A Biosocial Developmental Model of Borderline Personality: Elaborating and Extending Linehan's Theory

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Over the past several decades, research has focused increasingly on developmental precursors to psychological disorders that were previously assumed to emerge only in adulthood. This change in focus follows from the recognition that complex transactions between biological vulnerabilities and psychosocial risk factors shape emotional and behavioral development beginning at conception. To date, however, empirical research on the development of borderline personality is extremely limited. Indeed, in the decade since M. M. Linehan initially proposed a biosocial model of the development of borderline personality disorder, there have been few attempts to test the model among at-risk youth. In this review, diverse literatures are reviewed that can inform understanding of the ontogenesis of borderline pathology, and testable hypotheses are proposed to guide future research with at-risk children and adolescents. One probable pathway is identified that leads to borderline personality disorder; it begins with early vulnerability, expressed initially as impulsivity and followed by heightened emotional sensitivity. These vulnerabilities are potentiated across development by environmental risk factors that give rise to more extreme emotional, behavioral, and cognitive dysregulation.

Keywords: borderline personality disorder (BPD), developmental psychopathology, child, adolescent, self-inflicted injury

Borderline personality disorder (BPD), which is characterized by persistent and pervasive cognitive, emotional, and behavioral dysregulation, is among the most severe and perplexing behavioral disorders. BPD is a heterogeneous phenotype and results from a polythetic criterion set of which only five of nine behavioral features are required for a diagnosis. Thus, two individuals receiving a diagnosis of BPD could potentially overlap on only one diagnostic criterion. The BPD phenotype is broadly defined by features of emotion dysregulation, impulsivity, identity disturbance, problematic interpersonal relationships, and suicidal/self-injurious behaviors, among others (American Psychiatric Association, 2000). Currently, the developmental trajectory or trajectories that lead to BPD in adulthood remain unclear. Despite steady progress toward outlining the etiology of BPD (see Lenzenweger & Cicchetti, 2005), longitudinal research is still needed to identify biological vulnerabilities, environmental risk and protective factors, and patterns of homotypic and/or heterotypic continuity that lead to the diagnosis.

When compared with research on other psychological disorders, such as depression and antisocial personality disorder (ASPD), research on the development of BPD has been strikingly sparse. This is troubling, given the high rates of morbidity and mortality associated with the disorder. Data suggest that BPD affects from 1.2% to almost 6% of the general population, approximately 10% of those who seek outpatient services, and as many as 20% of those who undergo inpatient treatment (Grant et al., 2008; Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). Moreover, up to 10% of those who meet criteria for BPD eventually commit suicide, this rate is 50 times that observed in the broader population (American Psychiatric Association, 2001). Thus, BPD is associated with tremendous emotional and financial burdens to individuals, families, and society. In light of these costs, identifying precursors to the disorder must be a priority.

According to current definitions, personality disorders are characterized by persistent patterns of behavior that are rigid and pervasive and that emerge in late adolescence or early adulthood (American Psychiatric Association, 2000). However, in spite of increasing evidence to suggest that BPD can be identified reliably among adolescents (Miller, Muehlenkamp, & Jacobson, 2008), the clinical diagnosis of BPD is rarely given before the age of 18, and there is a dearth of empirical longitudinal research with these youth. In accordance with a developmental psychopathology perspective, we assume that there are etiological precursors to BPD (e.g., Crowell et al., 2005) and that identifying these precursors will lead to more effective prevention and treatment (see Beauchaine & Marsh, 2006; Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). Therefore, in this article, we elaborate on

probable mechanisms underlying the development of BPD. We also expand upon Linehan's (1993) theory by examining the etiology of impulsivity, independent of emotion dysregulation, in the developmental trajectory leading to BPD. This elaboration draws from current research on biological vulnerabilities and psychological risk factors to bring a developmental psychopathology approach to Linehan's biosocial theory.

Linehan's Biosocial Theory

Linehan's biosocial theory of BPD (1993) is among the most thoroughly delineated etiological models of borderline pathology (for other models, see Fonagy, Target, & Gergely, 2000; Judd & McGlashan, 2003; Kernberg, 1967, 1975, 1976). According to Linehan, BPD is primarily a disorder of emotion dysregulation and emerges from transactions between individuals with biological vulnerabilities and specific environmental influences. The dysfunction proposed by Linehan is one of broad dysregulation across all aspects of emotional responding. As a consequence, individuals with BPD have (a) heightened emotional sensitivity, (b) inability to regulate intense emotional responses, and (c) slow return to emotional baseline. Furthermore, from Linehan's perspective, the construct of emotion (and thus of emotion dyregulation) is very broad and includes emotion-linked cognitive process, biochemistry and physiology, facial and muscle reactions, action urges, and emotion-linked actions. Emotion dysregulation subsequently leads to dysfunctional response patterns during emotionally challenging events. Linehan suggested a number of possible biological substrates of emotional dysregulation (e.g., limbic dysfunction). However, the literature on the biology of psychological disorders was extremely limited when Linehan first articulated her theory.

In addition, Linehan proposed that the development of BPD occurs within an invalidating developmental context. This invalidating environment is characterized by intolerance toward the expression of private emotional experiences, in particular emotions that are not supported by observable events. Furthermore, although invalidating environments intermittently reinforce extreme expressions of emotion, they simultaneously communicate to the child that such emotional displays are unwarranted and that emotions should be coped with internally and without parental support. Consequently, the child does not learn how to understand, label, regulate, or tolerate emotional responses and instead learns to oscillate between emotional inhibition and extreme emotional lability. The child also fails to learn how to solve the problems contributing to these emotional reactions.

An Elaboration and Extension of Linehan's Theory

Both the basic and the applied literatures have advanced substantially over the last several decades and now provide emerging insights into the biological correlates and substrates of BPD. Similarly, research on family interaction patterns contributing to the development of psychological dysfunction—including emotion dysregulation—has expanded and become more specific (e.g., Granic & Patterson, 2006). Thus, it is now possible to explore Linehan's biosocial theory in detail and provide a more current and comprehensive outline of the etiology of BPD. In addition to examining etiological mechanisms in more detail, we extend Linehan's (1993) theory. In particular, Linehan focused almost exclu-

sively on early emotional development and placed no focus on trait impulsivity independent of emotion. It is important to note here that Linehan's theory was developed to guide treatment strategies and therefore concentrated primarily on links between emotional experience and behavior. Accordingly, therapeutic strategies modifying any component of emotional experience were found to change functioning across all associated feeling states and behaviors. This finding provided evidence in support of a unitary system governing both emotion and impulsive, mood-dependent behavior.

In contrast, in this review we explore the possibility that the action component of emotion may also be influenced by trait impulsivity, independent of emotion. We assert specifically that early impulsivity is a predisposing vulnerability for a substantial subset of those who eventually meet criteria for BPD. This subset consists of those who continue as adults to manifest impulsive behaviors, including suicidal and nonsuicidal, self-injurious behaviors. This impulsive subtype not only captures some of the most severely affected individuals with BPD, but also encompasses the majority of those who eventually meet criteria for the disorder. Indeed, evidence suggests that as many as 40%–90% of individuals with BPD either engage in nonsuicidal self-injury or make a suicide attempt (American Psychiatric Association, 2004). Therefore, we propose that impulsivity is among the earliest emerging traits among those who later receive a BPD diagnosis, a position that Linehan did not consider in her original biosocial theory. We also present data to support that early impulsivity is a predisposing vulnerability for both current and future difficulties with emotion regulation.

This extension of Linehan's theory is likely the product of conceptualizing BPD from a life span developmental perspective. Whereas impulsivity and emotion dysregulation are almost invariably linked by the time borderline pathology is canalized, impulsivity and emotional dysregulation may emerge independently and sequentially during development and thus contribute to different aspects of functioning. Moreover, a review of etiological mechanisms further reveals that many biological correlates of BPD are similar to those observed across impulse control disorders. Therefore, our extension is also informed by possible etiological overlap between borderline pathology and attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD), substance use, and antisocial pathology. Although BPD is distinct from many (though not all) impulse control disorders in the degree to which emotional lability predominates the clinical presentation, exploration of the extensive literature on the development of impulse control disorders allows for a richer understanding of possible pathways to BPD.

