7

Borderline Personality Disorder

Rebekah Bradley

Emory University

Carolyn Zittel Conklin

Cambridge Health Alliance and Harvard Medical School

Drew Westen

Emory University

Borderline personality disorder (BPD) is one of the most prevalent, most widely studied, and yet most controversial of the personality disorders (PDs) described in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association [APA], 1994). Its public health significance arguably rivals that of any other diagnostic syndrome. Patients with BPD constitute 20% of psychiatric

inpatients and 10% of patients seen in outpatient mental health clinics (APA, 1994) and are high consumers of emergency room services, crisis lines, and psychiatric consultations requested by other medical services (Ellison, Barsky, & Blum, 1989; Forman, Berk, Henriques, Brown, & Beck, 2004; Gross et al., 2002; Reich, Boerstler, Yates, & Nduaguba, 1989; Zanarini, Frankenburg, Hennen, & Silk, 2004). Between 70% and 75% of BPD patients have a history of at least one self-injurious act (Clarkin, Widiger, Frances, Hurt, & Gilmore, 1983; Cowdry, 1992), and quick calculations with available statistics (APA, 1994; McGlashan, 1986; Samuels et al., 2002; Stone, 1993; Torgersen, Kringlen, & Cramer, 2001) indicate that of the 6 million individuals currently estimated to have BPD in the United States alone, between 180,000 and 540,000 will die by suicide.

In this chapter, we provide a broad overview of the state of knowledge of BPD. We begin by briefly describing the evolution of the diagnosis and contemporary controversies regarding the construct itself and the way it should be defined. We then discuss the assessment of BPD. Next we examine what is known about the development and developmental course of BPD, including its etiology, longitudinal stability, and prognosis. The final section considers treatment approaches, including a number of relatively recent empirical developments in the psychotherapy of BPD.

The Borderline Diagnosis: Evolution and Diagnostic Controversies

The concept of "borderline" has undergone a substantial evolution since its early identification by psychoanalytic clinical theorists, who first identified the construct as "pseudoneurotic schizophrenia," "as-if personality," and eventually "borderline state" (Knight, 1953, 1954). In this section, we briefly describe the evolution of the construct. We then examine contemporary controversies and diagnostic dilemmas in the understanding and diagnosis of the borderline construct.

Evolution of the Borderline Construct

Initially the term *borderline* referred to individuals who seemed neither neurotic nor psychotic but were somewhere in between. This was the conceptualization that Kernberg (1967) later elaborated in his concept of borderline personality organization (BPO). By "personality organization," Kernberg meant enduring ways of feeling, thinking, behaving, experiencing the self and others, and dealing with unpleasant realities. In Kernberg's view, patients with borderline personality organization tend to use drastic, immature ways of dealing with impulses and emotions (e.g., behaviors such as cutting and defensive maneuvers such as denial of obvious realities). They

are not psychotic but can become cognitively more disorganized than most people, particularly under stress, and have difficulty maintaining balanced views of the self and significant others ("splitting" their representations into all good and all bad).

Over time, the concept of borderline as a level of disturbance (originally between neurotic and psychotic) shifted from this broader construct to the more specific diagnostic category first defined in the third edition of the DSM (DSM-III; APA, 1980). Kernberg's concept of borderline influenced the description of the disorder in DSM-III, which has remained intact, with small modifications, for the last 20 years. However, his concept of border-line as a form of personality organization is a broader construct that describes a *level* of personality sickness that encompasses many of the DSM-IV PDs, including all the Cluster A (odd, eccentric) PDs; the Cluster B (erratic, dramatic) PDs, with the exception of some higher functioning nar-cissistic patients; and the more disturbed subset of patients within each of the Cluster C (anxious, fearful) PDs.

Like most diagnoses, the construct of BPD first emerged from the work of prescient clinical observers who attempted to identify patterns of covari-ation among symptoms not previously understood, followed by research aimed at refining the construct. The initial efforts to establish a more empir-ically grounded concept of BPD actually began prior to DSM-III with the work of Grinker, Werble, and Drye (1968), who suggested the first empiri-cally derived diagnostic criterion set for the borderline syndrome. This was followed by development of the Diagnostic Interview for Borderline Personality Disorder (DIB; Gunderson & Kolb, 1978; Gunderson, Kolb, & Austin, 1981; Gunderson & Singer, 1975). As editor of DSM-III, Spitzer developed potential diagnostic criteria for BPD by reviewing clinical and research literature and consulting with clinicians expert in treating border-line patients. He then collected data in a national survey of psychiatrists who evaluated the selected criteria. The resulting set of distinguishing borderline characteristics (Spitzer, Endicott, & Gibbon, 1979) became the basis for the BPD criteria in the DSM-III (APA, 1980). This resulted in BPD's becoming an official psychiatric disorder rather than a level of personality structure or disturbance. DSM-IV defines the essential features of BPD as a "pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity that begins by early adulthood and is present in a variety of contexts" (APA, 2000, p. 706).

Current Controversies and Diagnostic Dilemmas

Like the other PDs, the BPD diagnosis in DSM-IV emerged through over half a century of clinical observation, which largely generated the criteria for the disorder in DSM-III (and instruments for assessing it), followed by 25 years of research aimed at refining the diagnosis. The criteria for the disor-der clearly capture a group of severely impaired patients frequently seen in

mental health settings. However, a number of problems limit the clinical util-
ity and validity of the diagnostic criteria for BPD. We focus here on three:
heterogeneity of symptom presentation, categorical diagnosis, and excessive
comorbidity with other Axis II disorders as well as Axis I disorders.

With respect to heterogeneity, a patient can receive the BPD diagnosis in
over 150 different ways based on varying combinations of the nine criteria
for the disorder (Skodol, Gunderson, Pfohl, et al., 2002). Put another way,
two patients may both be diagnosed with BPD while sharing only one symp-
tom in common. This fact has important clinical implications because sub-
types of BPD seem to exist that do not reflect random variation among
criteria but rather meaningful, patterned heterogeneity, such as internalizing
and externalizing subtypes of the disorder (Bradley, Zittel, et al., 2005;
Conklin & Westen, 2005; Conklin, Bradley, & Westen, 2006; Westen &
Shedler, 1999b; Zittel & Westen, 2002).

With respect to categorical diagnosis, the DSM approach to classification
assumes that PDs represent categorically distinct classes of psychopathology.
However, most research on classification of PDs favors a dimensional rather
than a categorical understanding of PD (e.g., Clark, Livesley, & Morey,
1997; Trull, 2001; Widiger, 1995). Consistent with this overall trend in per-
sonality research, research on BPD, including research applying taxometric
analysis (Meehl, 1995), suggests that the disorder is likely best represented
dimensionally and does not represent a distinct taxon (e.g., Rothschild,
Cleland, Haslam, & Zimmerman, 2003).

With respect to comorbidity, research using both DSM-III and DSM-IV
criteria indicates high levels of comorbidity with other PDs, particularly anti-
social PD, avoidant PD, dependent PD, and paranoid PD (Becker, Grilo,
Edell, & McGlashan, 2000; Gunderson, Zanarini, & Kisiel, 1991, 1995;
Oldham et al., 1992; Stuart et al., 1998). This finding suggests that the diag-
nostic criteria do not adequately capture a disorder distinct from other dis-
orders or from a general personality pathology dimension. Indeed, many of
the DSM-IV PDs—including paranoid, schizoid, schizotypal, antisocial,
histrionic, and sometimes dependent—are consistent with borderline per-
sonality organization as defined by Kernberg. With the exception of
schizoid, all of these PDs show high comorbidity with DSM-defined BPD,
tending to cluster together in studies of adaptive functioning, and disorders
such as avoidant, narcissistic, and obsessive-compulsive generally showing
better adaptive functioning (e.g., Skodol, Gunderson, McGlashan, et al.,
2002; Skodol, Gunderson, Pfohl, et al., 2002; Tyrer, 1996). In any case, the
comorbidity of BPD with other Cluster B PDs (histrionic, antisocial, and nar-
cissistic; Fyer, Frances, Sullivan, Hurt, & Clarkin, 1988) as well as with dis-
orders such as avoidant and schizotypal PDs (Barasch, Kroll, Carey, &
Sines, 1983; Pfohl, Coryell, Zimmerman, & Stangl, 1986), is highly prob-
lematic, particularly given that schizotypal and avoidant individuals tend to
be socially withdrawn, whereas BPD is associated with fear of aloneness and
the trait of extraversion (e.g., Lynam & Widiger, 2001). Borderline PD also
shows high comorbidity with most nonpsychotic Axis I disorders, notably

mood, anxiety, substance use, and eating disorders (e.g., Zanarini et al., 1998; Zimmerman & Mattia, 1999).

In response to these problems, DSM task forces and PD work groups since DSM-III have attempted to adjust diagnostic criteria with the goal of making BPD less redundant with other diagnoses. For example, the Axis II Work Group for DSM-IV rewrote the DSM-III-R criterion "affective instability: marked shifts from baseline mood to depression, irritability, or anxiety, usually lasting a few hours and only rarely more than a few days." In hopes of better discriminating between major depression and BPD, the word "depression" was replaced with "dysphoria"; in hopes of better differentiating between the mood lability seen in cyclothymic disorder and the unstable affect seen in BPD, the phrase "marked shifts . . . [of] mood" was replaced by "marked reactivity of mood." Such efforts do not appear, however, to have substantially reduced the comorbidity of BPD with other disorders, raising questions about whether the diagnosis remains, in Akiskal's (1996; 2004) words, "an adjective in search of a noun."

In summary, the development of diagnostic criteria for BPD in DSM-III laid the groundwork for a surge of research on the disorder. BPD is now the most highly researched PD and has the strongest empirical evidence regarding its phenomenology, etiology, and treatment. Nevertheless, the research that was in large measure fostered by the presence of DSM criteria since 1980 has resulted in the identification of a number of problems with the diagnosis that remain to be resolved.

Assessment

The Diagnostic Interview for Borderline Personality Disorder (DIB; Gunderson & Kolb, 1978; Gunderson et al., 1981; Gunderson & Singer, 1975) was the gold standard procedure for assessing BPD in the decade following the definition of operational criteria for the disorder in DSM-III. However, what quickly became apparent was that any sample of BPD patients could differ in unknown ways from any other sample, depending on the presence of comorbid PDs. Researchers addressed this problem with the development of structured interviews designed to assess all of the DSM PDs. The advantage of these instruments was that they assessed the range of personality pathology defined by the DSM. The disadvantage was that, in less time than it typically takes to administer the DIB (a semistructured interview for a single disorder), they attempted to assess the roughly 10 PDs defined by the various versions of the DSM since DSM-III. To economize the assessment of these disorders, interviews came to emphasize more the behavioral manifestations of the disorder (e.g., cutting) over the functional or "structural" aspects of personality that originally defined the disorder in the clinical literature. In turn, the diagnostic criteria for the disorder shifted toward readily observable behaviors that could be assessed by structured interview, leading to the possibility of the procedural tail wagging the conceptual dog (Westen, 1997).

Methods for assessing BPD generally rely on patients' self-reported symp-toms using either structured interviews or questionnaires. A full review of such measures is beyond the scope of this chapter (see Clark & Harrison, 2001, for a review). However, we briefly present the relative strengths and weaknesses of these approaches.

The currently accepted diagnostic gold standard for the assessment of BPD is a standardized structured interview yoked to DSM criteria (e.g., the Structured Clinical Interview for DSM-IV Personality Disorders [SCID-II; First, Spitzer, Gibbon, & Williams, 1997]; the Structured Interview for DSM-III-R Personality Disorders [SIDP; Pfohl, Blum, Zimmerman, & Stangl, 1989]). A primary advantage of this approach is that it asks questions about each criterion directly, ensuring adequate coverage for a DSM-IV diagnosis. A second advantage is reliability, particularly when this approach is compared with the method more common in clinical practice of conducting unstructured interviews with patients before referring to DSM or International Classification of Diseases (ICD) diagnostic criteria, which yields low interrater reliability (Mellsop, Varghese, Joshua, & Hicks, 1982; Satorius et al., 1993).

