

Borderline personality disorder: A comparison between children and adults

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Recently, more empirical studies have been devoted to the investigation of borderline personality disorder (BPD) in children and adolescents. Against this background, the purpose of the current review is to compare research findings on diagnostic-related phenomena in child and adolescent samples with those in adult samples to establish the utility of the BPD construct in childhood and adolescence. A search of relevant publications reported in Pubmed and PsycInfo from 1940 (the first clinical descriptions of BPD in childhood) to 2006 was carried out. A total of 58 studies were included. The review of the adult literature was not exhaustive but relied on excellent existing and comprehensive reviews of the adult literature carried out in the past 5 years. Although significant differences seem to exist between juveniles and adults in diagnostic-related phenomena associated with BPD, these can be explained by the principle of heterotypic continuity in development. Moreover, enough overlap between juvenile and adult BPD has been observed to warrant further empirical investigation into the construct of juvenile BPD. Specific areas for future research in juvenile BPD suggested by this review include studies of comorbidity, measure development, and the use of neurobiological measures such as functional neuroimaging (Bulletin of the Menninger Clinic, 71[2], 85-114)

Adult borderline personality disorder (BPD) is suggested to be a relatively stable personality disposition and a particularly devastating disorder (Bleiberg, 2001). Epidemiological estimates reveal point prevalences for the disorder at approximately 1%–2% (Lenzenweger, Loranger, Korfine, & Neff, 1997; Swartz, Blazer, George, & Winfield, 1990; Torgersen, Kringlen, & Cramer, 2001). One in every 100–200

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people may therefore be affected with BPD (Lenzenweger & Cicchetti, 2005). In addition, the American Psychiatric Association (2000) reported that 10% of psychiatric outpatients and 20% of inpatients suffer from BPD, and that BPD is associated with severe psychosocial impairment and a mortality rate 50 times higher than that of the normal population. In addition, Zanarini, Frankenburg, Khera, and Bleichmar (2001) demonstrated histories of excessive mental health use among individuals diagnosed with BPD. Taken together, these findings point to the need for the early identification of and intervention for those with BPD features.

Despite this urgency, most mental health professionals have viewed personality as lacking in cohesiveness and durability prior to the age of 18 (Crick, Murray–Close, & Woods, 2005). The identification of BPD is therefore strongly discouraged during childhood and adolescence, with the unfortunate result that we still do not know whether the phenomenology, correlates, risk factors, and rates of BPD in childhood and adolescence resemble those of adult BPD.

There has been a renewed surge of interest in juvenile BPD, as reflected in the fact that a special issue of *Development and Psychopathology* was recently devoted to the topic (Lenzenweger & Cicchetti, 2005). Understanding the relationship between childhood/adolescent borderline traits/disorder and BPD in adulthood will add to this growing body of literature, but this requires a systematic review of similarities and differences between diagnosis–related phenomena across adults and youth. Without such knowledge, the applicability of the BPD diagnosis to children and adolescents remains dubious, and planning and evaluation of evidence–based practices aimed at preventing the development of full–blown BPD pathology may be limited.

To this end, the purpose of this review is to compare research findings on diagnostic–related phenomena in child and adolescent samples with those in adult samples to establish the utility of the BPD construct in childhood and adolescence. The review of the child and adolescent literature is exhaustive, in that a search in PubMed and PsycInfo from 1940 (the first clinical descriptions of BPD in childhood) to 2006 was carried out to review all empirical work with the following keywords: “personality disorder,” “Cluster B,” “borderline personality disorder,” and “borderline,” as well as “children,” “adolescent,” and “adolescence.” Only studies that included child and adolescent samples were considered (for reviews of retrospective studies investigating childhood precursors of adult BPD, see Paris, 2003, or Zanarini, 2000). A total of 58 studies were included. In contrast, the review of the adult literature is representative. Recent reviews of the adult literature by Skodol et al.

(Skodol et al., 2002a; Skodol et al., 2002b; Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004), and Siever, Torgersen, Gunderson, Livesley, and Kendler (2002) are recommended for additional references.

The review is structured around a discussion of similarities and differences in several diagnosis-related phenomena, which are also listed and summarized in Table 1 for quick reference: diagnostic criteria, diagnostic instruments, questionnaire measures, prevalence, comorbid disorders, course, environmental risk factors, neurocognitive and/or biological risk factors, and genetic factors. The article concludes with future research directions that may increase confidence in the construct of juvenile BPD.

Diagnostic criteria

Early authors viewed borderline pathology as a developmental concept. For example, Kernberg (1967) defined “borderline personality organization” as an intrapsychic organization that fell somewhere between neurotic and psychotic organizations. In this definition, borderline pathology was characterized by identity diffusion (vs. cohesion), primitive defenses, and variably intact reality testing. In the child literature, Masterson (1978) described borderline personality disorder as the end result of intense abandonment feelings, clinging to the maternal figure, and failure to achieve autonomy after successfully resolving the separation-individuation phase of development. As a result of this failure to separate and individuate from the primary attachment figure, individuals with borderline pathology are severely intolerant of being alone (Masterson & Rinsley, 1975).

The concept became a formal diagnosis in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 1980), which described BPD as a personality disorder marked by instability in multiple areas, including interpersonal behavior, mood, and self-image. Although the diagnosis has evolved since that time, instability across a variety of domains remains the hallmark of the diagnosis. The nine diagnostic criteria in the latest version of the *DSM* (APA, 2000) can be organized into four areas of psychopathology (Lieb et al., 2004): affective (inappropriate and intense anger, chronic feelings of emptiness, affective instability); cognitive (paranoid ideation and severe dissociative symptoms, identity disturbance); behavioral (impulsivity, suicidal behavior or self-mutilation); and interpersonal (unrealistic fears of abandonment by loved ones, unstable and intense relationships). Individuals who exhibit symptoms in all four of these areas simultaneously can be discriminated from individuals with other

