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Borderline Personality Disorder and the Emerging Field of Developmental Neuroscience

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Over the past 2 decades there has been a dramatic shift in understanding of personality disorders, such as borderline personality disorder (BPD). What was historically viewed as an entrenched pattern of antagonistic, interpersonally dependent, and uncorrectable conduct is now seen as the outcome of complex—yet modifiable—developmental processes. The borderline label, which once inspired such harsh opprobrium in clinical communities that early diagnosis was considered taboo, is now increasingly applied to adolescents who are receiving effective treatment and desisting from a borderline trajectory. Research examining the developmental origins and early manifestations of BPD is increasing rapidly, making it an appropriate time to take stock of current developmental research and articulate an agenda for the future. We identify 4 challenges that continue to impede innovative research on borderline personality development: (a) inadequate attention to continuity and discontinuity across development, (b) medical and diagnostic systems that localize personality pathology within the individual, (c) the lingering belief that biological research is antithetical to contextual/interpersonal understandings of psychopathology (and vice versa), and (d) reluctance to reach across disciplinary and developmental boundaries to identify creative paradigms and foster innovative discovery. In order to overcome these challenges, we propose an approach to future research on adolescent borderline pathology that integrates developmental psychopathology, social and affective neuroscience, and personality theory perspectives. This intersection—the developmental neuroscience of personality pathology—offers theoretical and methodological advantages over disciplinary isolation and is fertile ground for generating novel hypotheses on the development and prevention of BPD.

Keywords: borderline personality disorder, social and affective neuroscience, personality theory, developmental psychopathology

Borderline personality disorder (BPD) is a complex and highly prevalent psychiatric diagnosis that was first recognized officially in the third edition of the *Diagnostic and Statistical Manual for Mental Disorders* (*DSM*; American Psychiatric Association [APA], 1980). However, written reports of borderline patients date back even farther, to early descriptions of those who "fell on the border" between "neurotic" and "psychotic" patient groups, as they were characterized at the time (e.g., Stern, 1938). For many reasons, these borderline patients did not fare well in standard treatment and often engendered a sense of futility and frustration among providers (see Linehan, 1993). In an attempt to understand this complex diagnosis, etiological theories of BPD were prevalent and predated official recognition in the *DSM*. It was acknowledged that personality disorders (PDs) had childhood origins (see, e.g., Masterson & Rinsley, 1975; Sherman, 1938). Similarly, Haviland (1929) noted that borderline pathology does not emerge "out of a clear sky but develops slowly and insidiously over long periods of time" (p. 624). In spite of this longstanding awareness, programmatic research on early identification of borderline traits and prevention of BPD is a relatively recent development (e.g., Crowell, Beauchaine, & Linehan, 2009; Gratz et al., 2009; Sharp et al., 2011; Stepp, Pilkonis, Hipwell, Loeber, & Stouthamer-Loeber, 2010).

This recent surge in developmental research on BPD coincides with other important trends in the field. First, there is an accumulation of empirical work linking PDs with traditional personality theory (Costa & Widiger, 1994; Kendler, Myers, & Reichborn-Kjennerud, 2011; Trull & Widiger, 2015). This research emphasizes the importance of understanding psychopathology *dimensionally* rather than categorically (Widiger & Trull, 2007), which reveals continuities from normative to disordered personality functioning and across different PDs. Second, developmental psychopathologists recognize that adaptive and maladaptive trajectories emerge through complex transactions between correlated biological vulnerabilities, contextual risk/protective factors, and developmental stage (Beauchaine & McNulty, 2013). Similar to personality theory, the developmental psychopathology perspective is also inherently dimensional because behaviors that are adaptive in one context or life stage can be maladaptive in another and vice versa (Cicchetti & Rogosch, 1996). Thus, artificial diagnostic boundaries oversimplify developmental processes and ignore other factors that make psychopathology dynamic rather than static (see

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Crowell, Puzia, & Yaptangco, 2015). Third, increasingly sophisticated measurement tools now allow for a richer understanding of biology *within* a nuanced and ever-changing social context. Indeed, biological adaptations to environmental cues begin at conception (Babenko, Kovalchuk, & Metz, 2015; Bock, Wainstock, Braun, & Segal, 2015) and are shaped continually by environmental and social exposures (e.g., Gatzke-Kopp, 2011). Over time, these day-to-day experiences shift biological reactivity and interpersonal patterns, creating the enduring patterns that define personality.

