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A Developmental Neuroscience of Borderline Pathology: Emotion Dysregulation and Social Baseline Theory

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Abstract Theoretical and empirical research has linked poor emotion regulation abilities with dysfunctional frontolimbic circuitry. Consistent with this, research on borderline personality disorder (BPD) finds that frontolimbic dysfunction is a predominant neural substrate underlying the disorder. Emotion regulation is profoundly compromised in BPD. However, BPD is also associated with broad impairment across multiple domains, including impulse control, interpersonal relationships, and cognitive functioning. To date, BPD research has focused largely on single areas of dysfunction, failing to account for overlap at either the biological or behavioral levels of analysis. We examine the literature on frontolimbic dysfunction in BPD within the context of Coan's social baseline theory. Social baseline theory proposes that healthy human functioning is dependent upon adequate social support and that, at baseline, biological systems are adapted to operate interdependently rather than independently. The social baseline perspective is particularly useful for understanding borderline personality development because the impulsive and emotionally dysregulated behaviors common among those with BPD occur almost invariably within an interpersonal context. We discuss clinical and research implications of this work.

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Borderline personality disorder (BPD) is a severely impairing behavioral disorder characterized by emotion dysregulation, poor interpersonal functioning, cognitive difficulties, and high rates of impulsive, self-damaging behaviors (American Psychiatric Association [APA], 2000). Epidemiological surveys estimate the disorder affects between 1–6% of American adults (e.g., Grant et al. 2008; Torgersen et al. 2001). Of those, approximately 70% self-injure and 8–10% complete suicide eventually (see Black et al. 2004). Borderline personality disorder is the most prevalent Axis II condition observed in psychiatric hospitals and is among the more costly diagnoses facing the health care system currently (Bender et al. 2001; Comtois et al. 2003; Trull et al. 2003). Thus, effective intervention and prevention of BPD is an urgent priority.

Advances in treatment almost invariably follow from an improved understanding of etiology, including biological and environmental mechanisms of risk (see Beauchaine et al. 2008). Preventative interventions in particular must address multiple etiological factors because emerging biological systems are sensitive to environmental moderation across development. Indeed, it is well recognized that BPD, along with nearly every psychiatric disorder, results from complex biosocial interactions that begin in early childhood (for a review see Beauchaine et al. 2009; Crowell et al. 2009).

The literature on the developmental neuroscience of BPD lags behind that of many other clinical disorders (see e.g., Kaufman and Charney 2001). This may result from the longstanding belief that BPD is a disorder of adulthood with few identifiable developmental precursors (see Crick et al. 2005). Furthermore, traditional symptom-based

approaches to understanding psychopathology do not lend themselves well to a neuroscience perspective. Biological systems rarely align neatly with single areas of dysfunction, particularly during development (Charney et al. 2002; Hyman 2007). Instead, neurobiological overlap likely accounts for much of the phenotypic covariation observed in attachment formation, affect regulation, behavioral control, and adult interpersonal relatedness. Recent advances in social and affective neuroscience may provide insight into these features of borderline personality development, allowing for more sophisticated etiological models.

Borderline Pathology and the Diagnostic and Statistical Manual

According to the current Diagnostic and Statistical Manual (DSM-IV; APA 2000), a BPD diagnosis is made when a person meets five of nine criteria for several years and also experiences significant functional impairment. These criteria are listed in Table 1. Because the diagnosis is based on a polythetic criterion set, there are several different BPD phenotypes and two affected individuals could overlap on only a single feature. However, those with BPD typically show dysfunction across several domains, which can be categorized into four broad areas of dysregulation: emotion (e.g., mood lability, anger), interpersonal (e.g., frantic efforts to avoid abandonment), cognitive (e.g., dissociation), and behavioral (e.g., impulsive behaviors; BPDRF 2006). To date, research on BPD has largely examined single areas of dysfunction, with studies of emotion dysregulation dominating the empirical literature (see Kuo and Linehan 2009).

It is not surprising that emotion dysregulation has been a focus of clinical and research attention. Poor affect regulation is central to most conceptualizations of BPD (APA 2010; Linehan 1993) and by adulthood, emotion dysregulation is the most prominent feature of BPD (Lieb et al. 2004). Therefore, negative affectivity, emotional lability, and mood-dependent behavior are likely central to the etiology of borderline pathology. However, negative emotionality confers broad, rather than specific risk for psychopathology (Beauchaine et al. 2009). To improve intervention, it is important to identify causal processes that potentiate risk uniquely for BPD, in addition to general developmental mechanisms contributing to maladaptation (Rutter and Sroufe 2000). From the developmental psychopathology perspective, this requires attention to multiple, interacting factors and how these shape behavior across the life span.

Table 1 Diagnostic criteria for 301.83 borderline personality disorder

A pervasive pattern of instability of interpersonal relationships, self-image and affects and marked impulsivity beginning by early adulthood and present in a variety of contexts as indicated by five (or more) of the following:

(1) Frantic efforts to avoid real or imagined abandonment

(2) A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation

(3) Identity disturbance markedly and persistently unstable self-image or sense of self

(4) Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)

(5) Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior

(6) Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria irritability or anxiety usually lasting a few hours and only rarely more than a few days)

(7) Chronic feelings of emptiness

(8) Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights)

(9) Transient, stress-related paranoid ideation or severe dissociative symptoms

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Developmental Psychopathology and Borderline Personality

Borderline personality disorder typically emerges as an identifiable syndrome by late adolescence to adulthood. Furthermore, personality disorder diagnoses are intended to capture stable, enduring, and maladaptive behavior patterns (APA 2000). Thus, practitioners are understandably reluctant to diagnose personality disorders among adolescents. In contrast to a traditional diagnostic perspective, which requires dysfunction at threshold levels, the developmental psychopathology approach examines continuities and risks across the life span (Sroufe and Rutter 1984). This perspective is well suited for understanding personality trajectories, since personality structure is relatively stable across development (Caspi 2000). However, existing research on the development of personality disorders is relatively new and there is little consensus regarding which temperamental antecedents best mark risk for borderline pathology (BPDRF 2006).

In particular, there is very limited research examining how personality and temperament are expressed behaviorally and within interpersonal contexts (Caspi et al. 2003a, b; Funder 2001). Among adults it is often assumed that personality is pervasive and stable. Yet personality in children is almost certainly a dynamic process that emerges gradually through Temperament × Context interactions.

Personality and psychopathology are both developmental processes that are shaped via complex risk and protective factors (Sroufe 1997). Developmental psychopathologists are interested in high-risk environments and patterns of maladaptation that pre-date the emergence of psychiatric diagnosis. Recent evidence suggests that both maladaptive traits and contextual risks for BPD can be identified prior to adulthood, suggesting that the probability of developing BPD may be higher for some individuals (Beauchaine et al. 2009; Crowell et al. 2009). Identifying those on a trajectory leading to poor emotional and behavioral control is fundamental to early intervention and prevention efforts.

Emotion Regulation and Frontolimbic Development as Interpersonal Processes

Conceptualizing Emotions and Emotion Regulation

Emotions are evolutionarily maintained capabilities that promote the behaviors necessary to survive and thrive (Cole et al. 2004; Ekman 1992). An emotion consists of both appraisal and response tendencies that often function seamlessly (Cole et al. 2004; Gross 1998b; Walden and Smith 1997). Emotional responses are automated, rapid, and dynamic, allowing a person to react quickly to salient information (Cole et al. 2004; Ekman and Friesen 1976; Gross 1998b). However, not all responses are appropriate to every context and therefore require some form of modulation. Because there is variability in emotion regulation abilities, and because poor regulation characterizes many diverse psychiatric conditions, the construct has been a focus of psychopathology research (Davidson et al. 2000). Yet, few have examined biosocial changes in emotion regulation across the life span with attention to the interpersonal context in which psychopathology emerges.