Table 1. Similarities and differences between adults and children in diagnostic-related phenomena of BPD

| | Adults | Children |
|---------------------|--|--|
| Diagnostic criteria | <p>A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation.</p> <p>Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating).</p> <p>Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days).</p> <p>Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights).</p> <p>Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior.</p> <p>Identity disturbance: markedly and persistently unstable self-image or sense of self.</p> <p>Chronic feelings of emptiness.</p> <p>Frantic efforts to avoid real or imagined abandonment.</p> <p>Transient, stress-related paranoid ideation or severe dissociative symptoms. (APA, 2000)</p> | <p>A pattern of unstable and intense interpersonal relationships characterized by alternation between extremes of overidealization and devaluation and/or marked distortion of the nature of the relationship.</p> <p><i>Example:</i> Describing teacher as a “girlfriend,” chronic inability to maintain friendships despite wish to do so.</p> <p>Impulsiveness in at least two areas that are potentially self-damaging (e.g., reckless risk taking, running away, stealing, substance abuse, sex, binge eating).</p> <p><i>Example:</i> Walking across railroad bridge railing, sniffing glue.</p> <p>Affective instability: marked, rapid shifts from baseline mood to depression, irritability, or anxiety lasting less than a few hours and only rarely more than a few days; episodes may include transient distortions of reality.</p> <p><i>Example:</i> Early-afternoon anxiety attack with persecutory delusions, followed by successful participation in soccer game in late afternoon.</p> <p>Inappropriate, intense anger or lack of control of anger (e.g., frequent displays of temper, constant anger, recurrent physical fights).</p> <p><i>Example:</i> Easily provoked, frequent fights, threatens and attempts to throw therapist out window.</p> <p>Recurrent suicidal threats, gestures, or behavior or self-mutilating or self-endangering acts.</p> <p><i>Example:</i> Carves boyfriend’s name into arm, multiple episodes of being struck by car.</p> <p>Marked and persistent disturbance in self-perception and self-presentation characterized by confusion regarding two of the following: gender identity or roles, friendships, socially appropriate behaviors, school or career plans, self-image.</p> <p><i>Example:</i> Chronic cross-dressing, running for class president despite having no friends.</p> <p>Chronic feelings of emptiness or boredom.</p> <p><i>Example:</i> Chronic complaints of boredom, unable to invest in appropriate activities.</p> <p>Frantic efforts to avoid, or major preoccupation with, real or imagined abandonment (do not include suicidal or self-mutilating behavior).</p> <p><i>Example:</i> Continual concern that therapist will not be there at next appointment, refusal to leave house while parent is at work. (Goldman, D’Angelo, DeMaso, and Mezzacappa, 1992)</p> |

| | | |
|--------------------------------------|--|--|
| <p>Diagnostic instruments</p> | <p>Diagnostic Interview for Borderline Patients (DIB) (Gunderson, Kolb, & Austin, 1981) Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV) (Zanarini et al., 1996) International Personality Disorder Examination (IPDE) (Loranger, 1999) Structured Interview for DSM-IV Personality (SIDP-IV) (Pfohl, Blum, & Zimmerman, 1997) Personality Disorders Interview-IV (PDI-IV) (Widiger et al., 1995) Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II) (First et al., 1995)</p> | <p>Personality Disorder Examination (PDE); (Lenzenweger, Loranger, Korfine, & Neff, 1997) Diagnostic Interview for Borderline Patients (DIB) (Gunderson, Kolb, & Austin, 1981) Diagnostic Interview for Borderline Patients-Child Version (C-DIB) (Greenman, Gunderson, Cane, & Saltzman, 1986) Structured Clinical Interview for DSM-III-R Personality Disorders (SCID-II) (Spitzer, Williams, Gibbon, & First, 1989) SWAP-2000-A (Westen & Muderrisoglu, 2003)</p> |
| <p>Questionnaire measures</p> | <p>Dimensional Assessment of Personality Pathology-Basic Questionnaire (DAPP-BQ) (Livesley, 1998) Schedule for Nonadaptive and Adaptive Personality (SNAP) (Clark, 1993) Schedler-Westen Assessment Procedure (SWAP-2000) (Westen & Schedler, 1999) Personality Disorders Questionnaire (PDQ) (Hylet, 1994)</p> | <p>Borderline Personality Features Scale (BPFS-C) (Crick, Murray-Close, & Woods, 2005)</p> |
| <p>Prevalence</p> | <p>Community samples: 0.7% (Torgensen, Kringlen, & Cramer, 2001) 1.3% (Lenzenweger, Loranger, Korfine, & Neff, 1997) 1.8% (Swartz et al., 1990)</p> <p>Clinical samples: 10% of psychiatric outpatients (Widiger & Weissman, 1991) 20% of psychiatric inpatients (Widiger & Weissman, 1991) 43% of young adult (ages 18-37) inpatients (Grillo et al., 1998) 47% of adults (Becker, Grilo, Edell, & McGlashan, 2002)</p> <p>Gender differences: 74% of all patients with BPD are women (Widiger & Frances, 1989).</p> | <p>Community samples: 42% of 72 children in community showed some form of personality disturbance (Korenblum, Marron, Golombek, & Stein, 1990) 46% Cluster B in a sample of 13-year-old schoolchildren (Golombek, Marron, Stein, & Korenblum, 1986) 3% of 9 to 19-year-olds with severe borderline disturbance (Johnson, Cohen, Brown, Smalles, & Bernstein, 1999) 14% in high school students (Chabrol, Montovany, Chouicha, Callahan, & Muller, 2001)</p> <p>Clinical samples: 49% of inpatients (ages 12-18) (Grilo et al., 1998) 43% of inpatients (mean age, 15.5 years) (Levy et al., 1999) .02% of consecutive referrals (mean age = 10.8 years) (Goldman, D'Angelo, DeMaso, & Mezacappa, 1992)</p> <p>Gender differences: 53% of inpatients (Becker, Grilo, Edell, & McGlashan, 2002) 15% of males and 17.2% of females in a community study met criteria (Bernstein et al., 1993)</p> |

Table 1. (cont.)

