The aetiology of borderline personality disorder (BPD): contemporary theories and putative mechanisms

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Abstract

This article presents an overview of current knowledge regarding the aetiology of Borderline Personality Disorder (BPD). It begins with a brief synopsis of early research and theory, and discusses how changing conceptualisations of BPD have impacted on our aetiological knowledge. Contemporary theories are described and presented within a developmental psychopathology framework. Deficient co-regulation and social communication in infancy are purported to underpin emotional dysregulation and social cognition deficits across development. These mechanisms are further potentiated by maladaptive social experiences in a series of positive feedback loops. Prospective research provides preliminary evidence for the reciprocal (or mediating) effects of maladaptive experiences and childhood dysregulation. Moving forward, cohort studies may incorporate neurobiological assessments to examine the biological systems underpinning phenotypic (e.g., impulsivity, disturbed relatedness) covariation.

There has been a sea change in our understanding of the aetiology of Borderline Personality Disorder (BPD). Early research focused on associations with childhood trauma, and relied on retrospective reports from clinical samples [1-3]. Theoretical models underpinned by these early studies hypothesised that childhood sexual abuse was a major risk (and aetiological) factor for BPD [4]. While trauma theories were popular in the 1980s [5], they presented an overly simplistic account of the development of BPD. It has since been recognised that severe abuse occurs in only a minority of BPD patients, and that childhood maltreatment is a non-specific risk factor for a diverse range of psychopathologies [6]. In the 1990s, trauma theories were superseded by multifactorial models, describing interactions between diatheses and stressors. Models varied in focus (i.e., cognition, attachment, emotion, trauma, social context) though all acknowledged the importance of psychosocial risk factors [7-10]. Of these models, Linehan's biosocial theory [8] is perhaps the most influential, laying the foundations for successive theories [11].

Over the last 10 years, classic diathesis-stress models have been extended to reflect a shift in focus towards the developmental precursors of BPD [12]. Concomitantly, empirical studies have increasingly focused on BPD antecedents, symptoms, or disorder from early adolescence onwards [13*]. Current theoretical models are situated within a developmental psychopathology framework [12] and employ a cross-discipline approach (e.g., social and affective neuroscience, personality theory) to consider aetiological pathways at several levels of analysis including genetic, neural, behavioural, familial, and social [11, 14, 15]. Common to these theories, risk factors are believed to impinge upon psychological processes undermining personal and interpersonal functioning across developmental stages.

Contemporary theories: common mechanisms in the aetiology of BPD

Contemporary theories for the development of BPD may be broadly categorised into those emphasising the aetiological role of emotional dysregulation [11*] or maladaptive social cognition [16*]. A comprehensive model for the aetiology of BPD (see **Figure 1**) should describe how different domains of dysfunction interactively contribute to the development of the disorder across the lifespan [17**]. Below follows a brief overview of these current theories (please refer to recommended reading for further explication).

The Biosocial Developmental Model (BDM) of BPD presents an extension of Linehan's biosocial model. The original model hypothesised that BPD is primarily a disorder of emotional dysregulation, arising from maladaptive transactions between biological vulnerabilities and an invalidating environment [8]. The BDM extends this model by considering development from a lifespan perspective [11]. Impulsivity is identified as an early trait vulnerability for emotion regulation difficulties. Thus, impulsivity and emotional dysregulation are hypothesised to emerge independently and sequentially, and are potentiated by environmental risk factors in a series of reciprocal transactions (e.g., an impulsive child in a high-risk environment may be unable to inhibit extreme emotions in the face of inconsistent parenting). Over time, maladaptive transactions contribute to negative social and cognitive outcomes, and by mid-adolescence the individual develops a set of maladaptive coping strategies. These maladaptive traits exacerbate risk for BPD by evoking further negative responses from the environment, and disrupting healthy social development.

Selby's Emotional Cascades Model (ECM) of BPD [18] complements the BDM by explicitly articulating putative processes underpinning the potentiation of emotional, cognitive, and behavioural dysregulation. The ECM states that emotional dysregulation is amplified and exacerbated by rumination in a positive feedback loop, which leads to "cascades of emotion." These cascades of emotion magnify negative affect, causing the individual to resort to extreme behaviours (e.g., self-injury) as a distraction technique. Within a broader framework, several factors contribute to the maintenance of this dysregulation cycle via bottom-up or top-down processes. Child abuse, for example, can lead to distorted cognitions which then contribute to further dysregulation cascades. In an outward process, behavioural dysregulation could elicit negative reactions from others (e.g., accusations of manipulation), further propagating dysregulation cascades. It is proposed that this network of interactions eventually leads to the emergence of BPD.