Instead, emotion regulation is almost always defined as an individual process. Regulation is thought to occur internally, when a person influences which emotion is felt, when the emotion occurs, or how it is experienced and expressed behaviorally (Gross 1998a). Emotion dysregulation is defined as a person's failure to modify emotions in any or all of these ways, often leading to context-inappropriate emotions and behaviors (Gratz and Roemer 2004). By and large, neuroscientists operate on the assumption that emotion regulation and dysregulation occur within, rather than between, individuals.

Emotion Regulation and Frontolimbic Circuitry

Researchers have used functional Magnetic Resonance Imaging (fMRI) to localize emotion regulation to frontolimbic regions of the brain (e.g., Davidson and Irwin 1999). These regions include, among other structures, the amygdala, hippocampus, hypothalamus, dorsolateral and right dorsomedial prefrontal cortices (PFC), orbital frontal cortex (OFC), anterior cingulate cortex (ACC), and insula (Banks et al. 2007; Davidson et al. 2000; Goldin et al. 2008). Voluntary regulation of negative emotion activates these and other structures that are connected functionally and structurally (Goldsmith et al. 2008). Thus, researchers hypothesize that frontolimbic circuitry underlies effortful regulation of emotions. In other words, individuals recruit PFC resources to modulate subcortical responses to emotional stimuli and inhibit behavioral impulses (see Fuster 2002).

Dysfunction of frontolimbic circuitry also appears to contribute to emotion dysregulation (e.g., Bechara et al. 1994; Damasio 1994; Sanchez-Navarro et al. 2005). Across several studies, those with psychiatric conditions show different activation and functional connectivity within frontolimbic structures (e.g., Davidson et al. 2000; Taylor and Liberzon 2007). When depressed adults use reappraisal (i.e., construing an emotional situation in non-emotional terms; Gross 1998a) to down-regulate emotions, their amygdala activation increases. In contrast, when healthy adults apply this strategy their amygdala activation decreases, likely indexing more effective emotion regulation (Goldsmith et al. 2008).

Among 9–13 year-old children, internalizing vs. externalizing behavior problems are each associated with different frontal and cingulate cortex activity (Moadab et al. 2010). This suggests that even in childhood, distinct structures may contribute to over- as opposed to under-regulation of frontolimbic networks. Taken together, these studies reveal that emotion regulation is an effortful endeavor, which can be measured biologically. However, biological studies often neglect to address the social context in which emotion dysregulation develops and is maintained. This is a significant limitation given consistent evidence that Biology × Environment interactions can promote lasting difficulties with self-control among vulnerable individuals (Caspi et al. 2002, 2003a, b; Rutter and Sroufe 2000).

Re-Conceptualizing Emotion Dysregulation and Frontolimbic Dysfunction: Social Baseline Theory

We hypothesize that emotion regulation is better understood as an individual *and* interpersonal process. This begins with early attachment and continues to later peer and romantic relationships. The developmental mechanisms we review are likely common to both typical and maladaptive outcomes. However, a social neuroscience perspective has not been applied broadly to the literature on BPD, even though

there are rich theoretical models that translate well to the clinical literature. Social baseline theory (SBT; Coan 2008, 2010), in particular, addresses interrelations across multiple systems, including social affiliation, emotion regulation, and behavioral dyscontrol. This provides a useful framework for understanding the precise domains of functioning that are impaired in BPD. Although research on attachment and BPD is not new (see e.g., Levy 2005; Lyons-Ruth 2008; Macfie 2009), studies to date have focused largely on patterns of attachment among those with BPD. In contrast, SBT outlines the neural mechanisms by which relationships regulate (or fail to regulate) biology and behavior across the life span.

Social baseline theory proposes that all people "are hardwired to assume close proximity to conspecifics, and to utilize social proximity as a baseline affect regulation strategy" (Coan 2008, 2010, p. 213). Among the many advantages of social proximity, there are three biological principals that make attachment across the lifetime an evolutionary necessity: risk distribution, load sharing, and economy of action (Coan 2010; Krebs et al. 1993). *Risk distribution* is how the species benefits from the probabilistic distribution of risk, not only through lower likelihood of falling prey, but also through the distribution of effort toward vigilance to predators, maintenance of thermal energy, and predation. *Load sharing* suggests that in addition to broader social support, it is necessary to identify a trusted companion who assists with important health- and safety-enhancing behaviors. These include resource acquisition, vigilance for environmental threats, caring for one another's needs, and nurturing of offspring. Finally, *economy of action* implies that all organisms opt to conserve energy by optimizing the ratio of resources expended to those acquired. Thus, attachment and interpersonal relationships serve to distribute the cost of difficult (i.e., metabolically expensive) life activities through co-regulation.

Emotion regulation, along with other PFC mediated self-regulatory tasks, is metabolically intensive (Gailliot and Baumeister 2007). Throughout life, co-regulation of emotions functions to conserve metabolic resources and increase survival. Indeed, it is well-established that early attachments promote emotion regulation skills and the ability to form healthy adult relationships (Butner et al. 2007; Coan 2008; Diamond and Fagundes 2008; Fonagy et al. 2000; Gillath et al. 2005; Lyons-Ruth 2008). From the SBT perspective, these early experiences of co-regulation (or lack thereof) provide the foundation for continued co-regulation into adulthood. Without this, resources deplete more rapidly, which further disrupts social affiliation, affect regulation, and skills that rely on the PFC, such as problem solving, attention allocation, cognition, and planning (Coan 2010; Fuster 2002).

The Development of Co-Regulation and Borderline Pathology

Borderline personality disorder is a complex phenotype and etiological trajectories leading to BPD are invariably characterized by equifinality and multifinality (Beauchaine et al. 2009). However, affective dysregulation is a core feature of BPD, with one study finding that 90% with the diagnosis meet this criterion (Zanarini et al. 2004). Moreover, affective instability is the diagnostic feature associated most strongly with treatment utilization among those with BPD (Bagge et al. 2005). For these reasons, trajectories leading to dysregulated emotion likely overlap with those that lead to BPD (Lieb et al. 2004). Social baseline theory couches emotion dysregulation within an interpersonal context. This is consistent with findings that the developing frontolimbic system is affected by environmental inputs, such as the availability of caregivers to skillfully co-regulate a child's emotional distress (e.g., Belsky and de Haan 2011; Roth and Sweatt 2011; see also Mead et al. 2010 for a review).

Current understanding of borderline personality development could be enriched by incorporating some of the key assumptions of SBT. *First*, the early attachment relationship serves as the initial source of co-regulation. Filial bonding typically occurs quickly and unconditionally during a period of rapid neural development. During this time, neural links are formed between the PFC and structures that underlie emotion and memory (e.g., amygdala, nucleus accumbens, hippocampus; Coan 2008; Hofer 2006). This may contribute to early variability in assumptions about the social world and the perceived likelihood of experiencing co-regulation during emotional situations.

Second, these and other neural structures likely support attachment and maintenance of friendship bonds in childhood and adolescence (Porges et al. 1996; Rockhill et al. 2009; Shields et al. 1994; Shipman and Zeman 2001; Snyder et al. 1997). Ideally, effective co-regulation will have occurred earlier in life and, consequently the neural structures implicated in self-control will have already begun to form and strengthen. Children with better self-control are more likely to be accepted by peers (Shields et al. 2008) and may be less likely to use problematic strategies for obtaining emotional support (Crick et al. 2005), which could further alienate potential sources of co-regulation (e.g., relational or physical aggression, self-injury, substance use).