Developmental Neuroscience of Personality and Personality Pathology

These trends in the field fall broadly within three major domains of psychological research: personality theory, developmental psychopathology, and social/affective neuroscience. Although each field has a unique history, impassioned adherents, and an extensive literature, there are significant areas of overlap and complementarity. Thus, an increasing number of studies fall at the imperceptible intersections between disciplines (e.g., Uchino, 2009). For example, social and affective neuroscientists study neurobiological correlates of emotional, social, and behavioral processes (Barrett, 2012). Developmental psychopathologists are similarly interested in understanding biological processes, especially how biology changes across development, shapes developmental outcomes, and is influenced by context (e.g., oppression, culture, neighborhood context, interpersonal relationships; Sroufe & Rutter, 1984). At the intersection of these two fields, researchers examine the complex effects of social relationships on developing neurobiological systems (Steinberg, 2008). In a similar manner, researchers integrating personality theory with developmental psychopathology explore the emergence of PDs over time (e.g., Crowell et al., 2009) and also how personality traits (e.g., hostility; Smith, Glazer, Ruiz, & Gallo, 2004) relate to emotional and physical health outcomes across development. Personality theorists study the dynamic organization of behavior, emotion, and thought within a person and how those individual differences develop over time to create a stable and coherent identity (e.g., Allport, 1961). At the juncture between personality theory and social and affective neuroscience, researchers examine biologically based emotional systems that underlie individual differences in personality (Davidson, Jackson, & Kalin, 2000). These themes and their overlap are illustrated in Figure 1. As this brief sampling of work reveals, research integrating across these disciplines often promotes creative advances in the field.

Relative to the two-discipline interactions across these fields, the center of the Venn diagram is still underexplored. However, there is interest in understanding the emergence of personality and psychopathology within a biosocial developmental context. Existing work that falls at the center of these fields could be labeled the *Developmental Neuroscience of Personality* (see, e.g., Hughes,

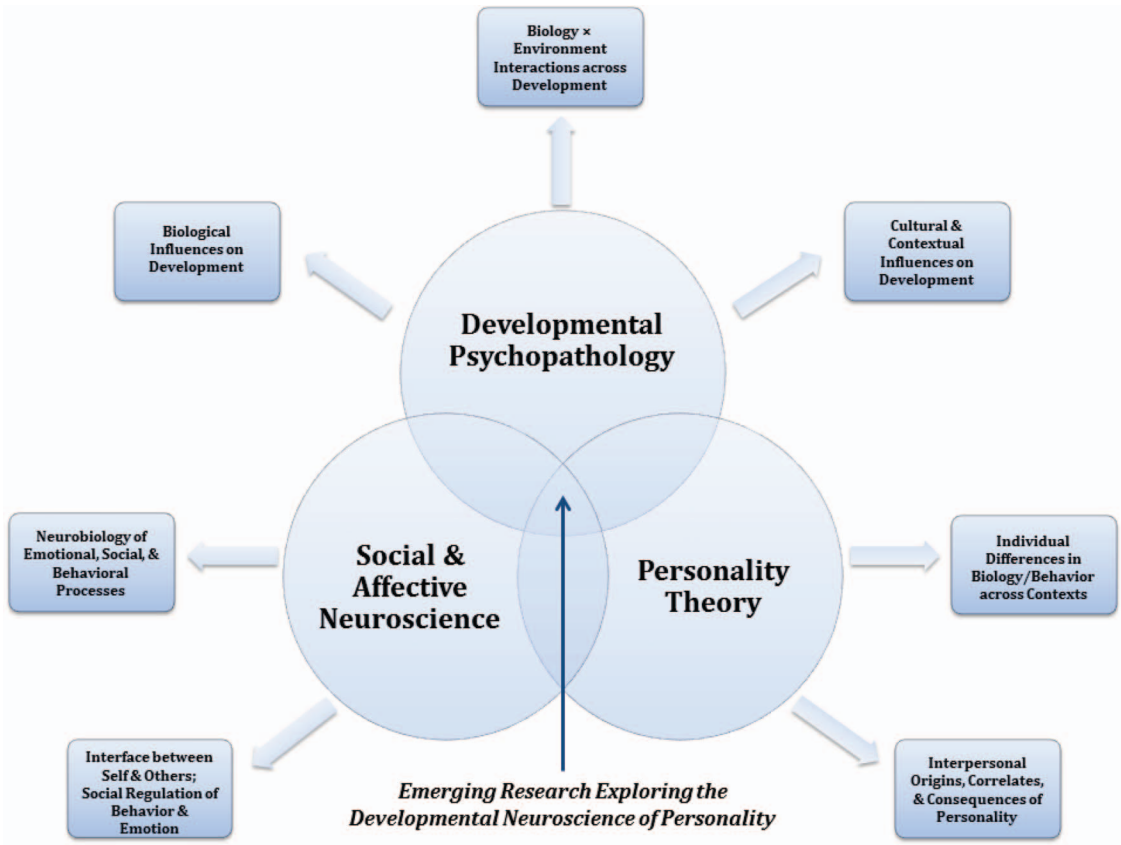

Figure 1. Conceptual origins of a developmental neuroscience approach to personality and personality disorders. See the online article for the color version of this figure.