In a shift of emphasis, Fonagy and colleagues' [16] socially-oriented model of BPD places epistemic trust and social communication as central to the early development of BPD. BPD is hypothesised to emerge from a failure of communication deriving from an early breakdown in the capacity to forge learning relationships. If there is an absence of non-verbal ostensive cues

(e.g., eye contact, turn-taking) from the caregiver, epistemic trust will not be stimulated. Instead, the infant becomes persistently vigilant or closed off to the communication of social knowledge. In families with abusive or hostile caregivers, epistemic mistrust becomes entrenched in an adaptive process. Across development, epistemic hypervigilance may manifest as an over-interpretation (i.e., hypermentalisation) of the motives of others, and personality dysfunction will be maintained in a self-perpetuating cycle of social dysfunction and mentalisation difficulties. Mentalisation difficulties, in turn, lead to emotional dysregulation further disrupting the ability to mentalise [19].

Hughes and colleagues' developmental model of BPD [17] more explicitly combines emotional and social domains by considering the role of frontolimbic dysfunction within the context of Coan's social baseline theory [20]. It is hypothesised that emotion regulation is an individual and interpersonal process, and that relationships play an important role in regulating biology and behaviour across the lifespan. In a departure from Fonagy's model, the emphasis is on the co-regulation of emotions rather than the communication of social knowledge. Successful co-regulation during infancy (e.g., via secure attachment relationships) provides the foundation for later co-regulation, by strengthening the neural structures underpinning self-control. Children with poor self-control, in contrast, are less likely to be accepted be peers precluding additional sources of co-regulation across the developmental trajectory [21**]. An over reliance on self-regulation is purported to deplete prefrontal cortex resources, subsequently potentiating ineffective and/or impulsive emotion regulation strategies.

Prospective evidence for putative mechanisms

Cross-sectional findings provide a platform from which we can hypothesise about the developmental trajectory of BPD [22]. Nevertheless, we need prospective studies to clarify *how*, and in *what order*, various factors contribute to the development of BPD. There is a small but growing body of prospective studies examining mediational or reciprocal associations between environmental risk factors (e.g., maladaptive parenting), individual processes (e.g., worsening self-control), and the manifestation of BPD symptoms across early development. The current review focuses on these studies, as they explicitly test some of the mechanisms delineated in the above theories. The interested reader is directed towards Stepp, Lazarus [23*] for a

comprehensive review of individual precursors and environmental risk factors prospectively associated with BPD.

Four studies considered the aetiological role of negative social interactions (i.e., parenting, bullying) in the development of BPD symptoms. In the Greifswald Family study, Reinelt and colleagues found that negative mother-child interactions (i.e., rejection and overprotection) mediated the longitudinal transmission of BPD symptoms from mother to adolescent child [24]. These findings indicate one potential mechanism (i.e., maternal parenting) via which familial risk can contribute to the development of adolescent BPD. In the first of two studies from the high-risk Pittsburgh Girls cohort, Stepp and colleagues found that harsh parenting moderately tracked the trajectory of BPD symptoms from 14 to 17 years, suggesting a reciprocal relationship between these two factors [25]. In a sophisticated extension to this study, Hallquist, Hipwell [26**] found that the effects of harsh punishment on the development of adolescent BPD were partially mediated by the earlier reciprocal effects of harsh parenting and poor self-control. These findings appear to support aspects of biosocial developmental model (BDM) by demonstrating that a coercive cycle of harsh parenting and reduced self-control may partly explain the association between environmental risk and subsequent BPD. Also providing some support for the BDM, Winsper, Hall [21**] demonstrated in the Avon Longitudinal Study of Parents and Children (ALSPAC) that bullying in late childhood strongly mediated the association between earlier childhood dysregulation and the emergence of adolescent BPD. Although not directly tested, we hypothesised two potential (and interacting) mechanisms underpinning this mediational association. First, and consistent with social cognition/regulation models, we hypothesised that bullying may exacerbate hypermentalisation causing "emotionally labile patterns of interaction." Second, we suggested that the social stress related to bullying may "work itself under the skin" impacting on an already vulnerable stress response, further potentiating emotional and behavioural dysregulation.

Four studies examined the mediating effects of individual dysfunctional features. In a second study from the ALSPAC cohort, Lereya, Winsper [27] demonstrated several pathways via which childhood dysregulation (i.e., nightmares and daytime emotional and behavioural dysregulation) mediated associations between earlier risk factors (temperament, abuse, and maladaptive

