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

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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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C. Sharp and J.L. Tackett (eds.), *Handbook of Borderline Personality Disorder in Children and Adolescents*,
DOI 10.1007/978-1-4939-0591-1_2,

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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