Finally, frontolimbic circuits are also implicated in adult attachment formation, trust, affiliation, and attraction (Fertuck et al. 2006). By adulthood, people who have low expectations for co-regulation may be more likely to chronically deplete PFC resources by using independent self-regulation as the baseline strategy. Unfortunately, such

chronic self-reliance often leads to "regulatory failure" (Gailliot and Baumeister 2007), which can potentiate ineffective and/or impulsive emotion regulation strategies. Differences in frontolimbic circuits are found consistently in fMRI studies of BPD (Bohus et al. 2004; Domes et al. 2009).

In this review, we examine each of the three assumptions of SBT and how insufficient co-regulation across the life span may lead to behavioral and interpersonal difficulties, such as those seen in BPD. Social baseline theory integrates attachment theory with an emerging literature on neural depletion following independent self-regulation (Coan 2008). Most attachment researchers propose a developmental trajectory of increasing behavioral control such that, by adulthood, nearly all self-regulation is independent (e.g., Mikulincer and Shaver 2008). In contrast, SBT suggests that the process by which early attachment leads to adaptive adult outcomes is via healthy expectations of continued co-regulation in adulthood. The inability to identify, utilize, and maintain these social supports may contribute to emotional fatigue, dysregulated behaviors, and differences in frontolimbic circutry observed among adults with BPD (Bohus et al. 2004; Domes et al. 2009). From this perspective, borderline pathology can be understood not only as a disorder of emotion dysregulation but also one of insufficient co-regulation across the life span.

Biological Mechanisms of Early Attachment

Attachment figures serve as the initial source of social affect regulation and load sharing. Thus, these early relationships appear to have a lasting effect on attachment style and emotion regulation (Coan 2008; Diamond and Hicks 2005; Meany 200; Schore 1996). Animal research with rat pups and their mothers has found that maternal care affects DNA methylation, altering future cortisol regulation and temperament of the pups (Meany 2001). For humans, the caregiver-child relationship is the first experience whereby the child learns to influence the caregiver's emotions and behaviors. Similarly, the child experiences self-regulation of behavior and emotions through the caregiver's actions (Cole et al. 2004). Researchers find that compared with adults, children recruit greater regions of the PFC during independent affect regulation tasks (Levesque et al. 2004). This is likely due to immature emotion regulation skills and the considerable neural resources required for self-control in the absence of parental support. Thus, the attachment relationship appears to offset the extremely high cost of independent emotional and behavioral control.

Early attachment status also predicts emotion regulation abilities and attachment style in adulthood (Diamond and Hicks 2005; Diamond et al. 2006, Lyons-Ruth 2008). Thus, co-regulation not only conserves resources, but may also teach independent self-regulation and strategies for utilizing other individuals as regulatory resources; processes that are likely subserved by increased axonal connectivity within frontolimbic substrates (Posner and Rothbart 2007). Functional connectivity between the dorsomedial PFC, amygdala, and the hippocampus may also be one mechanism by which early relationships become an "internal working model" of attachment (Hofer 2006). In other words, the developing frontolimbic system is sensitive to social inputs and structurally encodes expectations of distress alleviation and security provision from attachment figures (Coan 2010). Across development, the amygdala tags emotional stimuli while the hippocampus consolidates the associated contextual cues into long-term memory. Through this process, the behavior of attachment figures becomes stored as neural representation. The amygdala is also sensitive to signs of threat and, via input to the hypothalamus, functions to regulate stress hormones and facilitate social soothing (Kemeny 2003; McEwan 2007).

Reciprocal projections between the PFC and the amygdala, hippocampus, and hypothalamus contribute to memory formation and conditioned learning, including the appraisal of emotional stimuli and activating the appropriate motivated behavior (Davidson and Irwin 1999; Rolls 2007). Through this process, conditioned responses to attachment figures are encoded within medial, orbital, and dorsolateral circuits of the PFC, serving as markers of threat or protection. These associations strengthen (i.e., become internal working models) through dopaminergically mediated experiences of security (Coan 2010). Similarly, oxytocinergic activity in the hypothalamus, nucleus accumbens, ventral tegmentum, and amygdala appear to influence attachment security (see Coan 2008). Researchers also find that increased endogenous opioid activity in the ACC underlies sensitivity to and greater distress from social rejection, which may be yet another mechanism by which attachment experiences are encoded within frontolimbic circuitry (Eisenberger et al. 2007; Way et al. 2009). According to SBT, the development and reinforcement of these circuits likely shape interpersonal relatedness across the life span.

Middle Childhood and Adolescence

Throughout childhood, the regulatory effects of the child-caregiver bond occur both in the presence of the attachment figure and through the mental representation of caregiver availability in response to threat. Under optimal conditions, the child and caregiver form a secure attachment. This leads to reasonable assumptions of co-regulation and a confident sense of "the self" in relation to key attachment figures. Developmental psychopathologists conceptualize this as an

"average expectable environment" (Cicchetti and Valentino 2006). Such environments include a range of conditions that promote normative developmental processes. Differences within this range serve as opportunities for individual development and promote variability in the phenotypic expression of genes. In some cases, however, there is a "failure" of the average expectable environment, as in cases of neglect or abuse. Under such circumstances, normal development is threatened.

A large number of adults with BPD report a history of neglect or abuse (e.g., Goldman et al. 1992; Soloff et al. 2002; Zanarini et al. 1989), although it is now clear that abuse is not a necessary antecedent to later borderline pathology (see Goodman et al. 2004). However, research on children with abuse histories can inform our understanding of BPD because such youth also manifest poor interpersonal relatedness and deficits of emotional and behavioral control (Shields et al. 1994).

Maltreated children are more likely than non-abused children to engage in antisocial, aggressive, withdrawn, and disruptive behaviors during play interactions, even when children are matched on key demographic variables (see Cicchetti et al. 1992; Shields et al. 1994). Moreover, children who display antisocial or aggressive behaviors are subsequently viewed as mean or attention seeking and tend to be less highly regarded by their peers (Asher and Coie 1990; Denham and Holt 1993; Salzinger et al. 1993). This can lead to rejection and enduring negative reputations within social groups (Shields et al. 1994). In turn, peer rejection produces functional changes in insular, ventrolateral PFC, ACC and ventral striatum activation among adolescents (Masten et al. 2009). Thus, problematic behaviors and peer affiliations may reinforce maladaptive assumptions about attachment and decrease the likelihood of receiving effective co-regulation through friendships. In addition to changing social expectations and behaviors, peer rejection may also produce lasting biological adaptations within these frontolimbic circuits.

By late adolescence, many youth with BPD can be diagnosed reliably (Miller et al. 2008). Moreover, mood dependent, impulsive behaviors (e.g., self-injury, substance use) often emerge during adolescence (Crowell et al. 2008). Recently, we have suggested that repetitive self-injurious behaviors in adolescence may represent one stage in a trajectory leading to adult BPD (Beauchaine et al. 2009; Crowell et al. 2009). From the perspective of SBT, self-injury likely occurs in the context of co-regulatory failure. This is consistent with findings that interpersonal stressors, such as conflict with parents, peer problems, or the end of a romantic relationship, often precipitate self-injurious behavior (Berman et al. 2006; Brown et al. 2002). Chronic interpersonal stress across both family and peer systems may ultimately be a strong predictor of later borderline pathology (Kobak et al. 2009). To our knowledge, however, there are no data that test this hypothesis sufficiently.

At each stage of development, the child is actively shaping her own trajectory through behavioral response patterns and via active attempts to resolve stage-salient tasks (Cicchetti and Cohen 2006). Thus, attachment formation is not merely a product of parental availability and support. Rather, child temperament and Temperament × Parenting interactions influence attachment patterns across the life span and shape the emerging adult personality (Bates et al. 1985; Rothbart et al. 2000). However, even though there is widespread acceptance of transactional/biosocial explanations of personality development, nearly every study of adolescent or adult BPD examines independent functioning, without any interaction with attachment figures (e.g., Buchheim et al. 2008; Silbersweig et al. 2007; Wingenfeld et al. 2009). Moreover, very few researchers have attempted to investigate individual differences in adult attachment using measures of neural activity.