parenting from 2-7 years) and adolescent BPD. Consistent with the emotional cascades model (ECM), we also found a significant pathway linking nightmares to BPD symptoms via an increased risk of daytime dysregulation. We speculated that nightmares may potentiate dysregulation over time via physiological (e.g., increased amygdala responsiveness) and cognitive (e.g., increase the tendency to ruminate) processes. Consistent with our findings, Bornovalova, Huibregtse [28] also found that childhood dysregulation (in the form of internalising and externalising disorders) significantly mediated the association between childhood abuse and BPD traits at 24 years. Together, these two studies support that dysregulation may partly account for the link between early maladaptive experiences and subsequent BPD. In an explicit test of the ECM, Selby and colleagues conducted micro-longitudinal assessments (over 2 weeks) to examine prospective associations between negative emotion, number of nightmares, and rumination. In support of their model, they found that daytime emotional cascades predicted subsequent nightmares, while BPD diagnosis interacted with baseline rumination to prospectively predict nightmares [29]. Finally, in a high-risk sample of mothers and their children, Carlson, Egeland [30] found that disturbances in self-representation during early adolescence significantly mediated the association between attachment disorganisation in infancy and BPD symptoms at 28 years. Consistent with the notions of Hughes, Crowell [17], these findings suggest that infants experiencing early relational disturbance have later problems with self-functioning, subsequently increasing risk of BPD.

Conclusions and future research directions

There has been a new wave of aetiological theories for BPD, with a shift in emphasis towards the early origins and developmental unfurling of the disorder. The study of BPD in younger community populations has facilitated the examination of some of the complex aetiological processes described in these theories. By tracking populations early in the developmental trajectory, we can assess risk factors when they are "aetiologically active" and prospectively test mediating and reciprocal effects between environmental and individual factors [13].

Recent prospective research has started to test potential mechanisms underpinning the early development of BPD. Studies offer some support for contemporary theories by demonstrating reciprocal (or mediational) links between maladaptive experiences (harsh parenting, bullying),

childhood dysregulation or disruptions in self representation, and subsequent BPD symptoms. It should be noted, however, that most of these studies examine BPD symptoms rather than the full clinical disorder, and only a minority use sophisticated methodology to explicitly examine reciprocal effects (i.e., though prospective, most studies examine one-way rather than bi-directional processes). Current theories (and empirical research) tend to focus on the aetiological underpinnings of emotional dysregulation and deficits in social cognition. While these are core elements of BPD, other pertinent features (e.g., identity disturbance, emptiness) currently receive little attention.

One pressing issue in our aetiological understanding of BPD is the non-specificity of highlighted risk exposures. Childhood maltreatment, for example, is associated with a range of psychopathological outcomes [31]. It is unclear why some vulnerable children exposed to risk develop BPD, while others develop different psychopathologies, i.e., "equifinal" and "multifinal" pathways [32, 33]. Non-specificity likely relates, in part, to current diagnostic categories, which tend to demonstrate heterogeneity and excessive comorbidity. Moving forward, researchers may adopt a dimensional approach (as articulated by the RDoC initiative) to explicate the neurodevelopmental underpinnings of intermediate phenotypes on the pathway to psychopathology [34]. While there may be several pathways to the development of BPD, early impairments in emotional and social domains appear to represent common denominators. We do not currently know the order (e.g., parallel versus sequential) in which these dysfunctions (and other core BPD phenotypes - cognitive and behavioural) unfold. Increased understanding of how different domains of dysfunction (e.g., disturbances in neural circuits and networks) are linked and potentiated by environmental effects could accelerate our understanding across a range of analyses from genes to behaviour [35].

At present our neurobiological knowledge relies on cross-sectional studies, with related methodological limitations [15, 36]. We now require prospective studies from conception onwards [37] with repeated environmental, phenotypical, biological, and clinical assessments. Studies should include genome wide analysis - GWAS [38, 39], as multiple genes are thought to moderate the impact of early life stress on the development of BPD [40]. Epigenetic studies

should also be utilised to help elucidate how child trauma influences gene-expression, thereby increasing vulnerability for BPD [36, 40].

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Figure 1. A tentative logic model delineating the pathogenesis of Borderline Personality Disorder (BPD) from conception onwards

This model combines recent theory to present an overview of our current knowledge regarding the aetiology of BPD (relevant references are indicated in the figure). Early origins include familial risk and prenatal exposures [37], which may heighten likelihood of temperamental predisposition and early deficits in caregiving [41*]. These features may contribute to disruptions in emotion co-regulation [17] and the transmission of social knowledge [16], having biological (e.g., frontolimbic dysfunction) and psychological (e.g., hypermentalisation) impacts. By later childhood/adolescence the individual may become embroiled in a series of "emotional cascades," which are further exacerbated by invalidating interactions or abuse [18]. Eventually (e.g., adolescence), a constellation of maladaptive traits coalesce [11], increasing risk of the clinical disorder via their evocative effects on the environment.

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**of outstanding interest

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Extending the developmental trajectory, this comprehensive systematic review examines studies providing evidence for the intergenerational transmission of BPD from mother to child. Potential mechanisms of transmission are discussed in detail.