Crowell, Uyeji, & Coan, 2012). We hypothesize that thoughtful integration across these three disciplines will continue to yield novel hypotheses and experimental designs. Nonetheless, there are important challenges that must be overcome in order to position BPD research at the juncture of social and affective neuroscience, developmental psychopathology, and personality theory. Many of the existing challenges are vestigial holdovers from early psychological research yet continue to permeate modern approaches. In this article, we explore several key limiting factors as well as alternative viewpoints, innovative approaches, and future directions for research on the development of BPD and the developmental neuroscience of PDs more broadly.

Current Challenges Facing Research on the Development of BPD

There are several conceptual threats to innovative research on borderline personality development. First, many scholars and clinicians have a limited understanding of continuity and discontinuity across development, expecting that child and adolescent manifestations of risk should be similar to the adult diagnosis or, if not, the conditions are probably not related (Beauchaine & McNulty, 2013). Second, our current medical and diagnostic systems continue to localize personality pathology within the individual, neglecting an abundance of research showing that many aspects of functioning are context dependent (see, e.g., Hopwood, Schade, & Pincus, 2014; Schaffer, Barak, & Rassovsky, 2015). This may account for any number of unnecessary diagnoses that could be more appropriately conceptualized as context and learning-dependent variations of similar underlying vulnerabilities. Third, there is a lingering belief that biological research is antithetical to contextual/interpersonal understandings of psychopathology (and vice versa). Finally, there is a reluctance to reach across disciplinary and developmental boundaries to identify creative paradigms and foster innovative discovery.

In this review, we explore each of these challenges in turn, with an emphasis on recent innovative research of relevance to the development of BPD. We argue that researchers increasingly look to early development (e.g., in utero stress exposure) to identify familial, contextual, temperamental, epigenetic, and diagnostic precursors of adult psychopathology. Such work would benefit from a multiple-levels-of-analysis approach in which biological response are measured in vivo during environmental risk exposures (e.g., family conflict) as well as during protective interactions (e.g., validation). In addition, developmental psychopathology paradigms can be extended into adult development in order to better understand how adult friendships and romantic relationships support and/or hinder emotion regulation, wellbeing, and health over time. Innovative research in the field of social neuroscience suggests that emotion regulation can be conceptualized as a dyadic process in adulthood (Coan, Kasle, Jackson, Schaefer, & Davidson, 2013) and across development (Hughes et al., 2012), yet few studies of BPD examine biological mechanisms of emotion regulation with and without coregulatory support. Thus, even though there is a growing body of research examining adolescent precursors to BPD, there are many challenges that continue to hinder a developmental neuroscience approach to personality pathology from conception to older adulthood.

Challenge One: Inadequate Understanding of Developmental Continuity and Discontinuity

Developmental psychopathologists assert that psychopathology emerges via the same mechanisms and principles that shape all aspects of human development. Thus, psychopathology is similar in many ways to developmental changes in physical characteristics or improvements in emotion regulation skills over time. Identifying precursors and trajectories leading to clinical problems may facilitate early identification of those at highest risk and could illuminate the factors that lead one person to continue along a maladaptive pathway while another desists (see Sroufe, 2013). However, psychiatry and clinical psychology have long been handicapped by an assumption that disorders of childhood should either look similar to the adult diagnosis or be given an alternate diagnostic label. For example, adolescent BPD was widely rejected until relatively recently, with many citing evidence that such youth go on to develop a range of adult psychopathology as their primary justification (Biskin, Paris, Renaud, Raz, & Zelkowitz, 2011; Lofgren, Bemporad, King, Lindem, & O'Driscoll, 1991). Personality theorists, however, have not faced similar obstacles and, as a result, there is broad support for the notion of continuity and change in personality traits. Indeed, personality is understandably complex and emerges due to endogenous biologically based traits (McCrae et al., 2000), which are shaped into enduring behavior patterns by proximal (e.g., parent–child interactions; Crowell et al., 2013) and more distal influences (e.g., cultural; Triandis & Suh, 2002). Stability *and* change are an expected part of personality development. Because PDs likely fall at the extreme of basic personality traits, one would expect similar stability and change in the emergence of PDs.

