develop addictions or experience trauma or become overwhelmed when their behaviors take them down paths they are unprepared for. Another group that may be particularly at risk are those adolescents who are confronted with the task of assuming a sexual identity that is not culturally approved and confronts the adolescent with the possibility of being alienated from peers and family. In addition, for hypersensitive adolescents, parental separation, especially when accompanied by conflict and geographical moves to far away cities that make parents more difficult to access and which is associated with a loss of friends and challenges them to integrate into a new social circle and adapt to a new academic environments, can provoke breakdowns and identity diffusion which present like BPDs and is associated with the onset of suicidal and self-harming behavior. It is possible that parental mental illness, chaotic family environments where parents respond with inappropriate physical aggression to adolescent self-assertion, and bids for separation can also lead to breakdowns that result in identity diffusion in sensitive or vulnerable adolescents.

Neurobiological Developments at Adolescence

From a neurobiological perspective, adolescence is considered to be one of the most optimal but at the same time vulnerable periods for the development of cognition, especially of higher order thinking, reasoning, problem solving, and risk taking (Reyna, Chapman, Dougherty, & Confrey, 2012; Steinberg, 2008). Over the period of adolescence and extending into early adulthood, dramatic brain changes take place in the frontal lobe regions that subserve reasoning, problem solving, decision making, and higher order reasoning. If these abilities are developing so rapidly during adolescence, why do adolescents seem more emotionally reactive and vulnerable to making bad decisions when against their better judgment and engage in risky behaviors, especially when under the influence of emotions and peers? Casey, Jones, and Somerville (2011) have proposed an imbalance model of adolescent brain development. They point out that during adolescence, the limbic system is functionally mature at a time when the prefrontal systems are still developing, so that adolescents are much more vulnerable to the influence of the reward-sensitive limbic. This is consistent with the widely used dual processing model which holds that people often use reflexive or automatic, intuitive affect-driven heuristic processes although they are capable of more reflective, controlled rational processes (Evans, Venn, & Feeney, 2002; Reyna, 2004). In neurobiological terms the reflexive processes are mediated by the affect-related subcortical systems while the reflective component is subserved by the prefrontal cortex (Galvan et al., 2012; Galvan, 2013). This model is supported by the increase in evidence that dual systems involving both cognitive and affective processes are involved in our evaluations of situations so that decisions result

from an interaction between more thoughtful processes and more experience-based, affective, heuristic, and motivational processes (Damasio, 1994; Epstein, 1994; Evans, 2008; Lerner & Keltner, 2000; Lieberman, 2000; Loewenstein, Weber, Hsee, & Welch, 2001; Schneider & Caffray, 2012; Stanovich & West, 2000).

From the dual systems perspective BPD pathology, including dysregulated negative affect, impulsive and aggressive behavior, and interpersonal difficulties, can be seen as related to deficits in the reflective and executive control processes, coupled with a biased reflexive process where there is an automatic hypersensitivity to negative social cues (Koenigsberg et al., 2009), an expectation of untrustworthiness (King-Casas et al., 2008), and increased negative affect (Sadikaj, Russell, Moskowitz, & Paris, 2010).

Affects, Affect Regulation, and Aggression

High levels of negative affect and difficulties in affect regulation are considered to be problems commonly experienced by individuals with BPD (Lenzenweger, Clarkin, Fertuck, & Kernberg, 2004). This is even more problematic during adolescence when the intensity of emotional states and visceral urges are amplified. In contrast to treatment models focusing more on cognition and cognitive interventions, we give a central place to affects, with a special emphasis on working with negative affects such as aggression and fear. Affect is considered to play a vital role in guiding behavior (Schneider & Caffray, 2012) with affect-laden intuitions acting as a fast and frugal heuristic (Gigerenzer, 2000) for quickly and automatically judging whether stimuli in the environment are positive or negative (Slovic, Finucane, Peters, & MacGregor, 2002, 2004). In patients with BPD reflexive social cognitive processing is distorted by negative affect especially in contexts that evoke threat. Given that attention and processing resources are likely to prioritize potential threats, this might leave fewer resources for attentional and reflexive processes that can help with top down regulation.

Intense negative affect is also central in our conceptualization because we consider that it is at the heart of the mechanism of projection, which can be seen in cognitive terms as a process where the person does not recognize his/her own affects such as aggression, and is convinced that it comes from the other. Our treatment is specifically designed to stabilize extreme affects by systematically getting the patient to become aware of the split and polarized, positive and negative affective representations in peak affect states and under the impact of projection, and then to slowly integrate these representations. This reduces the extreme distortions in representation and cognitive processing maintaining and escalating the states of intense negative affect and making the resources of the cognitive system available to develop more accurate integrated representations of self and others.

Sexuality in Adolescence

The integration of sexuality into identity is one of the important challenges of adolescents. The physical and hormonal changes associated with puberty and sexual maturation challenge adolescents to integrate being sexually active into their previous prepubertal identity where sexuality was prohibited by parents and society. Sexual interest, first sexual experiences, falling in love and developing an intimate sexual and emotional relationship, and negotiating dependency needs in a close relationship with a partner are important developmental steps that have to be negotiated during adolescence. Adolescents with BPD and histories of physical, sexual, and emotional abuse within the family, especially where this is superimposed on preexisting difficulties in responding to their attachment needs, can be expected to find it difficult to develop sexual and emotional intimacy. They are known to have earlier onset of first intercourse, a higher risk of sexual victimization, and date rape

(Sansone, Barnes, Muennich, & Wiederman, 2008), but may also be at increased risk for perpetrating abuse (Zanarini, Frankenburg, Reich, Hennen, & Silk, 2005). In addition BPD is associated with greater risk taking in the sexual arena involving promiscuity and impulsively entering into sexual relationships. Others may experience sexual inhibition, avoidance (Zanarini et al., 2003), and fears related to physical and emotional intimacy that becomes a serious obstacle to establishing intimate relationships. In addition to personality, trauma-related factors are considered to be important for understanding these reactions (Trippany, Helm, & Simpson, 2006). Furthermore there is evidence that the onset of sexual relationships coincides with the onset of BPD symptoms in a third of BPD patients (Zanarini et al., 2003). Given the high probability of difficulties relating to risk taking, victimization, and intimacy in the area of sexuality, and the fact that these difficulties may emerge or be particularly pertinent during adolescence, we consider that clinicians need to be alert and open to exploring and thinking about difficulties in this area.

Adolescence is also a time when sexual identity or sexual orientation is defined. We consider that the question of sexual identity needs to be considered carefully when adolescents present with concerns in this area. While adolescents, like adults, with BPD may frequently switch between having sex with one gender to the other (Reich & Zanarini, 2008) it is important to identify those adolescents who are actually struggling with important difficulties around assuming their sexual identity, and treat them appropriately. Adolescents who become aware that they are not heterosexual may present with features that could potentially be confused with identity diffusion as they experiment and explore different sexual orientations and roles. The assumption of a homosexual identity is more challenging and takes longer, and is presumably only achieved by early adulthood, usually between the ages of 22 and 24 (Isay, 1986). Parental rejection and loss of the family as a support system may increase conflicts and anxieties about separation and becoming independent. Adolescents who experience acute rejection and ejection from the family home are at risk for running away, are more vulnerable to engaging in prostitution and substance abuse (Sugar, 1997), at higher risk for attempting suicide especially when confronted with intimidation (Renaud, Berlim, Begolli, McGirr, & Turecki, 2010), and more likely to consult mental health services.

Identity, Identity Diffusion, and Identity Crisis

Identity is one of the central concepts in the area of personality development and is central in our conceptualization of personality disorders. It is the subjective part of personality, and self-understanding, self-concept, self-as-subject, and self–other differentiation are all terms that have been used at times, interchangeably with identity. While these constructs share many features, they are not necessarily synonymous. As Erikson (1968) stated earlier, identity consists of a sense of inner sameness, continuity within oneself and in our interactions with others over time, being distinct with others, and a sense of inner agency. It reflects the awareness of one's individuality and one's allegiance to the ideology and culture of his group. It implies a sense of purpose, intentionality, and mastery.

In Akhtar and Samuel's review of the concept (1996), identity is constituted of a realistic body image, an awareness of a core gender identity (male or female), gender roles (feminity or masculinity), and sexual orientation (heterosexual or homosexual), a subjective self-sameness across situations and smooth transitions between various self-representations emerging under diverse social circumstances, a temporal continuity, a true capacity to recognize the positive and negative traits in oneself and in others that conveys a sense of authenticity, an ethnic identity constituted of cultural values, verbal and nonverbal modes of expression, and patterns of interpersonal behaviors, and finally a conscience that reflects the capacity to respond to rewards and punishments, to experience remorse and guilt, and to work for ideals. By the same token, an integrated identity also involves an integrated

realistic view of significant others that tolerates the complex integration of positive and negative features of their personality, and the capacity to maintain such a view even under conditions of temporary conflict or mood-inflicted negative affective interactions with them.

In our opinion, school-aged children have already attained a good level of integration of these components. Indeed, identity is a lifelong process that has its roots in children's earliest interactions with the environment. Children's identification and introjects are precursors of the process of identity formation in adolescence. In fact, identity formation starts where the usefulness of identification ends. During adolescence, these components of identity are remodeled under the psychobiological changes. The adolescent feels himself in the grip of overwhelming instinctual impulses that he must rapidly learn to master. As stated by Jacobson (1964), "adolescence is life between a saddening farewell to childhood—i.e., to the self and the objects of the past—and a gradual, anxious-hopeful passing over many barriers through the gates which permit entrance to the as yet unknown country of adulthood" (p. 161). The adolescent must free himself from his attachments to persons who were all important during childhood; he must also renounce his former pleasures and pursuits more rapidly than at any former developmental stage. Preparing himself to leave home, he must reach out for adult sex, love, and responsibility, for personal and social relations of a new and different type, for new interests and sublimations, and for new values, standards, and goals as an adult. This necessitates a complete reorientation, leading to structural and energetic transformations, redistributions of emotional investment, and to a drastic overhauling of the entire psychic organization. This remodeling of the adolescent's identifications, values, and ideals and its interrelationship with the development of his new identity, feelings, and object relations find an echo in the states of his shifting mood and emotional turmoil.

An important diagnostic problem with adolescent is to differentiate between the syndrome of identity diffusion that underlies personality disorders and the identity confusion (or crisis) that can be accounted for by normal development in adolescence (Erikson, 1968).

Identity crisis (*confusion*) refers to the frequent, time-limited dissociation between the perception of an adolescent of himself/herself, on the one hand, and the perception of that adolescent by the family and the social environment in general, not fully grasping the profound internal transformations occurring as part of puberty and adolescence. The lack of confirmation of the perception of self in the interactions with significant others may induce a sense of alienation and confusion in adolescents who, however, present a well-integrated view of their present self and an integrated view of significant others. An adolescent's conflicts around regressive dependency and rebellious assertion of autonomy may convey a picture of emotional instability and interpersonal conflicts that, however, may not correspond to a syndrome of identity diffusion.

Identity diffusion. In the case of identity diffusion, there is a general lack of integration of the concept of the self reflected in contradictory self-experiences that cannot be reconciled, serious distortions in the views of significant others, the typical development of sharply split, idealized, and persecutory object relations, and extreme oscillations of self-esteem. Moreover, the syndrome of identity diffusion typically is reflected in serious discontinuities in the self-concept and unrealistic evaluation and affective distortions in the relations with significant others, significant failure in the social life at school, in school performance, in the relations at home, serious dissociation between sexual behavior and emotional intimacy, and the possible development of antisocial behavior.

Integrated (*consolidated*) *identity*. An adolescent's capacity to describe an internal state of turmoil from the perspective of an implicit-integrated view of self, and an integrated view of the most important members of his/her family in spite of turmoil and conflicts in their relations, the presence of a well-integrated system of moral and ethical values, the commitment to ideals beyond self-serving objectives, the capacity for friendships in depth with peers, good functioning

at school, and evidence for the capacity of romantic love, all point to the presence of an integrated identity. He/she has attained a level of self-reflection (or mentalization) that permits this self-examination.

Fig. 22.1 Object relation dyad: self–other representations linked by affects

Definitions and Core Constructs Using a Psychodynamic Perspective

In essence we consider that patients with BPD suffer from identity diffusion where their representations of self and significant others are polarized and unstable and may oscillate rapidly from idealized to persecutory. We consider that affect, and particularly negative affect, and the predominance of aggressive internalized object relations over idealized ones (Kernberg, 2006) are central to this failure of psychological integration and interfere with the use of mentalization and impede the development of integrated representations of self and others. This is because, in an effort to protect the idealized segment of self and other representations, these patients use dissociation and splitting mechanisms as well as other primitive defenses such as projection, projective identification, omnipotence and omnipotent control, devaluation, denial, and primitive idealization. Identity diffusion manifests clinically in the incapacity to have a reasonably accurate and nuanced assessment of self and others, a lack of understanding, empathy, and the normal tact in interpersonal situations, and incapacity to maintain intimate relationships and commit in depth to work.

Contemporary Object Relations Theory

A fundamental premise of this psychodynamic conceptualization and treatment of adolescents with personality disorders is that the observable behaviors and subjective disturbances reflect pathological features of underlying psychological structures. A psychological structure is a stable and enduring pattern of mental functions that organize the individual's behavior, perceptions, and subjective experience. A central characteristic of the psychological organization of adolescents with severe personality disorders is the lack of integration of the psychological structures of an integrated conceptualization of self and of significant others, that is the syndrome identity diffusion.

In object relations theory it is emphasized that the drives described by Freud—libido and aggression—are always experienced in relation to a specific other: an object. Internal object relations are the building blocks of psychological structure and serve as the organizers of motivation and behavior. These basic building blocks of psychic structure are units made up of a representation of the self, an affect related to or representing a drive, and a representation of the other (the object of the drive). These *units of self*, *other*, and the *affect* linking them are referred to as *object relations dyads* (Fig. 22.1). It is important to note that the "self" and the "object" in the dyad are not accurate internal representations of the entirety of the self or the other, but rather are representations of the self and other as they were experienced in specific affectively charged moments in time in the course of early development in primary attachment relationships, and defensively distorted in the course of intrapsychic development.

The individual with a normal personality organization has first an integrated concept of self and of significant others which is captured in the concept of identity. This concept includes both a coherent internal sense of self and a pattern of behavior that reflects self-coherence. Such a coherence of self is basic to self-esteem, enjoyment, and the capacity to derive pleasure from relationships with others and from commitment to work, school, or other responsibilities. A coherent and integrated sense of self contributes to the realization of one's capabilities, desires,

and long-range goals. Likewise, a coherent and integrated conception of others contributes to a realistic evaluation of others involving empathy and social tact. An integrated sense of self and significant others permits the development of intimacy and stability in love relationships and the harmonious integration of tenderness and eroticism in such relationships.

In essence, our basic assumption in the application of contemporary object relations theory is that all internalizations of relationships with significant others, from the beginning of life, have different characteristics under the conditions of peak affect interactions and low affect interactions. Under conditions of peak affect activation—be they of an extremely positive, pleasurable or an extremely negative, painful mode—specific internalizations take place framed by the dyadic nature of the interaction between the baby and the care-taking person, leading to the setting up of specific affective memory structures with powerful motivational implications. Object relations theory assumes that these positive and negative affective memories are built up separately in the early internalization of these experiences and, later on, are actively split or dissociated from each other in an effort to maintain an ideal domain of experience of the relation between self and others, and to escape from the frightening experiences of negative affect states. Negative affect states tend to be projected, to evolve into the fear of "bad" external objects, whereas positive affect states evolve into the memory of a relationship with "ideal" objects. This development evolves into two major, mutually split domains of early psychic experience, an idealized and a persecutory or paranoid one, idealized in the sense of a segment of purely positive representations of self and other, and persecutory in the sense of a segment of purely negative representations of other and threatened representation of self. This early split experience protects the idealized experiences from "contamination" with bad ones, until a higher degree of tolerance of pain and disappointment, and more realistic assessment of external reality under painful conditions evolves.

This early stage of development of psychic representations of self and other, with primary motivational implications—move toward pleasure and away from pain—eventually evolves toward the integration of these two segments, an integration facilitated by the development of cognitive capacities and ongoing learning regarding realistic aspects of self and others interacting under circumstances of low affect activation. The normal predominance of the idealized experiences leads to a tolerance of integrating the paranoid ones, while neutralizing them in the process. In simple terms, the child recognizes that he/she has both "good" and "bad" aspects, and so does mother and the significant others of the immediate family circle, while the good aspects predominate sufficiently to tolerate an integrated view of self and others.

This state of development, referred to by Kleinian authors (Klein, 1940; Segal, 1964) as the shift from the paranoid-schizoid to the depressive position, and by ego psychological authors as the shift into object constancy, presumably takes place somewhere between the end of the first year of life and the end of the third year of life. Here, Mahler's (1972a, 1972b) research on separation-individuation is relevant, pointing to the gradual nature of this integration over the first 3 years of life.

Peter Fonagy's (Fonagy & Target, 2003; Bateman & Fonagy, 2004, 2006) referral to the findings regarding mother's capacity to "mark" the infant's affect that she congruently reflects to the infant points to a related process: mother's contingent (accurate) mirroring the infant's affect, while marked (differentiated) signaling that she does not share it while still empathizing with it, contributes to the infant's assimilating his/her own affect while marking the boundary between self and other. Under normal conditions, then, an integrated sense of self ("good and bad"), surrounded by integrated representations of significant others ("good and bad") that are also differentiated among one another in terms of

their gender characteristics as well as their status/role characteristics, jointly determines normal identity.

One central consequence of identity diffusion is the incapacity, under the influence of a peak affective state, to assess that affective state from the perspective of an integrated sense of self. The particular mental state may be fully experienced in consciousness, but cannot be put into the context of one's total self-experience: this implies a serious loss of the normal capacity for self-reflection, that is, for mentalization: under conditions of a peak affect state, a balanced and integrated representation of self and other is not possible.

This state of affairs has an important implication for the technique of TFP: the interpretation of splitting and other derived primitive defensive operations that bridge the emotional barrier between contradictory but conscious mental states fosters mentalization by integrating the mutual split representations of self and others. The development of an integrated representation of self facilitates the self-reflective function regarding the particular peak mental state under consideration. In short, interpretation of primitive defense mechanism fosters mentalization.

The major proposed hypothesis regarding the etiological factors determining severe personality disorders or borderline personality organization is that, starting from a temperamental predisposition with the predominance of negative affect and impulsivity or lack of effortful control, the development of disorganized attachment, exposure to physical or sexual trauma, abandonment, or chronic family chaos predispose the individual to the abnormal fixation at the early stage of development that predates the integration of normal identity: a general split persists between idealized and persecutory internalized experiences under the dominance of corresponding negative and positive peak affect states. Clinically, this state of affairs is represented by the syndrome of identity diffusion, with its lack of integration of the concept of the self and the lack of integration of the concepts of significant others.

Assessment

A careful diagnostic assessment is an essential precondition for the indication of the TFP-A treatment, and that assessment must include (1) the assessment of the adolescent symptomatology and behaviors; (2) the exploration of the parents' current maintenance of, as well as conflicts with, the adolescent's behaviors and symptoms; (3) a thorough developmental history to identify the roots of the disorders; and (4) the assessment of the adolescent's level of personality integration as reflected in the moment-to-moment interactions with the interviewer, his functioning at school or work, and through his peers and other social relations. The main goal is to get an as complete as possible picture of the adolescent level of functioning and level of personality organization and to distinguish his perturbations between normal identity confusion commonly encountered at adolescence and identity diffusion that is associated to personality disorders and to which this treatment is focused.

Assessment of the Adolescent Symptomatology and Disturbed Behaviors

The diagnostic criteria for a personality disorder as identified in the DSM-IV (American Psychiatric Association, 2000) (and now the DSM 5) are used for the assessment of PD in adolescents with an additional criterion that the onset must be traced at least to the early school years to respect the basic definition of PD as enduring maladaptive patterns of thinking, feeling, and behavior that are relatively stable over time.

Parental Contribution or Maintenance of the Disorder

As Freud (1905) stated, the adolescent detachment from parental authority, even though painful, is one of the most significant and necessary psychic achievements of the human mind. The adolescent

has to loosen his bonds with his family to be able to gain the instinctual freedom, a sense of autonomy, and to be able to assume full responsibility for his actions, thoughts, and feelings. Parents' roles are twofold: both to foster connectedness with their adolescent, and to encourage realistic steps toward autonomy, exploration of choice, speaking out, and questioning parental values. However, parental attitudes may convey a resistance to the adolescent's effort to separate or, on the contrary, underestimate or neglect the importance to accompany the adolescent through this delicate and anxiety-driven process. These parental attitudes may contribute to the development or to the maintenance of the disorder or, in severe cases, engender pervasive disturbances over the adolescence period (see Jacobson, 1964, pp. 200–209).

The therapist has to look for parental attitudes that did not permit the child's normal individuation or foreclose the adolescent's normal separation. For example, contradictory emotional and educational parental attitudes, early experiences of severe disappointment and abandonment, having grown up in an atmosphere of emotional poverty because of parents' personality disorders, emotional instability, confusion, or incapacity to love, or inordinate or repressive attitudes toward the adolescent's sexual behavior.

Developmental History: Assessing High Risk for Development of PD

Most of the personality disorders in adolescence can be traced back into infancy and childhood periods either through temperamental dispositions or through environmental hazards. In the patient's history, the clinician looks for indications of high risk factors for the development of PD. We look in the child history for signs of temperamental predisposition to the predominance of negative affect and impulsivity or lack of effortful control. On the environmental side, we look for signs of development of disorganized attachment, exposure to physical or sexual trauma, abandonment, or chronic family chaos predisposing the adolescent to the abnormal fixation at the early stage of development that predates the integration of normal identity.

The Personality Assessment Interview

There are different ways to get a clinical impression of the adolescent's sense of his personality, self, or identity. His actual behaviors, verbal and nonverbal, in the interaction with the examiner are a valuable source of information. He can also be asked to describe himself and significant figures such as parents, teachers, or friends with questions such as the following: (1) Can you describe yourself? (2) How do you see yourself physically, in front of the mirror? (3) How do you feel about changes in your body? (4) Can you describe your parents? and (5) Can you describe your best friend?

The Personality Assessment Interview (PAI), developed by Selzer, Kernberg, Fibel, Cherbuliez, and Mortati (1987) and derived from the structural interview developed by Kernberg (1981), focused specifically on the moment-to-moment interaction between the interviewer and the adolescent. The underlying hypothesis of the interview is that the patient's experience of the interview taps into his fantasies and influences his style of interaction with the examiner. The 60-min interview is conceived to elicit the basic components of the personality and its governing principles of organization and adaptation. The PAI technique consists of systematically asking questions that involve self-representation, object representation, mentalization capacities, affects, and cognitions as the patient talks. The interviewer asks at the beginning of the session: (1) what have you been told about this interview or meeting with me? and subsequently in the session: (2) now that we have been together for 15 min or so, how does what happened compare with your initial impressions, and what do you expect the rest of the meeting will be like?; and (3) What have you learned about yourself, about me, and what do you imagine I have understood so far?

The PAI is well suited for adolescents because the interviewer does not inquire about their private life. Furthermore, it helps to differentiate between normal identity confusion and elements of identity diffusion by giving signs of a preserved sense of self under challenging interactions.

Finally, the PAI helps to assess the capacity of the adolescent to benefit from psychotherapy, and specially TFP-A, because it taps such functions as attention, memory, reality testing, mentalization, and the capacity to sustain a working alliance under high affective interactions.

At the end of the assessment sessions, the therapist offers his understanding of the problem, and gets a sense of the capacity of the adolescent to recognize that he has a problem and whether he is able to commit himself to coming independently to sessions. This working toward a recognition and acceptance that there is a problem is in many ways a precondition for a successful treatment, as the adolescent is unlikely to be motivated to make a commitment to the therapy. The adolescent is much more likely to recognize that he has a problem when the therapist reformulates his behavior as internally, unconsciously, or developmentally motivated, and something which he does not have control of, or does not want to have control of, at the moment. We recommend TFP-A when there is the combination of the adolescent's capacity to take some responsibility for his own problems and to see that he needs to work to solve them and of little evidence of an antisocial personality disorder proper and few secondary gains from the illness.

Treatment

TFP-A is a psychodynamic treatment for borderline adolescents delivered in individual sessions ideally twice a week but not less than once a week (TFP-A manual available on request). The major objectives of the TFP-A are gaining better behavioral control, increasing affect regulation, developing more intimate and gratifying relationships with family, peers, or close friends, and engaging in a productive life as well as investing in school and future goals. This can be achieved through the development of integrated representations of self and others, the modification of primitive defensive operations, the resolution of identity diffusion that participates in the fragmentation of the adolescent's internal world, and recognizing and facilitating every attempts made by the adolescent to face normal developmental challenges which are often confounded or hijacked by the pathology itself. TFP-A interventions are composed of a series of tactics, strategies, and techniques geared to achieve these goals.

Tactics: Contracting and Setting the Priorities

The treatment tactics are the tasks the therapist must attend to in every session to create the necessary environment for the therapeutic work. They include how to prevent and address complications that may arise in the course of the treatment and how to choose the priority theme to address.

Contracting

The first key tactical aspect of TFP-A is establishing a contract between the adolescent, his/her parents, and the therapist. The contracting phase serves two purposes: first, to create the conditions for the therapist's position of neutrality which is the necessary precondition for the analysis of the transference, and second, to establish a treatment frame that protects the adolescent and the treatment from dangerous acting out, unconstructive parental involvement and considers the particular reality of the adolescent in terms of relations to home, school, and street.

Neutrality and authority. Adopting a position of technical neutrality requires open and direct spelling out of the boundaries of the treatment situation and their rationale to the adolescent and his parents, and an active, patient, and persistent educational work with the parents around the fact that they continue to maintain full authority outside the treatment sessions. The therapist may provide recommendations regarding some problematic interactions at home, but assumes no executive authority in this regard. The authority of the therapist is limited by the spatial boundaries of his office; what happens outside may trigger his counsel or advice, but is not his responsibility. Setting up regular joint meetings with the adolescent and parents should permit

them to ventilate many of problems that emerge. For example, some parents may abandon their responsibilities, "dumping" the patient in the hands of the therapist; or else, parents may feel envious and resentful of the therapist's influence on their child, threatened by the therapist's potentially undermining their authority, or by the therapist's not sharing their particular ethical values and moral demands involving their child. In the case of female adolescents, parents may strictly forbid all sexual behavior—while the adolescent acts out her rebellion in unprotected sex, the therapist may have to help the parents find a more effective way to protect their child. Full discussion of all issues regarding authority in the joint meetings of adolescent, parents, and therapist should permit such a gradual clarification and assurance of the therapist's position of technical neutrality, and permit teasing out the adolescent's developing transference reactions.

Parent involvement and parental authority. Contract setting also requires agreements involving the parents and sometimes the school and even legal authorities, in addition to the direct contract setting with the adolescent. The fact that parents keep their legal authority until the adolescent's majority gives them the right to be informed on a regular basis of his progress and the evolution of the treatment. However, the therapist has to create a space protected from the parents' intrusion where the adolescent can have the experience of autonomy and individuation. The therapist needs to systematically address all the behaviors that can put this space in jeopardy, and help to identify strategies that will protect the treatment from unnecessary intervention from the parents. This is not an easy task and requires good clinical judgment from the part of the therapist because parents also may need to carry out significant controls of the adolescent's life outside the sessions involving school, social life, the street, and the adolescent's family and friends. Borderline adolescents tend to evoke powerful emotional reactions in their family that will influence the family's transference reactions to the therapist, and represent a heightened influence on the treatment by the adolescent's transference acting out involving his family, including their willingness to collaborate with the treatment in terms of facilitating the adolescent's coming to sessions, being responsible regarding financial arrangements, and following through with jointly agreed-upon structuring of the adolescent's life outside the sessions.

Parents may need help with adolescents' efforts at omnipotent control at home, with severe acting out at home or in relation to the school. Sometimes conflicts between the parents may be expressed in their lack of clarity regarding the support for the treatment, the responsibility for the adolescent's attendance, payment, and the responsibility for both of them to attend scheduled joint meetings with therapist and adolescent. Sometimes the suggestion may be made that parents enter into couple therapy. In the case of separated or divorced parents, all these difficulties may become even greater.

Confidentiality. Another important issue at the contracting stage is confidentiality, how adolescents' and family "secrets" are to be handled, and collateral sources of information. The general principle should be that all information regarding the adolescent and its sources should be shared with the adolescent himself. All the material from the sessions of therapist and adolescent is confidential, with the exception of issues that the therapist considers essential to protect the adolescent and the treatment. If information is to be shared outside the treatment, the adolescent would be informed first about that intention by the therapist to provide the possibility of a full discussion before the therapist proceeds. In those cases, it would be the responsibility of the therapist to indicate why such action seems to him essential to protect the adolescent's social standing, physical health, psychological well-being, or even survival. This, of course, is particularly relevant with adolescents who present chronic suicidal and parasuicidal behavior, where the responsibility of the therapist, the adolescent, and the family has to be clearly spelled out. On this latter difficult issue, we follow Yeomans, Clarkin, and Kernberg's (1992) suggestion for the treatment of adult BPD patients.

Setting the Priorities

A second crucial tactical aspect of TFP-A is setting the priorities and determining a sharp focus on what is going on in the sessions itself.

The priorities for interventions are similar to those of adults and derived from danger signals that override the general criteria for selection of material to be explored: First, threat to life, particularly suicidal intentions or behavior. Second, threat to treatment, represented by both refusal to come to sessions and indirect indications that patients and family are considering its disruption. Third, deceptiveness in the hours, indicating a predominance of "paranoid" or "psychopathic" transferences that need to be explored. In general, chronic deceptiveness takes a high priority for elaboration over an extended period of time in some cases, and regularly reveals underlying paranoid transferences: the adolescent fears that to "confess" certain issues or feelings would provoke criticism, rejection, retaliation, or punishment by the therapist, parents, or others. Fourth, severe acting out, either inside the sessions or outside, usually indicates affective dominance of the related material, and needs to be explored in the transference to being the material into the verbal realm. Fifth, trivialization: sometimes, the only thing the therapist can diagnose is that the content of the hours seems to be trivial; there is no particular affect activation nor affect "freezing," both transference manifestations and countertransference dispositions are relatively quiet, and the question may be raised with the adolescent: what are we talking about? What is the relevance of all this? Are we leaving out important issues? In other words, the "looping" technique (reflecting on the recent interaction) may be used to interpret the defensive functions of trivialization.

The selection of material to be explored depends on what is predominant in the adolescent's affect, and, if that is not clear, what is predominant in the transference, and, if that were not clear either, what is predominant in the countertransference. At the same time, the awareness of the problems in reality that are dominant in the adolescent's life permits the therapist to intervene regarding these issues even in sessions in which affect dominance does not appear clearly.

The frequent stimulation of the adolescent in terms of what his/her reactions are to what is going on in the session and to what has been discussed, the adolescent's thoughts about what has been happening in the session, all are ways to stimulate the adolescent to explore his/her self-experience and the experience of the therapist's interventions in the session. Following the therapist's interpretive efforts, his raising the question with the adolescent, what is his/her understanding of why the therapist has said what he said, and a repetition of certain subject matters in this "reflective loop" may apparently reduce the amount of material that can be taken up in each session, but, in fact, increases the possibility of helping the adolescent to become aware of his mental states and of the mental states of the therapist, the process of "mentalization." In the process, the therapist may evaluate the adolescent's self-representations and object representations, their projections, the intensity of affect activation, and the extent to which the adolescent's cognition is framing his affective experience and giving evidence of the adolescent's developing reflective capacity and empathy.

External reality. A major tactical focus in adolescent TFP-A is the attention to external reality: where the adolescent stands regarding his/her actual developmental tasks. How is he/she doing at school, at home, in social life, and regarding personal well-being? Borderline adolescents who are involved with drugs, alcohol, cutting behavior, and neglect of their self-care in daily life require careful monitoring of these behaviors, and ongoing evaluation of their implications in terms of transference acting out. Transference analysis and consideration of external reality have to remain closely linked. As mentioned before, the therapist's awareness of dominant problems in the adolescent's relation with reality helps bring in those issues at times when intense transference–countertransference turbulence seems to direct the focus of the

treatment almost exclusively onto what is going on in the sessions.

Sexuality and erotic life. A sensitive tactic and particularly important focus in TFP-A is the adolescent's erotic life, an area that is a conventional cultural taboo in terms of communications between adolescents and adults, and may emerge only in subtle indirect ways in erotic behavior in the session, in erotic countertransferences, and in a clear discrepancy between the adolescent's erotized behavior on the one hand, and complete absence of information regarding erotic experiences and behaviors in the adolescent's external life, on the other. The management of erotic transferences may present a difficulty in terms of showing up in countertransference reactions while the adolescent studiously avoids any reference to his/her erotic fantasy or behavior. Tactful pointing out of the erotic implications of the adolescent's behavior in the hour, and its contrast with no reference to erotic experiences outside the hours, as if an important aspect of life were missing in the adolescent's experience, may bring the subject into full exploration. Here, direct, open discussion of sexual issues in a non-erotized context, and without taking the side of "superego" determined criticism nor rebellious stimulation of "sexual freedom" permit opening up this important area of adolescents' life experience, including the difficulty talking about sexual inhibitions, polymorphous sexual behavior and fantasies, and confusion and anxieties over their sexual wishes or inhibitions.

Affect storms. The development of affect storms is a particularly frequent complication and tactical challenge in the treatment of adolescents. The adolescent should be free to express his/her affects in the hour as long as there is no physical attack on the therapist or the office, nor sexual behavior during the sessions, and the adolescent's voice volume is contained by the office door and arrangements. It is important for the therapist to respond in affective terms that correspond to the affect activation of the adolescent, without entering into yelling matches nor impulsive affective expression himself. In the case of opposite developments, with severe affective freezing and inhibition, the therapist has to be prepared to gradually interpret the transferential implications of that development as well. At times, severe affect freezing is a defense against the potential of a strong affect storm.

Acting out. A major tactical task, at times, is to systematically analyze acting out outside as well as within the sessions, in order to transform it into a cognitively framed emotional experience that can be shared and jointly explored in the sessions. By the same token, the transformation of severe somatization into an equally shared cognitively framed affective experience that can be explored in the transference is an application of the same principle. Splitting processes in adolescents often take the form of a dissociation between severe acting out, on the one hand, and completely "non-related" affective reactions, anxiety, and depression, on the other. For example, an adolescent may have presented serious failures in school, and gross neglect of work that threatens him/her with academic failure, on the one hand, with no apparent concern and worry about it, and, on the other, nightmares or unexplained anxiety without any apparent content. Here, the major task is to overcome the mutual splitting of acting out and its corresponding affects, often linked with other defensive operations such as denial of the potential destructive effects of acting out behavior, and the unconscious enactment of guilt over competitive, self-assertive, or sexual gratification.

Strategies: Toward Integration of Split-Off Internalized Object Relations

The treatment strategies have to do with the long-term objectives of the treatment and are geared toward the integration of the "split-off" internalized self and other representations. They are guidelines to stay focused on the main task of

working on the adolescent's internal world even though the therapy session or the external life is chaotic.

Activation of Split-Off Object Relations

The main strategy of TFP-A consists in the facilitation of the (re)activation in the treatment of split-off internalized object relations of contrasting persecutory and idealized nature that are observed and interpreted as they are experienced in the transference. As in the adult treatment, the adolescent is instructed to carry out free association. The therapist restricts his role to careful observation of the activation of regressive, split-off relations in the transference, to help the adolescent to identify them, and to interpret their segregation in the light of his enormous difficulty in reflecting on their own behavior and on the interactions they get involved in.

Identity integration. The strategies revolve around the central object of identity integration as it is facilitated by the interpretive process to be described below. The interpretation of split-off internal states is based upon the assumption that the activation of one particular internal dyad determines the adolescent's perception of the therapist at any given moment. Activation of a dyad can involve rapid role reversals of the self and object representations that comprise the dyad: the adolescent may identify with the role of victim while projecting a corresponding object representation onto the therapist, while, 10 min later, he might angrily threaten the therapist who then is in the role of the victim. Engaging the adolescent's observing ego in this phenomenon paves the way for interpreting the conflicts that keep his internal world fragmented. After the adolescent's identification with both poles of a given dyad is explored, the work can address conflicts between opposite dyads—those reflecting the persecutory segment of the internal world and those representing the ideal segment. These dyads, and the corresponding views of self and other, are separate and exaggerated. Until these representations are integrated into more nuanced and modulated ones, the adolescent will continue to perceive himself and others in exaggerated, distorted, and rapidly shifting terms. As we noted, the oscillation or alternative distribution of the roles of each dyad has to be differentiated from the split between opposite dyads carrying opposite affective charges. The final step of interpretation consists in linking of the dissociated positive and negative transferences, leading to integration and the resolution of identity diffusion.

The resolution of identity diffusion facilitates the modulation of intense affect dispositions as primitive euphoric or hypomanic affects are integrated with their corresponding fearful, persecutory, aggressive opposites. There is a significant integration of the adolescent's ego identity, as an integrated view of self—more complex, rich, and nuanced than the simplistic and extreme split-off self-representations—and a corresponding integrated view of significant others replace their split-off previous nature, and an experience of appropriate depressive affects, reflecting the capacity for acknowledging one's own aggression that had previously been projected or experienced as dysphoric affect, with concern, guilt, and the wish to repair good relationships damaged in fantasy or reality, becoming dominant. This step also brings about the mutual penetration and toning down of extreme, opposite affect states linked to all these representations. There is an increased capacity for affect control by the strengthening of their cognitive context as a consequence of the integration of self and object representations. In short, significant increase in cognitive framing of affective states improves mentalization—the capacity for realistic assessment of mental states of self and significant others, together with impulse control and enrichment of the overall subtlety and complexity of the assessment of social interactions. We will discuss how TFP-A then helps the adolescent use improved mentalization to resolve internal conflicts.

When severe identity diffusion and the corresponding splitting in transference developments are gradually overcome, the sessions may become more differentiated in their emotional implications, and acting out decreases. Severe turmoil in the sessions, while the external life of the adolescent normalizes, is a good indicator of progress in the strategic efforts of the therapist.

Techniques: Interpretation, Technical Neutrality, Transference, and Countertransference

The treatment techniques are the tools the therapist uses to address what is happening in the here and now in the service of accomplishing the overall strategy of integration.

The techniques of TFP-A are the same techniques described for adult TFP, with differences regarding the intensity, time dimension, and respective dominance of some of these technical approaches. They include interpretation, transference analysis, technical neutrality, and countertransference analysis.

Interpretation

The stages of interpretation include (1) clarification, that is, clarification of the communication of the adolescent, trying to reach the limits of the adolescent's self-awareness, before contributing with additional observations from the therapist; (2) confrontation, that is, tactful exploring of contradictions within the adolescent's communication, including his nonverbal behavior; and (3) interpretation per se, that is, formulation of a hypothesis regarding the unconscious implications of what has been clarified and confronted: first, in the unconscious meanings in the "here and now," and, only later, regarding the corresponding unconscious meanings in the "there and then."

