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PERSONALITY DISORDERS (C SCHMAHL, SECTION EDITOR)

Recent Advances in the Developmental Aspects of Borderline Personality Disorder

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Abstract The aim of the current paper was to review the most recent advances in the developmental aspects of borderline personality disorder (BPD) over the last 3 years to highlight the most significant trends in the field. In so doing, we identify and discuss two exciting new trends: (a) an emphasis on the biological basis of adolescent BPD and (b) empirical evidence in support of long-held theories of the development of BPD. Together, these trends suggest that for the first time, empirical findings are beginning to emerge in support of complex and reciprocal biology × environment interactions over time in the development of BPD. We discuss the emerging literature and highlight the translational impact of this work for the assessment and intervention of adolescent BPD.

Keywords Borderline personality disorder · Adolescence · Development · Biology × environment interaction · Heritability · 5-HTTLPR · Frontolimbic · Inferior longitudinal fasciculus · Biosocial theory · Attachment · Mentalization · Genetic mediation · Diathesis-stress · Translational · Assessment · Treatment · Research Domain Criteria (RDoC)

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Introduction

Over the past 10 years, there has been a fivefold increase in empirical studies examining Borderline personality disorder (BPD) in adolescent populations [1]. Collectively, this research has firmly established the BPD construct in adolescence according to the well-established Robins and Guze [2] criteria for the validity of psychiatric disorder. Specifically, research over the last decade has demonstrated the validity of adolescent BPD in terms of its clinical description [e.g., 3], correlates and causes [e.g., 4, 5], studies that delimitate the disorder from other related syndromes [e.g., 6], follow-up studies that demonstrate a prototypical course and outcome of the symptoms [e.g., 7–9], and family studies that aim to identify a genetic basis of the biological phenomena associated with juvenile BPD [e.g., 10]. This research has also shown that adolescent BPD occurs at rates around 1 % [11] to 3 % [12, 13] in community samples. In clinical samples, rates are 11 % in outpatients [7], 33 % [14] and 43–49 % in inpatients [15].

This research has been thoroughly reviewed over the last 10 years [e.g., 16, 17]. However, research on adolescent BPD continues at an unprecedented pace. Therefore, our aim in the current paper was to review the most recent advances in the developmental aspects of BPD over the last 3 years (since 2011) in order to highlight the most significant trends in the field. In so doing, we identify and discuss two exciting new trends: (a) an emphasis on the biological basis of juvenile BPD and (b) empirical evidence in support of long-held theories of the development of BPD. Together, these trends

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suggest that for the first time, empirical findings are beginning to emerge in support of long-held developmental theories, demonstrating that BPD is the result of complex and reciprocal biology×environment interactions over time. While empirical evidence in support of such interactions has been evident for other adolescent disorders, such studies have been slow to emerge for BPD, but as reviewed here are now on the increase. Naturally, these trends will have translational impact. We therefore also discuss the effects of these trends on advances in the assessment of BPD in youth as well as the emergence of treatment outcome studies specific to BPD pathology in adolescence.

Biological Basis of Juvenile BPD

Genetic Factors BPD has been shown to be moderately heritable in adults, although specific genes are yet to be identified [17]. Heritability rates of 0.42 [18], 0.69 [19], and 0.60 [20] have been demonstrated in adults.

In the last 3 years, two studies have been published on the heritability of borderline traits in adolescents. Belsky et al. [21] examined borderline-related features in 1116 pairs of twins aged 12. The correlation for BPD traits between MZ twins was found to be 0.66 compared to 0.29 for DZ twins. Genetic factors were found to account for 66 % of the variance in borderline traits, suggesting very similar heritability for adolescents compared to adults. Bornovalova et al. [8] found that borderline traits were moderately heritable, with average heritability across age of approximately 0.3–0.5. Developmentally, heritability appeared to increase from ages 14 and 18. Importantly, this study also showed that both stability and change of BPD traits were influenced profoundly by genetic factors and modestly, but increasingly, by non-shared environmental factors, underscoring the etiological significance of young people progressively selecting their own environment, as well as support for a stress–diathesis approach to BPD, which we will return to later.

