Etiology of Borderline Personality Disorder

Disentangling the Contributions of Intercorrelated Antecedents

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Abstract: A substantial body of research points to several variables relevant to the etiology of borderline personality disorder (BPD), notably childhood physical and sexual abuse, childhood family environment, and familial aggregation of both internalizing and externalizing disorders. However, these variables tend to be correlated, and few studies have examined them simultaneously. A national sample of randomly selected psychologists and psychiatrists described 524 adult patients with personality disorders. Family environment, parental psychopathology, and history of abuse all independently predicted BPD symptoms in multiple regression analyses. Sexual abuse contributed to the prediction of BPD symptoms over and above family environment, although family environmental factors such as instability partially mediated the effect. The results converge with recent studies using very different samples and methodologies.

Key Words: Borderline personality, personality, SWAP-200, etiology, child abuse, sexual abuse, family environment.

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Borderline personality disorder (BPD) is a relatively prevalent psychiatric disorder estimated to affect 1% to 2% of the population (Paris, 1999; Torgersen et al., 2001) and 10% to 25% of patients presenting in clinical settings (DSM-IV, American Psychiatric Association, 1994). Even more individuals appear to have subclinical features of the disorder (Westen and Arkowitz-Westen, 1998). Research over the period of the past 20 years has identified several factors likely to contribute to the development of BPD, including history of childhood abuse, unstable or otherwise toxic family environment, and family history of psychopathology. To what extent each of these factors, which tend to be highly correlated, contribute independently to the etiology of the disorder is only beginning to be understood.

Research has consistently identified a link between abuse, particularly childhood sexual abuse, and BPD (*e.g.*, Herman et al., 1989; Ogata et al., 1990; Zanarini, 1997). Herman et al. (1989) found that 81% of BPD patients had histories of childhood abuse, including both physical (71%) and sexual (67%). In a community-based longitudinal study of personality disorders (PDs), Johnson et al. (1999) found that childhood physical, sexual, and emotional abuse increased risk for development of 10 of the 12 DSM-IV PDs. However, when they adjusted for the effects of co-occurring PDs, only the cluster BPDs remained significantly related to experiences of childhood maltreatment. Zelkowitz et al. (2001) found that children who experience childhood sexual abuse (CSA) are four times more likely to develop BPD than those who do not.

Although the link between BPD and CSA is relatively consistent, rates of CSA in BPD patients vary considerably across studies, and physical abuse is only inconsistently linked to BPD (Golier et al., 2003; Westen et al., 1990). A recent meta-analysis found that the effect of CSA is small to moderate ($d = .28$; Fossati et al., 1999), suggesting that it accounts for only part of the variance. Several studies suggest that although the presence of abuse is itself important, its more specific characteristics may be relevant to the etiology of BPD and borderline symptoms more generally. Of particular significance are severity, chronicity, and age of onset of sexual abuse, and co-occurrence (and severity) of other forms of abuse and neglect (McLean and Gallop, 2003; Silk et al., 1995; Yen et al., 2002; Zanarini et al., 2002).

A methodological difficulty in establishing the link between childhood abuse and later psychopathology, including BPD, is that most research on traumatic precursors to PDs has not assessed the broader family environment. Given that severe and recurrent abuse tends to occur in families that do not acknowledge, prevent, or interrupt it, and that children exposed to one risk factor are more likely to be exposed to others (Rutter, 1992, 2002), the impact of sexual or physical abuse can be difficult to disentangle from broader family and parental dysfunction. For example, sexual abuse often co-

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occurs in the context of physical and emotional abuse and of neglect (Dong et al., 2003; Faust et al., 1995; Zanarini et al., 1999), and multiple types of abuse and neglect are related to higher risk for later psychopathology (Bifulco et al., 1998; Green et al., 2000). Nash et al. (1993) found that much of the impairment in women with a history of CSA was accounted for by family environment. Other studies, although identifying a significant contribution of family factors such as problematic attachment relationships, poor parental care, and emotional abuse and neglect, suggest that CSA also makes an independent contribution to psychopathology, including symptoms of BPD (Boney-McCoy and Finkelhor, 1996; Hill et al., 2000; Melchert, 2000; Molnar et al., 2001). To what extent sexual abuse contributes to BPD symptoms over and above other family environment factors is unclear (Russ et al., 2003).