Clarification. In TFP-A, clarification acquires a particular importance, and is an extended technical approach, practically in each session. The technique of exploring in "loops" what the adolescent thinks about what he has said, about the reaction of the therapist, where the adolescent stands now regarding what he has said, all this involves clarification in the sense of an exploration of the adolescent's conscious and preconscious awareness of his mental state. It is an essential technique leading to "mentalization," that is, a clearer understanding of the affective state of self and others as a motivational affective development. As mentioned before, clarification includes significant aspects of the adolescent's life outside the sessions, and the utilization of expressions of the adolescent that stem from sources such as diaries or literary productions, drawings, and the adolescent's detailed description of important friends and people in his environment. The adolescent's enthusiastic wishes to tell about experiences he has had outside the sessions are encouraged to lead to detailed narratives, within which the adolescent's reactions and reflections may then be explored.

Confrontation. Equally, confrontation becomes a very important technique, and, particularly in the early stages of treatment, the focus on the adolescent's behavior in the sessions, inviting the adolescent to explain whatever caused the attention of the therapist, and inviting him to reflect on his own attitudes as he is developing a narrative in the session, all are important roads to exploring transference dispositions. This do not, however, necessarily lead to immediate transference interpretation, but, rather, to the relationship of what evolves in the session with the adolescent's behaviors outside the hours, thus facilitating the analysis of transference dispositions expressed toward third parties before direct interpretation of the transference in relation to the therapist. In a somewhat different sense, confrontation in the sense of challenging may also become an important technique in the case of significant secondary gain that may have to be vigorously questioned and resolved in order for it not to become a major obstacle to the treatment progress. For example, the therapist questions the adolescent's failure to do his homework, smoking pot at school, and putting him at risk sexually, and then considers what can be done to avoid placing both the adolescent and his treatment at risk. Discussion often leads to the understanding that these behaviors are resistances to experiencing parts of the internal world. These situations are quite challenging because the therapist may have to abandon momentarily his therapeutic neutrality in order to protect treatment and adolescent.

Interpretation. As mentioned before, interpretation starts out very carefully with extra-transferential interpretations, and tentative

efforts to link the content of different sessions, establishing a continuity of contents that originally may have been presented split off from each other. The therapist starts with what is affectively dominant in each session based on the therapist's combined assessment of the adolescent's verbal communication, nonverbal communication, and the countertransference. In the early stages of the treatment, much of the information is carried by nonverbal behavior and the therapist's countertransference reactions.

The therapist also follows the principle of interpreting from surface to depth, from the defensive sides of the conflict to the impulsive side of it, a general principle of psychoanalytic technique that becomes particularly important in adolescents, given the risk of adolescents receiving any new information brought in by the therapist as an authoritarian "brainwashing." It is important to start out with observations shared by the adolescent and the therapist regarding the reality of a certain fact that the therapist then may develop in further depth. If, to begin with, no common element of thinking or appreciation may be found regarding an issue the therapist thinks is important, the interpretation may have to begin simply with the therapist sharing with the adolescent that he has a particular view about a certain issue but believes that the adolescent may have a different one, and is interested in sharing with the adolescent the fact that there are two potentially incompatible views of that issue. In general, analyzing the defensive function before the deeper, impulsive one of a certain conflict is facilitated by the fact that the defensive operations are closer to consciousness than the dissociated, projected, or repressed ones. The formulation of the defensive aspect, its motivation, and only then, what it is defending against can be facilitated by a tentative, open-ended style of communication of the therapist's thinking, always sharing it as something to be examined in the same way as a statement of the patient. All of what has been said makes interpretation a slowed down process, or rather, it assumes a lengthy preparatory process that only culminates with the hypothesis about an unconscious meaning once abundant evidence on the road to that interpretation has already emerged.

Technical Neutrality

Technical neutrality has been defined as intervening from a position that is equidistant to the sides of a patient's internal conflicts, as from the viewpoint of an "observing third party" (Clarkin, Lenzenweger, Yeomans, Levy, & Kernberg, 2007). Technical neutrality implies equidistance from impulses, prohibitions against impulses, the acting ego, and external reality, and an identification not only with the observing part of the adolescent's ego but also with general humanistic values that favor and support life, respect for the individual, physical health, and emotional well-being. Technical neutrality does not imply a cold, rejecting, or uninterested objectivity, but a warm, concerned, objective way of looking at the adolescent's internal conflicts. It is an essential position for the therapist in order to be able to analyze credibly transference developments. It doesn't imply a lack of countertransference reactions—even intense ones—as long as the therapist's interventions occur at a point where he has regained his internal objectivity.

Technical neutrality may have to be abandoned temporarily when the adolescent's well-being, the treatment, or others are threatened. In that case, the therapist may have to intervene with limit setting, and has to be prepared to follow up, over a period of time, with exploring fully the reasons for which he had to abandon technical neutrality, the significance of the conflicts that were activated in this context, all of it leading to the gradual analytic interpretive reinstatement of technical neutrality. Structuring, limit-setting interventions that involve the adolescent's home, school, or social life, may create significant and unavoidable complications to the therapist's efforts to reinstate his position of technical neutrality. Under conditions when the adolescent's sexual behavior, drug or alcohol abuse, antisocial behavior, or problems with the law requires energetic interventions from the parents, and these interventions may be

complicated by authoritarian, even sadistic behavior from them, the therapist's efforts to maintain the structure of the treatment are particularly difficult, and enormous efforts may be required to differentiate his interventions from the authoritarian behavior of the parents. A careful equilibrium between respecting the privacy of the adolescent's sexual behavior and protecting the adolescent from dangerous expression of it, maintaining confidentiality while remaining within the boundary of legal dispositions, is a major challenge that has to be confronted from the viewpoint of what are the minimum moves away from technical neutrality that protect the adolescent's well-being and the viability of the treatment.

Transference Analysis

As in the case of adults, it is important to interpret both positive and negative transferences to prevent that, with the strong predominance of negative transferences typical in severe personality disorders, the therapist conveys the impression to the adolescent that he is "all bad." This becomes particularly relevant in the case of severe narcissistic transferences, with their tendency of dismissal and devaluation of all the therapist's suggestions. To point out, for example, the adolescent's capacity to be openly critical of the therapist as a way to stress the positive aspects of courage in his communications may help a patient who otherwise feels that he is always involved with a critical therapist.

Transference interpretation usually starts out with significant exploration of transference displacements to external figures. The interpretation of the transference in relation to the therapist himself may be opened as a "playful" invitation to the adolescent to express in fantasy and playful action in the session what his experiences or thinking about the therapists is. It is important to foster the cognitive framing of the adolescent's feelings in this regard in terms of the adolescent's developmental level. The therapist may make an appropriate bridging from play to verbal, symbolic communication of the meaning of the adolescent's experience and their interaction. The therapist suggesting to the adolescent that acting out behavior as well as somatization may, at times, express feelings that the adolescent wouldn't dare to express toward the therapist represents another bridging effort to bring these manifestations into a verbalized, affective, and symbolic context.

Countertransference

Countertransference, in its contemporary view, corresponds to the therapists total emotional reaction to the patient. In Kernberg's model a destinction is made between acute and chronic countertransference reactions. Acute reactions may manifest at a particular moment such as when the therapist is surprised by the intensity of the affect or the type of thought he might find himself having about the patient. Chronic reactions on the other hand is a much more stable emotional disposition towards the patient—these distinctions will be discussed further in our manual (Normandin et al., in press). The severity of the adolescent's acting out in the sessions and outside the sessions may promote strong countertransference reactions, reflecting concern both for the adolescent and over the risk that the treatment will be interrupted by adolescent's behavior that cannot be tolerated by the parents, and provokes their hostile reactions against the therapist. The intense, consistent dismissal and devaluation of the therapist's interventions in the case of adolescents with severe narcissistic personality disorder may, over a period of time, seriously disturb the therapist's sense of security, raising intense feelings of failure, and provoke the temptations to giving up on the adolescent. The therapist may lose sight of any positive transference manifestations. The adolescent's wishes for maintaining a dependent relationship with the therapist, in spite of the adolescent's constant attacks on him, may be missed. Erotic countertransferences to sexually seductive adolescent may disturb a therapist more than corresponding countertransferences evoked by adult adolescents, stirring up profound oedipal prohibitions against intergenerational activation of sexual desire. The therapist needs to tolerate these experiences in himself/herself in order to observe and come to understand them fully, and

neither act on them nor communicate them directly to the adolescent, but use these reactions as material to be woven into transference interpretations. The general preparedness of the therapist to be alert to the risk of either adopting a seductive "freedom fighter" attitude toward the adolescent or to become the "policeman" for inefficient parents should provide a general frame helping the therapist to maintain an objective stance regarding his countertransference temptations.

There are times when the treatment is "blocked." There may be weeks of "non-understanding," or a pervasive sense of hopelessness that interferes with the active work with transference and countertransference. Tolerance of such periods with an openness, at times, to share with the adolescent the impression that the treatment has come to a standstill may open up new information about transference and countertransference lines. There are adolescents with severe self-destructive tendencies and the unconscious tendency to destroy whoever tries to extend them a helping hand, associated with the syndrome of malignant narcissism that may seriously limit the effectiveness of the treatment. We have to accept that not everybody can be helped with this treatment, or even with treatment in general. The major prognostic indicators, corresponding to those for adults, are the adolescent's remaining capacity for non-exploitive object relations, the absence of antisocial features and of secondary gain, and the adolescent's intelligence and demonstrated potential for creative functioning in some areas. A supportive family environment may be a major positive contributing factor to supporting the treatment with a very disturbed adolescent.

Conclusion

TFP-A as we have described is a psychodynamic treatment that was adapted for adolescents following several studies that provide support for TFP as an evidence-based treatment for adult patients with BPD (Clarkin et al., 2004; Doering et al., 2010; Levy et al., 2007). In clinical practice we have been using TFP techniques for adolescents over the past 20 years, but the impetus to formalize the treatment and its adaptations came initially from Paulina Kernberg who was convinced that early diagnosis and intervention with children and adolescents was both a priority and realistic. The present climate is much more favorable for diagnosing and treating BPD in adolescence, and early skepticism has been replaced by enthusiasm and encouragement for developing and adapting treatments for adolescence. The time is ripe for formalizing and describing the adaptations that clinicians have been making to existing treatments in order for their adolescent BPD patients also to benefit from manualized treatments. We are encouraged by the satisfaction of seeing adolescents resume a normal development, by the fact that clinical psychology students can be trained to apply this treatment model in their work with adolescents, by the spirit of cooperation between those working on adapting treatments for adolescents, as well as the scientific advances and interest in the development of this problematic at adolescence. We see the treatments as complimentary, serving different populations, and with many common elements. While the treatments commonly address emotional regulation and mentalization, the unique component of TFP-A is its capacity to support normal development while addressing and changing the path of the development of personality through addressing extreme affects and split-off self and object representations. The effective integration of the adolescent's self-concept and his concept of significant others, that is, the development of a normal ego identity corresponding to a normal adolescent developmental stage, will facilitate the adolescent's resumption of normal psychological growth. It will show that the optimal features of TFP are not educational or reeducative efforts, but the establishment of the adolescent's internal freedom to enrich his internal experience and develop creative relationships in school, work, love, friendship,

family, and social life. We have shown that it is possible to train graduate students in psychology to become competent TFP therapists with adolescents, and we are now conducting a study to collect evidence of the efficacy of TFP-A.

Appendix: Clinical Vignette

Case of Jacob

Jacob, aged 13½, was brought to the clinic by his parents after his school threatened to expel him because he physically assaulted another student. In spite of above average intellectual ability, he was failing at school and had a long history of oppositional behavior at school and at home. At home, his parents were at a loss as to how to deal with his swings from being oppositional, provocative, and argumentative to being stubbornly silent and passive aggressive, or overly dependent, infantile, and submissive at other times. They were also concerned about the extent to which he was bullying his younger brother. In addition he was eating uncontrollably and never seemed satisfied, and as a result was becoming increasingly overweight. In terms of his early history, his mother described him as a demanding and hypersensitive baby. Her first impression of him at birth was that there was something in the way that he looked at her that evoked a fear in her that he would suck her dry.

In therapy, Jacob habitually slouched in his chair and seemed to cut off and became morosely silent and exaggeratedly tired and sleepy the moment he entered the therapy room. This contrasted sharply with how he behaved when the therapist fetched him in the waiting room when he seemed evidently happy to see her, talking on the way to the therapy room about computer games, card collections, and television series and being obviously pleased when he could see that she new what he was talking about. The main difficulty however was his extreme and prolonged silences during the sessions. While it is common for adolescents to be silent especially during the beginning phase of treatment, in Jacob's case his silence went far beyond this. An intense paranoid reaction was apparent and he acts like someone who has been dropped behind enemy lines. Jacob used his silence so that he could feel in control of the relationship, and while this defended him from revealing and facing a much more sensitive dependent side, it also left him with a very restricted inadequate range of interpersonal responses, and evoked frustration and rejection in others, who felt devalued and treated as if they were trying to control him. His peers did not tolerate his superiority and haughtiness and humiliated and rejected him when he responded like that, something he was highly sensitive to and unable to defend himself from, except through aggressive retaliation.

The following extract is from a session after a humiliating experience at a summer camp where his characteristic stubbornness and refusal to participate in any activity provoked mockery and rejection from the other boys. For example, he refused to prepare for a 3-days survival excursion, something that could potentially place the other members of the group at risk. He found the rejection by his peers extremely humiliating and difficult to tolerate, but had no other strategy to repair and reinsert himself socially and consequently remained rejected and isolated. When he was no longer able to tolerate this situation, he phoned his father and asked him to come and fetch him.

In this session the therapist uses clarification, confrontation, and interpretation to address the dyad that Jacob sets up with the therapist where he induces her to become the controlling object.

Th: Do you have any further thoughts about the meeting we had with your parents?

Pt: No, but I guess we are obligated to talk about it. (The therapist had the impression here that this was said without hostility, and that Jacob actually wanted to speak about it, but that he would only do this in the context of an interrogation where he set up a dyad, where he was the victim and the therapist the torturer).

Th: Does it mean that you don't want to share your thoughts because you have the impression that I am forcing you?

Pt: Let's say, just as a question, what is your point in asking me. If YOU want to, we will end up talking about the meeting, about what you have seen (He has successfully reestablished the victim–torturer dyad, even though the family session had ended up with a feeling of cooperation).

Th: If I understand you correctly, you seem to think that I have something in mind and you will have to hear it no matter if you like it or not, no matter if you want it or not... It may be important to try to understand why you see it like that; either you are right but then we have a problem because I am certain that you know that therapy is not about a therapist imposing on a person in need; or there is something in you right now that need to see me as imposing my own point of view on you.

Pt: (Interrupting the therapist) I have nothing to say!

Th: Wait a minute Jacob, are you answering my first question or commenting on what I just said? Right now, I was questioning the fact that you stay with the impression that I am forcing you to talk, that you have no choice and this situation leaves us with two options; either you are right or this way of seeing me help you in someway.

Pt: I have the choice to leave if I want... I also have the right to remain silent and sleep for an hour (said in a somewhat haughty tone).

Th: (smiling) Yes, this is right.... and by doing that you can be freed from having to decide between the two ways of seeing the situation.

Pt: (Nodding his head with a triumphant smile). Yes!

Th: You smile.... As if now you are the one who is in control of the situation, and of me.

Pt: Yes!

Th: What I am wondering now is that during the session with your parents you were able to share what you have been experiencing at the camp and seemed to be able to participate in the discussion actively and honestly. I am wondering if anything has happened since then to explain why you seem now to behave as if I am against you.

Pt: I don't know; nothing has changed.

Th: This is interesting... do you remember how you were able to talk during that session?

Pt: No, I am the same.

Th: Right now could you say that there is a part of you that is convinced that I am controlling you so much so that you feel justified to not respond.... I understand that.

Pt: (looking more vulnerable) But I have the right to stay silent, you just said so, what is the problem?

Th: Right now we are stuck because you are so convinced that I want to control you or force you that you don't see any other possibilities but to oppose. This thought seems so strong that you even forgot how it was during that session and specially at the end of that session. It looks as if something terrible is going to happen between you and me. Do you have any idea?

Pt: I am sure that you will want to dig, and dig, and dig, and find another fault...

Th: Hah! Hah! Again this is quite interesting and I think we have to try to understand what is going on right now. What I understood at that meeting was that you had a good reason for not wanting to stay at the camp anymore, and that your parents didn't understand that. They were not able to understand the reason it was so difficult for you to stay at the camp. .....What I understood from what you were saying is that it was difficult for you to stay there because you could not bear to be humiliated for being French and being treated as different from Quebecers and put aside. You found it difficult to protect yourself and defend yourself. I don't think your parents knew that side of you at least it is not a side of you that you show to them. Most of the time, they see quite a different side of you. It seems that there is a side of you that feels easily hurt, easily humiliated, and you are not able to protect yourself and then there is that other side that acts as if nothing happened.

Pt: (silent but listening).

Th: When we look at what is happening between you and me right now, you seem to be engaged in a similar struggle with me. Because you are deeply convinced that I will find faults, that I will humiliate you, you don't have any other choice but to start a battle and being really

decided to win that battle. But do you see how this side clashes with the one side at the camp who could not tolerate being joke at and humiliated. So, there is that part of you that is very "defiant," very "arrogant" as your parents would say, and that other part of you that is very very sensitive, easily hurt, and defenseless. What do you think?. . .. Is it possible?

Pt: (mumbling) It is possible!. . .. . . I don't know. . .... Anyway, what is the problem?

Th: I guess we have a problem. I say "we" because I think that what happened at the camp is serious because it shows a part of you that needs help to learn to protect yourself. But when I offer my help you don't see it like help, you have the conviction that I will humiliate you even more, that I won't let you say what you want to say. . . that I will do whatever I want with you, that I will force you to talk, that I will torture you. . . So we have a problem because it seems like the only way you think you can protect yourself from me is to stand up against me. It is okay in a way because I think you get some sort of a reassurance from that. It is like saying to yourself: "So it is me who is in control here, nothing will happen to me! . . .. It is OK. . .. in the sense that you communicate something important there, but deep down, there is a problem. . .And the problem is that it is your only card. When you get into a situation like that, let's say with your parents for example, . . . they react quite strongly when you enact this role because you don't have any other card in your pocket. With your classmates, or with the other boys at the camp, you couldn't use that card, or maybe you used it, I don't know. But, am I right if I say that if you were inflexible with them, they will go away or they will continue to provoke you and hurt you."

Pt: That is true. They were laughing at me.

Th: Your card, the only card you have in your game, which is to be opposed. . . to stand up. . . was not working there and it left you exposed.

Pt: Hum, hum. . . yes.

Th: Yes!. . . I find you quite courageous for saying "yes" like during the meeting with your parents, I also found you courageous for tolerating being there with them while they were obviously angry and depreciative of you. Courageous for staying there. You didn't subside into your chair, you didn't fall asleep. You did not provoke your parents too much. You were able to tell me enough about what happened with the boys at the camp and the issue around your French accent so much so that I could understand how difficult it must have been for you at camp. I found you courageous because you, in a way, admitted that it had nothing to do with finding the camp boring, that it had nothing to do with the fact that it was not what you were expecting.

Pt: (looking engaged and interested).

Th: And I don't know if you had noticed something then, but your father changed his attitude towards you just before the end of the meeting. He mentioned that you have expressed remorse for having him to drive all the way to the camp to fetch you and that you were searching somehow for ways to repair it by offering to pay for the expenses.

Pt: I know, I remember.

Later in the session:

Th: I wonder if we can understand that famous incident where you assault one of your classmates last fall. I wonder if there is a link between being unable to protect yourself when you feel humiliated and exploding?. . . You know between the fact that this person had probably provoked you by humiliating you and that the only way you could find at that moment, to stop the torture, to protect yourself was by hitting him. . ..

Pt: Yes, he did not want to stop. The girl too, was provoking me (revealing by the same token that he had been assaulting at least one other classmate).

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HYPE: A Cognitive Analytic Therapy-Based Prevention and Early Intervention Programme for Borderline Personality Disorder

23

Andrew M. Chanen, Louise McCutcheon, and Ian B. Kerr

Introduction

Borderline personality disorder (BPD) is a leading candidate for developing empirically based prevention and early intervention programmes because it is common in clinical practice, it is among the most distressing and functionally disabling of all mental disorders, it is often associated with help-seeking, and it has been shown to respond to treatment, even in those with established disorder. Moreover, BPD can be reliably diagnosed in its early stages and it demarcates a group with high levels of current and future distress, morbidity and mortality, making intervention a clinically justified and humane response. Data also suggest considerable flexibility and malleability of BPD traits in youth, making this a key developmental period during which to intervene.

Accordingly, we have developed the Helping Young People Early (HYPE) programme, a comprehensive and integrated indicated prevention and early intervention programme for youth (15–25 years of age). HYPE includes both a service model and an individual therapy, and incorporates the principles of cognitive analytic therapy (CAT) into both components.

CAT is a time-limited, integrative psychotherapy that arose from a theoretical and practical integration of elements of psychoanalytic object relations theory and cognitive psychology, subsequently developing into an integrated model of development and psychopathology. CAT is practical and collaborative in style, with a particular focus upon understanding the individual's problematic self-management and interpersonal relationship patterns and the thoughts, feelings and behavioural responses that result from these patterns. A central feature in CAT is the joint (patient–therapist) creation of a shared understanding of the patient's difficulties and their developmental origins, using plain-language written and diagrammatic 'reformulations'. These form the basis for understanding self-management and relationship problems both within and outside therapy, assist the patient to recognise and revise their dysfunctional relationship patterns and assist the therapist to avoid or recover from collusion with such relationship patterns. CAT has particular advantages for early intervention in BPD, especially because its integrative and 'transdiagnostic' approach encompasses the myriad co-occurring problems, which are the norm in this patient group, within the overall treatment model.

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Prevention and Early Intervention Makes Sense

The long-term outcomes for adult American patients with established BPD are now well recognised. By 10 years, 85 % of adults with BPD 'remit' (no longer meet five or more DSM-IV BPD criteria) (Gunderson et al., 2011), rising to 99 % at 16 years (Zanarini, Frankenburg, Reich, & Fitzmaurice, 2012). This so-called remission tends to be stable but *recovery* is more elusive. When recovery is defined as 2 years of both remission of BPD symptoms and good social and vocational functioning (Zanarini, Frankenburg, Reich, & Fitzmaurice, 2010), only half of adult BPD patients will recover by 10 years. One third of those recovered will later 'relapse'.

It is now evident that BPD is associated with severe distress and persistent functional disability, which is at least as severe as that associated with major depression (Gunderson et al., 2011). There is also high family and carer burden (Hoffman, Buteau, Hooley, Fruzzetti, & Bruce, 2003) and high rates of continuing resource utilisation (Horz, Zanarini, Frankenburg, Reich, & Fitzmaurice, 2010). Despite persistent help seeking, 8–10 % of adults with BPD will die by suicide (Paris & Zweig-Frank, 2001; Pompili, Girardi, Ruberto, & Tatarelli, 2005).

Notwithstanding the significant achievements of the past two decades of treatment research for adults with BPD (e.g. Bateman & Fonagy, 2009; Giesen-Bloo et al., 2006; Linehan et al., 2006), the overall outcomes from such interventions have been relatively modest. Moreover, many evidence-based interventions are complex and lengthy. Their implementation and availability are limited in most healthcare systems and they tend to be offered only to those patients who are 'motivated' to enter into treatment, leaving the majority of BPD patients untreated, undertreated or subject to unhelpful interventions with high a likelihood of iatrogenic harm and demoralisation (of patients and staff) (Mulder & Chanen, 2013).

These data support a *prima facie* case for developing prevention and early intervention programmes for BPD to complement established treatment services. These are intended to be made available earlier in the course of the disorder and offered to a wider variety of individuals and carers who access the health system. This chapter outlines the rationale for developing such programmes, and why combining *indicated prevention* and *early intervention* is currently the best alternative. It also describes the application of this theory to a frontline, 'real world' clinical setting in Melbourne, Australia where the HYPE programme has been operating for over a decade. HYPE is a comprehensive indicated prevention and early intervention programme that includes both a service model and an individual therapy, which incorporates the principles of CAT.

BPD in Young People

Despite longstanding general agreement that personality disorders (PDs) have their roots in childhood and adolescence (APA, 1980), diagnosing PDs prior to age 18 years has been more controversial than diagnosing PDs in adults (Chanen & McCutcheon, 2008b), but this is no longer justified (National Collaborating Centre for Mental Health, 2009; National Health and Medical Research Council, 2012). BPD is increasingly seen as a lifespan developmental disorder (Tackett, Balsis, Oltmanns, & Krueger, 2009) that is similarly reliable and valid when applied to adolescents or adults (Chanen, Jovev, McCutcheon, Jackson, & McGorry, 2008; Miller, Muehlenkamp, & Jacobson, 2008), is not reducible to other diagnoses (Chanen, Jovev, & Jackson, 2007), and can be identified in day-to-day clinical practice (Chanen, Jovev, Djaja, et al., 2008).

In fact, BPD might be better considered as a disorder of younger people, with a rise in prevalence from puberty and a steady decline with each decade from young adulthood (Johnson et al., 2000; Samuels et al., 2002; Ullrich & Coid, 2009). Limited data suggest that BPD occurs in approximately 3 % of community-dwelling (Bernstein et al., 1993; Moran, Coffey, Mann, Carlin, & Patton, 2006) and up to 22 % of outpatient (Chanen et al., 2004; Chanen, Jovev, Djaja, et al., 2008) adolescents and young adults.

BPD (or dimensional representations of BPD) in young people demarcates a group with high morbidity and a particularly poor outcome. BPD uniquely and independently predicts current psychopathology, general functioning, peer relationships, self-care and family and relationship functioning (Chanen et al., 2007). It also uniquely predicts poor outcomes up to two decades into the future, such as a future BPD diagnosis, increased risk for other mental disorders (especially substance use and mood disorders), interpersonal problems, distress and reduced quality of life (Cohen, Crawford, Johnson, & Kasen, 2005; Crawford et al., 2008; Winograd, Cohen, & Chen, 2008).

A Practical Strategy for Prevention and Early Intervention

The above data suggest that BPD is a leading candidate for developing empirically based prevention and early intervention programmes because it is common in clinical practice, it is among the most distressing and functionally disabling of all mental disorders, it is often associated with help-seeking (cf. schizotypal or antisocial personality disorders, (Tyrer, Mitchard, Methuen, & Ranger, 2003)), and it has been shown to respond to treatment, even in those with established disorder. Moreover, BPD can be reliably diagnosed in its early stages and it demarcates a group with high levels of current and future morbidity and mortality. Data also suggest considerable flexibility and malleability of BPD traits in youth (Lenzenweger & Castro, 2005), making this a key developmental period during which to intervene, and adolescent BPD features have been shown to respond to intervention (Chanen, Jackson, et al., 2008, 2009; Schuppert et al., 2009, 2012).

Aims of Prevention and Early Intervention

Prevention and early intervention for BPD should primarily aim to alter the life-course trajectory of young people with borderline personality pathology by attenuating or averting associated adverse outcomes and promoting more adaptive developmental pathways. It should not be narrowly focused upon the diagnostic and symptomatic features of BPD, as these naturally attenuate over time.

Antisocial personality disorder (ASPD) provides a useful model for such purposes. There is a remarkable amount of information about childhood-onset and adolescent-onset conduct disorder (CD) and the developmental pathways leading to ASPD, along with associated outcomes such as substance abuse, mental disorders and poor physical health (Moffitt et al., 2008). These data logically give rise to potential 'universal' (whole population), 'selective' (asymptomatic but with risk factors) and 'indicated' (symptomatic but not 'case level' disorder) preventive interventions (Mrazek & Haggerty, 1994), along with early intervention for the established phenotype (Weisz, Hawley, & Doss, 2004; Woolfenden, Williams, & Peat, 2002).

Although the time course and form of early manifestations of BPD are likely to differ from ASPD, the two disorders have substantial phenotypic overlap and similar objectives might be realised for BPD through identifying appropriate risk factors and antecedents for intervention.

What Form Should Intervention Take?

Risk Factors: Implications for Universal and Selective Prevention

We have reviewed the findings from prospective longitudinal studies of community samples and studies of young people with borderline pathology elsewhere (Chanen & Kaess, 2012). These suggest a variety of genetic, neurobiological, psychopathological and environmental risk factors for BPD. However, a fundamental drawback of these data is that their specificity for BPD appears to be limited (Chanen & Kaess, 2012), making these findings less than informative for the purposes of prevention.

Stand-alone universal (whole population) prevention of BPD is not currently feasible because

BPD is not sufficiently prevalent to justify whole population approaches and it is unclear exactly what form or 'dose' of intervention would be appropriate. Similarly, selective prevention (targeting those with risk factors for BPD) is currently impractical because many of the risk factors for BPD (particularly environmental factors) more commonly lead to, or are associated with, outcomes other than BPD (i.e. multifinality; Cicchetti & Toth, 2009). This should not diminish the importance of intervention for some risk factors (e.g. child abuse and neglect) as primary objectives because they are undesirable, immoral or unlawful. However, many factors (e.g. poverty and inequality) require a major social and political change and are unlikely to have a major impact on BPD prevention in the near future. Also, it is difficult to design studies with adequate statistical power to demonstrate the efficacy or effectiveness of universal and selective prevention (Cuijpers, 2003). Some of these problems would be overcome if current universal and selective programmes (e.g. parent training programmes) were to measure multiple syndromes as outcomes, and the above data constitute a strong case for including BPD as one of these syndromes.

Precursor Signs and Symptoms: Implications for Indicated Prevention

Prospective longitudinal data indicate that certain temperamental characteristics and early onset mental state or behavioural problems that are analogous to characteristics of BPD are precursors to the emergence of the BPD phenotype but do not predict its onset with certainty. These include attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), conduct disorder, substance use, depression and deliberate self-harm (DSH), along with the actual features of BPD. However, it is technically imprecise to refer to many of these phenomena as 'risk factors' (Kraemer et al., 1997), as these same phenomena are later used to define PD. Eaton, Badawi, and Melton (1995) refer to the signs and symptoms from a diagnostic cluster that precede a disorder but do not predict its onset with certainty as *precursor signs and symptoms*.

Maternal reports of childhood temperament are related to BPD in adolescence or adulthood, up to 30 years later (Carlson, Egeland, & Sroufe, 2009; Crawford, Cohen, Chen, Anglin, & Ehrensaft, 2009). Substance use disorders during adolescence, particularly alcohol use disorders, also specifically predict young adult BPD (Rohde, Lewinsohn, Kahler, Seeley, & Brown, 2001; Thatcher, Cornelius, & Clark, 2005) and there are strong prospective data that disturbances in attention, emotional regulation and behaviour, especially the disruptive behaviour disorders (CD, ODD, ADHD) in childhood or adolescence are independent predictors of young adult BPD (Burke & Stepp, 2012; Carlson et al., 2009; Stepp, Burke, Hipwell, & Loeber, 2012). Moreover, one study suggests that for adolescent BPD symptoms, difficulties with emotion regulation and relationships might precede problems with impulse control (Stepp et al., 2012).

DSH is a core feature of BPD (Leichsenring, Leibing, Kruse, New, & Leweke, 2011) and retrospective reports from adults with BPD indicate childhood-onset of DSH in more than 30 % and adolescent-onset in another 30 % (Zanarini et al., 2006). However, DSH is surprisingly under-researched as a potential precursor to BPD. Although DSH is relatively common among adolescents and young adults (Nock, 2010) and is associated with a range of clinical syndromes, there is evidence that repetitive DSH, which is less frequent, might differ from occasional DSH (Brunner et al., 2007). BPD can be diagnosed in the majority of female adolescent inpatients with DSH (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006) and the likelihood of meeting the diagnosis of BPD is greater in adolescents endorsing both DSH and suicide attempts compared with individuals reporting DSH or suicide attempts alone (Muehlenkamp, Ertelt, Miller, & Claes, 2011). Also, the number of BPD criteria met is predictive of whether or

not an adolescent has engaged in DSH or attempted suicide (Jacobson, Muehlenkamp, Miller, & Turner, 2008).

There is now clear evidence that dimensional representations of BPD features have similar stability in adolescence and adulthood (Chanen, Jovev, McCutcheon, et al., 2008). Evidence is emerging that the underlying dimensions of BPD features (conceptualised as impulsivity, negative affectivity and interpersonal aggression) are also stable in children (Crick, Murray-Close, & Woods, 2005; Stepp, Pilkonis, Hipwell, Loeber, & Stouthamer-Loeber, 2010). Only one study has specifically measured childhood or adolescent PD features as a predictor of later PD over multiple assessments from childhood to adulthood (Cohen et al., 2005). PD symptoms in childhood or adolescence were the strongest long-term predictors, over and above disruptive behaviour disorders and depressive symptoms (Bernstein, Cohen, Skodol, Bezirganian, & Brook, 1996; Cohen, 1996; Cohen et al., 2005; Kasen, Cohen, Skodol, Johnson, & Brook, 1999), of later DSM-IV cluster A, B or C PD. Overall, the data support a normative increase in BPD traits after puberty, perhaps bringing the problems associated with BPD to clinical attention. As this wanes in early adulthood, partly due to maturational or socialisation processes (Cohen et al., 2005), a group is revealed that is increasingly deviant compared with their peers (Crawford et al., 2005) and that might more closely resemble the 'adult' BPD phenotype. This suggests that young people displaying BPD features are a major group from which the adult BPD phenotype arises.

In short, signs and symptoms appear from childhood through to adolescence that resemble aspects of the BPD phenotype and presage its later appearance in adolescence or emerging adulthood. Certain early temperamental and personality features, internalising and externalising psychopathology and specific BPD criteria are all candidate precursor signs and symptoms. However, more work needs to be done to gain a better understanding of the role these factors play in the developmental pathways to BPD and to increase their specificity for BPD.

The data reviewed above suggest that 'indicated prevention' (Chanen, Jovev, McCutcheon, et al., 2008) is currently the 'best bet' for prevention of BPD. This targets individuals displaying precursor (i.e. early) signs and symptoms of BPD. Although the BPD phenotype is not robustly identifiable in children, its underlying dimensions can be measured, appear to be relatively stable and could be directly targeted. Moreover, typical child and adolescent psychopathology (e.g. disruptive behaviour disorders, DSH, substance use, depressive disorders) might additionally be regarded as targets for indicated prevention of BPD, rather than separate domains of psychopathology that might then be renamed in adulthood. Two programmes have been developed that directly target sub-syndromal borderline pathology in adolescents (Chanen, Jackson, et al., 2008; Chanen, McCutcheon, et al., 2009; Schuppert et al., 2009), while concurrently targeting syndromal BPD.

Early Detection and Intervention

Early detection and intervention for BPD is now justified and practical in adolescence and emerging adulthood (Chanen, Jovev, Djaja, et al., 2008; National Collaborating Centre for Mental Health, 2009; National Health and Medical Research Council, 2012) and consequently, we have developed and researched a novel early intervention programme (Chanen, Jackson, et al., 2008; Chanen, McCutcheon, et al., 2009). This programme should be differentiated from conventional BPD treatment programmes that are applied to individuals who have established, complex and severe BPD but who happen to be less than 18 years old. Treatment for this latter group should already be considered part of routine clinical practice in adolescent mental health (National Collaborating Centre for Mental Health, 2009; National Health and Medical Research Council, 2012).

The HYPE Programme: Indicated Prevention and Early Intervention for BPD Using Cognitive Analytic Therapy

Cognitive Analytic Therapy

CAT is the core of the HYPE therapeutic model and is the *lingua franca* of the team. CAT is a time-limited, integrative psychotherapy that has been developed in the United Kingdom over the past 30 years (Ryle & Kerr, 2002). CAT arose from a theoretical and practical integration of elements of psychoanalytic object relations theory and cognitive psychology, developing into an integrated model of development and psychopathology. Key features of the CAT model of development and psychopathology are outlined in Fig. 23.1.

The self is seen in CAT to be characterised by an 'internalised' repertoire of relationship patterns, acquired throughout early and subsequent development. When development is suboptimal (as in the development of personality disorders) and early caregiving interactions are less nurturing or even destructive, these relationship patterns will be internalised and used or re-enacted inappropriately and/or inflexibly.

CAT is practical and collaborative in style, with a particular focus upon understanding the individual's problematic relationship and self-management patterns and the thoughts, feelings and behavioural responses that result from these patterns. A central feature in CAT is the joint (patient–therapist) creation of a shared understanding of the patient's difficulties and their developmental origins, by means of plain-language written and diagrammatic 'reformulations'. These form the basis for understanding relationship problems both outside and within therapy and assist the patient to recognise and revise their dysfunctional relationship and self-management patterns. Because of its collaborative style and strong relational focus, CAT has been increasingly used with more complex and relational types of disorder, especially BPD (Ryle, 2004), where it has a specific model and treatment approach (Ryle, 1997a).

CAT has particular advantages for early intervention in BPD. Its integrative and 'trans-diagnostic' approach encompasses co-occurring problems (e.g. other personality pathology, mental state and substance use disorders) within the overall treatment model, rather than seeking separate interventions. Also, CAT sees 'psychological mindedness' as a goal of therapy, rather than a prerequisite. Youth, especially those with BPD, rarely present as 'therapy ready' in any traditional sense and they often have limited and/or adverse experiences of mental health services or therapy. Finally, while CAT is essentially a talking-based therapy, the model can be modified for use with less verbal patients or those with intellectual/learning difficulties and can also encompass a range of other (e.g. behavioural) approaches.

Routinely, 16 CAT sessions (plus whatever case management is required) are offered to each patient, with four post-therapy follow-up sessions (at 1, 2, 4 and 6 months) to monitor progress and risk. This is negotiable to a lesser amount, especially for those who are ambivalent about treatment, but can be extended up to 24 sessions, if needed.

The CAT Approach to BPD

Key features of the CAT model of therapy for BPD are outlined in Fig. 23.2.

CAT adopts a dimensional approach to the conceptualisation of degrees of damage to and dysfunction of the self. From a CAT perspective, BPD is seen as a severe and complex disorder frequently characterised by considerable comorbidity. The self is understood as operating in states ranging from normal multiplicity through to those of overt dissociation (Ryle & Fawkes, 2007; Ryle & Kerr, 2002). Lesser degrees of damage to the self are characterised by the presence of mildly dysfunctional or maladaptive reciprocal role procedures for coping, located within a more integrated self that is capable of self-reflection, empathic interactions with others and an advanced capacity for executive function. However, more severe degrees of damage are characterised by failure of integration of the structures of the self (notably, its repertoire of reciprocal roles and reciprocal role procedures), and by lack of self-

- The model is predicated on a fundamentally relational and social concept of self; this implies that individual psychopathology cannot be considered apart from the sociocultural context in which it arose and within which it is currently located.
- In the context of individual genetic and temperamental variation, early socially meaningful experience is internalised as a repertoire of reciprocal roles.
- A reciprocal role is a complex of implicit relational memory that includes affect and perception and is characterised by both child-derived and parent/culture-derived poles; a role may be associated with a clear dialogical 'voice'.
- Enactment of a reciprocal role always anticipates or attempts to elicit a reciprocal reaction from a historic or current other.
- Reciprocal roles and their recurrent procedural enactments determine both subsequent interpersonal interactions and also internal dialogue and self-management.
- All mental activity, whether conscious or unconscious, is rooted in and highly determined by our repertoire of reciprocal roles.
- Human psychopathology is rooted in and highly determined by a repertoire of maladaptive or unhealthy reciprocal roles.
- More severe and complex damage to the self may occur as a result of chronic developmental trauma/deprivation, resulting in dissociation and disruption of the repertoire of reciprocal roles and consequent impairment of self-reflective and executive function. These phenomena are accounted for in the 'multiple self-states model' of borderline personality disorder.