Beyond behavioral genetic designs to determine heritability estimates, only one study has, thus far, used a molecular genetic design to examine genetic polymorphisms associated with adolescent BPD. Hankin et al. [22•] demonstrated an association between 5-HTTLPR and BPD traits in 9–15 year olds that mirrored prior findings in adults. Specifically, carriers of the short allele of 5-HTTLPR exhibited the highest levels of borderline traits. Importantly, discriminant validity analyses showed that 5-HTTLPR was associated specifically with BPD traits, but not with depressive symptoms that often co-occur with BPD traits. 5-HTTLPR is an important gene to examine in relation to borderline traits because of its association with several core features of BPD, including emotion dysregulation and lability as well as stress reactivity and impulsivity [23]. Several other candidate genes have been examined in adult samples of BPD, including tryptophan hydroxylase (TPH) [24], 5HT2a [25], 5HT2c [26], and monoamine oxidase [27], which, together with the findings for 5-HTTLPR in adolescence, suggest a genetic predisposition for serotonergic abnormality associated with BPD [28]. Thus, while several developmental theories of BPD have emphasized the interaction of biological and environmental influences in the development of emotion dysregulation in BPD, molecular and behavioral genetic studies are beginning to delineate more explicitly the exact biological mechanisms in these interactions.

Neural Correlates Over the last 3 years, we have seen an increase in developmental studies to examine whether the neural correlates of core adult borderline features can be replicated in adolescence. These studies have focused mostly on structural abnormalities (reduced volume) of the anterior cingulate cortex (ACC) and atypical ACC/orbitofrontal cortex (OFC) coupling.

At least 11 neuroimaging (all structural) studies have been conducted in adolescent BPD [for a review, see 28]. Similar to findings in adults, structural imaging research has demonstrated volume reduction in the frontolimbic network in BPD with adolescents, to include the *orbitofrontal cortex* [29, 30] and the *anterior cingulate cortex* [31]. With regard to the *amygdala*, only two studies have been conducted. The first found no difference in amygdala volume between borderline and healthy control adolescents [32]. In the second study [33•], 20 female borderline adolescents were compared with 20 psychiatric and 20 healthy controls. Group differences were found for the right and left hippocampus and the right amygdala. Additionally, significant volume reductions in frontal (right middle frontal gyrus, orbital part of the inferior frontal gyrus bilaterally) and parietal regions (superior parietal gyrus bilaterally) were found in adolescents with BPD compared with controls. Only one other study has examined *hippocampal volume* in adolescence [32]. Here, negative findings with no differences between borderline adolescents and healthy controls were demonstrated. Finally, re-analyzing the same adolescent data from Chanen et al. [32] insular volume reductions were found in impulsive adolescent BPD compared to non-impulsive adolescent BPD [34] and superior temporal gyrus volume reductions were found in violent adolescent BPD compared to non-violent BPD [35].

Mixed or negative findings regarding brain areas typically associated with trauma (hippocampus and amygdala) for adolescent BPD (at least based on current structural studies) underscore the importance of examining the neural correlates of core features of psychiatric disorder earlier in the developmental course of the disorder. Specifically in the context of BPD, doing so reduces the effects of duration of illness factors upon brain morphology such as severity of the duration of the illness, treatment, cumulative traumatic events, associated

lifestyle factors or the co-occurrence, or duration of common mental disorders [36]. It also underscores the importance of biological × environmental interactions that manifest differently depending on age [37]. In other words, consistent with developmental psychopathology principles, it is possible that clearer findings for amygdala and hippocampal abnormalities begin to emerge as children age through adolescence into adulthood precisely because the environment interacts with the brain in a reciprocal fashion over time. Thus, the combined effects of biological and environmental factors influence each other synergistically rather than linearly.

Beyond volumetric studies, alterations have been identified in white matter pathways involved in emotion regulation and emotion recognition [38, 39•]. These studies, which have used diffusion tensor imaging (DTI), demonstrate abnormalities in early abnormal functional connectivity among brain regions relevant to BPD. Specifically, New et al. (2013) conducted DTI tractography in 38 BPD patients (14 adolescents, 24 adults) and 32 healthy controls (13 adolescents, 19 adults). They found bilateral tract-specific decreased fractional anisotropy (FA) in inferior longitudinal fasciculus (ILF) in borderline adolescents compared to adolescent controls. ILF FA was significantly higher in adolescent controls compared to BPD adolescents, BPD adults, and adult controls. The comparatively lower FA in BPD adolescents compared to adolescent controls suggests that the normal developmental "peak" in FA that should be achieved in adolescence does not occur in borderline adolescents further underscoring the notion of a OFC–amygdala disconnect in BPD. Such functional abnormality in the coordination among brain regions that may persist through adulthood provide important corroboration for long-held theoretical views on the developmental aspects of BPD, as we will discuss in the next section.

