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Developmental precipitants of borderline personality disorder

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Abstract

Traditional theories regarding the etiology of borderline personality disorder have focused on poor attachment figures and/or traumatic experience. The present review posits an additional pathogenic course for this disorder. Specifically, the proposed mechanism involves a basic disruption of the neural hardware that supports the formation and maintenance of unconscious emotional memory, hardware essential for the formation of early attachments. It is further theorized that this early disruption has ongoing effects on both behavioral and concomitant neural development. Within this model, adolescence is described as a period of intense change that serves as the tipping point for the onset of borderline personality disorder.

"All you need is love," sang the Beatles. More appropriate, however, may have been something along the lines of—"all you need is a reciprocal exchange of correctly expressed, perceived, and internalized attachment-related affect." Somehow, that is just not as catchy.

Human beings are fundamentally prosocial animals who rely on interactions with conspecifics for feedback and guidance on a vast array of life events ranging from simple daily activities like basic nourishment, to more complex life-altering decisions like whether to accept a job overseas. These essential personal interactions are uniquely disrupted in individuals suffering from borderline personality disorder (BPD).

BPD is a personality disorder in which patients exhibit highly unstable affect, mood, behavior, object relations, and self-image. Traditional theories regarding the etiology of this disorder have focused on environmental contributors, such as poor rearing conditions or traumatic experience. The present review suggests an additional pathogenic course for this disorder. Specifically, the proposed mechanism involves a basic disruption of the neural hardware that supports the creation of unconscious emotional memory, which is the foundation of early attachment. We further suggest that, akin to tossing a stone in a pond, the effects of this early disruption have a rippling effect in both behavioral and concomitant neural development. Based on this notion, we develop a model that describes adolescence as a period of intense change where these early perturbations begin to evidence themselves in terms of psychopathology.

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Etiology of BPD

There is a rich clinical and theoretical literature on the affective and interpersonal components of BPD; however, there is little empirical work in these areas. Although clinical research suggests that BPD is more common among females, recent epidemiological work (Torgerson, Kringlen, & Cramer, 2001) suggests that BPD may be equally common in males. This discrepancy in prevalence estimates may be explained by several factors: (a) women may be more likely to seek treatment

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for BPD, (b) symptoms of BPD might cause more impairment in females relative to males, or (c) gender bias in the categorical description of this disorder may favor females.

Given the prevalence of females in BPD clinical settings, female samples have informed much of the theoretical and empirical investigation in this area. Most etiological theories suggest that the disorder develops from some combination of biological or temperamental predisposition and a toxic family environment. Kernberg (1996) argues that BPD results from the combined influence of genetic, constitutional, psychodynamic, and psychosocial forces. Although there is some evidence that BPD and related traits tend to run in families (Baron, Gruen, Asnis, & Lord, 1985; Nigg & Goldsmith, 1994) it is unclear to what extent shared pathology is due to genetic versus environmental influences.

Disordered personality may be considered an unusual and maladaptive expression of normal personality traits, thus, some scholars argue that it may be inappropriate to consider personality disorders distinct, independent categories. Indeed, Depue and Lenzenweger (2005) suggest that personality disorders may be better understood as fluid, multidimensional entities, produced by a confluence of traits on neurobehavioral dimensions that exist within the space of normal personality. Within this model, personality traits are best defined as the product of the interaction of neurobehavioral systems. These systems may be part of an evolutionary process designed to produce flexible behavior that is critical and adaptive for reproductive success and survival. Thus, neurobehavioral systems respond to interpersonal events, producing a complex emotional response to stimuli. Personality disturbance, therefore, appears when these emotional responses are maladaptive, essentially inappropriately intense or absent. For BPD, this may mean that the emotional lability and frantic fears of abandonment may be manifestations of a complex interplay of anxious and affiliative (increased negative emotion, decreased positive emotion, and diminished constraint) behaviors that may emerge (a) to less specific interpersonal stimuli or (b) with more intensity.

Problems in interpersonal relationships may be a function of the borderline's frequent cognitive distortions. Borderlines appear to have a cognitive style, in which they display, "A tendency toward global perceptions with a loss of attention to detail, distortion of the meaning of an event, patterns of confusion, and spotty amnesias" (Kroll, 1988). Clinicians have noted that much of the cognitive distortion of borderline individuals is a function of a frantic fear of abandonment or rejection. Given that a large percentage of individuals with BPD have a concurrent depression or anxiety disorder (Gunderson & Phillips, 1991; Zanarini, Frankenburg, Dubo, et al., 1998), it is difficult to disentangle the relative contribution of Axis I pathology to such distortion. However, it is of note that the quality of the cognitions thought to underlie the borderline's distortions may be particularly interpersonal in nature. Dysphoric cognitions uniquely associated with BPD compared to other personality disorders reflect themes of paranoia and being tortured or abused, among others (Zanarini, Frankenburg, DeLuca, et al., 1998).

Recent attempts to elucidate these cognitive distortions have involved investigation of information-processing mechanisms in individuals with BPD. Neuropsychological data suggests that there is no *specific* cognitive impairment unique to BPD. The role that affective memory plays in these difficulties is as yet unclear. Two studies of neuropsychological functioning in BPD have revealed difficulties in memory and visual perception (O'Leary, Brouwers, Gardner, & Cowdry, 1991; Swirsky–Sacchetti, et al., 1993). Problems in memory functioning appeared to be positively related to the complexity of the information. Difficulty in visual perception appears to reveal a tendency to rely on global scenes at the expense of specific details.

A natural extension of this work would be to include affectively salient stimuli. Sprock, Rader, Kendall, and Voder (2000) explored this issue by including a modified Stroop task utilizing anger- and sadness-related words (presumably because these words should have particular significance to a BPD sample). Further, they employed a story-recall paradigm to assess memory for complex verbal material and

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the influence of emotional themes on recall. Finally, the researchers presented participants with a verbal-recall task with interference. Two lists of eight words were presented followed by an interference involving a control condition of serial sevens, or an emotional interference that required subjects to tell a story about Thematic Apperception Test Card #13 MF, which often evokes stories of rape or murder. There was no difference in performance on any of the tasks, even those involving emotional stimuli, between BPD, depressed, and normal subjects.