Fig. 23.1 Key features of the cognitive analytic therapy model of development and psychopathology

reflective capacity and problems associated with a lack of a coherent and continuous sense of identity (Kerr, 2005; Ryle, 1997b, 2004). Such a disorder is also typically characterised by extreme psychological distress that might manifest as a stress-related dissociation into different self-states as well as extreme coping procedures. Dissociation is also conceived of as the principle mechanism through which developmentally abusive, traumatic and depriving interpersonal experiences have a deleterious effect on the developing self. The damage is considered to occur in the context of likely neurobiological vulnerability through, for example, impaired impulse control and/or proclivity to dissociation in the face of (psychological) trauma (Ryle & Kerr, 2002).

This conceptualisation addresses and largely accounts for the range of psychopathology encountered in BPD, in particular the tendency under pressure to switch suddenly and apparently unpredictably between different self-states, with their associated differing reciprocal roles and reciprocal role procedures (Pollock, Broadbent, Clarke, Dorrian, & Ryle, 2001). These switches between self-states represent some of the most problematic and challenging enactments encountered in working in any capacity with people with BPD, often causing such patients to be seen as 'difficult' or 'hard to help'—at least in the absence of a coherent model accounting for these interactions. Another advantage of the CAT model in this context is its explicit and

- Proactive and collaborative ('doing with') style, stressing the active participation of the patient/client.
- Aims at non-judgemental description of, and insight into origins and nature of, psychopathology conceived as procedural enactments of reciprocal roles and associated dialogical voices, and of a tendency under stress to dissociate into different self-states.
- Aims to offer a new form of non-collusive relationship with a benign, thoughtful other that the patient/client can internalise in the form of new reciprocal roles and that enables the exploration of new perceptions of self and new ways of interacting with others; this is conceived of in terms of recognition and revision of maladaptive reciprocal role procedures.
- Therapy is aided by the early collaborative construction of written and diagrammatic reformulations (conceived of as psychological tools) by the end of the initial phase of therapy. These serve as 'route maps' for therapy and also as explicit narrative and validating testimonies.
- Therapy subsequently focuses on revision of maladaptive reciprocal role procedures and associated perceptions, affects and voices as they are evident in internal self-to-self dialogue and self-management, through enactments in the outside world, and also as manifest in the therapy relationship (as transference and countertransference).
- Further techniques may facilitate this ranging from challenging of dialogical voices to behavioural experiments, mindfulness exercises, 'empty chair' work or active processing of traumatic memories.
- The focus from the beginning is on a time limit (whether in individual therapy or CAT-informed approaches in other settings); 'ending well' is seen as an important part of therapy (experience of new reciprocal roles), and as a means of addressing issues surrounding loss and of avoiding protracted and collusive relationships.
- Social rehabilitation is an important although often neglected aspect of therapy.

Fig. 23.2 Key features of the cognitive analytic model of therapy for borderline personality disorder

robust relational framework, which can help make sense of the frequently challenging relational dynamics, both individual and systemic, which represent a core feature of these disorders. The model can provide a *lingua franca* for teams and, ideally, to others involved in the care of the individual with BPD, which enables considered responses rather than 'knee-jerk' reactions to be made to 'difficult' and challenging patient behaviours (reciprocal role enactments) through use of tools such as an extended 'contextual' reformulation, even if formal therapy as such is not being offered to the patient. This can reduce staff stress, team splitting and burn out (Caruso et al., 2013; Thompson et al., 2008), and in turn improve the delivery of patient care.

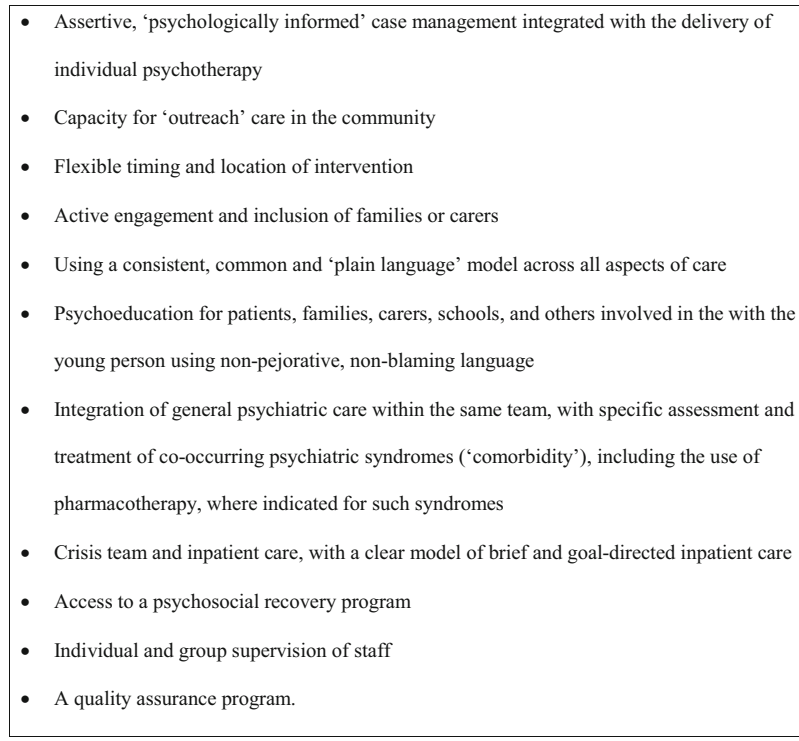

Fig. 23.3 Key elements of a team-based, integrated early intervention for BPD

Principles of Indicated Prevention and Early Intervention for BPD

There is such a great emphasis in the treatment literature for BPD on providing individual psychotherapy that it leads to the misleading conclusion that lengthy individual therapy is both necessary and sufficient for the treatment of all individuals with BPD. Little prominence is given to the service delivery models that support the provision of individual therapy for BPD (Mulder & Chanen, 2013), the emerging evidence that 'high quality care' for BPD might be as effective as 'branded' psychotherapies (Bateman & Fonagy, 2009; Chanen, Jackson, et al., 2008; McMain et al., 2009) or that intermittent care might be worthy of empirical investigation (Paris, 2007).

The HYPE model addresses these issues by defining a model of service delivery separately from the practice of individual psychotherapy, while using a common language and tools for the integration of both components. It also uses time-limited, intermittent treatment as its primary mode of intervention. The key features of this model are listed in Fig. 23.3 and elaborated in the following sections.

A Dimensional View of BPD

An indicated prevention and early intervention programme for BPD needs to adopt a dimensional view of BPD and to recognise its heterogeneity and 'comorbidity'. A dimensional view of BPD combines sub-syndromal (indicated prevention) and syndromal (early intervention) BPD. This also avoids unnecessary disputes about whether someone is eligible for the programme because of arbitrary diagnostic thresholds when there is a clear need for care. Nonetheless, operational criteria for personality pathology should be rigorously applied, often supported by semi-structured interview. This is especially so because DSH is relatively common among adolescents and young adults (Nock, 2010) and although it is commonly associated with BPD, it is also associated with a range of

other clinical syndromes, which often present clinically as 'blends' of psychopathology, rather than prototypical 'adult' syndromes.

Fitting the Treatment to the Patient (Not the Patient to the Treatment)

The very nature of BPD makes it unrealistic to expect that young people with BPD will organise themselves to attend regularly in the early phase of treatment. Rather, increased capacity for self-care and self-management is a goal of treatment. Expectations about and tolerance of disruptive behaviour needs to match the phase of intervention, while always being mindful of the safety of patients, carers and clinicians.

Youth with BPD often have difficulty fitting in with (adult) clinicians' expectations to attend appointments regularly and on time. HYPE adopts a flexible (time and location of appointments) and transparent (processes and policies) approach to engagement. When clinicians' needs (e.g. duty of care) might be experienced as being at odds with the patient's expressed needs, this is acknowledged. The CAT model facilitates this discussion through the early establishment of common ground. Our approach to challenges to engaging and treating young people and strategies for managing these difficulties are described elsewhere (Chanen & McCutcheon, 2008a; McCutcheon, Chanen, Fraser, Drew, & Brewer, 2007).

Responsibility for attendance is progressively handed over to the patient. Early in treatment, young people are actively followed up (e.g. telephone calls, letters and home visits) with a focus upon barriers to attendance. The early, joint development of a shared understanding of the patient's difficulties is used to promote this discussion and allows the therapist to be aware of collusion with the patient's dysfunctional relationship patterns. Early in therapy, therapist collusion might be deliberate and strategic (e.g. home visits to a passive, angry and controlling patient) to facilitate a dialogue promoting change.

Easy Accessibility

Early intervention programmes need to be offered to everyone presenting for care, rather than 'cherry picking' participants based upon non-evidence-based assumptions or judgemental attitudes about 'suitability' for therapy. Access to and use of high quality care does not require a commitment to regular psychotherapy.

Not everyone who is offered intervention will accept it and 'easy access' needs to be complemented by a mechanism for 'easy exit' after a defined period (usually 6 weeks) of vigorous attempts at engagement. Exit should also be accompanied by an invitation to return when needed.

Because co-occurring psychopathology is the norm in BPD, programmes need to have limited exclusions for co-occurring psychopathology, especially substance use disorders. Also, as described above, some of this psychopathology represents precursor signs and symptoms for BPD. Co-occurring psychopathology should be addressed within the overall BPD treatment plan, rather than provide a reason to fragment the patient and their care. This is particularly important in BPD, as every increase in the number of agencies involved also increases the potential for miscommunication. Multi-agency involvement is typical for this patient group. HYPE case manager/therapists adopt the same active, open, transparent and collaborative attitude with all concerned. The jointly constructed reformulation is used (with the patient's consent) within the CAT approach to promote a shared, plain-language understanding of the patient's difficulties that ensures all are 'singing from the same song sheet' and minimises professional disputes or 'splits' (Kerr, 1999). This model also facilitates advocacy on behalf of the young person.

Time-Limited and Intermittent Intervention

Time-limited intervention is a means of providing the young person with an opportunity to practice what they have learned in treatment and sets the expectation at this early stage of illness of living a fulfilling and functional life. Given the young age of this patient group, it is also a means of avoiding prolonged and/or collusive relationships from developing. Pragmatically, it also increases the capacity of the

programme to see a sufficient number of individuals to achieve its prevention aims.

Limiting Iatrogenic Harm

The time limit also serves to limit the potential for iatrogenic harm, which is unusually high in BPD (Mulder & Chanen, 2013) and a particular risk associated with early diagnosis and intervention.

Service Context

The HYPE programme (Chanen, McCutcheon, et al., 2009) is part of Orygen Youth Health (McGorry, Parker, & Purcell, 2007), the government-funded youth mental health service in western and north-western metropolitan Melbourne, Australia. Orygen services a catchment population of approximately 160,000 15–25-year-olds and offers a comprehensive mental health service for severe mental disorders.

Referral and Initial Assessment

Youth with BPD commonly seek clinical help but opportunities for early intervention are frequently missed (Chanen et al., 2007). Referrals are made to Orygen's single point of entry and are usually precipitated by symptoms of another disorder (e.g. major depression), not BPD *per se*. First episode psychosis patients are always allocated to Orygen's Early Psychosis Prevention and Intervention Service, regardless of comorbidity.

Selection of Patients

The primary inclusion criterion for HYPE is having three or more DSM-IV BPD criteria. Previously published data (Chanen, Jovev, Djaja, et al., 2008) indicate that 39 % of non-psychotic patients assessed at Orygen meet this threshold. This threshold reflects HYPE's mixed indicated prevention and early intervention mission (Chanen, Jovev, McCutcheon, et al., 2008), recognises the dimensional nature of BPD (Zimmerman, Chelminski, Young, Dalrymple, & Martinez, 2012), and reduces practical disputes about 'eligibility' when there is a clear clinical need for intervention, such as when there is prominent parasuicidal behaviour, impulsivity and affective instability, without meeting the threshold for a categorical diagnosis of BPD.

HYPE has no specific exclusion criteria for other forms of psychopathology in recognition of the heterogeneity of BPD, where comorbidity is the norm at any age (Chanen et al., 2007). Low IQ is not a contraindication to treatment in HYPE, provided the individual has sufficient verbal skills to participate in the programme.

Patients are not compelled to attend HYPE. Those with substance use problems or a history of overt aggression are asked not to attend appointments while intoxicated and to respect the safety of themselves and others while at Orygen. However, there is no 'behavioural contract' with new patients, as this is often experienced as both provocative and an invitation to a battle for control. Rather, these issues are addressed if and when they arise during referral, assessment or treatment, using the CAT model.

Screening and Assessment of BPD in Young People

Despite its high prevalence in clinical services, many clinicians lack the skills or confidence to assess BPD in young people. BPD often complicates assessment, frequently causing patients to feel intruded upon or overwhelmed. Operationally, a BPD criterion is defined as 'present' if it is displayed outside any period(s) of other major mental disorder(s), such as major depression, and there has been a recurrent pattern for 2 or more years (1 year longer than required for adolescents in the DSM-IV). Clearly, many PD features are exacerbated by other periodic mental disorders but they must be present, at least to some degree, outside of these periods.

Sometimes, distinguishing mental state from trait-based problems can be difficult but our overall experience is that the process (described elsewhere, Chanen, McCutcheon, et al., 2009) is usually uncomplicated. Assessment can be

facilitated by using a screening instrument, such as the 15 BPD items from the Structured Clinical Interview for DSM-IV Axis II disorders (SCID-II) Personality Questionnaire and its operating characteristics in outpatient youth have been described elsewhere (Chanen, Jovev, Djaja, et al., 2008). A score of 13–15 (out of 15) indicates a possible BPD diagnosis and 9–12 a possible sub-syndromal BPD diagnosis. Detailed clinical assessment for BPD is then conducted, supplemented by a semi-structured BPD interview (e.g. SCID-II BPD module).

Treatment Model

The elements of HYPE's integrated, team-based treatment model are described above (Principles of Early Intervention). A single practitioner (called a case manager) provides both psychotherapy and case management and all patients are jointly managed with a psychiatrist (or senior psychiatric trainee) and reviewed weekly by the entire treating team. The reasoning behind this model is both pragmatic and theoretical. First, integrating therapy, case management and psychiatric care minimises the number of clinicians involved, reducing opportunities for disputes or 'splits' among professionals. Second, combining therapy with case management provides opportunities to generalise progress in therapy to other problems and situations. Third, the costs involved in having two clinicians (therapist and case manager) per patient are relatively higher, as the work is never divided *pro rata*. Finally (and in our view most importantly), a team-based approach, provides a supportive environment for clinicians and facilitates the development of a 'common language' through a shared model of BPD and appropriate interventions for the disorder.

Although they are combined, the model clearly distinguishes between therapy and case management in order to avoid therapy sessions being 'hijacked' by day-to-day crises. Case management is defined as work that focuses upon general psychiatric care, housing, educational or vocational issues, family matters, liaison with other services and agencies and the management of suicidal crises or deliberate self-injury. Therapy is defined as time spent using the therapeutic approach and specific tools of CAT (see below), reflecting upon how and why the presenting problems have emerged and recur and the development of more adaptive ways of coping in the context of a benign and supportive therapy relationship. Although sessions normally observe a 'fifty minute hour', shorter sessions are possible, depending upon the capacity of the individual to manage therapy. This allows therapists to address patients' often unpredictable needs by offering some case management in addition to therapy within a realistic time frame. If the minimum amount of therapy (usually 25 min) is not achieved, another therapy session is scheduled in its place, preferably in the same week. If therapy sessions are repeatedly disrupted, this becomes a focus for the therapy itself.

Consent, Confidentiality and 'Informed Refusal'

Verbal informed consent is routinely obtained from the young person, along with parental or guardian consent. The right to and limits of confidentiality are clearly outlined to all involved at the outset and a clear statement is always made that 'duty of care' will prevail and that the safety of the young person and others is paramount.

BPD directly and adversely affects young people's capacity to access and use treatment services. Failure to attend appointments and other forms of non-communicative behaviour are expected and are not immediately interpreted as refusal of treatment. HYPE emphasises engagement and outreach, initially to inform potential patients about the actual nature of the treatment programme (often dispelling unfounded fears) and the risks and benefits of participating or not. Following 6 weeks of vigorous efforts to engage the young person (at least weekly phone calls, letters and home visits, where appropriate), non-attendees are discharged with an invitation for re-referral. A clear message of refusal is always respected, unless duty of care considerations must prevail.

The Episode of Care

Our clinical experience is that most youth drop in and out of treatment and prefer time-limited therapy contracts. This notion of 'intermittent' therapy for personality disorders has received some support in the literature (Paris, 2007). The CAT time limit does not preclude future episodes of CAT, either completing the balance of the 16-session intervention or in the form of 'booster' sessions. The emphasis in CAT is upon having an agreed ending, which is usually achieved. For those patients who do have a planned ending (as opposed to dropping out), the usual practice is to discharge them after their first follow-up appointment.

Family Involvement

Family conflict is a prominent feature of adolescent PD and 37 % of HYPE patients are not living with any biological parent by mean age 16 years (Chanen et al., 2007), rising to 57 % by mean age 19 years (Chanen, McCutcheon, et al., 2009). Consistent with young people's preferences, the HYPE intervention is mostly individually based but the usual practice is to at least involve family members or carers in assessment, treatment planning and psychoeducation and to provide support within the limits of confidentiality and resources. The primary aim of this involvement is to facilitate engagement and change in the patient. Where indicated, HYPE offers more formal family intervention sessions, conducted by the primary therapist and another HYPE clinician, as appropriate, within the overall CAT model.

Psychoeducation, Stigma and Discrimination

The BPD diagnosis is communicated with cautious optimism, based upon the natural history of improvement in BPD traits (Chanen, Jovev, McCutcheon, et al., 2008), the evidence supporting the effectiveness of the HYPE intervention (Chanen, Jackson, et al., 2008, 2009), and the natural limitations of such interventions. Education and training for patients and professionals about the nature of BPD in young people emphasises that they have infrequently entered into the mutually hostile relationship with the health system that often characterises adult BPD. There is little need to 'undo' iatrogenic complications or adopt defensive or discriminatory institutional practices, such as prohibiting inpatient care.

Pharmacotherapy

There are no methodologically sound studies of pharmacotherapy for BPD in young people. Psychotherapy and case management are given primacy in the treatment model and pharmacotherapy is presented as an adjunctive collaborative endeavour for co-occurring mental state (Axis I) disorders, such as mood or anxiety disorders, within the CAT model. The potential for polypharmacy is monitored (and discouraged) through weekly clinical review meetings.

After Hours Response and Inpatient Care

Written management plans are developed for all patients and made available electronically to Orygen's 24-h crisis team. These outline the jointly developed formulation of the patient's difficulties, current management plan and specific recommendations for management during acute crises that are based upon the shared formulation and goals developed with the patient. HYPE's primary aim is to promote appropriate self-care and self-management skills for community living and to minimise the risk of iatrogenic harm. Inpatient care is usually only used when all options for community treatment have been exhausted. Admission is usually voluntary, infrequent, brief and has specific goals. HYPE case managers work with inpatient and crisis teams to facilitate a 'common language', to minimise collusion with patients' problems and to achieve the goals of admission.

Treatment Fidelity and Supervision

Treatment fidelity and completion of the tasks of an episode of care (e.g. assessment, management planning, attendance, engagement and risk management) are monitored weekly. In common with most BPD treatment models, supervision is an

integral part of HYPE. It aims to support clinicians, allow time for reflection and to ensure a high standard of care. CAT supervision occurs weekly in small groups (two or three participants) and there is a peer group case discussion every 2 weeks. Individual case management supervision occurs once every 2 weeks.

Discharge

An explicit aim of HYPE is to promote support networks independent of mental health services and to avert unhelpful involvement with the mental health system. However, this is at odds with BPD patients' high needs for treatment of recurrent mental state disorders (Chanen et al., 2007) and their intolerance of aloneness. Referrals are often made to external, non-mental health networks for post-discharge support. Patients are also encouraged to practice what they have learned in therapy and to delay seeking further psychotherapy until their 6-month follow-up review. This does not preclude further case management or treatment of mental state disorders, as necessary. However, this is infrequently required.

Case Example: Madison

Madison was a 17-year-old female student living with her parents on the outskirts of a large city. She was referred from an Emergency Department, following an overdose of an unspecified number of tablets (paracetamol/acetaminophen, ibuprofen and zopiclone), combined with alcohol. She reported that she wanted to kill herself because her boyfriend wanted to end their 3-year relationship.

Madison reported 1 year of increasingly severe and persistent major depressive symptoms, increasing suicidal ideation, at least one other suicide attempt and several incidents of superficial cutting of her arms and abdomen. Concurrently, she also reported periods of dietary restriction and binge eating, gaining 20 kg (44 lb). She denied any history of anxiety, manic or psychotic disorder and there was no history of childhood inattention or hyperactivity.

These symptoms occurred on a background of longstanding relationship instability, impulsive behaviour (spending, alcohol and marijuana use, binge-eating), affective instability, feelings of emptiness and recurrent episodes of derealisation that lasted several minutes to an hour. She also reported 3–4 years of fluctuating low-grade depression, lack of motivation, feelings of worthlessness and suicidal ideation.

Madison began smoking tobacco and marijuana, and binge drinking alcohol with friends up to three times per week between the ages of 12 and 15 years. More recently, she only engaged in impulsive substance use (approximately weekly) when she felt low or upset.

Madison was the eldest of three girls and lived in an intact family. She was a planned pregnancy. She was described as a relaxed baby and generally her early childhood was unremarkable.

Her father was in the armed forces and the family relocated frequently and they struggled financially. Madison's father was often away for many months and her mother took part-time jobs outside the home. Madison and her younger sister were often left in the care of military friends or neighbours. Sometimes they were left unsupervised.

Madison changed schools frequently and her reading difficulty was not picked up until grade 4. At age 11, she disclosed that a male babysitter had sexually abused her several years earlier and she received six sessions of psychiatric care.

The family settled in one place when Madison began secondary school and her third sister was born. Madison's difficulties became substantially worse and her parents responded with increasing control and restrictions. This was met with increasing rebellion, which in turn exacerbated her parents' anxiety and authoritarian responses. She started dating a 16-year-old male and at age 14, she dropped out of school. She ran away from home for several days, used drugs and was sexually assaulted. Eventually, she was placed in foster care for 6 months, which allowed her to re-engage with school and for the conflict to settle enough for her to return home.

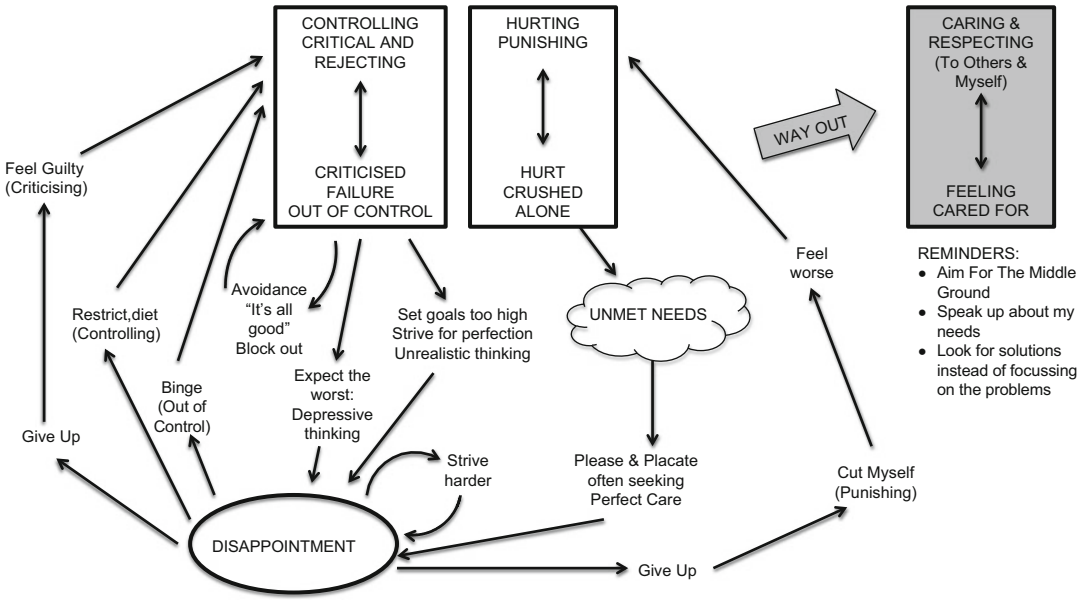

Fig. 23.4 Madison's sequential diagrammatic reformulation

Treatment

Madison was offered the standard HYPE programme, including 24 sessions of CAT, psychologically informed case management and general psychiatric treatment. Initial case management included two sessions with Madison alone, and one with her parents for an introduction to the service, assessment and psychoeducation. Around half way through the therapy, the therapist also visited her school to facilitate the transition into her final year of secondary school.

The shared goals for her treatment were to treat her major depression, reduce her risk-taking and to understand the relational patterns that drove her to feel bad about her ownself and to binge-eat.

Madison had regular psychiatric reviews during her 10-month episode of care as part of the team-based care. Antidepressant use was discussed early in her care but Madison declined. She did not receive any pharmacotherapy.

The first four sessions of CAT focused both upon her current difficulties and exploration of her developmental experiences. Madison felt cautious about discussing family relationships, resulting in a noticeable sense for the therapist to 'tread carefully'. She often answered the therapist's enquiries by saying that things were 'all good' and that she knew that her parents loved and cared for her. This was usually followed by bewildered silence, during which she sometimes stated that she couldn't understand why she felt so bad.

Madison's caution in talking about her early experiences was explored, especially the possibility that she might feel judged or criticised. Madison was able to identify that she had learned from her parents to set high goals, and to judge herself as a failure if she didn't manage to achieve these goals.

The first relationship pattern (Reciprocal Role) that was clearly identified was the one in which she felt others were *controlling*, *critical and rejecting* towards her (see Fig. 23.4). Initially, she could only state that in response to this, she felt overwhelmed, upset, angry and not good enough. Words that best described these feelings were added and changed over the next few sessions, in order to better capture the self-state and responses they elicited.

Madison was able to notice that she often expected others to be critical of her efforts, and this commonly led to her avoiding situations in

which she felt that she might be scrutinised. Discussion of her substance use revealed that this was a highly effective way of avoiding difficult emotional states. Over time, she was also able to acknowledge that this strategy was only effective in the short-term. She was surprised to consider that perhaps her 'risk-taking' and substance use looked 'out of control' to others, and therefore invited others to attempt to control her more. This exploration of the dyadic nature of the relationship pattern was a surprise and seemed to be engaging to her. The exploration of patterns during the early sessions was tentative and the therapist was able to sketch out some of these patterns to assist in keeping the sessions collaborative and open, and to demonstrate a sense of shared exploration of her experiences. This was a preliminary Sequential Diagrammatic Reformulation (which was developed into Fig. 23.4 over the course of therapy).

As well as making sense of the historical relational themes in her family, Madison was able to talk about her problems with her current boyfriend, which had precipitated the referral. Madison felt that their relationship had gone well for 2 years but, over the past 18 months, they had broken up and reconciled several times. She identified that she spent most of the time fearing he would leave, and therefore attempting to placate him, in the desperate hope that he would return her love and that she would feel 'perfectly' cared for.

Discussion explored how the second Reciprocal Role pattern (Hurting and Punishing—Hurt, Crushed, Alone) had been internalised and was often enacted 'self-to-self' (when Madison was overcome by the distress of feeling like a failure) or enacted by others to her (in response to her impulsive risk-taking). Examples included when she verbally abused herself for becoming angry or for breaking her diet or when she became angry and punishing towards her sisters or her mother or boyfriend. Madison discovered that she spent a lot of time feeling depressed and guilty and thinking about punishing or killing herself.

Madison came to understand that these patterns developed because, as a young child, she had been very sensitive to her mother's isolation and worries. She learned to please her mother and to try to protect and look after her younger sister. The family moved around so often that Madison became good at making friends quickly. However, she also learned to not trust others fully, waiting for something to go wrong or for people to reject her or let her down.

Madison also identified that when she felt that she wasn't living up to others' or her own expectations (e.g. to do well at school), she felt guilty and turned to self-punishment. She also became increasingly disillusioned and rebellious. It was easier to excel at being bad than being good. She also learned that alcohol and drugs took away her feelings and concerns quickly and efficiently, even if only for a short time. She also discovered that her increasingly rebellious and dangerous behaviour had unanticipated consequences because it elicited either greater control and restrictions (e.g. from her father) or rejection from others (e.g. teachers and some peers). It also led to her disengagement with school and this invited self-criticism about her lack of purpose in life.

These discussions and discoveries were jointly summarised in her prose 'Reformulation Letter' (Fig. 23.5), which was read aloud at session 4.

Madison missed the subsequent two sessions after the letter was read out. She later said that she had obtained a job after school and in the excitement had not thought of calling to cancel. However, this allowed a conversation about possibly feeling criticised by the letter and also about her needs and those of her therapist. This led to an agreement to attempt to contact to reschedule in the future.

The middle phase of therapy was spent exploring and detailing the patterns initially outlined, and developing a clearer understanding of how these had emerged and how they were enacted between her and others as well as with herself.

Madison was very focused on issues in the present such as her relationship with her boyfriend, completing her secondary schooling and her weight. She was able to engage in the process of monitoring the identified patterns, and to work toward devising new strategies. She was able to

Fig. 23.5 Excerpts from Madison's reformulation letter

Dear Madison,

We have started trying to understand how your feelings of sadness, anger and depression started. When we first met these were so consuming, you felt you couldn't go on and had tried to take your own life.

You remember moving around a lot as a child, following your father who worked in the army. Your family often had to stick together and were cut off from friends and relatives who could support you and your parents. You feel protective of your Mum, and know that it was tough for her looking after you and your sisters on her own for long periods of time while Dad was away. You feel that she tried hard to give you attention and care, but also you can see that she relied on you a lot. On the one hand this might have felt special but on the other, it also led to you expecting more and more of yourself. You tried hard to please Mum, to do the right thing and to be the 'perfect child' you thought she wanted. I guess that the more she relied on you, the harder you probably tried to be the 'support' that she seemed to need? When you couldn't always keep this up, or know what she wanted, you started to feel guilty and angry with yourself. It seems that you developed high expectations of what you should be able to achieve. Whatever you did, it had to be perfect and when it wasn't, you would be upset and angry with yourself. Perhaps this was your way of trying to manage the unpredictable world that constant moving around created. It also led to a feeling of almost constant dissatisfaction and unhappiness, because things were never good enough and you often blamed yourself.

As you grew older, you took on more responsibility for helping your Mum, and felt more and more guilty about having any needs of your own at all. Even when other people had hurt you, you covered this up feeling ashamed, blaming yourself.

By high school, you were feeling so trapped and unhappy, and you were sick of trying to be the 'good girl'. You started staying out and smoking dope, trying to take away those sad feelings and to feel you were in charge of things yourself – even though this also meant that things got worse. You felt that your parents were always criticising, blaming, and making unreasonable demands of you. You felt angry and thought that you might as well go and do all those bad things they accused you of! When you were 14, things finally seemed to snap. After a fight at school, you ran away and slept wherever you could for a week, mostly smoking dope with your friends. Your parents tried to pull you back into line. There were lots of arguments and you felt you had to fight and resist them.

You went to live with Tina and her family for 6 months, and there you felt more understood. There were some attempts to get you all talking more, and your parents let you know they loved you and wanted you back. You realised that things were not working out very well, and you worked hard to try to settle down. In particular, you stopped smoking dope as much and you felt a bit less angry. Then things changed for you again when Tina moved away.

You went home and tried to sort things out with your parents. You tried doing a course but then went back to school to do year 11. Most of this time, you felt down and that nothing could make you happy. You began bingeing when you felt upset. You would feel even more disappointed and guilty after these episodes. This made it harder to let people know you were upset and you got better and better at keeping it all locked inside. You also learned how to cut off from your feelings, to look from the outside like you were coping. You have become so good at this and others often don't really know how you feel. This keeps them out of your business, but it also means they can't support you either. By pushing others away, you stop them from being able to care and support you, even though this is actually often what you really want from them. It also means that you often go on feeling lonely.

It seems that all through the ups and downs of the last few years, your relationship with Will has been important. When you first started seeing him, he seemed so perfect. He was older and exciting and everything seemed so good. It felt like you were the centre of his world and this was just what you had been hoping for. After a few months, you began to feel that he wasn't always interested in you the way you wanted he to be. He wanted to spend more time with his friends than with you, and you felt overlooked and ignored. The more you asked of him, the more he pushed you away. So you tried bottling it up inside and not letting him know how you felt. This just led to more disappointment. When you broke up a year ago, you felt so devastated that you started to really punish yourself. As if this all meant that you were somehow a 'bad' person. Whether you do this by bingeing, harming yourself or bending over backwards to please others, none of these solutions lead to you feeling any better. Mostly they all lead to you feeling worse and more stuck.

Madison, it seems that while you did have some experiences of feeling cared for, by Mum and others, you have often found yourself feeling it is not enough, or hoping for 'more perfect' care from others. This frequently leads to feeling disappointed and let down when they cannot give you this. Similarly, your expectations of yourself are so high, that you are bound to feel 'let down' and disappointed with yourself. The solutions you tried were self-punishment and avoidance, but these make you feel depressed and haven't led you out of these vicious circles.

Regards,
(therapist)

Fig. 23.6 Excerpts from Madison's 'goodbye letter'

Dear Madison,

It seems to me that over the time that I have known you, you have been keen to sort things out better and to learn how to do this for yourself. I have seen you get better and better at letting people know what you think and what you need. You have been practicing how to be more assertive, and have been able to let me know when you were not sure where our sessions were headed, or when you thought we should talk more about a particular issue. You have also started to consider which friends treat you the way you want to be treated. These are very important skills that we all need to learn, and I feel confident that you can go on developing these skills into the future.

I said to you last week, that I feel this therapy is just the beginning. Not of a life of therapy, but a life of reflecting on what works for you and what doesn't. It is the beginning for you in lots of ways and this is bound to be both scary and exciting. I hope you can look back on this time as having been one in which you learned some skills that will help with this. There are still aspects of this work which may need more attention than others parts. For you, I wonder whether you still need to look out for your harsh Critical Voice, which tends to make little of your achievements and stops you enjoying the results of your hard effort? I hope that you can get better at turning this voice down so you can smell the roses a little more!

Madison, you have been very reliable, and thorough, and this tells me about how committed you are to sorting things through, even when this is tough. I know that you are a determined person, and that this will stand you in very good stead through the ups and downs ahead. I have also been very impressed by the strong caring side of you. You see injustice and things that are not right and want to do something about them. I think the world needs more people like you!

Last week we talked about the mixed feelings you have about finishing therapy. I too will miss our meetings and will look forward to the follow-up sessions to hear how things are going for you. I also know that it has been an achievement for you to complete this therapy, and I would like to congratulate you on doing well. I am sure that you will probably have other moments of doubt, sadness and even despair in the future. Nevertheless, I feel confident that you can overcome these.

I wish you all the best,
(therapist).

reflect and to consider what she wanted from the therapy relationship, and was able to accept being challenged by her therapist when she appeared to be avoiding a particular topic or issue.

As Madison gradually became more trusting, open and able to reflect upon the relationship patterns being enacted, her mood improved and her risk-taking decreased. She became more able to challenge her high expectations, especially about her school performance, and she became more open with what she wanted from others.

As she approached the final few sessions, she expressed some reticence about whether she would be able to manage after termination. However, she felt generally proud of her achievements thus far and reassured by her therapist's confidence that she could continue the work begun in therapy on her own.

One of the main challenges that Madison faced at the end of therapy was her difficulty applying her newly developed strategies to her frequent binge eating. She attempted to focus upon healthy eating, rather than dieting, and to take a more 'non-judging' and 'accepting' position in relation to her disappointment about her weight and body. By the end of treatment, her binge eating had reduced but not completely resolved and Madison still felt disappointed.

In the final session, her therapist read her a *goodbye letter* (Fig. 23.6) that summarised her therapist's view of Madison's progress through therapy. Madison declined the invitation to write her own *goodbye letter*, opting for a verbal discussion of her experience of the therapy in the final session. Madison was offered four follow-up appointments but only felt the need to attend two of these, at 1 and 2 months after termination and chose not to make the final two follow-up appointments.

Remaining Barriers and Potential Risks for Prevention and Early Intervention

Despite evidence of sufficient reliability and validity for the BPD diagnosis in young people,

stigma is a key lingering barrier to the early diagnosis of BPD in day-to-day clinical practice. BPD is highly stigmatised among professionals (Aviram, Brodsky, & Stanley, 2006) and it is also associated with patient 'self-stigma' (Rusch et al., 2006). This fuels the perception that the diagnosis is 'controversial' (Chanen & McCutcheon, 2008b) and clinical experience suggests that many clinicians will deliberately avoid using the diagnosis in young people with the aim of 'protecting' individuals from harsh and/or discriminatory practices.

While concerns about stigma are genuine and the response is well intentioned, we believe that this practice runs the risk of perpetuating negative stereotypes, reducing the prospect of applying specific beneficial interventions for the problems associated with BPD, and increasing the likelihood of inappropriate diagnoses and interventions and iatrogenic harm (such as polypharmacy).

There is now robust support for the early diagnosis of BPD. The UK National Institute for Health and Clinical Excellence (NICE) (National Collaborating Centre for Mental Health, 2009) and the Australian National Health and Medical Research Council (National Health and Medical Research Council, 2012) guidelines for BPD support the diagnosis of BPD in adolescents and the forthcoming ICD-11 is proposing to remove age-related caveats on the diagnosis of PDs (Tyrer et al., 2011). Moreover, the ICD will include the identification of subthreshold personality pathology. These innovations foster not only the early diagnosis of BPD but also the identification of subthreshold BPD, supporting the aims of indicated prevention and early intervention. However, this will bring into the clinical realm, young people (and adults) who might once have been considered 'colourful' and potential benefits are accompanied by potential risks associated with 'medicalising' common problems; risks that are not confined to the field of BPD (Mulder, 2008).