| | Adults | Children |
|---------------------------|---|--|
| Comorbid disorders | <p>Axis I disorders comorbid with BPD</p> <p>Major Depressive Disorder (Skodol et al., 1999; Zanarini et al., 1998a)</p> <p>Panic Disorder (Zanarini et al., 1998a)</p> <p>Social Phobia (Zanarini et al., 1998a)</p> <p>Posttraumatic stress disorder (Zanarini et al., 1998a)</p> <p>Eating Disorder Not Otherwise Specified (Zanarini et al., 1998a)</p> <p>Substance Use Disorders (Trull et al., 2000) to 64.1% (Zanarini et al., 1998a)</p> <p>Prevalence of other Axis II disorders in BPD:</p> <p>Antisocial Personality Disorder (Becker et al., 2000)</p> <p>Self-defeating Personality Disorder (Zanarini et al., 1998b)</p> <p>Paranoid Personality Disorder (Zanarini et al., 1998b)</p> <p>Avoidant Personality Disorder (Zanarini et al., 2004)</p> <p>Dependent Personality Disorder (Zanarini et al., 1998b)</p> | <p>Axis I disorders comorbid with BPD:</p> <p>Externalizing disorder (Eppright, Kashani, Robison, & Reid, 1993); (Myers, Burkner, & Otto, 1993); (Guzder, Paris, Zeligowitz, & Feldman, 1999)</p> <p>Major depression (McManus, Lerner, Robbins, & Barbour, 1984)</p> <p>Bipolar disorder (Kutcher, Marton, & Korenblum, 1990)</p> <p>Substance abuse (Grilo, Levy, Becker, & McGlashan, 1996)</p> <p>Prevalence of other Axis II disorders in BPD:</p> <p>Adolescent BPD shows broad distribution of clusters A, B, and C (Becker, Grilo, Edell, & McGlashan, 2000): Paranoid (7%), Schizoid (0%), Schizotypal (12%), Histrionic (10%), Narcissistic (9%), Avoidant (12%), Dependent (9%), Obsessive-compulsive (6%), Passive-aggressive (29%).</p> |
| Course | <p>Remission rates:</p> <p>McLean Study of Adult Development: 34.5% of 290 patients with BPD met criteria for remission at 2 years, 49.4% at 4 years, and 68.6% at 6 years (Zanarini et al., 2003).</p> <p>Collaborative Longitudinal Personality Disorder Study: 59% of BPD patients met criteria for remission at 12 months (Shea et al., 2002).</p> <p>Factors associated with poor prognosis (diagnostic stability):</p> <p>Childhood sexual abuse (Paris, 1993)</p> <p>Incest (Stone, 1990)</p> <p>Affective instability, impulsivity, substance abuse, and an increased number of comorbid Axis I and/or Axis II disorders (Skodol et al., 2002b)</p> <p>Factors associated with good outcome:</p> <p>High IQ (e.g., Stone, 1990)</p> <p>Low narcissistic entitlement, absence of parental divorce, and presence of self-destructive acts at the time of admission (Plakun, 1991).</p> | <p>Remission rates:</p> <p>44% of diagnosed children were found to be free of their initial personality difficulties at 3-year follow-up (Marion, Connolly, Kutcher, & Korenblum, 1993).</p> <p>Two thirds of adolescent inpatients with BPD were free of BPD at 2-year follow-up (Garnet, Levy, Matranah, Edell, & McGlashan, 1994).</p> <p>85% of adolescent inpatients were free of BPD at 3-year follow-up (Meijer, Goodhart, & Treffers, 1998).</p> <p>A 59% linear decline in the number of BPD features between ages 16 and 22 (Johnson, Smailes, Cohen, Brown, & Bernstein, 2000).</p> <p>Cluster B symptoms highly stable across an 8-year period from early adolescence to early adulthood (Crawford, Cohen, & Brook, 2001a)</p> <p>Factors associated with poor prognosis:</p> <p>Antisocial personality disorder symptoms before age 15 predicted later BPD (Goodman, Hull, Clarkin, & Yeomans, 1999).</p> <p>Over half of conduct-disordered patients developed BPD or Antisocial personality disorder (Modestin, Matutut, & Wurmle, 2001).</p> <p>Internalizing and externalizing problems at ages 10-14 each predicted Cluster B symptoms 2 years later: Cluster B symptoms predicted externalizing problems at follow-up (Crawford, Cohen, & Brook, 2001b).</p> <p>Factors associated with good outcome:</p> <p>Unknown</p> |

| | | |
|---|---|--|
| <p>Environmental risk factors</p> | <p>Retrospectively measured: Abuse and neglect during childhood (Battle et al., 2004; Links, Steiner, Offord, & Eppel, 1988; Shearer, Peters, Quayman, & Ogden, 1990; Westen, 1990; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1989; Zanarini et al., 1997) Experiences of disturbed parental involvement during childhood (Goldberg, Mamm, Wise, & Segall, 1985) Childhood problems of anxiety and tolerating separations, frustration and mood regulation (Reich & Zanarini, 2001) Childhood attachment problems (Agrawal, Gunderson, Bjarne, Holmes, & Lyons-Ruth, 2004) Parental separation and loss (Paris, Zweig-Frank, & Guzder, 1994a, 1994b) Symptoms of externalizing disorder combined with abnormal neuropsychological functioning, physical abuse and separations (Greenman, Gunderson, Cane, & Saltzman, 1986)</p> | <p>Concurrently measured: Trauma, neglect, maltreatment, and separation (Famularo, Kinscherff, & Fenton, 1991; Johnson, Smailes, Cohen, Brown, & Bernstein, 2000; Kestenbaum, 1983; Lofgren, Bemporad, King, Lindem, & O'Driscoll, 1991; Ludolph et al., 1990; Rogosch & Cicchetti, 2004) Exposure to sexual and physical abuse (Goldman, D'Angelo, DeMaso, & Mezzacappa, 1992) Serious parental psychopathology, including depression, substance abuse, or antisocial personality (Goldman, D'Angelo, & DeMaso, 1993)</p> |
| <p>Neurocognitive- and/or biological concurrent correlates</p> | <p>Physiological findings: Hyperarousal to emotional stimuli in daily life (Bohus, Schmahl, & Lieb, 2004) Hypoarousal in response to affect-eliciting pictures (Bohus, Schmahl, & Lieb, 2004) Endocrinological findings: HPA axis hyperresponsiveness (Rinne et al., 2002) Structural findings: Reduced hippocampal and amygdala volumes (e.g., Driessen et al., 2000) Reduced frontal and orbitofrontal volumes (Tebarth van Elst et al., 2003) Functional findings: Anterior cingulate cortex, orbitofrontal and dorsolateral prefrontal cortex, hippocampus, and amygdala abnormalities (e.g., Lieb et al., 2004) Altered baseline metabolism in prefrontal regions (Soloff et al., 2003) Differential activation in amygdala (e.g., Donegan et al., 2003; Herpetz et al., 2001)</p> | <p>Neuropsychological findings: Executive functioning (Rogosch & Cicchetti, 2005; Zelkowitz, Paris, Guzder, & Feldman, 2001)</p> |
| | <p>Neurotransmitters implicated: Serotonin (e.g., Coccaro, Bergeman, Kavoussi, & Seroczynski, 1997; Gurvits, Koenigsberg, & Siever, 2000) Dopamine (e.g., Friedel, 2004)</p> | |

Table 1. (cont.)