A potential criticism of this work is that test stimuli are not specifically interpersonally salient, and thus will not activate BPD-specific schemas. Thus, general words associated with depression might not be uniquely relevant to BPD individuals. There is a strong empirical basis for this hypothesis. Cognitive biases for specific emotional states have been noted in several mental disorders. Mood-congruent memory biases have been demonstrated in individuals with depressive disorders (Mineka & Nugent, 1995). Further, it appears that the emotional and/or cognitive deficits associated with each disorder are often circumscribed to disorder-specific features. For example, individuals with posttraumatic stress disorder (PTSD) display enhanced sensitivity only to emotional lexical stimuli that are related to the traumatic experience (Foa, Feske, Murdock, Kozak, & McCarthy, 1991; McNally, Kaspi, Riemann, & Zeitlin, 1990). These findings suggest that trauma-related psychopathology may involve effects specific to one emotional system rather than a general state of heightened emotionality.

To investigate this issue in BPD, Korfine and Hooley (2000) utilized a directed forgetting paradigm. Researchers instructed a group of BPD patients from a day treatment program, a group of BPD individuals from the community, and a group of normal controls to either remember or forget three types of words: borderline, neutral, and positive. Consistent with hypotheses, both borderline groups showed increased recall of borderline words in the forget condition. In short, BPD individuals remembered more of the borderline words that they were instructed to forget but only when the words were particularly relevant to BPD (e.g., abandon, reject, emptiness). The authors speculated that participants with BPD were unable to inhibit rehearsal of BPD words when they were presented with the instructions to forget. This disinhibition is unique to stimuli that are interpersonally salient, and does not occur in the presence of positive or neutral words.

Indeed, it appears that the addition of an affective component that is uniquely salient to BPD individuals may affect information processing. Such an interaction between cognition and affect would suggest that borderlines might have difficulty on other types of tasks of emotion recognition and emotion management. Levine, Marziali, and Hood (1997) found that borderlines differed from normal subjects on four measures of processing emotional information, suggesting that borderlines have more limited capacities for processing emotional information related to self and others, recognizing facial expressions of emotion, and integrating conflicting or ambiguous emotional states.

In addition, tasks that require effortful processing of stimuli may present a unique challenge to individuals with BPD. Lenzenweger, Clarkin, Fertuck, and Kernberg (2004) examined the role of attention and control in frontally mediated tasks (the Wisconsin Card Sort Test [WCST]) in relation to emotional dyscontrol. Whereas participants with BPD were not impaired relative to normal, healthy controls on tasks that employed working memory or sustained attention, BPD participants did exhibit impairment on the WCST. Similar to the conclusions of Korfine (1998; Korfine & Hooley, 2000), the authors suggest that diminished control may interfere with tasks that require effortful processing.

A few etiological explanations have focused on the interaction between temperament and specific environmental attributes. Linehan (1993) focuses on the emotional dysregulation that is pathognomonic to BPD. She argues that a biological predisposition (perhaps limbic in origin) interacts with an invalidating and interpersonally alienating family environment resulting in a disruption of the emotion regulation system. This theory views

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child sexual abuse (CSA) as a model by which to understand the etiology of BPD rather than a prerequisite of the disorder. According to this perspective, an invalidating environment in general is required for the disorder to develop, and CSA represents a prototypical example of extreme invalidation. This chronic invalidation of emotional experiences may result in a disruption of the emotion regulation system. As a result, the borderline patient develops extreme emotional vulnerability and deficits in the ability to regulate emotions. As such, the borderline will experience high sensitivity to emotional stimuli, increased intensity of emotional states, and a slow return to emotional baseline relative to nonborderline patients. Experience with highly emotional events may lead to an overactivation of the limbic system, which may render the individual more sensitive to emotional material. The result may be analogous to "kindling," where emotional sensitivity becomes heightened to a pathological level resulting in misinterpretation of interpersonal stimuli (Hartman & Burgess, 1993).

Benjamin (1996) asserts that borderline pathology is best observed within a social and interpersonal context. The borderline individual's misunderstanding of seemingly innocuous events, her rapid mood swings, and her frequent cognitive distortions make maintaining a relationship with a borderline a particularly daunting task. Using interpersonal behavior as a proxy by which to illuminate borderline pathology, Benjamin (1992) offers an explanation of the intrapsychic functioning of the borderline. In general, she argues that the interpersonal experiences in early childhood affect the patterns of adult relationships. Specifically, Benjamin (1992) postulates that the instability in relationships, affect, and self-image in BPD are an extension of childhood environments in which the child was exposed to traumatic experiences, simultaneously idealized and devalued, and encouraged to be and rewarded for being dependent upon the family.

According to object relations theory, the borderline individual experiences disturbances in interpersonal functioning that reflect underlying disturbances in "object–world" representations. These representations are the merging of cognitions relating to self and others and the affects attached to those representations. If these representations are flawed or biased by differential memory processing and retrieval, this may account for a great deal of the interpersonal problems experienced by BPD individuals (Kernberg, 1976, 1996).

Attachment

The first systematic examination of attachment behaviors involved the study of imprinting behavior by Lorenz (1935). Lorenz noted what he believed to be an innate behavioral program, in which the newborn bird formed a rather rigid behavioral bond with a moving object. Exposure to this object must occur during a critical period immediately following birth. Unfortunately, one caveat of this phenomenon is that the object with which this bond is formed may or may not be useful as a mother. There are numerous reports of investigators imprinting birds on everything from human beings to garden rakes. It is clear that without the interference of science this process is effective in securing recognition between baby and mother bird. Although human infants do not exhibit imprinting per se, they do exhibit a wide range of complex attachment behaviors, the nature of which has been empirically delineated.

As conceptualized by the British psychoanalyst John Bowlby, attachment behaviors are those that have the predictable outcome of maintaining proximity/contact with the mother. Further, Bowlby proposed that the presence of these behaviors was evidence of a biologically based behavioral system that serves to promote the viability of the infant by the formation of a bond with its mother (Bowlby, 1969). This bond, according to Bowlby, occurs within the first year of life, and promotes the survival of the infant by ensuring the proximity of a protecting adult. The way in which maternal behavior and infant physiology interact is no doubt reciprocal and largely dependent on the infant's stage of development. In a broader sense, mammalian-infant attachment, unlike imprinting in birds, is not a singular, instantaneous event. Infant attachment repre-

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sents a constellation of various behavioral–physiological interactions that begin prenatally and continue to evolve postnatally (Insel, 1997). Bowlby (1969) described this assemblage of attachment-related processes as "internal working models." Internal working models describe a process thought to begin during infancy, when, based on experiences, the child forms a cognitive model of an attachment figure (Kirsh & Cassidy, 1997). Particularly important aspects of these models, according to Kirsh and Cassidy, concern the availability and responsiveness of the attachment figure. Although Bowlby viewed internal working models as remaining open to new input, he believed that, because they tend to operate outside the realm of consciousness, over time they become increasingly resistant to change. These models have also suggested that attachment guides children's behavior, feelings, and processing of social information (Kirsh & Cassidy, 1997). An interactive relationship between attachment and information processing is based on the notion that such a relationship would be adaptive by providing psychological protection to the individual as well as by helping to preserve the relationship with the attachment figure. For example, according to Main (1996) an individual whose bids for care have been rejected or belittled may develop a nonconscious strategy for reducing attention to, or memory for, attachment-related stimuli. Such a cognitive strategy would help ensure that the attachment system is activated only when absolutely necessary, thereby reducing the risk of alienating an attachment figure who did not wish to respond. Although attachment theorists have long proposed that representations of attachment would influence a child's information processing related to social relationships, empirical examination of this proposition has been rare.