Developmental Psychopathology

Our elaboration of Linehan's biosocial theory, described below, should be considered within the broad perspective of developmental psychopathology. Within this framework, BPD can be viewed as an outcome of multiple interacting risk factors, causal events, and dynamic processes (e.g., Cicchetti, 1984). The developmental psychopathology perspective places emphasis on the mechanistic processes that underlie psychopathology at several levels of analysis, including genetic, neural, behavioral, familial, and social (Cicchetti, 2008). This approach also examines how and why psychiatric conditions emerge and evolve over time and how

psychopathology is influenced by the interaction between individuals and contexts across development (Cummings, Davies, & Campbell, 2000). This framework is particularly well suited for understanding the emergence of problems during adolescence and the continuity of these problems throughout adult development (Cicchetti & Rogosch, 2002). Therefore, consistent with a developmental psychopathology approach, we describe how reciprocal transactions between predisposing biological vulnerabilities and environmental risk factors shape the development of BPD among vulnerable individuals. For example, impulsive and emotionally sensitive children who are placed in high-risk environments may experience considerable difficulty inhibiting extreme emotions in the face of invalidation by family members, inconsistent use of punishment, and escalation of anger during interactions.

The developmental psychopathology approach is also well suited for examining the emergence of emotion dysregulation among impulsive youth. There is a substantial literature to suggest that extreme impulsivity expressed early in life may represent the first stage on a trajectory leading to later difficulties with both behavioral and emotional dysregulation (for a review, see Beauchaine & Neuhaus, 2008). Evidence also suggests that temperamental disinhibition increases risk for disorders across both the internalizing and the externalizing spectra (e.g., Hirshfeld-Becker et al., 2002; Patterson, Degarmo, & Knutson, 2000) and may therefore predispose individuals to a wide range of adverse, multifinal outcomes. Our model extrapolates from this literature and suggests that the development of emotional lability seen in BPD is similar to that seen in other impulse control disorders. In approaching this literature, we have taken a largely behavioral definition of impulsivity, as we subscribe to the notion that impulsivity consists of "behavior that is socially inappropriate or maladaptive and is quickly emitted without forethought" (Oas, 1984, p. 142, 1985, p. 142). Accordingly, we do not assume causal mechanisms in the etiology of disinhibited behavior, and both psychological and biological factors are presumed to contribute to the development of BPD. Prior to elaborating on our developmental model,

we briefly explore terminological and conceptual issues that contribute to current understanding of BPD.

Terminological and Conceptual Issues

At the time the diagnostic criteria for BPD were adopted, several ongoing controversies were being expressed in the literature (Gunderson & Singer, 1975; Spitzer, Endicott, & Gibbon, 1979). These included whether BPD (a) represented a stable set of personality traits or a transient psychotic state, (b) was truly distinguishable from schizophrenia, (c) represented a unitary concept or rather two or more subtypes, and (d) was an appropriate diagnostic label. Early diagnostic criteria were remarkably similar to those used in the current version of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2000). Not surprisingly, controversies over the borderline label and the notion of BPD as a unitary diagnostic entity remain. Nevertheless, it is now clear that the term *borderline*—which was coined to indicate pathology on the borderline between neurosis and psychosis—is incorrect, imprecise, and dated. These sentiments were present prior to publication of the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1980), when Spitzer et al. (1979) suggested a more appropriate label of "unstable personality disorder." A similar label was ultimately adopted in the *ICD-10 Classification of Mental and Behavioural Disorders* (World Health Organization, 1992), which identifies BPD as "emotionally unstable personality disorder, borderline type." Current diagnostic criteria for BPD are listed in Table 1.

The borderline label is at least as inappropriate for pediatric populations as it is for adults. When the label is applied to children and adolescents, common adaptations include "borderline pathology," "borderline features," or "borderline traits." In part, these terms were coined to capture the belief that personality is in flux during development (see Crick, Murray-Close, & Woods, 2005). However, these constructs are sometimes used loosely, have unknown predictive validity (e.g., Guzder, Paris, Zelkowitz, &

Table 1
Diagnostic Criteria for Borderline Personality Disorder

Diagnostic criteria for 301.83 Borderline Personality Disorder
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:
(1) Frantic efforts to avoid real or imagined abandonment
(2) A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation
(3) Identity disturbance markedly and persistently unstable self-image or sense of self
(4) Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)
(5) Recurrent suicidal behavior, gestures, or threats, or self-mutilating 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)
(7) Chronic feelings of emptiness
(8) Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights)
(9) Transient, stress-related paranoid ideation or severe dissociative symptoms

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Marchessault, 1996), and still maintain the problematic "border-line" label. Another alternative label is "multiple complex developmental disorder" (MCDD; Cohen, Paul, & Volkmar, 1987). As with youth with borderline pathology, those diagnosed with MCDD are described as having dysregulated affect, intense anxiety, poor social skills, interpersonal deficits, and episodic thought disorder (Towbin, Dykens, Pearson, & Cohen, 1993). The labels of "childhood schizophrenia" and "borderline syndrome of childhood" (Petti & Vela, 1990) have also been used to capture early emerging borderline traits. Given evidence suggesting that most children and young adolescents with borderline-like features do not go on to develop either schizophrenia or BPD (Lofgren, Bemporad, King, Lindem, & O'Driscoll, 1991), the MCDD diagnostic label may be most appropriate for this population. However, it is unlikely to be appropriate for youth who go on to develop BPD in adulthood, a population that has not yet been satisfactorily identified (Paris, 2005). In part, it has been a challenge to identify this population due to a lack of research with youth at risk for BPD, and to a lack of measures for assessing such risk. However, the recent development of measures designed to reliably assess developmental precursors to BPD represents a promising area for future research (for a borderline personality features scale and a review of other measures, see Crick et al.). In this review, we use the term *borderline pathology* to describe the presence of borderline-like traits among those who do not yet meet full criteria for BPD. However, this terminology will likely change when future diagnostic manuals dispense with the "borderline" label for adults.

A Biosocial Model of BPD

The biosocial developmental model presented here is based on theoretical and empirical evidence reviewed below, which suggests the following:

1. Poor impulse control likely emerges early in the development of borderline pathology, and this may account for the overlapping biological vulnerabilities for BPD and other impulse control disorders.

2. The development of extreme emotional lability characteristic of BPD is shaped and maintained by the caregiving environment and is based on characteristics of the child (e.g., baseline emotional sensitivity) and the developmental context.

3. Reciprocal reinforcing transactions between biological vulnerabilities and environmental risk processes potentiate emotion dysregulation and more extreme behavioral dyscontrol and thereby contribute to negative cognitive and social outcomes.

4. By mid- to late adolescence there is a constellation of identifiable features and maladaptive coping strategies that indicate heightened risk for later BPD.

5. These traits and behaviors may exacerbate risk for BPD across development, due to evocative effects on interpersonal relationships and social functioning and via interference with healthy emotional development.

Below, each of these points is addressed in turn, following an overview of current research on biological vulnerabilities and psychosocial risk factors that have been theoretically or empirically linked with BPD, the development of BPD, or related disorders. Understanding possible biological and psychosocial precursors to the diagnosis is critical, as childhood manifestations of BPD risk are unlikely to take the same form as the adult diagnosis. This prohibits a simple downward extension of adult diagnostic criteria. The data informing this biopsychosocial formulation are drawn largely from adults and older adolescents with BPD. We subsequently integrate these findings with the developmental literature and propose a theoretical and testable biosocial developmental model.

