

BRIEF REPORT

Neuropsychological Dysfunction in Children with Borderline Personality Disorder Features: A Preliminary Investigation

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Borderline personality disorder (BPD) is often linked to neuropsychological deficits, yet few studies have examined BPD or its features and concomitant neuropsychological dysfunction in childhood. This study examined children with borderline features ($n = 21$) using the Coolidge Personality and Neuropsychological Inventory for Children and compared them to controls ($n = 21$) with features of at least one personality disorder, but not BPD. As hypothesized, the BPD group scored significantly higher than the control group on the Attention Deficit/Hyperactivity Disorder, Executive Functions Deficits, Mild Neurocognitive Disorder, Conduct Disorder, and Oppositional Defiant Disorder scales. It appears that behavioral disturbances associated with BPD are linked strongly with neuropsychological dysfunction. Because none of the children with BPD features in the present sample had a history of traumatic brain injury (TBI), it appears likely that TBI in the histories of adult BPD patients may not be the cause of BPD, but traits such as anger and impulsivity in BPD may cause TBI. Clinical implications and future research directions are discussed. © 2000 Academic Press

Borderline personality disorder (BPD), characterized by a pervasive and chronic pattern of unstable relationships, affective instability, self-damaging impulsivity, and identity problems, is a debilitating disorder believed to be present in 1 to 2% of the population (American Psychiatric Association, 1994). Livesley, Jang, Jackson, and Vernon (1993) found that identity problems as well as affective lability were highly heritable (59 and 49%, respec-

The CPNI is available, *pro bono*, for research.

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tively) in a sample of 175 adult twin pairs. They postulated that a stable sense of self might require regulatory and integrative processes free from early neuropsychological dysfunction.

Indeed, a growing body of research supports the comorbidity of brain dysfunction in BPD, including an early history of organicity, such as a prior head injury (van Reekum, 1993), minimal brain dysfunction (Murray, 1979), childhood hyperactivity (Soloff & Millward, 1983), and learning disabilities, brain trauma, encephalitis, or epilepsy (Andrulonis, Glueck, Stroebel, & Vogel, 1980). Notably, Kimble, Oepen, Weinberg, Williams, and Zanarini (1997) found that adult patients with neurological dysfunction were six times more likely to be diagnosed as having BPD than patients without some form of neurological dysfunction. Neurological soft signs have also been found to be associated with adult BPD patients (Gardner, Lucas, & Cowdry, 1987; Quitkin, Rifkin, & Kline, 1976). Andrulonis and Vogel (1982), in a study of 106 adult BPD patients, statistically derived three subtypes: (a) BPD without a prior history of organicity, (b) BPD with an acquired history of neurological dysfunction, and (c) BPD with an early history of organicity and/or developmental deficits, such as learning disabilities or attention-deficit/hyperactivity disorder (ADHD). They proposed that the latter two groups should be considered part of a spectrum of organic brain dysfunction syndromes whose patients are most often male with a family history of drug and alcohol abuse.

Most studies to date, however, have examined the comorbid evidence of neuropsychological dysfunction in adult BPD patients in retrospective designs, despite evidence that BPD or its associated traits may be diagnosed early (Bemporad, Smith, Hanson, & Cicchetti, 1982; Kernberg, 1983; Lewis, 1976; Verhulst, 1984). It may be useful to study BPD in childhood or early adolescence in order to assess current comorbid symptoms of neurological dysfunction [e.g., ADHD, executive function (EF) deficits of the frontal lobes, or mild neurocognitive disorder (MND)].

The purpose of the present pilot study was to examine a group of children with borderline features and to compare them to a matched control group who had features of at least one personality disorder, but not BPD. It should be noted that the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV*; American Psychiatric Association, 1994) does not list any neuropsychological deficits in the diagnostic features or associated features of BPD. The *DSM-IV* does mention ADHD as a common co-occurring Axis I disorder. It was hypothesized that children with BPD features would have greater neuropsychological dysfunction, including EF deficits, and that they would be more likely to have associated clinical disturbances of ADHD, Conduct Disorder (CD), and Oppositional Defiant Disorder (ODD) compared to control children.

METHOD

Participants and Procedure

The present clinical sample was obtained from a variety of settings (e.g., private practice, outpatient psychiatric hospital, and schools). The parents of the children (typically the mother) were asked to complete the 200-item, Coolidge Personality and Neuropsychological Inventory for Children (CPNI; Coolidge, 1998; Coolidge, Thede, & Stewart, 2000) along with a demographic survey that assessed a variety of relevant issues, including prior childhood illnesses, diseases, and head injuries. The parents were recruited through psychology students who received extra credit for their recruitment of respondents. The parents received no remuneration and were simply asked to volunteer. Informed consent was obtained. None of the respondents were aware of the hypotheses of the study at the time that they completed the CPNI. The CPNI and the demographic survey was completed in a single session, either at the university, at home, or in a clinical setting.