Empirical Evidence in Support of Long-Held Developmental Theories of BPD

So far, we have discussed the biological basis for the developmental aspects of BPD. In so doing, it was clear both from genetic and neural substrate studies that simple linear models of psychopathology where biological risk factors will predict the onset and maintenance of BPD are most likely inadequate to accurately capture and describe the development of BPD. Consistent with the developmental psychopathology principle of multifinality [40], it is more likely that children who display early signs of borderline pathology will follow different trajectories depending on the interaction of both biological and environmental risk and protective factors that they encounter through development. Accordingly, even before the above biological studies have pointed to the possibility of biology × environment interaction models for the development of BPD, most etiological theories for the development of BPD have favored a diathesis–stress approach. In this section, we discuss the two most prominent diathesis–stress approaches and present recent empirical evidence of biological × environment interactions to support these.

Biosocial Theory A very well-delineated etiological model of BPD was originally put forward by Linehan [41] and recently expanded by Crowell, Beauchaine, and Linehan [42]. In this model, a complex, heterotypic trajectory from childhood vulnerability to adult BPD begins with heritable trait vulnerabilities in the form of emotional sensitivity and reactivity in the original model [41] or trait impulsivity in the extended model [42]. These trait vulnerabilities result in the acquisition of poor emotion-regulation skills primarily through aberrant socialization mechanisms in the family context (i.e., an invalidating family environment), ultimately culminating in the complex disorder of BPD. The focus on trait impulsivity as the key underlying pathogenic process in BPD is also reflected in Paris's [43] diathesis–stress model of BPD.

In one of the first attempts to empirically validate the interaction components of the biosocial theory for BPD, a longitudinal design was used [44•]. The authors examined whether the biologically based temperamental traits of harm avoidance (HA) and novelty seeking (NS), internalizing and externalizing disorders, trauma, and perceived invalidating parenting style, measured around age 15 years, contributed to the risk of BPD five years later. Results indicated that adolescent internalizing disorders as well as the interaction of HA and perceived maternal overprotection predicted the risk of BPD five years later. The important point here is that a difficult temperament alone did not predict later BPD, but a difficult temperament in interaction with an invalidating (overprotected) environment predicted BPD. The authors suggested that an overprotecting parental style may be inhibiting the offspring's developing capacity to deal with her own emotions in self-determination.

In another interaction study, albeit not longitudinal [45], the authors examined the interrelationships among two temperamentally based traits (affective dysfunction and impulsivity), emotional abuse, and borderline features in a sample of 225 children aged 11 to 14 years. Results provide support for the role of both trait vulnerabilities and environmental stressors in childhood borderline features. Further, findings highlight the moderating role of affective dysfunction in the relationship between emotional abuse and childhood borderline features, such that elevated borderline features were more strongly associated with emotional abuse in the presence of high affective dysfunction.

Mentalization-Based Theory The attachment and mentalization-based theory of BPD [46, 47] posits that a vulnerability to failures or misinterpretations of seeing actions in

terms of underpinning mental states may account for core features of BPD. Importantly, Fonagy and colleagues have argued that as the child's attachment relationships have an important role to play in the acquisition of social cognitive capacities, disruptions of early attachment experiences can derail social-cognitive development, thereby leading to BPD [48]. Emergent mentalizing capacity is therefore the result of both genetic factors (inherited theory of mind capacity and sensitive temperament) and environmental factors (adverse family or other environment). Recently, this theory was further delineated by suggesting that mentalizing dysfunction in BPD is present not in the form of failure or suppression, but in the form of excess mentalizing (hypermentalizing) [49•].

The notion of a constitutional diathesis in the form of relational reactivity that interacts with the environment is also evident in Gunderson and Lyons-Ruth's [50] gene–environment developmental model. Specifically, hypersensitivity to interpersonal stressors contributes to the development of a disorganized–ambivalent form of attachment, leading to an escalation of problematic transactions between primary caregiver and child and, ultimately, BPD. As espoused in another diathesis–stress approach to BPD [51], the caregiving environment is not necessarily in and of itself abnormal or traumatic, but in the interaction with a vulnerable or hyperbolic temperament may put an individual at risk for developing BPD.