Emerging Adulthood

A growing literature suggests, however, that adult attachment relationships are represented biologically and can be assessed using fMRI. In our work (Coan et al. 2006), we have examined neural correlates of adult co-regulation. Specifically, we have used fMRI to study women's brain activation in response to intermittent, mild electric shocks. Threat cues and shocks were delivered during three separate conditions: holding a husband's hand, no hand, or the hand of an anonymous male experimenter. The findings confirmed that social contact functions to regulate emotional responding during stress. Relative to the other two conditions, when women held their husband's hand they showed attenuated activation in the neural circuits that subserve emotional and behavioral threat responses. Moreover, marital quality predicted neural responses, such that women with healthier marriages showed lower threat-related activation in the right anterior insula, superior frontal gyrus, and hypothalamus while holding their husband's hand. In other words, women in happy relationships were able to more effectively "outsource" their emotion regulation to an attachment figure, thereby reducing their own metabolic demands (see Coan 2010).

This type of co-regulation can be thought of as a form of "on-line" social support (cf., Coan 2011). However, internal models of attachment are also crucial to successful adult functioning. For example, during an fMRI task that involved contemplating negative relationship scenarios, adults with insecure attachment styles showed greater activation in emotion-related brain regions when compared with securely attached adults (Gillath et al. 2005). Furthermore, there was an inverse relation between activation of

the anterior temporal pole (a paralimbic structure) and the orbital frontal cortex, such that those with the greatest attachment anxiety showed the least frontal activation and the greatest paralimbic activation. The authors hypothesize that insecurely attached individuals may be less able to modulate emotional processes via top-down control from frontal regions.

In theory, attachment history provides a working template of interpersonal behaviors, which can be accessed to regulate emotions at a lower metabolic cost, even when the attachment figure is not readily available. This extends to pair-bond formation, where attachment history likely influences present-day emotion regulatory activities, including the decision to cede some of the regulatory effort to the potential mate (Coan 2010). Among adults with an insecure attachment history, these processes may be disrupted.

Applications to Borderline Personality Disorder

Emotion related neural activity is a focus of BPD research. These studies find that when compared with typical controls, those with BPD have increased amygdala activation and decreased ACC activation in response to fear, emotional pictures, fearful faces, and abandonment scripts (Donegan et al. 2003; Herpertz et al. 2001; Minzenberg et al. 2007; Schmahl et al. 2003). During emotionally challenging tasks, such as the recall of unresolved life events or facing attachment threats alone, adults with BPD show increased activation of the amygdala, insula, and parahippocampal regions relative to controls. (Beblo et al. 2006; Buchheim et al. 2008; Schnell et al. 2007). In some of these studies, adults with BPD also show increased activation in cortical regions involved with regulatory efforts, including the ACC, medial PFC, and OFC compared with typical participants. Those with BPD also differ from controls in response to neutral stimuli, activating similar regions of the brain in response to both neutral and emotional stimuli (Schmahl et al. 2004; Schnell et al. 2007; Wingenfeld et al. 2009).

During tasks that tap behavioral control, especially within the context of negative emotion, adults with BPD show absent or decreased activation of the cingulate, ventromedial PFC, medial OFC, and subcortical reward regions, along with increasing amygdala activity (Kraus et al. 2010; Silbersweig et al. 2007; Völlm et al. 2007). Adults with BPD also recruit a larger number of brain regions during memory tasks compared with typical controls, suggesting that cognitive tasks are more taxing or effortful for those with the diagnosis (Mensebach et al. 2009). Thus, fMRI studies reveal broad disruption of frontolimbic circuitry during emotional, cognitive, and behavioral tasks. Importantly, activation within frontolimbic circuitry is intricately related to neurotransmitter and neuropeptide functioning (see Coan 2008; Eisenberger et al. 2007). Although beyond the scope of this review, both neurotransmitters and neuropeptides are involved in attachment formation, mood regulation, and behavioral control (see Crowell et al. 2009; Stanley and Siever 2010 for recent reviews). Serotonin, dopamine, endogenous opioids, oxytocin, and vasopressin play a central role in the development of BPD.

Finally, co-regulation in the partner relationship is an act of trust. Research on the development of trust emphasizes mutual interdependence, which includes both a cooperative intention and expectation (Loomis 1959). Mutual interdependence occurs when both partners have a shared goal, a desire to reach the goal, and awareness that collaboration is essential. Among adults with BPD, there is evidence that trust is impaired, both behaviorally and in terms of neural response patterns (King-Casas et al. 2008). Specifically, compared with typical controls, those with BPD are more likely to rupture trust during an economic investment game. This break down of trust occurs in two ways. First, those with BPD are less likely to repay investors in a manner that leads to profit for both players. Second, participants with BPD are less likely to engage in "coaxing behaviors" (i.e., viewing small investments as a sign of broken cooperation and repairing via large repayments).

In contrast, typical controls are more likely to coax the investor, which serves to repair trust and increase the overall amount of money earned for both participants (King-Casas et al. 2008). The authors report differences in anterior insula activity during this task. While typical controls had strong anterior insula activation when offering a small repayment (i.e., a norm violation), those with BPD did not show such activation when engaging in trust-rupturing behavior. The authors suggest that "the norms used in perception of social gestures are pathologically perturbed or missing altogether among individuals with BPD" (p. 806).

An alternative interpretation is that those with BPD are inexperienced at trust. This includes trusting that another's behavior is well-intended and also trusting that one's own behavior will be well received. Based on the findings of King-Casas and colleagues, control participants appear to experience distress when failing to repay investments whereas those with BPD do not. The lack of anterior insula activation may suggest that those with BPD do not experience conflict when cheating the other participant out of money, perhaps because they assume the partner is also attempting to cheat them. Although the authors did not assess motivations or cognitions, those with BPD appear to struggle with representing other people's motives accurately (Linehan 1993). They may therefore feel justified in taking as much money as possible, whenever it is available. In

other words, those with BPD behave as though the world is scary, awful, and untustworthy. In real-world situations this likely manifests as an inability to trust potential co-regulators. Linehan (1993) suggests that such doubt is a consequence of an invalidating family environment—one which inconsistently rejects, ignores, invalidates, or reinforces emotional responses. Implicit in this description of an "invalidating environment" is insufficient co-regulation by the attachment figure.

Social Baseline Theory and Borderline Personality Disorder

To summarize, SBT suggests that as a social species we are adapted biologically to seek close proximity to other people. An implication of this model is that social isolation *defies* the baseline assumption and is therefore intrinsically threatening and punishing. Unfortunately, for many adults with BPD isolation is not unusual, but is rather a chronic state promoting loneliness, self-injury, and completed suicide (Paris and Zweig-Frank 2001; Soloff and Fabio 2008). Strong social support is a known protective factor against suicide, whereas interpersonal loss is a consistent risk factor (Heikkinen et al. 1997; Kelly et al. 2000; Pagano et al. 2004). We suggest that social isolation in adulthood has its roots in early attachment, Temperament × Parenting interactions, and lifelong experiences of insufficient co-regulation. Over time these experiences become represented neurobiologically as deficits of frontolimbic circuitry, consistent with fMRI studies of adults with BPD.

A Developmental Progression

We have described a trajectory that begins with insufficient co-regulation in early attachment relationships. As with any dyadic process, co-regulation is likely shaped by interacting characteristics of both the caregiver and child. There are several factors that may contribute to poor co-regulation abilities by the caregiver. For example, limited social support, insufficient resources, psychological struggles, partner violence, or the caregiver's own attachment history might contribute to difficulties with soothing or regulating a child (Macfie 2009; Shipman and Zeman 2001). Although many children adapt successfully to environmental stressors, those who are vulnerable biologically may not. In the case of abuse or neglect, even a child with no biological vulnerabilities may experience lasting psychological and biological adaptations to stress, leading to behaviors that are maladaptive in other contexts (Mead et al. 2010).