Biological Correlates of BPD

Research on biological dysfunction in BPD has centered on structural, neurochemical, and genetic vulnerabilities. Several neurotransmitter systems, including serotonin, dopamine, vasopressin, acetylcholine, noradrenaline, and gamma-aminobutyric acid, have recently received attention in the empirical literature. Researchers have also explored relations between borderline pathology and dysfunction of the peripheral nervous system. Finally, evidence suggests that BPD has a heritable component (e.g., Torgersen, 2000; Torgersen et al., 2008). This is consistent with research indicating that impulsivity, such as that seen in BPD, is roughly 80% heritable, has clear neuroanatomical correlates, and predisposes to a number of psychiatric conditions (see Beauchaine & Neuhaus, 2008). However, as relations between genes and complex behaviors are rarely straightforward, an integrated and dynamic understanding of biological and psychosocial contributions to development is required (see also, Beauchaine, Klein, Crowell, Derbidge, & Gatzke-Kopp, in press).

Behavioral genetics and family studies. Behavioral genetics research on the heritability of BPD has produced contradictory results, due in part to methodological differences between studies, but also to the heterogeneity of the BPD phenotype (Skodol et al., 2002). One of the largest and most methodologically sound twin studies to date (92 monozygotic and 129 dizygotic Norwegian twin pairs) produced a best fitting model in which 69% of the variance in symptoms was attributable to additive genetic effects and 31% of the variance was attributed to nonshared environmental effects, with no shared environmental effects (Torgersen et al., 2000). In this study, the concordance rate for BPD was 38% among monozygotic twins and 11% among dizygotic twins, which suggests a strong genetic component. In a separate family history study, Silverman et al. (1991) found that risk for affective instability and impulsivity was greater in the relatives of individuals diagnosed with BPD than in relatives of individuals with other personality disorders or schizophrenia.

Serotonin. Evidence suggests that both impulsive aggression and affective instability are related to specific genetic polymorphisms and functional impairments within the central serotonin (5-HT) system. This follows from overwhelming evidence suggesting that deficits in 5-HT functioning are associated with BPD-related conditions and behaviors, such as mood disorders, suicidal and nonsuicidal self-injury, and aggression (e.g., Kamali, Oquendo, & Mann, 2002). Several direct tests of 5-HT functioning in individuals with BPD support an association between the

disorder and deficits of the 5-HT system. Pharmacologic challenge tests designed to assess central 5-HT activity (e.g., fenfluramine, *m*-chlorophenylpiperazine, buspirone, ipsapirone) have been conducted in several samples with personality disorders, including individuals with BPD, with findings suggestive of reduced central 5-HT activity (Coccaro, Astill, Herbert, & Schut, 1990; Coccaro & Kavoussi, 1997; Moss, Yao, & Panzak, 1990; Soloff, 2000). These results reveal that the relation between aggressive, mood-dependent behavior and 5-HT activity is common to many personality disorders and may not be specific to BPD (Gurvits, Koenigsberg, & Siever, 2000).

Genetic studies have also focused on 5-HT candidate genes. Briefly, there are seven 5-HT receptor types (5-HTR1–7) and several subtypes within each of these classes (e.g., 5-HTR1A, 5-HTR1B). There are also several studies that have examined the role of genes coding for the serotonin transporter (5-HTT) and tryptophan hydroxylase (TPH), a rate-limiting enzyme in the biosynthesis of 5-HT (Skodol et al., 2002). Findings suggest that individuals with BPD have fewer platelet 5-HTT binding sites, which is likely due to polymorphisms of the 5-HTT gene (Greenberg et al., 1999). 5-HTT plays a significant role in the reuptake of 5-HT from the synaptic cleft. Individuals who are either heterozygous or homozygous for the short allele (*s/s* or *s/l*) of the 5-HTTLPR (a common polymorphism of the 5-HTT gene on chromosome 17) appear to have faster 5-HT reuptake and are at greater risk for developing depression following childhood maltreatment (Caspi et al., 2003). The *s* allele also has been associated with harm avoidance and impulsivity (for a review, see Goodman, New, & Siever, 2004). Finally, research has indicated a correspondence between individuals with the *s* allele and greater amygdala activation to fearful stimuli as measured by functional MRI (Hariri et al., 2002). Additional 5-HT genes have been studied for their relation with BPD, impulsive, and self-injurious behaviors, but findings require further replication (Anguelova, Benkelfat, & Turecki, 2003; Du, Bakish, Lapierre, Ravindran, & Hrdina, 2000; Huang, Grailhe, Arango, Hen, & Mann, 1999; Rujescu, Giegling, Sato, & Möller, 2003; Turecki et al., 2003). Researchers have also explored polymorphisms of the *U* and *L* alleles of the *TPH* gene (TPH is a rate-limiting enzyme of 5-HT synthesis) and the *TPH* gene's relation to impulsive aggression (for a review, see Gurvits et al., 2000). Zaboli et al. (2006) used a risk haplotype analysis to explore the TPH-1 and TPH-2 isoforms in a sample of suicidal Caucasian women with BPD. Zaboli et al. concluded that TPH-1 was associated with BPD in their sample. Their finding providing further evidence for a link between functioning of the 5-HT system and impulsive aggression.

Dopamine. Although no studies have tested dopamine (DA) functioning directly in individuals diagnosed with BPD, there is an emerging consensus that DA dysfunction contributes to the affective, cognitive, and behavioral traits seen in the disorder (Friedel, 2004; Skodol et al., 2002). Nevertheless, the specific mechanism of DA dysfunction (hypo- versus hyperfunctioning) remains unresolved. Joyce et al. (2006) found a significant association between the 9-repeat allele of the DA transporter (*DAT1*) and BPD in two independent samples of depressed adults diagnosed with BPD. *DAT1* is involved in the reuptake of DA from the synaptic cleft within the mesolimbic DA system and likely affects the amount of DA available for neurotransmission. Although Joyce et al. imply that the genetic variations associated with *DAT1* in BPD result in

hyperdopaminergic functioning (leading to the psychotic-like features of BPD), the same *DAT1* abnormalities have been associated far more consistently with trait impulsivity arising from hypodopaminergic states (Sagvolden, Russell, Aase, Johansen, & Farshbaf, 2005). In fact, findings from numerous research groups indicate that the same polymorphism of the *DAT1* gene (i.e., the 9-repeat allele) identified by Joyce et al. is linked to ADHD (Kim, Kim, & Cho, 2006) and other externalizing behavior patterns, including both CD (Young et al., 2002) and alcohol use among individuals high in novelty seeking (Bau et al., 2001). Moreover, stimulant medications (e.g., methylphenidate), which are commonly used in treatment of ADHD, function primarily by inhibiting *DAT1* receptors, thereby increasing striatal DA activity. Finally, theories of hyperdopaminergic functioning are inconsistent with (a) the linkage between high central DA activity and positive affectivity (Ashby, Isen, & Turken, 1999) and (b) the finding that low central DA activity predicts negative affectivity and trait irritability (Laakso et al., 2003), which are broad features of BPD. Taken together, these findings suggest that the impulsivity and negative affectivity characteristic of BPD are more likely related to hypodopaminergic functioning (see also Gatzke-Kopp & Beauchaine, 2007).