Although neurocognitive models of human attachment have been rare, there has been significant focus on affiliation, which is thought to be one of the primary underpinnings of attachment. Particularly at birth, but in actuality throughout the life span, human beings rely on one another for survival. Given this, it is reasonable to think of a relatively innate and general neurobehavioral system of behaviors that enables affiliation (see Depue & Lenzenweger, 2005, for a review). As defined by Depue and Lenzenweger, affiliation "reflects enjoying and valuing close interpersonal bonds and being warm and affectionate." They go on to further describe affiliation as reflecting reward processed, and specifically, two important phases of goal acquisition: appetitive and consumatory. The appetitive processes serve to bring individuals into closer proximity with their desired affiliative objects. In humans, this may be expressed as approach behaviors such as desire, wanting. When close proximity to a rewarding goal is achieved, appetitive processes give way to consumatory ones. Consumatory processes elicit intense feelings of pleasure, gratification, and in terms of physiology produce restful and satiety. Thus, where appetitive approach processes bring an individual into contact with incentive stimuli, consumatory processes bring behavior to a gratifying conclusion (Hilliard, Domjan, Nguyen, & Cusato, 1998, as cited in Depue & Lenzenweger, 2005). Depue and Lenzenweger posit that the subjective experience of warmth and affection reflects this capacity to experience consumatory reward. It is also thought that this general capacity is thought to apply to a number of affiliative stimuli, and through this it serves as an essential part of additional psychobiological processes that facilitate the development and maintenance of longer term affective bonds, the sort that form the basis of attachment. Because of the fact that these ideas about affiliation and attachment are based in large part on the reward system, it is intuitive to look to the dopaminergic system for neurochemical evidence of this process.

Distal (i.e., visually based) affiliative cues are known to provoke dopamine (DA)-facilitated incentive–reward motivation, desire, wanting, and approach. As affiliative stimuli are reached, more proximal (i.e., tactile) affiliative stimuli strongly activate μ-opioid release. This cascade promotes an intense state of reward, warmth, affection, and physiological quiescence, and brings approach behavior to a gratifying conclusion. Throughout this entire sequence of goal acquisition, the contextual cues associated with approach to the goal, and the cues specifically related to

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the goal, are associated with the experience of reward. DA and μ-opiates play a central role in strengthening the association between contextual cues and reward. Thus, these two neuromodulators are essential for establishing the stimulus reward pairings that are the basis of our preferences and memories for specific stimulus and contextually based affiliations (Depue & Lenzenweger, 2005).

An additional neurochemical player in the game of affiliation and attachment is the hormone oxytocin. Oxytocin is produced in the hypothalamus, and released into the bloodstream through axon terminals in the pituitary. Part of a family of hormones, oxytocin can be traced phylogenetically to invertebrates. Ancestral substances have been implicated in various forms of nonmammalian reproductive behaviors. Oxytocin is unique, however, as it is found exclusively in mammals. It is not surprising that oxytocin has been implicated in prototypically mammalian functions. For instance, it has an important role in milk ejaculation during nursing and uterine contraction during labor. Thomas Insel has speculated that the role of oxytocin in the uterus and mammary tissue necessary for providing the physiological support of offspring may be parallel to that in the brain, where oxytocin subserves the motivational changes essential for maternal care (Insel, 1997). There has also been a great deal of work that has underscored the importance of oxytocin in complex forms of social memory such as offspring recognition, and pair bonding (see Bielsky & Young, 2004, for a review). It is thought that social memory is highly reliant upon the expression of oxytocin receptors in the nucleus accumbens (a portion of the caudate nucleus well known for its role in reward and learning). Further, the expression of oxtytocin receptors in this brain region facilitates brain DA release (Insel, 2003). Finally, oxytocin's effects on maternal bonding can be disrupted via blockade of μ-opiod receptors, underscoring the relation between oxytocin and opioid peptides. In both monkeys and humans, dulling the effect of μ-opiates in the reward centers of the brain with drugs has the effect of inducing individuals to seek social contact (Kendrick, 2004). Taken together, these findings suggest a neurochemical recipe for both immediate and long-term social bonds. The DA and opioid system sets up reward contingencies for achieving close proximity to an affiliative object, which may be any number of people or things. The approach to this object if driven by dopaminergic activity, and the satisfaction felt after achieving proximity, is mediate by the μ-opiates. Over time, specific affiliations may form. The memory for these specific affiliative objects (e.g., the smell of one's offspring) is regulated by the effects of oxytocin on the DA and μ-opiate systems.

Human beings are fundamentally social mammals, and social mammals regulate each other's neurophysiology and alter the internal structure of each other's nervous systems through the synchronous exchange of affect. For these interactions to exert long-lasting influences on behavior, it is necessary that they exist in memory. It is possible, then, that the memory of the attachment relationship is an enduring neural structure that exerts a significant influence on emotional behavior related to attachment behavior. The internalization in memory of affective information over time has consequences of utmost importance for the successful development and future function of the developing organism. In an infant or child, the gradual internalization in memory of the affectively laden interchange with his/her surroundings may organize and determine the structure and function of neurobehavioral systems, and the psychology of the child's mind, which are fundamentally intertwined (Amini et al., 1996). Further, the relationship between affect and memory has been described as a reciprocal one, in which the character and development of affect is as much influenced by memory processes as these processes are influenced by affect. Important to understanding how deficits in this system may contribute to the emergence of BPD is a more thorough understanding of memory development.