Conclusion and Future Perspectives

BPD should now be seen as a lifespan developmental disorder with substantial ramifications across subsequent decades. Consequently, intervention at any stage should aim to alter the life-course trajectory of borderline personality pathology, not just its diagnostic features. At present, there is sufficient evidence to support diagnosing and treating the BPD syndrome when it first appears becoming part of routine clinical practice. This has never actually been precluded in the DSM-IV but has been explicitly adopted by the NICE and NHMRC guidelines for BPD (National Collaborating Centre for Mental Health, 2009; National Health and Medical Research Council, 2012) and it is likely to be supported by the ICD-11. There are also data showing that targeting subsyndromal borderline pathology through indicated prevention is a promising and clinically justified approach and that the benefits of intervention appear to outweigh the risks. However, this approach requires further development and evaluation over longer periods in order to ensure that there are no significant 'downstream' adverse effects.

Indicated prevention and early intervention also offer a unique platform for investigating BPD earlier in its developmental course, where duration of illness factors that complicate the psychopathology and neurobiology of BPD can be minimised. This might make more sense of the confusing array of biological and psychopathological research findings in BPD.

In the future, a more detailed understanding of individual and contextual risk factors, precursors, pathways and mechanisms for the development of BPD might enable the development of universal or selective preventive approaches, but these are likely to require the joint effort of research groups aiming to prevent the range of major mental disorders. 'Clinical staging' (McGorry, 2010) for BPD, which is analogous to disease staging in general medicine, offers a potential integrating framework for selecting appropriate interventions and predicting outcome. A key implication of such an approach is that treatment needs will differ by phase or stage of disorder, and by socio-

cultural context (Kirmayer, 2005; Paris & Lis, 2013) with the possibility that interventions might be more benign and/or effective in earlier phases of BPD.

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Dialectical Behavior Therapy

24

Alec L. Miller, Mary T. Carnesale, and Elizabeth A. Courtney

Dialectical behavior therapy (DBT) is a comprehensive, multimodal psychosocial treatment originally developed by Linehan (1993a) to treat chronically suicidal adults diagnosed with borderline personality disorder (BPD). DBT has become the gold standard evidence-based treatment for the treatment of both suicidality and BPD (Linehan et al., 2006). There are more than 15 randomized controlled trials conducted at 12 different sites demonstrating that DBT is more effective than comparison treatments in reducing suicide attempts, non-suicidal self-injury (NSSI), inpatient hospitalizations, and other BPD symptoms while improving outpatient treatment compliance and numerous quality of life outcomes (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004; Linehan et al., 2006). Since the introduction of Linehan's treatment manual in 1993, DBT has been widely disseminated throughout the world where it is used in multiple therapeutic settings and adapted for many other patient populations including adolescents (Miller, Rathus, Leigh, Landsman, & Linehan, 1997; Miller, Rathus, & Linehan, 2007). Below we provide a review of DBT and the adaptations made for suicidal adolescents diagnosed with BPD (Table 24.1).

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Theoretical Foundations

The theoretical foundations of DBT—dialectical philosophy, Zen practice, behaviorism, and biosocial theory—provide the frame for treatment to target multiple problems and behavioral patterns. An understanding of these theories is necessary to conduct DBT effectively.

Dialectical Philosophy

In the process of developing the treatment for chronically suicidal adults with BPD, it became evident to Linehan (1993a) that an exclusive focus on change was too emotionally dysregulating and ultimately invalidating for patients. Alternatively, she learned that becoming solely acceptance oriented was equally problematic, as patients felt hopeless and/or invalidated by the lack of change orientation. Consequently, Linehan wisely synthesized the change-orientation taken from behavior therapy with the acceptance-orientation taken from Zen philosophy and Western Contemplative Practices to more effectively balance her therapeutic interactions, which resulted in patients both feeling better understood while also becoming more motivated to change. This dialectical synthesis of acceptance and change is most fundamental to

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Table 24.1 Randomized controlled trials for dialectical behavior therapy with adolescents

Authors	Subjects/setting/inclusion criteria	Design	Treatment	Outcome
Principal Investigator: Lars Mehlum M.D., Ph.D.	Setting: Outpatient	RCT	DBT: individual therapy, multi-family skills group, telephone consultation, DBT consultation team	At 16-week posttreatment individuals in DBT group had significantly greater reductions in self-reported and clinician rated depression, hopelessness, suicidal ideation, self-harm, and BPD symptomatology compared to EUC group
	Inclusion: history of deliberate self-harm and at least two BPD features		EUC: weekly individual therapy (CBT or psychodynamic) and supportive family therapy as needed	
	DBT: $n = 39$		Duration: 16 weeks	
	EUC: $n = 38$			
Principal Investigator: Emily Cooney, Ph.D.	Setting: Outpatient	RCT	DBT: individual therapy, multi-family skills group, telephone consultation, DBT consultation team	Individuals in DBT were more compliant with treatment. There were no significant differences between groups in reductions in emotion dysregulation, NSSI, and suicidal ideation
	Inclusion: history of suicide attempt or self-injury in the past 3 months		TAU: weekly individual therapy and family therapy as needed	
	DBT: $n = 14$		Duration: 26 weeks	
	TAU: $n = 15$			
Principal Investigator: Tina Goldstein, Ph.D.	Setting: Outpatient	RCT	DBT: alternating weekly family skills training and individual therapy, telephone consultation, pharmacotherapy. Skills were adapted for bipolar symptomatology	DBT group had significantly greater improvement in suicidality, nonsuicidal self-injurious behavior, emotion dysregulation, and depressive symptoms posttreatment compared to SOC group
	Inclusion: adolescents diagnosed with bipolar I, II, or mixed, who had an acute manic, mixed, depressive episode in the 3 months prior to study entry		SOC: pharmacotherapy, individual and/or family	
	DBT: $n = 14$		Duration: 12 months	
	SOC: $n = 6$			
Principal Investigator: Marsha Linehan, Ph.D.	Setting: Outpatient	RCT	DBT: individual therapy, multi-family skills group, telephone consultation, DBT consultation team	Study recruitment in progress
	Inclusion: suicidal ideation, more than one incident of self-injury or suicide attempt, and difficulties with emotional and behavioral dysregulation		ISGT: individual and supportive group therapy	

BPD borderline personality disorder, *RCT* randomized controlled trial, *DBT* dialectical behavior therapy, *CBT* cognitive behavioral therapy, *NSSI* non-suicidal self-injury, *TAU* treatment as usual, *EUC* enhanced usual care, *SOC* standard of care

DBT and led to the description of the therapy as DBT.

Dialectics provide both a worldview perspective on the fundamental nature of reality and the

framework for the therapeutic approach. According to dialectical philosophy, change is constant, there is no absolute truth, and apparent contradictions can both bear truth (Linehan, 1993a, 1993b; Miller et al., 2007). Dialectics provide a method to embrace and skillfully navigate conflict rather than refute and fight against it. Linehan (1993a) captures dialectics in treatment with the image of a patient and therapist on a seesaw. Both sides are constantly in flux as each member is sliding up and down until both try to move towards the middle to reach a balance. Balance is achieved through the synthesis of polarities, and with it are new truths and change.

Dialectical philosophy informs DBT interventions by emphasizing balance and acknowledging the truth in opposites with the use of "both/and" rather than "either/or" (e.g., I am doing the best I can, and I can do better; My mom loves me, and she sometimes says hurtful things). Dialectics provide strategies for balancing not only acceptance and change, but also flexibility and stability, challenging and nurturing, and deficits and capabilities. Finally, dialectics also involve movement, speed, and flow. This strategy suggests keeping the session moving and the patient off balance, so as not to get stuck or polarized with the patient.

Zen Practice

The technology of acceptance within DBT is derived from Zen practice as well as Western Contemplative Practices (e.g., Hanh, 1976; Watts, 1961). DBT focuses on acceptance and validation of thoughts, feelings, and behaviors as they are in the present moment. This is taught through mindfulness, which is the philosophy and practice of attending, nonjudgmentally, to the present (Kabat-Zinn, 1990). Mindfulness has been incorporated into a number of different treatment approaches, which have garnered empirical support (e.g., Marchand, 2012). Mindfulness is used to improve attentional control and increase awareness and acceptance of one's experience as it is, with the objective of ultimately reducing suffering and increasing the potential capacity for pleasure.

Behaviorism

Behaviorism provides the foundation for the technology of change within DBT. The conceptualization of problem behaviors is based on the principles of learning theory (for a review, see Goldfried & Davison, 1976). Specifically, behavioral chain analyses are used to understand the function of maladaptive behaviors, how they are maintained, and how they can be replaced with more adaptive behaviors. Problem-solving strategies, including skills training, contingency management, exposure therapy, and cognitive modification are the core change strategies, and specific solutions are generated to address problematic links in the behavioral chain. Behavioral principles are employed in individual therapy, skills group, and family sessions in an effort to reinforce adaptive behaviors and extinguish maladaptive behaviors.

Biosocial Theory

Linehan's biosocial theory (see also Chap. 11 by Crowell and colleagues, current volume for an update on the biosocial theory) provides a compassionate explanation of the etiology and maintenance of BPD. In this model, BPD is conceptualized as a disorder of the emotion regulation system that involves emotional vulnerability and an inability to regulate emotions. This is considered to be the outcome of a transaction between a dispositional emotional vulnerability and an invalidating environment. Unlike the diathesis-stress model, which maintains that there is an underlying vulnerability that is awaiting activation by an event in the environment, the biosocial theory is based on the idea that the individual and the environment transact in providing conditions for emotion dysregulation (Linehan, 1993a).

According to the developmental psychopathology perspective, the development of BPD is influenced by characteristics of the child, the caregiver, the environmental context, and transactions among these (Crowell, Beauchaine, & Linehan, 2009). Examining the emergence of problems during adolescence helps to understand

how biological and environmental risk factors contribute to different aspects of functioning and how the developmental trajectory differs among teens. Crowell et al. (2009) suggest that by mid-to-late adolescence, a constellation of identifiable features and maladaptive coping strategies are present and represent a heightened risk for the development of BPD. Other researchers have found that while some teens might be diagnostically subthreshold for BPD, they have comparable levels of impulsivity, suicidal thinking, self-injury, emotion dysregulation, and psychological distress to teens who meet full DSM-IV BPD diagnostic criteria (Brightman, Rathus, Ortiz, & Miller, under review).

Emotional Vulnerability

Emotional vulnerability is characterized by certain biological predispositions of the individual that may be genetic, neurobiological, and neurochemical (Linehan, 1993a; Miller et al., 2007). This includes a high sensitivity to emotional stimuli, increased emotional reactivity, and a slow return to emotional baseline. Individuals with high sensitivity to emotional stimuli have a very low threshold for an emotional reaction; they react quickly. Emotional reactivity is characterized by intense emotional reactions. Lastly, a "slow return to baseline" means that the emotions are long lasting rather than brief. In one recent study of adolescents, investigators found intense anger and affective instability were the most salient DSM-IV diagnostic features of BPD (Brightman et al., under review).

Neurobiological researchers have examined the construct of emotional sensitivity and reactivity and found that adults with BPD have differences in the volume and activity of brain structures related to emotion and impulsivity. Studies show that hippocampal and amygdala volumes, prefrontal lobe and prefrontal cortex, and the anterior cingulate gyrus (ACG) are measured to be smaller among patients with BPD compared to controls (for a review, see Bohus, Schmahl, & Lieb, 2004; Lis, Greenfield, Henry, Guile, & Dougherty, 2007; Schmahl & Bremner, 2006). Individuals with BPD also demonstrate increased activation in the amygdala when exposed to negative emotional stimuli (e.g., Donegan et al., 2003; Herpertz et al., 2001). This is notable as the amygdala is the center of the emotion memory system and plays an important role in the generation of negative emotions such as fear and anger. Differences in frontal lobe and prefrontal cortex are also significant because the prefrontal cortex deals with judgment, decision-making, impulsivity, planning, reasoning, and inhibiting aggression, and individuals with BPD demonstrate difficulty in these areas.

While most research has been conducted on adults with BPD, research has also been done with adolescents to assess volumetric abnormalities in the ACG and the anterior cingulate cortex (ACC). Studies show a decrease in volume in the left ACC in adolescents with BPD (Whittle et al., 2009) and decreased ACG volume in adolescents with BPD/MDD (Goodman et al., 2011). These findings suggest that neurobiological vulnerabilities may occur early in the developmental course of BPD.

There is also evidence that deficits in neurotransmitter systems including serotonin (e.g., Kamali, Oquendo, & Mann, 2002), and possibly dopamine, monoamine oxidase, and vasopressin are likely associated with the impulsive, aggressive, and self-injuring features of BPD (for a review, see Crowell et al., 2009). Crowell et al. (2009) also suggest that emotional lability may be accounted for by deficits in cholinergic and noradrenergic systems, which are sensitive to change and environmental inputs across the lifespan. Lastly, Crowell et al. (2009) propose that impulsivity is the earliest emerging trait for BPD and that there is a possible etiological overlap between BPD and other impulse control disorders including attention-deficit/hyperactivity disorder, conduct disorder, substance use, and antisocial pathology.

This research provides an explanation for some BPD symptoms and behaviors from a neurobiological, genetic, and developmental perspective. The impact of these vulnerabilities are related to deficits in emotion modulation including difficulties in: (1) inhibiting mood-dependent dysfunctional behaviors, (2) organizing behavior

in the service of goals, independent of current mood, (3) increasing or decreasing physiological arousal as needed, (4) distracting attention from emotionally evocative stimuli, and/or (5) experiencing emotion without either immediately withdrawing or producing an extreme secondary negative emotion (Linehan, 1993a; Miller et al., 2007). DBT treatment strategies aim to specifically target these emotion regulation deficits.

Invalidating Environment

Because not all emotionally vulnerable individuals go on to meet criteria for BPD, Linehan (1993a) also describes the necessity of a coexisting "invalidating environment" that in transaction with an emotionally vulnerable child/adolescent facilitates the development of BPD. An invalidating environment is one that responds inappropriately and erratically to the expression of private beliefs, experiences, and actions such that painful emotions and experiences are trivialized, misunderstood, punished, or dismissed. These environments may include family members, peers, teachers, coaches, therapists, and medical doctors (Miller et al., 2007). Sometimes an invalidating environment is a "poor fit" for the child because there is a discrepancy between the demands of the environment and the capabilities of the child (Thomas & Chess, 1985). This occurs in families experiencing different socioeconomic and life stressors, as well as in various school environments and communities. Additionally, while this may include experiences of physical abuse, sexual abuse, neglect (Herman, Perry, & van der Kolk, 1989; Zanarini, 2000), and bullying (Sansone, Lam, & Wiederman, 2010), invalidation also occurs inadvertently and within multiple contexts.

The primary characteristics of invalidating environments are that individuals are perceived as being over-reactive, unmotivated, manipulative, and/or undisciplined. For instance, a friend or parent might tell a teen, "you are over-reacting, it's not that bad," or "you are just doing this to get attention." Invalidating environments also emphasize controlling emotional expression (especially negative affect), are intolerant of displays of emotions, and oversimplify the ease of solving problems. For example, a teacher or parent may tell a teen, "just calm down, and let it go" or "you just don't care enough."

While invalidation may be inadvertent, individuals nonetheless receive the message that their emotional experiences are wrong or inappropriate. Consequently, the individual fails to learn when to trust his/her own emotional responses, and eventually may adopt a self-invalidating style and questions whether his/her own experience and interpretation of events are valid. Further, escalated emotional displays often become necessary to evoke a helpful response from the environment, and over time the child learns to oscillate between emotional inhibition and extreme emotional lability (Linehan, 1993a).

There are several significant consequences of being pervasively invalidated, including a failure to learn how to: (1) label private experiences, including emotions, in a manner normative in the larger social community, (2) tolerate distress or form realistic goals and expectations, and (3) trust one's own emotional and cognitive responses as valid. As a result, the emotionally vulnerable individual develops a skills deficit in which he/she does not know how to understand, modulate, and manage emotional experiences, and ultimately becomes self-invalidating.

Functions and Modes of DBT with Teens

DBT flexibly addresses multiple problems, while targeting suicidal and NSSI behaviors first and foremost, regardless of psychiatric diagnosis. An assumption of DBT is that effective treatment for multi-problem teens must be comprehensive and multimodal in order to address five specific functions. These functions include: (1) improving motivation to change and replacing maladaptive behaviors with adaptive behaviors to build a life worth living, (2) increasing capabilities and skills, (3) ensuring that newly acquired skills and

behaviors generalize to daily life, (4) structuring the environment to support clients and therapists when needed, and (5) enhancing and maintaining therapist motivation and capabilities for conducting effective therapy (Linehan, 1993a; Miller et al., 2007). Thus, as opposed to other forms of treatment delivered in a single modality, DBT includes multiple components to address these functions: individual therapy, multifamily skills group, intersession telephone coaching for patients and caregivers, family therapy, and therapist consultation team.

Improve Motivation to Change and Build a Life Worth Living

The goal of individual therapy is to improve motivation to change and reduce behaviors antithetical to a life worth living. The individual therapist is the primary therapist and functions as the "quarterback" of treatment and is responsible for: (1) assessing problem behaviors and skills deficits, (2) problem solving for these maladaptive behaviors to manage life-threatening behaviors and generalize skills, (3) organizing other treatment modes to address all problem areas, and (4) monitoring and ensuring progress towards treatment targets. Individual therapy is scheduled weekly for 50–60 minutes and is structured based on a four-stage model organized in hierarchical severity. Most research has been done on pretreatment and Stage I, which are discussed further below (see Linehan, 1993a for a review of Stages II–IV).

Pretreatment involves orientation and commitment to treatment with a focus on establishing a therapeutic alliance and defining primary target behaviors and goals for treatment. The primary focus of Stage I treatment is on stabilizing the patient and achieving behavioral control. Stage I treatment is conducted according to the following target hierarchy: (1) life-threatening behaviors (e.g. suicide, self-harm), (2) therapy-interfering behaviors (e.g., nonattendance, noncompliance, and hostile behaviors towards therapist), (3) quality-of-life-interfering behaviors (e.g., substance use), and (4) behavioral skills deficits. Sessions are organized based on this target hierarchy as reported on the teen's self-monitoring form—the diary card—that is completed daily and brought to all individual therapy sessions. The agenda for the session is structured in this way so that the highest priority targets—suicidal thoughts, behaviors, and NSSI—are addressed first. A significant portion of individual therapy is spent conducting behavioral chain and solution analyses of the teen's target behaviors. Individual therapy requires knowledge and application of all problem-solving and validation strategies, stylistic strategies (i.e., irreverent and reciprocal communication), case management strategies (i.e., consultation-to-the patient and environmental intervention), dialectical strategies, commitment strategies, and all other protocols (e.g., suicide risk assessment and management) (for a full review, see Linehan, 1993a; Miller et al., 2007). For an example of a therapist utilizing some of these strategies, please see the sample script in the Appendix.

Enhance Capabilities

The biosocial theory assumes that teens have not learned the skills necessary to effectively cope with their emotions and behaviors. Therefore, skill building is emphasized, and the teen and his/her caregiver participate in a weekly multifamily skills group to increase behavioral skills. This mode of treatment that differs from standard DBT in that skills group is not solely for clients. The purpose of involving family members is for parents to receive training in DBT skills to help manage the teen, reduce conflict at home, and serve as additional coaches to the teens (Miller et al., 2007). Rathus and Miller (in press) have described the application of DBT to teens and families in their soon-to-be-published DBT skills training manual for adolescents.

Multifamily skills group requires at least a 16–20-week commitment, and it is organized into five modules: mindfulness, distress

tolerance, interpersonal effectiveness, emotion regulation, and walking the middle path. These modules target five domains of dysregulation (i.e., emotional, behavioral, interpersonal, cognitive, and self-dysregulation) that were broadly derived from the DSM-IV BPD diagnostic criteria. For example, mindfulness targets dysregulation of the sense of self (i.e., not aware of your feelings, goals, and values) and cognitive dysregulation (e.g., depersonalization and dissociation). Distress tolerance targets behavioral dysregulation and impulsivity. Emotion regulation targets affective instability, and interpersonal effectiveness addresses problems with relationships. Rathus and Miller (2000) developed a skills module entitled "Walking the Middle Path," after applying standard DBT to teens and families and recognizing the need for additional skills to help families navigate their challenges more effectively.

Walking the Middle Path provides psychoeducation and skills regarding typical adolescent-family dilemmas, how to think and act dialectically, how to validate, and how to effectively apply behavioral principles. The specific dilemmas include: (1) making light of problem behaviors versus making too much of typical adolescent behaviors, (2) forcing independence too soon versus holding on too tight, and (3) being too strict versus too loose. This module addresses how to effectively manage these issues by teaching participants how to reduce extreme thinking, feeling, and acting, through learning dialectical thinking as well as managing cognitive distortions (e.g., all-or-nothing thinking, emotional reasoning). A key feature of dialectical thinking is employing "both/and" thinking instead of "either/or" thinking. Validation skills are taught and practiced so that parents and teens can learn how to both reduce invalidation while more effectively validating each other and themselves. Lastly, caregivers and teens learn behavioral principles (e.g., reinforcement, extinction, effective punishment, and shaping) in order to increase desired behaviors and reduce unwanted behaviors in themselves and others.

Ensure Generalization of Skills to Daily Life

There are three primary reasons for the adolescent to contact the therapist between sessions. The first reason is for intersession coaching, which is used not only to help the teen generalize new skills and behaviors to their natural environment but also to provide emergency crisis intervention (e.g., if the teen has self-harm urges). The primary therapist briefly assesses and coaches the teen on using DBT skills to solve a problem, or more likely, on how to tolerate distress *before* engaging in maladaptive behavior (e.g., cutting). For this reason, telephone consultation is available 24/7. Caregivers are also encouraged to utilize this coaching opportunity while they are attending the multifamily skills training group in order to effectively apply DBT skills when navigating challenging issues pertaining to their adolescent. Steinberg, Steinberg, and Miller (2011) developed guidelines related to telephone calls in DBT with teens and caregivers.

The second reason for calling the therapist is for clients to share "good news" in between sessions with the objective of breaking the link between maladaptive behaviors and therapist attention and allowing opportunities for shaping and positive reinforcement of incremental gains. The third reason for intersession telephone contact is to allow the teen and therapist to address any issues, misunderstandings, or emotional reactions he/she may have had with the other during the previous interaction without having to wait until the next session to resolve it.

In addition to telephone calls, individual therapy and family therapy sessions also contribute to skills generalization. Behavioral and solution analyses are conducted in individual therapy to understand target behaviors and instead apply skillful behavior both in and out of session. Family sessions are conducted as needed in response to family crises, if considerable emotional turmoil is centered on the home environment, and/or if a parent's behavior is reinforcing

dysfunctional behavior. Family sessions help with skills generalization, as family members can serve as skills coaches at home and by providing in vivo opportunities to practice skills.

Structure the Environment to Support Teens and Therapists

Because teens are usually still in their invalidating environments, DBT helps structure this environment to improve the treatment's effectiveness and be sure that others reinforce skillful rather than maladaptive behaviors. DBT intervenes with family members by including them in multifamily skills group and integrating them as needed into individual and family therapy sessions. Additionally, family members who participate in the multifamily skills group are permitted to call skills trainers as needed for coaching on how to implement newly acquired skills. Structuring the environment is also achieved through contacting providers of ancillary treatments, including psychiatrists and school personnel. The primary therapist often coaches the teen on how to present DBT to other providers; however, therapists may also need to facilitate communication to offer education and orientation to DBT strategies. DBT family therapy sessions are often scheduled after the teen and therapist have had a chance to develop their therapeutic alliance. The targets of these sessions are often to teach validation and other interpersonal effectiveness skills before moving into problem solving (Miller, Glinski, Woodberry, Mitchell, & Indik, 2002).

Enhance and Maintain Therapist Motivation and Capabilities

DBT assumes that effective treatment of BPD requires attention to both the teen's and the therapist's behavior and experience of therapy. As such, all DBT therapists are required to attend a weekly consultation team meeting. The goals of the consultation team are to improve therapists' motivation and capabilities for conducting effective therapy. As "therapy for the therapists," the consultation team addresses stress and burnout associated with treating suicidal and multi-problem teens as well as difficulties delivering adherent DBT. Consultation team members follow a set of team agreements that facilitate these goals. This is a critical modality of DBT treatment that can easily get short-shrift when practitioners, agencies, and resources are stretched. It is important to protect this mode just as one would the other modalities mentioned above.

Adaptations for Adolescent DBT

Several modifications to standard DBT were made to provide treatment that is developmentally appropriate and relevant for teens and their families (Miller et al., 2007; Miller, Rathus, Linehan, Wetzler, & Leigh, 1997). Inclusion criteria for most adolescent outpatient DBT programs include teens, ages 12–19, with histories of suicidal behavior, NSSI, current suicidal ideation, and BPD features (Miller et al., 2007). In the past 10 years, however, many adolescent programs have broadened the inclusion criteria for adolescents who have general emotional and behavioral dysregulation and who are not necessarily suicidal or have BPD features and is being applied in various settings, including outpatient, inpatient, residential, forensic, school, and medical settings. Comprehensive adolescent DBT has been divided into two phases of treatment. The first phase is comprehensive and includes all of the modes mentioned above, including the multifamily skills training group. Once a teen graduates from that group, they are eligible for the Adolescent DBT Graduate Group. The graduate group functions as a continuation/maintenance phase of treatment following the acute phase of treatment to assist the teen to strengthen capabilities and motivation and develop a sense of mastery.

The length of the initial phase of DBT treatment (typically 16–24 weeks) has been reduced from standard DBT (1 year) in an effort to increase the likelihood that teens will commit to

and complete treatment. As a result, fewer skills are presented in each module and the skills are presented in more depth (Miller et al., 2007). Lastly, the skills training handouts in the manual have been modified so that the language, terminology, and visual layout are more developmentally appropriate for teens. Moreover, additional skills handouts were developed to supplement the original material developed by Linehan (Rathus & Miller, in press).

Empirical Research

There has been considerable empirical support for the efficacy of DBT in treating adult women with BPD who have chronic suicidal behavior and NSSI. To date, 12 randomized controlled trials (RCTs) have demonstrated that DBT is more effective compared to treatment as usual (TAU) in reducing NSSI, suicide attempts, and other impulsive behaviors associated with BPD (Koons et al., 2001; Linehan, Armstrong, Suarez, Allmon, & Heard, 1991; Linehan et al., 2006; van den Bosch, Koeter, Stijen, Verheul, & van den Brink, 2005; Verheul et al., 2003). Given the strong evidence demonstrating DBT's effectiveness in treating suicidal adults with BPD, investigators chose to adapt DBT for suicidal adolescents, many of whom had BPD features.

DBT was initially adopted and adapted for use with suicidal adolescents by Miller and colleagues (Miller, Rathus, Leigh, et al., 1997; Miller et al., 2007; Rathus & Miller, 2002). Several randomized and nonrandomized studies have examined DBT's effectiveness in the treatment of adolescents with suicidal behavior, NSSI, and BPD features. In the first nonrandomized controlled pilot study for suicidal, multi-problem youth, Rathus and Miller (2002) evaluated the effectiveness of a 12-week DBT program. Individuals in the DBT group ($n = 29$) were compared to adolescents receiving TAU ($n = 82$), which comprises 12 weeks of individual supportive psychodynamic therapy and weekly family sessions. Inclusion criteria for DBT included one suicide attempt within the last 16 weeks and a minimum of three BPD features as measured by the SCID-II. Adaptations to the original Linehan DBT model for adults included the inclusion of families in skills training groups (multifamily skills group), revising the skills handouts to include examples more relevant to teenagers, and shortening the length of treatment to 12 weeks. Results indicated that individuals in the DBT group had a higher rate of treatment completion and fewer psychiatric hospitalizations, compared to adolescents in the TAU group. Posttreatment adolescents in the DBT group had significant reductions in depressive symptoms and in all four problem areas targeted in DBT (i.e., confusion about self, impulsivity, emotion dysregulation, and interpersonal problems). These promising results led to the further development of DBT for adolescents (Miller et al., 2007) which modified standard DBT to include a specific focus on commitment strategies, included family members in the multifamily skills group, and added a fifth skills module entitled "Walking the Middle Path."

In a study examining adolescents' self-report of the usefulness of DBT skills and their impact on BPD problem areas, adolescents reported statistically significant reduction in all four BPD problem areas as measured by the Life Problems Inventory (Rathus & Miller, 1995) after completion of a 12-week DBT program (Miller, Wyman, Glassman, Huppert, & Rathus, 2000). These salient findings suggest that after only 12-weeks, DBT reduced BPD features according to adolescent self-report measures. In a more recent study (Campbell, Rathus, Miller, & Smith, in press), adolescents and caregivers were surveyed after completing their DBT multifamily skills training and reported Walking the Middle Path Skills to be most helpful in assisting them in reducing their family conflicts.

Randomized Controlled Trials (RCTs) of DBT with Adolescents

Since the development of DBT for adolescents, four RCTs have been conducted or are underway. The first clinical trial was recently completed in

Oslo, Norway under the direction of Lars Mehlum, M.D., Ph.D. ("Long Term Efficacy of DBT-A in Adolescents With Repetitive Self-harming and Suicidal Behaviours"). Seventy-seven adolescents (12–18 years) with a history of deliberate self-harm who met two DSM-IV criteria for BPD were randomly assigned to 16 weeks of DBT or enhanced usual care (EUC). DBT treatment comprised weekly individual psychotherapy, multifamily skills group, telephone coaching, and family therapy, and pharmacological treatment as needed. The DBT treatment team met regularly for consultation meetings, expert DBT consultants provided monthly supervision and a reliable DBT adherence coder provided detailed written adherence coding notes for therapists and team leaders. Adolescents receiving EUC received 16 weeks of weekly individual therapy and supportive family and/or pharmacological treatment as needed. Both groups received similar dosage of individual therapy. Results from posttreatment assessments indicated that DBT appeared to be more effective than EUC at reducing deliberate self-harm, suicidal ideation, depression, and hopelessness (Mehlum et al., 2012).

Another RCT that evaluated DBT for suicidal adolescents with BPD features was conducted in New Zealand by Emily Cooney, Ph.D. and colleagues (Cooney et al., 2012). Twenty-nine adolescents with a history of a suicide attempt or NSSI in the past 3 months were randomly assigned to DBT or TAU. This study, which contained more methodological and procedural limitations than the prior study, found no significant differences between groups in reductions in NSSI, suicide attempts, emotion dysregulation, and depressive symptoms.

A third randomized trial evaluating DBT for adolescents diagnosed with bipolar disorder was recently conducted by Tina Goldstein, Ph.D. and colleagues at the University of Pittsburgh (Goldstein et al., 2012). This RCT was based on earlier pilot work evaluating and adapting DBT for bipolar youth (Goldstein, Axelson, Birmaher, & Brent, 2007). DBT was seen as a preferred treatment for bipolar youth given that emotion dysregulation is a crucial clinical feature in bipolar youth (Leibenluft, Charney, & Pine, 2003) and enhancing emotion regulation capacities is a primary target in DBT. Goldstein et al. (2012) conducted an RCT of DBT with 20 adolescents diagnosed with bipolar I, II, or mixed, who had an acute manic, mixed, depressive episode in the 3 months prior to study entry. Twenty adolescents were randomized to DBT ($n = 16$) and standard of care (SOC) ($n = 4$). The DBT intervention was delivered over 1 year and consisted of 24 weekly sessions of family skills training (single family), and individual therapy, with sessions alternating each week between these two treatment modalities. Treatment continued with 12 weeks of additional sessions after the first 6 months (monthly family skills training and individual therapy). Adolescents were also provided with telephone consultation. Treatment modifications were made from standard DBT for adolescents, by adding a bipolar disorder psychoeducation module, tailoring skills to specifically apply to bipolar symptomatology. Results indicated that individuals in the DBT group had significantly greater reduction in suicidality, NSSI, emotion dysregulation, and depressive symptoms at the 12-month time point compared to teens in the SOC group. It is important to note that subjects were not stratified based on suicidal behavior, with individuals in the DBT group engaging in more suicidal behavior at pretreatment compared to the SOC group. Despite these limitations, these findings support the feasibility and effectiveness of DBT with a pediatric bipolar population.

A fourth randomized trial of DBT with suicidal adolescents is being initiated by Linehan, McCauley, Asarnow, and Berk (2012) at University of Washington and UCLA, called "Collaborative Adolescent Research on Emotions and Suicide" (CARES). The inclusion criteria are adolescents (13–17 years of age) with suicidal ideation, more than one incident of NSSI or suicide attempt, and difficulties with emotional and behavioral dysregulation (BPD features). Adolescents are randomized to DBT or individual and supportive group therapy (IGST). Both treatment modalities include 6 months of

individual and group treatment and adolescent and parent assessments over a 1-year period. We await the results from this trial.

Adaptations of DBT to Other Adolescent Psychiatric Populations

Although DBT was originally developed to treat suicidal behavior and BPD, in recent years there is interest in the application of DBT to other populations and settings. Adaptations of DBT for adolescents (Miller et al., 2007) have recently shown promising results in treating adolescents with a broad range of psychiatric and behavioral problems such as bipolar disorder, eating disorders, oppositional defiant disorder, substance abuse, and juvenile delinquency (for a broader review of adaptations of DBT with adolescent populations, see Groves, Backer, van den Bosch, & Miller, 2011). One possible explanation for DBT's adaptability across a range of disorders is the transdiagnostic nature of emotion dysregulation (Ritschel, Miller, & Taylor, 2013). Transdiagnostic approaches suggest that emotion regulation difficulties are the central component in the development and maintenance of many psychological disorders. This conceptualization would suggest that interventions targeting emotion regulation deficits, such as DBT, are likely to be highly effective in improving individuals' abilities to better understand, tolerate, and regulate affective states and consequently help reduce secondary behavioral, cognitive, self and interpersonal problems.

Adaptations of DBT in Diverse Settings

Applications of DBT with adolescents in naturaliatic, residential (Sunseri, 2004), forensic (Shelton, Kesten, Zhang, & Trestman, 2011; Trupin, Stewart, Beach, & Boesky, 2002), and inpatient (Katz, Cox, Gunasekara, & Miller, 2004; McDonell et al., 2010) settings also show promising results. Sunseri (2004) reviewed outcome measures of 68 adolescent females receiving DBT. Within this sample, 85 % met criteria for BPD. Results indicated that DBT was effective in reducing premature terminations, defined as engaging in NSSI and then having to go to a psychiatric hospital and not subsequently reengaging in DBT treatment. In addition, DBT treatment was effective in significantly reducing the number of days adolescents were psychiatrically hospitalized due to NSSI and the length of time patients were held in restraints or placed in seclusion. This data supports that DBT is a feasible treatment for suicidal, multi-problem adolescents with BPD features in residential care settings.

DBT has also been adapted for juvenile offenders in correctional facilities (Shelton et al., 2011; Trupin et al., 2002). Trupin and colleagues compared two mental health correctional residencies, one in which offenders received DBT. DBT skills modules were taught to residences during 60–90 minute groups once to twice per week. A DBT consultation team for staff met weekly and milieu staff was available to provide skills coaching for residents as needed. Results indicated that over a 10-month period, suicidal acts, aggressive behaviors, and class disruption significantly decreased in individuals receiving DBT. A more recent study has evaluated DBT's effectiveness with incarcerated adolescent males (Shelton et al., 2011) by implementing DBT skills groups for 16 weeks in a correctional facility. Results indicated a significant reduction in aggression and in the number of disciplinary tickets. These encouraging findings suggest that implementing DBT in juvenile forensic settings is associated with a reduction in problematic behaviors.

Katz et al. (2004) successfully adapted DBT with adolescents in an inpatient hospital setting. They implemented DBT on an adolescent inpatient unit and compared treatment outcomes to suicidal adolescents on a TAU unit receiving psychodynamically oriented treatment. All adolescents recruited for the study were recently hospitalized for suicidal ideation or a recent suicide attempt. Individuals on the DBT unit received twice a week individual therapy that included review of diary cards and behavioral chain and solution analyses

and ten daily manualized skills group sessions. In addition, staff members created a DBT milieu on the unit, which included allowing patients to request and receive coaching from milieu staff and a weekly DBT consultation team meeting. Adolescents on the TAU unit received daily psychodynamic psychotherapy group and once a week psychodynamically oriented individual therapy sessions. At discharge, the DBT unit had a statistically significant reduction in behavioral incidents compared to the TAU ward. At the 1-year follow-up, both groups reported a statistically significant decrease in depressive symptoms, suicidal ideation, and NSSI. There were no significant differences between groups on these outcomes; however, the effect sizes were greater for DBT compared to the TAU group. A more recent study similarly implemented and evaluated DBT for youth in long-term, inpatient psychiatric care (McDonell et al., 2010) and found that individuals receiving DBT engaged in less NSSI, were prescribed less psychotropic medications, and had greater improvements in global functioning.

Adapting DBT for Children

Given that DBT has become the gold standard for treating suicidal adults and has been considered a promising treatment for suicidal adolescents, some investigators considered it timely to evaluate the effectiveness of DBT for children with suicidal behavior. Perepletchikova et al. (2011) adapted DBT for children by modifying handouts to include cartoons, larger font sizes, less text per page, and changing language to a second-grade reading level. Additional modifications included the development of new skills such as the "STOP" skill, aimed at building attentional awareness and decreasing impulsivity (distress tolerance), and the "Surfing Your Emotion" skill that teaches children to regulate emotional arousal (emotion regulation). The presentation of didactic materials has also been augmented by experiential exercises such as games, role plays, and multimedia presentations such as video cartoon clips to demonstrate skills. This adaptation of DBT for children was tested in a non-clinical setting with students from regular education classes ($n = 11$). At baseline, the children had mild to moderate symptoms of depression, anxiety, and suicidal ideation. The intervention included group skills training twice a week for 6 weeks. Parental and child reports supported the acceptability and feasibility of implementing DBT for children. In addition, at posttreatment, children reported significant decreases in depressive symptoms and suicidal ideation. Based on these findings, Perepletchikova and colleagues continue to develop DBT skills training for children, as well as adapting DBT for individual therapy with children, and developing a caregiver training modality (Perepletchikova et al., 2011). Currently, Perepletchikova, at New York Presbyterian Hospital is conducting the first randomized clinical control trail adapting DBT for children in a residential care setting (DBT-C res).

DBT in School Settings

Findings from the Substance Abuse and Mental Health Services Administration (SAMHSA, 2012) survey indicate that youth ages 12–17 are most likely to receive mental health care in educational settings, with an approximate 2.9 million children receiving mental health treatment in schools in 2010. Based upon these findings, implementing DBT in schools appears to be an important area to explore. Mazza, Dexter-Mazza, Murphy, Miller, and Rathus (in press) are publishing a DBT skills manual to be applied by educators in school settings based on their work with schools in Seattle as well as in several middle and high schools in New York. Some of the NY schools have employed Rathus and Miller's (in press) skills manual in applying comprehensive DBT to their high school students in an effort to retain some of their multi-problem youth within district. Catucci and colleagues found DBT to be promising at Ardsley High School at improving school attendance, and

reducing the number of disciplinary referrals by 50 % at school (Catucci, 2011; Mason, Catucci, Lusk, & Johnson, 2009).