Vasopressin and monoamine oxidase. Some investigators have hypothesized that vasopressin, a neurotransmitter involved in the expression of both aggressive behavior and pair bonding, is associated with borderline pathology (Gurvits et al., 2000; Teicher, Andersen, Polcari, Anderson, & Navalta, 2002). Increased levels of vasopressin consistently correlate with higher levels of aggression in animal studies (e.g., Ferris & Potegal, 1988) and correlate negatively with 5-HT functioning among individuals who meet criteria for personality disorders (Coccaro, Kavoussi, Hauger, Cooper, & Ferris, 1998). It is believed, based on these and other findings, that the vasopressin and 5-HT systems may interact to promote aggressive behavior (see Delville, Mansour, & Ferris, 1996). Similarly, monoamine oxidase (MAO), an enzyme involved in the breakdown of monoamine neurotransmitters, has been hypothesized to contribute to borderline pathology. There are two forms of MAO: A and B. Research on the MAOA gene grew rapidly following a publication by Brunner, Nelen, Breakefield, Ropers, and van Ost (1993) that suggested that a point mutation in the gene was associated with violent behavior and possibly risk for suicide. Polymorphisms in the MAOA gene appear to interact with risky rearing environments to potentiate impulsive and aggressive behavior. For example, Caspi et al. (2002) found that the high-risk allele of the MAOA gene resulted in high levels of aggression only in combination with early child maltreatment. This finding may have implications for the emergence of BPD. Platelet studies of MAOB have consistently found a negative correlation between peripheral MAOB and impulse control disorders, including ADHD, antisocial personality, criminality, BPD, pathological gambling, and alcohol/drug abuse (see Zuckerman & Kuhlman, 2000).

In summary, functional deficiencies in 5-HT and possibly DA, MAO, and vasopressin are likely associated with the impulsive, aggressive, and self-injuring features of BPD. However, emotional lability, a hallmark of the disorder, may be better accounted for by deficits in the cholinergic and noradrenergic systems and by elevated hypothalamic–pituitary–adrenal axis responding. As discussed below, such associations do not imply biologic determin-

ism. Indeed, biological systems are highly sensitive to environmental inputs across the life span.

Acetylcholine. Cholinergic neurons innervate several structures within the brain that are involved in emotion regulation, among them, the amygdala, the hippocampus, and the dorsal tegmental complex, portions of both the striatum and the cingulate cortex. Furthermore, the effectiveness of antidepressants may be due in part to the inhibition of nicotinic acetylcholine receptors (NAChRs; Shytle et al., 2002). NAChRs contribute to the regulation of several mood-related processes and physiological functions, such as sleep, arousal, fatigue, anxiety, pain processing, food intake, and cognitive functions (Gotti & Clementi, 2004). The primary theory of cholinergic dysfunction in mood disorders suggests that complex interactions between the cholinergic and adrenergic systems lead to some depressive features (see Shytle et al., 2002). An imbalance in cholinergic versus adrenergic system activity could develop following prolonged exposure to stress. Thus, as a consequence of the stress response, central acetylcholine turnover is increased and leads to chronic increases in heart rate, blood pressure, dysphoria, depression, anxiety, irritability, aggression, and hostility. All are traits associated with BPD.

Noradrenergic dysfunction. The noradrenergic (NE) system is believed to subserve individual differences in reactivity to the environment, irritability, mood regulation, social affiliation, and affect (Cloninger, 2000; Gurvits et al., 2000; Skodol et al., 2002). Medications that selectively target the NE system appear to be more effective than SSRIs at treating some forms of depression, in particular melancholia (Pinder, 2004). Furthermore, increases of NE activity achieved with reboxetine lead to increased social engagement and cooperation and to reduced self-focus among randomly assigned typical volunteers (Tse & Bond, 2002). Evidence indicates that tyrosine hydroxylase (the rate-limiting enzyme of biosynthesis of NE) can be up- or down-regulated through environmental and pharmacological mechanisms. For example, NE depletion that occurs as a result of exposure to chronic stress leads to up-regulation of tyrosine hydroxylase proteins in rats (e.g., Melia et al., 1992). However, further studies of those with BPD are needed (see Paris et al., 2004).

Hypothalamic–pituitary–adrenal axis. Evidence also suggests that chronic stress leads to elevated hypothalamic–pituitary–adrenal (HPA) axis responding. Furthermore, animal models suggest the HPA axis and the central NE system interact with one another, leading to dysregulated stress responses. In particular, chronic stress leads to NE facilitation of HPA reactivity, and this facilitation appears to be the means through which exposure to severe or repeated stress results in extreme dysregulation among vulnerable individuals (Pardon, Ma, & Morilak, 2003). There is also increasing evidence that the HPA axis is involved in suicidal behavior. This evidence comes from studies that used the dexamethasone suppression test (DST), a cortisol challenge that indexes HPA axis reactivity. Nonsuppression of cortisol in response to the DST marks HPA axis hyperactivity, which in turn predicts heightened suicide risk over impressive intervals of time (Lester, 1992). For example, Coryell and Schlesser (2001) followed a group of depressed patients over 15 years and found that those who were cortisol nonsuppressors on the DST were at 14-fold greater risk of death by suicide than were those who suppressed cortisol in response to the DST. However, several cross-sectional studies have failed to find an association between suicidal behavior and cortisol nonsuppression on the DST (e.g., Pitchot, Reggers, Pinto, Hansenne, & Ansseau, 2003), and further work is needed.

Central and peripheral correlates of BPD. Each of the neurotransmitter systems discussed above operates within multiple interconnected neural structures that are believed to play a role in the expression of BPD. Both emotion dysregulation and impulsive aggression have neurobiological substrates that have been described in several reviews (Beauchaine, 2001; Beauchaine, Katkin, Strassberg, & Snarr, 2001; Brendel, Stern, & Silbersweig, 2005; Davidson, Putnam, & Larson, 2000; Goldsmith & Davidson, 2004; P. A. Johnson, Hurley, Benkelfat, Herpertz, & Taber, 2003). A recent series of studies of BPD among adolescent females suggests that there are differences in brain maturation that are measurable with electroencephalography (Ceballos, Houston, Hesselbrock, & Bauer, 2006; Houston, Ceballos, Hesselbrock, & Bauer, 2005). These studies and others suggest that BPD is associated with deficits of frontolimbic circuitry (Brendel et al., 2005), including the orbitofrontal cortex and the ventrolateral prefrontal cortex, as well as the amagdyla, hippocampus, fusiform gyrus, anterior cingulate cortex, basal ganglia, and thalamus. Of note, the orbitofrontal cortex, prefrontal cortex, and basal ganglia are all rich in DA neurons and are also implicated in almost all forms of externalizing behavior among males (Gatzke-Kopp & Beauchaine, 2007).

There are several theories outlining specific relations between frontolimbic dysfunction and the vulnerabilities for emotion dysregulation and impulsivity (see work by Davidson and colleagues, e.g., Davidson, Putnam, & Larson, 2000; and Mann, 2003). These theories suggest broadly that prefrontal circuitry involved in inhibiting behavioral responses is insufficient in light of overpowering limbic activity (see also Le Doux, 1992; Shaw et al., 2005). Indeed, functional MRI (fMRI) data indicate that changes in amygdala and prefrontal activation are observed reliably in studies in which individuals are instructed to regulate emotional responses (e.g., Schaefer et al., 2002). Findings also suggest an association between the amygdala–prefrontal circuit and genetic variations in the serotonin transporter (Heinz et al., 2004). In a study of social cooperation, King-Casas et al. (2008) found that individuals with BPD had reduced activity of the bilateral anterior insula during a trust game. These and other data (see Caspi et al., 2003) reveal that emotion dysregulation, interpersonal conflict, and impulsivity are complexly interrelated and that transactions between genetic, environmental, and neuroanatomical risk may contribute to the development of BPD.

Autonomic functioning. There is a rich theoretical literature linking measures of the autonomic nervous system to the central nervous system substrates of various psychological conditions (for a review, see Beauchaine, 2001; Beauchaine et al., 2001). Of particular relevance to the development of emotional lability may be the functioning of the parasympathetic nervous system (PNS). PNS activity can be indexed by respiratory sinus arrhythmia (RSA), a marker of vagal influences on heart rate fluctuations across the respiratory cycle (also referred to as vagal tone; Berntson et al., 1997). Individual differences in RSA are associated with social affiliative behaviors and emotion regulation capabilities, with reduced RSA conferring risk for psychopathology and heightened RSA buffering against risk (Beauchaine, 2001; Katz & Gottman, 1997; Shannon, Beauchaine, Brenner, Neuhaus, & Gatzke-Kopp, 2007). Consistent with this research, reduced RSA has been observed across numerous populations characterized by

poor emotion regulation, including severe conduct problems (Beauchaine et al., 2001), nonsuicidal and suicidal self-injury (Beauchaine et al., 2001; Crowell et al., 2005), trait hostility (Sloan et al., 1994), and both depression and anxiety disorders (Lyonfelds, Borkovec, & Thayer, 1995; Rechlin, Weis, Spitzer, & Kaschka, 1994; Rottenberg, Wilhelm, Gross, & Gotlib, 2002; Thayer, Friedman, & Borkovec, 1996; Yeragani et al., 1993). Thus, it is likely that individuals at risk for BPD will also show reduced RSA, a hypothesis that we are testing.