However, this approach to PD diagnosis has limitations. First, rates of comorbidity are extremely high, with the average patient receiving any PD diagnosis receiving 4 to 6 of the 10 DSM-IV PDs by structured interview and often even more by questionnaire (see Westen & Shedler, 1999a). Although this problem stems at least in part from the overlap among the DSM-IV dis-orders themselves, other approaches to diagnosis, such as assessing the patient's match to a prototype of the disorder, show similar external corre-lates indicative of diagnostic validity while substantially decreasing estimates of comorbidity (Westen, Shedler, & Bradley, 2006). Second, neither struc-tured interviews nor questionnaires correlate strongly with consensus diag-noses made using all available data collected over time by teams of clinicians who not only have access to data from other informants but also know the patients well (e.g., Pilkonis et al., 1995; Pilkonis, Heape, Ruddy, & Serrao, 1991; Skodol, Oldham, Rosnick, Kellman, & Hyler, 1991). The third and most central problem of this approach is reliance on the self-awareness among a group of patients (PD patients) who, almost by definition, are likely to have distorted views of themselves and others. For example, Oltmanns, Turkheimer, and their colleagues have demonstrated across multiple samples that although lay informants converge remarkably well in assessing their peers' personality pathology, aggregated peer assessments tend to correlate only on the order of $r = .20$ to .30 with self-reports (Clifton, Turkheimer, & Oltmanns, 2003; Klein, 2003; Oltmanns, Melley, & Turkheimer, 2002; Thomas, Turkheimer, & Oltmanns, 2003). This relatively modest level of self-informant agreement is only slightly lower than meta-analytic estimates, which are in the mid .30s (Klonsky, Oltmanns, & Turkheimer, 2002). For the more overt symptoms of BPD, such as self-mutilation and suicidal ideation, self-report biases are less likely to be problematic. For more subtle

personality symptoms, and particularly for externalizing symptoms (see Fiedler, Oltmanns, & Turkheimer, 2004), these biases may be more problematic. Unfortunately, the more subtle personality symptoms appear to be the most stable indicators of the disorder (Grilo et al., 2004; Zanarini, Frankenburg, Vujanovic, et al., 2004).

Another approach to the assessment of personality, including BPD, relies on the use of a systematic clinical interview paired with psychometrically valid instruments for rating data gathered in the interview. Westen and Shedler (1999a; 1999b) developed a Q-sort instrument designed to quantify the judgments of experienced clinical interviewers, combining clinical description with statistical prediction. Clinically experienced observers sort the 200 items of the SWAP-II Q-sort (or its progenitor, the SWAP-200) based either on their observation of a patient over time in treatment or on data ascertained using a systematic clinical interview, the Clinical Diagnostic Interview (CDI; Westen & Muderrisoglu, 2003, 2006; Westen, Muderrisoglu, Fowler, Shedler, & Koren, 1997). The CDI differs from structured PD interviews in that it does not primarily ask patients to describe themselves (although it does not avoid face-valid questions about behaviors, intentions, or phenomenology, such as whether the patient has self-mutilated or thought about suicide). Instead, it asks patients to provide detailed narratives about their symptoms, their school and work history, and their relationship history, focusing on specific examples of emotionally salient experiences. From these data (or from all available clinical data, if the clinician is describing a patient in ongoing treatment), the clinician-informant makes judgments about the ways the patient characteristically thinks, feels, regulates impulses and emotions, views the self and others, and behaves in significant relationships, and these are reflected in the clinician's placement (ranking) of the items.

Several recent studies using the SWAP-200 or the newly developed SWAP-II have focused on BPD (Bradley, Zittel, et al., 2005; Westen, Bradley, & Shedler, 2005; Westen & Shedler, 1999a, 1999b; Zittel & Westen, 2005). These studies indicate that SWAP-based assessment of BPD predicts external correlates, such as adaptive functioning and developmental history, in ways predicted by prior research (Zittel & Westen, 2005). These data also highlight the importance of understanding not only stress-dependent behaviors that are hallmarks of BPD (e.g., self-harming behavior) but also those characteristics (e.g., depressed mood, anxiety, hopelessness) that are characteristic of the everyday experience of BPD patients but not necessarily distinctive to them because they are common in psychiatric samples (Bradley, Zittel, et al., 2005).

In addition, these studies identify aspects of BPD not captured fully by the nine DSM BPD criteria, which are probably better understood as indicators of a latent construct than as the signs and symptoms that exhaustively define the disorder. For example, SWAP-based data provide a more thorough description of affect dysregulation among BPD patients. Specifically, data

obtained using the SWAP-200 and SWAP-II reveal that emotion dysregulation in BPD comprises a tendency for emotions to spiral out of control, a tendency to become irrational under stress, and a dependence on others to regulate emotions.

All approaches to assessment have their limitations, and the SWAP is no exception. The most central limitation of most data obtained on BPD so far using the SWAP is that these data rely, like most studies using structured interviews and questionnaires, on the perspective of one informant (in this case, the clinician; in the modal study of BPD, the informant is the patient). Future research using all assessment procedures needs to triangulate data gathered from multiple sources, including self-reports, quantified clinical judgments, informant ratings (e.g., friends and family), and laboratory tasks.

Etiology of BPD

Research on BPD implicates a broad array of factors in the etiology of BPD, including biological/genetic factors, separation and loss, childhood abuse, global family environment, and disrupted attachments. Research on the etiology of BPD has largely addressed each of these domains separately and hence has not yet established models for their combination and interaction, although such work is under way. We will first review research for each of these etiologic factors and then summarize the current status of the field with respect to understanding their interplay.

Biological and Genetic Factors

Clearly, personality traits are heritable (see Plomin, Chipuer, & Loehlin, 1990), although the extent to which genetic transmission contributes to the development of BPD has yet to be fully understood. Nevertheless, a growing number of studies, including two preliminary twin studies (Nigg & Goldsmith, 1994; Torgersen, 1980; Torgersen et al., 2000), suggest the importance of familial aggregation. In a recent review of family studies of BPD, White, Gunderson, Zanarini, and Hudson (2003) found little support for familial links between schizophrenia or bipolar disorders and BPD, some support for familial links with major depression, and stronger support for familial aggregation of impulse spectrum disorders, including BPD itself. As we describe below, research that addresses both main effects and interactive effects in combination with environmental traumas is likely to prove more fruitful (see Nigg & Goldsmith, 1994; Torgersen, 1980; Torgersen et al., 2000; White et al., 2003).

An alternate approach to understanding the heritability of BPD is to look at subsyndromal markers, or endophenotypes, of the BPD construct (e.g., affect dysregulation and relationship instability). A recent study (Zanarini,

Frankenburg, Yong, et al., 2004) found that although the diagnosis of BPD showed familial aggregation, both specific BPD criteria and the broader BPD symptom categories of affect, cognition, impulsivity, and interpersonal relationship disturbance showed even stronger familial aggregation and discriminated better between the relatives of BPD probands and those of comparison subjects. This idea is consistent with theory and research conceptualizing BPD as the extreme presentation of aspects of heritable temperament or traits (e.g., impulsivity, neuroticism, and affective lability; see Paris, 2003; Skodol, Gunderson, Pfohl, et al., 2002, for reviews). Impulsive aggression is a central characteristic of Cluster B Axis II disorders, particularly BPD and APD (Coccaro, Bergeman, & McClearn, 1993; Goodman & Yehuda, 2002; Skodol, Gunderson, Pfohl, et al., 2002), and shows substantial heritability. However, the data on familial aggregation are difficult to interpret because of the complexity in disaggregating heritable temperamental and family environment effects (i.e., having a parent with borderline or related psychopathology increases the likelihood of adverse childhood events).

A burgeoning literature on the neurobiology of BPD focuses primarily on two trait aspects considered central to BPD: affect instability/dysregulation and impulsivity/impulsive aggression (e.g., Siever & Davis, 1991). Most functional neuroimaging research rests on the premise that BPD is associated with hyperreactivity to emotional stimuli, which should be manifest in such neural responses as heightened activation of the amygdala (Donegan et al., 2003; Herpertz et al., 2001). Several studies using functional magnetic resonance imaging (fMRI) do indeed find increased amygdala reactivity when these individuals are exposed to emotion-related stimuli, particularly faces (Donegan et al., 2003; Herpertz et al., 2001). Interestingly, BPD patients appear to show greater amygdala reactivity to neutral faces as well, perhaps supporting prior research linking BPD to a malevolence attribution style (Nigg, Lohr, Westen, Gold, & Silk, 1992; Westen, 1991b). Another study (Schmahl, Vermetten, Elzinga, & Bremner, 2003) identified decreased amygdala volume in BPD subjects (Driessen et al., 2000). Currently, the field is witnessing an explosion of fMRI research with BPD patients that is likely to elucidate the nature of the disorder (e.g., by examining links between amygdala reactivity and hypoactivity of cortical circuits that would normally regulate it, such as the ventromedial prefrontal cortex). At the same time, such research should be treated cautiously from an etiologic standpoint. Finding that BPD patients show greater amygdala reactivity, for example, is important, but it does not go far beyond a neural translation of the definition of a disorder characterized by emotional reactivity. Complicating matters, the pattern of data across studies may be complex because of the complex ways BPD patients try to regulate their affects. For example, the amygdala of BPD patients who dissociate may be relatively *less* reactive than that of non-BPD patients (Schmahl et al., 2004).

The majority of the research looking at the biological basis of impulsivity/ impulsive aggression focuses on the role of reduced serotonergic responsivity

(for reviews, see Skodol, Gunderson, Pfohl, et al., 2002; Soloff, Lynch, Kelly, Malone, & Mann, 2000). For example, some research associates lower levels of 5-hydroxytryptophan (e.g., Mann, 1998) with increased self-harming and suicidal behaviors. Using positron-emission tomography (PET), Leyton et al. (2001) identified an inverse relationship between alpha-methyl-L-tryptophan (converted to alpha-methyl-serotonin) and impulsivity as measured by errors on a go/no-go task. Other studies (De La Fuente et al., 1997; Soloff, Meltzer, Greer, Constantine, & Kelly, 2000) suggest that orbital prefrontal dysfunction may be associated with increased aggression via inhibition of limbic regions. Other neuropsychological studies implicate impaired functioning on laboratory tasks designed to evaluate planning and decision making abilities (Bazanis et al., 2002; Lenzenweger, Clarkin, Fertuck, & Kernberg, 2004), which could implicate prefrontal circuits more broadly.

Separation and Loss

A considerable body of research points to separation from or loss of parental figures during childhood as etiologically relevant to BPD. For example, a meta-analytic review found that 20% to 40% of BPD patients had experienced traumatic separations from one or both parents (Gunderson & Sabo, 1993). Childhood histories involving lengthy separations from, or the permanent loss of, one or both parents have been found to discriminate BPD patients from patients with schizophrenia, depression, and other PDs (Akiskal et al., 1985; Bradley, 1979; Frank & Paris, 1981; Goldberg, Mann, Wise, & Segall, 1985; Gunderson, Kerr, & Englund, 1980; Links, Steiner, Offord, & Eppel, 1988; Paris, Nowlis, & Brown, 1988; Soloff & Millward, 1983; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1989).

In evaluating the relationship of separation and loss to BPD, however, factors such as the child's age, nature and duration of the separation or loss, and availability of nurturant, enduring surrogate caregivers in the absence of the primary caregiver need to be taken into account. For example, a classic study of depression (see Brown & Harris, 1989, for details) found a constellation of symptoms resembling BPD to be highly prevalent among patients who had a peculiar kind of separation history (which they labeled "aberrant"), in which the mother appeared to have left the children for no "socially acceptable" reason (e.g., she abandoned her children for months because of her own instability). It is important to note when interpreting these findings that causal direction is not clear due to possible genetic confounds or gene-environment interactions.