Nelson-Gray et al. (2006) implemented a 16-week DBT skills group in outpatient and school settings for non-suicidal oppositional defiant adolescents and found promising results. Similarly, Hanson (2012) recently implemented a DBT program at Lincoln High School in Portland, OR with reportedly promising results. This school developed a DBT program for course credit that involves weekly skills groups, individual sessions, parenting training, and telephone consultation for adolescents. Although the results from this intervention have yet to be published, findings indicate that students report decreased anxiety, social stress, anger, and depression, and increased school attendance and GPA (Hanson, 2012). These authors wonder whether DBT may become a primary prevention intervention. That is, whether introducing the life skills of DBT to elementary school students (e.g., fourth and fifth grade) may help buffer emotionally vulnerable children from developing BPD.

Future Directions

To date, there are no empirically validated treatments for adolescents with BPD. Empirical support of DBT for this adolescent population has yielded compelling results that continue to warrant further evaluation. The largest randomized trial of DBT with suicidal adolescents with borderline personality features conducted in Norway suggests DBT is more effective than EUC at reducing many of the coexisting problems of adolescent BPD, including deliberate self-harm, depression, and hopelessness. Future research on the effectiveness of DBT components, mediators, and moderators is also needed to better understand the mechanisms underlying improvement within DBT. Mediational research will help understand the relative contributions of DBT strategies and their association with improvement in BPD symptomatology. Theorists (Chapman & Linehan, 2005) propose that the primary mechanism of change in DBT is the "reduction of ineffective action tendencies linked with dysregulated emotions." A recent research study with female adults with BPD supported this proposal with results indicating that DBT skills use fully mediated the likelihood of suicide attempts, the decrease in depressive symptoms over time, and partially mediated the likelihood of NSSI occurrence (Neacsiu, Rizvi, & Linehan, 2010). An important area of research would be to replicate these meaningful findings with suicidal youth with BPD features. Another remaining research question is whether DBT skills training alone, which may be effective for some less severely impaired adults (Koons et al., 2006) and oppositional defiant adolescents (Nelson-Gray et al., 2006), is effective for other adolescent populations. Randomized treatment component analysis studies are needed to evaluate the effectiveness and necessity of various treatment modes in DBT.

Fledgling research of DBT with youth in school settings as well as with younger children in outpatient settings is exciting and requires more controlled trials. DBT skills training in schools may be effective at primary, secondary, and tertiary prevention levels. From a primary and secondary prevention standpoint, might teaching DBT skills serve as a prophylaxis against vulnerabilities to BPD? And, for those already exhibiting signs and symptoms of BPD in childhood, would providing a comprehensive DBT program in schools be more feasible to help treat this condition earlier in life?

From a developmental perspective, childhood and adolescence are significant periods of vulnerability for the onset of BPD symptoms. There have been significant strides in demonstrating that DBT is an effective intervention for youth with BPD symptomatology. In addition, establishing preventive-focused DBT programs appears to be a promising avenue of research. We hope this chapter has offered a review of DBT for youth as well as raised some questions to guide the future directions of research in this arena.

Appendix: Sample Script

Patient is a 15-year-old Dominican female who presented to our DBT program following a recent suicide attempt. She presented with symptoms of depression, anxiety, oppositionality, and BPD. She has a history of intermittent suicidal ideation, NSSI, and significant school refusal. Her siblings are not currently living at home due to long-term psychiatric hospitalization and residential treatment.

Therapist: So do you think DBT is a treatment you'd like to do?

Patient: Yea, definitely. I went through the intake, I should do this.

Therapist: Really? It's a lot of work, and well, a lot of time. Are you sure you really can devote two afternoons a week to this therapy? (Devil's Advocate).

Patient: Yea, I can do that.

Therapist: Ok, I know that in your prior treatment experiences, it has been difficult for you to attend one session per week. How will you be able to do twice weekly sessions in DBT?

Patient: I guess I'll just have to come. I don't want to go back to the hospital or be like my siblings. My mom can help me too.

Therapist: Maybe we can talk about the pros and cons of doing DBT versus not doing DBT? What are the possible benefits as well as "cons" of doing this treatment? (Pros and Cons).

Patient: I guess the cons are that it takes a lot of time, and I might not always want to come. Also, I'll have to talk about things I don't want to talk about. The group could be boring too. The pros are that I won't have to go to the hospital, the skills might help me, and I guess it might get my mom off my back.

Therapist: Ok, great. When you say the skills might help you. . ..in what ways? Be specific.

Patient: It might help me feel less depressed and anxious and maybe feel better about going to school. And maybe even stop feeling suicidal and cutting myself.

Therapist: That sounds appealing to you if that all came to be. And how about the pros and cons about not doing DBT at all and just continue in your life the way you are now?

Patient: Well, some positive things are that I'll have more time because I won't have to come here and do the group. I could hang out with my friends, watch TV, or nap if I wanted.

Therapist: And the cons?

Patient: Well, I could end up in the hospital again, or I might end up hurting myself worse than before.

Therapist: Ok, it's really up to you then. If you look at these two things and try to balance out the pros and the cons, which weighs more?

Patient: Well, I don't know. I mean I have tried therapy before and nothing changed. I could just deal with it on my own.

Therapist: That's an option; you could try doing it yourself. How well has that been working so far though?

Patient: Not very well.

Therapist: I guess if you didn't do the therapy now, like you said, there's the chance that you will go back to the hospital again, miss more school, who knows maybe even have to go to residential treatment like your sister, or even worse you may end up dead.

Patient: I don't want those things. I guess I really should try this given all of the cons.

Therapist: Ok, so the treatment is 20 weeks, which is 5 months, and it is a 50-minute individual session per week plus a 2-hour skills group per week. Can you commit that you'll come to all of these appointments and do this treatment for the next 5 months?

Patient: Woah, 5 months. That's a really long time.

Therapist: That does feel like a long time, huh? OK what do you think is doable for you?

Be realistic. Could you commit to at least 15 weeks? (Door-in-the-face strategy).

Patient: That still feels long.

Therapist: How about ONLY 10 weeks then?

Patient: Well, 10 weeks is half. I guess that sounds better.

Therapist: And will you agree that during these 10 weeks you won't cut yourself, hurt yourself in another way, or try to kill yourself? Because the therapy won't work if you're dead (Irreverent communication strategy). So do you agree to take those ways of solving problems off the table?

Patient: Yes, I won't cut and I won't try to hurt myself. I can page you if I think about those things.

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Working with the Parents of Children and Adolescents with BPD

25

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Introduction

The pediatrician and psychoanalyst Donald Winnicott said: "There is no such thing as a baby, there is a baby and someone." This statement captures the reality that a human baby cannot exist on its own. To see the infant as an individual completely separate from its caregiver misses the essential nature of the child's utter dependence on another person.

In a similar sense, there is no such thing as a borderline child or adolescent. As we read earlier (Chap. 11), genes account for about 60 % of what makes up BPD and the environment the other 40 %. The interplay between family and child or caregiver and child is always a factor in the pathology of BPD and, as such, families are critical to the healing process. It is not the child alone who needs to heal but the entire family.

Family therapy often seems to be a good idea in working with the BPD child and its family. It is not just the adolescent who is suffering; typically the entire family is living with the disruption and pain that accompanies the symptoms of BPD. Mental health professionals almost reflexively prescribe family therapy, and yet standard family therapy can make many of the behaviors that are seen as needing to change much worse. How is it that standard family therapy can make the situation worse and what can be done about it?

Family therapy can be a high risk/high gain strategy for families who have a child with BPD. Unless carefully conducted, the treatment can have the unintended effect of increasing many of the self-destructive behaviors that are core to BPD. On the other hand, when carefully constructed, family therapy can reduce these self-destructive behaviors, increase effective family functioning, and teach broad skills that go beyond simply dealing with BPD. The aim of this chapter is to describe the family-based work we do at the McLean Hospital with adolescents with BPD and their families. The broad approach is based on the work of Dr. Alec Miller and colleagues (2006) who adapted dialectical behavior therapy (DBT) for adolescents. Below, we describe the approach. First we review what the difficulties were with historical approaches to working with BPD adolescents. Next, we outline the broad goals of family therapy including psychoeducation and a more explicit look at the various behaviors commonly exhibited by adolescents with BPD and their families. We then move to what the actual structure of the family work looks like before and conclude the chapter with a call for the need for more research into this critical piece of the therapeutic process.

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Problems with Standard Family Therapy

Of all the psychotherapeutic modalities, family therapy may be the one that requires family members to be most skillful in managing their emotions. There is a minimum of three people in the room; the child, the therapist, and the parent. Frequently, there are two parents and sometimes a sibling as well. Occasionally, an individual therapist might sit in. Having so many perspectives during often-heated conversations can be hard to manage. They are bound to evoke powerful feelings in all the participants, especially the adolescent. As a group, adolescents with BPD, especially if they self-injure, are well known to have poor capacity in managing and modulating their emotions effectively (Klonsky, 2009; Klonsky & Muehlenkamp, 2007; Nock, 2010; Walsh, 2006). In fact, self-injury may be one of the most effective strategies these teens have for managing intense emotional experiences.

Consequently, unstructured, traditional open-ended family therapy is often too emotionally intense for the adolescents to manage. All too often, they storm out of the room or stay put, folding their arms across their chest and shut down emotionally. While BPD behavior may seem ineffective from one perspective, it makes perfect sense from another. If a child learns that screaming at the top of his or her voice is an effective way to get a parent to back down, the child will continue to do this. Coauthor Dr. Michael Hollander, PhD, has written, "Family therapy for BPD adolescents is akin to teaching someone to swim by throwing them out of a boat in the middle of a deep lake. Some people would quickly give up trying to get to shore and just go under, like our emotionally shut down clients. Others would flail about for the longest time, exhausting themselves unproductively, like our clients who exhibit intense emotional displays. In either case, learning how to swim spontaneously is not likely to be the outcome" (Hollander in Walsh, 2012).

In order for a family therapy to be effective, adolescents with BPD and their parents have to be able to tolerate intense emotions without any avoidant or escape behavior. Often we are asking these families to do something they don't yet have the skill to do.

Parents often come to treatment feeling worried, annoyed, afraid, ashamed, angry, and guilty. Healthy interpersonal development for the child, such as independence and autonomy, has usually been derailed by BPD behavior. The family therapist must have an expert understanding of BPD and how the symptoms of BPD are transactional in nature. Without this understanding, the therapy blows up and all participants become frustrated, hopeless, and despairing. In many cases, adolescents will refuse to do family therapy because they feel that they have done it in the past and it either "did not work" or "made the situation worse." Their perspective often has more than a grain of truth to it. On the other hand, there are some very compelling reasons to prescribe family therapy.

Goals of Family Therapy for BPD

Family therapy for BPD has three overarching goals:

1. Assessment of family functioning and psychoeducation about BPD that includes all the family members and orients them as to the function of behaviors commonly seen in BPD.
2. Decreasing the behaviors within the family that erode family functioning, such as contextually inappropriate emotional displays, invalidation, and emotional avoidance.
3. Increasing behaviors that lead to more effective family functioning, such as age-appropriate and normative roles, validation, and curiosity about others' behaviors and mind states. In order to best achieve these goals it is often useful to think of stages of the family work. The first stage is assessment and psychoeducation. These goals are clearly visible in the family therapy session script provided in Appendix.

Assessment and Psychoeducation

Generally, two or three sessions are required to conduct a thorough assessment of family

functioning and to begin to educate the family about BPD. Families need to be oriented to what will be expected of them and to how the work is going to proceed. It is a time for developing a consensus about the issues to be addressed and the hope for goals. Family members should know that each member's current capacity to do family work will be assessed. In addition, the members should be helped to understand that as important as family work is, other interventions might be needed before family treatment can be pursued. For example, it may be the case that the parent of a BPD child notices BPD symptoms in himself or herself and the parent may need his or her own individual therapy. At the end of the orientation, family members and the clinician should have a good sense about whether family therapy is indicated at this point in time, what is going to be discussed in treatment, and what changes the family can expect (Hollander in Walsh, 2012).

In addition, the therapist uses this period to help family members understand the functions that BPD behaviors like self-injury play in the child's life. Psychoeducation about BPD will help modulate parental worries and support family members in gaining a more compassionate understanding of their child's struggles. It is critical that the therapist finds the "wisdom" in all the behaviors exhibited by child and parents and to help all the family members understand the reasons behind the various behaviors. This is *not* an endorsement of the behavior, but rather a way of making sense of why family members are doing what they are doing. At the end of the assessment the therapist and family may decide that they are ready to enter formal family therapy, or the therapist may recommend that the family spend time developing the emotion regulation and interpersonal skills that will be critical to successfully do family treatment. For instance, the therapist may suggest that the first several months of treatment be primarily focused on helping the family—as a unit or individually—acquire the skills taught in DBT.

Themes in Psychoeducation

There are some common themes experienced by families of children with BPD, and psychoeducation involves not only education about the disorder but also understanding the types of behaviors and feelings that are generated by the often-conflictual interactions. The following is an overview of some common BPD themes and behaviors that may be reviewed when educating the family about the disorder.

Parental contribution to the problem. Parents of children with BPD often ask themselves and their clinicians if they are to blame, or what their role was in their children having BPD. There is little literature on how family interactions with a child lead to the development of BPD and in fact, few methodologically sound studies have even been conducted to research family relationships and BPD (but do see Chap. 17 for an emerging literature on this topic). Various researchers have tackled the question and described types of parental dysfunction associated with the development of BPD. Zanarini et al. (2000) developed the concept of "biparental failure" finding that people with BPD were significantly more likely than Axis I controls to report having caretakers of both sexes deny the validity of their thoughts and feelings, fail to provide them with needed protection, neglect their physical care, withdraw from them emotionally, and treat them inconsistently.

An earlier paper, Norden, Klein, Donaldson, Pepper, and Klein (1995), characterized the relationships of people with BPD with their parents as poor and that they were more likely to have been sexually abused. Paris and Zweig-Frank (1997) also reported higher levels of early abuse but also cautioned that abuse itself did not explain BPD, and that many of the control subjects who had been sexually abused did not go on to develop BPD. Finally, Fruzzetti, Shenk, and Hoffman (2005) describe family interactions that are "invalidating, conflictual, negative, or critical," in their transactional model of the development of BPD, one consistent with Linehan's biosocial theory (1993).

What parents need to know is that the few studies on the matter show that there appears to be early invalidation and at times abuse (see Chap. 16) in many children who have gone on to develop BPD. It is also true no parent wants to have a child with BPD, and that at times the nature of early invalidation is less an act of commission and more a function of lack of awareness and knowledge about BPD.

Regardless of the parents' possible contribution to the etiology of the patient's BPD symptoms, family treatment should focus less on meaning making and searching for "causes" of the patient's dysfunction, and more on here-and-now interactions and the transactional nature of his or her difficulties. The therapist needs to keep in mind that the parents have also been influenced and behaviorally shaped by their child—it is very much a two-way street.

Self-injury. Self-injury in various forms is common in BPD and there are very few experiences that are more frightening, confusing, or worrisome to parents than when a child self-injures. When one of the intrinsic roles of parenting is to protect a child and prevent harm from occurring, the self-injury by a child challenges the parents to do everything in their power to act and prevent ongoing harm. In many cases parents who have a child who self-injures experience intense levels of guilt and shame. The guilt can be that of self-blame for the behavior or that perhaps they should have been aware of it sooner. Shame can come from feeling that family members and friends are judging the parenting as inadequate, or taking a child into social settings where exposed scars identify the child as "troubled." It is important that self-injurious behaviors are spoken about in an open and direct fashion. Trying to protect the parent from feelings of shame by not discussing self-injury puts the child at great risk, as those who self-injure have an approximately 30-fold increase in suicide over those who do not (Cooper et al., 2005).

Suicidality. Parents need to know that suicide is a serious complication of BPD and research shows that personality disorders are estimated to be present in more than 30 % of people who commit suicide, in about 40 % of people who make suicide attempts, and in about 50 % of psychiatric outpatients who die by suicide (American Psychiatric Association, 2003). In clinical populations, the rate of suicide in BPD is estimated to be between 8 and 10 % (Black, Blum, Pfohl, & Hale, 2004), a rate far greater than that in the general population. Parents also need to know that since 60–70 % of patients with BPD make suicide attempts (Gunderson, 2001), unsuccessful attempts are far more frequent than completed suicides in this population. In adolescent inpatients, BPD has also demonstrated an incremental contribution to risk for suicidal behaviors above and beyond depression (Sharp, Green, Venta, Pettit, & Zanarini, 2012).

Emotional dysregulation. Central to BPD in adolescents is the difficulty they have in regulating their emotions (see also Chaps. 9 and 13). This does not simply mean that the adolescent has high emotional sensitivity and reactivity, but rather combines the following elements (Gratz & Roemer, 2004):

- A lack of awareness, or understanding, or capacity to identify or accept emotions
- A developmental lack of adaptive strategies for regulating the intensity and/or duration of an emotional response
- An inability or unwillingness to experience emotional distress as part of pursuing desired goals
- The inability to engage in goal-directed behaviors when experiencing distress

For parents to understand that emotion regulation difficulties are a function of the disorder and have transactionally evolved over time, rather than being an act of premeditated and intentional choice, can help them develop more compassion for their child as the work progresses.

Relationship with siblings. Siblings can further complicate the picture. Parents often struggle with how to explain BPD behavior to the other children in the family. Parents understandably worry about scaring younger siblings; however, pretending that "all is well" can lead to confusion and unspoken fear. Older children can become judgmental and impatient with their BPD sibling. They may condemn their sibling's behavior as

well as their parents' management of it. They may accuse the BPD child of taking all the family's time and financial resources. Alternatively, older kids can become overprotective of their BPD sibling and in so doing unwittingly reinforce the dysfunctional behavior. The therapist needs to determine at what point siblings should be brought into sessions in order to educate them, give them a voice in the process, and ensure that they can support more functional behavior in all family members. Generally this will be after the child with BPD and the parents have had an opportunity to strengthen their communication and be more skillful with one another. Therapists need to be flexible in their approach with siblings, given the wide age range and varying relationships. It is often useful to meet individually with siblings in order to assess their needs and to pitch the psychoeducation to their developmental level.

Estrangement. BPD adolescents not uncommonly feel lost and estranged from their families. They frequently feel a sense of defectiveness and a belief that they are a disappointment to their parents, leading them to withdraw from family relationships. The shame and guilt surrounding their behaviors and their worry about parental response often lead them to avoid seeking support when they need it most. They can also blame their parents for the very behavior the parents are trying to manage.

Adolescent and family secondary targets. The work of Alec Miller, Jill Rathus, and Marsha Linehan on pervasive dysfunctional family patterns is a helpful tool in evaluating family difficulties and providing guidelines for resolution (Miller, Rathus, & Linehan, 2007; see also Chap. 22). They posit three dialectical dilemmas common to families with suicidal and borderline adolescents. One goal of treatment is to help the family achieve resolution of the following problematic patterns, which usually involves moving toward the center of the dialectical extremes. These dialectical dilemmas are an essential piece in both psychoeducation and assessment of the family, and families will frequently recognize their own tendencies as the following are elucidated:

The three dialectical dilemmas are as follows:

1. Authoritarian control vs. excessive leniency
   Authoritarian control means keeping very tight reins on all of the child's behavior, at times using coercive methods to maintain control. When this happens and especially if paired with quick attempts by the parent to solve what they see as problem behavior, the adolescent may feel intensely misunderstood and invalidated. When this occurs, the likelihood is that emotional dysregulation increases and that the interaction between child and parent devolves into an emotional storm. Parents whose understanding is inaccurate, especially when under emotional distress, may search for confirming evidence to support their view, closing their minds to what their child is saying. When the conflict between parent and child endures, it not only takes a toll on the adolescent–parent relationship but can lead to increased tension between parenting partners as well, further increasing family dysfunction. On the other hand, excessive leniency means that the parent does little if anything to manage the child's behavior and allows the child to do almost anything. This degree of permissiveness is at odds with the developmental imperative that a child be in the position of having to face restrictions, and then dealing with consequences of defying the rules or having to solve the problem or deal with difficult emotions.
2. Pathologizing normative behavior vs. normalizing pathological behavior.
   Pathologizing normal behavior is the act of labeling typical adolescent behavior as "wrong" or as something that needs to be treated. For instance, a parent may feel that a child's anxiety about going to school is a serious problem that needs immediate psychiatric attention. Sometimes parents are so worried about their child that they see "problems" in everything their child does.

   Normalizing pathological behavior, on the other hand, is the act of seeing all of a child's behavior as typical and normal. For instance, whereas it may be normal for an older

adolescent to experiment with drugs, smoking marijuana on a daily basis and being unable to attend to his or her schoolwork and family obligations because of this is not normative.

3. Forcing autonomy vs. fostering dependency
When parents push their child to do more than he or she is developmentally or constitutionally capable of, they are forcing autonomy. Parents who thrust excessive responsibility on an adolescent, or who hasten separation and self-sufficiency, regardless of the adolescent's capacities, fall into this category. Fostering dependency is the opposite extreme of stifling the adolescent's natural developmental strivings for independence. This also plays out with parents who over-function in the caretaking role, intervening in the world for their teens and depriving the adolescent of the experience and practice of negotiating on his or her own behalf.

Decreasing the Behaviors Within the Family That Erode Family Functioning

It is often good for the therapist to begin the assessment by having family members speak about the strengths in their family. Doing so helps the family start on a more balanced and hopeful note. It also keys the clinician to areas of resiliency within the family. During this assessment, it can be very helpful to explore times in the family's history when they functioned more effectively and how it was that they were able to do so. The therapist should assess each family member's capacity to *validate* and to understand the perspectives of others. Validation is the recognition that the experience of another is valid. Validation does not endorse or accept behavior; it simply recognizes that the behavior, which includes individual emotional experience, makes sense given the circumstances. When a family member feels validated, his or her distress level usually goes down. When this happens, the person is far more capable of understanding life's problems and potentially applying more effective solutions. Validation is listening empathetically and with authentic curiosity.

Invalidating statements that are blatantly hostile or judgmental comments should be blocked by the therapist and reframed in more behaviorally specific terms. A statement like "she is shaking her leg" is something that all in the room can see and is more effective than "she is trying to distract me by fidgeting." Such reframing should be neutral while addressing the speaker's emotional state.

Generally speaking, hostility and negative judgments arise from hurt feelings, worry, or anger. When these moments arise, the therapist will have the opportunity to get a read on each family member's capacity to tolerate distress, to regulate his or her emotions, and to be interpersonally effective (Linehan 1993). If the family has real trouble with validation, distress tolerance, and emotional regulation, it is likely that some skill building will need to happen before family treatment proper is initiated.

The therapist should assess whether guilt and shame have become debilitating and are compromising effective parenting skills. When this happens parents can be overwhelmed by uncertainty, helplessness, and inaction. Alternatively, some parents' emotional distress can lead them to take on a far more rigid stance. They move from helplessness to "take action," becoming certain about the motivations and intentions of the child. Unfortunately, even when parental understanding is accurate, the manner in which parents present their ideas is often very problematic as it is too often short on validation. When this occurs the adolescent may have real trouble accepting parental advice.

The therapist needs to help families understand that anger can and will appear in family work, but that unbridled anger can have particularly deleterious effects on the process. It can not only disrupt the therapy by shutting down communication but also potentially induce fear in other family members.

Excluding a Parent from Family Work

Although it is very rare, there are situations when one parent might be excluded from a family therapy. For example, one parent may be ready and capable while the other parent may be emotionally, behaviorally so easily dysregulated or

cognitively limited that their presence in family therapy disrupts effective therapy and prevents learning to take place. In situations where the parent is so enduringly devaluing of the child, the therapist, or the other parent, that cost to the treatment outweighs the possible benefit, the recommendation could be to exclude the parent.

Any exclusion of a parent should be done after consultation with other treatment team members, after clear and direct communication with the parent, and with clear instruction as to how the parent can rejoin family therapy. Given that the genetic load of BPD appears to be about 60 % it is not unusual that parents have traits of BPD. Most parents benefit tremendously from parent psychoeducation and family work, and many use the opportunity to get into treatment themselves. Again, it is incumbent upon the therapist to be clear, direct, and transparent when making these recommendations.

Structure of Family Therapy

The Pretreatment and Commitment Stage

Once psychoeducation and family assessment is complete and the broad goals of family therapy have been reviewed, the next step is a collaborative development of clearly defined, narrower, and individualized goals. The clinician should work at gaining consensus as to the shape and form of the problems. The clinician needs to be particularly active in this phase of the treatment and highly vigilant for signs of emotional dysregulation. When that happens, the therapist's interventions are aimed at articulating and clarifying the wisdom in each family member's point of view. The skill for the therapist is to stay empathically validating of each person in the family.

Sometimes parents present as so anxious that they come across as "needy," helpless, and confused, wanting the therapist to "fix" their child, without believing that they can be any part of the solution. It may be very difficult for the therapist to tolerate such helplessness on the part of a parent. It is essential however that the clinician finds compassion for each participant; without this capacity, the clinician cannot formulate interventions that will enhance *family* functioning. In those instances when a therapist encounters difficulty generating compassion, or notices that he or she is becoming emotionally dysregulated himself or herself, it is useful to get a consultation from a colleague or another team member.

The family therapist needs to be comfortable being active at managing the treatment hour so as to minimize the effects of emotional dysregulation yet also knowing when to let things play out. It cannot be emphasized enough that it is incumbent on the therapist to find the wisdom in each participant's perspective and to give clear voice to it while not validating ineffective behavior. This is easier said than done. It is often difficult for clinicians to find and maintain compassion and empathy for each family member and his or her point of view. Therapists with children of their own may be more aligned with the parents in the room. Younger therapists and therapists without children may be more in tune with the adolescent's perspective. Sometimes therapists are too focused on the adolescents' distress and at other times therapists can find the adolescent behavior so reprehensible that they ally with the parents' perspective. These common clinical pitfalls are best resolved through the use of consultation.

After the goals of therapy have been agreed upon, the next step is for the therapist to obtain a firm commitment from each participant about the work ahead. All too often therapists skip this part of the treatment because they assume that everyone has a commitment to the family work. The commitment should be clearly delineated, explicit, and structured. The commitment should focus on attendance, doing homework, and working toward the shared family goals. There should be agreement as to who will attend family sessions. At times it may involve the child and one parent, at other times the child and both parents, and yet at other times the child and his

or her sibling or siblings. There can be many configurations. Mostly though, it will be the child with BPD and one or both parents. Siblings can be asked to join on an issue-by-issue basis.

It is useful to plan regular reviews about the treatment. If the treatment is going to be ongoing and weekly, a review every 10 or 12 sessions is a reasonable time frame. The function of the review is to monitor progress toward goals and to introduce new goals if needed.

In many cases the family therapist is not the individual therapist for the adolescent, and as such a plan for communication between the family therapist and individual therapist needs to be established. This insures that critical information that could influence the family therapy is known. The two therapists need to decide together what information will be shared, and this agreement needs to be communicated to the patient and the family.

Family Therapy Proper

If the family as a whole seems to have sufficient capacity to manage family therapy, then treatment can proceed. The following are guidelines to help the family therapist organize the complex and often perplexing aspects of family work.

1. The therapist should work at establishing (or reinstating) age-appropriate roles for parent(s) and child. Faulty parenting is frequently cited as a major causal factor in the development of child psychopathology (Goodman, 2003; Newman & Stevenson, 2005), and on the other hand parents often say that they "if only there were a manual for parenting." The concept of faulty parenting is not only poorly defined but also rarely takes into account parental depression, physical abuse, poverty, single parenthood, parental substance and alcohol abuse, or simply lack of knowledge and skill in the face of worrisome and dangerous behaviors on the part of an adolescent with BPD.
2. The family therapist needs to assist the parents in establishing or reinstating normative family roles and tasks. The American Academy of Child and Adolescent Psychiatry published the following list "Facts for Families (American Academy of Child and Adolescent Psychiatry, 2012)". These may be useful prompts for structuring some initial discussions in family therapy.
   - Providing a safe and loving home environment
   - Creating an atmosphere of honesty, mutual trust, and respect
   - Allowing age-appropriate independence and assertiveness
   - Developing a relationship that encourages your child to talk to you
   - Teaching responsibility for their belongings and yours
   - Teaching basic responsibility for household chores
   - Teaching the importance of accepting limits
   - Teaching the importance of thinking before acting
3. The therapist helps the family learn what defines typical adolescent behaviors and recognize what behaviors "cross the line." The therapist then works with parents to establish clear rules and the need to enforce them in a predictable fashion which has to be balanced with the willingness to negotiate on some issues. These negotiations should take into account the adolescents' readiness to manage increased freedom and responsibility. It is useful in this context to refer back to the Adolescent Secondary Targets covered earlier in the chapter. The therapist can also help the family to understand and have compassion for aspects that are often challenging for an adolescent with BPD, such as deficits in the capacity for thinking before acting. Once there is a shared view of the problem, families are better able to support a compromised family member. Parents can get confused by what is BPD behavior and what is normal adolescent development. Distinguishing between these can be difficult, and so the clinician needs to have a clear understanding of typical adolescent development. For example, as the child

moves toward a more consolidated sense of self, she can struggle with a sense of identity, feel awkward about her body, and be powerfully influenced by her friends and peer group. It is easy to see how these behaviors could be confused with the third criterion of BPD in the DSM; identity disturbance as evidenced by marked and persistent unstable self-image or sense of self. However, the educated therapist will take into account the age and the degree of the behavior in determining whether it has crossed the line from normal development to pathology. In this example, one would expect to see this kind of behavior in younger adolescents, but not in older individuals.

Research on Family Work

Much of the research in family work in BPD has been done by the National Education Alliance for Borderline Personality Disorder (NEABPD) an advocacy group that developed Family Connections, a 12-week manualized education program for relatives of people with BPD. Different from clinician-led treatment, Family Connections is led by trained family members, and is based on the strategies of standard DBT and DBT for families. Similar to the goals of family work as outlined above, the Family Connections program provides (a) psychoeducation on BPD, (b) coping skills, (c) family skills, and (d) opportunities to build a support network for family members. Their findings indicate that families who completed the course showed significant reductions in grief and burden, and a significant increase in mastery from pre- to post-group assessment and that these gains were maintained at 6 months post-baseline (Hoffman et al., 2005). These findings were replicated by a Swedish group (Rajalin, Wickholm-Pethrus, Hursti, & Jokinen, 2009) who found that the Family Connections intervention led to a significant reduction in burden, improved psychological health, and an increase in well-being regarding the relationship with highly suicidal patients. A 2007 replication study of the Family Connections model by Hoffman found that participants showed significant improvements on all well-being variables, including significant reductions in depression (Hoffman et al., 2007).

Future Research

Although BPD is the most researched of the personality disorders, it does not receive the funding of other major mental illnesses. Research on family work in BPD is essentially nonexistent and the need to develop and research models of family therapy that lead to better outcomes is needed.

Conclusions

In summary, family therapy is critical in the treatment of child and adolescent BPD. The following are guidelines for clinicians:

- In most circumstances, family therapy includes the parent(s) and the BPD youth. Siblings and extended family may attend on occasion to address specific issues.
- Understand that not all families are capable of using the treatment immediately or effectively. Family members who are prone to explosive emotion dysregulation may require interventions such as individual DBT in order to become ready for family treatment.
- Family therapy begins with validation, assessment, and psychoeducation about BPD.
- Family therapy should be structured, with clearly defined and mutually agreed-upon behavioral goals.
- Family work should not be aimless or "waiting for something to come up."
- Each session should work from an agenda. Generally, the behaviors that are most likely to cause damage, behaviors such as suicide and self-harm, should be prioritized. Next would be the family behaviors that threaten or interfere with the therapy.
- Family therapy can be among the most emotionally activating of psychological treatments. Asking an adolescent and his or her problem behavior be the focus of extended discussion can generate a lot of dysregulated emotion.
- The therapist has to consistently validate all members, avoid taking sides, and

emphasize the wisdom in each member's perspective.

- The therapist has to walk the fine line between decreasing the unfiltered expression of emotion and increasing effective communication about the internal psychological states of family members.

The therapist models validation, compassion, effective limit setting, and an unknowing, curious stance regarding each family member's private experience.

Appendix: Family Therapy Script

Amanda is a 16-year-old adolescent who presented to our residential DBT program after being kicked out of her boarding school for self-injury which, in turn, precipitated a suicide attempt. Her parents were furious at her as they had "pulled strings" to get her into the school, and she in turn was furious with them as during her brief hospitalization after the suicide attempt they had gone into her room and "they read my journal." The parents admitted they were terrified that she would kill herself and angry that she was keeping to herself and that she had a "secret life" that included drug use and sex with boys and girls. The family and Amanda have been oriented to family therapy and have committed to do the treatment. This is their first family session.

Therapist: We have an hour and a lot to get through. I want you each to set a goal for this session and then if we have time at the end we can deal with other stuff that might come up. When each person talks I want the others to listen and not interrupt. I'll be the referee and make sure that everyone has more or less equal time, agreed? Amanda you go first.

Amanda: I want to know why they read through my stuff. It is none of their business.

Mother: I want to know how long this cutting business has been going on and if she is doing it for attention.

Father: I want Amanda to know that we are not going to put up with her behavior any longer and to get all the work we went to get her into the school.

Therapist: OK I want you all to work with the idea that each person is doing the best that they can at every given moment and yes we all can and must do better. Also I want you to consider that it is not in any of your interests to intentionally make life more difficult for anyone else. That is unless you woke up this morning intending to do so! In other words I want to make sure that you all can work with being curious rather than certain.

Amanda: Yea, I can do that, but they can't.

Father: You see what I mean. That is disrespectful and we won't stand for that.

Therapist: If we agree that each is doing the best they can then Amanda can you accept that your father will try and Father can you accept that in this moment Amanda is doing as best she can given that this is her first family meeting in months.

Father and Amanda nod.

Therapist: Amanda go ahead.

Amanda: So OK it's none of their damned business what I write in my diary, so why do they have to go through my stuff?

Therapist: Did you ask them?

Amanda: No.

Therapist: Can you?

Amanda: So why did you go trough my stuff? Why did you read my diary?

Mother: Honey I am so sorry. It's just with all your cutting and suicide we were so worried about you and we were worried that other people were influencing your behavior that you had fallen into a bad crowd?

Amanda: Bad crowd? You mean like your so-called friends who get drunk every weekend at our house?

Father: That's it I won't stand for it. One more disrespectful comment and I'm done.

Amanda: You see I told you he couldn't do it.

Therapist: OK now everyone let's slow down and take a breath. Remember the goals you set at the beginning of the session. If all you do is argue, you go back to the pattern of behaviors that got you here in the first place and you'll never reach your goals. Father, by insisting on Amanda behaving in a certain way and threatening to walk out to get her to do so you lose an

opportunity to understand Amanda's perspective, and can lead to what we term invalidation as it implies that she is capable of doing something that might come easily to you. Amanda by going after your mother in the way that you did it takes you away from your goal of wanting to know why they read your journal. Can we all agree to get back to sticking to the goals, unless of course you want to change the goals!

Mother: That makes sense. I know that we are running out of time and I am terrified about the cutting. Is it just for attention?

Therapist: Amanda how long have you been cutting?

Amanda: 9 months.

Therapist: Mother can you see how if she was doing it for attention, keeping it hidden for 9 months would be a lousy way to get attention? Self-injury in various forms is common in BPD and there are very few experiences that are more frightening, confusing, or worrisome to parents than when a child self-injures. It must be terrifying to think that all you want to do is protect your child and you can't prevent the harm she is doing to herself. Amanda can you help you mother understand more about the cutting and how it helps you? When was the last time you cut?

Amanda: 3 weeks ago.

Therapist: What was going on then?

Amanda: My dad was on my case about how he had done everything to get me into school and how I had screwed everything up. Then what happened. I got really angry.

Therapist: Was it just anger?

Amanda: Well I was sad just before that but it quickly turned to anger.

Therapist: Then what happened.

Amanda: I went to the bathroom and cut myself.

Therapist: Why did you do that?

Amanda: Because I felt better after I cut.

Therapist: Parents, what you see as a problem, the cutting, for her is a *solution* to the problem of how she feels in the moment. So cutting is both a problem and a solution to the problem. Amanda's task is to learn other solutions to intense emotions and your task is to get more curios about her experience without judging it. We still need to answer Amanda's question of why you read her diary but I want to get to dad's goal. . ..

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Systems Training for Emotional Predictability and Problem Solving (STEPPS)

26

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Introduction

STEPPS (Systems Training for Emotional Predictability and Problem Solving) is a manualized, cognitive-behavioral, skills-based group treatment program originally developed in the USA for adults with borderline personality disorder (BPD) (Black, Blum, Pfohl, & St. John, 2004, 2012; Blum, Bartels, St. John, & Pfohl, 2002); the manual was also adapted for use in the UK (Blum, Bartels, St. John, & Pfohl, 2009), and the program is widely used in the Netherlands under the title VERS (Van Wel et al., 2006). The program is evidence based, as designated by the National Registry for Evidence-Based Practices (NREPP 2012). STEPPS was developed as a supplement to, not a replacement for, the patient's ongoing support system and treatment regimen, e.g., individual therapy and medication. Data from six studies, including two randomized controlled trials, show that STEPPS plus treatment as usual (TAU) led to significant reductions in depression symptoms and greater improvements in BPD-related symptoms (Black et al., 2008, Blum et al., 2008, Blum, Pfohl, St. John, Monahan, & Black, 2002, Freije, Dietz, & Appelo, 2002, Harvey, Black, & Blum, 2010, Van Wel et al., 2009). Surveys of both clients and therapists showed high levels of acceptance (Blum, Bartels, et al., 2002, Freije et al., 2002). The program is easily taught (typically 2-day on-site training workshops) to therapists from widely varying theoretical and professional backgrounds.

Although it was originally conceptualized as an outpatient program, STEPPS has been successfully adapted and implemented in a variety of settings, including inpatient units (Boccalon et al., 2012) partial hospital, day treatment programs, residential treatment facilities, substance abuse treatment, and correctional settings including both male and female offenders in prisons and community corrections (Black et al., 2008, Black, Blum, & Allen, 2013). Depending on the facility's schedule and intellectual level of participants, lessons may be broken down into shorter components at more frequent intervals. In this chapter, an adaptation of STEPPS for adolescents in the UK will be described. We will first provide a brief overview of the STEPPS program, followed by an explanation of how the systems approach is applied. This will be followed by a description of the components of STEPPS and the elements that make up the structure of the sessions (lessons). Finally, the chapter will focus on a description of how the program was adapted for adolescents in the UK.

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Overview of the STEPPS Program

The STEPPS outpatient program for adults, which meets for twenty 2-h weekly group sessions, combines a manual-based, cognitive-behavioral, skills training approach with a systems component, which will be described in more detail in the next section. Group facilitators (two facilitators for a group of 6–10 patients are suggested) follow a detailed lesson plan for each week. Group members receive a packet with the lesson materials each week, including a session agenda, a description of the skill to be mastered which is read aloud in the group, worksheets to reinforce the content, and homework assignments. Patients are encouraged to share their notebooks and lesson materials with reinforcement team members and to review homework assignments with their individual therapist. Lessons in the workbook contain artwork, essays, and poems contributed by former STEPPS participants and current group members are encouraged to bring in similar materials to help reinforce the material. The facilitator guidelines contain suggested additional readings, songs, and a variety of relaxation activities.