Memory Development

Memory processes are frequently described as being either explicit or implicit in nature

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(Sherry & Schacter 1987; Squire, Knowlton, & Musen, 1993). Simply, the explicit memory (EM) and implicit memory (IM) systems appear to be fundamentally different neurophysiological processes that rely on different sets of neural structures and have different physiological properties. Damage to temporal lobe structures impairs EM while leaving some forms of IM unaffected (Eslinger & Damasio, 1985), whereas damage to the basal ganglia impairs some forms of IM while leaving EM capacities largely intact (Squire et al., 1993). EM and IM tasks also result in different patterns of neural activity as detected by positron emission tomography and by EEG. In addition, EM and IM seem to have a different pharmacology, as evidenced by differential responses to anxiolitics and alcohol, both of which impair EM, but not IM (see Amini et al., 1996, for a review).

Most relevant to the present discourse, however, is that EM and IM show a different course of development over the life cycle. The developmental courses of IM and EM, not surprisingly, seem to parallel the development of the neurological structures thought to underlie the respective systems. Infant monkeys exhibit poor performance on tasks that depend on the operation of EM (Bachevalier & Mishkin, 1984).

In marked contrast, some forms of implicit learning and memory appear to be behaviorally functional quite early in life. Implicit or procedural memory has been described as being both functionally and structurally more archaic. For the most part, IMs can be regarded as the mnemonic component of learned action patterns. Simple organisms can learn patterns of action without any detailed episodic recall, again, like human infants. Sherry and Schacter (1987) have reported that, in terms of its storage strategy, procedural memory is the opposite of EM. Whereas EM preserves the specifics of events, IM preserves the general principles for action and ignores the specifics of the situation. Thus, learning a procedure involves setting parameters and following general rules. Detailed EM would interfere with this process (Donald, 1991). Donald provided an excellent example of this theory:

> . . . in learning to catch a ball one must learn the principle of tracking a moving object, no matter what the speed of the object, the starting point, or one's initial posture at the time it is thrown. It would be cumbersome to remember the exact speed, starting point and position of each successful practice catch; a new throw is unlikely to match any specific counterpart in practice. (p. 150)

This perspective has received support from research on nonhuman primates. Infant monkeys have shown habit or skill-based learning as early as 3 months of age (Bachevalier, 1990). In addition, there is clear evidence that human infants, despite the absence of EM function, are able to acquire *operational* long-term memories. These "habits" or "procedures" of which infants and young children are capable can be thought of as products of IM (Schacter & Moscovitch, 1984).

IM most frequently describes skills and "automatic" operations that are not stored with respect to times or places. The human organism is capable of demonstrating long-lasting changes in behavior in response to information and experiences that have no conscious representation. IM has been described as a system that preserves information that is not available for conscious recollection or awareness; however, this information creates enduring and observable changes in behavior (Milner, Corkin, & Teuber, 1968). In addition, evidence suggests that the IM system is capable of functioning early in the neurodevelopmental life of nonhuman primates (Bachevalier, 1990). Conjugate reinforcement paradigms have been used in human infants to demonstrate that infants as young as 3 months can be conditioned to kick their foot to move a mobile (Rovee–Collier & Hayne, 1987). Amini et al. (1996) review a great deal of evidence supporting the hypothesis that human infants are equipped with a functional memory system at birth, a system that is most adequately suited for implicit learning. Furthermore, it is conceivable that IM represents an inherent property of the mechanism used by the brain to store information, specifically, Hebbian synapses. This kind of memory, which may be paramount in the function of affect and attachment, may be phylogenetically older than the

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consciousness, EM, with which we are most familiar.

It has been established that information can be analyzed at a level including emotional/affective meaning without reaching awareness (Bruner & Postman, 1947). This and similar findings have lent support to the contention that affective information is processed (at least in part) in the IM system. As mentioned above, one of the ways in which IM operates is by extracting the rules that appear to govern particular covariations between stimuli. It has been shown that if subjects are presented with affective stimuli, the nature of which reflects the presence of an underlying rule that governs or predicts a covariation, subjects will learn the rule and proceed to apply it without their awareness (Hill, Lewicki, Czyzewska, & Schuller, 1990).

In this view, from the first day of life, mother and infant are participating in a vital exchange of signals that will have crucial implications for neurodevelopment, and hence, for the nature and capacities of the adult that infant will become. From their earliest encounter, the mother is participating in the regulation of the homeostatic state and the neurodevelopment of the infant with whom she is engaging. All mammals are perpetually dependent upon the synchronous exchange of affect with others for stability; early life, however, represents an important period during which input heavily influences and shapes the organism. At this time in life, implicit memories of the experience of the attachment relationship are laid down, well before the capacity for EM of events and concepts is present. The underlying patterns and regularities in the attachment relationship are thus detected, extracted, encoded, and stored. In this manner the growing infant acquires knowledge regarding what relationships are like, how they are conducted, and according to which "rules" behavior is regulated. However, because the operative memory system is implicit, the knowledge gained is in the form of generalizations and rules extracted from experience, and it later operates to influence behavior in an unthinking, reflexive manner. In a process that may be somewhat analogous to the learning of motor skills, people proceed in later life to enact attachments in accordance with the rules or prototypes they have extracted based on their prior experience. If their experience contained aberrancies, they will likely proceed to extract aberrant rules and generalizations, and they may enact them later without being able to have conscious knowledge of why they do so. Because of the interplay between the phenomenon of attachment and memory, the prior experience of inadequate attachment figures may result in an inner structure that is incapable of optimal self-regulation. In addition, because of the preference for familiar patterns that seems operative in attachment (most vividly illustrated in the primitive bond of imprinting), early exposure to an inadequate attachment figure may also predispose an individual to recognize and engage preferentially with a particular brand on inadequacy in relationships.

The IM system is (a) operational early in life, perhaps at birth; (b) is capable of extracting and storing prototypes and complex rules that govern covariations from repeated exposure to examples; and (c) appears to be involved in at least some forms of unconscious processing of affective information. Once learned implicitly, rules may exert a self-perpetuating bias for interpreting later experiences in a light consistent with past experience, whether later experience is objectively consistent with past experience or not.

Affective Development

Charles Darwin was among the earliest people to theorize about different types of affect in an integrated way (Darwin, 1872). Darwin saw affect as a behavioral function that originated and persisted in human beings for the same reason that any aspect of function originated and persists in any species: because of its ability to advance survival and reproductive fitness. In this way he saw affect as an intrinsic part of the human neurobehavioral repertoire. In addition, Darwin viewed affect as originating in our phylogenetic past, part of which we share with other animals. Affect clearly has powerful experiential aspects; therefore, it would be an oversimplification to explain affect as little more than subjective internal experience; rather, it is a complex

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physiological process with its roots in evolutionary history. As a dynamic process stemming from a neurological system that has been conserved over a considerable evolutionary time span, affect must serve functions that enhance survival and fitness. The functional role of affect, therefore, is required to be multifaceted and highly complex.