Children on a borderline trajectory will display few prototypical BPD symptoms during early development. This is unsurprising given that hallmark BPD criteria require time to emerge and/or are anchored to behaviors that are unlikely to appear before adolescence. For example, establishing a pattern of unstable and intense interpersonal relationships typically requires some degree of agency in relationships as well as a breadth of social experiences. Such conditions are not typical to the developmental context of young children. As a second example, impulsivity is a criterion for many diagnoses yet the exemplars provided for a BPD diagnosis include reckless spending, risky sexual behavior, substance abuse, and irresponsible driving (APA, 2013; Centers for Disease Control and Prevention, 2014; Dir, Coskunpinar, & Cyders, 2014). In other words, impulsivity in BPD is illustrated in the *DSM* with adolescent/adult behavior patterns.

Although BPD-specific manifestations of impulsivity and emotion dysregulation do not present in childhood, underlying trait vulnerabilities such as trait impulsivity and trait anxiety appear early and can develop into characteristic BPD features over time (see Crowell & Kaufman, in press). For example, the combination of hyperactivity, impulsivity, and attention problems is identifiable in childhood (Lynam, 1996) and shows high stability from age 17 months to 8 years (Galéra et al., 2011). Fewer than 15% of preschoolers diagnosed with attention-deficit/hyperactivity disorder (ADHD) are well-adjusted by early adolescence (Lee, Lahey, Owens, & Hinshaw, 2008), placing this group at high risk for PDs and related diagnoses. As one example, girls with a childhood diagnosis of combined-type ADHD show elevated risk for suicide at-

tempts and self-injury relative to controls, in spite of diagnostic instability over time (Hinshaw et al., 2012; Swanson, Owens, & Hinshaw, 2014). Although many youth with ADHD will not go on to develop BPD, there is accumulating evidence that impulsive children are on a high-risk developmental trajectory (Beauchaine, 2015).

Researchers exploring the developmental neuroscience of personality pathology must look beyond diagnostic continuity over time (which is low) to trait-level continuity (which is much higher). Impulsive traits are highly heritable and have a high degree of stability across development, affecting social relationships and the development of emotion regulation skills (Crowell & Kaufman, in press). For example, there is some evidence to suggest that behaviors of impulsive youth can have an evocative effect on parent–child (Burt, McGue, Krueger, & Iacono, 2005) and peer social relationships (Burt, 2009). These patterns often continue into young adulthood as those with combined-type ADHD report lower satisfaction in their romantic relationships and demonstrate higher observed negativity and lower positivity during relationship conflict (Canu, Tabor, Michael, Bazzini, & Elmore, 2014).

These interpersonal patterns appear to have biological implications across development. When social neuroscientists examine neural responses to threat, women with higher relationship satisfaction respond with decreased neural threat reactivity (Coan et al., 2013). The authors hypothesize that higher relationship quality is associated with decreased self-regulatory effort (i.e., regulation comes more easily). In other words, adults in healthy relationships may experience emotion regulation benefits similar to those experienced by securely attached infants. For many reasons, those with BPD may not experience the social or biological benefits of coregulation in family and romantic relationships. Accordingly, participants with BPD show distinct patterns of behavioral and neural activity during laboratory tasks that require trust (e.g., King-Casas et al., 2008). Thus, social norms, trust, and biological response patterns differ among those with BPD, which is consistent with a developmental neuroscience perspective. From infancy onward, biologically vulnerable youth are at higher risk for negative outcomes, mediated by social and biological processes that shape emerging self-regulatory systems. The complexity of these processes means that the outward manifestation of core traits will change across development, resulting in distinct diagnoses. However, those who appreciate the stability of underlying trait vulnerabilities will be better positioned to predict and prevent BPD.

Challenge Two: Systems That Localize Personality Pathology Within the Individual

Historically, BPD has been conceptualized as an individual problem and is typically treated in individual therapy. This approach may be insufficient, given mounting evidence that interpersonal and contextual factors shape the emergence and maintenance of this condition (see Hopwood et al., 2014; Schaffer et al., 2015). The *DSM* presents BPD as a stable, enduring, and pervasive condition, despite evidence that (a) personality and behavior patterns emerge and change gradually through Biology × Environment interactions, (b) social factors play an important role in how borderline pathology is expressed, and (c) BPD symptoms may shift or remit over time as circumstances change (see Crowell et al., 2009; Hopwood et al., 2014; Hughes et al., 2012; Johnson et al., 2000; Lenzenweger & Desantis Castro, 2005). Disrupted interpersonal functioning is well-represented among BPD diagnostic criteria (e.g., fears of abandonment, chronically unstable social functioning) and researchers find that many borderline symptoms are shaped through interpersonal processes (Beauchaine, Klein, Crowell, Gatzke-Kopp, & Derbidge, 2009; Kernberg, 1967). Although biosocial explanations of personality development are widely accepted, many studies of adolescent and adult BPD focus on independent functioning without sufficient consideration of interpersonal processes or developmental context (Hughes et al., 2012).