The Systems Approach

An important goal of the systems approach is to provide the person with BPD and members of their system (treatment providers, family members, significant others, and friends) with a common language to communicate about BPD and the specific emotion management and behavioral skills that group members are learning to manage their disorder. Those system members are identified by the patient as those with whom they regularly share information about their disorder, and they are referred to as the patient's "reinforcement team." The manual includes a suggested lesson plan for at least one education session for reinforcement team members; additional sessions may be added by group facilitators as desired. Patients attending the program are encouraged to become STEPPS experts and to help teach their reinforcement team how to respond with the STEPPS "language." The systems component encourages the client to include peers, family, and others for reinforcement, and reduces the tendency to focus on one individual (e.g., their individual therapist) who runs the risk of being alternately over-idealized and devalued. For clients receiving individual therapy, we ask the therapist to agree to support the program by reviewing the workbook materials provided to the patient each week. Patients are often pleasantly surprised by the capacity for change that their reinforcement team members demonstrate when provided with education and a vocabulary of consistent, familiar responses. One patient remarked, "I cannot believe how much my family has changed since I've been in this program!"

In addition to the basic 20-week STEPPS program, which patients have the option to repeat, there is an optional 1-year, twice-monthly follow-up group program, called STAIRWAYS (Blum & St. John, 2008).

Components of STEPPS

There are three main components to the STEPPS program:

1. *Awareness of illness*. Individuals with BPD often view BPD as a pejorative term, believing it implies they are fatally flawed and, therefore, hopeless and helpless. They may alternately blame themselves and others. The term *emotional intensity disorder* (EID) is often more easily embraced and reflects the way the disorder is experienced. The awareness component emphasizes psychoeducation about the thought patterns, feelings, and behaviors that are symptoms of the disorder and helps group members identify their own schemas (referred to as cognitive filters) that drive their thoughts, feelings, and behaviors.
2. *Emotion management skills training*. There are five basic skills (distancing, communicating, challenging, distracting, and managing problems) group members learn to manage the cognitive and emotional aspects of BPD/

EID. Using the information learned in the awareness component and identifying the cognitive filters activated in a particular situation, assists the individual to more accurately anticipate and predict stressful situations which are likely to lead to increased emotional intensity, and to be more confident in their ability to use their skills to manage their symptoms. There is an optional unit on managing emotional intensity during the holiday season.

3. *Behavior management skills training*. Patients learn eight behavioral skills (setting goals, eating behaviors, sleeping, exercise, leisure, physical health, abuse avoidance, and relationship behaviors). These functional areas often break down through the disruptive interplay between the emotional intensity episodes and the increasingly unempathic and unresponsive social environment. In other cases, dysfunctional lifestyle behaviors (poor nutrition, erratic sleep patterns, substance abuse, etc.) increase the frequency of emotional intensity episodes, which contribute to further deficits in these functional areas. Learning skills to manage these behavioral areas helps keep them under control during episodes of emotional intensity.

Structure of STEPPS Sessions (Lessons)

Sessions have the look and feel of a classroom seminar and group members often refer to attending STEPPS sessions as "coming to class." They are given a binder or folder for their weekly lesson packets and patients often view their binders as a resource they can turn to during stressful situations. Key concepts, such as a brief description of each skill, are described on pocket-sized cards to be carried in a pocket or purse, emphasizing the "portability" of their skills beyond the STEPPS sessions (a detailed description of the STEPPS manual is available at www.steppsforbpd.com). Participants sit at a conference table facing a board. There is a short break between the first and second hours. Each weekly session is organized around a particular skill. Some skills require more than one session to teach.

Participants are asked to monitor their thoughts, feelings, and behaviors over the 20 weeks of the program; this enables them to become aware of and to monitor improvements in the intensity and frequency of their emotional episodes. They are introduced to the Emotional Intensity Continuum (EIC), a Likert-like scale from 1 to 5 using the metaphor of pots on a burner. At level 1 (feeling calm and relaxed), there is no heat under the pot; at level 5 (feeling out of control) the pot is boiling over. This allows participants to recognize early warning signs of an impending outburst; new skills become the tools to prevent the heat from getting too hot, and reduce the chances of the pot boiling over. Group members are asked to complete the EIC daily and to estimate the amount of time at each level during the week. Participants achieve a more balanced view of themselves and are often surprised to learn that they frequently have substantial periods of time when they are not at the fifth level. With this and other metaphors, abstract concepts are more concrete and understandable. As participants progress, they are asked to monitor the new skills they used to manage their emotional intensity. Patients gradually become more aware of emotional triggers that may lead to outbursts.

During a group session, an individual with BPD/EID may try to reframe an emotional experience as the result of some personal or interpersonal problem. While there is an opportunity for participants to respond and share experiences relevant to the skill being taught, the structure does not allow the group or facilitator to spend a significant amount of time focusing on one group member who may be in crisis. One effect of the structured format is to model how to acknowledge problems and offer support, while still imposing reasonable limits on the scope of the interaction so the main goal of the session is not lost. Group leaders must be prepared to reframe problems in the context of the symptoms of the disorder and the cognitive schemas (filters). The

rule to use is: focus on the disorder (managing the emotional intensity), not the content.

Each session begins with participants completing the Borderline Evaluation of Severity over Time (BEST) scale, a self-report instrument developed to help rate symptoms specific to BPD and which has demonstrated good internal consistency and sensitivity to change (Blum, Bartels, et al., 2002, Pfohl et al., 2009). By putting their weekly BEST scores on a graph, patients are able to see the variation in symptom severity over time, and to observe that during the 20 weeks, the increased use of their skills and positive behaviors leads to a gradual decrease in their scores.

The Skills Monitoring Card lists both the emotion management and behavioral skills being taught and allows clients to indicate which skills they used during the previous week. Reinforcement team members receive an abbreviated version of this card with a list of specific responses using the STEPPS language. This allows family members and others to respond to the STEPPS participant consistently; receiving consistent responses from all parts of their system helps decrease the emotional intensity a patient may experience from a perceived crisis, and may decrease the emotional intensity that family members and others often experience from the feelings of frustration felt when the patient with BPD/EID calls repeatedly.

STEPPS for Adolescent Groups

An application of STEPPS for adolescents was developed in the Netherlands in 2009 by Schuppert et al. (2009) with significant modification to the model, emphasizing the concept of locus of control. A randomized controlled pilot study was carried out with 43 young people aged 14–19 years in five mental health centers in the Netherlands. Subjects were assessed before and after random assignment to Emotion Regulation Training (ERT, as the program is known in that country) plus TAU ($n = 23$), or to TAU alone ($n = 20$). Assessment included measures of BPD symptoms, locus of control and internalizing and externalizing behavior. Both groups showed reductions in symptoms of BPD over time. There was a significant increase in internal locus of control in the ERT plus TAU group, with no significant change in the TAU only group. The ERT subjects reported a greater sense of control over mood swings with a higher level of attribution of changes to inner control factors. There was a high level of attrition in this study and the authors highlighted difficulties inherent in treating and researching an adolescent population, as well as the need for researchers to develop more age-appropriate assessments.

The main author of this chapter set out to create a version of STEPPS for adolescents for an English speaking audience, bearing in mind the systems within the healthcare in the UK and some of the specific challenges of the current economic climate. Considering the adolescent population itself, there was recognition that individuals within the group of young people from ages 13 to 18 may differ considerably in developmental phase and in the degree to which the material would need to be modified to be suitable. In order to create a starting platform for this, the aim was to begin with the older group, the 16–17-year-olds. With this group, it was assumed that the existing STEPPS manual for the adult population might need less modification and could be piloted, with some initial minor adjustment. The rationale was that this group might be able to relate to the same manual structure initially, and that they could be involved in helping us to rewrite it to make it more suitable. Further modifications for the younger age group could be our next step.

We also believed that running a STEPPS group with young people who would most likely be brought to the groups by their parents or carers, or who would have parents/carers still closely involved in their education, development, and emotional needs, provided us with an opportunity to involve them differently from the standard STEPPS group.

Initial Modifications to the Manual

Our first step was to consider the stage in the care pathway and the issues around diagnosis. This had been a stumbling block for some time, as clinicians had been very wary of diagnosing young people, and the UK authors were also working with a legacy of clinicians who believed (and perhaps still believe), that you cannot diagnose personality disorder in a young person or at least that you should not do so for a multitude of reasons. In spite of work being done elsewhere on early intervention with young people (described in this publication), it proved difficult to overcome this resistance. Several years of meetings, presentations, and other discussions met with similar responses of resistance to identifying and providing any interventions with young people who were (by their perception) going to be "labeled." All of this was occurring in a setting of considerable upheaval resulting from organization change, where the working groups set up to carry out this project kept changing, with people leaving for various reasons, working groups needing to be reestablished, and other agendas taking precedence. This is not to say that individual clinicians and a core of individuals in the working groups were not recognizing the gap in service provision and attempting to work with some of the presenting issues in various other groups (for example, self-esteem). Fortunately this led to the ability to draw together a group of clinicians with an interest and considerable skill in engaging and working with young people facing the kinds of challenges which STEPPS addresses.

The first issue was to work with a way of identifying the core problem to be addressed, without applying the diagnostic label "Borderline Personality Disorder." In STEPPS, this label is changed during the very first lesson to "Emotional Intensity Disorder" because it is recognized that

- People come to groups with a wide variety of experiences with this diagnosis, ranging from extremely negative to somewhat positive, where they have embraced and perhaps even over-identified with it. It is recognized that it is probably useful to create a more level playing field by using another descriptor as a more useful way of referring to what STEPPS is addressing.
- Some people are actually referred to groups without having been told what their diagnosis is, or with another label, such as bipolar illness, which may or may not be helpful (and, of course, may be a comorbidity which needs to be addressed). With referring clinicians recognizing that the pattern is one which is likely to be addressed in the STEPPS group (i.e., with significant features of BPD) the opportunity was created to be helpful to these individuals in managing episodes of emotional upset and destructive coping mechanisms without the need to apply another psychiatric label to them.
- The term "Emotional Intensity Disorder," while not officially recognized in any diagnostic system, has been found to be much more acceptable to service users. In their opinion, (and in the opinion of many others), this is a much more descriptive term for what people are struggling with, one which is easily understandable and not as unacceptable to them as "borderline personality disorder."

In considering this as it applied to the manual for young people, it was felt that the opportunity to bypass the issue of labeling in the spirit of early intervention would be even more fully met if we changed our terminology to "Emotional Intensity Difficulty" or even "Emotional Intensity." The latter was eventually rejected, as it was felt important by the main author of STEPPS that people should not be given the message that there was something "wrong" with being emotionally intense. People are taught in STEPPS that there are ways to cope with emotions when they become intense so that one does not have to resort to destructive and potentially addictive ways of coping. If intense emotions are managed constructively and channeled into something positive, there is no reason to label them in any negative way. Sperry (2003) differentiates between "personality

disorder" and "personality style," the former referring to "functioning that is characterized by specific DSM-IV diagnostic criteria," while the latter refers to "high adaptive functioning behavior for a particular personality type" (Sperry, 2003, p. 11). Sperry states, "The basic goal of treatment is to facilitate movement from personality-disordered functioning to adequate personality-style functioning or even to optimal functioning" (Sperry, 2003, p. 11). This "optimal functioning" is assumed in STEPPS not only to be a goal of treatment, but to indicate that the underlying "style" in this group of people in fact indicates a personality with many "positive" characteristics, a message to counteract the very poor self-esteem that is common amongst people given the diagnosis of BPD. Sperry's description of the personality style which corresponds with BPD is as follows:

- Tend to experience passionate, focused attachments in all relationships. Nothing in the relationship is taken lightly
- Emotionally active and reactive, they show their feelings and put their hearts into everything
- Tend to be uninhibited, spontaneous, fun-loving, and undaunted by risk
- Tend to be creative, lively, busy, and engaging individuals. They show initiative and can stir others into activity
- Imaginative and curious, they are willing to experience and experiment with other cultures and value systems
- Regularly tend to be deeply involved in a romantic relationship with one person (Sperry, 2003, p. 83).

Within a population of young people who have not yet been given a diagnosis, a focus on enhancing the positive aspects of this personality and deflecting behaviors and perceptions away from further development or consolidation of a "disorder," would present a more hopeful stance to say the least.

In line with the decision to avoid the term "borderline," the BEST (Borderline Evaluation of Severity over Time), was changed to QuEST (i.e., "Quick" Evaluation of Severity Over Time).

In terms of further modifications made to the manual at the initial stage, we considered the time period over which groups would be running. In the standard STEPPS group for adults, every attempt is made to run the groups consistently every week. Ideally there would be no breaks. In reality since some groups are bound to run when a major holiday season would occur, the STEPPS manual provides an extra chapter entitled "The Holiday Season." The ethos of the content makes it most applicable to the course being run with a break over the Christmas period, although it could be adapted slightly to be used for holiday breaks over Easter. Breaking the course for the long summer holidays in August is generally not thought to be a good idea, as this leaves too large a gap in the flow of continuity of the course, where skills build sequentially on one another. The reality of running a STEPPS course with this service user group means that ideally the course would run during term-time, with planned breaks around mid-term holidays. In order to fit the whole course into a time period without too many external breaks, the course facilitators decided to condense two of the topic areas slightly. When the topic of challenging is taught, the adult version of STEPPS allows for two sessions in which new content is covered and a third which is for review. The decision was made to condense this to two sessions overall, incorporating revision into the second lesson. Similarly, lessons 15 and 16 dealing with areas for behavior change were merged, with a focus on participants beginning gradually in one area of their choice. This reduced the overall length of the course to 18 weeks.

Our final modification before our first group was to consider the role of parents and carers.

Carer and Family Involvement

In the standard STEPPS groups run for adults, the course involves one evening session for carers or family members during which they are provided with information about BPD, the STEPPS course and what it addresses, and how they might

support the person on the course through helping to reinforce skills taught on the course.

Young people in the groups, being under age, would all still have family members or carers involved in their care. Most young people coming to the group would also be brought to the group by family members, who would either wait for the 2 h period or would be coming back to collect their family member. This provided, we thought, a clear opportunity for closer involvement of the families in the process.

With this in mind, we believed the pilot study would provide us with an opportunity to examine the effectiveness of increasing the family engagement by involving them more closely in the course. In the adult groups run in Iowa, family members are also given the opportunity to attend some of the groups (by prior arrangement, and with the permission of all group members). This would involve the family member sitting in the same room, observing the group. Interestingly, in Sussex, we have generally encountered strong resistance within our adult groups for involvement of any "outsider" once the group had been established. Considerable preparation has to be done within each new group in anticipation of the family/carer evening session, as our group attendees have reacted with general anxiety and mistrust. We have found it necessary to reassure them repeatedly that no personal discussions of any kind would be allowed, that the evening was about information-giving and setting up skills reinforcement, and also that they were invited to be present if they so chose. For our young people's pilot study, the working group after some discussion, concluded that we would invite parents to form a parallel group, which would run alongside the main group but in another room and with different facilitators. The aim of this group would not be to provide a STEPPS group for them, but would take them through the material week-by-week, so they would be able to have an in-depth understanding of the content, and how they might work with their son or daughter in the most acceptable, helpful, and constructive way. This would also provide us with an opportunity after the group, for their feedback.

Consideration was given to how much communication there would be between the two groups. The decision was to keep this to a minimum. This was done to allow both groups to feel they were able to speak freely in their group and that what they had said would not be communicated to members of the other group by the facilitators. Any communication would be up to them.

The Study

The Population

Young people were recruited from across West Sussex through communication with community teams and from group facilitators' own caseloads, using the following descriptive illustration of the symptom features likely to be found in this group:

Levels of emotional intensity which may be described as problematic could include:

1. Fear and worry that someone important in your life is tired of you or planning to leave you. You may go to extremes to keep someone from leaving you.
2. Unstable and stormy relationships and friendships because of quickly changing your opinions about others, such as thinking someone is completely wonderful, and just as quickly deciding they are the worst person you've ever known.
3. Frequent or extreme changes in how you see yourself, such as shifting from feeling confident about who you are to being very unsure of who you are, or what your goals and values are.
4. Being impulsive and engaging in risky behaviors (like risky sexual behavior, using drugs or alcohol, driving recklessly, etc.) without thinking of the consequences.
5. Feeling very sad and/or hopeless, purposely doing something to injure yourself, or making a suicide attempt.
6. Experiencing very rapid mood changes several times a day, often going from feeling depressed to normal to angry or anxious very

quickly. Minor events seem to cause major shifts in mood.
7. Feeling very empty.
8. Temper outbursts or problems with anger leading to relationship problems, physically hurting other people, or breaking things (your own or other people's).
9. When you are stressed, feeling paranoid or distrustful of people you usually trust. Having brief blackouts or periods of time when you forget what has happened (not due to using drugs or alcohol).

The above characteristics should be a long-term feature of your personality, and not be as a result of a physical illness, use of substances, or only relating to one or two specific situations.

(Above based on American Psychiatric Association (2000), DSM-IV-TR, (2013) DSM-5 and BEST/QuEST, STEPPS Group Treatment Program (2002, 2012))

The age group was limited to 16–17 years of age at the start of the group.

A total of ten young people were invited to attend for assessment, and eight presented for the first session.

Method

Screening and Assessment

Potential participants were invited to an assessment session where they were asked to complete the Millon Adolescent Clinical Inventory (MACI) (Millon, Millon, Davis, & Grossman, 1993/2006). This is a 160-item questionnaire which takes about 20 min to complete. It provides information on 31 scales across both DSM-IV-TR (APA, 2004), Axis I and II, including "Borderline Tendency." This was done to formally establish the presence of features of BPD.

Prior to start of the group, participants were asked to fill in a QuEST, a Strengths and Difficulties Questionnaire (SDQ), and a Moods and Feelings Questionnaire (MFQ). The SDQ is a brief behavioral screening questionnaire for 3–16-year-olds (depending on their understanding and literacy), with 25 items. It measures psychological attributes on five scales: emotional symptoms (five items), conduct problems (five items), hyperactivity/inattention (five items), peer relationship problems (five items), pro-social behavior (five items). The MFQ is a 32-item questionnaire based on DSM-III-R criteria for depression. It consists of a series of descriptive phrases regarding how the subject has been feeling or acting recently over the previous 2 weeks. Additional items (e.g., loneliness, feeling unloved) were also included because of their clinical significance to the construct.

The parents/carers group members were asked to fill in a QuEST rating the young person, and were given parent's versions of the SDQ and MFQ.

The above measures (except for the MACI) were repeated after the group ended (last session). In addition, group members in both groups were asked to fill in a Group Evaluation Questionnaire, a brief list of open and closed questions about the group. One week later, both groups participated in a focus group discussion facilitated by independent research assistants to obtain further feedback about their experience of the group. These were recorded and transcribed verbatim and subjected to a thematic analysis.

Procedure

Groups were set up to run in the early evening (5–7 pm) so that working carers/parents were more likely to be able to attend. Two rooms in the same building were booked for the whole length of the group.

For the first meeting, all the young people and their families/carers were gathered in one of these rooms. Facilitators welcomed them, introduced themselves, and explained that the young people would be taken to another room, and that thereafter there would be no communication between the two groups about what was taking place in the group, to ensure a sense of trust and safety in speaking freely. (The usual caveat to this was given, that is that in the event of a high risk situation, appropriate steps would

need to be taken and confidentiality of necessity set aside as required.) The young people were then taken out of the room and introduced to the venue where they would go through the rest of the course.

After this, communication between facilitators of the two groups was kept to a minimum. There was no sharing of any of the paper work done after the group (the keeping of Group Process Notes and Individual Record Sheets), and discussions of what had occurred in the group were restricted to generalities or practical issues.

Both groups were set up in a classroom/seminar type environment, with tables or desks, and the availability of whiteboards and flipcharts. In addition, there was material for running the groups, such as art/craft materials, aids for relaxation, stacks of ongoing monitoring forms and worksheets, and the lesson packets which are handed out weekly. The groups ran for 2 h, with a 15 min break roughly halfway during which refreshments were provided.

For the young people:

After the introductory session (Lesson 1), lessons settled into a pattern of weekly monitoring, checking of homework tasks from the previous week, and working on the EIC with examples from experience, followed by the next lesson after the break.

For the families and carers:

Participants were given the opportunity to update others in the group on how the week had gone, with examples of how they might have seen skills in action or had been involved in helping to reinforce skills. Following the break, the same lesson pack as the young people were getting was handed out, and facilitators took them through it. This was done in the spirit of informing them what their family member would be doing, and they were not specifically asked to complete any of the exercises for themselves, nor encouraged to think about how any of the skills might be relevant to their own lives.

From the five facilitators who ran the sessions, three were also reinforcers for some of the young people attending the group. One clinician ran the parents group and two clinicians ran the young people's group. The young people felt that it was helpful to have their reinforcement clinician facilitating the groups as it encouraged them to attend and to begin to use the skills learned in the session throughout the week.

Throughout the group some facilitators were able to keep in communication with the young people's reinforcement teams. The young people seem to value and trust in the facilitators that they would support them in ensuring that their reinforcement team would be made aware of any difficulties they had mainly relating to their self-harm behavior. However, some young people had little or no contact with their reinforcement team either because they chose not to attend the appointed sessions or because the reinforcer clinician was not available.

The young people were initially concerned that their parents/supports were in the same building, but quickly realized that they would be kept separate. They were concerned that the facilitators would discuss the group with their parents/supporters. The facilitators reassured the young people that they would not break confidence and would not be engaging in any conversation with their supporters unless absolutely necessary.

Results

Eight young people presented for the start of the group. Two were accompanied by both parents. One was accompanied by a mother and sister. One was unaccompanied. Over the course of the group, one of the parents attended very erratically because of childcare issues and eventually stopped attending altogether. Of the eight young people starting the group, three dropped out, the group ending with five. One of these was the young person without a parent in the families' group. In the parents/carers group, a total of six people stayed with the group to the end: one couple, two mothers, and the mother and sister dyad.

We were unable to analyze the questionnaires statistically because of small numbers, and although drops in scores were observed in most

cases, it is impossible to say whether these were statistically significant. These drops were observed in the questionnaire scores filled in by the young people, as well as the scores obtained from their families. We report the qualitative outcomes of the study here as well as our observations on the setting up and running of a new systemic intervention within service settings in a state of flux.

Results from the young people's group:

The young people who participated in the adolescent pilot group were forthright with their view about the manual and in many things they were all in total agreement. They struggled initially with the classroom approach and the "paper work" and some (deliberately?) did not bring their folders to the first two or three sessions.

Fortunately this issue was soon resolved. Interestingly the aspects that the facilitators presumed that the young people would not like were very different from the reality. It was thought by the facilitators that the group members would find 20 weeks too long and that they would just stop coming after 12 weeks. In actual fact, the young people grew much more cohesive as a group after the first 7 or 8 weeks and were all very sad when the group ended. Several sent texts to the facilitators on the following two Tuesdays after the group finished saying how much they missed the group or that they didn't know what to do with themselves on a Tuesday now that they didn't have the group. It was widely assumed by the facilitators that the homework would be an issue. The young people invited to attend the group were potentially in the middle of an important academic year at school when the group began and it was felt that this would be an obstruction to the homework. We were only partly right. It was not actually the completion of the homework that the young people had difficulty with. The issue was admitting that they had actually done it. At the start of every session the question: "who has done the home work?" was met with a grim silence. We soon learned to dispense with this question, as it became clear in each session that actually most of them had done the work but it was just not acceptable to admit doing it.

The manual was hard for many of the young people to take in. There were a lot of ideas and concepts that they found difficult to understand such as the difference between emotions, thoughts, and physical sensations. However, they were much more sophisticated than we gave them credit for and the language of the manual was much less problematic than we anticipated. The first half of each session was often spent revising the content from the previous lesson and this really did help to cement the concepts for all participants.

The parts of the lessons such as the stories and poems written by previous STEPPS participants did not resonate with the young people in the way that we hoped. We were never sure if it was because they reminded them too much of literature lessons at school or because they could not compare their experiences to fiction. The poems did, however, provide a very useful discussion point. The fact that some of them were universally loathed by all of the group members helped unite the group, and this feedback from the young people about how much they hated them will guide modifications.

Some of the statements made by the young people (both written and in the focus group) presented contradictions: on the one hand, they were critical of the material and stated that the group had made no changes to them or even that raising awareness through attending the course had in fact made things worse for them:

- "I think once you are into that frame of mind, you can't write it down to stop from self-harming. And that was kind of what we were meant to do. And I don't think that's helpful at all because when you sit down and write about it, you can either go one of two ways, where you sit and think about it and then just get more and more built up or it can make you feel better, but I think for most of us, it didn't make us feel better, it perhaps made us worse. I know for definitely for me it did."
- (re filters) "I think it's quite overwhelming, because I was sort of looking at it for about 5 minutes and staring blankly and thinking I really don't want to get into this sort of thing. It brought up a lot of shit."

On the other hand, statements were made such as:

- "...it has just helped me realize like... to be honest I've been on CAMHS for like 9 years or something now, and this course has basically been the closest I have ever got to actually being told I have anything. And it's helped more than those 9 years."
- "I'm more in control than I was and I think about it more than I used to because before I would just be like right this is my solution and go straight for it now I use different techniques to try and stop me from self-harming."
- "I've changed in my personality: who I am and stuff. Like before you wouldn't get a word out of me I'd just like sit there like 'I aint talking'. And now I will talk. And I don't feel so nervous."
- "I'm more aware of my emotions so.... I have realized like when to step away from stuff ... and not carried on, I have got out of situations."

And some ambivalence:

- "It's helped me to put things into categories so I know what's kind of wrong and what's alright, if that makes sense. It's helped in ways, but in other ways it hasn't really helped, but...yeah, it's kind of hard to describe it, because I don't know what it has helped, and what it hasn't, all I know is that it has helped with certain kinds of stuff."

There was generally also ambivalence about parents being involved, but general agreement that they wished they had had this course earlier in their lives:

- "I ... think that they should make it for younger people really. Um. Yeah before they kind of get stuck in this kind of mentality. It's easier when they are younger...to change your mindset kind of thing."
- "I think if I was around 13 when I was to do this, when I had not long been harming, I think it would have changed me so much, and I don't think I would still be harming now because I would have been taught about it."

Others: "Yeah, yeah, yeah" [Agreement].

Results from the parents/carers group:

Feedback from the parents/carers group was generally much more positive than from the young people's group. They experienced their group as positive and saw more positive change in their family member. Some comments:

- [re the developing of awareness] "I think when you just come into something like this, for the young person, if something is given a name, you're not really understanding it, but to have it put into context like this, I think this is what inspired us to accept this is the kind of things your young person has been going through. And you haven't been aware of a lot of that, so yeah it's good just to have it down that it's clear and concise and this is the reasoning behind what's happened is happening."
- "... its always going to be difficult when you have so many people with so many different personalities—but I think the structure... the first one's a bit of a blur anyway because you're coming into a room full of people you don't know, you're scared, you feel like you're being judged and the rest of it, so you've got all this guilt on your own, and then you come in so, the structure...all the way through has been comforting."
- "we were saying that... it was a cathartic space and it wasn't therapeutic although we were able to help each other sometimes with certain experiences that we've had or that sounded familiar...and we got to know each other and we were saying in those first few weeks we were like terrified, hardly anyone spoke, and it was like 'what do you mean, we have to read?' you know. But as we've moved forwards we've all sort of become mates and talked to each other and sympathized with each other."
- "and its been nice, well not been nice, that's really the wrong phrase to use—maybe when some of the other kids have also had a bad week that you think, actually its not just us, we're not going wrong with what we're learning, it is just another blip, so I think for all of us this has been kind of rewarding to hear that you are doing the right thing but they are, your kids are going to have weeks where its not going to be perfect."

The group overwhelmingly expressed positivity about the mutual support they felt from others. There was general agreement that they had learned skills for themselves, for example,

- "It has been a truly positive thing and we have all. . . learned so much from it."
- "That relaxation technique; I do that every morning in a traffic jam!"
- "Before I only had a limited number of words I could use—like anxious—now I have a lot more words, and I can use other peoples' examples."

Asked whether they had noticed changes in their young family member:

- "I'll never be a 100 % sure of how she is really feeling. I think she personally has masked it so well over the last 2 or 3 years, she says she is fine and then the next thing, she is in hospital. It's hard but I certainly noticed a difference being on this course."

And changed the relationship between them:

- "And I says on the way home 'how do you think the course has changed you, and changed us?' and she said. . .what was it? Because you understand, I feel like we can talk about it rather than it being something that we're all scared to talk about."
- [Describing a specific problem that had occurred earlier]. . . "I think it was just a realization that I was able to let her sort out her problems with my help without really over reacting and making the situation a lot worse. Because if I had done what I would normally do and shouted and got cross she would have gone. And I wouldn't have seen her 'til the next day sometime. So yeah, but she stayed at home, we stayed home together that evening, watched a DVD, had a great evening together, and it was just because I had dealt with it differently."
- "How I react to [x] now or even half way through is completely different to how I would have reacted in the past. I mean I was more likely to tell her to grow up and stop being silly. . .[elaborating on a specific incident] we feel like we can trust her more. And hopefully the trust is building up, it's taken this course I think to get to that."

Some of the more problematic issues raised by both groups related to the availability, consistency, and quality of reinforcement from the wider team and their anxieties around what might follow after the course ended—whether any more help was available, whether they could access STAIRWAYS (the 1-year follow-up program mentioned previously), the loss of the mutual support coming from the group, and whether positive changes would last.

Discussion

Although this was a very small group, we believe the results present a strong case for proceeding with further development of STEPPS for young people. Verbal feedback during and after the course, backed up by the thematic analysis suggest that the course was helpful in bringing about a change in attitude, in perceptions, in symptoms, and in behavior in an 18-week period. A few specific issues will be elaborated further below.

The drop out rate: Drop out rates from groups with this population (all ages), tends to be relatively high, many studies reporting between 30 and 40 % (Freije et al., 2002; Harvey et al., 2010; Linehan, 1993; Yeomans et al., 1994). It should be borne in mind, however, that reasons for dropping out of groups are not always negative, and in our experience (anecdotal evidence only), individuals frequently report having derived some benefit from only a few sessions. Future studies might shed light on this by attempting to discover what the optimal number of sessions for effecting meaningful change is.

Resource issues, positive and negative: While the STEPPS group was running, it was noticed by the reinforcement teams that the young people reduced their demands/use on other services. The group appeared to hold the young people in a way that allowed them to feel supported and to an extent understood by the other group members and the facilitators.

The STEPPS course for adolescents had been discussed within the NHS trust with many CAMHS clinicians. Clinicians agreed that the most complex and at times challenging young

people with emotional intensity difficulties, who demanded many hours of clinician time, through therapy, individual and family, and many hours in emergency/crisis intervention, services which included police and inpatient hospital both general and psychiatric services, would benefit from the STEPPS program. STEPPS offered an evidence-based psychoeducation program that would enable the young people to learn and practise skills to manage their emotions. STEPPS also gave the young people a place to explore their emotional intensity difficulties, and negative- and positive-coping behaviors in a nonjudgmental way.

The pilot group facilitators were skilled CAMHS clinicians who had an interest in BPD and working with groups of young people. The clinicians were also prepared to make the time commitment for a minimum of 18 weeks, which would include working into the evening 4–8 pm.

The time commitment from five clinicians would be approximately 3–4 h a week. This would not include the administration and photocopying that was required each week to produce the session packs and input the weekly data collected from the young people. While this time commitment initially felt excessive, we found that time spent liaising with other agencies and managing crises were significantly reduced. There were fewer presentations at A&E with episodes of self-harm during the STEPPS program than was usual for the young people.

Observations following the ending of the course presented clear evidence for careful consideration of what needs to follow after STEPPS. Participants from both groups expressed a strong sense of loss after the end of the group, with some reported deterioration of symptoms in some of the young people. Further work on systemic support is needed, as one of the issues facing this group apart from the losses of the group was the milestone of the 18th birthday, with issues of losing CAMHS support as they were discharged either from services or across to adult services.

Further changes to the manual: Throughout the course and during feedback sessions, many helpful suggestions were made for further refinement of the manual. These will now be applied in preparation for further research.

Conclusions

The outcome of this initial pilot with adolescents has been extremely positive and indicative of the value of further development. Much more work needs to go into systemic support around running the groups, arranging for robust follow-on from the group and development of ongoing therapeutic input where required. A more extensive research study will aim to confirm initial impressions of significant change in attitude, understanding, and behavior in the young group participants, in perceptions, skills, and helpful involvement of their parents/carers, and in improvements in the relationships between them. The pilot has helped highlight some of the challenges and point the way forward.

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Appendix: A "Typical" STEPPS Lesson

Every STEPPS lesson is structured according to an agenda, which is handed out with the notes. The agendas are all broadly similar. This provides a predicable pattern, which helps the participant to feel less anxious and enables them to concentrate on the new material being presented each week.

Described here is Lesson 10, which is the first of two on "Managing Problems." By this stage, the participant has been introduced to the concept of emotional intensity difficulties (as an alternative to a diagnosis of BPD) and has been given a series of emotion management skills to underpin the work on behavior change to follow (See Chap. 27 for a description of these). Comments in square brackets explain the process.

Agenda

- Complete a QuEST scale and record the score [Symptom measure which is done weekly]
- Relaxation [A brief relaxation session, each week introducing a different method so that participants have a choice]
- Review *EIC* [Here there is an opportunity for participants to describe how the past week has been, and for the group to share how they have filled in the 5-point EIC form relative to any incidents they experienced. There is usually an example done in the lesson on the white-board, with all encouraged to comment, make suggestions, and share their own responses.]
- Review Skills Monitoring Card [Participants have been encouraged to use a tick list of skills every day.]
- Review of homework exercises from the previous week.
- Presentation of the week's lesson and homework exercises.

In the Managing Problems lesson, participants begin with problem identification and potential solution strategies, but with particular attention paid to understanding the role of "filters" (schemas/core beliefs) in contributing to the intensity of their reactions and the obstacles in their way. All the skills learned so far are brought to bear in understanding how they might find a possible solution and how to overcome resistances and self-sabotaging which may have played a role in past failures. The group contributes by making suggestions, encouraging each other, and sharing experiences of what has been helpful for them. In the lessons to come, feedback from each of the previous lesson is used to build the skills of the next.

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Part VI

Conclusions and Future Directions

Borderline Personality Disorder and DSM-5: New Directions and Hopes for the Future

27

Merav H. Silverman and Robert F. Krueger

Despite advancements in research on and treatments for borderline personality disorder (BPD), which have relied on the current formulation of the diagnosis as presented in DSM-IV, growing evidence argues for reassessing how we understand the BPD diagnosis, in the context of a broader sea change in our understanding of personality disorder (PD) psychopathology. An increasing body of research, driven by a strong theoretical framework and fueled by empirical studies, provides evidence for remodeling the overall framework of PDs in an effort to make the diagnoses more valid, more descriptive, and more clinically relevant. In this chapter we discuss certain of the main challenges to DSM-IV PDs and the process of developing the PD sections in DSM-5. We particularly focus on the implications for research and treatment, with an eye toward the impact of changes for children and adolescents.

Over the past 30 years, there has been a proliferation of research on BPD. To date, research on BPD has relied on the model of the disorder presented in DSM-III and the modifications of the diagnosis found in subsequent revisions of the DSM (III-R, IV, and IV-TR). This research has shed light on the etiology, course, and prevalence of the disorder. In conjunction with an improved understanding of the disorder, progress has been made in creating and disseminating treatments for BPD; in recent years, a number of treatments have been developed for BPD, which have been validated empirically and manualized, allowing for wider distribution and improved quality of care for borderline patients. While the majority of the contemporary research on BPD has been in adult samples, recent studies have verified the positive value of identifying and treating BPD at younger ages (Chanen et al., 2007). This finding, along with a general interest in early interventions for psychopathology, means that there now exists a growing literature about BPD and BPD symptoms in children and adolescents. Using the current diagnostic criteria set for BPD found in DSM-IV-TR (henceforth in this chapter, DSM-IV), a great deal of progress has been made, furthering our understanding of the disorder, garnering public attention for the disorder, and improving treatments for very impairing symptoms.

Still, despite the progress that has been made using the current model of personality pathology, there is a compelling case for using what we know about basic personality to shed light on PDs, thereby bridging the gap between these two, currently disparate, areas of research (Markon, Krueger, & Watson, 2005; Widiger, Livesley, & Clark, 2009). This, along with a growing literature on the underlying structure of PDs, which provides an evidence-based alternative to the current medical-categorical model of PDs, has coincided with the preparation of

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DSM-5. This has brought the question of the future of PD diagnoses to the forefront of both researchers' and clinicians' minds.

As it stands in DSM-IV, in order to receive a diagnosis of BPD, a person must exhibit an enduring pattern of instability, manifested in interpersonal relationships, self-image, affect, and impulsivity. This pattern must be evidenced in early adulthood and must be present in many areas of functioning. DSM-IV identifies nine criteria indicative of BPD. In order to receive a diagnosis of the disorder, a person must show five or more of the following symptoms: efforts to avoid abandonment, unstable interpersonal relationships, unstable self-image or sense of self, impulsivity, suicidal and self-injurious behaviors, unstable affect, feelings of emptiness, difficulty with controlling anger, and transient, stress-related paranoid, delusional, or dissociative symptoms. The current diagnostic criteria are made up of a combination of specific behavioral symptoms, similar in feel to symptoms one would see in Axis I diagnostic criteria, and more enduring, trait-like criteria.

Though the current diagnosis captures many features of BPD, and has been a useful tool for better understanding and treating patients with the disorder, a growing body of research highlights the shortcomings in the current BPD diagnosis. Enough evidence had accumulated such that, with the revision of DSM-5 approaching, many in the field thought it was time to reconsider the structure of the disorder. Many researchers involved in the DSM revision process felt that the categorical diagnosis of BPD did not represent the most compelling articulation of the dysfunctions present in patients with BPD. They also identified the shortcomings of the DSM-IV diagnosis as a clinical tool, noting that it did not capture the ways that patients present in clinics and hospitals. The challenges to the BPD diagnosis, as with the other PD diagnoses, are not simply abstract or academic. Rather, they have a direct bearing on clinicians' ability to conceptualize cases and develop treatment plans for severely impaired patients.

With a sense that the limitations in the BPD diagnosis were hindering treatment of and research on the disorder (and with similar concerns existing for the other PDs, as well), the researchers and clinicians who were chosen to serve on the Personality and Personality Disorder (P & PD) Work Group for DSM-5 proposed alterations to the framework of the PD section of the DSM. The members of the work group were selected for their extensive research on PDs and their experience working with these patients. Throughout the process, BPD in particular captured the attention of many in the field, due to its prominent status as the most researched and treated of the PDs. Because of this, both researchers and clinicians cared a great deal about the future of the disorder.