Emotional development is the process by which children learn to recognize, identify, and communicate affective states. Extreme emotional experiences are thought to influence an individual's interpretation and reaction to affective information. This effect of experience suggests that emotion and memory are intimately related. Further, this relationship may involve neurophysiological functioning that is affected by both genetics and environmental events. Cicchetti and Tucker (1994) argue that brain function and development are governed primarily by one's active strivings for self-organization. As a result, portions of brain development that involve self-organization may be particularly sensitive to environmental influences at certain life stages. The implication inherent in this argument is that neural development may be uniquely goal oriented. Depending upon one's experiences, neural development may proceed to incorporate specific pathways intended to facilitate behavior that is uniquely adaptive.

In the toddler years, the infant enters a new phase of development, what some have called the separation/individuation phase of development (Mahler, 1975). These years are marked by the full maturation of the perceptual and motor systems. The child also makes great strides in the development of cognition, memory (especially representational memory), and language. With increased perceptual and motor skills, the child becomes able to turn their attention away from the attachment relationship toward an active interest in the external environment. The child experiences various states of elation and considerable pleasure in mastery by using this increased autonomy to effectively negotiate the new world and discover new abilities (Mahler, 1975).

The toddler years are also characterized by great strides in cognitive development. The child develops the capacity for genuine mental representations, symbolic representations as opposed to more rudimentary schemas (Greenspan & Lourie, 1981). The capacity for symbolic representation sets the stage for the integration and differentiation of the representational world and the development of an organized representation for the psychological sense of self (Kohut, 1971).

During this phase, the heretofore relatively autonomous lines of affective and representational development become integrated. From this point in development and thereafter, affective states become associated with the inner experience of self and object representations. The emergent complex constellations of affective states and internal representations of experiences are primarily affective in nature and only secondarily ideational. Affect experience undergoes a change in function. Affect now functions not only as a response to external stimulation or as a communication to the caregiver but also as a signal for the ongoing changes within the representational world. Certain constellations of internal self-representations, object representations, or both may become activated or deactivated in particular situations. The changes in affective experiences correspond to shifts in internal representations as much as to shifts in external stimulation. In this sense, affective states become partially separated from external stimulation and interactions with others with whom they were originally associated. However, total memory capacity during this phase of development is unstable, so the emerging organization of representations is vulnerable to fragmentation in response to the contingencies associated with this developmental period (Brown, Mounts, Lamborn, & Steinberg, 1993); all of this changes with the onset of adolescence.

What is strikingly clear throughout every stage of affective development is the reliance of the individual on a tightly interwoven synergy between cognitive and emotional processes, there are individuals (LeDoux, 1994) who have argued that these two processes are nearly inextricable. Many species are critically dependent upon the synchronous exchange of affect with one another for survival; early life, however, represents an important pe-

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riod during which input heavily influences and shapes the organism. Among the functions that affect serves then, is that of providing an innate language that possesses seemingly "hard wired" predispositions alongside mechanisms that enable rapid, perhaps even single-exposure, lessons. This type of learning provides the scaffolding for split-second communication between social animals (LeDoux, 1994). Because the expression and interpretation of affective information does not exclusively depend on explicit information or learning, it is a communication and learning process that begins to operate immediately after birth.

Adolescent Development

Philosopher Jean Jacques Rousseau wrote: "We are born twice over; the first time for existence, the second time for life; once as human beings, and later as men or women." In his view, adolescence was a recapitulation of infancy; a time of intense and rapid change where many of the early lessons are transformed to suit adult life.

There is an ongoing debate over whether or not adolescence is characterized by a distinct stage of affective development. The assumption commonly made is that adolescence is a period of marked emotional variability and intensity. However, there have been surprisingly few studies on affect in adolescence. There is evidence that adolescents (compared with children and adults) do not manifest significantly greater variation or liability in the emotional states (Greene & Larson, 1991). Overall, there is no compelling evidence in support of the view of the so-called "storm and stress hypothesis" of normal adolescence. Although the transition from childhood to adolescence may be characterized by an intensification of certain affective states, adolescent development progresses in the direction of greater consistency and stability of affective states (Brown et al., 1993). Brown speculates that adolescent development brings forth a variety of speech patterns that include an emotive language. Thus, locating what is characteristic of adolescent affect may be found less in the type and structure of affect and more in the way it is expressed.

More characteristic of adolescent affect is the way in which it is transformed by cognitive maturation. Adolescence marks the transition from concrete operational thinking to formal operational thinking (Piaget, 1954). With respect to affect, affect is translocated from the self to self-in-relationship, a domain where the complex unfolding of affective states in both the self and other can be mutually recognized. The adolescent has the capacity to discern future feelings and to make subtle distinctions between fine nuances of emotion. Moreover, affective states become integrated with formal thought operations. For this transition to take place all of the requisite building blocks must be sturdy and available for assembly. It is as if adolescence is the performance of an orchestra, whose sections have been practicing independently for some time, and it is finally time for the group performance. If the conductor taps the music stand, raised the baton, and the horns are out of tune, or absent all together, the resultant sound will suffer. Simply put, the intense transformation of adolescence relies on the integrity of the developmental processes (both behavioral and neurobiological) that have preceded it. It is not surprising that adolescence is also a time when the chances of nonadaptive change significantly increase. Because of the nature of human development, specifically its tendency to build on itself, it is possible for a relatively uneventful developmental process to unmask a previously hidden diathesis. To use another analogy, not being able to steer a car is not a problem prior to the age at which one is expected to be able to drive. If one is not able to learn to steer, however, the entire process of learning to drive a car is likely to collapse. As a result of trying to learn to drive, one now has knowledge of a previously silent "steering disorder." Quite simply, adolescent development requires the acquisition of increasing complex cognitive, social, and emotional process that rely heavily on those that precede them, and it is conceivable that the expectations associated with adolescent development may in and of themselves serve to uncover and perhaps even exacerbate previously unseen diatheses.