Psychosocial Risk Factors for the Development of BPD

Family psychopathology. There is a long history of research on psychopathology among the family members of individuals with BPD. The focus of this research has changed over time, depending on the preferred etiological formulations of the investigators (for a review, see White, Gunderson, Zanarini, & Hudson, 2003). Researchers initially investigated disorders in the schizophrenia spectrum, then mood disorders, and more recently impulse control disorders. These studies reveal that, among individuals with BPD, there is virtually no relation between BPD and schizophrenia spectrum disorders, a moderate relation between BPD and major depressive disorder, and a significant familial aggregation of impulse control disorders. Thus, the literature on the familial aggregation of psychopathology among those with BPD is positive for both mood and impulse control disorders, which likely confer vulnerability for BPD via both biological and social mechanisms.

Disrupted attachment relationships, invalidating environments, and child maltreatment. Disrupted interpersonal relationships have long been described as a risk factor for the development of borderline pathology (Gunderson, 1996; Linehan, 1993). Many theorists have hypothesized that histories of disrupted attachment relationships are common among those with BPD (e.g., Levy, Meehan, Weber, Reynoso, & Clarkin, 2005). Maternal sensitivity and related attachment processes are believed to play a particularly important role in human development. Theories of attachment posit that effective parent–child interaction patterns (proximity seeking, touching, and soothing in childhood and fostering security and autonomy in adolescence) lead to the development of an enduring emotional connection between children and their caretakers (see Bowlby, 1990). Attachment research with humans has focused traditionally on behaviors such as maternal sensitivity and reciprocity (Ainsworth, Blehar, Waters, & Wall, 1978) and given minimal consideration to the role of genes or other biological influences (see David, Borelli, Beauchaine, & Waters, in press). However, a compelling line of research by Lakatos and colleagues (Gervai et al., 2005; Lakatos et al., 2002; Lakatos et al., 2000) indicates that dopamine DRD4 polymorphisms in children are related to disorganized attachment patterns with their parents. This suggests that gene–environment interactions may influence attachment processes across development. Furthermore, preliminary evidence from the same lab suggests that the lack of the T.7 haplotype of the *DRD4* gene may constitute a resiliency factor in the development of early attachment difficulties (infants who did not carry the T.7 haplotype were more likely to develop a secure attachment to their mother). Among individuals diagnosed with BPD, increasing evidence suggests disrupted attachment processes. A consistent association between BPD and insecure attachment, particularly the unresolved, preoccupied, and fearful sub-

types, was found in a review of 13 adult attachment studies, (Agrawal, Gunderson, Holmes, & Lyons-Ruth, 2004). However, the relation between childhood attachment experiences and the development of BPD cannot be known without prospective, longitudinal research.

As reviewed above, Linehan (1993) proposed that the development of BPD occurs in part due to an invalidating family environment. There are some prospective data to support the notion that emotional underinvolvement by parents impairs a child's ability to socialize effectively. J. G. Johnson et al. (2002) found that children raised in such environments are at increased risk for engaging in suicidal behaviors and making suicide attempts, even after controlling for parental psychopathology. Despite the retrospective nature of nearly all studies relating abuse and self-injurious behaviors (for a review, see Gratz, 2003), results are remarkably consistent with those from prospective studies, suggesting that adolescents and young adults with an abuse history are about three times more likely to engage in suicidal behaviors than controls (Brown, Cohen, Johnson, & Smailes, 1999; Dube et al., 2001). Similar findings from the literature on nonsuicidal self-injury suggest that childhood trauma is a significant risk factor for the initiation of "self-destructive behavior" (Green, 1978) but that a lack of secure attachment may maintain the behavior (van der Kolk, Perry, & Herman, 1991). These findings are clearly relevant to the development of BPD.

An environment characterized by neglect, physical abuse, emotional abuse, or sexual abuse is extremely invalidating. However, the exact nature of the relation between childhood abuse and the development of BPD has been the subject of extensive debate. Some investigators (e.g., Zanarini, 2000) have presented evidence that a high percentage of individuals with BPD report a history of neglect (92%), physical abuse (25%–73%), and sexual abuse (40%–76%). Thus, many have described abuse as a critical risk factor and even a central etiological factor in the development of BPD (Herman & van der Kolk, 1987; Soloff, Lynch, & Kelly, 2002). Some researchers have sought to characterize BPD as a form of chronic posttraumatic stress disorder, given the high rates of abuse reported (Herman, Perry, & van der Kolk, 1989). Conversely, other researchers have criticized the retrospective nature of these reports of abuse and have highlighted the importance of not viewing any single event as the most important risk factor for the development of BPD (Zanarini et al., 1998). The current consensus in the literature is that even though a history of abuse is common among individuals with BPD, it is neither necessary nor sufficient for the development of the disorder (e.g., Zanarini et al., 1997).

Sociocultural correlates of BPD. The potential impact of broad environmental influences, such as culture, race–ethnicity, neighborhood, and/or socioeconomic status (see Chavira et al., 2003), on the development of BPD is an area that has received relatively little research attention. Evidence to date suggests that BPD has been identified in all of the countries in which it has been studied (e.g., Ono & Okonogi, 1988; Pinto, Hemangee, Shobha, Bharati, & Dewan, 2000), has roughly the same prevalence (typically on the basis of small samples; see Lieb et al., 2004), and shows similar rates of heritability cross-culturally (e.g., Distel et al., 2008). Taken together, these findings appear to indicate that the relative impact of culture on the development of BPD is limited. However, recent evidence from a large epidemiological

study assessing BPD among 34,653 American adults suggests that BPD has a higher prevalence than has been previously documented (5.9%) and that there appear to be significant differences in the prevalence of the disorder across racial–ethnic groups (Grant et al., 2008). Nationwide, there are greater rates of BPD among Native American men (13.2%) and relatively lower rates among Asian women (2.5%). A higher prevalence of BPD was found among those with lower incomes. However, future research is needed to determine how culturally driven experiences may serve as protective or risk factors for the development of BPD and/or whether current diagnostic criteria are culturally appropriate (Grant et al.).

A Biosocial Developmental Model

According to the developmental psychopathology perspective, the development of BPD is likely influenced by characteristics of the child, the caregiver, the environmental context, and dynamic transactions among these characteristics. In this review, we propose that early biological vulnerabilities for impulsivity and emotional sensitivity may contribute to temperamental and behavioral qualities of youth on a BPD trajectory. Given the literature reviewed above suggesting that BPD cannot be diagnosed reliably among children and young adolescents and is rarely diagnosed even among older adolescents, much of the evidence in support of this proposition is extrapolated from adult populations. In light of these limitations, the developmental literature on BPD has been understandably sparse. However, the fact that co-occurrence of impulsivity and emotion dysregulation is not unique to BPD allows etiological hypotheses to be drawn from the literatures on CD and ASPD, among other disorders (e.g., substance use disorders).