Many of the criticisms of the current model of BPD are not specific to BPD but rather pertain to the entirety of the PD section of DSM-IV. These limitations have been discussed at length elsewhere (Clark, Livesley, & Morey, 1997; Krueger & Eaton, 2010; Krueger, Markon, Patrick, & Iacono, 2005; Westen and Shedler, 2000; Widiger & Trull, 2007) but will be covered here in brief. Four key limitations, in particular, have proven vexing in the current diagnostic system and serve as the primary intellectual impetus behind the argument for restructuring PD diagnoses in DSM-5. These include polythetic and dichotomous diagnoses, excessive comorbidity of psychiatric disorders, inadequate coverage of psychiatric dysfunction using our current diagnostic categories, and lack of diagnostic stability over time.

The first part of this chapter will explore these theoretical challenges to the current conception of BPD and some of the ways that these conceptual difficulties hinder further research, case conceptualization, and treatment. Next, we will outline the process by which the PD section for DSM-5 was developed, and will explain, in brief, the final PD sections in DSM-5. Subsequently, we will explore some of the future directions and anticipated changes moving forward, beyond the publication of DSM-5. Special attention will be paid to the ways that these changes may impact

research and treatment of BPD and BPD symptoms in children and adolescents. It is important to emphasize that the goal of these proposed changes is to provide a more precise method of describing and diagnosing BPD in order to improve the quality of research and to aid clinicians in their diagnosis and treatment of the disorder. Ultimately, the intention is that these changes will better serve those researching BPD and improve treatments for patients suffering from the disorder.

Challenges to DSM-IV Personality Disorders

Polythetic Dichotomous Diagnoses

In our current diagnostic system, PD diagnoses are conceptualized as polythetic dichotomies. The polythetic nature of the diagnoses indicates that many different combinations of symptoms can lead to the same diagnosis and that not all symptoms for a specific diagnosis need to be met in order for a person to meet for that diagnosis. Dichotomous means that diagnoses are established using a threshold of symptoms. If persons present with the number of requisite symptoms for a diagnosis, or higher than that number, they would meet for a disorder. Alternatively, if they present with a subthreshold number of symptoms, they would not receive a diagnosis. In this way, the diagnoses are considered as a yes/no distinction, with no room in the rubric for gradations of a disorder.

Before DSM-III-R, certain PDs, such as dependent PD, were diagnosed using monothetic criteria sets, meaning that all symptoms had to be present in order to receive that diagnosis. Other PDs were diagnosed using polythetic criteria sets (Oldham, 2005). Revisions for DSM-III-R included making all of the PD criteria sets polythetic, after determining that monothetic diagnostic systems were not reliable. Monothetic criteria sets were considered too restrictive to capture the heterogeneity in these disorders; therefore, the writers of DSM-III-R converted all of the PDs to polythetic criteria sets (Pfohl, Coryell, Zimmerman, & Stangl, 1986; Widiger, Frances, Spitzer, & Williams, 1988). As compared to monothetic criteria sets, polythetic criteria sets allow for more flexibility in the diagnoses by allowing phenotypic variation in the symptom manifestations of the disorders (Cooper, Balsis, & Zimmerman, 2010).

Polythetic criteria sets mean that patients can present with only a portion of the criteria that define a disorder; as long as they have met the symptom threshold, they receive the given diagnosis. The specific number of symptoms that serves as the threshold varies from one PD to the next. For example, in order to receive a diagnosis of BPD a person must endorse five diagnostic criteria or more out of nine possible criteria; for obsessive–compulsive PD a person needs to meet four or more out of a possible eight criteria. Practically, a polythetic classification system of PDs means that it is possible for there to be very little symptomatic overlap among patients with the same diagnoses. For BPD, specifically, there are 256 different combinations of symptoms that all result in a person receiving a diagnosis of the disorder. Another way to think about this is that two patients presenting with a diagnosis of BPD may only share one symptom in common. For obsessive–compulsive PD, mentioned above, the reality is even starker, in that two patients may not share any disorder-specific symptoms but might nonetheless receive the same diagnosis. This extreme level of phenotypic variation creates challenges for research and treatment of PDs.

In addition to the problem of arguably too much symptom variation within the current system, polythetic criteria sets also introduce the challenge of developing diagnostic cutoffs. Whereas disorders diagnosed using monothetic criteria sets did not need a threshold, in a polythetic diagnostic system a specific point needs to be established where above a certain symptom count, a person meets for a diagnosis (Oldham, 2005). A person that meets for one symptom less than that diagnostic cutoff point, from a diagnostic standpoint, is considered to be free of personality pathology. From a clinical standpoint, it would be hard to imagine that this

person would be treated as if he or she suffered from no features of the disorder. Using categorical classification, though, there would be no way to classify subthreshold impairment.

Deciding where to set appropriate diagnostic cutoffs for PDs has proven quite challenging, as there is no clear boundary above which it is obvious that a person's behavior is pathological. Furthermore, it is unclear whether more symptoms (or even above threshold symptom counts as opposed to below threshold symptom counts) necessarily indicate a worse clinical presentation or course. For example, it is likely that there are certain combinations of four BPD symptoms, which would be subthreshold for a BPD diagnosis, yet might indicate a worse clinical course than a different combination of five BPD symptoms, which would receive a BPD diagnosis. This is largely due to the fact that all PD symptoms are not viewed as having equal clinical weight, though they are all treated equivalently in the current DSM. Thus, clinicians already will emphasize certain symptoms more heavily in their PD diagnoses than other symptoms (Cooper, Balsis, & Zimmerman, et al., 2010). Still, there is no room in the current diagnostic system to indicate differing severity depending on specific symptoms nor is there a way to indicate a subthreshold combination of symptoms that still warrants significant clinical attention.

Furthermore, the current diagnostic cutoffs are not based on empirical evidence, but instead have been chosen somewhat arbitrarily (Kamphuis & Noordhof, 2009). Often the argument given to justify the thresholds is that they are generally about half of the symptoms of a given disorder. Though establishing a demarcation between the presence and lack of a disorder is necessary and has utility, particularly for treatment and communication of patient information, the cutoff points in the current categorical system have little scientific basis. Using a model of psychopathology aside from the medical-categorical system, such as a hybrid or dimensional model, would change our reliance on specific diagnostic cutoffs (Hopwood & Zanarini, 2010; Morey & Zanarini, 2000; Skodol et al., 2011). Within a more dimensional system it would be possible to indicate impairments, even if they were subthreshold, which would lessen the importance of chosen thresholds. Regardless of what kind of system is employed, it is important that cutoffs are meaningful.

An additional problem with the current diagnostic cutoffs is that no distinction is made based on patient severity. A patient who meets five criteria for BPD, and gets a diagnosis of BPD, is not described any differently than a patient who meets for all nine criteria, even though meeting for nine criteria in most cases implies a worse prognosis and a different course of treatment. The challenges of diagnostic cutoffs extend as far as to each individual symptom, many of which exist on dimensions but are rated as either present or absent. For example, symptoms such as identity confusion or entitlement most certainly exist on some sort of continuum as opposed to on a yes or no dichotomy, as they are currently assessed (Westen et al., 2003). Though it may always be necessary to identify boundaries in order to have a meaningful classification system, it is important that the choices for cutoffs, even at the level of symptom severity, are guided by theory and validated models.

The combined impact of polythetic dichotomous diagnoses means that in our diagnostic system for BPD, we include a variety of patients who share very little symptom overlap and we simultaneously exclude many patients from receiving diagnoses despite potentially severe clinical presentations. This creates problems for research and for treatment development. Though the decision to change all of the PD diagnoses from monothetic to polythetic criteria sets gave the diagnostic categories more flexibility, and has since been standard in PD diagnostics, the current classification system includes diagnoses, which are very broad, lacking specificity as a result of the heterogeneity of symptomatic manifestation. Simultaneously, it can also be argued that the diagnoses are too narrow, providing no way to identify certain patients with subthreshold levels of personality symptoms or to differentiate more severe BPD patients from milder presentations.

Psychiatric Comorbidity

One of the most challenging diagnostic issues across the board in DSM-IV is the high rate of comorbidity for psychiatric patients. Using DSM-IV diagnoses, there is widespread comorbidity between BPD and other Axis I and Axis II psychopathology. Like in all fields of medicine, diagnostic criteria sets are designed to help guide clinicians toward the correct diagnosis of each patient and to enable differentiation between one disorder and other similar disorders. In theory, this would enable a clinician to understand a specific case as it relates to a paradigmatic example of that diagnosis. The supposed overlap between a patient's clinical presentation and the DSM's delineation of a diagnostic criteria set should cue a clinician to a narrow set of treatment options.

Still, in DSM-IV, there are very few stipulations about meeting for multiple disorders simultaneously. Thus, as they are conceptualized in DSM-IV, psychiatric diagnoses co-occur routinely and to such an extent that it becomes challenging to argue that the diagnoses represent distinct clinical entities (Mineka, Watson, & Clark, 1998). The question of diagnostic comorbidity, and what it means for our understanding of psychopathology, and is particularly pertinent to PDs, as they have been evaluated on a separate axis in DSM-IV and therefore routinely co-occur with diagnosed Axis I disorders (Clark, 2005; Krueger, 2005; Krueger & Markon, 2006).

The high comorbidity rates seen in patients with BPD seem to indicate that this problem might be more ubiquitous in this diagnosis than in other diagnoses. The rates of psychiatric comorbidity frequently complicate case conceptualization and treatment of BPD (Zanarini et al., 1998a). One study of inpatients with BPD found that borderline patients had comorbid PDs at the following rates: 31 % for odd cluster disorders, 73 % for anxious cluster disorders, and 40 % for dramatic cluster disorders (Zanarini et al., 1998b). In a study of the rates of comorbidity between BPD and Axis I psychiatric diagnoses, 75 % of individuals with a lifetime BPD diagnosis met criteria for a lifetime mood disorder and 73 % met criteria for a lifetime substance abuse disorder (Grant et al., 2008). In a study looking at the longitudinal course of anxiety disorders in borderline patients, 80 % of the borderline patients met for a simultaneous anxiety disorder at baseline (Silverman et al., 2012). Another recent study found that the rates of comorbid Axis I and Axis II disorders and BPD in adolescents were particularly high, compared to a clinical control group, particularly for mood, eating, dissociative and substance abuse disorders, and for cluster C personality disorders (Kaess et al., 2013).

This is not an exhaustive list of studies on BPD comorbidity but these selected studies should indicate the extent to which this problem exists within the BPD diagnosis. Further, the literature on this topic points to the degree to which it is a vexing problem for researchers, trying to conceptualize and differentiate these disorders. From a theoretical and philosophical standpoint, comorbidity begs questions about the nature of our diagnostic system and the individual integrity of each DSM diagnosis, if disorders so frequently co-occur. From a practical standpoint, the problem of comorbidity impacts the ability for researchers to study and develop treatments for disorders. In order to conduct research on a treatment for BPD, is it important for participants to meet for BPD alone? If the answer is no, then it would be hard to know whether positive improvements in the participant were due to changes in BPD or other comorbid disorders. If the answer is yes, then any participants in a treatment study would be aberrant from the typical BPD patient, who often presents with multiple disorders. Therefore, findings from this hypothetical treatment study would be hard to generalize. These questions are not simply theoretical; high rates of comorbidity serve to hinder effective research about the nature, course, and treatment for BPD.

From a clinical standpoint, the issue of comorbidity raises questions about which treatments to use. For many Axis I and Axis II disorders there are specific empirically validated therapies, which

have been shown to be effective for treating those disorders. A great deal of progress has been made in the arena of validated treatments for BPD. A number of treatments have been designed specifically to treat BPD, such as dialectical behavioral therapy (DBT), mentalization-based treatment (MBT), transference-focused psychotherapy, and schema-focused therapy, among others (Bateman & Fonagy, 2004; Clarkin, Yeomans, & Kernberg, 1999; Linehan, 1993; Young, Klosko, & Weishaar, 2003). Still, when patients suffer from multiple disorders, for example BPD and an eating disorder or BPD and an anxiety disorder, it is difficult for clinicians to know which disorder should be tackled first, or whether both disorders should be treated simultaneously. If a clinician treats a patient with multiple disorders, which given the high rates of comorbidity is not unusual, what would be the ideal treatment to use, for that patient, with that combination of disorders?

We are in a new age for treatments for psychiatric disorders, with a growing acknowledgement of the importance of using empirically validated treatment modalities. Yet the problem of comorbidity means that it is difficult for a clinician to know how to target multiple disorders simultaneously, even when there are validated treatments for the individual disorders. Recently, there have been efforts to modify existing treatments in order to target more than one disorder at a time, when those disorders are commonly comorbid with one another. For example, DBT has been modified to treat BPD comorbid with drug dependence (Linehan et al., 1999, 2002). Still, this method of treatment development is inefficient, as it is nearly impossible to tailor existing treatments to include all of the various possible combinations of comorbidities. The question of comorbidity in a medical-categorical classification system, then, ultimately impacts the type and quality of care that patients receive and creates challenges for the clinician looking for a proper course of action for treating a patient. Taken together, the research suggests that the problem of comorbidity in the BPD diagnosis is prevalent, pertinent for adolescents with BPD diagnoses, and impacts the quality of patient treatment and care.

Inadequate Coverage

Comorbidity is a problem in that diagnoses do not seem to reflect distinct entities. A seemingly opposite problem in the current diagnostic classification system is that of the ubiquity of the "not otherwise specified" (NOS) label. An NOS qualifier can be appended to any Axis I or Axis II diagnosis and is given when a clinician determines that a patient suffers from a certain class of psychiatric disorders but does not meet enough symptoms to receive a diagnosis within that category. Thus, an NOS diagnosis describes the overall framework of a person's dysfunction but provides no incremental specificity about the nature of the impairments. Unlike comorbidity, which is the result of multiple distinct categorical diagnoses, the NOS diagnosis indicates that a certain amalgam of symptoms is impairing, but is impossible to further categorize beyond the family of disorders in which it falls (Widiger & Trull, 2007).

A diagnosis of personality disorder—NOS (PDNOS) is given when a patient meets general diagnostic criteria for a PD but not the full criteria for any single PD. The general diagnostic criteria for a PD indicate impairments in cognition, affect, interpersonal relationships, and impulsivity, without specifying details of impairment. Studies looking at the prevalence and usage of the PDNOS diagnosis found that the best estimate of relative prevalence of PDNOS, as compared to Axis II prevalence without PDNOS, was in the range of 21–49 % (Verheul & Widiger, 2004). In reality, it is the most frequently assigned PD diagnosis (Clark, Watson, & Reynolds, 1995). A common example of a PDNOS diagnosis is when a patient presents with features of multiple personality disorders but does not meet criteria for any single PD. Thus, the only descriptive information in the diagnosis is that a patient is suffering from multiple symptoms of maladaptive personality, which cause clinically significant distress or impairment. No information is given about the nature of the impairments or what the patient is experiencing. In a study of the PDNOS

diagnosis, patients receiving a diagnosis of PDNOS did not differ significantly from those that met for a single specific PD (Verheul, Bartak, & Widiger, 2007). Furthermore, in terms of quality of life and psychosocial functioning, subjects in the study with PDNOS had a higher degree of pathology, similar to that of subjects with a specific PD diagnosis, and not to Axis I patients or healthy individuals (Coccaro, Nayyer, & McCloskey, 2012). This research lends credence to the clinical severity of patients with a PDNOS diagnosis and the importance of properly treating these patients. It also supports the notion that DSM-IV is not providing adequate coverage for patients with pathological personality traits.

Despite the seriousness of the PDNOS diagnosis, this diagnosis conveys nothing about the specific maladaptive patterns and behaviors that characterize patients. This makes developing an effective treatment plan based on the diagnosis nearly impossible. In some ways, it is worth thinking about PDNOS as an example of a polythetic diagnosis writ large. Similar to the problems for research and treatment created by the fact that 256 different combinations of symptoms all fall under the category of a BPD diagnosis, there are potentially thousands of symptom combinations that all warrant the PDNOS diagnosis.

Needless to say, there is very little that binds PDNOS as a unitary diagnosis. As a clinician, knowing how to treat an amalgam of unspecified symptoms that are characterized by an enduring, stable, and pervasive pattern of a combination of maladaptive cognitions, affect, interpersonal functioning, and problems with impulse control, and which leads to clinically significant distress, is quite daunting. The lack of descriptive information about the disorder also hinders the ability to produce a conclusive body of research about PDNOS. Aforementioned studies have established the necessity of maintaining PDNOS as a diagnostic category and have shown that the disorder can be quite impairing. It is important, then, to have a way to identify these patients. Still, as it stands, it is challenging to conduct meaningful research on such a nebulous disorder, with literally thousands of potential presentations. It is equally challenging to establish treatment standards for this patient population.

Lack of Diagnostic Stability

To date, PDs have been distinguished from Axis I psychopathology in that they are considered to be more stable, pervasive, and enduring than Axis I disorders (Gunderson & Pollack, 1985; Widiger & Shea, 1991). According to DSM-IV, one of the defining features of PDs is that they onset during adolescence and remain stable over time. Thus, the very nature of a PD diagnosis rests on its stability and the enduring nature of the dysfunctions present. Research has shown that the stability associated with PDs, and specifically with BPD, in reality is an elusive concept. In a longitudinal study on the course of BPD, Zanarini et al., (2012) found that after 16 years of prospective follow-up, 99 % of the patients that had originally met for BPD had experienced at least a 2-year remission of symptoms. For a disorder that by its very definition is expected to be stable over time, these results are surprising.

Furthermore, according to DSM-IV, BPD is supposed to begin before a person reaches adulthood and continue into adulthood. Studies on the temporal stability of adolescent BPD, though, have produced results that are inconsistent with this criterion. In a study of a small sample of hospitalized borderline adolescents, only two out of the 14 cases continued to receive a diagnosis of BPD after 3 years post-hospitalization (Meijer, Goedhart, & Treffers, 1998). In a similar study of patients with BPD in the community, researchers found that less than one third of the adolescents who met for BPD at baseline continued to meet criteria for the disorder after 2 years of follow-up (Bernstein et al., 1993).

Examining these findings, Bornovalova et al., (2009) point out that many of the studies looking at the temporal course of BPD in adolescence use dichotomous diagnoses of BPD (as it is defined in DSM). As explained above, a dichotomous diagnostic system means that an affirmative

diagnosis of BPD can hinge on a single diagnostic criterion, making it relatively easy to transition from meeting to not meeting the diagnosis. This is regardless of whether a person continues to exhibit a number of symptoms and traits associated with the disorder. Chanen et al. (2004) conducted a study comparing the BPD diagnosis in adolescents measured categorically to a dimensional measurement of BPD. They found the categorical stability to be low over time in adolescents, but that stability of the diagnosis was considerably higher when measured dimensionally. More generally, a paper from the Children in the Community study on the developmental course of PDs found that nearly all PD symptoms decline linearly between the ages of 9 and 27 (Johnson et al., 2000). Thus, despite the fact that stability over time has been considered a defining feature of PDs generally, and of BPD specifically, empirical studies evaluating the stability of the diagnosis have shown that it generally remits over time, in both adults and children. This problem is compounded by our current categorical diagnostic system. It is probable, as previous research suggests, that a dimensional model of diagnosing PDs would be more effective at capturing personality pathology present in children and adolescents, by better accounting for shifts in personality that are age appropriate.

DSM-5 and Hopes for Beyond

Over the last 20 years, both the theoretical and practical shortcomings of the DSM-IV diagnostic criteria for PDs, as described above, were significant enough to inspire many researchers in the field to consider DSM-5 as an opportunity for constructive evolution. The shortcomings enumerated above as well as data supporting a dimensional model of personality pathology in large-scale samples indicated that the future of PD diagnoses generally, and BPD specifically, rested on developing a dimensional approach to diagnosis (Benjamin, 1993; Clark, 1993; Cloninger, Svrakic, Bayon, & Przybeck, 1999; Livesley, 1998; Miller, Morse, Nolf, Stepp, & Pilkonis, 2012; Widiger & Costa, 1994). Some of the most salient elements of the process of developing this dimensional model of BPD for DSM-5 will be discussed and the changes proposed for DSM-5 will be outlined briefly.

The process of developing DSM-5 highlighted the challenges of creating a unified definition of BPD based on the vast research literature that exists, as well as accounting for the various individual opinions in the field. Additionally, untenable ideas that were part of the brainstorming process were made public on the DSM5.org website, and people balked at what seemed to be an overly radical and rash departure from the current model. Ultimately, the final decision for DSM-5 was to include two forms of the PD section. The first model, presented in Section II of DSM-5, is the model of PDs delineated in DSM-IV (diagnostic criteria and codes). In Section III of DSM-5 there is an alternative model of PDs, meant to address the shortcomings of DSM-IV, and designed for further research. The intention is that the constructs developed in Section III will become features of Section II in future editions of the DSM, upon further research and validation. Some of the watershed moments in coming to this final decision will be discussed below, as well as an explanation of the alternative model of PDs.

First, though, it is important to recognize that the creation of the DSM has always been a political process. Scientific research necessarily has no end date. In an effort to create a provisional diagnostic system, which has many important practical implications, people create arbitrary deadlines and end dates. No matter when these dates are set for, they inevitably come in the middle of the research process. Thus, each DSM revision hinges on research that has been completed, and cannot include the lines of research that are still in progress. Furthermore, the revision process is often dictated by work groups, which are based on traditional DSM chapter headings, such as mood disorders, anxiety disorders, and personality disorders. Given what we know about psychopathology, and how much biological, clinical, and diagnostic overlap exists between disorders, this method for writing

the DSM does not allow for substantial cross-fertilization or collaboration between the different work groups. Ultimately, the process of writing the DSM ends up eclipsing potentially fruitful and elucidating avenues of research.

DSM-5 was overseen by a task force, comprising primarily psychiatrists, and was co-chaired by David Kupfer, MD, and Darrel Regler, MD. The American Psychiatric Association (APA) Board of Trustees in turn oversaw the task force. The individual members of the task force also served as chairs of the specific work groups. In thinking about changes in PDs generally for DSM-5, some members of the Personality and Personality Disorder (P & PD) Work Group felt committed to maintaining continuity with DSM-IV, despite the drawbacks. Other members, in response to the limitations of the traditional medical-categorical perspective on psychopathology, pushed for changes based on an individual difference perspective, in which PD psychopathology would be understood using quantitative models developed from analysis of observable signs and symptoms. In these models, data would delineate the constructs, thereby allowing them to be more inductive than deductive. That is to say, the constructs of individual PDs would flow from the data itself, as opposed to from preconceived notions of a disorder, based primarily on clinical experience, as this approach has frequently been shown to be flawed (Grove, 2005). This iterative method of diagnosis development differs from past revisions of the DSM, in which the data collection has primarily been dictated by the existing diagnostic categories, circumscribing the realm of possible findings.

Using this novel guiding principle, many different quantitative individual difference models were considered and inspiration was drawn from both basic personality and personality disorder research (Harkness & McNulty, 1994; Livesley, 2003; Widiger, Costa, & McCrae, 2002). Meetings about the future of PDs in DSM-5 began as early as 2004. Researchers in attendance agreed that a dimensional focus was necessary for guiding the thinking about the structure of PDs. There was enthusiasm for developing a new dimensional approach to better articulate the way personality pathology exists, and as a way of creating continuity between the literatures on basic personality and maladaptive personality. Under the direction of the task force, a hybrid model of PDs was developed, combining the elements of both a dimensional and categorical diagnostic system. Upon completion, in November 2012, the task force endorsed this novel model of PDs, but the board of trustees voted to maintain the DSM-IV PD categories. The final decision was to include the DSM-IV PDs, with the criteria unchanged, in Section II of DSM-5, and to include the alternative model of PDs in Section III of DSM-5 (Krueger, 2013).

In the DSM-5 alternative model, PDs are characterized by impairments in personality functioning and pathological personality traits. Only six out of the current ten PD diagnoses are explicitly included in this model. They are antisocial, avoidant, borderline, narcissistic, obsessive–compulsive, and schizotypal PDs. In addition to these specific PDs, a novel feature found in Section III is the diagnosis of PD-Trait Specified (PD-TS), which is given when the general diagnostic criteria for a PD are met, meaning the presence of impairments in personality functioning and pathological personality traits, but none of the six specified PDs is appropriate. We will explain PD-TS below, but the hope is that it will help fill the diagnostic niche of PDNOS, while providing descriptive information about the exact maladaptive traits present. In this way, PD-TS will avoid the pitfalls of the PDNOS diagnosis, such as inadequate coverage and lack of clinical utility, by providing important clinical information and diagnostic coverage of this patient population.

The general criteria for PD in the alternative model require two determinations, which represent the hybridism of the model. The first determination is that there is a level impairment in personality functioning, either within the person's concept of self, or interpersonally. At the same time, in order to meet for a PD in the alternative model, one or more pathological personality traits must be present. The other general criteria for a PD in Section III of DSM-5 include relative stability over time tracing back to

adolescence or early adulthood; relatively pervasive and inflexible across situations; not better explained by another medical condition or substance use; and not better understood as normal for a person's developmental stage or sociocultural environment. All DSM-5 Section III PDs described by criterion sets and PD-TS meet these general criteria, by definition.

In the alternative model of PDs, impairments in personality functioning are defined as disturbances in self- and interpersonal functioning, but they are measured on a continuum. Self - functioning involves identity and self-direction; interpersonal functioning involves empathy and intimacy. In order to measure these, the alternative model provides The Level of Personality Functioning Scale, measuring personality functioning from no impairment to severe impairment (Bender, Morey, & Skodol, 2011). Thus, though there continue to be cutoffs for delineating specific PDs in this model, there still exists a way to indicate subthreshold impairment. All people, regardless of the presence or absence of personality pathology, can be described using the Level of Personality Functioning Scale.

Beyond the assessment of impairment in personality functioning, the alternative model for PDs has a system for assessing and recording pathological personality traits. Pathological personality traits are organized into five broad domains: Negative Affectivity, Detachment, Antagonism, Disinhibition, and Psychoticism. These domains consistently map onto the highly replicated Five-Factor Model (FFM), and can be viewed as the maladaptive extreme ends of the domains of FFM personality traits (Fruyt et al., 2013; Thomas et al., 2013). These domains have also been further broken down into 25 more specific trait facets, providing space to give more descriptive information about a person's specific personality profile and impairments. Maladaptive traits were identified through an iterative process starting with 37 trait facets which were ultimately narrowed down to 25 trait facets empirically (Krueger et al., 2012; Krueger et al., 2011). These traits represent the maladaptive poles on personality dimensions with polar opposites. Noting the high scores on the opposite poles can be important as well, as they can serve as protective factors, and may facilitate treatment or positive coping. Again, this system offers a dimensional way of describing all people's personalities, regardless of whether impairment is present or not. This rubric is important because it provides a way of assessing and describing subthreshold personality pathology. Having a way to describe personality traits can also be helpful in treating people with Axis I disorders such as anxiety and mood disorders. Personality plays an important role in all psychopathology and functioning and, thus, having a system for describing personality traits is a huge step forward in the diagnostic system.

In addition to impairment in personality functioning and the presence of pathological personality traits, the model of PDs in Section III of DSM-5 requires that these features are relatively pervasive and relatively stable. They are supposed to be pervasive in a range of contexts, maladaptive, and inflexible, meaning that these patterns lead to disabilities in social, occupational, or other domains. Similarly, the impairments in functioning and traits are supposed to be relatively stable over time. It is important to highlight the word *relatively* which is used in the alternative model of PDs in DSM-5. Despite the research discussed above, which has shown that personality disorders do change and improve over time, the language from DSM-IV does not accommodate for the changes in the disorder over time. As laid out in the PD section developed for DSM-IV, the BPD diagnosis is primarily defined by specific symptom patterns, which have been shown to be mutable over time, as opposed to the more enduring trait and affective features of the disorder (Hopwood & Zanarini, 2010). Thus, the fact that the diagnosis has been defined by its stability does not optimally describe the disorder in nature

The alternative model for DSM-5 lays out room for change in the diagnosis or symptoms, incorporating research about disorders in time. Even personality traits, which are considered to be relatively stable over time, do change over the course of the life-span (Roberts & DelVecchio,

2000; Roberts, Walton, & Viechtbauer, 2006). Hence the alternative model acknowledges that maladaptive personality patterns can evolve over time. Without this understanding, a discussion of treatments seems futile. Hopefully this linguistic change will augur an increased focus on developing treatments for these challenging disorders more broadly (for example, most contemporary treatment research on PDs focuses primarily on BPD). It is important to also note that DSM-5 did away with the multiaxial system, which had Axis I and Axis II disorders assessed separately. This change in the structure of the DSM serves as an acknowledgment that personality disorders are not different in kind than Axis I disorders, another way of indicating the importance for developing treatments for these disorders (Markon, 2010).

The six PDs specified in DSM-5 are all characterized by the general impairment in personality functioning, but also by specific personality traits that make up the disorder. In order to meet for BPD in the alternative model in DSM-5, a person must exhibit four or more pathological personality traits out of seven. The traits include Emotional Lability (as an aspect of Negative Affectivity), Anxiousness (Negative Affectivity), Separation Insecurity (Negative Affectivity), Depressivity (Negative Affectivity), Impulsivity (Disinhibition), Risk Taking (Disinhibition), and Hostility (Antagonism). Specifically, in order to receive a diagnosis of BPD, a person must endorse at least one of the following: impulsivity, risk taking, or hostility. Thus, a BPD diagnosis hinges on a general assessment of overall personality impairment as well as a confluence of specific maladaptive personality traits.

One could argue that this system seems likely to perpetuate the problems described earlier with polythetic diagnoses seen in DSM-IV. An initial response is that these traits have been shown to have good coverage of DSM-IV BPD (Hopwood, Thomas, Markon, Wright, & Krueger, 2012). As Trull (2005) argues, it is important that a dimensional model of personality pathology is coordinated with DSM-IV PDs in order to ease the transition between a categorical model and a dimensional system. Thus, the specific PD categories laid out in the alternative model of DSM-5 can be seen as a middle ground between the DSM-IV categorical system and a purely dimensional model of psychopathology. More noteworthy for clinical purposes, though, is that in Section III of DSM-5, if a person meets for a set of maladaptive traits that are not covered in one of the six diagnostic categories, there still remains a way of identifying and describing personality pathology through the PD-TS diagnosis. Using categorical diagnoses, if someone does not meet for the specific symptoms and traits delineated in the ten PD categories, they would not receive a diagnosis, or they would receive the problematic PDNOS diagnosis. In the new model, there is the flexibility to give a general diagnosis of PD-TS while still indicating specific maladaptive traits and levels of personality functioning descriptions.

A clinician diagnosing a patient with PD-TS would indicate which maladaptive personality trait or traits are present, giving an indication of general impairment and specific information about the dysfunctional personality presentation. This is the novelty of the PD-TS diagnosis, as clinicians will have information about all five domains of personality and will be able to capture the scope of a person's personality functioning, not limited to a specific diagnostic label. In order to meet for PD-TS, a person would have to meet for moderate or greater impairment in personality functioning and would also have to meet for one or more pathological personality trait domains (Negative Affectivity, Detachment, Antagonism, Disinhibition, and Psychoticism) or specific trait facets within those domains.

In addition to providing an efficacious solution to the problem of PDNOS, this new trait-based system also ameliorates some of the aforementioned diagnostic problems associated with the high rates of comorbidity. A trait-based diagnostic system would make it unnecessary to diagnose an individual with multiple PDs. Instead, people meet for personality impairment and personality domains and facets. Even if persons do meet for one of the six specific PDs, clinicians would still have the classification tools to indicate whether they meet other personality

features. Meaning, even if they do meet for one of the specific PDs, if they present with other maladaptive personality traits, there is a framework through which to record those as well. For example, psychoticism is not included as a specific trait necessary to receive a diagnosis of BPD under this model. Still, if a patient endorses features of psychoticism, such as cognitive or perceptual dysregulation, it would be important for a clinician to have this information and to have a method with which to convey this information to others involved in this patient's case. Using the model in Section III of DSM-5, a patient would not receive a second, separate PD diagnosis. This would mean one would no longer see patients presenting with multiple PDs. While the issue of comorbidity with other disorders previously on Axis I remains, this dimensional model provides one method for solving the diagnostic problem of comorbidity among PDs. As many Axis I disorders could be understood in a more dimensional fashion as well, Section III of DSM-5 provides a window for reconceptualizing psychopathology in a way that would solve certain of the most nagging diagnostic problems.

The trait aspect of the model has been operationalized using the Personality Inventory for DSM-5 (PID-5; Krueger et al., 2011), and levels of personality impairment can be measured using the Level of Personality Functioning Scale. The PID-5 can be administered via self-reports filled out by the patient or in its informant report form (Markon, 2013). The PID-5 is another of the novel features of DSM-5 Section III in that it directly ties an assessment instrument to the DSM and enables clinicians to assess the models of personality variation described in the DSM using an instrument owned and distributed by the APA. Although the PID-5 is copyrighted by the APA, it is also freely available for clinical use and for research, allowing for a more fluid transfer of information between the clinical and research communities, by providing a unifying measure of assessing PDs.

Overall, the changes that will appear in Section III of DSM-5 help address many of the concerns with the DSM-IV PD (which will also appear in DSM-5) diagnostic criteria, that were described above. Additionally, these changes, if implemented, could have positive impacts on research, diagnosis, and treatment of BPD, which would hopefully influence the quality of care and treatment for adolescents and children experiencing personality pathology. Though the outcome of the decisions made regarding PDs in DSM-5 reflects ambivalence and uncertainty in the field, it also indicates the investment that researchers and clinicians have in better understanding and more correctly describing PDs. Moving forward, hopefully the energy and momentum that were created in the process of conceiving and writing DSM-5 can propel the field to continue putting research time and money into fleshing out a more accurate and useful conception of PDs, to further the field in research and patient care.

The Upside to Change

It is important to acknowledge that many of the features of Section III of DSM-5 as well as other suggestions proposed in this chapter can seem drastic. Much of the opposition to the changes proposed for DSM-5 and for BPD, specifically, has revolved around the structural and institutional challenges inherent in doing a systematic overhaul of the PD section in the DSM. Changes would have reverberations to areas as far reaching as insurance coverage, governmental funding, and the structure of psychiatric hospitals, in addition to having implications for current lines of research and centers of study, all based on DSM-IV categories. Additionally, improving the diagnostic validity also requires discarding elements of diagnosis that have come to feel synonymous with BPD. Fundamental changes such as these can be difficult. Still, it is important to note the ways that changes in the diagnosis could positively impact clinical care.

One of the primary arguments for maintaining categorical diagnostic criteria sets is that they create a unified conception of a disorder, thereby aiding treatment development and helping clinicians to hone in on a specific treatment

plan for a patient with a given diagnosis (Kendell & Jablensky, 2003; Kraemer, Noda, & O'Hara, 2004). Yet for BPD, the diagnostic confusion delineated above manifests in the array of validated treatments available for the disorder, all of which focus on different features of the diagnosis. For example, the most frequently utilized and highly researched treatment for BPD is DBT. DBT was originally developed by Marsha Linehan to treat parasuicidal and borderline women (Linehan 1987a, 1987b) and is now used broadly, including for treating adolescents with borderline features (Rathus & Miller, 2002). DBT rests on Linehan's biosocial theory of BPD, which is essentially a diathesis-stress model of the disorder. At its core, the biosocial theory of BPD argues that these patients have a biological propensity toward emotional vulnerability, or the trait of experiencing frequent and intense emotional reactions, which interacts with an "invalidating environment" and results in dysfunctional behavioral patterns (Linehan, 1993).

Based on this theory, emotional vulnerability, which is reflected in the affective symptoms of the DSM-IV BPD diagnosis, rests at the core of the disorder and the other behaviors and symptoms associated with BPD stem from this basic vulnerability. Hence, DBT operates primarily to teach patients the skills necessary to regulate their emotions while also focusing on improving secondary skills deficits, through modules such as interpersonal effectiveness and distress tolerance (Shearin & Linehan, 1994). Still, despite the fact that this is the most frequent treatment for BPD, given the current polythetic diagnostic system, it would be possible to meet criteria for BPD without primarily endorsing the BPD symptoms that seem most tied to problems with emotion regulation. Though this presentation of the disorder is likely uncommon, in this scenario, one could argue that DBT would not be targeting the core dysfunction and would not be ideal, even for this borderline patient.

Similarly, other treatments for BPD assume different core dysfunctions that reflect different understandings of the essential nature of BPD. For example, MBT, also mentioned above, is another empirically validated therapy for BPD developed by Bateman and Fonagy (2004). MBT describes the dysfunctions we see in patients with BPD as stemming from their inability to mentalize. By this, they refer to the disorganized attachment experienced by borderline patients in their early relationships and their inability to think about mental states as distinct from the mental states of others, "yet potentially causing actions" (Bateman & Fonagy, 2004, p. 36). This implies that the fundamental dysfunction for people with BPD described by Bateman and Fonagy is both cognitive and interpersonal. They hypothesize that BPD patients have an inability to understand the underlying thoughts that might lie behind the overt behavior of others, particularly in emotionally charged interpersonal situations. This inability causes difficulties in forming intimate relationships and ultimately in self-regulation, leading to maladaptive behaviors, emotion dysregulation, and impulsivity. This guiding theory of BPD places interpersonal relationships at the center of disorder, with emotion dysregulation resulting from cognitive difficulties in relationships. Still, it is possible to meet criteria for BPD without endorsing any symptoms pertaining to interpersonal relationships and, thus, a treatment that focuses on developing a capacity for engaging effectively in interpersonal relationships might not pertain to certain patients with BPD.

These two examples of different treatments for BPD each with a different basic understanding of the mechanisms behind the disorder, underscoring different DSM-IV symptoms, highlight the challenges inherent in the current diagnostic system for treatment development. When a disorder like BPD is based on polythetic, categorical symptoms, it becomes hard to develop a unified conception of the disorder, as presentation can vary substantially between cases. Widely varying clinical presentations mean that treatment development necessarily emphasizes and focuses on specific aspects of the diagnosis, to the exclusion of others.

Furthermore, the validated and effective use of these treatments for other disorders indicates that the treatments likely are not targeting disorder-specific dysfunctions. Rather, it is

possible to understand these treatments as targeting clinical features found in people with BPD, but also people with other psychiatric disorders, explaining their generalizability. For example, DBT targets emotion dysregulation, akin to the lower order personality trait of emotional lability. Though emotional lability is seen in BPD, it can also be found in diverse psychiatric disorders. Thus, it can be argued that DBT effectively targets emotional lability, explaining its effectiveness in treating other disorders, where problems with emotion regulation are also central. As a result, DBT has also been used for treating eating disorders (Linehan & Chen, 2005), suicidality in adolescents and adults (Rathus & Miller, 2002), and depression in older adults (Lynch, Morse, Mendelson, & Robins, 2003), to name just a few examples. MBT operates under the assumption that an inability to mentalize is the key dysfunction among borderlines, but it, too, has been developed for use in other populations, such as for eating disorder patients (Skårderud, 2007). It can be argued that patients with certain other disorders also have difficulty mentalizing, explaining its potential effectiveness for more widespread use.