According to social neuroscience and developmental psychopathology perspectives, mental health conditions arise when children's biologically based temperamental factors are poorly matched to the environment (Cicchetti & Posner, 2005; Rutter & Sroufe, 2000). Thus, in order to better understand risk for BPD, we must investigate salient developmental contexts in which personality traits emerge (e.g., Hallquist, Hipwell, & Stepp, 2015). Given that early development sets the stage for later functioning, the family system is particularly important (Hughes et al., 2012; Stepp, Whalen, Pilkonis, Hipwell, & Levine, 2012). Caregivers serve as the initial source of social, affective, and behavioral regulation and can have a lasting influence on children's functioning as they age (Diamond, Fagundes, & Butterworth, 2012; Laurent, 2014; Lyons-Ruth, 2008). Parent–child transactions affect youth brain regions responsible for executive functioning, emotion regulation, learning, memory, and interpersonal affiliation such as the prefrontal cortex, limbic structures, the hypothalamus, and their functional connectivity (Hughes et al., 2012). Importantly, children are not only receiving and responding to parental cues, but are actively shaping their caregivers' behavior and carving out their own trajectories (Cicchetti, 2006; Hallquist et al., 2015). For a number of reasons, including gene-environment correlations, biologically vulnerable youth are likely to make substantial demands on their caregivers and these demands may exceed the family's capacity to provide consistent support (Crowell, Puzia, & Yaptangco, 2015; Stepp et al., 2014).

Cutting edge longitudinal research has begun to explore complex transactions between individual-level characteristics and family context in the pathogenesis of BPD. Stepp and colleagues have utilized data from the Pittsburgh Girls Study to examine a high-risk cohort of low-income families. The authors examined the bidirectional effects of parenting strategies and BPD symptoms, finding that adolescents who expressed higher levels of BPD symptoms across ages 14 through 17 were more likely to experience harsh punishment and low warmth from parents during this period. Furthermore, BPD symptoms predicted an increase in harsh parental punishment during the following year (Stepp et al., 2014). Another study found a significant interaction between youth negative emotional reactivity (assessed via self-report, ecological assessments, as well as direct observation) and exposure to family adversity (e.g., poverty, crowded housing, parental incarceration, single-parent household), where exposure to adversity strengthened the association between negative emotional reactivity and BPD symptoms (Stepp, Scott, Jones, Whalen, & Hipwell, 2016). Although hostile dyadic parent/adolescent interactions appear to increase BPD symptom severity among youth, maternal support and validation appears buffer these effects, even among those who are particularly vulnerable (Dixon-Gordon, Whalen, Scott, Cummins, & Stepp, 2016; Whalen et al., 2014). For example, Dixon-Gordon and colleagues (2016) found that maternal interpersonal

emotion regulation strategies like problem solving and displays of support and validation moderated the relation between a child's negative affect and BPD symptoms. Furthermore, maternal problem solving behavior coupled with low support and validation resulted in a stronger negative affect–BPD association among adolescents, whereas maternal problem solving behavior in the context of high support led to a weaker negative affect–BPD association. This work is consistent with theories that loved ones can serve a coregulatory function and that such relationship processes are relevant to the manifestation of BPD symptoms (see Hughes et al., 2012).