Childhood Abuse

Early writing on BPD (Stern, 1938) focused on the etiologic role of childhood abuse, noting that "actual cruelty, neglect, and brutality by the parents

of many years' duration are factors found in these patients. These factors operate more or less constantly over many years from earliest childhood. They are not single experiences" (p. 470). This early observation has been corroborated in the empirical literature, with numerous studies identifying a link between abuse, particularly childhood sexual abuse, and BPD (e.g., Ogata et al., 1990; Silk, Lohr, Ogata, & Westen, 1990; Westen, Ludolph, Misle, Ruffins, & Block, 1990; Zanarini, 1997). In Herman, Perry, and van der Kolk's (1989) sample of BPD patients, 81% had childhood histories that included abuse, both physical abuse (71%) and sexual abuse (67%). In a community-based longitudinal study of PDs, Johnson, Cohen, Brown, Smailes, and Bernstein (1999) found that experiences of childhood physical, sexual, and emotional abuse increased risk for development of virtually all of the DSM-IV PDs. However, when they adjusted for the effects of co-occurring PDs, only the Cluster B PDs remained significantly related to experiences of childhood maltreatment. Zelkowitz, Paris, Guzder, and Feldman (2001) found that people who had experienced childhood sexual abuse were four times more likely to develop BPD than those who had not. Not surprisingly, some studies did not find such a significant link between childhood abuse and BPD (see Fossati, Madeddu, & Maffei, 1999). The association between child abuse and BPD exists in the context of multiple, interactive genetic, environmental, and social factors, and the idea of one-to-one correspondence is an unfounded oversimplification (Bradley, Jenei, & Westen, 2005; Paris, 1997).

In addition to the presence/absence of abuse, several studies suggest that characteristics of abuse, including severity, age of onset, and number of types of abuse experienced, contribute to degree of impairment related to borderline pathology (McLean & Gallop, 2003; Silk, Lee, Hill, & Lohr, 1995; Yen et al., 2002; Zanarini et al., 2002). Studying BPD in adolescents, Ludolph et al. (1990) suggested that cumulative trauma, rather than a single traumatic event, appears to be more relevant to the development of BPD (see also Weaver & Clum, 1993). Thus, although not all patients with BPD have experienced childhood trauma, the current research indicates that traumatic experiences are a salient component of the developmental history of many individuals who develop BPD.

Family Environment

More generally, an unstable, nonnurturing family environment appears to contribute to the development of BPD. In adolescent patients, for example, the tendency to misunderstand people's actions and intentions (poor understanding of social causality) characteristic of BPD shows a strong association ($r = \sim.50$) with a simple metric of family instability, namely, the number of times the family moved (Westen, Ludolph, Block, Wixom, & Wiss, 1990). Much of the literature on traumatic precursors to PDs (and other psychiatric symptoms, such as depression) has not taken into account the impact of family

environment, making it difficult to disentangle the impact of sexual or physical abuse from the overall family context within which abuse typically occurs, such as family chaos, disrupted attachments, multiple caregivers, parental neglect, alcoholism, and/or evidence of affective instability among family members (Dahl, 1995; Gunderson & Phillips, 1991; Ogata et al., 1990).

Studies of adverse childhood events have linked the number of such events to multiple adverse medical and psychiatric outcomes (Dong et al., 2004; Edwards, Holden, Anda, & Felitti, 2003). Research that has considered several of these variables together with regard to the etiology of PDs has often found that the context within which abuse occurs (e.g., problematic attachment relationships, emotional abuse, and neglect) is as strongly associated with BPD as the presence or absence of physical or sexual abuse (Johnson et al., 2001; Ludolph et al., 1990; Zanarini et al., 1989). For example, a recent study of the relationship between childhood abuse, family environment, and BPD found that family environment partially mediated the relationship between abuse and level of BPD symptoms (Bradley, Jenei, et al., 2005), although abuse showed a substantial unmediated relation to BPD. In other words, sexual trauma predicted BPD, but part of its impact reflected the effects of an unstable, nonnurturing family environment. The dearth of research on the relation between abuse and family environment is particularly problematic in the case of BPD, which is associated not only with the attribution of malevolence on others but also with fears of abandonment and aloneness that may be related to neglectful, absent, or unstable parenting (see Gunderson, 2001; Zanarini & Frankenburg, 1997).

One attempt to clarify this literature suggests that insecure attachment to parental figures, coupled with emotionally unstable or neglectful family environments, may account for the development of BPD, whereas physical or sexual abuse may account for symptom severity (Salzman, Salzman, & Wolfson, 1997). Other models suggest that sexual abuse may account for some of the severity of impulsive symptoms in BPD, such as self-mutilation, suicide attempts, substance abuse, promiscuity, running away, and assaultiveness (e.g., Westen, Ludolph, Misle, et al., 1990). Zanarini and Frankenburg (1997) reviewed research on the etiology of BPD and distinguished three types of trauma that they felt better explained the pathogenesis of BPD. Type I trauma includes "unfortunate but not entirely unavoidable or unexpectable experiences," including prolonged early separations, chronic insensitivity to the pre-borderline child's feelings and needs, and serious emotional discord in the family, perhaps leading to separation or divorce. Type II trauma includes experiences of verbal and emotional abuse, neglect of age-appropriate physical needs, and circumscribed episodes of parental psychiatric illness. Type III trauma includes experiences of clear physical and sexual abuse, chronic psychiatric illness in caretaker or caretakers (particularly Axis II psychopathology and substance abuse), and a generally chaotic and dysfunctional home environment (e.g., parents repeatedly engaging in shouting matches, children physically assaulting one another, constant

disregard of family rules and invasions of other family members' bound-
aries). Zanarini and Frankenburg (1997) estimated that approximately half
of borderline patients report a childhood characterized by type I and/or type
II trauma, and the remaining half of borderline patients report a childhood
characterized by all three types of trauma.

Attachment

Attachment theory (Bowlby, 1969, 1973) provides a framework for some
of the most important recent theory and research on the etiology of BPD (see
Agrawal, Gunderson, Holmes, & Lyons-Ruth, 2004, for a meta-analytic
review). Research on attachment in BPD focuses on an infant's or young
child's experience of unpredictable, frightening, and/or abusive caregiving
that interrupts the formation of coherent internal working models of
relationships. This presumably results in an inability to predict, understand,
and flexibly respond to the actions of significant others (Lyons-Ruth &
Jacobvitz, 1999; Main, Kaplan, & Cassidy, 1985). Attachment theorists
describe a child's attachment status as "disorganized-disoriented" when the
child is faced with an unsolvable dilemma: separation from a caregiver
causes distress; this activates proximity seeking; the caregiver is unavailable,
unpredictable, or frightening; and the infant or child is left without coherent
strategies for making sense of or obtaining security from the caregiver. This
disorganized-disoriented attachment pattern in infancy (similar to the char-
acterization of "unresolved with respect to loss and trauma" in adult attach-
ment) is marked in laboratory studies by incoherent and ineffective attempts
to self-regulate following a separation from a caregiver. Instead, disorga-
nized-disoriented infants demonstrate seemingly undirected or contradictory
behavior, such as freezing, rocking, or head banging.

Research on disorganized attachment in children (beyond infancy) high-
lights perceptions of parental figures as unpredictable, unavailable, and
frightening. In one series of studies, children with disorganized attachment
were more likely to respond to pictures of distressed children separated from
their parents with stories depicting violent harm to the child or others
(Kaplan, 1987; Main et al., 1985). In other studies, parents are described as
unavailable, frightening, or frightened (Solomon, George, & De Jong, 1995),
and dolls representing the child engage in angry/violent and idiosyncratic/
odd behavior (Cassidy, 1988). Interestingly, these findings parallel research
on adolescent and adult patients with BPD using storytelling procedures
such as the Thematic Apperception Test (TAT), which finds that BPD is
associated with negative emotional tone of relationship descriptions and par-
ticularly by malevolence attributions (Westen, 1991b; Westen, Lohr, Silk,
Gold, & Kerber, 1990; Westen, Ludolph, Block, et al., 1990).

Because "unresolved" is a qualifier rather than one of the three primary
attachment patterns coded categorically from the Adult Attachment Interview

(AAI), the most common attachment pattern associated with BPD is preoccupied (analogous to anxious/ambivalent in infancy and childhood). A combination of unresolved and preoccupied attachment has been associated with BPD in adolescents as well as adults (Nakash-Eisikovits, Dutra, & Westen, 2002; Westen, Thomas, Nakash, & Bradley, 2006). In general, preoccupied attachment in combination with the unresolved qualifier resembles the interpersonal style of BPD patients, marked by rejection sensitivity, alternation between anxious preoccupation and anger with attachment figures, and incoherent strategies for attempting to make intimate contact with others.

Recent work integrating object relations and attachment theories connects insecure or disorganized attachment to BPD symptoms using the concept of mentalization (Fonagy, Target, Gergely, Allen, & Bateman, 2003). Mentalization refers to the ability to make sense of one's own and others' actions by reflecting on and understanding their mental states (including feelings, beliefs, wishes, and ideas). In healthy development, this capacity is developed in the context of attachment relationships with primary caregivers, during which infants and children develop internal working models of self, others, and relationships (Bowlby, 1988). According to Fonagy, an inability to make sense of one's own and others' mental (and particular emotional) states not only results from interactions with inexplicable caregivers but also renders anticipation of attachment figures' actions impossible, leading to difficulty in self-regulating emotion.

Difficulties with mentalization can also be seen in an implicit or explicit belief in a one-to-one correspondence between one's perceptions (of situations, others' feelings and motives, etc.) and reality, and hence in an inability to consider possible alternate interpretations. This often leads to instability of interpersonal relationships, as whatever emotion one feels in reaction to others (e.g., anger, happiness) is perceived as directly and unquestionably reflective of the other's feelings or intentions (e.g., intent to harm), whether correctly or incorrectly perceived. According to this model, deficits in mentalization also contribute to an unstable sense of self and a sense of emptiness.

Interaction of Biological and Psychosocial Risk Factors

Despite the relatively neat categorization of putative risk factors presented above, research dating back to Harlow's monkeys should lead to circumspection in making distinct attributions to nature or nurture in the etiology of BPD or any other psychiatric disorder. What begins as a biological vulnerability may lead to a cascade of environmental events, just as what may begin as an environmental effect may become "hard-wired." Data on the interplay of risk factors in the development of BPD do not exist at this point, primarily because BPD is not officially diagnosed until age 18 and comprehensive longitudinal studies of the development of personality disorders beginning in infancy have yet to be conducted. However, a number of studies in domains related to BPD (e.g., childhood sexual and physical abuse,

attachment disorganization, impulsivity and depressed mood) have demon-strated that both psychological and biological influences play important roles (see Judd & McGlashan, 2003, for a review).

Two areas of research are particularly relevant to BPD. Caspi, Moffitt, and colleagues (Caspi et al., 2002; Caspi et al., 2003) have focused on gene-environment interactions in a large longitudinal sample in New Zealand. In a landmark study (2002), they found that a functional polymorphism in the promoter region of the serotonin transporter (5-HTT) gene moderated the influence of stressful life events in both childhood and adulthood on subse-quent depression. Stressful events in adulthood, as well as abuse in childhood, predicted subsequent depressive symptoms and suicidality—two features that in combination often point to the presence of BPD—in individuals with the short allele of the 5-HTT promoter as compared to individuals homozygous for the long allele. In a second study (2003), they found that a functional polymorphism in a gene regulating monoamine oxidase (MAO) moderated the relationship between child abuse and antisocial behavior in adulthood.

Research on the neurobiology of early life stress also highlights the impor-tance of the interaction of biological and environmental factors in the devel-opment of psychopathology. These studies suggest that early life stress modifies brain circuits involved in stress regulation, resulting in a type of "biological priming" that interacts with genetic vulnerabilities to increase the risk of later psychopathology (Heim, Meinlschmidt, & Nemeroff, 2003). Though none of the research to date directly addresses the development of BPD, the types of early life stress studied (notably early separation from mother in animal analogue studies and childhood sexual and physical abuse in human studies) as well as the domains of documented outcomes (e.g., depression and substance abuse) are germane to an understanding of the interaction of genetic and biological risk factors in BPD.

Prognosis and Natural Course of the Disorder

A small but growing body of research on the longitudinal course of BPD exists, and although the findings of these studies are not entirely consistent, several broad characterizations emerge. One is that patients tend to lose their BPD diagnosis over time. For example, the longitudinal McLean Adult Development Study found a remission rate of 35% at 2 years, 50% at 4 years, and 69% at 6 years (Zanarini, Frankenburg, Hennen, & Silk, 2003).