The final clinical sample consisted of 21 children (M age = 11.1, SD = 3.6, range = 5 to 17 years; M education = 5.9 years, range = K–12; 9 male and 12 female; 67% Caucasian) whose T score on the borderline scale of the CPNI was greater than or equal to 70 (98th percentile or greater in the normative sample). A control group (n = 21), matched on age, was selected from a large sample of convenience (n = 654) that was initially collected as part of the norming process for the CPNI. The control children were selected if they had at least one personality disorder scale T score greater than or equal to 60 (84th percentile or greater), but not the borderline scale. The control group averaged 1.4 elevated personality disorder scales per child (T score > 60), and those scales were as follows: Schizoid, 33%; Obsessive-Compulsive, 29%; Conduct Disorder, 24%; Histrionic, 14%; Dependent, 14%; Narcissistic, 10%; Avoidant, 10%; Schizotypal, 5%; and Paranoid, 5%. Their mean age was 11.2 (SD = 3.8, range = 5 to 16; M education = 5.7, range = K–11; 14 males and 7 females; 67% Caucasian). It is also important to note that according to the demographic survey, none of the parents reported a prior head injury for the children in either the clinical or the control groups.

Measure

The CPNI has a threefold purpose: (a) to assess 12 personality disorders (including BPD) as well as CD according to the criteria in the *DSM-IV*; (b) to measure neuropsychological dysfunction including ADHD (according to the criteria in *DSM-IV*), MND (according to the criteria in Appendix B of *DSM-IV*), EF deficits, and maturational delays; and (c) to assess other *DSM-IV* Axis I clinical disorders such as ODD. The CPNI uses a 4-point Likert scale (1 = *strongly false*, 2 = *more false than true*, 3 = *more true than false*, and 4 = *strongly true*) and is designed to be filled out by a primary caregiver. The CPNI also has a validity scale to measure the level of the respondent's denial. The CPNI normative sample consists of 329 children ages 5 to 17 years old. The 12 personality disorder scales of the CPNI have a median scale reliability of .72 and a median test–retest reliability of .81. Specific scale characteristics on the CPNI used in the present study are as follows: the BPD scale has a scale reliability of .62, a test–retest reliability of .67, and contains 9 items covering the nine criteria for BPD in *DSM-IV*. The CD scale has a scale reliability of .79, a test–retest reliability of .87, and contains 15 items covering the 15 criteria in *DSM-IV*. The ADHD scale has a scale reliability of .91, a test–retest reliability of .83, and contains 18 items covering the 18 criteria for ADHD in *DSM-IV*. The EF deficits scale has a scale reliability of .86, a test–retest reliability of .81, and contains 8 items that assess decision-making difficulties, organizational impairment, troubles with planning, perseveration, and sequencing difficulties. The ODD

scale has a scale reliability of .84, a test-retest reliability of .67, and contains eight items covering the eight criteria in *DSM-IV*. The MND scale has a scale reliability of .89, a test-retest reliability of .77, and contains 16 items covering the five major categories of symptoms (memory impairment, disturbances in executive functioning, disturbances in attention or speed of information processing, impairment in perceptual-motor abilities, and language impairment) for the disorder listed in Appendix B of *DSM-IV*. The Maturation Delay scale has a scale reliability of .54, a test-retest reliability of .81, and contains 5 items assessing encopresis, enuresis, and delays in motor and language skills. Validity studies support the use of the CPNI in various clinical and nonclinical settings (e.g., Coolidge, Aksamit, & Becker, 1994; Coolidge et al., 1992; Coolidge, Thede, & Jang, 1999; Coolidge, Thede, & Stewart, 2000; Coolidge et al., 1990; Friedman, 1998; Philbrick, 1990; Reilman, 1993).

RESULTS

Multiple *t* tests were performed between the two groups using Holms' (1979) modified Bonferroni correction to control for experimental Type I and Type II error rates. Results of these analyses are summarized in Table 1. Consistent with the initial hypotheses, the BPD group was significantly elevated on all six scales compared to the control group. The median correlation of effect size was $r = .45$, ranging from $r = .43$ for the Maturation Delay and the MND scale to $r = .65$ for the ODD scale (where $r > .371$ is considered a large effect; Rosnow & Rosenthal, 1996). In order to determine the specific areas of neuropsychological dysfunction, the 16 items on the MND scale were analyzed individually by group. This analysis revealed that the BPD group had a higher mean than the control group on all 16 items while six of these items were significantly different between the two groups.