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Germane to the investigation of the neurophysiological mechanisms that may underlie an individual's ability to successfully integrate cognitive, emotional and social processes is the neurodevelopmental trajectory of the functional connectivity between medial temporal lobe structures and frontal cortices. LeDoux (1994) has stressed the importance of the amygdala in fear detection and conditioning describing it as "a neural system that evolved to detect danger and produce rapid protective responses without conscious participation." The central nucleus of the amygdala has been described as essential for the expression of autonomic and somatic fear responses elicited by both learned and unlearned threats. These responses are controlled through efferent connections from the central amygdala to brainstem nuclei (Rogan & LeDoux, 1996). The amygdala has also been characterized as a higher order "convergence zone" for the social homeostatic and survival-related meanings of complex stimuli (Damasio, 1994). Taken together, these lines of evidence describe the amygdala as a structure that has evolved to help the human animal recognize and learn the emotional meaning of stimuli in their environment, and produce appropriate behavioral responses.

The production of appropriate behavior may rely heavily on the functionality of the frontal cortex. The frontal cortex has been shown to contribute to a wide range of human behavior; it has been implicated in behavioral inhibition, decision making, and abstract thinking. One striking difference in the development of the prefrontal cortex relative to other cortical areas is the continuation of synaptic pruning into young adulthood. This decrease in synaptic density during adolescence coincides with the emergence of newly entwined cognitive and emotional phenomena. The secondary process that is likely to be taking place during this time is the fortification of synaptic connections that will remain into adulthood. There has been further speculation that this "use it or loose it" process may represent the behavioral, and ultimately, the physiological, suppressing of immature behaviors that have become obsolete due the novel demands of adulthood (Casey, Giedd, & Thomas, 2000). One can imagine that a response to a particular event in the environment will be potentiated by repeated exposure and subsequent strengthening of the relation between that event and the generation of the appropriate response. The delayed maturation of this brain region allows the individual to adapt to the particular demands of their unique environment. Many researchers have documented that while there are age-related decreases in gray matter in the prefrontal cortex, the overall cortical volume does not change significantly. It is not surprising that the cortical volume remains stable because of simultaneous increases in white matter volume that may be equally important in terms of functionality. The greater volume of frontal white matter observed during adolescence is likely the result of greater axonal myelination. It has been well established that myelination has a direct impact on the speed and efficiency of neural processing. At the level of the neuron, increased myelination leads to increased action potential propagation speed and reduced signal attenuation. At a macroscopic level this type of maturation facilitates synchrony and coordination, both regionally and across the whole brain.

One specific frontal region within which increases in myelination have been observed is the anterior cingulate, an area known for its central role in the mediation of emotional, attentional, motivational, social, and cognitive behaviors (Vogt, Finch, & Olson, 1992). A significant positive relationship between age and total anterior cingulate volume (which has been attributed to increases in white matter) has been well documented (Casey et al., 1997). It is thought that this relationship may reflect improved cortical–cortical and cortical–subcortical coordination. The observed projections from both cortical and subcortical regions to the cingulate in adult subjects is known to contribute to the coordination and regulation of cognitive and emotional processes. A critical question with regard to human development has been the exact developmental course of these projections. Activity of the dorsal cingulate has been shown to play a crucial role in autonomic control and the conscious interpretation of somatic state. Further, maturation of

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the dorsal cingulate has been correlated with self-control and behavioral inhibition. Finally, the anterior cingulate is believed to play a specific executive role in the integration of autonomic responses with behavioral effort (Critchley, Mathias, & Dolan, 2001). This maturation may set the stage for the integration of emotion and cognition, which is fundamental to the formation of social attachments.

Relevant to the establishment of reciprocal influence between the amygdala and the prefrontal cortex is the time course of this connectivity. Medial temporal lobe structures are functionally mature very early in life, while the human frontal cortex does not reach full functional maturity until after puberty. Therefore, it is plausible that early in development, many aspects of behavior may be regulated by medial temporal lobe structures. Further, it is conceivable that during later development, frontal regions begin to exert a more powerful influence, so that what were once largely survival-based "unconscious" behaviors become increasingly entwined with more "conscious" cognitive and social processes. To date, however, few studies have explored the developmental trajectory of this functional connectivity. It has been established that during adolescence there is a substantial increase in the density and myelination of projections between medial temporal regions and frontal cortices (for reviews of the specific circuits, see Benes, Vincent, Molloy, & Khan, 1996; Cummings, 1995). Investigations using nonhuman primates have suggested that selective aspects of normal frontal development may be reliant on medial temporal lobe integrity (Bertolino et al., 1997). Additional studies of nonhuman primates support the idea that early damage to the amygdala has a deleterious effect on later social and emotional learning and behavior, while lesions of the amygdala in adulthood do not produce the same types of behavioral impairments (Bachevalier, 1991). It is not clear, though, how these findings extend to human development. It is possible, however that the functionality of early developing brain regions (i.e., temporal lobe structures) may serve to guide, at least in part, later developing regions (i.e., frontal cortices). Taken together, the studies above suggest a model of functional connectivity that in early life begins with medial temporal influence of frontal cortices, and later is driven by frontal regulation of medial temporal lobe structures.

Implications

To illustrate the infant–parent bond, consider the analogy that it takes two hands to clap. Although a lack of sound indicates no clapping, it is hard to say for sure which hand is not cooperating. Traditional models of attachment disruption focus on a disordered or inadequate attachment figure (see Agrawal, Gunderson, Holmes, & Lyons–Ruth, 2004, for a review); again this is simply one side of the possible equation. Modern attachment theorists have concluded that sociability is a natural consequence of a beneficial infant/parent attachment and, further, that the quality of being securely attached is preserved from infancy to later childhood. (Note that the use of "secure" here is not meant to imply a category of attachment, but rather the quality of attachment. There has been considerable empirical evidence that the categorization of attachment relationships into "types" may be misleading. More parsimonious models have suggested that variation in patterns of attachment is largely continuous rather than categorical. See Fraley & Spieker, 2003, for a review of these arguments.) These inferences have generated considerable debate because of the possibility that the temperamental qualities of infants might influence early anxious behavior, as well as later social interactions with their peers, independent of their attachment to their parents (Kagan, 1996). Although the current literature is replete with reports of how poor early attachment predicts adult psychopathology, there has been a paucity of reports that have gone beyond that basic relationship to more closely examine how and why. We propose that there may be a subtype of BPD where the observed disturbances in socioemotional cognition are the result of a deficit on the infant side of the parent/infant dyad. Simply, we suggest that neurophysiological-based deficits in the infant's implicit affective memory system may disrupt the earliest of attachment relationships. This deficit is then magnified

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by the development that takes place during adolescence.