In addition to outlining potential biological vulnerabilities, we have reviewed environmental risk factors that have been linked to BPD. We emphasize, consistent with a developmental psychopathology perspective, that it is the transaction between biological and psychosocial variables across development that contributes to the emergence of BPD. Moreover, we assert that BPD, a disorder characterized by extreme emotion dysregulation that affects thought and behavior, has temperamental and behavioral precursors that emerge at different times over the course of development and with varying levels of predictive specificity. Thus, future research will likely determine (a) which markers can be identified early, (b) which markers are general risk factors for adverse outcomes, (c) which if any are specific to BPD, and (d) how vulnerabilities and risk factors interact both with protective factors and with one another to predict equifinal and multifinal outcomes.

We present our biosocial developmental model in Figure 1. Below we describe briefly the model and then present etiological hypotheses that emerge from this developmentally informed conceptualization of BPD. As indicated, biological vulnerabilities affect children's temperament (child contribution), which in turn affects environmental contexts (high-risk transaction), which subsequently affect children's biological functioning in several of the mood- and emotion-subserving systems described in earlier sections. The high-risk transaction is illustrated as an escalating process (indicated by the spiral arrow) in which qualities of the child and the caregiver lead to intensification of extreme emotional displays and reinforcement of emotional lability. We suggest that such interactions increase risk for emotion dysregulation, in par-

ticular high emotional sensitivity, intense and more frequent responses to emotional stimuli, and a slow return to emotional baseline. Behavioral and cognitive reactions to emotional situations are dysregulated as a consequence of emotional lability and result in distorted information processing, difficulties regulating actions to achieve non-mood-dependent goals, problems controlling emotion-linked behavior, and shutting down/freezing.

Figure 1 reflects Linehan's (1993) theory that the relation between psychopathology/emotion dysregulation and persistent cognitive, emotional, social, and behavioral outcomes is mediated by a history of increasingly more extreme and more disorganizing emotional responses. When these reactions occur repeatedly over months and years, emotion dysregulation becomes traitlike and outcomes such as social isolation, hopelessness, sadness, shame, anger, and repetitive impulsive behaviors (among others; see rectangle box for examples) become canalized. These traits and behaviors (which likely first emerged in instances of extreme dysregulation) become increasingly frequent and reinforcing via their discovery as an emotion regulation and/or avoidance strategies. Thus, we assert that early vulnerability interacts with learning history to shape and maintain dysregulated emotional, behavioral, interpersonal, and cognitive aspects of the "self" and thereby create the "borderline" personality.

Etiological Hypotheses of BPD

In order to extrapolate developmental hypotheses drawn, in part, from a literature on adults, one must illustrate that there is some degree of continuity from early temperamental predispositions to later adult personality. Indeed, although evidence suggests that diagnosable BPD does not emerge until late adolescence, there is a rich developmental literature linking early temperament with later personality (for a review, see Halverson, Kohnstamm, & Martin, 1994). Moreover, both empirical and theoretical work suggests that many aspects of adult personality emerge early in childhood and are driven largely by biological predispositions (Caspi, 2000; Kagan & Snidman, 2007). For example, Caspi and colleagues found that temperamental variables at age 3 predict adult personality structure, the emergence of psychopathology, and antisocial behaviors in adulthood (Caspi, 2000; Caspi et al., 2003). In particular, youth who were identified as undercontrolled were subsequently at greater risk for developing externalizing disorders, whereas those who were inhibited were at greater risk for developing internalizing disorders (Caspi, 2000). Thus, it appears that temperamental differences that can be observed early in life are linked to adult personality, patterns of interpersonal relations, and the development of psychopathology (see also Beauchaine & Neuhaus, 2008; Kagan, 2008). In the remainder of this article we synthesize research on known biopsychosocial correlates of BPD with the developmental literature in proposing five testable hypotheses.

> *Hypothesis 1:* Poor impulse control and emotional sensitivity are early biological vulnerabilities for BPD.

When viewed cumulatively, studies conducted to date suggest that BPD is associated with deficits in both impulse control and emotion regulation. Family studies reveal significant familial aggregation of both impulse control disorders and mood disorders
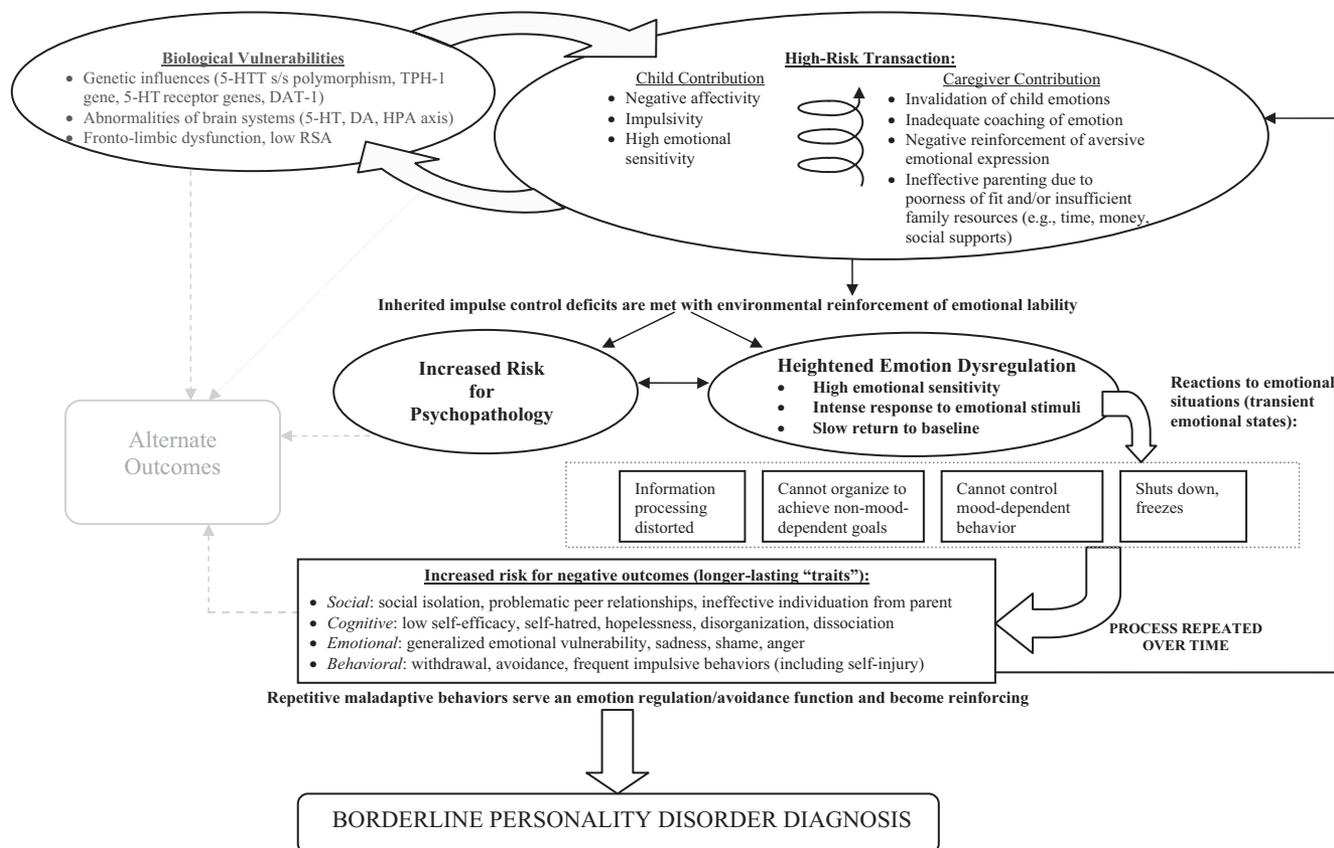
Figure 1. A biosocial developmental model of borderline personality. 5-HT = serotonin; DA = dopamine; HPA = hypothalamic–pituitary–adrenal; RSA = respiratory sinus arrhythmia. Figure adapted from "The Development of Borderline Personality and Self-Injurious Behavior," by S. E. Crowell, T. P. Beauchaine, and M. F. Lenzenweger, 2008, in T. P. Beauchaine & S. Hinshaw (Eds.), *Child Psychopathology* (p. 528), Hoboken, NJ: Wiley. Copyright 2008 by John Wiley & Sons, Inc. Adapted with permission.

among those diagnosed with BPD (White et al., 2003). This makes BPD unique when compared with other disorders of childhood, such as ADHD and depression, which appear to involve more circumscribed biological vulnerabilities that are more often restricted to dysfunction in specific neural systems (Beauchaine, 2001). Yet, like BPD, some disorders do appear to result from biological vulnerabilities across multiple neural systems. Examples include CD and ASPD, which are associated with dysfunction in mesolimbic and mesocortical structures that subserve impulse control and with disruption in medullary functions that subserve emotion regulation and emotional expression (see Beauchaine, 2001; Beauchaine, Gatzke-Kopp, & Mead, 2007; Marsh, Beauchaine, & Williams, 2008). Thus, we draw from these related literatures in our exploration of potential biological underpinnings of BPD.