Another potential confound in establishing the etiological significance of abuse in BPD is that heritable temperamental contributions can be confused with family environment effects. Borderline pathology is linked to a number of heritable personality traits, such as impulsivity, neuroticism, and affective lability (Paris, 2003; Skodol et al., 2002a). For example, impulsive aggression, which is partially heritable, is a central characteristic of cluster B disorders, particularly BPD and APD (Coccaro et al., 1993; Goodman and Yehuda, 2002; Nigg and Goldsmith, 1994; Skodol et al., 2002b; Torgersen, 1980; Torgersen et al., 2000). Making matters more complicated is that these traits, if also present in parents, are likely to make genetic, environmental, and interactive contributions to the developing child's personality pathology and Axis I symptomatology. A recent multivariate analysis of risk factors for borderline features in a sample of college students (Trull, 2001) found the influence of parental psychopathology to be largely mediated by temperamental traits of negative affectivity and trait disinhibition, although the study design did not permit the researchers to distinguish whether these traits reflected genetic or environmental influences.

The present study attempts to contribute to this literature by evaluating the relative contributions of childhood physical and sexual abuse, family environment, and history of parental psychopathology to the development of BPD symptoms. In addition, we attempted to determine whether family environment mediates or partially mediates the relationship between childhood abuse and BPD symptoms.

METHODS

We used a practice network approach, in which randomly selected, experienced clinicians provide data on patients that can be aggregated across large samples (Westen and Harnden-Fischer, 2001; Morey, 1988; Shedler and Westen, 2004; Westen and Chang, 2000; Westen and Shedler, 1999a, 1999b, 2000; Wilkinson-Ryan and Westen, 2000). Elsewhere we have addressed in detail the rationale for clinician-report data, including advantages and limitations (Dutra et al., 2004; Westen and Shedler, 1999a, 1999b; Westen et al., 2003; Westen and Weinberger, 2004). The primary advantage is that clinicians are trained, experienced observers, with skills and a normative basis with which to make inferences and recognize nuances in psychopathology. Clinician-report instruments are less vulnerable to defensive and self-presentational biases than self-reports (Shedler et al., 1993; Westen et al., 1997). Further, clinical observation is generally longitudinal, rather than based on a single interview or questionnaire completed on a single day. This can be particularly useful in studying symptoms and personality processes that wax and wane or are subject to mood-dependent reporting biases.

The most important objection to the use of clinicians as informants is the possibility of biases in clinical judgment (Garb, 1998). The research literature on clinical versus statistical prediction is often misinterpreted as suggesting that clinicians are poor informants, whereas this literature actually shows that statistical aggregation of data (whether self-reports, clinician-reports, court records, and so forth) is generally superior to global, informal aggregation of the same data (Grove et al., 2000; Meehl, 1954; Westen and Weinberger, 2004). Recent research suggests that clinicians can in fact make highly reliable and valid judgments if their observations and inferences are quantified using psychometric instruments. For example, correlations between treating clinicians' and independent interviewers' assessments of a range of variables on measures designed for use by experienced clinicians tend to be large, typically greater than .50 (generally ranging from $r = .50$ to .80; Hilsenroth et al., 2000; Westen and Muderrisoglu, 2003; Westen et al., 1997), and clinician-report personality data are associated with a range of variables in theoretically predicted ways, such as measures of adaptive functioning, attachment patterns, and family and developmental history (Nakash-Eisikovits et al., 2002; Westen et al., 2003). The structure of clinician-report data using well validated instruments for lay informants (*e.g.*, the Child Behavior Checklist; Achenbach, 1991) is also virtually identical to that obtained using more traditional informants (Dutra et al., 2004; Russ et al., 2003), suggesting that clinicians are not in fact idiosyncratic respondents. Concerns about potential respondent biases when a single informant per case provides all the data, although legitimate, apply equally to most of studies in psychiatry, which rely exclusively on a single informant (the patient, whether by interview or questionnaire). Empirically, clinician theoretical orientation has predicted little variance in recent research when clinicians are asked to describe a specific patient rather than their beliefs or theories of psychopathology (*e.g.*, Shedler and Westen, 1998, 2004).