There are several reviews that emphasize biological differences among individuals at risk for BPD or suicide (Beauchaine et al. 2009; Crowell et al. 2009; Mann 2003). Although not a focus of this review, biological differences in serotonergic or dopaminergic functioning appear to underlie early temperament (e.g., Sheese et al. 2007). Variability in infant temperament likely also affects parent's abilities to co-regulate infant distress. Specifically, children who have higher levels of trait impulsivity or negative affectivity (i.e., a "difficult temperament") may challenge their parent's efforts at behavioral and emotion regulation. Developmental psychopathologists have long observed that impulsive children are harder to parent (Mash and Johnston 1990). However, coercive parenting styles appear to contribute uniquely to the development of emotion dysregulation among impulsive youth (Beauchaine et al. 2007; Patterson et al. 1989; Patterson et al. 1984).

Within the SBT model, the inability of early attachment figures to provide consistent co-regulation may overwhelm developing frontolimbic circuits. This could contribute to borderline personality development through two social mechanisms. First, the failed development of successful co-regulation through early attachment disrupts learned self-regulation and associated frontolimbic connections. Second, insufficient co-regulation influences the development of internal working models of attachment. Each pathway could increase the likelihood that an adolescent will utilize maladaptive regulation strategies.

Healthy peer regulation provided early in the development of BPD could ease the burden of independent regulation and promote new skills. Unfortunately, chronically taxed frontolimbic circuitry heightens risk for self-regulation failure—leading to impulsive behaviors, disrupted social processing and affiliation, poor emotion regulation, and cognitive difficulties—which then further delay the development of frontolimbic circuitry. Thus, early biological adaptations to stress may potentiate later risk via Biology × Peer interactions. Social baseline theory provides a framework for understanding how insufficient co-regulation could chronically tax the biological *and* social structures that promote self-control.

Limitations and Implications

Biological mechanisms of development are complex. In this review we have focused on structural adaptations to early environments. However, functional changes in synaptic connectivity are the result of physiological and neurochemical processes that continue across the life span. Several biological mechanisms likely underlie the development of BPD, including genetic and epigenetic influences on neurotransmitter systems. Because research on borderline personality development is still limited, many hypotheses put forward in this review remain to be tested. However, SBT is consistent with both interpersonal and dialectical

theories of borderline personality development (Fonagy 2000; Linehan 1993).

Frontolimbic dysfunction is common to many psychiatric conditions and SBT may also apply to other disorders characterized by dysregulated affect, behavior, interpersonal relatedness and/or cognitions. While not unique to borderline personality development, SBT illustrates several mechanisms that may account for disrupted frontolimbic circuitry in BPD. Moreover, BPD is one of the few disorders where interpersonal distress is reportedly a key precipitant of dysregulated behavior (APA 2000). This suggests that insufficient co-regulation may be experienced more intensely by individuals with BPD or for those who are at risk for the disorder.

When BPD is viewed from the developmental psychopathology perspective, intervention should not be postponed until an individual meets diagnostic criteria. Rather, family and school-focused prevention can begin at an earlier age, possibly reducing risk for any number of multifinal outcomes. An important implication of SBT is that attachment can be viewed as a lifelong process. Thus, relationships at any stage of development have the potential to alter the presentation of BPD and promote healing. For this to occur, emotion regulation must be understood as an interdependent process for all people, not just those with BPD.

Clinical Implications

Social baseline theory suggests that interpersonal dependency is necessary for healthy emotional functioning. However, many existing treatments operate at the individual level and attempt to alter cognitive processes. Such interventions ask that a person engage in resource-intensive self-regulation, often without adequate social support. Thus, even clients who appear highly competent in the therapy room (i.e., in the presence of effective co-regulation), may falter in real-world contexts. Because cognitive strategies rely heavily on independent self-control, they are unlikely to be effective until social networks are engaged. Preventative interventions will also be more effective if parent, teacher, and peer support are enlisted.

Dialectical behavior therapy (DBT) is an effective treatment for BPD and adolescent self-injurious behaviors (Linehan 1993; Miller et al. 2007). Social baseline theory may help explain some of the unique factors that contribute to DBT's success. First, DBT includes 24-h telephone consultation. This relationship with the therapist may function as a form of co-regulation until the client is skillful enough to re-engage social supports. Second, DBT includes skills classes in addition to individual therapy. This provides another form of social support (through both skills coaches and other group members). Indeed, skills are an important ingredient of treatment and reduce affective instability, identity problems, and negative interpersonal relatedness, even when initial distress and engagement in individual therapy are controlled statistically (Stepp et al. 2008). Based on SBT, it may be most effective to focus on enhancing distress tolerance and interpersonal effectiveness skills early in therapy. Skills that tax frontal lobe resources (i.e., mindfulness and emotion regulation) should be taught later. Offering intervention to a parent or romantic partner may also improve co-regulation and offer lasting benefit.

Research Implications

There are no studies that test co-regulation theories directly among those with BPD or those at high risk for the diagnosis. Future research could employ imaging and basic science methodology to augment existing research on emotion regulation. Specifically, this work should expand beyond independent self-regulation tasks to explore social affect regulation and its development. Moreover, studies addressing the neurobiology of BPD should attempt to integrate research on frontolimbic activation with emerging research on neurotransmitter and neuropeptide systems. Prospective studies of infants, children, and adolescents could also examine neurobiological mechanisms of attachment and how early attachment experiences shape borderline personality development. To date, the majority of BPD research has approached the diagnosis from an individual-illness perspective. This fails to account for competencies that many individuals with BPD possess when supported adequately. Future research should attempt to incorporate an understanding of the interpersonal context in which personality dysfunction emerges and is maintained, as it is the same context that will ultimately provide the structure and support for emotional health.

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References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author. Text Revision.

American Psychiatric Association. (2010). DSM-5 Development. Retrieved April 14, 2010, from http://www.dsm5.org/Pages/Default.aspx

Asher, S. R., & Coie, J. D. (Eds.). (1990). *Peer rejection in childhood.* New York: Cambridge University Press

Bagge, C. L., Stepp, S. D., & Trull, T. J. (2005). Borderline personailty disorder features and utilization of treatment over two years. *Journal of Personality Disorders, 19*, 420–439.

Banks, S. J., Eddy, K. T., Angstadt, M., Nathan, P. J., & Phan, K. L. (2007). Amygdala-frontal connectivity during emotion regulation. *Social, Cognitive, and Affective Neuroscience, 2*, 303–312.

Bates, J. E., Maslin, C. A., & Frankel, K. A. (1985). Attachment security, mother-child interaction and temperament as predictors of behavior-problem ratings at age three years. *Monographs of the Socieity for Research in Child Development, 50*, 167–193.

Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. K. (2007). Polyvagal theory and developmental psychopathology: emotion dysregulation and conduct problems from preschool to adolescence. *Biological Psychology, 74*, 174–184.

Beauchaine, T. P., Neuhaus, E., Brenner, S., & Gatzke-Kopp, L. (2008). Ten good reasons to consider biological processes in prevention and intervention research. *Development and Psychopathology, 20*, 745–774.

Beauchaine, T. P., Klein, D. N., Crowell, S. E., Derbidge, C., & Gatzke-Kopp, L. M. (2009). Multifinality in the development of personality disorders: a Biology × Sex × Environment interaction model of antisocial and borderline traits. *Development and Psychopathology, 21*, 735–770.