Research on externalizing psychopathology suggests that impulsivity is highly heritable, although its specific expression is linked with environmental opportunities (Beauchaine & Neuhaus, 2008; Beauchaine et al., 2008; Cadoret, Leve, & Devor, 1997; Hinshaw, 2002; Krueger et al., 2002). In fact, the emergence of more severe behavioral and emotional dysregulation among impulsive children appears to be largely dependent on environmental risk exposure (Jaffee et al., 2005; Lynam et al., 2000; Patterson et al., 2000). Emotional lability and dysregulated anger appear to be particularly sensitive to environmental experience and may be overlaid onto temperamental impulsivity as a consequence of repeated negative reinforcement within the family (see above). Stressors in early development appear to alter already compromised dopaminergic, noradrenergic, and serotonergic pathways and may also contribute to hormonal alterations in the developing brain (for a review, see King, Tenny, Rossi, Colamussi, & Burdick, 2003). Heightened sensitivity to environmental stress may therefore mediate the relation between biological vulnerabilities and the emergence of disorders such as BPD.

Although there is limited research on the development of emotion dysregulation in BPD, research on the development of CD and ASPD suggests that broad emotion dysregulation emerges across the preschool and the middle school years among vulnerable (e.g., impulsive) children (Beauchaine et al., 2007). Data to support this assertion come from a series of psychophysiological studies with externalizing preschool children (Crowell et al., 2006), middle school children (Mead et al., 2004), and adolescents (Beauchaine et al., 2001). These studies indicate that differences in psychophysiological markers of emotion dysregulation (e.g., RSA) are

present among impulsive middle school children and adolescents but are not yet apparent among impulsive preschoolers. This suggests that at least some of the biological vulnerability for emotion dysregulation is shaped and later maintained by environmental influences. This interpretation is consistent with Linehan's theory of emotion dysregulation evolving in invalidating environments and with findings indicating that (a) emotion regulation (and RSA) are socialized within families (Calkins, 1997; Shipman & Zeman, 2001) and (b) about 50% of RSA—a consistent marker of emotional adjustment—is environmentally determined (Kupfer et al., 2005). Finally, early emotion regulation abilities, which are marked by high RSA, appear to buffer children from developing psychopathology in adverse rearing environments (e.g., Katz & Gottman, 1997). Thus, emotion dysregulation, though partly heritable, is also sensitive to environmental input. As a result, high-risk family environments may contribute to the development of dysregulated affect among impulsive and/or emotionally sensitive children. This leads to our second hypothesis.

Hypothesis 2: Broad emotion dysregulation is fostered and maintained within an invalidating developmental context.

Although we suggest that the emotionally sensitive child is vulnerable to developing BPD, this vulnerability is likely exacerbated within an invalidating caregiving environment (Linehan, 1993). Within this context, a child's expressions of emotion are often rejected by the family and life's problems are oversimplified. Consequently, the child is not taught how to modulate emotional arousal or cope with distress. Because emotional expressions are poorly tolerated within the caregiving environment, the child does not learn how to label and understand his or her emotional experiences, and the interpretation and communication of emotions is made increasingly difficult. Furthermore, in an invalidating environment, extreme emotional displays are often needed to generate helpful responses from caregivers. Thus, the family haphazardly punishes communication of negative emotions while intermittently reinforcing extreme emotional outbursts and actions.

A parallel model of the development of emotion dysregulation in CD and ASPD has received considerable empirical support. This literature outlines the processes through which emotional lability is shaped within the families of children with ADHD, leading to the development of CD among those raised in high-risk family environments but not among those raised in protective family environments (Beauchaine et al., 2007; Patterson, Chamberlain, & Reid, 1982; Patterson, DeBaryshe, & Ramsey, 1989; Patterson, Dishion, & Bank, 1984; Snyder, Edwards, McGraw, & Kilgore, 1994; Snyder, Schrepferman, & St. Peter, 1997). As articulated by Patterson and colleagues, repeated escalating exchanges between impulsive at-risk children and their parents serve as training for emotionally labile interaction patterns. Negative social exchanges (involving both punishment and negative reinforcement of emotional outbursts; see above) shape the behavior of both the parent and the child. Over time, this pattern results in increased frequency, intensity, and affective valence of negative interactions. The child learns that highly aversive behaviors and labile expressions of emotion are effective in obtaining desired ends, and this further disrupts parenting efforts.

Common to both the Linehan and Patterson models is the notion that family processes lead to increasingly more extreme emotional displays on the part of both the parent and the child. It is through such processes that emotional lability is shaped within the family context. In the case of antisocial pathology, this model of emotional lability has been confirmed in longitudinal studies where negative reinforcement of aggression predicts the later emergence of antisocial and emotionally labile behavior (Snyder et al., 1997). Furthermore, reinforcement of aggressive and emotionally labile interaction styles likely contributes to the development of antisocial behavior by fostering ineffective means of dealing with social conflict. Emotionally labile patterns of interacting are common to BPD as well. Thus, the literature demonstrating that operant processes shape emotional lability across development provides empirical support for Linehan's (1993) biosocial model and for our third hypothesis.

Hypothesis 3: Reciprocal transactions between biological vulnerability and environmental risk potentiate emotion dysregulation and lead to more extreme behavioral dyscontrol.

It is important to emphasize, as we have in Figure 1, that an invalidating developmental context is likely shaped by specific characteristics of both the caregiver and the child and their interactions over time. As reviewed above, certain child characteristics, such as impulsivity, are biologically driven and present early in development. An example from the developmental literature includes the "difficult child" exemplar described by Thomas, Chess, and colleagues (Thomas, Chess, & Birch, 1968; Thomas, Chess, Birch, Hertzig, & Korn, 1963). After nearly 50 years of research, the difficult child construct has been revised substantially; researchers have identified three broad dimensions of infant temperament: extraversion/surgency, negative affectivity, and effortful control (e.g., Rothbart & Rueda, 2005). Effortful control, which contributes to both emotional and behavioral regulation, is defined as "the ability to inhibit a dominant response to perform a subdominant response, to detect errors, and to engage in planning . . . and self regulation" (Rothbart & Rueda, 2005, p. 169). Accumulating data indicate that the neural substrates of effortful control overlap with those implicated in BPD and externalizing behavior problems, among them the prefrontal cortex and the anterior cingulate cortex (Rothbart & Rueda, 2005). Children at risk for BPD are also likely to be high on negative affectivity, which is characterized by discomfort, frustration, shyness, sadness, and nonsoothability.