Neuropsychological research of BPD patients suggests that a hallmark of the disorder is general dysfunction of the frontal and limbic circuitry (see Tebartz van Elst, 2003, for a review). Anatomical investigations of patients with BPD have found relative reductions in gray matter volume in both the limbic and frontal systems of the brain. The most common anatomical finding among patients with BPD is reduced amygdalar and hippocampal volume (Brambilla et al., 2004; Driessen et al., 2000; Schmahl, Vermetten, Elzinga, & Bremner, 2003; Tebartz van Elst, 2003). Functional studies of these structures have revealed metabolic differences associated with BPD. Specifically, the amygdala has been shown to be hypermetabolic in BPD patients (Herpertz et al., 2001).

One might consider these anatomical differences from a developmental perspective. It is possible that there is a subtype of individuals for whom it is difficult to form implicit affective memories from birth. We propose this is a result of early dysfunction of the amygdala/hippocampal complex. Starting in early infancy, these individuals would possess unstable affect, poor implicit affective memory, and overt biological markers, such as abnormal hippocampal and/or amygdala functionality. Further, it is suggested that a hyperactive amygdala in infancy produces a highly anxious, affectively unstable infant, while an underactive hippocampus, in combination with a preexisting deficient amygdala, underlies the disturbances in implicit affective memory.

As a result of deficits in their implicit affective memory systems, these individuals are theorized to be unable to benefit from their attachments to their primary caregiver(s). There has been much debate as to the nature of the hippocampal differences observed in BPD patients. Specifically, it has been suggested that these differences are the result of the stress or trauma associated with the disorder. This reasoning has also been applied to similar volumetric differences observed among individuals with PTSD (for reviews, see Hull, 2002; Sala et al., 2004). There is, however, compelling empirical evidence that hippocampal differences may exist prior to the onset of psychopathology. In a twin study disorder severity in PTSD patients who were exposed to trauma was negatively correlated with the hippocampal volume of both the patients and the patients' trauma-unexposed identical co-twin. Furthermore, severe PTSD twin pairs (both the trauma-exposed and -unexposed members) had significantly smaller hippocampi than non-PTSD pairs. We suggest that it is, in fact, the very early disruption of limbic development that not only prevents the individual from making necessary attachments, but also creates a memory system that is ill equipped to cope with trauma. The important distinction between PTSD and BPD (although they arguably share a number of characteristics) is the difficulty with interpreting emotion, and creating habit-based memories based on emotionally laden information; this is the domain of the amygdala, which we also propose to be developmentally dysfunction in BPD. Taken together, the above evidence is consistent with a developmental pattern of hyperactive (meaning that emotional information is constantly being updated, to the extreme that it is not functional), but ineffective affective memory (spinning one's wheels) associated with BPD. It is additionally conceivable that, over time, unstable affective perceptions of relationships contribute to a cognitive style that is constantly updating IM, such that there are few, if any, long-term affective habits or procedures associated with the attachment figure. (Note that it is also conceivable that this cognitive style partly underlies splitting behavior in BPD.)

In this way BPD has something important in common with obsessive–compulsive disorder (OCD). Although this may not be intuitive, both populations have difficulty trusting their feelings regarding their memory. OCD patients have been shown to have normal memory for events. However, patients with OCD consistently self-report uncertainty around the accuracy of their memories and on neuropsychological memory tests are more likely to report low "feeling of knowing" with regard to memory for stimuli. Interestingly, these self-reports do not correlated with actual memory performance (Tuna, Tekcan, & Topcuoglu, 2005). In a study examining neural activity, metabolism in the right hippocampus, was

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associated with the severity of obsessive–compulsive symptoms, but not with memory for stimuli (Kwon et al., 2003). Perhaps there are features of BPD that resemble an interpersonal form of OCD, where there is a compulsive need to check on the status of relationships due to a feeling of uncertainty regarding the veracity of one's memory. In sum, it is offered that the volumetric differences in hippocampal volume are a relatively generic risk factor for psychopathology, and that it is the co-occurrence with amygdalar dysfunction early on that predicts the eventual appearance of BPD.

Traditional neuropsychological tasks have largely failed to reveal gross differences between BPD and control subjects. Intelligence testing, such as performance on the Wechsler Adult Intelligence Scale, does not seem to discriminate borderline subjects from normal subjects or many of their Axis II counterparts. Likewise, borderlines are often characterized by good functioning in work environments relative to other patient populations, argues against a general neurological or cognitive impairment. Investigations of brain regions known to be primarily involved in cognition (e.g., the dorsolateral prefrontal area), have also reported no significant differences between BPD patients and nonpsychiatric control participants (Tebartz van Elst, 2003). Further, the specificity of interpersonal problems generally experienced by individuals with BPD suggests that there may be certain emotional features of social or interpersonal interactions that are highly relevant to this patient group. This notion has received empirical support from the work of Korfine (Korfine, 1998; Korfine & Hooley, 2000), which examined directed-forgetting performance in a BPD patient group. As described above, the results of this work indicated that a disinhibitory process may be at the root of BPD patients' tendency to remember BPD-relevant words that they were instructed to forget. Whether early trauma is a cause or a symptom of BPD is not known (see Horesh, Sever, & Apter, 2003, for a review of this issue). It may simply be that a person who has BPD may be more likely to interpret or remember events as traumatic. Further, the ruminations associated with BPD may indicate memory deficits associated with traumatic stimuli, these ruminations may actually be indicative of a memory system trying (unsuccessfully) to consolidate the events, take away the implicit meaning, and "move on," a process that is often difficult for BPD patients. This thinking is also reflected by the idea that the symptoms associated with BPD are ". . . the manifestations and consequences of complex interactions among cognitive–affective units that are triggered by a specific situational contingencies and, through synergistic and recursive activation, lead to characteristic patterns of action, thoughts, and feelings" (Meyer & Pilkonis, 2005, p. 240).

As has been previously argued, one consequence of the early disturbance in the amygdala and hippocampus is subsequent disruption of the systems that rely on these structures during development, namely the frontal cortex. Frontal deficits have been reported in both the orbitofrontal cortex (OFC) and the anterior cingulate cortex (ACC) among patients with BPD (Tebartz van Elst, 2003). Perturbations to the OFC have been linked to impulsivity, irritability, and emotional instability. This area of the brain receives much input from the limbic system, and is primarily responsible for mediating functions critical to social behavior, including reward–punishment responses, recognition of emotional expression in others, identifying social signals and violations, and inhibition of behaviors that may compromise social rewards or lead to punishment. The OFC in borderlines has also been found to be hypometabolic. Interestingly, this same hypometabolism is seen in violent individuals, behavior somewhat consistent with BPD pathology (Soloff, 2003). Given its central role in the integration of emotion and cognition described in detail above (see section on adolescent development) it is not surprising that abnormalities in this region have been found in conjunction with BPD. Interestingly, and consistent with the ACC's role in second-order body representations, the volume of this region was correlated with incidents of self-injurious behavior among BPD patients (Tebartz van Elst, 2003). Finally, when education level, alcohol use, abuse, major depressive disorder, and PTSD were controlled for the OFC and ACC

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volumes remained significantly lower than nonpsychiatric controls, the observed neurological differences are not likely to be the direct result of environmental influence (Tebartz van Elst, 2003). In sum, the BPD patient's amygdala tends to be overreactive, increasing vigilance and evaluating threat in stimuli at higher rates than normal individuals; however, due to frontal deficits, BPD patients are less likely than nondiagnosed individuals to appropriately evaluate and modulate such reactions. The increasingly complex social and emotional demands of adolescence likely push the system to its "tipping point."