Beblo, T., Driessen, M., Mertens, M., Wingenfeld, K., Martina, P., Rullkoetter, N., et al. (2006). Functional MRI correlates of the recall of unresolved life events in borderline personality disorder. *Psychological Medicine, 36*, 845–856.

Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition, 50*, 7–15.

Belsky, J., & de Hann, M. (2011). Parenting and children's brain development: The end of the beginning. *Journal of Child Psychology and Psychiatry, 52*, 409–429.

Bender, D. S., Dolan, R. T., Skodol, A. E., Sanislow, C. A., Dyck, I. R., McGlashan, T. H., et al. (2001). Treatment utilization by patients with personality disorders. *American Journal of Psychiatry, 158*, 295–302.

Berman, A. L., Jobes, D. A., & Silverman, M. M. (2006). *Adolescent suicide: Assessment and intervention* (2nd ed.). Washington, DC: American Psychological Association.

Black, D. W., Blum, N., Pfohl, B., & Hale, N. (2004). Suicidal behavior in borderline personality disorder: prevalence, risk factors, prediction, and prevention. *Journal of Personality Disorders, 18*, 226–239.

Bohus, M., Schmahl, C., & Lieb, K. (2004). New developments int eh neurobiology of borderline personality disorder. *Current Psychiatry Reports, 6*, 43–50.

BPDRF. (2006). Borderline personality disorder research foundation: Phenotype conference. Retrieved August 12, 2007 from http://www.borderlineresearch.org/publications/phenotype_conf.html.

Brown, M. Z., Comtois, K. A., & Linehan, M. M. (2002). Reasons for suicide attempts and nonsuicidal self-injury in women with borderline personality disorder. *Journal of Abnormal Psychology, 111*, 198–202.

Buchheim, A., Erk, S., George, C., Kächele, H., Kircher, R., Martius, P., et al. (2008). Neural correlates of attachment trauma in borderline personality disorder: a functional magnetic resonance imaging study. *Psychiatry Research: Neuroimaging, 163*, 223–235.

Butner, J., Diamond, L., & Hicks, A. (2007). Attachment style and two forms of affect coregulation between romantic partners. *Personal Relationships, 14*, 431–455.

Caspi, A. (2000). The child is the father of the man: personality continuities from childhood to adulthood. *Journal of Personality and Social Psychology, 78*, 158–172.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science, 297*, 851–854.

Caspi, A., Harrington, H., Milne, B., Amell, J. W., Theodore, R. F., & Moffitt, T. E. (2003a). Children's behavioral styles at age 3 are linked to their adult personailty traits at age 26. *Journal of Personality, 71*, 495–514.

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003b). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science, 301*, 386–389.

Charney, D., Barlow, D. H., Botteron, K., Cohen, J. D., Goldman, D., Gur, R. E., et al. (2002). Neuroscience research agenda to guide development of a pathophysiologically based classifications system. In D. J. Kuper, M. B. First, & D. A. Regier (Eds.), *A research agenda for DSM-V.* Washington, DC: American Psychiatric Association.

Cicchetti, D., & Cohen, D. (2006). *Developmental psychopathology (vol. 3)*. Hoboken: Wiley.

Cicchetti, D., & Valentino, K. (2006). An ecological-transactional perspective on child maltreatment: failure of the average expectable environment and its influence on child development. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology, vol. 3* (2nd ed.). Hoboken: Wiley.

Cicchetti, D., Rogosch, F. A., Lynch, M., & Holt, K. D. (1992). Resilience in maltreated children: processes leading to adaptive outcome. *Development and Psychopathology, 5*, 629–647.

Coan, J. A. (2008). Toward a neuroscience of attachment. *Handbook of attachment* (Vol. 2, pp. 241–265).

Coan, J. A. (2010). Adult attachment and the brain. *Journal of Social and Personal Relationships, 27*, 210–217.

Coan, J. A. (2011). The social regulation of emotion. In J. Decety & J. T. Cacioppo (Eds.), *Oxford handbook of social neuroscience.* New York, Oxford University Press.

Coan, J. A., Schaefer, H. S., & Davidson, R. J. (2006). Lending a hand: social regulation of the neural response to threat. *Psychological Science, 17*, 1032–1039.

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: methodological challenges and directions for child development research. *Child Development, 75*, 317–333.

Comtois, K. A., Russo, J., Snowden, M., Srebnik, D., Ries, R., & Roy-Byrne, P. (2003). Factors associated with high use of public mental health services by persons with borderline personality disorder. *Psychiatric Services, 54*, 1149–1154.

Crick, N. R., Murray-Close, D., & Woods, K. (2005). Borderline personality features in childhood: a short-term longitudinal study. *Development and Psychopathology, 17*, 1051–1070.

Crowell, S. E., Beauchaine, T. P., & Lenzenweger, M. F. (2008). The development of borderline personality and self-injurious behavior. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology* (pp. 510–539). Hoboken: Wiley.

Crowell, S. E., Beauchaine, T. P., & Linehan, M. M. (2009). A biosocial developmental model of borderline personality: elaborating and extending Linehan's theory. *Psychological Bulletin, 135*, 495–510.

Damasio, A. R. (1994). *Descartes' error: Emotion, reason, and the human brain*. New York: Avon Books, Inc.

Davidson, R. J., & Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in Cognitive Sciences, 3*, 11–21.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation–a possible prelude to violence. *Science, 289*, 591–594.

Denham, S. A., & Holt, R. W. (1993). Preschoolers' likability as a cause or consequence of their social behavior. *Developmental Psychology, 29*, 271–275.

Diamond, L., & Fagundes, C. (2008). Developmental perspectives on links between attachment and affect regulation over the lifespan. *Advances in Child Development and Behavior, 36*, 83–134.

Diamond, L. M., & Hicks, A. M. (2005). Attachment style, current relationship security, and negative emotions: the mediating role of physiological regulation. *Journal of Social and Personal Relationships, 22*, 499–518.

Diamond, L. M., Hicks, A. M., & Otter-Henderson, K. (2006). Physiological evidence for repressive coping among avoidantly attached adults. *Journal of Social and Personal Relationships, 23*, 205–229.

Domes, G., Schulze, L., & Herpertz, S. C. (2009). Emotion recognition in borderline personality disorder—a review of the literature. *Journal of Personality Disorders, 23*, 6–19.

Donegan, N. H., Sanislow, C. A., & Blumberg, H. P. (2003). Amygdala hyperreactivity in borderline personality disorder: implications for emotional dysregulation. *Biological Psychiatry, 54*, 1284–1293.

Eisenberger, N. I., Taylor, S. E., Gable, S. L., Hilmert, C. J., & Lieberman, M. D. (2007). Neural pathways link social support to attenuated neuroendocrine stress responses. *NeuroImage, 35*, 1601–1612.

Ekman, P. (1992). An argument for basic emotions. *Cognition and Emotion, 6*, 169–200.

Ekman, P., & Friesen, W. V. (1976). Measuring facial movement. *Environmental Psychology and Nonverbal Behavior, 1*, 56–75.

Fertuck, E. A., Lenzenweger, M. F., Clarkin, J. F., Hoermann, S., & Stanley, B. (2006). Executive neurocognition, memory systems, and borderline personality disorder. *Clinical Psychology Review, 26*, 346–375.

Fonagy, P. (2000). Attachment and borderline personality disorder. *Journal of the American Psychoanalytic Association, 48*, 1129–1146.

Fonagy, P., Target, M., & Gergely, G. (2000). Attachment and borderline personality disorder: a theory and some evidence. *Psychiatric Clinics of North America, 23*, 103–122.

Funder, D. C. (2001). Three trends in current research on person perception: positivity, realism, and sophistication. In J. A. Hall & F. J. Bernieri (Eds.), *Interpersonal sensitvity: Theory and measurement*. Mahwah: Lawrence Erlbaum Associates, Inc.