To what extent the instability in the borderline diagnosis (and other PD diagnoses) is an artifact of arbitrary cutoff points for categorical diagnosis, the mixed diagnostic criteria for BPD in the DSM (enduring personality characteristics interspersed with typically stress-dependent behaviors), or the limited test-retest reliability of structured interviews at intervals beyond 6 weeks is unclear. What *is* clear is that dimensional assessments of both BPD (number of criteria met) and traits associated with BPD (e.g., negative

affectivity, neuroticism) show far more temporal stability than categorical diagnoses (Lenzenweger, Johnson, & Willett, 2004; Skodol et al., 2005).

Although longer-term follow-up studies have the disadvantage of less structured diagnostic procedures, they provide additional data suggesting a course of general improvement if BPD patients can survive their 20s and 30s. In one long-term follow-up study, Paris and Zweig-Frank (2001) found a high remission rate consistent with more recent studies using structured interviews, with only 25% of patients still meeting BPD criteria at 15 years and 7.8% meeting criteria at 27 years. McGlashan (1986) found that BPD patients discharged from an intensive inpatient program fared best two decades following discharge. These data, consistent with other data on externalizing disorders such as antisocial PD and substance abuse disorders, suggest that BPD tends to "burn out" with age. In long-term follow-up studies by McGlashan (1986) and by Stone (1987; 1992), patients with better outcomes tended to be higher in intelligence, more talented in the arts, more physically attractive, and/or described as more likable than those with poorer outcomes. The more chronically impaired patients were more likely to have sustained problems with alcohol abuse, histories of severe physical or sexual abuse, severe problems with impulsivity, comorbidity for antisocial PD, or schizotypal features.

BPD, however, is marked by internalizing symptoms and interpersonal difficulties as well as the more notable externalizing symptoms. Data suggest that although the impulsive and aggressive (both other- and self-directed) features tend to improve with time, core personality attributes such as negative affect, emotion dysregulation, and difficulties in intimate relationships tend to persist, whether or not the person continues to meet formal criteria for BPD. Depressive and anxious symptoms tend to remain high even among "recovered" BPD patients. Likewise, problems related to interpersonal relationships, including social isolation and fear of abandonment, tend to endure over time (McGlashan, 1986; Paris & Zweig-Frank, 2001; Zanarini et al., 2003). There is some suggestion that long-term interpersonal patterns among BPD patients tend to be bimodal, with some patients tending to become socially isolated (likely as a way of regulating the intensely distressing interpersonal patterns that tend to exacerbate BPD symptoms), whereas others become better able to maintain committed relationships. Some patients become symptomatic again during midlife in response to separation, divorce, or death of a spouse (McGlashan, 1986; Paris & Zweig-Frank, 2001; Stone, 1987, 1992).

One of the major risks for BPD patients seen in these and other studies is suicide. Long-term studies of patients with BPD suggest a suicide rate in the range of 3% to 10% (Black, Blum, Pfohl, & Hale, 2004). Following patients over a 27-year period, Paris and Zweig-Frank (2001) found a 10.3% rate of suicide (most of which occurred before age 40). Two of the most robust variables predicting greater risk of suicide in BPD patients include substance abuse and comorbid depression, although the latter is characteristic of most BPD patients (Black et al., 2004; Fyer et al., 1988; Isometsa et al., 1996).

Treatment Approaches

Patients with BPD are often considered difficult to treat. The first treatments for borderline patients, which began to emerge in the 1950s and 1960s, were modified forms of psychoanalytic psychotherapy, two of which have recently been tested in randomized controlled trials (RCTs). Cognitive-behavioral therapies for BPD began to emerge in the 1980s, when Linehan developed dialectical behavior therapy (DBT; Linehan, 1993). Integrative therapies have not been tested empirically but are widely practiced. We describe each treatment approach in turn.

Psychodynamic Psychotherapy

Because of centrality of the construct of personality (or character) to its conceptions of treatment, and because of the legacy of early theories on borderline states and personality organization, psychodynamic approaches constitute the largest body of theoretical work on the treatment of BPD. Psychodynamic approaches to BPD all share a grounding in developmental psychopathology, arguing that the only way to understand the disorder is through understanding the way development has gone awry in the way the patient experiences the self and others, regulates emotions, and regulates impulses (see Bradley & Westen, 2005). Although psychodynamic psychotherapy for BPD varies widely, two approaches have predominated in the clinical literature. The first is Kernberg's (1975) approach, which represents a confluence of multiple psychoanalytic schools of thought, particularly ego psychology (which focuses on adaptive functions) and object relations theory (which focuses on interpersonal relationships and the representation of self and others). The second, derived from the self-psychological approach of Kohut (1977), is based on work by Adler and Buie (Adler, 1981, 1989; Adler & Buie, 1979; Buie & Adler, 1982), which focuses on identity and self-soothing.

Similar to all psychodynamic approaches to the treatment of BPD, Kernberg's approach attends to the interpersonal dynamics of BPD patients (e.g., fear of abandonment) and uses the relationship with the therapist to address distorted ways of understanding interpersonal relations. More specifically, Kernberg's perspective focuses on the importance of aggression in borderline patients (often projected onto others) and a defensive style marked by "splitting" (a tendency to see the self and others as all good or all bad). Kernberg's treatment focuses on confronting aggression and manipulation, helping patients attain more balanced views of the self and others, and interpreting conflicts impeding the capacity to love and work.

The self-psychological approach of Adler, Buie, and others assumes that borderline patients' problems lie less in their conflicts than in psychological deficits, particularly in their capacity to self-soothe. Whereas Kernberg tends to see the aggression of borderline patients as primary, the self-psychological approach views borderline patients' rage as secondary to other feelings, such

as the pain of abandonment. From this perspective, BPD is a developmental disorder derived from a failure to develop soothing images of primary caregivers that the person can call upon in times of distress. Thus the primary problem facing patients with BPD is the inability to self-soothe and the need to find others who can help them regulate their feelings as well as their self-esteem. This approach to treatment emphasizes empathic attunement with borderline patients and works to help them internalize soothing functions not developed in childhood.

Like most psychodynamic therapies, the ratio of theory to empirical outcome research is unfortunately high. However, two psychodynamic approaches to the treatment of BPD have recently been manualized (using principle-based manuals) and tested in RCTs. The first, based on Kernberg's work, is called transference-focused psychotherapy (TFP). The second, based on Fonagy's attachment research, is called mentalization-based treatment (MBT). Both share a primary focus on changing patients' mental representations of themselves and others.

Transference-Focused Psychotherapy

Transference-focused psychotherapy (Clarkin, Yeomans, & Kernberg, 1999; Yeomans, 2004) places primary emphasis on the poorly integrated representations that empirically are characteristic of patients with BPD (Westen, 1991b; Westen & Shedler, 1999a; Zittel & Westen, 2005). The treatment proceeds through a hierarchy of goals, moving from containment of suicidal and self-destructive behavior and establishment of a stable treatment frame to a focus on dominant relationship patterns. As the name implies, TFP focuses on clarification, confrontation, and interpretation within the context of the patient-therapist relationship. TFP focuses on present-oriented identification of the dominant relationship paradigm (e.g., idealizer-idealized, victim-victimizer) active in the patient-therapist relationship. This process includes observing and interpreting changes in this relational configuration (e.g., a switch in role from victim to victimizer or perpetrator) and increasing patient awareness of split representations of self and others.

The principle underlying TFP is that increased awareness and understanding of distortions and expectations the patient brings to relationships will lead to more coherent, integrated views of the self and others, which will in turn generate an increased ability to regulate emotions, particularly those emerging from interpersonal interactions. A preliminary study of TFP (Clarkin et al., 2001) evaluated 23 female patients in twice-weekly TFP over the course of 12 months. Examining pre-to-post change, the study found significantly reduced levels of suicide attempts, decreased severity of injury resulting from self-harming behavior, and fewer days and numbers of hospitalizations. A randomized controlled trial of TFP comparing it with supportive psychodynamic therapy (treatment as usual at Kernberg's site) and DBT has recently been completed (Clarkin, Levy, Lenzenweger, & Kernberg,

2004). Although all three treatments produced positive outcomes, of interest is that the hypothesized mediating variable (changes in the structure of representations of self and others) appeared linked to change only in the TFP condition.

Mentalization-Based Therapy

Mentalization-based therapy is a relatively recent effort to operationalize an approach to treatment grounded in attachment theory (Fonagy, Target, & Gergely, 2000). This approach focuses on developing increased mentalization capacities in BPD patients. One of the aims of the treatment approach is to help patients identify and understand emotions by clarifying and naming them, understanding immediate precipitants, understanding the emotion in the context of past and current relationships, learning to express the emotion appropriately, and learning to understand the response others are most likely to have in reaction to the patient's emotional expression (Bateman & Fonagy, 2003). The therapist maintains a "mentalizing stance" by focusing on and discussing the here-and-now mental states of the therapist and patient. Transference interpretations are kept simple and made with respect to relatively immediate or "experience near" circumstances (e.g., the patient's tendency to quit psychotherapy when she begins to feel too close to the therapist), avoiding historical interpretations (e.g., how this is related to her early experiences with her mother).

A preliminary study of MBT (Bateman & Fonagy, 1999) compared a control group ($n = 19$) of patients receiving "general psychiatric services" with patients ($n = 19$) participating in a psychoanalytically oriented partial hospitalization program based on an MBT approach. The maximum length of the partial hospitalization program was 18 months. All patients were assessed at 3-month intervals over an 18-month period. Results indicated decreased self-mutilation and suicide attempts; reduced length of inpatient hospitalization; and decreases in self-reported anxiety, depression, and interpersonal problems. Data collected at 18-month follow-up (e.g., 36 months from start of treatment) found that these treatment benefits were maintained (Bateman & Fonagy, 2001). These promising but preliminary data led to an outpatient adaptation of MBT with a more clearly operationalized treatment manual (Bateman & Fonagy, 2004), with a treatment study currently under way.

Cognitive-Behavioral Therapy (CBT)

The first and best studied CBT approach to treatment of BPD is DBT, a modular, manualized treatment program for patients with BPD that may be implemented in inpatient or outpatient settings. DBT relies on a combination of skills training, usually implemented in group format, in four areas: mindfulness, interpersonal effectiveness, distress tolerance, and emotion regulation.

In addition to skills training in a group format, DBT includes an individual therapy component with several distinctive features. These include a functional analysis of behavior (focusing particularly on self-harming and therapy interfering behaviors); a "validating" approach focused on depathologizing the patient's difficulties and emphasizing how impulsive and self-harming behaviors such as parasuicidal behavior are understandable, albeit not effective, efforts to manage distress or emotion dysregulation; and 24-hour therapist availability for suicidality coupled with behavioral principles intended to mitigate the need for such phone calls.

DBT is the only treatment for BPD that has been widely subjected to empirical scrutiny, and it has proven effective in decreasing suicide attempts, self-injurious behaviors (e.g., cutting, binge eating), and hospitalizations (Koons et al., 2001; Linehan, Armstrong, Suarez, Allmon, & Heard, 1991; Linehan, Heard, & Armstrong, 1993; Verheul et al., 2003). Other studies have found changes in a variety of domains, such as anger and dissociation (Linehan, Tutek, Heard, & Armstrong, 1994). A full review of studies on DBT is beyond the scope of this chapter (for reviews, see Robins & Chapman, 2004; Scheel, 2000; Westen, 2000). More recently, Young (Young, Klosko, & Weishaar, 2003) and Beck, Davis, and Freeman (2004) have developed more cognitive approaches to the treatment of PDs, including BPD. A recent study of long-term treatment using Young's schema-focused therapy produced promising results (Giesen-Bloo, et al., 2006).