Those who go on to develop BPD may be primed to struggle in coregulatory relationships. Some studies find that individuals with BPD are unusually reactive to changes in others' emotions and nonverbal cues, are more likely to interpret neutral stimuli as negatively valenced, and have stronger physiological and emotional responses to interpersonal stressors (Daros, Zakzanis, & Ruocco, 2013; Hopwood et al., 2014; Lawrence, Chanen, & Allen, 2011; Lyons-Ruth, Choi-Kain, Pechtel, Bertha, & Gunderson, 2011). Recent functional MRI (fMRI) work within the social and affective neuroscience domain demonstrates that interpersonal cues can interfere with emotion regulation and cognitive processing (Soloff, White, Omari, Ramaseshan, & Diwadkar, 2015). Soloff and colleagues (2015) found that adults with BPD display diminished executive functioning such as compromised attention, decision making, response inhibition, and episodic memory relative to typical controls when exposed to negative affective interpersonal stimuli. However, such effects were less robust when participants were presented with neutral and positively valenced interpersonal stimuli. Thus, negatively valenced interpersonal cues may actually interfere with key executive functions among individuals with BPD. When coupled with findings that those with BPD do not differ from typical controls on nonsocial context processing tasks (Schaffer et al., 2015), these results highlight the importance of the social environment on borderline pathology. Unfortunately, in spite of a growing literature examining BPD and social relationships, most interventions still consist predominantly of individual therapy. Researchers need to expand intervention and prevention work to families, peers, and romantic partners of those with BPD or BPD traits in order to be consistent with the basic science research on personality development.

Challenge Three: Poor Integration of Biological and Contextual Research Approaches

Although most modern etiological theories of psychopathology acknowledge the importance of both biological and environmental influences, relatively few research groups employ a multiple levels of analysis approach to studying personality pathology. Even fewer incorporate biological considerations into behavioral intervention/prevention planning and research (Beauchaine, Neuhaus, Brenner, & Gatzke-Kopp, 2008). Historically, many scholars were reluctant to shift their attention to genetic and neurobiological influences on psychopathology for fear that such work might deemphasize the importance of social processes or oversimplify complex human behavior to biologically based disease (e.g., Albee & Joffe, 2004). This concern is justified in cases where mental disorders are likened to physical diseases and presumed to arise predominantly from biological abnormalities (see Deacon, 2013, for a review). Indeed, our efforts would be better served by examining biological vulnerabilities in conjunction with contextual risk, as Biology × Environment interactions and correlations can account for more variance in behavioral outcomes than either in isolation (Caspi et al., 2002; Crowell et al., 2008).

From a developmental neuroscience perspective, multiple explanatory pathways are needed to inform our understanding of BPD. Research on genetic, neural, autonomic nervous system, hormonal, and environmental levels of functioning (e.g., familial, neighborhood, cultural) are each suited to different research questions (Deacon, 2013). These influences on development continually transact such that changes in one domain typically spur changes in several others (Beauchaine et al., 2008). For example, breakthroughs in epigenetic research have significantly advanced our understanding of the mechanisms underlying Gene × Environment interactions at the molecular level in response to the intrauterine environment. These interactive effects strongly contribute to observable behavior and psychopathology (Svrakic & Cloninger, 2010).

It has become increasingly evident that genetic influences on phenotypic expressions of human behavior are indirect and malleable. DNA is now conceptualized as building blocks that influence a person's potential for adaptation — associated *probabilistically* rather than deterministically with phenotypic outcomes (Svrakic & Cloninger, 2010). For example, longitudinal research by Hammen, Bower, and Cole (2015) recently revealed that a polymorphism of the oxytocin receptor gene (implicated in social affiliation) moderates the link between early levels of family discord and later BPD symptoms. Children with an AA/AG genotype appear more sensitive to familial relationship quality. Those with the AA/AG genotype who are reared in households characterized by low discord go on to develop few or no BPD symptoms. Yet when reared in a household characterized by moderate to high familial discord, those with this genotype go on to express clinically significant borderline pathology. Children with a GG genotype appear less sensitive to familial context and develop some BPD symptoms regardless of reported level of interpersonal discord. Thus genetically vulnerable children may actually be at lower risk for BPD when raised in protective contexts.

Cutting edge research is also beginning to uncover *how* environmental risk factors implicated in BPD development shape biological processes and increase risk for the disorder. For example, childhood maltreatment is one developmental antecedent to BPD (Battle et al., 2004; Joyce et al., 2003). A recent whole-genome methylation scan of participants with BPD indicates that exposure to such adverse experiences can affect epigenetic methylation patterns (Prados et al., 2015) and other research groups have linked such epigenetic effects to problematic developmental outcomes (e.g., Martín-Blanco et al., 2014). Although researchers consistently emphasize the importance of Biology × Environment interactions, few are conducting studies that utilize a multiple-levels of analysis approach. Researcher should build upon the existing research to more effectively test and improve etiological theories of BPD.