| Adults | Children |
|---|---|
| <p>Genetic factors</p> | <p>Unknown</p> |
| <p>Family aggregation studies: Higher rates of BPD in relatives of individuals with BPD, compared to base rates (Siever, Torgersen, Gundersen, Livesley, & Kendler, 2002)</p> | <p>Higher rates of BPD in relatives of individuals with BPD, compared to base rates (Siever, Torgersen, Gundersen, Livesley, & Kendler, 2002)</p> |
| <p>Twin studies: 0% concordance in DZ pairs, 11% in MZ pairs (Torgersen, 1984) 7% concordance in DZ pairs, 35% in MZ pairs (Torgersen, Kringle, & Cramer, 2001)</p> | <p>0% concordance in DZ pairs, 11% in MZ pairs (Torgersen, 1984) 7% concordance in DZ pairs, 35% in MZ pairs (Torgersen, Kringle, & Cramer, 2001)</p> |
| <p>BPD traits with high heritability coefficients: Reckless impulsivity (e.g., Coccaro, Bergeman, & McClearn, 1993) Emotion dysregulation (e.g., Livesley, Jang, & Vernon, 1998)</p> | <p>Reckless impulsivity (e.g., Coccaro, Bergeman, & McClearn, 1993) Emotion dysregulation (e.g., Livesley, Jang, & Vernon, 1998)</p> |

Note. Adult BPD references are representative, not exhaustive. Comorbid diagnoses are also limited to those that were significantly higher in patients with BPD compared to non-personality-disordered controls.

personality disorders (Zanarini, Gunderson, Frankenburg, & Chauncey, 1990).

Because clinical descriptions of BPD originated in the developmental literature, it is not surprising that juvenile BPD can be traced back to as early as the 1940s, usually formulated in a psychoanalytic framework (Bleiberg, 1994). Kernberg (1990) discusses a good example of such early descriptions is by Kestenbaum (1983), who described Velia who at age 5 had no friends, was prone by age 7 to severe and uncontrollable rage episodes, was unable to get along with other children, and abused younger children if she did not get her way. She had begun lying to her parents, stealing from her mother's purse, and tearing up her own clothes and hiding them.

In spite of high intelligence, Velia lacked the concentration to complete homework. She seemed constantly nervous, denied she had any problems, and at times behaved either like a good little girl or betrayed her anger and hostility in comments such as "How would you like it if I put a live rattlesnake on your plate?" This 7-year-old saw women as halloween witches and often became violently upset with her therapist. ("You are dead, I killed you"; "You are a mind reader and a witch"; "You made me think of awful things.")

She exhibited shifting levels of functioning, becoming wild once and throwing two dolls against the door, saying, "I could vomit; you are vomited up" and "There, I vomited you out of me." There were therapeutic sessions during which the child lost control and regressed to the point of soiling herself, although this happened infrequently. Out-of-control sessions were interspersed with sessions in which the child displayed positive emotions, telling her therapist that she wished to adopt her because she loved her as much as her grandmother.

Descriptions such as these were not very precise but laid the foundation for describing formal criteria for BPD in children and adolescents. Goldman, D'Angelo, DeMaso, and Mezzacappa (1992) were the first to adapt BPD criteria for use with children (see Table 1). With the publication of the fourth edition of the DSM (APA, 1994), provision was made for the first time for the diagnosis of BPD in children and adolescents. Adult BPD criteria were modified for application in youth by stipulating that BPD may be diagnosed in younger patients when maladaptive traits have been present for at least 1 year (in contrast to 2 years for adult BPD) and are pervasive, persistent, and unlikely to be limited to a particular developmental stage or an episode of Axis I disorder. The application of adult BPD criteria to children and adolescents

led to the use of a standardized set of criteria by which findings from different studies can be compared with one another (Sharp & Bleiberg, 2007). It also enables the use of adult assessment tools in younger samples. It is to the topic of diagnostic instruments that we turn next.

Diagnostic instruments

To obtain an accurate diagnosis of BPD, clinicians and researchers may use a variety of questionnaires or semistructured interviews, including the Diagnostic Interview for Borderline Patients (DIB) (Gunderson, Kolb, & Austin, 1981) and its revised version, the Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV) (Zanarini, Frankenburg, Sickel, & Yong, 1996); the International Personality Disorder Examination (IPDE) (Loranger, 1999); the Structured Interview for DSM-IV Personality (SIDP-IV) (Pfohl, Blum, & Zimmerman, 1997); the Personality Disorders Interview-IV (PDI-IV) (Widiger, Mangan, Corbitt, Ellis, & Thomas, 1995); the Personality Diagnostic Questionnaire (PDQ) (Hyler, 1994); the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II) (First, Spitzer, Williams, Gibbon, & Benjamin, 1997); and the Zan-BPD (Zanarini, 2003). These instruments provide a significantly more reliable diagnosis than an unstructured interview alone (Skodol et al., 2002a).

Most of the above-mentioned adult measures (see Table 1) have been used in either an unadapted or adapted form in child samples. For instance, several studies in childhood and adolescence (Garnet, Levy, Mattanah, Edell, & McGlashan, 1994; Grilo et al., 1995; Grilo, Levy, Becker, Edell, & McGlashan, 1996; Grilo et al., 1998; Kutcher, Marton, & Korenblum, 1990; Levy et al., 1999) have made use of the Personality Disorder Examination (PDE) (Lenzenweger et al., 1997). Similarly, Ludolph et al. (1990) modified the DIB for use in a sample of adolescent inpatient girls, while a child version of the DIB (C-DIB) (Greenman, Gunderson, Cane, & Saltzman, 1986) was developed for use in preadolescent children through applying minor modifications. Like its adult counterpart, the C-DIB includes 24 items that cover five subscales. The C-DIB has been used in chart review studies of children (Greenman et al., 1986; Guzder, Paris, Zelkowitz, & Marchessault, 1996) and showed interrater reliability of kappa = .72 in the latter study. It was used again by the same group in a series of studies based on direct interview with preadolescent children (Guzder, Paris, Zelkowitz, & Feldman, 1999; Guzder et al., 1996; Paris, Zelkowitz, Guzder, Joseph, & Feldman, 1999). The SCID-II was used by Eppright, Kashani, Robison, and Reid (1993) in a sample of juvenile adolescent offenders.

Recently, Westen, Shedler, Durrett, Glass, and Martens (2003) re-

ported on the development of an alternative measure called the Shedler–Westen Assessment Procedure–200 for Adolescents (SWAP–200A). It is a Q–sort instrument (ranking procedure as pioneered by Block, 1978) for assessing adolescent personality pathology. Preliminary studies indicate good external and internal validity for the measure.