Integrative Treatment

The three treatments described above represent only the most broadly known and best-evaluated approaches. A number of other psychodynamic, cognitive-behavioral, and integrative approaches to the treatment of BPD also exist but are beyond the constraints of this chapter (see Aviram, Hellerstein, Gerson, & Stanley, 2004; Blum, Pfohl, St. John, Monahan, & Black, 2002; Brown, Newman, Charlesworth, Crits-Christoph, & Beck, 2004; Ryle, 2004; Westen, 1991a, 2000). Some common elements, however, cut across most of these treatments for BPD, and make considerable sense in light of the nature of borderline psychopathology.

The first is the importance of establishing a clear framework for the treatment that spells out expectations and boundaries for both the therapist and the patient. A second is frequency and length of treatment, with each approach including some form of biweekly contact over the course of at least twelve months. Third, all of the treatment approaches for BPD attend closely to the dynamics of the relationship between the therapist and the patient and make discussion of this relationship a central aspect of therapy. Fourth, although the construct is framed differently, emotion and impulse dysregulation is at the center of virtually all approaches.

Fifth and finally, treatments tend to proceed through a series of stages, similar to those proposed in a stage-based approach to the treatment of the

near-neighbor construct of "complex posttraumatic stress disorder" (e.g., Brown, Scheflin, & Hammond, 1998; Herman, 1992). The first stage, linked to keeping the patient alive and in treatment, focuses on stabilization of behavior and reduction of emotion dysregulation (e.g., getting impulsive self-harmful behaviors under control). The second stage focuses on understanding of past experiences with a focus on how prior life experiences are manifest in current patterns (as opposed to exploration of the past as an archaeological dig, as in more classic psychoanalytic approaches). The third stage addresses reorganization of both internal (representational and affect-regulatory) and external (behavioral) processes related to interpersonal relationships. Although these principles remain untested, current funding priorities in the United States and Britain, where most of the treatment research on BPD has been conducted, do not encourage the testing of treatments of a duration (i.e., years) that virtually all experts on BPD, from Kernberg to Linehan, indicate are necessary for adequate, effective treatment of the disorder. In practice, however, we suspect that effective treatment of BPD likely requires flexibility and integration across treatment approaches, particularly given the mélange of personality problems and Axis I symptoms with which BPD patients typically present.

Future Directions for the Study of BPD

Research on BPD has expanded exponentially over the last 25 years. Here we briefly describe three domains we believe to be central to progress in research on BPD: identification of improved diagnostic criteria for BPD, approaches to BPD diagnosis, and exploration of potential subtypes of BPD.

How Can We Improve the Diagnostic Criteria for BPD?

Despite consensus on the problems with the current BPD diagnostic criteria, no such consensus exists on the best remedy. The least radical solution would be to continue with diagnostic business as usual by tinkering with the current diagnostic criteria to create a modestly improved criterion set (e.g., modifying the affective lability criterion to include both pervasive negative affect and affect dysregulation). This approach, however, has not solved the problems with the BPD diagnosis over the last 25 years, and it is unlikely that any set of 7 to 10 items will adequately capture this complex, multifaceted disorder while distinguishing it from near-neighbor disorders.

An alternative, more radical approach is a construct validation approach, which would take a large group of candidate criteria; collect data from a large, diverse sample without assuming any preexisting diagnostic groupings; and use statistical aggregation techniques to identify and validate emergent traits or configurations of traits. Practically speaking, it is unlikely that

the field will allow for elimination of the current BPD diagnosis (presuming this would be the result of full-scale construct validation). Although some resistance to wholesale reworking of the BPD diagnosis no doubt results from inertia and the comfort with the familiar (not to mention the time and money spent developing assessments for the current diagnosis), some rests on the fact that BPD is a clinically useful construct backed by a large body of research. Data of this sort collected with the use of self-report instruments have allowed researchers to construct a BPD prototype from the traits represented in the five-factor model (Trull, Widiger, Lynam, & Costa, 2003), and similar data have identified both a trait and a personality constellation heavily marked by borderline features, called emotion dysregulation or emotionally dysregulated PD, in both adolescents and adults (Shedler & Westen, 2004; Westen, Dutra, & Shedler, 2005; Westen & Shedler, 1999b; Westen, Shedler, Durrett, Glass, & Martens, 2003).

A compromise between business as usual and radical overhaul would be a hybrid, or "construct validation lite," approach. This procedure would involve selecting a group of patients with a moderate to high degree of match to the current BPD diagnosis and then identifying their most salient personality characteristics from an item pool that includes but is not limited to DSM-IV criteria. Using a broad range of both patients and candidate criteria would allow for a more comprehensive description of personality features of BPD without "throwing the baby out with the bathwater."

How Should We Diagnose BPD?

Regardless of the criteria ultimately used to diagnose BPD, a second question regarding diagnosis remains, namely, how diagnostic criteria should be applied to individual cases. As noted by Sokal (1974), taxonomy (developing a classification) and diagnosis (identifying cases) are independent aspects of the classification process. Researchers have proposed several alternatives to the current DSM procedure of making categorical (yes/no) decisions on each diagnostic criterion, counting the number of criteria met, and applying arbitrary cutoffs. The least radical is simply to use the same procedure except to dimensionalize it, using number of symptoms met as a dimensional diagnosis, perhaps supplemented by categorical diagnosis for clinical communication. A second, less conservative method is a prototype matching approach (see Westen & Bradley, 2005; Westen & Shedler, 2000; Westen et al., 2006), in which clinicians rate the resemblance between the patient and a diagnostic prototype (e.g., in the form of a paragraph descriptive of a prototypical patient with the disorder). A recently completed study (unpublished data) finds that clinicians can make dimensional diagnoses using prototype matching with a simple five-point scale (1 = description does not apply; 2 = patient has *some features* of this disorder; 3 = patient has *significant features* of this disorder; 4 = patient *has* this disorder, diagnosis applies; 5 = patient *exemplifies* this disorder, prototypical case) with

high reliability. Other research finds that this prototype matching approach decreases diagnostic comorbidity when compared with dimensional diagnoses made by counting symptoms, with slight improvements, rather than decrements, in construct validity (Westen et al., 2006). Finally, the most radical approach would be to eliminate the BPD diagnosis and replace it with a four- or five-factor trait diagnosis, and to "reconstruct" the BPD diagnosis if necessary using four or five factor prototypes as described by Widiger and his colleagues (Trull et al., 2003). To what extent one of these approaches is more empirically valid and clinically useful than the others will require head-to-head comparisons, in which multiple approaches are all tested in the same data set. Unfortunately, to date, research has typically tested each approach in isolation or in comparison with only the current DSM-IV approach.

Do Subtypes of BPD Exist?

The heterogeneity in the clinical presentations that can yield a BPD diagnosis raises the question of whether the diagnosis may include meaningful subtypes or subgroups. Grinker, Werble, and Drye (1968), who undertook the first empirical study of borderline pathology, conducted the first research identifying subtypes of BPD. Using a sample of 51 psychiatric inpatients, they identified four groups of BPD patients: a more psychotic group (which later influenced the schizotypal diagnosis in DSM-III), a more neurotic group, a "core" borderline group, and an "as-if" (identity-changing) group. Theorizing about subgroups of borderline patients rests primarily on clinical observation (e.g., Oldham, 2001; Stone, 1994). However, recent research using the SWAP-200 and SWAP-II has consistently produced two- and three-cluster solutions in DSM-IV–defined adults and adolescents with BPD (Bradley, Zittel, et al., 2005; Conklin, Bradley, & Westen, 2006; Conklin & Westen, 2005; Westen & Shedler, 1999b; Zittel & Westen, 2002). The first two subtypes have been replicated across all samples. The first is an internalizing dysregulated subtype marked by severe dysphoria and desperate efforts to manage it (e.g., through cutting or suicide attempts). The second is an externalizing dysregulated subgroup marked by a tendency to be rageful rather than depressed and to try to self-regulate by blaming or attacking others instead of oneself. The third is a histrionic-impulsive subtype marked by a tendency to experience both intense positive and intense negative emotions and attempts to regulate both positive and negative affect through impulsive and sensation-seeking behavior. Across both adolescent and adult samples, these subtypes have demonstrated meaningful differences with respect to external correlates indicative of construct validity, such as adaptive functioning and etiology. Moreover, although the DSM criteria may capture many aspects of the externalizing dysregulated subtype, the intense pain manifest in the internalizing subtype is not captured by the diagnostic criteria.

Conclusion

Borderline personality disorder is a complex disorder—or, more likely, spectrum of pathology—whose phenomenology, etiology, prognosis, and treatment researchers have made great strides in understanding since its official introduction into the psychiatric nomenclature in 1980 in DSM-III. Nevertheless, the diagnosis itself is clearly in need of revision to minimize artifactual comorbidity with the majority of the other nine PDs in DSM-IV and to maximize both its construct validity and clinical utility.

References

Adler, G. (1981). The borderline-narcissistic personality disorder continuum. *American Journal of Psychiatry, 138,* 46–50.

Adler, G. (1989). Uses and limitations of Kohut's self psychology in the treatment of borderline patients. *Journal of the American Psychoanalytic Association, 37,* 761–785.

Adler, G., & Buie, D. (1979). Aloneness and borderline psychopathology: The possible relevance of child development issues. *International Journal of Psychoanalysis, 60,* 83–96.

Agrawal, H. R., Gunderson, J. G., Holmes, B. M., & Lyons-Ruth, K. (2004). Attachment studies with borderline patients: A review. *Harvard Review of Psychiatry, 12,* 94–104.

Akiskal, H. S. (1996). The prevalent clinical spectrum of bipolar disorders: Beyond DSM-IV. *Journal of Clinical Psychopharmacology, 16,* 4S–14S.

Akiskal, H. S. (2004). Demystifying borderline personality: Critique of the concept and unorthodox reflections on its natural kinship with the bipolar spectrum. *Acta Psychiatrica Scandinavica, 110,* 401–407.

Akiskal, H. S., Chen, S. E., Davis, G. C., Puzantian, V. R., Kashgarian, M., & Bolinger, J. M. (1985). Borderline: An adjective in search of a noun. *Journal of Clinical Psychiatry, 46,* 41–48.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

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

Aviram, R. B., Hellerstein, D. J., Gerson, J., & Stanley, B. (2004). Adapting supportive psychotherapy for individuals with borderline personality disorder who self-injure or attempt suicide. *Journal of Psychiatric Practice, 10,* 145–155.

Barasch, J., Kroll, J., Carey, K., & Sines, L. (1983). Discriminating borderline personality disorder from other personality disorders: Cluster and analysis of the Diagnostic Interview for Borderline. *Archives of General Psychiatry, 40,* 1297–1302.

Bateman, A., & Fonagy, P. (1999). The effectiveness of partial hospitalization in the treatment of borderline personality disorder: A randomized controlled trial. *American Journal of Psychiatry, 156,* 1563–1569.

Bateman, A., & Fonagy, P. (2001). Treatment of borderline personality disorder with psychoanalytically oriented partial hospitalization: An 18-month follow-up. *American Journal of Psychiatry, 158,* 36–41.

Bateman, A., & Fonagy, P. (2003). The development of an attachment-based treat-
ment program for borderline personality disorder. *Bulletin of the Menninger
Clinic, 67,* 187–211.

Bateman, A., & Fonagy, P. (2004). Mentalization-based treatment of BPD. *Journal
of Personality Disorders, 18,* 36–51.

Bazanis, E., Rogers, R. D., Dowson, J. H., Taylor, P., Meux, C., Staley, C., et al. (2002).
Neurocognitive deficits in decision-making and planning of patients with DSM-
III-R borderline personality disorder. *Psychological Medicine, 32,* 1395–1405.

Beck, A. T., Davis, D. D., & Freeman, A. (2004). *Cognitive therapy of personality
disorders* (2nd ed.). New York: Guilford Press.

Becker, D. F., Grilo, C. M., Edell, W. S., & McGlashan, T. H. (2000). Comorbidity
of borderline personality disorder with other personality disorders in hospital-
ized adolescents and adults. *American Journal of Psychiatry, 157,* 2011–2016.

Black, D. W., Blum, N., Pfohl, B., & Hale, N. (2004). Suicidal behavior in border-
line personality disorder: Prevalence, risk factors, prediction, and prevention.
Journal of Personality Disorders, 18(3), 226–239.