An obvious empirical test of the attachment and mentalization-based model of BPD is to examine whether attachment relates to a mentalization-based variable, which in turn should relate to borderline features in adolescents. Recently, we tested this hypothesis in a sample of 259 consecutive admissions to an adolescent inpatient unit (M_{age}=15.42, SD= 1.43; 63.1 % female) [49]. An interview-based measure of attachment (Child Attachment Interview [52]) was used to obtain a dimensional index of overall coherence of the attachment narrative. An experimental task was used to assess hypermentalizing (Movie Assessment for Social Cognition [53]), alongside a self-report measure of emotion dyregulation. Our findings suggested that, in a multiple mediation model, hypermentalizing and emotion dysregulation together mediated the relation between attachment coherence and borderline traits, but that this effect was driven by hypermentalizing; that is, emotion dysregulation failed to mediate the link between attachment coherence and borderline features while hypermentalizing demonstrated mediational effects.

In a study with a similar design [54], a self-report scale of mindfulness was utilized as an indirect but easy-to-use way of measuring mentalization-related phenomena in a large sample. In a sample of 501 Italian high-school students, the authors showed that the relationship between attachment-based need for approval and borderline features was fully mediated by mindfulness effects. It is important to note here, however, that concepts of mentalization and mindfulness are not identical [55] although they partially overlap. The two concepts converge in that both concern enhanced awareness of and participation in internal experience; they diverge in that mindfulness entails explicit mentalizing with respect to the self (and the self's experience of other people/inanimate objects), while leaving out implicit mentalizing and mentalizing of others' experience. Furthermore, mindfulness, as present-centered attention, underscores non-judgmental acceptance per se, whereas mentalizing encompasses an evaluative activity aimed at constructing meaning from experiences that span across the past, present, and future.

Similarly, in another study [56] the Reading the Mind in the Eyes Test (RET; [57]), the Lack of Emotional Clarity Scale from the Difficulties in Emotion Regulation Scale [58], and a self-report attachment measure were administered to three groups of adolescents based on BPD ratings (high, average, and low). High-BPD adolescents were found to score significantly lower than low-BPD adolescents on the RET and significantly higher than both other groups on the DERS LEC. When the effect of the attachment was controlled for, the high-BPD group did not show any significant difference from the other groups on mentalization measures, suggesting that attachment insecurity accounted for differences in mentalizing capacity.

In another mediational study of attachment (environment)×biologically based psychology function interaction [59], it was demonstrated that positive and negative emotion regulation strategies were differentially implicated in the link between attachment insecurity and BPD features. Attachment security functioned as a buffer against adolescent BPD by enhancing positive emotion regulation strategies, while negative emotion regulation strategies served to dilute the protective effect of attachment and positive regulation strategies, culminating in clinically significant levels of borderline traits.

Other Biology× Environment Studies In an innovative recent study using a twin design [60••], the temperamental traits of behavioral disinhibition or externalizing (EXT; impulsivity and inability to inhibit undesirable actions) and negative emotionality or internalizing (INT; predisposition to experience depression, anger, and anxiety) in interaction with child abuse (CA) to predict borderline traits over time was investigated. Three causal models were tested as follows: a direct causal model (CA→BPD), a diathesis–stress model (INT/EXT× CA→BPD), and a genetic mediation model where the CA–BPD association was better accounted for by common genetic risk factors (that is, INT, EXT, or additive INT and EXT psychopathology could account for genetic or environmental influences common to CA and BPD). The authors found strongest support for a genetic mediation model where the association between exposure to traumatic events and BPD may be better accounted for by common genetic influences rather than the former causally influencing the latter.

A diathesis–stress interaction was also demonstrated in a longitudinal cohort study of 1116 pairs of same-sex twins

followed from birth through age 12 years [61••]. The authors demonstrated that children who were exposed to harsh treatment earlier in life exhibited more borderline features at age 12 years. This association was specific to children's personal experiences of harsh treatment (i.e., not attributable to features of their families that were shared with their co-twins) and, similar to the Bornavolova study, was environmentally mediated (i.e., not attributable to gene–environment correlation). That is, children who were exposed to greater maternal negative expressed emotion or physically abused developed more borderline traits compared to peers, co-twins, and genetically identical co-twins who endured less parental maltreatment. Also, children were especially vulnerable to developing borderline traits following experiences of harsh parental treatment in the presence of a positive family history of psychiatric disorder.