TABLE 1
A Summary of *t* Tests and Effect Sizes between the BPD and Control Groups on the CPNI Scales

Scale	BPD			Control			<i>t</i>	<i>p</i>	Effect size ^a
	<i>M</i>	(<i>SD</i>)	%	<i>M</i>	(<i>SD</i>)	%			
CD	73.2	(21.9)	71%	54.3	(12.5)	29%	3.43	.001	.47
ODD	67.6	(10.2)	81%	51.9	(8.1)	19%	5.51	.001	.65
MND	66.3	(14.7)	67%	54.4	(10.7)	29%	3.00	.005	.43
ADHD	65.0	(14.3)	57%	54.3	(7.3)	29%	3.06	.004	.44
EF	63.1	(10.6)	57%	52.3	(9.8)	14%	3.45	.001	.48
MATDEL	63.3	(18.6)	52%	50.0	(8.3)	14%	3.00	.005	.43

^a as determined by the correlation of effect size where $r > .371$ is considered large.

Note. Percentages indicate percentage in each group that meets criterion at greater than 1 standard deviation above the normative CPNI sample ($N = 329$) mean. CD = Conduct Disorder; ODD = Oppositional Defiant Disorder; MND = Mild Neurocognitive Disorder; ADHD = Attention Deficit/Hyperactivity Disorder; EF = Executive Function Deficits; MATDEL = Maturation Delay.

Four of these six items were EF deficits such as planning, organizing, and sequencing difficulties. The other two items were memory and perceptual-motor difficulties.

DISCUSSION

The results appear to (a) support the general hypothesis that a majority of BPD children do exhibit symptoms of neuropsychological dysfunction, (b) support the specific hypothesis that a majority of BPD children appear to have symptoms of ADHD and EF deficits that may be associated with frontal lobe dysfunction, and (c) support the general hypothesis that a majority of BPD children have emotional and behavioral problems consistent with CD and ODD symptom patterns.

One of the important aspects of the present study is that, unlike previous studies, it did not employ an adult retrospective review of their childhood. The present results are consistent with previous findings (e.g., Andrulonis & Vogel, 1982; van Reekum, 1993) that BPD involves a specific neurocognitive dysfunction that does not appear to be explained by motivational or intrapsychic conflicts. It appears that at least one aspect of their neuropsychological problems may be frontal lobe dysfunction because the present sample of BPD children was significantly elevated on the EF deficits scale, and there is support for the contention that parents may be valid reporters of their children's EF deficits (e.g., Gioia et al., 1999; Silver et al., 1999). In addition, they were elevated on the ADHD scale, which previously has been linked to frontal lobe or frontal-striatal monitoring systems as well (e.g., Hechtman, 1994; Pennington & Ozonoff, 1996; Sherman, 1997). Furthermore, these BPD children were significantly elevated on the CD scale that has also been linked to frontal lobe dysfunction (Pennington & Ozonoff, 1996).

Preliminarily, the results appear to lend weight to the hypothesis that a subtype of BPD may exist without a concomitant history of brain trauma. This BPD subtype may manifest itself early in the dual primary form of behavioral disturbances (as evidenced by CD and ODD) coupled with neuropsychological dysfunction (as evidenced by maturational delays, EF deficits, ADHD, and MND symptoms). None of the BPD children in this study had histories of traumatic brain injury (TBI), yet adult BPD studies frequently report comorbid TBI. Cautiously, it may be suggested that TBI may not be a primary cause of BPD but it appears possible that the disinhibition, impulsivity, and anger in BPD may sometimes cause TBI through reckless and dangerous behaviors. The results also appear to be consistent with Spreen's (1989) hypothesis that neuropsychological deficits and psychopathology frequently occur comorbidly because they are most likely a function of a common biological origin (and most likely, according to Spreen, of a genetic basis). At least one implication of these findings is that the psychotherapeutic treatment of BPD may be enhanced when psychopharmacological interven-

tions are also employed. It may also be important to note that if the neuropsychological dysfunction is clearly substantiated in BPD children and adolescents then purely psychotherapeutic interventions may be limited.

One major reservation in the present study is that there were no corollary clinical investigations of the children in the BPD group to determine whether they met official *DSM-IV* criteria for a diagnosis of BPD. Future studies might include structured interviews and behavioral assessments. There was also no clinical neuropsychological evaluation to assess the validity of high scores on the CPNI neuropsychological scales, although there is new and substantial evidence that families can reliably and validly report a patient's neuropsychological dysfunction (Kreutzer, 1999). Certainly an elevated score on the BPD scale of the CPNI is not necessarily indicative of BPD. However, numerous studies support the contention that personality disorders represent continua of normal to abnormal behavior, and, thus, dimensional approaches to classification have been frequently used in clinical research (e.g., Jang, Livesley, Vernon, & Jackson, 1996). The present study was also limited by the small sample size, it did not differentiate on the basis of gender and age, it was restricted to children without a prior history of head injury, and it was not determined whether there might be differences between the parents of the two groups in terms of their socioeconomic status and their education. Future studies should increase sample size and diversity of participants to increase the generalizability of the findings and allow for an analysis of individual difference variables.

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