Blum, N., Pfohl, B., St. John, D., Monahan, P., & Black, D. W. (2002). A cognitive-
behavioral systems-based group treatment for outpatients with borderline
personality disorder: A preliminary report. *Comprehensive Psychiatry, 43,* 301–310.

Bowlby, J. (1969). *Attachment* (Vol. 1). New York: Basic Books.

Bowlby, J. (1973). *Separation* (Vol. 2). London: Hogarth Press.

Bowlby, J. (1988). *A secure base: Parent-child attachment and healthy human devel-
opment.* New York: Basic Books.

Bradley, R., Jenei, J., & Westen, D. (2005). Etiology of borderline personality disor-
der: Disentangling the contributions of intercorrelated antecedents. *Journal of
Nervous and Mental Disease, 193,* 24–31.

Bradley, R., & Westen, D. (2005). The psychodynamics of borderline personality disorder:
A view from developmental psychopathology. *Development and Psychopathology,
17*(4), 927–957.

Bradley, R., Zittel, C., & Westen, D. (2005). Borderline personality disorder in adoles-
cence: Phenomenology and subtypes. *Journal of Child Psychology and Psychiatry,
46,* 1006–1019.

Bradley, S. J. (1979). The relationship of early maternal separation to borderline
personality in children and adolescents: A pilot study. *American Journal of
Psychiatry, 136,* 424–426.

Brown, D., Scheflin, A. W., & Hammond, D. C. (1998). *Memory, trauma treatment,
and the law.* New York: W.W. Norton.

Brown, G. K., Newman, C. F., Charlesworth, S. E., Crits-Christoph, P., & Beck,
A. T. (2004). An open clinical trial of cognitive therapy for borderline personal-
ity disorder. *Journal of Personality Disorders, 18,* 257–271.

Brown, G. W., & Harris, T. O. (1989). *Life events and illness.* New York: Guilford Press.

Buie, D. H., & Adler, G. (1982). Definitive treatment of the borderline personality.
International Journal of Psychoanalytic Psychotherapy, 9, 51–87.

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

Cassidy, J. (1988). Child-mother attachment and the self in six-year-olds. *Child
Development, 59,* 121–134.

Clark, L. A., & Harrison, J. A. (2001). Assessment instruments. In W. J. Livesley (Ed.), *Handbook of personality disorders: Theory, research, and treatment.* New York: Guilford Press.

Clark, L. A., Livesley, W. J., & Morey, L. (1997). Personality disorder assessment: The challenge of construct validity. *Journal of Personality Disorders, 11,* 205–231.

Clarkin, J. F., Foelsch, P. A., Levy, K. N., Hull, J. W., Delaney, J. C., & Kernberg, O. F. (2001). The development of a psychodynamic treatment for patients with borderline personality disorder: A preliminary study of behavioral change. *Journal of Personality Disorders, 15,* 487–495.

Clarkin, J. F., Levy, K. N., Lenzenweger, M. F., & Kernberg, O. F. (2004). The Personality Disorders Institute/Borderline Personality Disorder Research Foundation randomized control trial for borderline personality disorder: Rationale, methods, and patient characteristics. *Journal of Personality Disorders, 18,* 52–72.

Clarkin, J. F., Widiger, T. A., Frances, A., Hurt, S. W., & Gilmore, M. (1983). Prototypic typology and the borderline personality disorder. *Journal of Abnormal Psychology, 92,* 263–275.

Clarkin, J. F., Yeomans, F. E., & Kernberg, O. F. (1999). *Psychotherapy for borderline personality.* New York: Wiley.

Clifton, A., Turkheimer, E., & Oltmanns, T. F. (2003). *Self and peer perspectives on pathological personality traits and interpersonal problems.* Unpublished manuscript, University of Virginia, Charlottesville.

Coccaro, E. F., Bergeman, C., & McClearn, G. E. (1993). Heritability of irritable impulsiveness: A study of twins reared together and apart. *Psychiatry Research, 48*(3), 229–242.

Conklin, C. Z., & Westen, D. (2005). Borderline personality disorder as seen in clinical practice: Implications for DSM-V. *American Journal of Psychiatry, 162,* 867–875.

Conklin, C. Z., Bradley, R., & Westen, D. (2006). Affect regulation in borderline personality disorder. *Journal of Nervous and Mental Disease, 194*(2), 69–77.

Cowdry, R. (1992). *Psychobiology and psychopharmacology of borderline personality disorder.* Madison, CT: International Universities Press.

Dahl, A. (1995). Borderline disorders: The validity of the diagnostic concept. *Psychiatric Development, 3,* 109–152.

De La Fuente, J. M., Goldman, S., Stanus, E., Vizuete, C., Morlan, I., Bobes, J., et al. (1997). Brain glucose metabolism in borderline personality disorder. *Journal of Psychiatric Research, 31,* 531–541.

Donegan, N. H., Sanislow, C. A., Blumberg, H. P., Fulbright, R. K., Lacadie, C., Skudlarski, P., et al. (2003). Amygdala hyperreactivity in borderline personality disorder: Implications for emotional dysregulation. *Biological Psychiatry, 54*(11), 1284–1293.

Dong, M., Anda, R. F., Felitti, V. J., Dube, S. R., Williamson, D. F., Thompson, T. J., et al. (2004). The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect, 28,* 771–784.

Driessen, M., Herrmann, J., Stahl, K., Zwaan, M., Meier, S., Hill, A., et al. (2000). Magnetic resonance imaging volumes of the hippocampus and the amygdala in women with borderline personality disorder and early traumatization. *Archives of General Psychiatry, 57,* 1115–1122.

Edwards, V. J., Holden, G. W., Anda, R. F., & Felitti, V. J. (2003). Experiencing mul-
tiple forms of childhood maltreatment and adult mental health: Results from the
Adverse Childhood Experiences (ACE) study. *American Journal of Psychiatry,
160,* 1453–1460.

Ellison, J., Barsky, A., & Blum, N. R. (1989). Frequent repeaters to an emergency
service. *Hospital & Community Psychiatry, 40,* 958–960.

Fiedler, E., Oltmanns, T., & Turkheimer, E. (2004). Traits associated with person-
ality disorders and adjustment to military life: Predictive validity of self and peer
reports. *Military Medicine, 169,* 32–40.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1997). *Structured clin-
ical interview for DSM-IV personality disorders (SCID-II).* Washington, DC:
American Psychiatric Press.

Fonagy, P., Target, M., & Gergely, G. (2000). Attachment and borderline personal-
ity disorder. *Psychiatric Clinics of North America, 23,* 103–122.

Fonagy, P., Target, M., Gergely, G., Allen, J. G., & Bateman, A. (2003). The devel-
opmental roots of borderline personality disorder in early attachment relation-
ships: A theory and some evidence. *Psychoanalytic Inquiry, 23,* 412–459.

Forman, E. M., Berk, M. S., Henriques, G. R., Brown, G. K., & Beck, A. T. (2004).
History of multiple suicide attempts as a behavioral marker of severe psy-
chopathology. *American Journal of Psychiatry, 161,* 437–443.

Fossati, A., Madeddu, F., & Maffei, C. (1999). Borderline personality disorder and
childhood sexual abuse: A meta-analytic study. *Journal of Personality Disorders,
13*(3), 268–280.

Frank, H., & Paris, J. (1981). Recollections of family experience in borderline
patients. *Archives of General Psychiatry, 38,* 1031–1034.

Fyer, M., Frances, A., Sullivan, T., Hurt, S., & Clarkin, J. (1988). Comorbidity of
borderline personality disorder. *Archives of General Psychiatry, 45,* 348–352.

Giesen-Bloo, J., van Dyck, R., Spinhoven, P., van Tilburg, W., Dirksen, C., van
Asselt, T., et al. (2006). Outpatient psychotherapy for borderline personality
disorder: Randomized trial of schema-focused therapy vs. transference-focused
psychotherapy. *Archives of General Psychiatry, 63,* 649–658.

Goldberg, R. L., Mann, L. S., Wise, T. N., & Segall, E. A. (1985). Parental qualities
as perceived by borderline personality disorders. *Hillside Journal of Clinical
Psychiatry, 7,* 134–140.

Goodman, M., & Yehuda, R. (2002). The relationship between psychological
trauma and borderline personality disorder. *Psychiatric Annals, 32*(6), 337–345.

Grilo, C. M., Shea, M. T., Sanislow, C. A., Skodol, A. E., Gunderson, J. G., Stout,
R. L., et al. (2004). Two-year stability and change of schizotypal, borderline,
avoidant, and obsessive-compulsive personality disorders. *Journal of Consulting
and Clinical Psychology, 72*(5), 767–775.

Grinker, R. R., Werble, B., & Drye, R. (1968). *The borderline syndrome: A behav-
ioral study of ego functions.* New York: Basic Books.

Gross, R., Olfson, M., Gameroff, M., Shea, S., Feder, A., Fuentes, M., et al. (2002).
Borderline personality disorder in primary care. *Archives of Internal Medicine,
162,* 53–60.

Gunderson, J. G. (2001). *Borderline personality disorder: A clinical guide.*
Washington, DC: American Psychiatric Publishing.

Gunderson, J. G., Kerr, J., & Englund, D. W. (1980). The families of borderlines: A
comparative study. *Archives of General Psychiatry, 37,* 27–33.

Gunderson, J. G., & Kolb, J. E. (1978). Discriminating features of borderline patients. *American Journal of Psychiatry, 135,* 792–796.

Gunderson, J. G., Kolb, J. E., & Austin, V. (1981). The diagnostic interview for borderline patients. *American Journal of Psychiatry, 138,* 896–903.

Gunderson, J. G., & Phillips, K. A. (1991). A current view of the interface between borderline personality disorder and depression. *American Journal of Psychiatry, 148*(8), 967–975.

Gunderson, J. G., & Sabo, A. N. (1993). "Borderline personality disorder and PTSD": Reply. *American Journal of Psychiatry, 150*(12), 1906–1907.

Gunderson, J. G., & Singer, M. T. (1975). Defining borderline patients: An overview. *American Journal of Psychiatry, 132,* 1–10.

Gunderson, J. G., Zanarini, M. C., & Kisiel, C. (1991). Borderline personality disorder: A review of data on DSM-III-R descriptions. *Journal of Personality Disorders, 5,* 340–352.

Gunderson, J. G., Zanarini, M. C., & Kisiel, C. (1995). Borderline personality disorder. In W. J. Livesley (Ed.), *The DSM-IV personality disorders* (pp. 141–157). New York: Guilford Press.

Heim, C., Meinlschmidt, G., & Nemeroff, C. B. (2003). Neurobiology of early-life stress. *Psychiatric Annals, 33,* 18–26.

Herman, J. L. (1992). Complex PTSD: A syndrome in survivors of prolonged and repeated trauma. *Journal of Traumatic Stress, 5,* 341–377.

Herman, J. L., Perry, J., & van der Kolk, B. A. (1989). Childhood trauma in borderline personality disorder. *American Journal of Psychiatry, 146*(4), 490–495.

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*(4), 292–298.

Isometsa, E. T., Henriksson, M. M., Heikkinen, M. E., Aro, H. M., Marttunen, M. J., Kuoppasalmi, K. I., et al. (1996). Suicide among subjects with personality disorders. *American Journal of Psychiatry, 153*(5), 667–673.

Johnson, J. G., Cohen, P., Brown, J., Smailes, E., & Bernstein, D. P. (1999). Childhood maltreatment increases risk for personality disorders during early adulthood. *Archives of General Psychiatry, 56*(7), 600–606.

Johnson, J. G., Cohen, P., Smailes, E., Skodol, A. E., Brown, J., & Oldham, J. M. (2001). Childhood verbal abuse and risk for personality disorders during adolescence and early adulthood. *Comprehensive Psychiatry, 42,* 16–23.

Judd, P. H., & McGlashan, T. (2003). *A developmental model of borderline personality disorder: Understanding variations in course and outcome.* Arlington, VA: American Psychiatric Publishing.

Kaplan, C. J. (1987). *Children's responses to the loss of a parent: The interaction between the family and the intrapsychic mourning process.* Unpublished manuscript, University of Massachusetts, Amherst.