Challenge Four: Lack of interdisciplinary Life Span Research

As this review highlights, many important advances to the study of PD development involve research designs that are interdisciplinary, longitudinal, or both. In spite of these promising advances,

there is still a long way to go. One obvious barrier to research in this area is a dearth of interdisciplinary life span training programs. Many psychology departments house life span research within a developmental program and many clinical programs divide child and adult programs into different training tracks or offer limited child training (see, e.g., Trull & Prinstein, 2012). Similarly, psychiatry programs typically offer separate fellowships and board certifications for those interested in child versus adult training. There are also challenges associated with interdisciplinary research, which can affect scholarly productivity if groups are both large and heterogeneous (Cummings, Kiesler, Zadeh, & Balakrishnan, 2013). Such obstacles must be identified and overcome if we hope to identify early social and biological mechanisms of risk for the development of BPD and other PDs.

In a recent study, Bollen et al. (2009) examined clickstream data from over a billion users of online scientific publications to create a "map of science." Each node on their map represents a highly accessed journal within a scientific discipline. The relative distance between each pair of nodes is based upon user clicks over the course of a year across different journals (http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0004803). Social sciences are well represented in the map and form a scientific hub—connected to both humanities and natural sciences. Unfortunately, child psychology and social and personality psychology are on opposite sides of the map. In other words, these data show that readers of child psychology journals are unlikely to click on articles in social and personality journals and vice versa. Even though these data were collected in 2006, which is just prior to the recent explosion of work on PD development, the distance between child and personality/social psychology is far too great. Personality theorists could still benefit from a deeper understanding of temperament and family processes just as child psychologists should better understand adult development and outcomes.

Over the past decade it has become increasingly clear that understanding personality and PD development has implications for health and wellbeing. Specifically, there is emerging evidence that many adult diseases have their origins in emotional problems of childhood (see Crowell et al., 2015 for a review). Indeed, researchers examining health differences between those with remitted versus unremitted BPD find that nonremission is associated with obesity, chronic fatigue, fibromyalgia, osteoarthritis, diabetes, hypertension, and back pain, as well as poor health behaviors, such as lack of exercise, smoking, daily alcohol consumption, sleep and pain medication usage, and emergency room visits (Frankenburg & Zanarini, 2004). Thus, there is also a need for interdisciplinary research outside of psychology, especially into the health consequences of untreated PDs.

Implications and Future Directions

As research on borderline personality development has grown, the need for cross-disciplinary and integrative research has become increasingly clear. In this article, we highlight several challenges to such work as well as how innovative contributions from developmental psychopathology, social and affective neuroscience, and personality theory may help the field to overcome these challenges (see Table 1 for a summary). Each of the three subdisciplines reviewed here is complex and interdisciplinary in its own right. However, emerging research that combines central tenets across these fields has been particularly innovative and fruitful. We hypothesize studies taking a *developmental neuroscience approach to personality* will advance research, prevention, and intervention for those at highest risk.

Our review highlights how considering etiology—particularly biological and behavioral adaptations within and across social environments—could clarify the origins of BPD traits and their emergence across distinct stages of development. Understanding probabilistic associations between childhood traits and later BPD could help prevention efforts and could alleviate functional impairment associated with problems as they emerge. Defining features of BPD such as affective instability, identity problems, impulsivity, and self-inflicted injury each emerge from a combination of underlying biological vulnerabilities and environmental factors that reinforce and maintain psychopathology (Beauchaine & McNulty, 2013; Crowell et al., 2009; Kaufman, Montgomery, & Crowell, 2014; Linehan, 1993). Our review also presents converging evidence for a paradigm shift whereby diagnosticians could benefit from adopting a view of BPD as a cluster of emotions and behaviors rather than as a discrete and immutable diagnostic entity.

In our review of the literature it also apparent that although research on family context and BPD is increasing, broader environmental risks (e.g., neighborhood) are often overlooked and cultural influences are understudied. There are some data to indicate that residence in neighborhoods characterized by concentrated poverty, unemployment, and high rates of public assistance is associated with more PD symptoms, lower levels of functioning, and poorer social adjustment, even when accounting for socioeconomic status and ethnicity (Walsh et al., 2013). However, other indicators of neighborhood risk such as proximity to environmental contaminants, walkability, or ease of access to amenities need further investigation. Similarly, although research supports the BPD diagnosis cross-culturally (e.g., Loranger et al., 1994; Wong, 2013), further analysis is needed to uncover whether there are differences in prevalence or presentation of specific BPD symptoms in nonwestern cultures. The effects of language, cultural norms, values, and expectations on the development of BPD are largely neglected in the current literature.