Procedure

The data were collected as part of a study of personality disorders in the community. We surveyed a random national sample of psychiatrists and psychologists with at least 3 years experience postlicensure from the membership registers of the American Psychiatric and American Psychological Associations. Approximately one third of the clinicians initially contacted participated in the research, for a total of 524 participants. We asked clinicians to describe a patient currently in their care who met DSM-IV criteria for one of the 10 Axis II disorders and diagnoses that were included in DSM-III-R and the appendices to DSM-III-R and DSM-IV (depressive, passive-aggressive, self-defeating, and sadistic PDs). More complete details of the method and sampling procedure have been described elsewhere (Westen and Shedler, 1999a).

We solicited data on one patient per clinician to minimize biases resulting from rater-dependent variance. Clinicians provided no identifying information about the patient (such as name, initials, or Social Security numbers) and were instructed to use only information already available to them from their contacts with the patient so that data collection would not compromise patient confidentiality or interfere in any way with ongoing clinical work.

Measures

Measures of Personality Disorders

We obtained dimensional assessments of Axis II disorders in two ways. First, we asked clinicians to rate the extent to which the patient met criteria for each Axis II disorder (7-point rating scale: 1 = "not at all," 4 = "has some features," 7 = "fully meets criteria"). Validity of such ratings are supported by data from a recent study (unpublished data) in which clinicians made similar ratings and present/absent ratings for each of the Axis II criteria for all disorders randomly ordered. Clinicians' global ratings correlated r = .73 with number of criteria met for each disorder, a widely used dimensional measure of Axis II disorders (Livesley et al., 1993).

Second, we used PD scale scores from the Shedler-Westen Assessment Procedure 200 (SWAP-200), a Q-sort procedure for assessing personality pathology. To describe a patient, a clinician sorts each of 200 statements into eight categories, from those that are least descriptive of the patient (assigned a value of 0) to those that are most descriptive (assigned a value of 7). Thus, the procedure yields a numeric score (0 to 7) for each of 200 personality-descriptive statements. The patient's 200-item profile is then correlated with diagnostic prototypes of each of the Axis II disorders to yield scale scores for each PD (Westen and Shedler, 1999b). Research has shown high correlations between SWAP-200 descriptions made by treating clinicians and independent interviewers; between independent observers reviewing recorded interviews; and between clinician ratings and self-reported antisocial and borderline traits (Bradley et al., 2004; Shedler and Westen, 1998; Westen and Muderrisoglu, 2003). Personality disorder scale scores derived from the SWAP-200 correlate with a range of criterion measures in both adult and adolescent samples, such as history of suicide attempts, arrests, psychiatric hospitalizations, social support, Global Assessment of Functioning, and family and developmental history variables (Dutra et al., 2004; Nakash-Eisikovits et al., 2002; Westen et al., 2003).