In a rare study investigating the interaction of candidate molecular genetic and environmental pathogens and gender in predicting borderline features [62••], genetic variants of the oxytocin receptor genotype and the FK506 binding protein 5 gene CATT haplotype were investigated alongside multiple source reports of borderline features, conflictual relationships, attachment, self-harm, and suicide ideation in a group of maltreated vs. non-maltreated children. For both genes, maltreated girls appeared to be more at risk for endorsement of borderline features when they had minor alleles (i.e., AG or AA of OXTR and one to two copies of the FKBP5 CATT haplotype), but not when they possessed major alleles (i.e., GGfor OXTR and zero copies of the FKBP5 CATT haplotype). In contrast, maltreated boys appeared to be at increased risk for higher borderline symptoms when they had major alleles (i.e., GG for OXTR and zero copies of the FKBP5 haplotype). These effects were not obtained for maltreated boys who possessed the minor alleles (i.e., AG or AA for OXTR and one to two copies of the FKBP5 CATT haplotype). In other words, the three-way interaction for maltreated girls appears to reflect a diathesis–stress model where genotypes of the minor alleles are associated with increased risk for borderline symptoms in the presence of maltreatment (stress).

Summary As reviewed here, the two major trends in recent research regarding the developmental aspects of BPD demonstrate that the tools of the developmental psychopathology approach can be usefully applied to this topic in the form of biology×environment interactions. As we have argued elsewhere [63], evidence to date strongly suggests that constitutional (e.g., anxious, aggressive, or impulsive temperament; or genes associated with regulation of emotion or attachment relationships) and environmental factors (e.g., risk, trauma, parenting) both have etiological roles in the development of BPD. Specifically, they interact with each other over time, moderated by gender and developmental stage. Genes mark a vulnerability, but adversity in the social environment further triggers genetic propensities. Together, the biosocial and mentalization-based theories indicate that greater sensitivity to negative environmental perturbations is likely to be an important aspect of constitutional disadvantage [64] and that emotional dysregulation is an indication of this potential constitutional vulnerability. These vulnerable individuals are easily overwhelmed by negative social experiences (e.g., perceived or actual rejection, withdrawal from others, criticism, ambiguity), which compromise their capacity to assimilate and accommodate to their social environment, especially the attachment context. The same genetic vulnerability is also likely to increase the individual's potential to benefit from positive experiences under normal circumstances. Beyond a certain point, individuals who are sensitive to their social and attachment environment are likely to take an adaptive stance of self-protection, disengaging their capacity to attend to and appropriately respond to social cues. Because of this, they become unable to change (update) their mental representations and coping strategies by learning from experience. This stance, which has recently been termed by Fonagy and colleagues as *epistemic hypervigilance* [65], renders the individual impervious to positive events that would typically contribute to resilience. Interpersonal hypersensitivity and hypermentalizing may be seen as compensatory strategies or direct consequences of this adaptation, aiming to distance oneself from social (other) experience.

Translational Impact of Biology×Environment Interactions on Assessment

BPD has typically been assessed using self-report or interview-based measures. However, several BPD criteria are rather abstract (e.g., identity disturbance; lack of empathy) and require significant insight from the respondent, whether in a self-report or interview-based context [66]. For individuals with BPD, this is especially challenging because self-identified trait attributes constitute only one level of personality functioning and disregard the emergent nature of BPD which is the product of complex interactions between genetic, epigenetic, environmental, and trait dimensions over time [17, 42, 67, 68]. Moreover, self-identified trait attributes cannot account for the dynamic processes that give rise to the discrepancies between self-identified traits and behavioral manifestations typically characteristic of BPD [69]. Moreover, central to borderline pathology is a limited awareness of one's behaviors and their effects on others, especially in adolescence [70]. While multiple informants provide one solution to this problem, the emerging data on biology×environment interaction provide a novel approach to early identification and assessment of BPD in adolescents. In such an approach, any early risk factor may be used to potentially early identify an

adolescent at risk. For example, the core diagnostic features of BPD may be used (in particular identity disturbance, inappropriate anger, chronic feelings of emptiness, self-harm). Childhood disorders that may show heterotypic or homotypic patterns of continuity with BPD may be used (for example [in particular ADHD; see 71 for a review]) or early problem behavior (controlling and coercive behaviors towards attachment figures, poorly identified sense of self, hostile and distrustful view of the world, relational aggression, anger outbursts, affective instability).

