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

15

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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

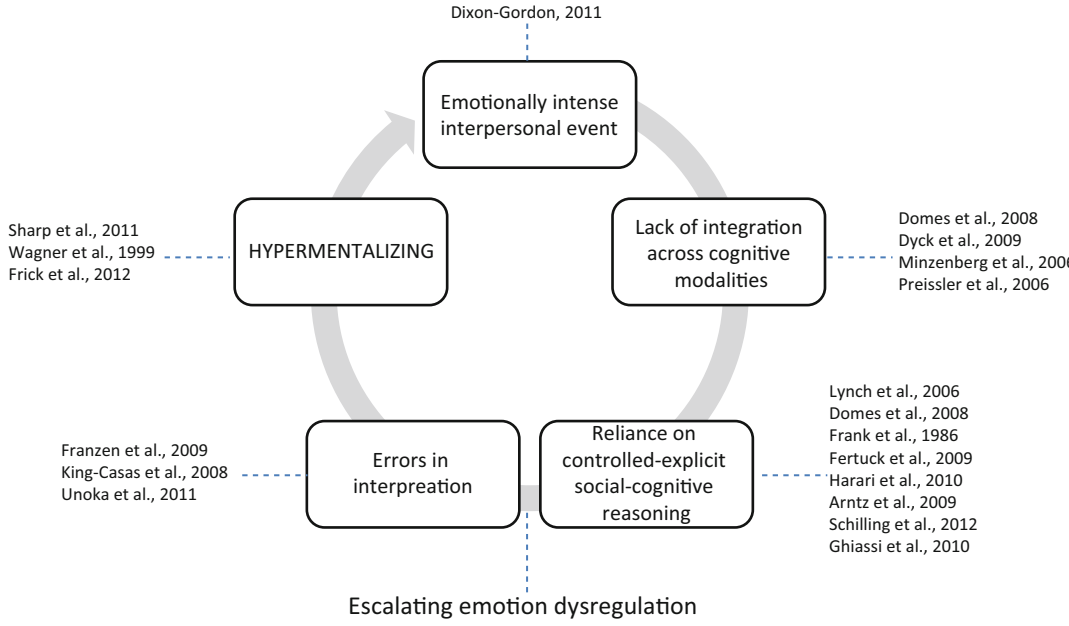

Fig. 15.1 The hypermentalizing theory of BPD

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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