Measures of Child Abuse, Family Environment, and Family History of Psychopathology: Clinical Data Form

The Clinical Data Form (CDF) assesses a range of variables relevant to demographics, diagnosis, adaptive functioning (GAF), and etiology. In previous research, we have found ratings of adaptive functioning (*e.g.*, GAF scores) to be highly reliable and to correlate strongly with ratings made by independent interviewers (Heim et al., 2004; Westen et al., 1997b). The CDF also assesses aspects of the patient's developmental and family history of potential relevance to etiology. Developmental history variables include quality of relationship with mother and father (1 = poor/conflictual, 7 = positive/loving); significant (more than 6 weeks) separations from a parent (0 = no, 1 = yes); general family stability (1 = chaotic, 7 = stable); general family warmth (1 = hostile/cold, 7 = loving); and physical abuse (rated as present/absent and in terms of severity, 0 = no physical abuse, 1 = occasional/mild, and 7 = frequent/severe) and sexual abuse (also rated as present absent and in terms of severity, 0 = no sexual abuse, 1 = exhibitionism, 4 = fondling, and 7 = penetration).

Considerable controversy exists regarding clinicians' judgment about history of sexual abuse. We asked clinicians to rate history of sexual abuse as present, unsure, or absent, and we instructed clinicians to mark as present only patients they felt confident had a history of sexual abuse. Recent research using the sampling procedures and methods used in this study finds that doctoral-level clinicians tend to be quite conservative in indicating confidence in sexual abuse history, tending to rate cases with questionable or ambiguous reasons for inference as unsure (Wilkinson-Ryan and Westen, 2000). When asked to identify reasons for their belief that a patient had a history of sexual abuse, more than 90% of clinicians cite items indicating involvement of authorities such as police or Department of Social Services, intact memories of sexual abuse before treatment, and corroboration from family members or court records, and most rely on the presence of multiple indicators. Clinicians' ratings of family environment tend to correlate strongly with data from a clinician-report version of the self-report Parental Bonding Inventory (Parker et al., 1979; Russ et al., 2003), which shows the same factor structure and correlates as the self-report version.

Finally, the CDF also measures family history of psychiatric disorders, presenting clinicians with a list of diagnoses and related problems (psychosis, bipolar disorder, major depression, anxiety disorder, alcohol abuse, prescription drug abuse, illicit substance abuse, criminality, suicide attempts and completed suicide) and asking them to check present, absent, or unsure for each in (a) first-degree relatives and (b) second-degree relatives. The form instructs clinicians to indicate presence only if they feel confident in their judgment, and to indicate absence or unsure if they are unclear, to minimize guessing and false-positives. Data from previous studies suggest that this method yields valid data (Westen and Shedler, 1999b), particularly with large sample sizes, in which the unreliability of any given judgment by individual clinicians tends to cancel over subjects. The relative validity of the data appears to reflect the fact that clinicians are very likely to have heard about a relative who has been in and out of psychiatric hospitals with delusions or hallucinations, has spent time in prison, has committed suicide, and so forth. Asking only about relatively obvious syndromes or diagnostic groupings (*e.g.*, history of any anxiety disorders, rather than specific anxiety disorders) has the advantage of preventing speculative diagnosis at a distance. Data comparing family history assessed by patient report with interviewing family members with structured interviews (Andreasen, 1986), like data comparing retrospective self-reports with objective (prospective) data on history of child abuse (Widom and Morris, 1997), suggest that underreporting tends to be more common than overreporting. Given that clinicians in this study ultimately rely on patients' reports, Type II error seems much more likely than Type I error, rendering the findings likely to be conservative.

RESULTS

Sample Characteristics

Psychiatrists comprised roughly 35% of the participants, with the remaining 65% being psychologists. The patients were 47% female and averaged 39 years old (SD, 11.53). The mean GAF score was 59.15 (SD, 14.69). Most were middle class (41%) or working class (38%). Patients were in treatment of a median of 24 sessions, suggesting that clinicians knew them well.