Fuster, J. M. (2002). Frontal lobe and cognitive development. *Journal of Neurocytology, 31*, 373–385.

Gailliot, M. T., & Baumeister, R. F. (2007). The physiology of willpower: linking blood glucose to self-control. *Personality and Social Psychology Review, 11*, 303–327.

Gillath, O., Bunge, S. A., Shaver, P. R., Wendelken, C., & Mikulincer, M. (2005). Attachment-style differences in the ability to suppress negative thoughts: exploring the neural correlates. *Neuroimage, 28*, 835–847.

Goldin, P. R., McRae, K., Ramel, W., & Gross, J. J. (2008). The neural bases of emotion regulation: reappraisal and suppression of negative emotion. *Biological Psychiatry, 63*, 577–586.

Goldman, S., D'Angelo, E., DeMaso, D., & Mezzacappa, E. (1992). Physical and sexual abuse histories among children with borderline personality disorder. *American Journal of Psychiatry, 149*, 1723–1726.

Goldsmith, H. H., Pollak, S. D., & Davidson, R. J. (2008). Developmental neuroscience perspectives on emotion regulation. *Child Development Perspectives, 2*, 132–140.

Goodman, M., New, A., & Siever, L. (2004). Trauma, genes, and the neurobiology of personality disorders. *Annals New York Academy of Sciences, 1032*, 104–116.

Grant, B. F., Chou, P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., et al. (2008). Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry, 69*, 533–545.

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment, 26*, 41–54.

Gross, J. J. (1998a). Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224–237.

Gross, J. J. (1998b). The emerging field of emotion regulation: an integrative review. *Review of General Psychology, 2*, 271–299.

Heikkinen, M., Henriksson, M. M., Isometsa, E., Marttunen, M. J., Aro, H. M., & Lonnqvist, J. K. (1997). Recent life events and suicide in personality disorders. *Journal of Nervous and Mental Disease, 185*, 373–381.

Herpertz, S. C., Dietrich, T. M., Wenning, B., Krings, T., Erberich, S. G., Willmes, K., et al. (2001). Evidence of abnormal amygdala functioning in borderline personality disorder: a functional MRI study. *Biological Psychiatry, 50*, 292–298.

Hofer, M. (2006). Psychobiological roots of early attachment. *Current Directions in Psychological Science, 15*, 84–88.

Hyman, S. E. (2007). Can neuroscience be integrated into the DSM-V? *Nature Reviews Neuroscience, 8*, 725–732.

Kaufman, J., & Charney, D. (2001). Effects of early stress on brain structure and function: implications for understanding the relationship between child maltreatment and depression. *Development and Psychopathology, 13*, 451–471.

Kelly, T. M., Soloff, P. H., Lynch, K. G., Haas, G. L., & Mann, J. J. (2000). Recent life changes, social adjustment and suicide attempts in patients with major depression and borderline personality disorder. *Journal of Personality Disorders, 14*, 316–326.

Kemeny, M. E. (2003). The psychobiology of stress. *Current Directions in Psychological Science, 12*, 124–129.

King-Casas, B., Sharp, C., Lomax-Bream, L., Lohrenz, T., Fonagy, P., & Montague, P. R. (2008). The reupture and repair of cooperation in borderline personality disorder. *Science, 321*, 806–810.

Kobak, R., Zajzc, K., & Smith, C. (2009). Adolescent attachment and trajectories of hostile-impulsive behavior: implications for the development of personality disorders. *Development and Psychopathology, 21*, 839–851.

Kraus, A., Valerius, G., Seifritz, E., Ruf, M., Bremmer, J. D., & Bohus, M. (2010). Script-driven imagery of self-inurious behavior in patients with borderline personality disorder: a pilot fMRI study. *Acta Psychiatrica Scandinavica, 121*, 41–51.

Krebs, J. R., Davies, N. B., & Parr, J. (1993). *An introduction to behavioural ecology*. London: Blackwell Science Ltd.

Kuo, J. R., & Linehan, M. M. (2009). Disentangling emotion processes in borderline personality disorder: physiological and self-reported assessment of biological vulnerability, baseline intensity, and reactivity to emotionally evocative stimuli. *Journal of Abnormal Psychology, 118*, 531–544.

Levesque, J., Joanette, Y., Mensour, B., Beaudoin, G., Leroux, J., Bourgouin, P., et al. (2004). Neural basis of emotional self-regulation in childhood. *Neuroscience, 129*, 361–369.

Levy, K. N. (2005). The implications of attachment theory and research for understanding borderline personality disorder. *Development and Psychopathology, 17*, 959–986.

Lieb, K., Zanarini, M. C., Schmahl, C., Linehan, M. M., & Bohus, M. (2004). Borderline personality disorder. *The Lancet, 364*, 453–461.

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York: Guilford.

Loomis, J. L. (1959). Communication, the development of trust, and cooperative behavior. *Human Relations, 12*, 305–315.

Lyons-Ruth, K. (2008). Contributions of the mother-infant relationship to dissociative, borderline, and conduct symptoms in young adulthood. *Infant Mental Health Journal, 29*, 203–218.

Macfie, J. (2009). Development in children and adolescents whose mothers have borderline personality disorder. *Child Development Perspectives, 3*, 66–71.

Mann, J. J. (2003). Neurobiology of suicidal behaviour. *Nature Reviews Neuroscience, 4*, 819–828.

Mash, E. J., & Johnston, C. (1990). Determinants of parenting stress: illustrations from families of hyperactive children and families of physically abused children. *Journal of Clinical Child and Adolescent Psychology, 19*, 313–328.

Masten, C. L., Eisenberger, N. I., Borofsky, L., Pfeifer, J. H., McNealy, K., Mazziotta, J. C., et al. (2009). Neural correlates of social exclusion during adolescence: understanding the distress of peer rejection. *Social, Cognitive, and Affective Neuroscience, 4*, 143–157.

McEwan, B. S. (2007). Physiology and neurobioogy of stress and adaptation: central role of the brain. *Physiological Reviews, 87*, 873–904.

Mead, H. K., Beauchaine, T. P., & Shannon, K. E. (2010). Neurobiological adaptations to violence across development. *Development and Psychopathology, 22*, 1–22.

Meany, M. J. (2001). Maternal care, gene expression, and the transmission of individual differences in stress reactivity across generations. *Annual Review of Neuroscience, 24*, 1161–1192.

Mensebach, C., Beblo, T., Driessen, M., Wingenfeld, K., Mertens, M., Rullkoetter, N., et al. (2009). Neural correlates of episodic and semantic memory retrieval in borderline personality disorder: An fMRI study. *Psychiatry Research, 171*, 94–105.

Mikulincer, M., & Shaver, P. (2008). Adult attachment and affect regulation. In J. Cassidy & P. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (2nd ed., pp. 503–531). New York: Guilford.

Miller, A. L., Rathus, J. H., & Linehan, M. M. (2007). *Dialectical behavior therapy with suicidal adolescents*. New York: Guilford.

Miller, A. L., Muehlenkamp, J. J., & Jacobson, C. M. (2008). Fact or fiction: diagnosing borderline personality disorder in adolescents. *Clinical Psychology Review, 28*, 969–981.

Minzenberg, M. J., Fan, J., New, A. S., Tang, C. Y., & Siever, L. J. (2007). Fronto-limbic dysfunction in response to facial emotion in borderline personality disorder: an event-related fMRI study. *Psychiatry Research: Neuroimaging, 155*, 231–243.

Moadab, I., Gilbert, T., Dishion, T. J., & Tucker, D. M. (2010). Frontolimbic activity in a frustrating task: covariation between patterns of coping and individual differences in externalizing and internalizing symptoms. *Developmental Psychopathololgy, 22*, 391–404.