Kernberg, O. (1967). Borderline personality organization. *Journal of the American Psychoanalytic Association, 15,* 641–685.

Kernberg, O. (1975). *Borderline conditions and pathological narcissism.* Northvale, NJ: Jason Aronson.

Klein, D. N. (2003). Patients' versus informants' reports of personality disorders in predicting 7½-year outcome in outpatients with depressive disorders. *Psychological Assessment, 15,* 216–222.

Klonsky, E. D., Oltmanns, T. F., & Turkheimer, E. (2002). Informant-reports of per-sonality disorder: Relation to self-reports and future research directions. *Clinical Psychology: Science and Practice, 9,* 300–311.

Knight, R. P. (1953). Borderline states. *Bulletin of the Menninger Clinic, 17,* 1–12.

Knight, R. P. (1954). *Psychoanalytic psychiatry and psychology.* In clinical and the-oretical papers from the Austen Riggs Center, Vol.1.

Kohut, H. (1977). *The restoration of the self.* Madison, CT: International Universities Press.

Koons, C. R., Robins, C. J., Tweed, J., Lynch, T. R., Gonzalez, A. M., Morse, J. Q., et al. (2001). Efficacy of dialectical behavior therapy in women veterans with borderline personality disorder. *Behavior Therapy, 32*(2), 371–390.

Lenzenweger, M. F., Clarkin, J. F., Fertuck, E. A., & Kernberg, O. F. (2004). Executive neurocognitive functioning and neurobehavioral systems indicators in borderline personality disorder: A preliminary study. *Journal of Personality Disorders, 18*(5), 421–438.

Lenzenweger, M. F., Johnson, M. D., & Willett, J. B. (2004). Individual growth curve analysis illuminates stability and change in personality disorder features: The longitudinal study of personality disorders. *Archives of General Psychiatry, 61*(10), 1015–1024.

Leyton, M., Okazawa, H., Diksic, M., Paris, J., Rosa, P., Mzengeza, S., et al. (2001). Brain regional alpha-[11C]methyl-L-tryptophan trapping in impulsive subjects with borderline personality disorder. *American Journal of Psychiatry, 158*(5), 775–782.

Linehan, M. M. (1993). *Skills-training manual for treatment of borderline personal-ity disorder.* New York: Guilford Press.

Linehan, M. M., Armstrong, H. E., Suarez, A., Allmon, D., & Heard, H. L. (1991). Cognitive-behavioral treatment of chronically parasuicidal borderline patients. *Archives of General Psychiatry, 48,* 1060–1064.

Linehan, M. M., Heard, H. L., & Armstrong, H. E. (1993). Naturalistic follow-up of a behavioral treatment for chronically parasuicidal borderline patients. *Archives of General Psychiatry, 50,* 971–974.

Linehan, M. M., Tutek, D., Heard, H., & Armstrong, H. (1994). Interpersonal out-come of cognitive-behavioral treatment for chronically suicidal borderline patients. *American Journal of Psychiatry, 151*(12), 1771–1776.

Links, P. S., Steiner, M., Offord, D. R., & Eppel, A. B. (1988). Characteristics of bor-derline personality disorder: A Canadian study. *Canadian Journal of Psychiatry, 33,* 336–340.

Ludolph, P., Westen, D., Misle, B., Jackson, A., Wixom, J., & Weiss, F. C. (1990). The borderline diagnosis in adolescents: Symptoms and developmental history. *American Journal of Psychiatry, 147,* 470–476.

Lynam, D. R., & Widiger, T. A. (2001). Using the five-factor model to represent the DSM-IV personality disorders: An expert consensus approach. *Journal of Abnormal Psychology, 110*(3), 401–412.

Lyons-Ruth, K., & Jacobvitz, D. (1999). Attachment disorganization: Unresolved loss, relational violence, and lapses in behavioral and attentional strategies. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research and clinical applications* (pp. 550–554). New York: Guilford Press.

Main, M., Kaplan, N., & Cassidy, J. (1985). Security in infancy, childhood, and adulthood: A move to the level of representation. *Monographs of the Society for Research in Child Development, 50*(1–2), 66–104.

Mann, J. (1998). The role of in vivo neurotransmitter system imaging studies in understanding major depression. *Biological Psychiatry, 44*(11), 1077–1078.

McGlashan, T. H. (1986). The Chestnut Lodge follow-up study: III. Long-term outcome of borderline personalities. *Archives of General Psychiatry, 43,* 20–30.

McLean, L. M., & Gallop, R. (2003). Implications of childhood sexual abuse for adult borderline personality disorder and complex posttraumatic stress disorder. *American Journal of Psychiatry, 160,* 369–371.

Meehl, P. E. (1995). Bootstraps taxometrics: Solving the classification problem in psychopathology. *American Psychologist, 50*(4), 266–275.

Mellsop, G., Varghese, F., Joshua, S., & Hicks, A. (1982). The reliability and Axis II of DSM-III. *American Journal of Psychiatry, 139,* 1366–1367.

Nakash-Eisikovits, O., Dutra, L., & Westen, D. (2002). Relationship between attachment patterns and personality pathology in adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 41,* 1111–1123.

Nigg, J. T., & Goldsmith, H. (1994). Genetics of personality disorders: Perspectives from personality and psychopathology research. *Psychological Bulletin, 115*(3), 346–380.

Nigg, J. T., Lohr, N. E., Westen, D., Gold, L. J., & Silk, K. R. (1992). Malevolent object representations in borderline personality disorder and major depression. *Journal of Abnormal Psychology, 101,* 61–67.

Ogata, S. N., Silk, K. R., Goodrich, S., Lohr, N. E., Westen, D., & Hill, E. M. (1990). Childhood sexual and physical abuse in adult patients with borderline personality disorder. *American Journal of Psychiatry, 147,* 1008–1013.

Oldham, J. M. (2001). Integrated treatment planning for borderline personality disorder. In J. Kay (Ed.), *Integrated treatment of psychiatric disorders* (Vol. 20, pp. 51–77). Washington, DC: American Psychiatric Publishing.

Oldham, J. M., Skodol, A. E., Kellman, H. D., Hyler, S., Rosnick, L., & Davies, M. (1992). Diagnosis of DSM-III-R personality disorders by two semistructured interviews: Patterns of comorbidity. *American Journal of Psychiatry, 149,* 213–220.

Oltmanns, T. F., Melley, A. H., & Turkheimer, E. (2002). Impaired social functioning and symptoms of personality disorders assessed by peer and self-report in a nonclinical population. *Journal of Personality Disorders, 16*(5), 437–452.

Paris, J. (1997). Childhood trauma as an etiological factor in the personality disorders. *Journal of Personality Disorders, 11*(1), 34–49.

Paris, J. (2003). *Personality disorders over time: Precursors, course, and outcome.* Washington, DC: American Psychiatric Publishing.

Paris, J., Nowlis, D., & Brown, R. (1988). Developmental factors in the outcome of borderline personality disorder. *Comprehensive Psychiatry, 29,* 147–150.

Paris, J., & Zweig-Frank, H. (2001). The 27-year follow-up of patients with borderline personality disorder. *Comprehensive Psychiatry, 42*(6), 482–487.

Pfohl, B., Blum, N., Zimmerman, M., & Stangl, D. (1989). *Structured interview for DSM-III-R personality—revised (SIDP-R).* Iowa City University of Iowa.

Pfohl, B., Coryell, W., Zimmerman, M., & Stangl, D. (1986). DSM-III personality disorders: Diagnostic overlap and internal consistency of the individual DSM-III criteria. *Comprehensive Psychiatry, 27,* 21–34.

Pilkonis, P. A., Heape, C. L., Proietti, J. M., Clark, S. W., McDavid, J. D., & Pitts, T. E. (1995). The reliability and validity of two structured diagnostic interviews for personality disorders. *Archives of General Psychiatry, 52,* 1025–1033.

Pilkonis, P. A., Heape, C. L., Ruddy, J., & Serrao, P. (1991). Validity in the diagnosis of personality disorders: The use of the LEAD standard. *Psychological Assessment, 31,* 46–54.

Plomin, R., Chipuer, H. M., & Loehlin, J. C. (1990). Behavioral genetics and personality. In L. A. Pervin (Ed.), *Handbook of personality: Theory and research* (pp. 225–243). New York: Guilford Press.

Reich, J., Boerstler, H., Yates, W., & Nduaguba, M. (1989). Utilization of medical resources in persons with DSM-III personality disorders in a community sample. *International Journal of Psychiatry in Medicine, 19*(1), 1–9.

Robins, C. J., & Chapman, A. L. (2004). Dialectical behavior therapy: Current status, recent developments, and future directions. *Journal of Personality Disorders, 18*(1), 73–89.

Rothschild, L., Cleland, C., Haslam, N., & Zimmerman, M. (2003). A taxometric study of borderline personality disorder. *Journal of Abnormal Psychology, 112*(4), 657–666.

Ryle, A. (2004). The contribution of cognitive analytic therapy to the treatment of borderline personality disorder. *Journal of Personality Disorders, 18,* 3–35.

Salzman, J. P., Salzman, C., & Wolfson, A. N. (1997). Relationship of childhood abuse and maternal attachment to the development of borderline personality disorder. In M. C. Zanarini (Ed.), *Role of sexual abuse in the etiology of borderline personality disorder* (pp. 71–91). Washington, DC: American Psychiatric Association.

Samuels, J., Eaton, W. W., Bienvenu, J., Clayton, P., Brown, H., Costa, P. T., Jr., et al. (2002). Prevalence and correlates of personality disorders in a community sample. *British Journal of Psychiatry 180,* 536–542.

Satorius, N., Kaelber, C., Cooper, J. E., Roper, M. T., Rae, D. S., Gulbinat, W., et al. (1993). Progress toward achieving a common language in psychiatry. *Archives of General Psychiatry, 50,* 115–124.

Scheel, K. R. (2000). The empirical basis of dialectical behavior therapy: Summary, critique, and implications. *Clinical Psychology: Science and Practice, 7*(1), 68–86.

Schmahl, C. G., Elzinga, B. M., Ebner, U. W., Simms, T., Sanislow, C., Vermetten, E., et al. (2004). Psychophysiological reactivity to traumatic and abandonment scripts in borderline personality and posttraumatic stress disorders: A preliminary report. *Psychiatry Research, 126*(1), 33–42.

Schmahl, C. G., Vermetten, E., Elzinga, B. M., & Bremner, J. (2003). Magnetic resonance imaging of hippocampal and amygdala volume in women with childhood abuse and borderline personality disorder. *Psychiatry Research: Neuroimaging, 122*(3), 193–198.

Shedler, J., & Westen, D. (2004). Dimensions of personality pathology: An alternative to the five-factor model. *American Journal of Psychiatry, 161,* 1743–1754.

Siever, L., & Davis, K. (1991). A psychobiological perspective on the personality disorders. *American Journal of Psychiatry, 148*(12), 1647–1658.

Silk, K. R., Lee, S., Hill, E. M., & Lohr, N. E. (1995). Borderline personality disorder symptoms and severity of sexual abuse. *American Journal of Psychiatry, 152,* 1059–1064.

Silk, K. R., Lohr, N. E., Ogata, S. N., & Westen, D. (1990). Borderline inpatients with affective disorder: Preliminary follow-up data. *Journal of Personality Disorders, 4*(2), 213–224.

Skodol, A. E., Gunderson, J. G., McGlashan, T. H., Dyck, I. R., Stout, R. L., Bender, D. S., et al. (2002). Functional impairment in patients with schizotypal, borderline, avoidant, or obsessive-compulsive personality disorder. *American Journal of Psychiatry, 159,* 276–283.

Skodol, A. E., Gunderson, J. G., Pfohl, B., Widiger, T. A., Livesley, W. J., & Siever, L. (2002). The borderline diagnosis: I. Psychopathology, comorbidity, and personality structures. *Biological Psychiatry, 51,* 936–950.