Although we asked clinicians to describe a patient with a specific Axis II diagnosis, secondary dimensional diagnoses were the norm on both clinician Axis II ratings and SWAP-200 PD scores. Thus, although 43 (8.2%) clinicians responded to our request to describe a patient with a diagnosis of BPD, when we asked clinicians to rate the extent to which the patient met criteria for each Axis II disorder on a 7-point rating scale, 121 (24.4%) rated their patients with a 4, 5, or 6, indicating patients with some or significant features of BPD. For the entire sample, the mean rating was 3.1 (SD, 1.9). Although the SWAP-200 is intended primarily as a dimensional measure of PDs, a *T* score $\geq$60 tends to indicate caseness for a given PD. In this sample, the average BPD *T* score was 49.9 (SD, 9.9), with 26.6% cases $\geq$60. With respect to developmental history variables, clinicians reported childhood sexual abuse in 16.2% of the sample, childhood physical abuse in 21.9%, and lengthy separations from a parent in childhood in 17.0%. Mean scores for family warmth, stability, and relationships with parents (7-point rating scales) are presented in Table 1.

Antecedents of BPD

In a first set of analyses, we examined zero-order correlations between our two measures of BPD (clinicians' 1–7 ratings and their SWAP-200 BPD scores) and specific items relevant to etiology from the CDF (ratings of abuse, family environment, and family history of psychiatric disorders). Table 1 reports the zero-order correlations, means, and SDs for each specific variable we examined. The data indicate significant associations between BPD symptoms and both sexual and physical abuse; family environment variables; and parental alcohol, drug, and anxiety disorders.

To assess the incremental predictive validity of childhood abuse variables, we next conducted hierarchical regression analyses to determine the degree to which measures of childhood abuse contribute to the prediction of BPD symptoms over and above measures of family environment and parental psychopathology. To maximize reliability of measurement, we standardized and aggregated the two measures of BPD (7-point ratings and SWAP-200 PD scores). To maximize reliability, reduce the number of predictor variables, and avoid problems of multicollinearity, we also created a composite family environment variable by standardizing and aggregating clinicians' ratings of family stability, family warmth, and parental relationships.

To assess the incremental predictive validity of childhood abuse variables in predicting BPD symptoms, we entered the composite family environment variable (and a related environmental variable, lengthy separations from parents) in the first step, and entered family history of disorders associated with BPD in the zero-order correlations (substance and anxiety disorders) in the second step. We then entered physical and sexual abuse in the third step.

As can be seen in Table 2, the overall model (family environment, lengthy separations, parental psychopathology, and childhood abuse) significantly predicted BPD symptoms ($F[6,442] = 15.05$; $p < .001$), accounting for 17% of the variance. In the first step, family environment accounted for a significant amount of the variance. However, lengthy separations did not make a significant contribution to BPD symptoms. Adding family psychopathology variables in the second step yielded a significant increase in prediction. However, the only significant predictor was family history of

TABLE 1. Correlations Among Potential Etiological Variables and BPD Scores

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. SWAP-200 BPD	—												
2. BPD 1–7 rating	.62***	—											
3. Family stability	−.32***	−.25***	—										
4. Family warmth	−.17***	−.19***	.31***	—									
5. Relationship with parents	−.27***	−.21***	.39***	.64***	—								
6. Lengthy separations	.05	.11*	−.11*	−.08	−.09*	—							
7. Sexual abuse	.18***	.22***	−.20***	−.19***	−.24***	−.14**	—						
8. Physical abuse	.18***	.24***	−.28***	−.49***	−.36***	−.13**	.36***	—					
9. Parental alcohol abuse	.07	.16***	−.28***	−.27***	−.24***	−.14**	.17***	.29***	—				
10. Parental drug abuse	.06	.10*	−.19***	−.12**	−.12**	−.08*	.22***	.20***	.30***	—			
11. Parental mood disorders	.03	.05	−.08	−.19***	−.08	−.13**	.06	.09*	.20***	.04	—		
12. Parental anxiety disorders	.06	.13**	.01	.01	.07	−.02	−.02	−.01	.01	.04	.16***	—	
13. Parental psychosis	−.03	.03	−.12**	−.05	−.05	−.08	.01	.08	.03	−.02	.07	−.004	—
N	499	472	524	524	524	524	524	524	524	524	524	524	524
M	.16	3.09	4.18	3.05	5.75	.17	.92	1.11	.34	.11	.30	.23	.03
SD	.18	1.94	1.72	1.26	2.27	.37	2.04	2.03	.48	.32	.46	.42	.16

*$p < .05$; **$p < .01$; ***$p < .001$.