Despite the increased reliability of the BPD diagnosis in both children and adults when a semistructured interview is used, however, the current conceptualization of BPD by the *DSM* is not without difficulties. For example, a review of the diagnostic criteria of BPD by Skodol and colleagues (2002a) has highlighted several criticisms aimed at its reliance on categorical method of diagnosis (e.g., Livesley, 1985; Widiger & Frances, 1985), among which are the lack of empirical support for diagnostic thresholds and the heterogeneity of the BPD diagnosis due to its polythetic criteria set. Alternate methods of diagnosing, such as dimensional models in which BPD is characterized as the extreme (and dysfunctional) expression of common personality traits, have been suggested (e.g., Livesley, 1998; Millon, 1991; Widiger & Sanderson, 1995). Given these difficulties, researchers who wish to demonstrate the existence of juvenile BPD will do well to take into account dimensional measures of borderline pathology when establishing diagnostic criteria. Only one dimensional measure of BPD has been developed for use in children and adolescents. We turn to a discussion of questionnaire measures next.

Questionnaire measures

To measure adult personality pathology dimensionally, several researchers have developed questionnaire measures focusing on traits that cut across personality disorders, such as the Dimensional Assessment of Personality Pathology–Basic Questionnaire (DAPP–BQ) (Livesley, Jang, & Vernon, 1998), the Schedule for Nonadaptive and Adaptive Personality (SNAP) (Clark, 1993), and the Shedler–Westen Assessment Procedure (SWAP–200) (Westen & Shedler, 1999). To date, research has not suggested a clear advantage of any of these measures, but all appear to measure affective, behavioral, and interpersonal dysregulation and dysfunction reliably in patients with BPD (Skodol et al., 2002a).

Only one dimensional measure has been developed for use in children. Crick and colleagues (2005) developed the Borderline Personality Features Scale–Child (BPFS–C). The BPFS–C is based on the borderline scale of the Personality Assessment Inventory (PAI) (Morey, 1991), which is a reliable and valid instrument used to assess borderline per-

sonality features among adults and includes four domains: affective instability, identity problems, negative relationships, and self-harm. The BPFS-C includes age-appropriate items adapted from the original PAI to reflect these four domains, including affective instability (e.g., “My feelings are very strong. For instance, when I get mad, I get really, really mad. When I get happy, I get really, really happy”); identity problems (e.g., “I feel that there is something important missing about me, but I don’t know what it is”); negative relationships (e.g., “I’ve picked friends who have treated me badly”); and self-harm (e.g., “I get into trouble because I do things without thinking”). The measure was recently validated in a normative sample of 400 (54% female) fourth through sixth graders. Higher scores on the scale were correlated with other indices of borderline behavior, for example, cognitive sensitivity, emotional sensitivity, friend exclusivity, and aggression. Furthermore, borderline features were also found to be moderately stable over the course of the study, with girls reporting higher levels of features than boys. Finally, BPFS-C scores were uniquely related to indicators of borderline personality pathology above and beyond scores on a depression scale for children.

The development and further validation of dimensional measures such as the BPFS-C is essential for shedding light on the phenomenology of BPD in childhood and adolescence. Not only does it enable the study of heterotypic continuity across development, but it also enables the use of BPD measures in community samples where subclinical levels of borderline pathology may exist. As such, these measures can be used for longitudinal follow-up of personality problems in community studies to further our understanding of the development of different trajectories associated with these disorders. Moreover, in identifying children with sub-clinical levels of personality disorder in clinical samples, clinicians are able to intervene before the full-blown disorder becomes entrenched.

Prevalence

Comprehensive surveys of community samples from the United States and abroad suggest that the point prevalence of BPD ranges from 0.7% (Torgersen et al., 2001) to 1.3% (Lenzenweger et al., 1997) or 1.8% (Swartz et al., 1990). Estimates are even higher in clinical samples. Approximately 10% of psychiatric outpatients and 20% of inpatients carry the BPD diagnosis (Widiger & Weissman, 1991). Rates are even higher among young adult inpatients (ages 18–37), with an approximate prevalence of 43% (Grilo et al., 1998). Furthermore, women are

overrepresented in this diagnosis, comprising 74% of all patients with BPD (Widiger & Frances, 1989).

Prevalence rates of BPD among child and adolescent samples vary depending on study design and sample characteristics but seem to be generally higher than reported for adults. For example, Levy et al. (1999) demonstrated a rate of 43% in 165 adolescent inpatients (mean age 15.5 years). Similar rates have been reported by Grilo et al. (1998), who reported 49%. In contrast, Goldman and colleagues (1992) assessed just under 2,000 consecutive referrals to an outpatient clinic through clinical assessments and the DIB-R. Only 44 children met criteria for BPD, of which 32 were boys (mean age = 10.8; *SD* = 3.6) and 12 were girls (mean age = 12.4; *SD* = 4.5).

Studies of prevalence rates in community samples also show inconsistent results. In a small community sample (*N* = 72), 42% of adolescents showed some degree of personality disturbance, of which 40% fell into a histrionic, borderline, narcissistic cluster (Korenblum, Marton, Golombek, & Stein, 1990). In another study, the same group reported a prevalence of 46% in a sample of 13-year-old school children (*N* = 63) (Golombek, Marton, Stein, & Korenblum, 1986). In contrast, Bernstein et al. (1993) found a rate of only 3% severe borderline disturbance in the largest community sample to date that investigated the prevalence of BPD in children (*N* = 733; 9- to 19-year-olds).

The clear sex difference in prevalence rates previously demonstrated for adults are not as clear for juvenile samples. As indicated in Table 1, in contrast to adult samples, comparable prevalence rates have been demonstrated for boys and girls in a community sample (Bernstein et al., 1993).

Comorbid disorders

Skodol and colleagues (2002a) reviewed 16 adult comorbidity studies that used structured diagnostic interviews for both Axis I and Axis II disorders. The prevalence of BPD in outpatients with comorbid major depressive disorder ranged from 1.5% (Benazzi, 2000) to 34% (Sullivan, Joyce, & Mulder, 1994). Prevalence rates are higher in individuals being treated for substance abuse, with estimates ranging from 16% (Nurnberg et al., 1993) to 61.4% (Grillo et al., 1997). Across the 13 studies in Skodol et al.'s (2002a) review that measured BPD, 594, or 27.5%, of the entire sample (*N* = 2,158) had BPD.