Furthermore, multimethod prospective longitudinal research is also greatly needed to examine how reciprocal transactions between biologically based traits and important social factors are refined into maladaptive personality functioning. Ideally, future studies would investigate: (a) contextual risk factors at multiple levels of analysis from the prenatal environment onward; (b) markers of genetic/epigenetic risk and temperamental characteristics during infancy; and (c) neurological functioning, familial psychopathology, and health behaviors across development. Multiple assessment approaches are also needed within the same samples across the life span. This work could include innovative methods, such as real-time observational assessments, epigenetic sequencing, experimental manipulations, and physiological activity monitoring. Furthermore, although studies of typical individuals in the personality theory domain have demonstrated moderate temporal stability of core personality traits (Specht, Egloff, & Schmukle, 2011; Vaidya, Gray, Haig, Mroczek, & Watson, 2008), we have a limited understanding of how extreme variations of core traits may shift over time or with intervention among those with BPD. Some have found evidence that shifts in broad personality

Table 1
Challenges and Future Directions for the Developmental Neuroscience Approach to Personality, Personality Disorders, and Psychopathology

Current challenges	Alternatives	Innovative approaches	Future directions
Inadequate understanding of continuity and discontinuity across development.	Do not assume a simple downward extension of adult diagnostic criteria into childhood. Define struggles and coping mechanisms as adaptive or maladaptive within the current developmental context. Understand that early vulnerabilities are associated probabilistically not deterministically with later outcomes.	Identifying transdiagnostic biological and environmental vulnerabilities that emerge early in the developmental trajectory and map how these factors manifest over the life span.	Enroll research participants based upon transdiagnostic traits and vulnerabilities, which will show more stability over time relative to *DSM* diagnoses.
Diagnostic and medical systems that define and treat personality disorders as an individual problem.	Developmental psychopathologists view caregivers and peers as critical for self-regulation and identity formation. Social and affective neuroscientists examine social regulation of behavior and emotion and its effects on neural systems. Personality theorists define personality as an enduring pattern of *interpersonal* situations.	Family-level approaches that examine a whole system's functioning. Social affective neuroscience research examining the moderating influence of relationships and context on key mechanisms promoting BPD traits from early development into adulthood.	Conduct treatment-outcome studies that include family members or significant others. Examine neurobiological and psychophysiological responses within a social context (e.g., during conflict or validation).
Biological research that neglects social, cultural, and family processes; contextual research that ignores biological vulnerabilities and/or biological consequences of social environments.	Many developmental psychopathologists focus on Biology × Environment interactions over time. Social and affective neuroscientists examine biological changes to social manipulations. Personality theorists examine negative and positive consequences of traits (e.g., neuroticism) on relationships and associated long-term health outcomes.	Longitudinal research using a multiple-levels of analysis approach to understand how social and biological risks interact within and across cultural contexts. Studies distinguishing Biology × Environment interactions from gene–environment correlations.	Researchers might examine how central and peripheral nervous system measures correspond to one another and behavioral responses to social manipulations targeting core BPD symptoms.
The relative dearth of interdisciplinary research, even within subdisciplines of psychology; the lack of novel downward extensions of theories and paradigms to high-risk child and adolescent samples.	Understanding that personality disorders do not emerge *de novo* in adulthood and explore the developmental origins of personality and psychopathology. Assuming that personality emerges iteratively across thousands of social and biological interactions. Conducting multi-method longitudinal research that extends adult protocols to younger samples.	Research examining the connections between fields can help to identify areas in need of novel interdisciplinary research. Studies examining health consequences of personality disorders reveal the need for further collaboration with medical researchers and providers.	Researchers could examine the long-term health consequences of adverse early experiences, including stress exposure in utero or chronic poverty.

Note. *DSM* = *Diagnostic and Statistical Manual of Mental Disorders*; BPD = borderline personality disorder.

traits predict shifts in BPD symptom presentation (see Wright, Pincus, & Lenzenweger, 2010).

When correlations and transactions among biological vulnerabilities and contextual risk factors are considered carefully, intervention strategies for all relevant levels of analysis become easier to identify (Beauchaine et al., 2013, 2008). For example, aiding families with vulnerable children should be a chief clinical priority, following evidence that social environments shape emotions, behaviors, and biological processes associated with BPD. If vulnerable youth are identified and treated early in the developmental

trajectory, it may prevent more severe neurological and behavioral symptoms from emerging. Furthermore, studies demonstrate that many personality traits are highly heritable (e.g., Krueger et al., 2002). Thus, parents of vulnerable youth could also have maladaptive traits and behaviors and, consequently, may require additional support to effectively manage the task of parenting a difficult child (Crowell et al., 2013). In light of these findings, we must make an ongoing commitment to foster life span interdisciplinary work, given the potential to decrease the burden of BPD.

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