TABLE 2. Hierarchical Regression Analyses Predicting Borderline Symptoms

Predictor	β	*t*	*p*	*R*	R^2	ΔR^2	*F* change	*p* change
Step 1				.35	.12	.12	31.37	.001
Family environment	−.34	−7.61	.001					
Lengthy separations	−.06	1.30	.19					
Step 2				.38	.14	.02	3.00	.03
Family environment	−.34	−7.12	.001					
Lengthy separations	−.05	1.17	.24					
Parental alcohol abuse	−.02	.35	.73					
Parental drug abuse	−.02	.40	.69					
Parental anx. disorder	.13	2.91	.004					
Step 3				.41	.17	.03	15.34	.001
Family environment	−.27	−.27	.001					
Lengthy separations	−.04	.84	.40					
Parental alcohol abuse	−.002	−.05	.96					
Parental drug abuse	−.01	−.23	.82					
Parental anx. disorder	.12	2.78	.01					
Child physical abuse	.09	1.75	.08					
Child sexual abuse	.14	3.04	.003					

anxiety disorders. As a conservative test of the hypothesis linking abuse to BPD, we entered abuse variables in step 3. As can be seen from the table, doing so yielded a significant *F* test, with childhood sexual abuse as a significant predictor of BPD symptoms and physical abuse showing a trend ($p = .08$).

Finally, in light of the correlations found in this as in other studies between abuse and family environment more generally, to examine the extent to which family environment mediates the relation between childhood sexual and physical abuse and BPD symptoms, we followed Baron and Kenny's (1986) recommendations for mediation analysis. We per-

formed two sets of regression analyses, one for sexual abuse and one for physical abuse. To assess family environment as a mediator of the impact of sexual abuse, we first conducted a regression with BPD symptoms as the criterion variable and childhood sexual abuse as the predictor variable ($\beta = .25$; $p < .001$). Next, we examined the relation between childhood sexual abuse and family environment ($\beta = –.26$; $p < .001$). We then entered family environment and childhood sexual abuse simultaneously into a regression equation with BPD symptoms as the criterion variable. We found a significant relationship between family environment and BPD symptoms ($\beta = –.30$; $p < .001$) and between childhood sexual abuse and BPD ($\beta = .17$; $p < .001$), although the standardized β for the latter was reduced. The Sobel (1988) z test was significant ($z = 4.35$; $p < .001$), indicating significant mediation. Thus, the data provide evidence for both a direct effect of child sexual abuse and an effect partially mediated by family environment (Figure 1).

We repeated the same procedures for testing family environment as a mediator of the relationship between childhood physical abuse and BPD symptoms. Childhood physical abuse predicted BPD symptoms, ($\beta = .26$; $p < .001$) and family environment ($\beta = –.43$; $p < .001$). When we entered family environment and childhood physical abuse simultaneously into a regression equation with BPD symptoms as the criterion variable, we found a significant relationship between family environment and BPD symptoms ($\beta = –.29$; $p < .001$) and between physical abuse and BPD symptoms, although the standardized parameter estimate for childhood physical abuse was reduced ($\beta = .13$; $p = .008$). As with childhood sexual abuse, the Sobel test was significant ($z = 5.22$; $p < .001$), indicating significant mediation. Thus, the data provide evidence for both a direct effect of child physical abuse and an effect partially mediated by family environment (Figure 2).