Furthermore, patients with BPD are much more likely to carry an Axis I diagnosis. For example, in Skodol et al.'s (1999) sample of 571 patients with personality disorders, 39.2% of those with BPD met criteria for one or more mood disorders, with major depression being the

most common diagnosis (31.3%). In another sample of 409 nonpsychotic outpatients, Zimmerman and Mattia (1999) found that all but 1 of the 59 patients with BPD had a concurrent Axis I diagnosis; 69.5% had three or more Axis I diagnoses. Zanarini and colleagues (1998a) interviewed 379 inpatients with BPD and 125 inpatients with other personality disorders. They found that a significantly higher percentage of those with BPD met *DSM-III-R* criteria for major depression (82.8%), panic disorder (47.8%), social phobia (45.9%), posttraumatic stress disorder (55.9%), and eating disorder not otherwise specified (26.1%), compared to those without BPD. Although the rate of substance use disorders was not higher than the rate in patients with other personality disorders, it was nonetheless fairly high (64.1%). This rate is consistent with the results of Trull, Sher, Minks-Brown, Durbin, and Burr's (2000) review of 17 studies that provided comorbidity rates of BPD and substance use disorders. Across studies that reported rates of nonspecified substance use disorder, 57.4% of individuals with BPD received a substance use disorder diagnosis. Across studies that provided rates of alcohol use disorders, 48.8% of participants with BPD met criteria for alcohol abuse or dependence. Across studies that provided rates of drug use disorders, 38% of individuals with BPD met criteria for drug abuse or dependence. Furthermore, rates of comorbid Axis I disorders decrease over time, yet remain relatively high. The largest follow-up study to date (Zanarini et al., 2004) reported that at the 6-year follow-up point, 75% of patients with BPD had a mood disorder, 60% had an anxiety disorder, 34% had an eating disorder, and 19% had a substance use disorder. However, remission of BPD was associated with sharper decreases in comorbid Axis I diagnosis. In summary, BPD is highly comorbid with Axis I disorders, particularly mood, anxiety, and substance use disorders.

In addition, BPD is often comorbid with other Axis II disorders. In a study of 50 adult inpatients (Becker, Grilo, Edell, & McGlashan, 2000), BPD was significantly comorbid with antisocial personality disorder (26% of the 50 patients with BPD also had antisocial personality disorder). However, in this sample, other Axis II disorders co-occurred with BPD frequently but at rates similar to those in inpatients without BPD. Zanarini and colleagues (1998b) found that patients with BPD could be discriminated from those without BPD by the comorbidity of paranoid, avoidant, dependent, and self-defeating personality disorders. Using the same sample, Zanarini et al. (2004) found that, by follow-up, both remitting and nonremitting patients with BPD had declining rates of most types of Axis II disorders. However, even 5 to 6 years after the initial assessment period, 59% of nonremitted border-

line patients met criteria for avoidant personality disorder, 45% for dependent personality disorder, and 27% for self-defeating personality disorder (*DSM-III-R* diagnoses were used). Such high rates of comorbidity have led some authors to suggest that BPD is primary in a hierarchy of personality disorders. Although some evidence exists to suggest the superordinate status of BPD with regard to severity of impaired functioning (e.g., Hopwood et al., 2006), the data do not support an overarching hierarchy with BPD at the top.

Few comorbidity studies have been conducted for juvenile BPD. Similar to adult BPD, juvenile BPD seems to be highly comorbid with antisocial behavior. In an incarcerated juvenile sample, almost half of female conduct-disordered adolescents met criteria for BPD, while a significant percentage of boys met criteria for antisocial personality disorder (Eppright et al., 1993). Similar findings were demonstrated in a sample of adolescent inpatients (Myers, Burket, & Otto, 1993). In addition, Guzder et al. (1999) found that conduct disorder was the only Axis I disorder that was significantly more prevalent in adolescents with BPD than in those without BPD. In addition, in a sample of female inpatients, Goodman, Hull, Clarkin, and Yeomans (1999) showed that antisocial personality disorder symptoms before age 15 predicted later BPD. In a sample of male adult inpatients, Modestin, Matutat, and Wurmlé (2001) showed that over half of the conduct-disordered patients developed borderline and/or antisocial personality disorder.

These findings were confirmed in the only longitudinal follow-up study so far to address the issue of antisocial behavior as a precursor to BPD in adulthood. Crawford, Cohen, and Brook (2001b) examined the relationship over time between Cluster B personality disorder symptoms (borderline, histrionic, and narcissistic symptoms) and comorbid internalizing and externalizing symptoms in a community sample of 407 adolescents. They tested the hypothesis that Cluster B symptoms reflect primary disturbances that give rise to co-occurring internalizing and externalizing symptoms, and they tested the alternative hypothesis that these Axis I symptom clusters reflect primary problems that interfere with normal personality development. Their findings did not clearly support one hypothesis over the other. Instead, their findings suggested that externalizing disorder in adolescence might precede BPD in girls, while Cluster B symptoms in adolescence might precede externalizing problems in boys.

Course

Skodol et al. (2002b) reported on 10 reviews of longitudinal studies of the course and stability of personality disorders and found less stability

and more heterogeneity in the course of BPD than would be suggested by the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*: “An enduring pattern of inner experience and behavior . . . [that is] stable and of long duration” (APA, 2000, p. 689). In contrast, dimensional (as opposed to categorical) measures of traits and functional impairment associated with BPD are more stable. More recently, two longitudinal studies have supported Skodol et al.’s review. Zanarini, Frankenburg, Hennen, and Silk (2003) demonstrated that 34.5% of 290 patients with BPD met criteria for remission at 2 years, 49.4% at 4 years, and 68.6% at 6 years, while approximately 25% of the sample never met criteria for remission. Among those who remitted, recurrence rates were low (only 5.9% over the 6 years of study). In a longitudinal study of 621 participants with schizotypal, borderline, avoidant, and/or obsessive-compulsive disorders, Shea et al. (2002) demonstrated that, relative to participants with major depressive disorder, significantly higher proportions of participants with each of the personality disorders remained at full criteria although the number of symptoms decreased. With regard to BPD specifically, 41% of those initially diagnosed remained at full criteria at 12 months, and the mean number of BPD criteria met by participants decreased from 6.8 to 4.2, a significant decrease.

Although these studies suggest less overall stability of the diagnosis, there are factors that are associated with greater stability. For example, poor prognosis is associated with childhood sexual abuse (Paris, 1993) and incest (Stone, 1990). Other factors predicting diagnostic stability include affective instability, impulsivity, substance abuse, and an increased number of comorbid Axis I and/or Axis II disorders (Skodol et al., 2002b). In contrast, factors associated with good outcome include high IQ (e.g., Stone, 1990), as well as low narcissistic entitlement, absence of parental divorce, and the presence of self-destructive acts at the time of admission (Plakun, 1991).