As implied above, hypothesized contributions of the caregiver include (a) a tendency to invalidate emotions and an inability to model appropriate expressions of emotion, (b) an interaction style that negatively reinforces emotional arousal, and (c) a poor fit between the child's temperament and parenting style. This final point is emphasized here because it highlights the Biology × Environment transactions that shape both child and caregiver behaviors. In theory, a child with low biological vulnerability may be at risk for BPD if there is an extreme discrepancy between child and caregiver characteristics or if the family's resources are extremely taxed (e.g., alcoholism, sibling with cancer). Such situations have the potential to perpetuate invalidation, because the demands of the child often exceed the ability of the environment to

meet those demands (Linehan, 1993). The converse is also likely; a biologically vulnerable child may be protected in a well-matched environment where strong family supports are in place. Such multifinal outcomes led Linehan to propose three primary types of families that increase risk for BPD: the disorganized family (e.g., one that is pervasively neglectful or maltreating), the perfect family (e.g., one in which expressing negative emotions is taboo), and the normal family (one characterized primarily by poorness of fit). It must be noted that caregiver characteristics are not necessarily fixed or preexisting. Rather, the caregiver is also a product of complex biological, social, and psychological transactions, including evocative effects of the child on parenting style.

Hypothesis 4: There are early behavioral indications of risk for BPD.

In this review we have emphasized that the developmental trajectory leading to BPD likely begins with biologically driven temperamental vulnerabilities. However, these early vulnerabilities have low predictive specificity and most likely indicate risk for a plethora of partially overlapping psychological conditions. By middle childhood, emotionally dysregulated and impulsive youth are most likely to meet or partially meet diagnostic criteria for a mood disorder comorbid with impulsive behaviors. Therefore, youth with comorbid internalizing and externalizing psychopathology likely represent one population at heightened risk for later BPD. This hypothesis is consistent with evidence suggesting that the combination of internalizing and externalizing psychopathology increases risk for both suicidal and nonsuicidal self-injury (e.g., Verona, Sachs-Ericsson, & Joiner, 2005), which occur commonly among adults diagnosed with BPD. Indeed, as reviewed above, there is an extremely high rate of self-inflicted injury among those with BPD, of whom approximately 40%–90% engage in nonsuicidal self-injury or make a suicide attempt at some point in their life (American Psychiatric Association, 2004). Moreover, retrospective evidence suggests that nearly 1/3 of adults with BPD reported first engaging in self-injury before the age of 12 and another 1/3 began such behaviors during adolescence (Zanarini et al., 2006). This suggests that it is a minority of those with BPD whose first episode of self-injury occurs after a BPD diagnosis can be ascertained reliably.

There is also considerable overlap between characteristics observed among self-injuring youth and those diagnosed with a personality disorder (for a review, see Miller, Rathus, & Linehan, 2007). Over two decades of research supports a relation between adolescent self-inflicted injury and personality disorders, particularly borderline and antisocial pathology (Brent et al., 1994; Clarkin, Friedman, Hurt, Corn, & Aronoff, 1984; Marton et al., 1989; Marttunen, Aro, Henriksson, & Lönnqvist, 1994; Pfeffer et al., 1991; Runeson & Beskow, 1991). In a review of 14 studies of completed suicide, Linehan, Rizvi, Welch, and Page (2006) found that 40%–53% of individuals met criteria for a personality disorder. Linehan et al. also reported a high correspondence between suicide and personality disorders among youth. Moreover, self-inflicted injury was an identifiable behavior that captured features of both impulsivity and emotion dysregulation. As with other impulsive and/or addictive behaviors, self-injury represents a failure to inhibit a dominant response in the service of more distal goals or objectives. Yet evidence also suggests that it serves an

emotion regulation function (e.g., Zlotnick, Donaldson, Spirito, & Pearlstein, 1997) and is often used as a strategy to reduce emotional distress. For these reasons, among youth who have not been identified previously, the manifestation of self-injurious behaviors indicates the need to assess for additional risk factors for a borderline diagnosis.

Hypothesis 5: Traits and behaviors indicative of BPD emerge earlier than a full diagnosis and may exacerbate risk for BPD.

According to the developmental model presented here, a taxed caregiving environment perpetuates emotional and behavioral dysregulation in the biologically vulnerable child. Theorists have proposed that early neurocognitive impairment may be one such vulnerability that moderates the relationship between an insecure/disorganized attachment and a later BPD diagnosis (e.g., Judd, 2005). Thus, traits and behaviors may manifest as early as birth and likely shape the developmental context within which BPD emerges. This process is dynamic and continues into adolescence and adulthood. For example, by adolescence, extremely dysregulated behaviors (e.g., self-inflicted injury, disordered eating, substance abuse) may also impact the parent–child relationship by lessening trust and increasing conflict and rigidity. Indeed, evidence suggests that there are high levels of parent–child conflict among self-injuring adolescents. Moreover, the interaction between low peripheral serotonin and high family conflict appears to increase risk for self-injurious behaviors among adolescent girls (Crowell et al., 2008). However, self-injury is certainly not the only maladaptive behavior that may occur in the developmental progression leading to BPD. In cases where self-injury is not present in the etiological formulation, it is possible that other repetitive maladaptive behaviors serve a similar function (e.g., purging or drug abuse). Although clinically it is important to understand each individual's behavioral repertoire, from an etiological perspective the manifest behavior is not as important as the function that behavior serves. In our model, we not only assert that problematic behaviors serve an emotion regulation/avoidance strategy but also suggest that these behaviors exacerbate risk for BPD via evocative effects on interpersonal relationships and via interference with healthy emotional development.

Limitations and Future Directions

In theory, each arrow of our model represents a testable hypothesis. Thus, future research could explore the relation between the biological vulnerabilities described above and early child temperamental characteristics, including behavioral reactivity, impulsivity, and emotional sensitivity. Longitudinal studies could assess parent–child dynamics and reciprocal influences between biological and environmental risk factors in a high-risk sample. Research could also evaluate the emergence of negative social, cognitive, emotional, and behavioral outcomes and their timing and relation with familial socialization processes. It is important to explore which social, cognitive, emotional, and behavioral outcomes emerge before diagnosable psychopathology and which emerge later, as well as whether there are individual differences in these processes. The extent to which psychopathology and emotion dysregulation are separable in the development of BPD is debatable, and it remains to be determined whether both are prerequi-

sites to the development of self-inflicted injury. Future research could explore whether repetitive maladaptive behaviors in the adolescent predict increases in family conflict or whether there is family conflict specifically around these maladaptive behaviors. One might hypothesize that a known act of self-injury reduces family conflict temporarily (e.g., in an effort to prevent further self-inflicted injury) but in the long run lessens the parent's sense of efficacy and ability to place appropriate demands on the adolescent.

Our model will certainly expand with the research on cultural and societal factors that influence the development of BPD. Although the development of BPD is clearly affected by multiple contextual factors, there are very limited data on which to base culturally informed developmental hypotheses on BPD. Although the prevalence of BPD appears to be higher among Native American populations and among those with incomes below $70,000 (Grant et al., 2008), the specific mechanisms by which such risk is increased require further exploration (see also Ziegenbein, Calliess, Sieberer, & Machleidt, 2008). Our model is also limited by an inability to determine which parent characteristics are present before a high-risk child is born and which emerge as a consequence of raising a challenging child. Finally, our model presents a number of biological vulnerabilities and environmental risk factors, many of which may increase risk for the development of other psychological disorders, such as antisocial behavior, which is often comorbid with BPD (Lenzenweger, Lane, Loranger, & Kessler, 2007) and which appears to overlap on common genetic vulnerabilities (Torgersen et al., 2008). Longitudinal research is clearly needed to determine what constellation of risk factors can predict the development of BPD with specificity.

In sum, the literature on BPD suggests a complicated process through which impulsivity and emotional lability are amplified within the family context. These processes results in numerous negative consequences, which in turn lead to emotion dysregulation, behavioral dyscontrol, and impairing psychopathology among some individuals. Our model also allows for diverse, mulitifinal outcomes. An important direction for future research is to follow children at risk for BPD longitudinally in order to determine which variables contribute to both risk and resilience. Such longitudinal research could also test each component of the model outlined in Figure 1. Although the etiology of BPD remains unknown, there is now sufficient research with which to begin testing developmental theories of BPD. It is through this line of research that the early identification and prevention of this debilitating condition and associated risk for suicide can be best achieved.

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