Skodol, A. E., Gunderson, J. G., Shea, M., McGlashan, T. H., Morey, L. C., Sanislow, C. A., et al. (2005). The Collaborative Longitudinal Personality Disorders Study (CLPS): Overview and implications. *Journal of Personality Disorders, 19*(5), 487–504.

Skodol, A. E., Oldham, J. M., Rosnick, L., Kellman, D., & Hyler, S. (1991). Diagnosis of DSM-III-R personality disorders: A comparison of two structured interviews. *International Journal of Methods in Psychiatric Research, 1,* 13–26.

Sokal, R. R. (1974). Classification: Purposes, principles, progress, prospects. *Science, 185*(4157), 1115–1123.

Soloff, P. H., Lynch, K. G., Kelly, T. M., Malone, K. M., & Mann, J. J. (2000). Characteristics of suicide attempts of patients with major depressive episode and borderline personality disorder: A comparative study. *American Journal of Psychiatry, 157,* 601–608.

Soloff, P. H., Meltzer, C. C., Greer, P. J., Constantine, D., & Kelly, T. M. (2000). A fenfluramine-activated FDG-PET study of borderline personality disorder. *Biological Psychiatry, 47*(6), 540–547.

Soloff, P. H., & Millward, J. W. (1983). Developmental histories of borderline patients. *Comprehensive Psychiatry, 24,* 574–588.

Solomon, J., George, C., & De Jong, A. (1995). Children classified as controlling at age six: Evidence of disorganized representational strategies and aggression at home and at school. *Development and Psychopathology, 7,* 447–463.

Spitzer, R. L., Endicott, J., & Gibbon, M. (1979). Crossing the border into borderline personality and borderline schizophrenia. *Archives of General Psychiatry, 36,* 17–24.

Stern, A. (1938). Psychoanalytic investigation and therapy in the borderline group of neuroses. *Psychoanalytic Quarterly,* 467–489.

Stone, M. H. (1987). Psychotherapy of borderline patients in light of long-term follow-up. *Bulletin of the Menninger Clinic, 51,* 231–247.

Stone, M. H. (1992). Borderline personality disorder: Course of illness. In J. F. Clarkin, E. Marziali, & H. Munroe-Blum (Eds.), *Borderline personality disorder: Clinical and empirical perspectives.* New York: Guilford Press.

Stone, M. H. (1993). Long-term outcome in personality disorders. *British Journal of Psychiatry, 162,* 299–313.

Stone, M. H. (1994). Characterologic subtypes of the borderline personality disorder: With a note on prognostic factors. *Psychiatric Clinics of North America, 17,* 773–784.

Stuart, S., Pfohl, B., Battaglia, M., Bellodi, L., Grove, W., & Cadoret, R. (1998). The co-occurrence of DSM-III-R personality disorders. *Journal of Personality Disorders, 12,* 302–315.

Thomas, C., Turkheimer, E., & Oltmanns, T. F. (2003). Factorial structure of pathological personality as evaluated by peers. *Journal of Abnormal Psychology, 112,* 81–91.

Torgersen, S. (1980). The oral, obsessive, and hysterical personality syndromes: A study of hereditary and environmental factors by means of the twin method. *Archives of General Psychiatry, 37*(11), 1272–1277.

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

Torgersen, S., Lygren, S., Oien, P. A., Skre, I., Onstad, S., Edvardsen, J., et al. (2000). A twin study of personality disorders. *Comprehensive Psychiatry, 41*(6), 416–425.

Trull, T. J. (2001). Structural relations between borderline personality disorder features and putative etiological correlates. *Journal of Abnormal Psychology, 110*(3), 471–481.

Trull, T. J., Widiger, T. A., Lynam, D. R., & Costa, P. T., Jr. (2003). Borderline personality disorder from the perspective of general personality functioning. *Journal of Abnormal Psychology 112*(2), 193–202.

Tyrer, P. J. (1996). Establishing the severity of personality disorder. *American Journal of Psychiatry, 153*(12), 1593–1597.

Verheul, R., van den Bosch, L. M. C., Koeter, M. W. J., De Ridder, M. A. J., Stinjnen, T., & van den Brink, W. (2003). Dialectical behaviour therapy for women with borderline personality disorder: 12-month, randomised clinical trial in the Netherlands. *British Journal of Psychiatry, 182,* 135–140.

Weaver, T. L., & Clum, G. A. (1993). Early family environments and traumatic experiences associated with borderline personality disorder. *Journal of Consulting and Clinical Psychology, 61*(6), 1068–1075.

Westen, D. (1991a). Cognitive-behavioral interventions in the psychoanalytic psychotherapy of borderline personality disorders. *Clinical Psychology Review, 11,* 211–230.

Westen, D. (1991b). Social cognition and object relations. *Psychological Bulletin, 109,* 429–455.

Westen, D. (1997). Divergences between clinical and research methods for assessing personality disorders: Implications for research and the evolution of Axis II. *American Journal of Psychiatry, 154,* 895–903.

Westen, D. (2000). Integrative psychotherapy: Integrating psychodynamic and cognitive-behavioral theory and technique. In C. R. Snyder & R. Ingram (Eds.), *Handbook of psychological change: Psychotherapy processes and practices for the 21st century* (pp. 217–242). New York: Wiley.

Westen, D., & Bradley, R. (2005). Prototype diagnosis of personality. In S. Strack (Ed.), *Handbook of personology and psychopathology* (pp. 238–256). New York: Wiley.

Westen, D., Bradley, R., & Shedler, J. (2005). Refining the borderline construct: Diagnostic criteria and trait structure. *Unpublished manuscript, Emory University, Atlanta, GA.*

Westen, D., Dutra, L., & Shedler, J. (2005). Assessing adolescent personality pathology: Quantifying clinical judgment. *British Journal of Psychiatry, 186,* 227–238.

Westen, D., Lohr, N., Silk, K. R., Gold, L., & Kerber, K. (1990). Object relations and social cognition in borderlines, major depressives, and normals: A thematic apperception test analysis. *Psychological Assessment, 2,* 355–364.

Westen, D., Ludolph, P., Block, M. J., Wixom, J., & Wiss, F. C. (1990). Developmental history and object relations in psychiatrically disturbed adolescent girls. *American Journal of Psychiatry, 147,* 1061–1068.

Westen, D., Ludolph, P., Misle, B., Ruffins, S., & Block, J. (1990). Physical and sexual abuse in adolescent girls with borderline personality disorder. *American Journal of Orthopsychiatry, 60,* 55–66.

Westen, D., & Muderrisoglu, S. (2003). Reliability and validity of personality disorder assessment using a systematic clinical interview: Evaluating an alternative to structured interviews. *Journal of Personality Disorders, 17,* 350–368.

Westen, D., & Muderrisoglu, S. (2006). Clinical assessment of pathological personality traits. *American Journal of Psychiatry, 163,* 1285–1287.

Westen, D., Muderrisoglu, S., Fowler, C., Shedler, J., & Koren, D. (1997). Affect regulation and affective experience: Individual differences, group differences, and measurement using a Q-sort procedure. *Journal of Consulting and Clinical Psychology, 65,* 429–439.

Westen, D., & Shedler, J. (1999a). Revising and assessing Axis II: 1. Developing a clinically and empirically valid assessment method. *American Journal of Psychiatry, 156,* 258–272.

Westen, D., & Shedler, J. (1999b). Revising and assessing Axis II: 2. Toward an empirically based and clinically useful classification of personality disorders. *American Journal of Psychiatry, 156,* 273–285.

Westen, D., & Shedler, J. (2000). A prototype matching approach to diagnosing personality disorders toward DSM-V. *Journal of Personality Disorders, 14,* 109–126.

Westen, D., Shedler, J., & Bradley, R. (2006). A prototype approach to personality diagnosis. *American Journal of Psychiatry, 163,* 838–848.

Westen, D., Shedler, J., Durrett, C., Glass, S., & Martens, A. (2003). Personality diagnosis in adolescence: DSM-IV Axis II diagnoses and an empirically derived alternative. *American Journal of Psychiatry, 160,* 952–966.

Westen, D., Thomas, C., Nakash, O., & Bradley, R. (2006). Clinical assessment of attachment patterns and personality disorder in adolescents and adults. *Journal of Consulting and Clinical Psychology, 74,* 1065–1085.

White, C. N., Gunderson, J. G., Zanarini, M. C., & Hudson, J. I. (2003). Family studies of borderline personality disorder: A review. *Harvard Review of Psychiatry, 11*(1), 8–19.

Widiger, T. A. (1995). Deletion of self-defeating and sadistic personality disorders. In W. J. Livesley (Ed.), *The DSM-IV personality disorders: Diagnosis and treatment of mental disorders* (pp. 359–373). New York: Guilford Press.

Yen, S., Shea, M. T., Battle, C. L., Johnson, D. M., Zlotnick, C., Dolan-Sewell, R., et al. (2002). Traumatic exposure and posttraumatic stress disorder in borderline, schizotypal, avoidant, and obsessive-compulsive personality disorders: Findings from the collaborative longitudinal personality disorders study. *Journal of Nervous and Mental Disease, 190,* 510–518.

Yeomans, F. E. (2004). Transference-focused psychotherapy in borderline personality disorder. *Psychiatric Annals, 34,* 449–454.

Young, J. E., Klosko, J. S., & Weishaar, M. (2003). *Schema therapy: A practitioner's guide.* New York: Guilford Press.

Zanarini, M. C. (1997). *Role of sexual abuse in the etiology of borderline personality disorder.* Washington, DC: American Psychiatric Publishing.

Zanarini, M. C., & Frankenburg, F. R. (1997). Pathways to the development of borderline personality disorder. *Journal of Personality Disorders, 11*(1), 93–104.

Zanarini, M. C., Frankenburg, F. R., DeLuca, C. J., Hennen, J., Khera, G. S., & Gunderson, J. G. (1998). The pain of being borderline: Dysphoric states specific to borderline personality disorder. *Harvard Review of Psychiatry, 6,* 201–207.

Zanarini, M. C., Frankenburg, F. R., Hennen, J., & Silk, K. R. (2003). The longitudinal course of borderline psychopathology: 6-year prospective follow-up of the phenomenology of borderline personality disorder. *American Journal of Psychiatry, 160*(2), 274–283.

Zanarini, M. C., Frankenburg, F. R., Hennen, J., & Silk, K. R. (2004). Mental health service utilization by borderline personality disorder patients and Axis II comparison subjects followed prospectively for 6 years. *Journal of Clinical Psychiatry, 65*(1), 28–36.

Zanarini, M. C., Frankenburg, F. R., Vujanovic, A. A., Hennen, J., Reich, D. B., & Silk, K. R. (2004). Axis II comorbidity of borderline personality disorder: Description of 6-year course and prediction to time-to-remission. *Acta Psychiatrica Scandinavica, 110*(6), 416–420.

Zanarini, M. C., Frankenburg, F. R., Yong, L., Raviola, G., Reich, D. B., Hennen, J., et al. (2004). Borderline psychopathology in the first-degree relatives of borderline and Axis II comparison probands. *Journal of Personality Disorders, 18,* 449–447.

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

Zanarini, M. C., Yong, L., Frankenburg, F. R., Hennen, J., Reich, D., Marino, M. F., et al. (2002). Severity of reported childhood sexual abuse and its relationship to severity of borderline psychopathology and psychosocial impairment among borderline inpatients. *Journal of Nervous and Mental Disease, 190*(6), 381–387.

Zelkowitz, P., Paris, J., Guzder, J., & Feldman, R. (2001). Diathesis and stressors in borderline pathology of childhood: The role of neuropsychological risk and trauma. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(1), 100–105.

Zimmerman, M., & Mattia, J. I. (1999). Axis I diagnostic comorbidity and borderline personality disorder. *Comprehensive Psychiatry, 40*(4), 245–252.

Zittel, C., & Westen, D. (1998). Conceptual issues and research findings on borderline personality disorder: What every clinician should know. *In Session, 4,* 5–20.

Zittel, C., & Westen, D. (2002). *Subtyping borderline personality disorder.* Presentation given at New Directions in Borderline Personality Disorder II, a conference jointly sponsored by the National Institutes of Mental Health and the Borderline Personality Disorder Research Foundation, New York.