Although few studies of the kind described above have been carried out in children and adolescents, the same pattern of instability in childhood and adolescent BPD appears to be described in juvenile samples. Garnet et al. (1994) carried out a short-term longitudinal study in a small sample ($N = 21$) of 15- to 19-year-olds diagnosed with BPD. Only one third of subjects still met criteria for BPD 2 years after baseline. Of all the BPD symptoms, emptiness and boredom were the most stable. Meijer, Goedhart, and Treffers (1998) demonstrated similar results in a group of 14 inpatients. Three years after baseline, only two subjects still met criteria for BPD. In a larger sample of adolescent female inpatients ($N = 156$), Levy et al. (1999) showed that personality disorder was not predictive of overall functioning or the presence of personality disorder

2 years after baseline, but did predict drug use and increased use of inpatient services. Levy and colleagues suggested that personality disorder in adolescence may be a point-in-time disturbance rather than enduring mode of functioning.

Although useful in providing preliminary data on the stability of BPD in childhood, the above studies were plagued with methodological difficulties: The sample sizes were small and the follow-up periods have stopped short of adulthood. Thus, these studies tell us little about continuity into adulthood or about predictors of juvenile BPD. In addition, they were conducted with adolescents in treatment. The low proportion of subjects retaining a BPD diagnosis may therefore reflect change due to treatment, not change due to time.

To rectify these methodological problems, Johnson, Cohen, Brown, Smailes, and Bernstein (1999) followed up a large community-based study of 9- to 16-year-olds. Children with a BPD diagnosis were 13 times more likely to still have the diagnosis at 2-year follow-up compared with those who did not have a diagnosis. Despite the bigger sample size in this study and overcoming the problem of confounding change due to time with change due to treatment, the study still could not shed light on the continuity of BPD into adulthood due to the short follow-up period.

The Children in the Community Study (CCS) under the leadership of Cohen improved these methodological limitations (Johnson, Smailes, Cohen, Brown, & Bernstein, 2000). Children with a mean age of 14 years were followed up at ages 16 and 22. The authors found a 59% linear decline in the number of BPD features over the study period, with a rank order stability of $r = .31$.

In another longitudinal study, Crawford, Cohen, and Brook (2001a) examined dramatic-erratic personality disorder symptoms (histrionic, borderline, and narcissistic symptoms) in a community sample of 407 adolescents to assess the meaningfulness of this diagnostic construct in young people. Cluster B symptoms were found to be highly stable across an 8-year period from early adolescence to early adulthood. When compared with internalizing and externalizing symptoms, Cluster B symptoms were more stable over time than these well-established Axis I symptom clusters. They were, however, also highly correlated with co-occurring internalizing and externalizing symptoms.

In summary, the diagnosis of BPD is not as stable in adults and youth as the *DSM* criteria would suggest. Fairly high percentages of individuals do not meet full criteria at follow-up after initial diagnosis. However, even in the absence of the BPD diagnosis, many of those with a history of BPD remain symptomatic and functionally impaired (Skodol

et al., 2002b), and numerous factors moderate the course of the illness—factors that remain unknown for juvenile populations.

Environmental risk factors

Although BPD is a complex condition without a single cause, several early environmental risk factors have emerged in the literature. Perhaps the most common event that increases risk is childhood neglect and abuse, particularly sexual abuse. Numerous studies indicate higher rates of abuse and neglect (e.g., Battle et al., 2004; Links, Steiner, Offord, & Eppel, 1988; Shearer, Peters, Quaytman, & Ogden, 1990; Westen, 1990). Furthermore, accounting for measurement differences, 40%–71% of inpatients with BPD retrospectively report having been sexually abused as children (e.g., Herman, Perry, & van der Kolk, 1989; Paris, 1994; Shearer et al., 1990; Westen, Ludolph, Misle, Ruffins, & Block, 1990; Zanarini, Gunderson, Marino, Schwartz, & Frankenburg, 1989; Zanarini et al., 1997), much higher than the base rates. However, by no means do all individuals who experience sexual abuse develop BPD, nor do all individuals with BPD have a history of childhood sexual abuse. This has led some researchers (e.g., Zanarini et al., 2002) to examine the association between the severity of childhood sexual abuse and the severity of borderline pathology, and to hypothesize that the secrecy of sexual abuse may be a particularly pathogenic aspect of the abuse (e.g., Jacobson & Herald, 1990).

Another potentially important risk factor appears to be the quality of an individual's early attachments. Gunderson (1996) hypothesized that individuals with BPD have core problems with aloneness and abandonment, secondary to insecure attachments. This hypothesis has been supported by numerous studies demonstrating that BPD patients have insecure attachments characterized by extreme neediness alternating with fear of involvement (Bartholomew, Kwong, & Hart, 2001). Furthermore, the increased reliance on transitional objects in adults with BPD likely reflects insecure attachment (Skodol et al., 2002a). Both abuse and insecure attachments are associated with what Linehan (1993) has described as the "invalidating environment." Linehan has theorized that a persistently invalidating environment interferes with a child's ability to develop the necessary skills for emotion regulation, distress tolerance, and self-management—all of which are generally deficient in individuals with BPD. Fonagy, Gergely, Jurist, and Target (2002) also suggest a central role for attachment relationships in the development of dysfunctional affect regulation and social cognition (mentalizing) in BPD. As of yet, no child data for juvenile BPD populations exist to support these convincing theoretical positions.

Other significant risk factors include experiences of disturbed parental involvement during childhood (Goldberg, Mann, Wise, & Segall, 1985), childhood problems of anxiety and intolerance of separations, frustration, and mood regulation (Reich & Zanarini, 2001); childhood attachment problems (Agrawal, Gunderson, Bjarne, Holmes, & Lyons-Ruth, 2004); parental separation and loss (Paris, Zweig-Frank, & Guzder, 1994a, 1994b); and symptoms of externalizing disorder combined with abnormal neuropsychological functioning, physical abuse, and separations (Greenman et al., 1986). Most of these studies have been retrospective in design and report that adult patients with BPD indicate higher rates of abuse and neglect during childhood (Battle et al., 2004; Links et al., 1988; Shearer et al., 1990; Westen, 1990; Zanarini et al., & Frankenburg, 1989; Zanarini et al., 1997).

Few studies have looked at whether any of the above distal or more proximal predictors and risk factors are also present in children and adolescents. However, those studies that exist have shown strikingly similar correlates to those of adult BPD. Such correlates include trauma, neglect, maltreatment and separation (Famularo, Kinscherff, & Fenton, 1991; Johnson et al., 2000; Kestenbaum, 1983; Lofgren, Bemporad, King, Lindem, & O'Driscoll, 1991; Ludolph et al., 1990; Rogosch & Cicchetti, 2004) and exposure to sexual and physical abuse (Goldman et al., 1992), as well as serious parental psychopathology, including depression, substance abuse, or antisocial personality (Goldman, D'Angelo, & DeMaso, 1993).