Pagano, M., Skodol, A. E., Stout, R. L., Shea, M. T., Yen, S., Grilo, C. M., et al. (2004). Stressful life events as predictors of functioning: findings from the Collaborative Longitudinal Personality Disorders Study. *Acta Psychiactra Scandinavica, 110*, 421–429.

Paris, J., & Zweig-Frank, H. (2001). A 27-year follow-up of patients with borderline personality disorder. *Comprehensive Psychiatry, 42*, 482–487.

Patterson, G. R., Dishion, T. J., & Bank, L. (1984). Family interaction: a process model of deviancy training. *Aggressive Behavior, 10*, 253–267.

Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. *American Psychologist, 44*, 329–335.

Porges, S. W., Doussard-Roosevelt, J. A., Portales, A. L., & Greenspan, S. I. (1996). Infant regulation of the vagal brake predicts child behavior problems: a psychobiological model of social behavior. *Developmental Psychobiology, 29*, 697–712.

Posner, M. I., & Rothbart, M. K. (2007). Research on attention networks as a model for the integration of psychological science. *Annual Review of Psychology, 58*, 1–23.

Rockhill, C. M., Vander Stoep, A., McCauley, E., & Katon, W. J. (2009). Social competence and social support as mediators beween comorbid depressive and conduct problems and functional outcomes in middle school children. *Journal of Adolescence, 32*, 535–553.

Rolls, E. T. (2007). Emotion elicited by primary reinforcers and following stimulus-reinforcement association learning. In J. A. Coan & J. J. B. Allen (Eds.), *The handbook of emotion elicitation and assessment* (pp. 137–157). New Yrok: Oxford University Press.

Roth, T. L., & Sweatt, J. D. (2011). Epigenetic mechanisms and environmental shaping of the brain during sensitive periods of development. *Journal of Child Psychology and Psychiatry, 52*, 398–408.

Rothbart, M. K., Ahadi, S. A., & Evans, D. E. (2000). Temperament and personality: origins and outcomes. *Journal of Personality and Social Psychology, 78*, 122–135.

Rutter, M., & Sroufe, L. A. (2000). Developmental psychopathology: concepts and challenges. *Development and Psychopathology, 12*, 265–296.

Salzinger, S., Feldman, R. S., Hammer, M., & Rosario, M. (1993). The effects of physical abuse on children's social relationships. *Child Development, 64*, 169–187.

Sanchez-Navarro, J., Martinez-Selva, J. M., & Roman, F. (2005). Emotional response in patients with frontal brain damage: effects of affective valence and information content. *Behavioral Neuroscience, 119*, 87–97.

Schmahl, C. G., Elzinga, B. M., Vermetten, E., & Sanislow, C. A. (2003). Neural correlates of memories of abandonment in women with and without borderline personality disorder. *Biological Psychiatry, 54*, 142–151.

Schmahl, C. G., Elzinga, B. M., Ebner, U. W., Simms, T., Sanislow, C., McGlashan, T. H., et al. (2004). Psychophysiological reactivity to traumatic and abandonment scripts in borderline personality and posttraumatic stress disorders: a preliminary report. *Psychiatry Research, 126*, 33–42.

Schnell, K., Dietrich, T., Schnitker, R., Daumann, J., & Herpertz, S. C. (2007). Processing of autobiographical memory retrieval cues in borderline personality disorder. *Journal of Affective Disorders, 97*, 253–259.

Schore, A. N. (1996). The experience-dependent maturation of a regulatory system in the orbital prefrontal cortex and the origin of developmental psychopathology. *Development and Psychopathology, 8*, 58–87.

Sheese, B. E., Voelker, P. M., Rothbart, M. K., & Posner, M. I. (2007). Parenting quality interacts with genetic variation in dopamine receptor D4 to influence temperament in early childhood. *Development and Psychopathology, 19*, 1039–1046.

Shields, A., Cicchetti, D., & Ryan, R. (1994). The development of emotional and behavioral self-regulation and social competence among maltreated school-age children. *Development and Psychopathology, 6*, 57–75.

Shields, A., Cicchetti, D., & Ryan, R. (2008). The development of emotional and behavioral self-regulation and social competence among maltreated school-age children. *Development and Psychopathology, 6*, 57–75.

Shipman, K. L., & Zeman, J. (2001). Socialization of children's emotion regulation in mother-child dyads: a developmental

psychopathology perspective. *Development and Psychopathology, 13*, 317–336.

Silbersweig, D., Clarkin, J. F., Goldstein, M., Kernberg, O. F., Tuescher, O., Levy, K. N., et al. (2007). Failure of frontolimbic inhibitory function in the context of negative emotion in borderline personality disorder. *American Journal of Psychiatry, 164*, 1832–1841.

Snyder, J., Schrepferman, L., & St. Peter, C. (1997). Origins of antisocial behavior: negative reinforcement and affect dysregulation of behavior as socialization mechanisms in family interaction. *Behavior Modification, 21*, 187–215.

Soloff, P. H., & Fabio, A. (2008). Prospective predictors of suicide attempts in borderline personality disorder at one, two, and two-to-five year follow-up. *Journal of Personality Disorders, 22*, 123–134.

Soloff, P. H., Lynch, K. G., & Kelly, T. M. (2002). Childhood abuse as a risk factor for suicidal behavior in borderline personality disorder. *Journal of Personality Disorders, 16*, 201–214.

Sroufe, L. A. (1997). *Emotional development: The organization of emotional life in the early years*. New York: Cambridge University Press.

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development, 55*, 17–29.

Stanley, B., & Siever, L. J. (2010). The interpersonal dimension of borderline personality disorder: toward a neuropeptide model. *American Journal of Psychiatry, 167*, 24–39.

Stepp, S. D., Epler, A. J., Jahng, S., & Trull, T. J. (2008). The effect of dialectical behavior therapy skills use on borderline personality disorder features. *Journal of Personality Disorders, 22*, 549–563.

Taylor, S. F., & Liberzon, I. (2007). Neural correlates of emotion regulation in psychopathology. *Trends in Cognitive Sciences, 11*, 413–418.

Torgersen, S., Kringlen, E., & Cramer, V. (2001). The prevalence of personality disorders in a community sample. *Archives of General Psychiatry, 58*, 590–596.

Trull, T. J., Stepp, S. D., & Durrett, C. A. (2003). Research on borderline personality disorder: an update. *Current Opinion in Psychiatry, 16*, 77–82.

Völlm, B., Richardson, P., McKie, S., Elliott, R., Dolan, M., & Deakin, B. (2007). Neuronal correlates of reward and loss in Cluster B personality disorders: a functional magnetic resonance imaging study. *Psychiatry Research: Neuroimaging, 156*, 151–167.

Walden, T. A., & Smith, M. C. (1997). Emotion regulation. *Motivation and Emotion, 21*, 7–25.

Way, B. M., Taylor, S. E., & Eisenberger, N. I. (2009). Variation in the mu-opioid receptor gene (OPRM 1) is associated with dispositional and neural sensitivity to social rejection. *Proceedings of the National Academy of Sciences, 106*, 15079–15084.

Wingenfeld, K., Rullkoetter, N., Mensebach, C., Beblo, T., Mertens, M., Kreisel, S., et al. (2009). Neural correlates of the individual emotional Stroop in borderline personality disorder. *Psychoneuroendocrinology, 34*, 2009.

Zanarini, M. C., Gunderson, J. G., Marino, M. F., Schwartz, E. O., & Frankenburg, F. R. (1989). Chilhood experiences of borderline patients. *Comprehensive Psychiatry, 30*, 18–25.

Zanarini, M. C., Frankenburg, F. R., Hennen, J., Reich, D. B., & Silk, K. R. (2004). Axis I comorbidity in patients with borderline personality disorder: 6-Year follow-up and prediction of time to remission. *American Journal of Psychiatry, 161*, 2108–2114.