Adolescence heralds the emergence of BPD as a clinical syndrome. Unfortunately, the increased focus on relationships in adolescence only exacerbates the long-standing attachment difficulties among those at risk for BPD. There are a number of reasons why relationships become central to adolescent development; however, from the neurobiologist's point of view they are an essential precursor to mate selection. Although it is obvious that human adolescence has come to represent a great deal more than simply reproduction, it is difficult to ignore some of the basic attainments of puberty. For example, it is now known that the sensitivity of the hippocampus to puberty-related increases in estrogen promotes greater growth in this structure among girls relative to boys, while sensitivity to androgen promotes greater amygdala growth in boys (Giedd, Castellanos, Rajapakse, Vaituzis, & Rapoport, 1997). It has been suggested that this difference may influence (although not exclusively) gender-based behavior associated with puberty.

Although there has been extensive investigation regarding the role of the amygdala in the fight or flight response, little attention has been paid to how a balance between this and the role of the hippocampus in the "tend and befriend" (Taylor et al., 2000) response could be effectively integrated for adolescent girls. Taylor and colleagues have reasoned that the adaptive value of fighting or fleeing may be lower for females, who often have dependent young and so risk more in terms of reproductive success if injured or dislocated. It is also the case that females of many species form tight, stable alliances, possibly reflecting an adaptive tendency to seek out friends for support in times of stress. Following a meta-analysis of extant animal literature Taylor and colleagues reported that relative to males, females' physical aggression and fear-related behaviors are less intense and more "cerebral." Specifically, females were more likely to display aggression and fear behaviors in response to specific circumstances, and these responses were less tied to physiological arousal. Thus, although both genders share the capacity for fight or flight, females seem to use it less. As previously discussed, oxytocin is known to contribute significantly to females' tendency to affiliate. In many mammals, and crossculturally in humans, females form especially close, stable attachments with other females, often kin. This is a process that takes on additional import following the onset of puberty. Before puberty priority had been given to the attachment relationship with the primary caregiver; following the onset of puberty there is a shift to peers and eventually romantic partners.

Adolescents spend an inordinate amount of time with friends. In a regular school day, teens have an average of 299 interactions with peers (Barker & Wright, 1951). During the limited time teens spend away from their friends, if they are not conversing on the phone or computer, they are likely *thinking* about their peer groups. Based on the empirical and theoretical data on the importance of social self-perceptions during the teenage years (Jacobs, Vernon, & Eccles, 2004), it is perfectly reasonable for teenagers to place great importance on their friendships. Adolescents turn to peer groups for emotional support and perceive group approval as an indication of social acceptability (Brown et al., 1993). These processes are all heavily reliant upon one's ability to form attachments, and retain a continuous and appropriately flexible affective sense of your relationships with others. During late adolescence peer groups become increasingly coed, and provide opportunities for sexual exploration and eventually mate selection. Assuming that the attachment relationship did not provide the necessary socioemotional foundation in childhood for individuals with BPD, it is inevitable that both peer and romantic relationships are highly dysfunctional for BPD patients (Agrawal et al., 2004).

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Given the combination of biology, social expectations, and hormonal events that take center stage during puberty, it is not surprising that women are 2–4 times more likely to seek treatment for and be diagnosed with BPD than are men (Swartz, Blazer, George, & Winfield, 1990). This etiological discrepancy also adds to the plausibility of the idea that amygdalar/hippocampal dysfunction is at the core of this disorder. It is plausible that the same biology in males is likely to manifest as antisocial personality disorder (see Paris, 2004, for a review of this idea). Proper adolescent development relies in great part on the processes that have preceded it. For example, by the time you are ready to seek out a mate, your language development should be complete. One must possess mastery over language, without devoting conscious energy to its construction, so that it may serve as a tool for establishing new relationships, and aid in the navigation of your social world. This is reflected in the fact that although it is possible to learn a new language after puberty, you will always speak with an accent. In terms of attachment, those who have not passed the appropriate developmental milestones will forever be easily discernable to those around them, if for no other reason than their "emotional accent." In terms of neuropathology, the changes that take place during puberty are effectively like attaching a 330 horsepower motor to a cardboard box to make a vehicle; it simply will not work. There is convincing evidence that the effect of having a developmentally dysfunctional amygdala/hippocampal complex undergo a biobehavioral growth spurt is certainly enough to provoke the symptoms associated with the emergence of BPD.

Cautionary Note

The prediction of adolescent or adult psychopathology from infancy and early childhood is modest because some infants with risk profiles are fortunate enough to encounter more-supportive environments later, while others living under adverse circumstances possess a temperament that enables them to develop effective coping styles. Many children who manifest symptoms do not encounter adverse circumstances until later in childhood (Kagan & Zentner, 1996). Despite these shortcomings, formulation of these types of models helps us to generate better descriptions of "at risk" populations, and are therefore worth consideration. Although the presented model is not meant to capture the full range of possible adult expression of early attachment disturbance, it may typify a unique subtype. This model is meant only as but one way of clustering developmental profiles that may be predictive of adult psychopathology. Kagan reminds us (1996):

> The actualization of most profiles—Axes I and II—requires the combination of at least three independent factors: a particular temperament, and environment that amplifies the particular vulnerability associated with the temperament, and stressors that will precipitate the symptoms.

Conceptualizing the relation between neural "hardware" and experiential "software," a permutation of the eternal nature/nurture debate, has kept psychologists, psychiatrists, and philosophers speculating for centuries. Given our recent advances in technology, it seems that we stand at the threshold of a great renaissance in the understanding of this relationship. Further, it is through careful characterization of not only the factors described above, but also their highly individualized, and mutually influential interplay, that will be better able to better identify populations "at risk."

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