Not only have these treatments been used successfully for other disorders beyond BPD, indicating that they are targeting some patient characteristics that are not disorder specific, but many of the treatments for BPD discussed above have been shown to be effective for treating BPD, despite operating on different theories of the disorder. Livesley (2012) argues that the various treatments for BPD, most of which focus on a single impairment, do not target the multiple etiologies or the heterogeneity of impairments present in patients with BPD. The practical and theoretical challenges of the diagnosis, therefore, hamper the effectiveness of the various treatments for the disorder. Livesley advocates for an integrated treatment approach, based on an understanding that the disorder is a "pervasive regulation disorder involving emotional, interpersonal, self, cognitive, and behavioral dyscontrol" (Livesley, 2012, p. 58). Given the diversity of symptom presentations in patients with BPD, treatment should begin with general mechanisms of change identified in all of the treatments and specific interventions should be utilized as different problems come to fore.

Not only have theoretical difficulties in the BPD diagnosis provided challenges for treatment development, but the strict categorical diagnosis has also negatively impacted treatment delivery for children and adolescents displaying BPD symptoms. As it stands, many clinicians are wary about giving out PD diagnoses to children and adolescents. Traditionally, literature about children's personality development has focused on temperament, which is viewed as biological, evident early in life, and stable throughout development, unlike personality traits. The theory holds that personality continues to change and evolve throughout childhood and adolescence and, thus, it is hard to make statements about a person's ultimate personality formation. Over time, temperament is believed to give way to more stable personality traits (Frick, 2004). To give a diagnosis to children and adolescents, some believe, denies the normal fluid developmental processes that occur during adolescence (Miller, Muehlenkamp, & Jacobson, 2008).

Research though now shows that personality traits can be identified reliably in childhood, and that certain traits can be measured as early as 3 years old (Halverson et al., 2003). Studies have also shown that childhood personality can link up with research on child psychopathology and with the adult personality-psychopathology literature (Tackett, 2006). Thus, there is evidence that understanding personality pathology in children and adolescents is not out of line with research on basic personality.

Still, as is described above, there is limited stability in PDs in children and adolescents, when they are measured categorically. Therefore, because BPD continues to carry a great deal of stigma (despite efforts to destigmatize the disorder) clinicians remain wary about attaching these labels to children and adolescents, especially if they may prove to be transient. Still, many traits associated with BPD are correlated with high-risk behaviors, and benefit from clinical attention and early intervention. Thus, finding a way to signify the presence

of these traits, without attaching the rigid BPD label, with its various clinical connotations, would be particularly useful for treating adolescents and children with BPD symptoms. More widespread use of the dimensional and trait-based diagnostic system presented in DSM-5 could allow clinicians to provide clinically pertinent information by delineating the relevant maladaptive traits exhibited by children and adolescents. In this way, clinicians could highlight the specific areas of concern for treatment. This would enable patients to get the treatment they need while hopefully lessening the long-term implications of a premature diagnosis of BPD.

Though the fields of personality research and treatment development continue to seem remote from one another in the scientific community, there are already exciting efforts to build bridges between these two fields. For example, researchers have been working on a novel personality-based treatment for adolescents, called *PreVenture*, which is a personality-targeted intervention for adolescent alcohol misuse. This early intervention program has been shown to have promising results for adolescent substance use (Conrod, Castellanos-Ryan, & Strang, 2010). The idea is that by intervening on the level of personality traits, we can more effectively prevent certain negative outcomes. This is one example of ways that a more personality-focused model of psychopathology could positively impact treatment. It also provides a model for pan-diagnostic treatments, which intervene on the personality level and thus can apply to an array of disorders.

The above is just one example of an exciting synthesis of research on basic personality, abnormal personality, and treatment. Ultimately, the DSM-5 process brought together people from a wide variety of research areas throughout the psychology and psychiatry communities and opened many conversations and opportunities for collaboration between researchers in diverse research areas. Despite frustration by many with the process and the outcome, it opened a discussion about a research and clinical area that is clearly of great importance for many. Instead of closing the discussion with the arbitrary end point of the publication of DSM-5, it is important that these cross-cutting conversations continue, and these research communities continue working together to best describe, classify, and treat psychopathology. Our hope is that moving beyond DSM-5, we will continue to see exciting collaborations between these various research areas, and ongoing discussions about classification, treatment, and research about BPD, PDs, and personality and psychopathology, particularly as they impact the youngest sufferers.

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Suggested Reading

Krueger, R. F., Deringer, J., Markon, K. E., Watson, D., & Skodol, A. E. (2012). Initial construction of a maladaptive personality trait model and inventory for DSM-5. *Psychological Medicine, 42*, 1879–1890. (This article introduces the PID-5, or the Personality Inventory for DSM-5, which provides an assessment tool for the personality traits described in Section III of DSM-5. This instrument is copyrighted by the APA (American Psychiatric Association) but it is freely available for use in clinical and research settings by downloading it from: http://www.psychiatry.org/practice/dsm/dsm5/online-assessment-measures#Personality.)
Livesley, W. J. (2012). Moving beyond specialized therapies for borderline personality disorder: The importance of integrated domain-focused treatment. *Psychodynamic Psychiatry, 40*, 47–74. (This article offers an argument for a more integrated and less specialized approach for treating BPD patients. Given well-established problems with DSM-IV and DSM-5 Section II personality disorder diagnoses and the changes suggested in Section III of DSM-5, this model for a more fluid attitude toward treating BPD patients provides a useful lens for thinking about treatment in light of suggested changes.)
Markon, K. E., Krueger, R. F., & Watson, D. (2005). Delineating the structure of normal and abnormal personality: An integrative hierarchical approach. *Journal of Personality and Social Psychology, 88*, 139–157. (This article provides a useful model for understanding the integration of normal and abnormal personality. The model of personality pathology presented in Section III of DSM-5 moves towards this integration, which is important for better synthesizing the currently disparate literatures of personality pathology and normal personality.)
Tackett, J. L. (2006). Evaluating models of the personality-psychopathology relationship in children and adolescents. *Clinical Psychology Review, 26*, 584–599. (In this article, Tackett explains how the personality and personality disorder literature pertain to adolescents and children, who are often left out of these models.)
Widiger, T. A., & Trull, T. J. (2007). Plate tectonics in the classification of personality disorder: Shifting to a dimensional model. *American Psychologist, 62*, 71–83. (This is a concise discussion of the most pressing intellectual and clinical problems found in the personality disorder model from DSM-IV, which has been carried to Section II of DSM-5.)

The Likely Classification of Borderline Personality Disorder in Adolescents in ICD-11

28

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The International Classification of Diseases, currently in its 10th revision (World Health Organization, 1992), is the official world classification of disease organised under the auspices of the World Health Organization. It is also the official classification of mental health disorders in the United States, despite the ubiquity of DSM, and although it tends to be overshadowed by its American competitor, it remains a respectable (Regier, Kaelber, Roper, Rae, & Sartorius, 1994) and widely used classification and also has the advantage of being easily linked to other disease systems, particularly in neurology. In the case of personality disorders, the recent decision of the American Psychiatric Association not to proceed with the DSM-5 recommendations for the reclassification of personality disorder (American Psychiatric Association, 2012; http://www.medscape.com/viewarticle/803884_8) has attracted more attention to the alternative new classification for personality disorders in ICD-11.

Status of Current Classifications of Personality Disorder

In approaching the ICD-11 classification of personality disorders the ICD-11 group were guided by several considerations: the unsatisfactory nature of current categorical classification (Livesley, Schroeder, Jackson, & Jang, 1994), the lack of stability of personality disorders as currently classified (Clark, 2007), the need to address the fact that personality dysfunction develops in childhood and adolescence but cannot be diagnosed until adult life, and the gross overlap between personality disorders as currently classified, leading to the wide use of the unsatisfactory diagnosis, PD-NOS (Verheul, Bartak, & Widiger, 2007; Verheul & Widiger, 2004). These considerations apply even more strongly to borderline personality disorder than most others. It is a condition that almost always appears in adolescence in some form, it is often associated with trauma and abuse, has a very erratic course with a wide range of outcomes, and tends to be diagnosed very frequently in association with other personality disorders. Recent research, particularly by Mary Zanarini and her colleagues, suggests that most patients lose the diagnosis of borderline personality disorder within several years of first diagnosis, although they and others have also found that despite symptomatic change there is continued impairment in terms of function (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2006, Zanarini, Frankenburg, Reich, & Fitzmaurice, 2012).

In addressing these problems, the ICD-11 work group for the revision of personality disorders decided on a radical solution to simplify the diagnostic system (Tyrer, Crawford, & Mulder, 2011; Tyrer, Crawford, Mulder, et al., 2011). The key

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aim of the World Health Organization in revising the ICD classification is to have a much simpler system with a good clinical utility. At the same time we recognised that it would cause great distress to some people to remove entirely the existing diagnostic system—as in the case of borderline personality disorder, it has been very productive in the development of treatments, including dialectical behaviour therapy (Linehan, Armstrong, Suarez, Allmon, & Heard, 1991), mentalising-based therapy (Bateman & Fonagy, 2004), schema-focused and cognitive behaviour therapy for personality disorders (Davidson et al., 2006; Giesen-Bloo et al., 2006), and Systems Training for Emotional Predictability and Problem Solving (STEPPS) (Blum et al., 2008), which have helped greatly in removing the old saw that personality disorders are untreatable.

Despite these advances, the diagnosis of borderline personality disorder remains controversial and most practitioners regard it as unsatisfactory. It also differs from other personality disorders in that its main operational criteria are symptoms rather than traits, it is fluctuating rather than pervasive, and its diagnostic usage by clinicians is much more like an Axis I than an Axis II disorder. I have argued the case that borderline personality disorder is neither a personality disorder nor borderline, as it satisfies all the criteria for a mixed mood disorder (fluxithymia, as the changes in mood are so dramatic and frequent; Tyrer, 2009). Although this may be a minority view, it was clear to the ICD-11 committee that it could not regard borderline personality disorder as a special case. Although some people argue for the diagnosis of borderline personality disorder on the grounds of clinical utility, as there is no doubt that the diagnosis is widely used and in our preliminary work for the World Health Organization we have found that it is diagnosed more often than any other personality disorder, it remains a diagnostic category without good empirical evidence for its existence. This perhaps is not surprising as the DSM and ICD diagnoses were all delineated by committees, who had very little evidence to go on in formulating their diagnostic categories and relied mainly on collective clinical experience.

The Proposed ICD Classification

The new classification abolishes all categories of personality disorder apart from the main one, the presence of personality disorder itself. Because the universal recognition of personality dysfunction is best represented on a continuum, different levels of severity indicate the band on the continuum where the person is at the time of the assessment. Of course, many people try and make a personality assessment as though it were a lifetime diagnosis, but there is abundant evidence that the severity of personality disorder fluctuates greatly over a relatively short time period. If this is acknowledged, it helps to destigmatise the diagnosis of personality disorder and also allows the diagnosis to be made in adolescence, so reassuring the practitioner that this is not necessarily a lifelong diagnosis that is going to stamp the sufferer indelibly.

The first stage in the diagnosis of personality disorder is testing whether the person's problems satisfy the general definition of personality disorder (Table 28.1). This is not dramatically different from either the DSM-IV or ICD-10 diagnoses of personality disorder; the difference exists in that as categorical diagnosis no longer exists the practitioner has to assess personality disorder in the round instead of being diverted into categorical cul-de-sacs. There is also no comment about the assessment of pathology of the self in contrast to its great emphasis in the DSM-5 proposals (Skodol et al., 2011), as the working group felt this was too difficult a concept to explore in a relatively short assessment and that the amount it added to diagnosis was small.

The second stage is the allocation of severity of personality disturbance. We identified a subthreshold level of disorder named personality difficulty but this is only allocated a Z-code in the classification; it refers to a disturbance in personality that may be only manifest intermittently at certain times or in particular environmental settings and in this respect is similar to what has been described as stress-induced personality disorder (Reich, 2002). Those with

Table 28.1 General definition of personality disorder (December 2012)

Essential features
A pervasive disturbance in the individual's enduring pattern of inner experience and behaviour manifest in at least two of the following areas: cognition (the way individuals think about themselves, others, and the world); emotional experience and expression; and patterns of behaviour
The disturbance produces significant problems in functioning that are particularly evident in interpersonal relationships
The disturbance must be manifest across a range of personal and social situations (i.e. they are not limited to specific relationships or 'triggering' stimuli or situations)
The disturbance is of long duration, having its onset in childhood or adolescence
Boundary with other disorders and normality:
The disturbance should not be due primarily to another mental disorder
The disturbance cannot be explained by social or cultural differences
The disturbance created in interpersonal relationships is not due to the physiological effects of a general medical condition or chronic substance use

Table 28.2 Definition of mild personality disorder (December 2012)

Essential features
The patient satisfies the requirements for the general definition of personality disorder
Problems with cognition, emotional experience, and expression are usually focused within one trait domain
These problems may involve some risk of harm to self or others but these are not major
The disruption created by problems in interpersonal relationships is mainly contained and does not spread to/involve occupational and other aspects of social function
Comorbid mental state pathology, if present, is not significantly influenced by the personality dysfunction

Table 28.3 Definition of moderate personality disorder (December 2012)

Essential features
The patient satisfies the requirements for the general definition of personality disorder
Problems in interpersonal relationships are marked, expected occupational and social roles are severely compromised, and most relationships are conflicted or absent
The personality dysfunction is likely to satisfy the criteria for more than one 'trait domain'
There is a clear risk of harm to self or others
Comorbid pathology in the form of other mental disorders is commonly associated as the personality pathology has a widespread influence on cognition, emotional experience, and expression

Table 28.4 Definition of severe personality disorder (December 2012)

Essential features
The patient satisfies the requirements for the general definition of personality disorder
There are severe problems in interpersonal functioning affecting all areas of life
The problems created by the severe personality disorder are such that they lead to a severe risk of significant harm to the self or others sufficient to cause long-term damage or endanger life
The individual's general social dysfunction is profound and the ability to perform expected occupational and social roles is absent or severely compromised
Comorbid mental state pathology in some form is very common

personality disorder have then to be allocated a severity level of mild, moderate, or severe using the features summarised (Tables 28.2, 28.3, and 28.4) (Crawford, Koldobsky, Mulder, & Tyrer, 2011; Tyrer, Crawford, & Mulder, 2011; Tyrer, Crawford, Mulder, et al., 2011). For each of these, the general requirements are monothetic: the individual is not just satisfying a set of

Table 28.5 Definition of detached domain (December 2012)

Domain status: The domain is a qualifier of the severity level of personality disorder and applies to mild, moderate, and severe personality disorder. It is intended to describe the features of a personality disorder, and is not a diagnosis
Core features: The central aspect of the detached domain is social indifference and impaired capacity to experience pleasure. Traits in the detached domain include aloofness, preference for solitary activities, unassertiveness, avoidance of interpersonal relationships (particularly close or intimate relationships), and reduced expression of emotions
Severity aspects: Individuals with moderate or severe personality disorder and marked detached traits are almost completely separated from other people, with very few or no attachment figures. They have limited awareness of the experience and motives of others, and so may misconstrue others' actions as threatening or hostile

Table 28.6 Definition of anankastic domain (December 2012)

Domain status: The domain is a qualifier of the severity level of personality disorder and applies to mild, moderate, and severe personality disorder. It is intended to describe the features of a personality disorder, and is not a diagnosis
Core features: The central aspect of the detached domain is concern over the control and regulation of behaviour. Traits in the anankastic domain include perfectionism, constraint, stubbornness, dutifulness, conscientiousness, deliberation, and order
Severity aspects: Individuals with moderate and severe personality disorder and marked anankastic traits manifest undue preoccupation with pursuing their perfectionistic ideal to the exclusion of pleasure and interpersonal relationships. Their rigidity if challenged may be met by aggressiveness

Table 28.7 Definition of dissocial domain (December 2012)

Domain status: The domain is a qualifier of the severity level of personality disorder and applies to mild, moderate, and severe personality disorder. It is intended to describe the features of a personality disorder, and is not a diagnosis
Core features: The central aspect of the dissocial domain is disregard for social obligations and conventions and the rights of others. Traits in the dissocial domain include callousness, lack of empathy, hostility and aggression, ruthlessness, and inability to maintain prosocial, goal-oriented behaviour
Severity aspects: Individuals with moderate and severe personality disorder and marked dissocial traits are combative and aggressive. They are dishonest, manipulative, and exploitative of others. They may hold themselves in excessively high regard and often are boastful and arrogant and expect admiration from others. They tend to be selfish and hedonistic, using others to meet their needs. They react to criticism with hostility and blame. What is generally called 'psychopathy' is an extreme manifestation of this trait domain and includes callousness, cruelty, and sadistic behaviour

criteria but the dysfunction created by his or her personalities classed by the extent to which it interferes with others and their mental states, its influence on trait domains (see below—with more severe personality disorders involving more than one), and the degree of risk to self and others. These definitions are still a 'work in process' but indicate the territory in which the clinician has to operate.

The level of severity is qualified by the description of domain traits. These indicate which of the main facets of personality are most prominent in the individual concerned. The final names of these domains are still under discussion but the four that are almost certainly going to be included are summarised (Tables 28.5, 28.6, 28.7, and 28.8). These are very similar to four of the Big Five traits of the NEO Personality Inventory (NEO-PI-R) (Costa & McCrae, 1992) and other studies looking at higher order traits in personality pathology (Widiger & Simonsen, 2005).

Again we are trying to use monothetic rather than polythetic criteria in encapsulating these.

Where Do Patients Currently Classified as Borderline Fit into the New Classification?

Firstly, it is possible for an individual to be diagnosed as having personality disorder at any

Table 28.8 Definition of distressed domain (December 2012)

Domain status: The domain is a qualifier of the severity level of personality disorder and applies to mild, moderate, and severe personality disorder. It is intended to describe the features of a personality disorder, and is not a diagnosis
Core features: The central aspect of the distressed domain is a persistent tendency to evaluate and respond negatively to the self, the world, and others. Traits in the distressed domain include sensitivity to scrutiny by others, self-consciousness, vigilance, fearfulness, pessimism, and emotional dysregulation
Severity aspects: Individuals with moderate and severe personality disorder and marked distressed traits may respond to even mildly stressful stimuli with dissociation and severe emotional distress, with vacillating mood states including anxiety, depression, and hostility, and they may engage in maladaptive behaviours to attempt to control their mood disturbance, including impulsive and self-destructive, behaviour

age. However, those who have demonstrated no personality problems in early life or early adult life are less likely to be diagnosed as having personality disorder than others, but it is not impossible. There is still argument in the ICD-11 about the usefulness of the diagnosis of personality change secondary to some other event, whether it is a traumatic one in terms of physical damage or a psychological one in terms of abuse or neglect. In coming to a diagnosis of personality disorder, there will be a tendency for many practitioners to think in terms of the old classification. This would be unfortunate. Officially, both ICD-10 and DSM-IV classifications have to identify that personality disorder is a general concept in the first instance. Unfortunately, because of the seductive attractions of labels such as 'borderline' many practitioners go straight to the category. They are going to have to get used to looking carefully at the severity of personality dysfunction before deciding which domains are most prominent.

Most people with a current diagnosis of borderline personality disorder are aware that they are part of a very heterogeneous group. It includes those people who do not seek any help and have been diagnosed, for example, as part of a national survey, a group of others who are seeking help because they can identify the features of borderline personality disorder but who are currently employed and have a series of reasonable relationships, another group who have a primary diagnosis of a mood disorder or post-traumatic stress disorder, and the final group that is very disturbed and likely to be in a forensic institution because of risk to themselves and others.

The trait domains that are likely to be associated with current borderline personality disorder are distressed and dissocial ones. There is a small degree of overlap with anankastic and detached personalities but these only have a small influence on the presentation of the condition. The advantage of the new classification is that the degree of risk to self and others is an important criterion determining severity, and so most people who are at high risk of self-harm will be classified as having severe personality disorder with the appropriate trait domains specified.

The big advantage of the ICD-11 classification system is that all patients with personality disorder basically have one diagnosis, with a small sub-classification indicating the trait domains, and so PD-NOS and mixed personality disorders have either no place in the classification or only a minor one.

Patients currently classified as having borderline personality disorder are a heterogeneous group who have variable response to treatment on different outcomes precisely because they are so heterogeneous. The new classification would allow them to be classified more consistently and, we hope, into homogeneous groups. Those currently classified as having borderline personality disorder are likely to fall into six separate groups: (a) mild personality disorder in the distressed trait domain, (b) mild personality disorder in the dissocial domain, (c) moderate personality disorder in the distressed and dissocial domain, (d) severe personality disorder in the distressed and dissocial domain, (e) moderate or severe personality difficulty with multiple domains, and (f) personality difficulty. In our preliminary field studies, we have found that most in-patients with borderline personality disorder qualify for moderate or severe personality disorders with two or three trait domains

involved. In outpatient and community mental health team settings there is a much greater proportion of those with mild personality disorder. We have not yet carried out any studies with adolescents but the basic structure of personality in this group fits the new system of classification reasonably well (Elliott, Tyrer, Horwood, & Fergusson, 2011).

Selection of Treatment for Those with Borderline Personality Disorder

One of the major problems in treating borderline personality disorder is the selection of patients for what is often a long and resource-intensive treatment. The simple diagnostic label of 'borderline personality disorder' is not of much value here. Epidemiological studies suggest that just under 1 % of the population has this diagnosis but clearly it would be quite inappropriate to consider most of these for an intensive psychological treatment. The advantage of the new diagnostic system is that guidelines for treatment of borderline personality disorder (e.g. NCCMY, 2009) would be able to specifically address severity of personality disorder in making their recommendations. My personal view is that it would be inappropriate to consider one of the complex psychological interventions for borderline personality disorder such as NBT, CBT, TFT, or STEPPS for anything other than moderate and severe personality disorders in which both distressed and dissocial domains are affected. This is not to say that treatments for mild personality disorder would be excluded but they would have to be different and probably need to be developed specifically for this condition.

Advantages of New Classification System in Assessing Adolescents with Borderline Symptomatology

There continues to be great controversy over the question of diagnosing personality disorder in adolescence. This is because the diagnosis is a pejorative one and implies permanence. The issue of permanence is a myth—no study shows personality disorder to persist without modification over time. Many with adolescent personality disorders will improve, but the presence of personality dysfunction is a major prognostic factor in adult mental pathology that cannot be ignored (Crawford et al., 2008). The ICD-11 classification offers help here by its grading of severity. People may be diagnosed as having moderate personality disorder at age 15 but by the age of 17 it may have changed to mild severity, and this ability to grade progress will help clinicians, patients, and families to understand both the ups and downs that may alter personality function and also response to interventions of all sorts.

It is a classification that is worth taking seriously.

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Some Overview Comments with an Eye to the Future

29

John G. Gunderson

Introduction

The many efforts to understand borderline personality disorder's (BPD) development in childhood and adolescence evidenced in this book testify to the ongoing interest in this subject, the wide range of approaches, the many obstacles that researchers face, and to the major questions that remain. It is beyond my ability to provide a synthesis of this diverse and adventuresome literature. What I will do is explore three topics relevant to the future development of this field, i.e., the implications of BPD's heritability, the identification of risk markers, and the potential to customize home environments that might derail BPD's onset. Even within this more focused discussion, I will not have completed the literature review required to claim anything close to comprehensiveness; rather, my comments will have rested heavily on those parts of the literature which have remained memorable to me. These limitations notwithstanding this chapter will develop the thesis that the time has come to conduct a large scale prospective study of children at risk for developing BPD that tests potentially preventative interventions.

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Genetics

In 2001 Sven Torgersen reported that BPD had an estimated heritability of 68 % (Torgersen et al., 2000). This report reversed his earlier report using a more modest sample that comorbid BPD was not heritable (Torgersen, 1984) and, in the process, irreversibly and dramatically changed considerations of BPD's etiology. The significance of BPD being significantly heritable (most estimates now place the level at about 55 % (Gunderson et al., 2011)) is still being only grudgingly appreciated. Until 2001, virtually all theories and research about the origins of BPD had stressed environmental factors, most notably, failed parenting, dysfunctional families, and childhood trauma.

An unrecognized, but critically important, failure in the earlier literature was its disregard for a pre-borderline child's genetically determined role in evoking those environmental adversities. The importance of a child's innate disposition on shaping his or her environment, i.e., specific genetic factors can predict certain types of environment (Reiss et al., 1995), had already been appreciated by geneticists and developmental psychologists, but it had not been appreciated within BPD's clinical or research literature. Within this perspective, the genetic disposition to BPD increases the likelihood of exposure to stressful life events (Distel et al., 2011) and increases the likelihood of stressors

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having traumatic effect (Orr et al., 2012; Verschoor & Markus, 2011).

Judging from the more general failure of the genome to have yielded significant insights into the origins of other more circumscribed and more heritable psychiatric disorders such as schizophrenia or bipolar disorder, it seems unlikely that BPD will yield clinically meaningful genetic answers in the foreseeable future. This conclusion is fortified by the complexity of BPD psychopathology which includes mood, behavior, cognitive/perceptual, and, most specifically, interpersonal components. While this breath and diversity of psychopathology have suggested that BPD represents the co-aggregation of four separable phenotypes, each with its own potentially discernible and polygenetic disposition, a series of studies have now consistently shown that BPD has a unitary unifying underlying structure (Gunderson et al., 2011). This, to me, somewhat surprising finding has strongly validated BPD's diagnostic integrity and provided the strongest evidence against radically changing its definition and for moving BPD to Axis I (Gunderson, 2013). Hence, despite my pessimism about clinical relevance, this finding has stimulated hopes that BPD's spectrum of genetic determinants will become identifiable as having a specifiable co-aggregation of genes.

The search for environmental determinants in BPD's development is handicapped by not knowing what its heritable disposition consists of. There are at present two viable candidates for what the genetic disposition will be. The first of these is *excessive emotionality* (Klein, 1977; Linehan, 1993) and the second is *interpersonal hypersensitivity* (Gunderson, 2007; Gunderson & Lyons-Ruth, 2008). *Excessive emotionality* is a highly prevalent positive symptom of BPD, has relatively strong stability compared to other symptoms, and is the central target of BPD's best validated treatment, i.e., Dialectical Behavior Therapy (DBT) (Linehan, 1993). DBT postulates that emotional dysregulation is the cause of the borderline patients' behavioral and interpersonal problems. *Interpersonal hypersensitivity* is the most clinically and phenomenologically discriminating symptom of BPD, also has good stability (especially intolerance of aloneness), and is central to treatments for BPD such as Transference Focused Psychotherapy (TFP) (Clarkin, Yeomans, & Kernberg, 2006), Mentalization-Based Treatment (MBT) (Bateman & Fonagy, 2004), and Good Psychiatric Management (GPM) (Gunderson & Links, in press). In this model, the borderline patients' emotional and behavioral symptoms are secondary to being excessively sensitive to perceived interpersonal slights.

Evidence-based therapies give token appreciation to BPD's heritable disposition (e.g., excessive aggression or emotional reactivity), but they have quite understandably emphasized psychosocial causes. This emphasis allows their therapeutic effectiveness to be understood as due to the corrective potential of social, cognitive, and interpersonal learning experiences. The success of those interventions has justifiably sustained the interest in understanding the psychosocial factors in BPD's development that this book documents. This contrasts with the almost exclusive attention now being given to genetic and neurobiological causes for those psychiatric disorders with even modestly effective psychopharmacological treatments. Therapies for BPD lacked, and still lack, any consistent or potent pharmacological agent. The unfortunate consequence of this is the continued absence of money for BPD-related research. The fortunate consequence is that BPD has sustained the development of psychosocial therapies and encouraged the interest in psychosocial causes for its development.

Identification of Risk Markers

Regardless of whether the latent genetic disposition for BPD is found to be *interpersonal hypersensitivity* or *excessive emotionality*, research into BPD's development needs to assess genetics, neurobiology, social adaptation, and phenomenology in children to identify markers of risk for adult BPD.

With respect to genetics, BPD occurs in about 2–3 % of the population, but in 11.5 % of the

first-degree relatives (FDRs) of BPD probands (White, Gunderson, Zanarini, & Hudson, 2003). Having a positive family history for BPD increases the likelihood of BPD in FDRs about fivefold, thereby constituting a significant marker of risk. Notably, when a positive family history is used to identify children at risk for schizophrenia, a disorder whose heritability is about 85 % and whose population prevalence is 1 %, having the disorder only doubles the risk in FDRs to about 2 %. The point here is that any search for children at risk for developing BPD should begin by finding children with a positive family history. Since children share exactly 50 % of their genes with parents, and 25 % with siblings, examination of children with BPD parents or from high density BPD family pedigrees provides the most clearly identifiable and significant risk for this disorder's development.

What follows is a proposal for what seems to me to be a logical progression of research to further identify risk markers for BPD:

1. *Retrospective reports by BPD patients*—Such reports have identified frequent parental separation/loss/illnesses (most especially fathers), transitional object dependency, and childhood histories of abuse, neglect, dysfunctional families, and parentification (Gunderson & Zanarini, 1989). They have also documented excessive angry conflicts, hostility, and poor communication within the families (Gunderson & Lyoo, 1997). As a footnote to preparing this chapter, I invited a group of borderline patients to describe what they think was the first indications that they would develop their disorder; they readily identified (a) severe separation anxiety, (b) always being angry, (c) homesickness, (d) self-consciousness, and (e) having narcissistic parents. These, I agreed, represent a pretty good set of risk markers.
2. *Retrospective reports by parents with BPD offspring*—Such reports generally confirmed the excess of angry alienation and early parental separations/losses and illnesses (Gunderson & Lyoo, 1997). (A notable study by Goodman et al. (2010) compared parental recall of the BPD offspring's childhood with that of their non-BPD siblings. They identified emotionality, hypersensitivity, and inability to self-soothe in infants, and academic difficulties, lack of friends, and the search for exclusive partnerships in grade school settings.)
3. *Contrast children at high risk with those with low risk*—Children at known risk for BPD include (a) those with BPD parents, (b) those from adverse home environments (e.g., prolonged absences; or with legal, childcare, or marital problems), (c) those with disorganized attachment (Lyons-Ruth, Choi-Kain, Pechtel, Bertha, & Gunderson, 2011), and (d) those with both internalizing and externalizing problems (Brezo et al., 2007). There have been relatively few studies of this sort. Assessments of such children should specifically seek out early signs of the two candidate phenotypes, i.e., *disturbed emotionality* or *interpersonal hypersensitivity*. Insofar as the disposition for BPD involves sensitivity and reactivity to environmental context, the phenomenological markers of risk are likely to be highly unstable. This suggests that risk markers for BPD may be less evident in a child's stable traits (e.g., anxiousness, aggressivity, attachment style) than in his or her responsivity to environmental stress (e.g., panic attacks, temper tantrums, separation distress).
4. *Experimental examinations of children at risk*—The inherent instability and reactivity of BPD encourage use of laboratory/experimental tests of response to situational change or to designed stressors. If, in addition, the pre-borderline is specifically more sensitive and reactive to interpersonal events that evoke the perceptions of rejection or abandonment, the experimental prompt should be designed to trigger those reactions (see, for example, Donegan et al., 2003). Within a naturalistic sampling, the significant fraction of children with transitional objects will include a subgroup who are particularly distressed by separation from them. They might be

expected to be at increased risk for adult BPD (Morris, Gunderson, & Zanarini, 1986). Strange situation (failed attachments) (Main & Solomon, 1990), still-face testing (Crandell, Patrick, & Hobson, 2003), and physiological measures of stress responsivity are examples (Herpertz et al., 2001; King-Casas et al., 2008). The naturalistic stress of transition from grade school to junior high school may provide an opportunity to observe the pre-borderline child's problems with stress responsivity that exceed both earlier and later ages (Livson & Peskin, 1967).

5. *Prospective follow along studies of children at risk*—As frequently noted in this book, there have already been landmark studies of this sort (e.g., Carlson, Egeland, & Sroufe, 2009; Cohen, Crawford, Johnson, & Kasen, 2005; Lyons-Ruth, Melnick, Patrick, & Hobson, 2007). The study that needs now to be done is one that starts earlier in childhood and that examines for the known or hypothesized risk markers. Such a study should include repeat measures of stress response, separation reactions, and emotionality. Two examples of previously researched childhood phenomenas that might characterize pre-borderline children—though this possibility was not considered—are temper tantrums (Caspi & Bem, 1990) and fears of death (Rose & Ditto, 1983). If the search for risk markers is conducted with infants, caretaker responses that exacerbate or diminish distressed responses are likely to be most fruitful. If the markers are identified in children, family systems and other social environmental phenomenon (e.g., school function, peer relationships, culture) can be studied to find their association with and consequences on a child's development.

When markers from different domains are assembled, their individual and aggregated strength as predictors of subsequent markers and, most significantly, of the onset of BPD can be calculated. A notable precedent is the finding that 25 % of children with conduct disorder go on to adult ASPD (Robins, 1966). For schizophrenia, the highly significant Treatment and Intervention in Psychosis (TIPS) (Hegelstad et al., 2012) study identified children as candidates by virtue of (a) a decline in school/social functioning score of more than 30 % during childhood/adolescence, (b) attenuated symptoms of paranoia, hallucinations, and delusions, and (c) a relative with schizophrenia (which, as noted, only increases the risk to 2 %). Children/adolescents with these markers had a 20–40 % likelihood of developing a psychosis. It was only later, in early adolescence, that these children developed the more schizophrenia-like prodrome, i.e., symptoms of social isolation and cognitive impairment (McGlashan, personal communication 6/15/13). It seems likely that we already have enough known or hypothesized early risk markers for BPD (e.g., an affected relative, prolonged parental loss/separation or illness, disorganized attachment, and excessive emotionality) that we could achieve an equivalent rate of prediction for adult BPD.

Designing Preventive (Customized) Home Environments

When children at risk for psychoses were identified, some received interventions of antipsychotic medication, casework, and psychoeducational multifamily group therapy. Those who received this were less likely to become psychotic for shorter times and with better functional outcomes (Hegelstad et al., 2012). When children at high risk for developing BPD became recognizable, what then could we propose as a preventive intervention?

To my mind the intervention should clearly be directed at the child's home environment. This is the most potent of the environmental influences on children and it will account for far more of the causal variance for BPD than was the case for the more genetically determinant disorder of schizophrenia. Moreover, we already know most of what an "anti-borderlinogenic" household would look like. Here the Guidelines for Families (Gunderson & Berkowitz, 2006) offer a model of

concerned responsiveness, foregoing angry reactivity, and establishing consistent limits—to which both parents adhere. There is little new or secretive within this formula for an "anti-borderlinogenic" family. It has become an important formula for professionals to underscore and remind parents who have borderline offspring—and I would propose the same formula is suitable for the family environment of the pre-borderline children. Such children will often have made it hard for parents to retain the calm consistent and generally reassuring environment that is particularly needed by such "special needs" children.

I think there may also be some other more BPD-specific guidelines for parenting of children at risk for developing BPD. One of these may be teaching parents how to help such children identify their feelings. Another might be letting children know that they can and do effect you; that is, from the response "I'm sorry you feel bad" to the more difficult "when you say that, it hurts my feelings." It may also be especially important that parents of pre-borderline children shelter them from parental disagreements about childcare (parents would be coached to resolve those before responding) or towards each other (implicitly inviting the child to take sides).

The more difficult question is whether parents of children at risk for BPD will be worried enough about their child and about the role of their childcare practices to accept the proposed coaching. Perry (1990) reported that parents with BPD offspring feel especially burdened because they are devalued. Such highly negative and critical perceptions of families by BPD offspring were found to contrast with the judgments by their parents who considered their families normal (Gunderson & Lyoo, 1997). While that finding could suggest such parents would be unreceptive to family interventions, I think that conclusion is premature. When parents receive psychoeducation about BPD's heritability and about its natural course and treatability, their receptivity to advice about modifying their parental habits usually, in my experience, is likely to be greatly improved.

We know that parents of BPD offspring feel very burdened and that this burden becomes more severe in adolescence when destructive acting out behaviors escalate (Goodman et al., 2010). By the time the children are diagnosed with BPD, parents report negative impacts on their emotional health (89 %), physical health (58 %), and marriage (56 %). We know that the burdens associated with self-destructive/suicidal behaviors and financial expectations subsequently escalate. Unfortunately, we have not yet prospectively assessed how burdened parents feel by their pre-BPD-diagnosed children.

At this point, we do not know whether parents of children who are identified as being at risk for BPD would respond to an opportunity to undertake a family-based intervention. Would they react defensively, i.e., "we're doing nothing wrong," or would they, after a good psychoeducation, respond with "we knew we have a difficult child. It's reassuring to now know we might be able to reduce his likelihood of becoming psychiatrically ill." If identification of the child at risk includes having a BPD relative (including one of the parents) this would likely increase receptivity. Beyond that, receptivity can be expected to correlate with how burdened the parents feel by their pre-BPD offspring.

An advantage of proposing a preventative intervention to parents whose child is at risk for BPD is that the intervention would not include medications with their known dangers of side-effects and their potential to adversely affect neurobiological systems. Moreover, by making the intervention within the child's family, the risk of the child becoming stigmatized is reduced.

Conclusions

Interest in the development of BPD has existed for a long time. While early studies were guided by the psychoanalytic reconstructions that gave emphasis to adverse parental responses in the separation-individualists phase (16–26 months) that were alleged to cause the pre-borderline child's aggression to be split off and/or that discouraged the child's development of independence/autonomy, the current body of research is much more stimulated by concepts of emotional dysregulation, failed attachment, and the search for a pre-borderline temperament.

Within this now considerable body of research, there are now many indicators of risk for BPD. Future researchers should know and learn from the literature identified throughout this book. As described, the identification of risk markers began with retrospective accounts by patients and families and has been followed by direct observations of children and adolescents who are at known risk. It is worth underscoring that the available descriptions of such children and their environments involve very different types of phenomena. Indeed the study of BPD's development is ideally suited for interdisciplinary studies starting with direct observation, but involving genetics, developmental psychology, and sociology. Research that relies solely on one discipline will prove far less likely to advance the field, in my opinion, than one that combines observations derived from multiple and diverse perspectives. I would add to this that future research into BPD's development should now primarily depend upon assessments of phenomena known to be BPD-relevant (i.e., should be hypothesis-driven), and not depend upon broad scale standardized assessments (i.e., should not be "fishing expeditions").

The implementation of early preventative interventions for families whose risk for BPD offspring is high is an exciting, albeit still futuristic, possibility. It is an extension of the thesis developed by Reiss, Neiderhiser, Hetherington, and Plomin (2000) by which personality and behavioral styles observed in children can yield formulaic prescriptions about what types of family environment will best enhance a child's innate potential for success and least encourage his or her dispositions towards disorder and dysfunction. The BPD field can learn greatly from the progress that has been achieved with children at risk for schizophrenia.

The clearest implication of this book and of this chapter is that the stage is near where a large scale probably multi-site prospective study can be successfully implemented to identify and follow children at risk and implement potentially preventative—and fortunately benign—interventions. I suspect that within the community of clinicians and scientists who have authored this book's chapters, the energy, creativity, and research talent will be found to undertake such a study. Good luck!

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