However, considering these risk factors *together* would be the most powerful assessment strategy. To this end, future research using complex research designs across multiple levels of analyses are needed to build algorithms of risk. We have previously suggested that the NIMH Research Domain Criteria (RDoC) may provide a powerful tool in this regard [72]. RDoC seeks to generate a psychiatric classification system using knowledge from basic behavioral neuroscience. Whereas the Robins and Guze system that we referred to in the beginning of this paper [2] sought to find the neurobiological substrates for DSM diagnosis, RDoC's starting point is knowledge of behavior-brain relations, most notably *interactions*, that can then be linked to clinical phenomena like borderline pathology [73]. To this end, RDoC provides a matrix whose *rows* cover five domains of function, including the Negative Valence Systems, Positive Valence Systems (approach/motivation), Cognitive Systems, Systems for Social Processes, and Arousal/Regulatory Systems. Each of the domains is associated with relevant constructs selected for the potential that a particular brain circuit or area could reasonably be specified that implements that dimension of behavior. The *columns* of the matrix represent seven different units of analysis including not only genes, molecules, cells, neural circuits, physiology, behaviors, and self-report, but should also include an additional column, namely, environment. Research that utilize constructs across rows and columns to delineate the interaction of risk and protective factors for BPD across multiple levels of analyses will significantly advance the field's capacity to early identify and treat adolescents at risk.

Translational Impact of Biology×Environment Interactions on Treatment

If it is true that BPD is the result of complex biology×environment interactions, it is unlikely that any one of the "branded" psychotherapies in isolation would be maximally effective. Unless a branded psychotherapy targets a cross-cutting psychological/biological factor that appears to consistently interact with environmental factors to cause or maintain BPD, the therapy would have limited effect.

Both mentalization-based treatment for adolescents (MBT-A) and dialectical behavior therapy [DBT; 41] target potentially cross-cutting biologically based psychological processes in this regard. MBT assumes that the development of BPD in adolescence and its treatment is grounded in a phase-specific compromise in the capacity to mentalize that occurs during adolescence [74] and incorporates monthly sessions of mentalization-based treatment for families (MBT-F). DBT synthesizes a change orientation from behavior therapy with an acceptance orientation from Zen philosophy specifically to target the emotional dysregulation, distress tolerance, and interpersonal difficulties in BPD and has been adapted for adolescents [75].

MBT has been shown to be effective in an RCT in a sample of self-harming adolescents [most of whom met criteria for BPD; 76]. Although no RCTs have been reported with samples exclusively comprising adolescents with BPD [75], DBT has been evaluated in adolescents with non-suicidal self-injury (NSSI) and two BPD criteria in Norway [77]. Similar to assessment, future intervention studies of adolescent BPD would benefit from the explicit integration of biological variables into the study design. Candidate molecular genetic and environmental pathogens alongside neural substrates can be assessed in the prediction of treatment outcome, thereby delineating the combination of biological and environmental factors that may predict or maximize treatment outcomes.

Conclusions

The field has come a long way in the last decade with regard to the assessment, diagnosis, and treatment of adolescent BPD. Accordingly, the DSM-5 states that BPD may be applied to children or adolescents "when the individual's particular maladaptive personality traits appear to be pervasive, persistent, and unlikely to be limited to particular developmental stage or another mental disorder" [78, p. 647]. In addition, both the ICD-11 and the UK national treatment guidelines [79] also now "legitimize" the diagnosis of BPD in adolescence. The field is now poised for the second generation of adolescent BPD studies that go beyond traditional scientific approaches espoused by the Robins and Guze approach to include the scientific approaches offered by Developmental Psychopathology and RDoC to fully characterize biology×environment interactions, not only to elucidate the various developmental trajectories for BPD but also to inform treatment.

Compliance with Ethics Guidelines

Conflict of Interest Carla Sharp and Sohye Kim declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- •• Of major importance

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