DISCUSSION

In this study, BPD symptoms were associated with three classes of variables identified in previous research and theory as relevant to etiology: childhood family environment, parental psychopathology, and childhood abuse. In multiple regression analyses, childhood sexual abuse contributed to the prediction of BPD symptoms over and above variables reflecting family environment; childhood physical abuse showed a trend in the same direction. We found evidence for

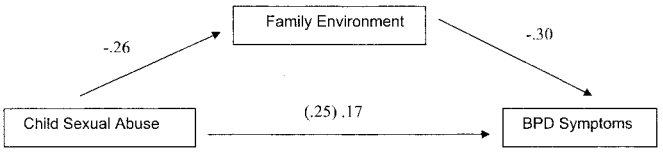

FIGURE 1. Mediation of the association between sexual abuse and BPD symptoms by family environment.

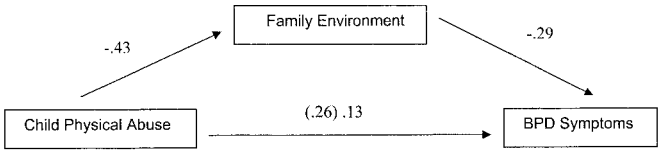

FIGURE 2. Mediation of the association between physical abuse and BPD symptoms by family environment.

partial mediation of the effects of both childhood sexual and physical abuse by family environment. Thus, our findings are consistent with other research (Nash et al., 1993b) suggesting that family environment may account for some but not all of the effects frequently attributed to abuse. Finally, familial psychopathology appears to have direct effects on borderline pathology, over and beyond its influence on family environment. The specificity to substance and anxiety abuse may suggest biological contributions (*e.g.*, toward impulsivity and negative affect, two of the salient features of the disorder; Skodol et al., 2002b) or environmental contributions not accounted for by global measures of parental pathology.

The study has three primary limitations. First, asking clinicians to describe a patient with a specific disorder (rather than collecting a random patient sample) could have somehow affected the pattern of findings. Although future research should attempt to replicate these findings using relatively nonselective samples (a project underway in our laboratory), several factors mitigate this concern. First, the findings are consistent with other research and with predictions, and it is difficult to imagine an explanation in terms of sampling artifacts that could make as much sense of the pattern of findings. Second, clinicians' selecting patients with a specific disorder should have attenuated the findings, leading to null rather than positive results, given that nearly over 480 of the 524 patients were selected for disorders other than BPD, and we used dimensional assessments on the entire sample as our criterion variables. Thus, if clinicians' theories of BPD influenced their ratings of family environment or abuse history, this should have led them to minimize such ratings in the 480 patients without a primary diagnosis of BPD.

A second limitation is that we did not operationalize BPD using DSM criteria. However, as noted, both clinicians' 7-point ratings and SWAP-200 PD scores are highly correlated with dimensional DSM-IV BPD diagnosis (number of BPD symptoms), and a relatively large body of research suggests that continuous measures of BPD tend to fare better than categorical diagnoses in predicting a range of variables (*e.g.*, Shea et al., 2002).

Third, as is the case with most research on BPD and sexual abuse, we relied on data from a single observer, in this case the treating clinician. Clearly the next step is to collect data from multiple sources, including multiple informants for variables such as family history of psychopathology and

family environment, and hospital and public records documenting abuse. However, the theoretical consistency of the findings, and their consistency with previous studies relying primarily on self-reports rather than clinician-reports, renders explanations in terms of artifacts of clinician observations unlikely. Once again, this is particularly true given that most of patients carried a diagnosis other than BPD, yet dimensional analyses across all 524 Ss demonstrated predicted associations across subjects selected for having an Axis II diagnoses other than BPD.

Implications

The results suggest that researchers interested in the association between sexual abuse and BPD and clinicians working with patients with BPD symptoms should assess a range of potentially significant etiological variables before attributing effects primarily to sexual abuse. Sexual abuse and physical abuse do appear to be important etiological contributors to borderline symptoms, but they typically occur in the context of a disturbed family environment, whose effects can be difficult to distinguish from the effects of abuse per se. Future research should address links between etiological correlates of BPD and specific characteristics of the disorder, such as affect dysregulation, identity disturbance, and impulsivity, which may have overlapping but distinct antecedents.

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