It should be noted that correlational evidence does not permit inference of causality; longitudinal studies have not yet been conducted to determine whether correlates that are measured concurrently are predictive in the sense of being a true etiological risk factor.

Neurocognitive and biological risk factors

Extensive evidence has documented the centrality of affective dysregulation and behavioral impulsivity in individuals with BPD (e.g., Bohus, Schmahl, & Lieb, 2004; Skodol et al., 2002a, 2002b). Two of many examples can be cited: In self-report formats, individuals with BPD report greater frequency and intensity of negative emotional experience (e.g., Stiglmayr, Shapiro, Stieglitz, Limberger, & Bohus, 2001), and individuals with BPD are between 2.12 and 3.53 times more likely to attempt suicide than healthy controls (Oquendo et al., 2007). Several studies have begun to look at neurological deficits associated with these aspects of BPD in particular (Berlin & Rolls, 2004; Bland, Williams, Scharer, & Manning, 2004; Dinn et al., 2004; Stevens, Burkhardt, Hautzinger, Schwarz, & Unkel, 2004; Wagner & Linehan, 1999). At

the physiological level, BPD has been associated with hyperarousal (e.g., increased heart rate) in response to emotional stimuli in daily life situations, although there is also evidence of hypoarousal in response to affect-eliciting pictures (Bohus et al., 2004). At the endocrinological level, hyperresponsiveness of the hypothalamic–pituitary–adrenal (HPA) axis has been documented in patients who have both BPD and a history of early traumatization (Rinne et al., 2002). At the structural level, BPD has been associated with reduced hippocampal and amygdala volumes, as well as reduced frontal and orbitofrontal volumes, relative to the regional volumes of healthy control subjects (e.g., Driessen et al., 2000; Tebartz van Elst et al., 2003). However, it is unclear whether these differences were present premorbidly, or whether they resulted from the disorder.

At the functional level, imaging studies of individuals with BPD have suggested abnormalities in the anterior cingulate cortex, orbitofrontal and dorsolateral prefrontal cortex, hippocampus, and amygdala (Lieb et al., 2004). For example, Soloff et al. (2003) demonstrated altered baseline metabolism in the prefrontal regions of BPD patients. fMRI studies have also shown differential activation in patients with BPD compared to controls, especially for the amygdala in response to emotional stimuli (Herpetz et al., 2001) and faces (Donegan et al., 2003). These brain regions are all involved in the neurotransmission of serotonin, which has also been investigated in BPD because of its association with impulsivity (e.g., Coccaro, Bergeman, Kavoussi, & Seroczynski, 1997). Both neuroimaging and pharmacological studies have implicated reduced serotonin in the impulsive and aggressive behaviors, as well as in the affective instability associated with BPD (Gurvits, Koenigsberg, & Siever, 2000). In addition to serotonin, dysfunctional dopaminergic transmission has also been suggested as a mechanism involved in affective dysregulation, impulsivity, and cognitive–perceptual impairment (e.g., Friedel, 2004).

Studies of the neurobiology of juvenile BPD are virtually nonexistent. Some neuropsychological studies have been conducted, which have demonstrated similar correlates for juvenile BPD as have been demonstrated for adults. Rogosch and Cicchetti (2005) showed that children with high levels of BPD precursors demonstrate deficits in the processing of the conflict attention network (a measure of executive functioning). Executive functioning difficulties associated with BPD symptoms were also demonstrated to be independent of conduct problems in 94 school-age children (Paris et al., 1999), while both executive functioning difficulties and trauma made independent contributions to the prediction of a BPD diagnosis in a sample of 86 school-age children in another study (Zelkowitz, Paris, Guzder, & Feldman, 2001).

Genetic factors

Numerous studies have provided evidence for familial aggregation of BPD or BPD traits (Siever et al., 2002), with higher rates of the diagnosis in relatives of individuals with BPD than in comparison subjects. However, these studies have not been able to tease apart genetic and environmental contributions to the development of the disorder. Research on genetic factors in BPD remains in the early stages. One twin study by Torgersen (1984) found a concordance rate of zero in 7 monozygotic (MZ) twin pairs and 11% in 18 dizygotic (DZ) twin pairs; however, as Siever et al. (2002) note, the small sample size limits interpretations of the findings. More recently, Torgersen et al. (2000) conducted a larger twin study, looking at 92 MZ and 129 DZ twin pairs. The concordance rate in MZ twin pairs was 35%, compared to 7% for DZ twin pairs, suggesting a strong genetic component in BPD. Furthermore, research has documented large heritability coefficients for BPD traits such as impulsive aggression (41%) (Coccaro, Bergeman, & McClearn, 1993) as well as higher-order personality factors such as emotion dysregulation (e.g., Livesley et al., 1998). There is also emerging evidence from twin studies (e.g., Livesley, Jang, & Vernon, 2003) that borderline pathology may reflect a combination of several latent traits, each of which is directly influenced by genetic and environmental factors, rather than a single heritable entity (Skodol et al., 2002b).

To our knowledge, no genetic studies of BPD symptomatology or disorder have been conducted using child or adolescent samples.

Conclusion

The purpose of this review was to compare research findings on diagnostic-related phenomena in child and adolescent samples with those in adult samples to establish the utility of the BPD construct in childhood and adolescence. Similarities in diagnostic-related phenomena across children and adults do not of course guarantee the validity of the BPD construct in juveniles. In fact, the principle of heterotypic continuity suggests developmentally determined manifestations of disorder that differ across age ranges. However, if some continuity can be established between adult and juvenile BPD, confidence in the construct of the latter is significantly bolstered (Sharp & Bleiberg, 2007).

As summarized in Table 1 and discussed in the review, juvenile BPD overlaps with adult BPD in important ways. For instance, similarities between juvenile BPD and adult BPD have been demonstrated in its diagnostic criteria, interview-based measures, comorbidity with antisocial behavior, the stability of the diagnosis, and environmental risk

factors. This review also testifies to some differences between adults and children. For instance, prevalence rates for juvenile BPD seem to be generally more elevated compared with adults, and the overrepresentation of BPD in adult females seems not to be reflected in child populations.

Despite these differences, the similarities between adult and juvenile BPD seem to outweigh the differences. However, it is also clear from this review that significant research has yet to be conducted before the diagnostic category of juvenile BPD will be included in the psychiatric nomenclature. Specific areas for future research include studies of comorbidity, measure development, and the use of neurobiological measures such as functional neuroimaging. In addition, there is a clear need for longitudinal follow-up studies that are aimed at describing the early precursors of BPD.

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