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The Role of Childhood Trauma in Differences in Affective Instability in Those With Personality Disorders

By Marianne Goodman, MD, Daniel S. Weiss, PhD, Harold Koenigsberg, MD, Vladimir Kotlyarevsky, MS, Antonia S. New, MD, Vivian Mitropoulou, MA, Jeremy M. Silverman, PhD, Karen O’Flynn, MD, and Larry J. Siever, MD

ABSTRACT

Background: This study examined the relationship of self-reported histories of childhood trauma to measures of affective instability in a sample of unmedicated outpatients with various personality disorders (n=174).

Methods: Childhood trauma was measured by the Childhood Trauma Questionnaire. Affective instability comprises at least two dimensions: affective lability, assessed using the Affective Lability Scale, and affective intensity, assessed using the Affective Intensity Measure.

Results: A history of emotional abuse was the only trauma variable that significantly correlated with the affect measures in the total sample (r=.20). More fine-grained analyses revealed that the relationship of emotional abuse and affective instability measures varied as a function of both gender and personality disorder type. In subjects with borderline personality disorder, the correlation for emotional abuse was greatly attenuated for both Affective Lability Scale (r=.10) and Affective Intensity Measure (r=.15) total scores.

Conclusion: This suggests that nontrauma-related factors may be more predominant in affective dyscontrol in individuals with borderline personality disorder.

CNS Spectr 2003;8(10):763-770

INTRODUCTION

Affective instability is characterized by alterations in the capacity of individuals to regulate and modulate their experience of mood states in terms of intensity, frequency, and stability. Affective instability is a frequent feature of the psychopathology of those carrying a personality disorder diagnosis. In contrast to the disturbances in Axis I mood disorders, such as major depression and bipolar disorder, which are typically more enduring departures from euthymia in the negative or positive direction, the affective dysregulation in Axis II disorders is characterized by markedly unstable affective states that are disturbingly transient and often rapidly reversible, and are more responsive to stimuli in the environment—both intimate and interpersonal. Emotional reactions to interpersonal precipitants, such as separation, criticism, disappointment, and frustration, may be especially intense. The impairments in the capacity to modify either the intensity or duration of predominantly negative emotional reactions disrupt the execution of one’s goals1 and result in significant behavioral and interpersonal difficulties. Despite its relative pervasiveness in the disruption of normal functioning in those with a variety of personality disorder difficulties, affective instability is explicitly included as a diagnostic criterion only for borderline personality disorder (BPD).

The earliest writings on the modern concept of personality disorders came from psychodynamic and psychoanalytic scholars2 who emphasized the relationship of stable affect to the development of self-esteem and the ability to maintain appropriate relationships. Impaired affect regulation was seen...
not only as a consequence of the disruption of early attachment but was understood to lead to disturbed attachments in adulthood, because of maladaptive responses to emotionally provocative interactions, and an accompanying unstable sense of self, identity diffusion, and continuing and self-fulfilling problematic interpersonal relationships. Our group has shown in a sample of adult patients with personality disorders, that affective instability is associated with identity disturbance, chronic feelings of emptiness, and suicidality. 

Empirical data have shown that affective states in adults are known to influence the way in which the world is experienced, encoded, and retrieved from memory. Presumably, this is true in children as well. A child with emotional vulnerability and impaired emotional regulation will be more sensitive and reactive to the inherent frustrations, disruptions, and separations in normal development. Efforts to mitigate these feelings include displays of distress that can range from clinging to tantrums. These can lead to chronic, repetitive, and maladaptive interactional patterns with others—the key feature of a personality disorder. Intense negative feelings may also affect self-image through the individual’s awareness of their own lack of capacity to modulate affect and, therefore, produce enduring feelings of shame, humiliation, and core defectiveness—additional key features of personality disorders.

The empirical literature on affective instability in personality disorders, including BPD, is limited but consistent with its presence. Cowdry and colleagues studied patients with BPD, with major depression, premenstrual syndrome, and normal controls and rated their mood for 14 straight days using visual analog scales. BPD subjects had the most variability in mood and each rating in mood had almost no relationship with the previous day. Levine and colleagues presented data that demonstrated those with BPD had greater intensity of negative emotions, lower levels of emotional awareness, and impaired ability to process mixed emotions as compared with controls.

In a study of hospitalized female BPD patients, the number of borderline traits was positively associated with affect intensity and affect control. A report from Koenigsberg and colleagues on a subset of the current data included 152 subjects with personality disorder and 42 with BPD, observed that affective instability in BPD did not involve all affects, but rather involved anger and anxiety and the oscillation between depression and anxiety. BPD diagnosis was significantly related to a measure of affect intensity but when age, gender, and comorbid Axis I disorder were controlled for, the association did not hold.

Herper et al. used measures of skin conductance response, heart rate change, and startle response to the viewing of emotionally laden slides, and found that BPD subjects did not show a general hyperresponsivity to environmental events, but rather lower electrodermal responses than a cluster B personality comparison group. The literature provides a somewhat variable picture depending on the measures that are used.

Emotional dysregulation is also the centerpiece of a more recent view of disordered personality, specifically, Linehan and colleagues a treatment-derived view of the etiology of BPD. Recognizing that crucial features of BPD were emotional vulnerability and maladaptive emotion regulation strategies, their treatment approach concentrated on these features, making basic vulnerability and poor regulation key concepts. These problems may be heightened in families that

### TABLE 1. COMPOSITION OF SUBJECTS BY PERSONALITY DISORDER DIAGNOSIS

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Percentage of Total Sample (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BPD</td>
<td>61</td>
</tr>
<tr>
<td>Narcissistic</td>
<td>30</td>
</tr>
<tr>
<td>Antisocial</td>
<td>14</td>
</tr>
<tr>
<td>Histrionic</td>
<td>31</td>
</tr>
<tr>
<td>Schizoid</td>
<td>22</td>
</tr>
<tr>
<td>Schizotypal</td>
<td>49</td>
</tr>
<tr>
<td>Paranoid</td>
<td>52</td>
</tr>
<tr>
<td>Dependent</td>
<td>18</td>
</tr>
<tr>
<td>Avoidant</td>
<td>53</td>
</tr>
<tr>
<td>Passive-aggressive</td>
<td>33</td>
</tr>
<tr>
<td>Obsessive-compulsive</td>
<td>40</td>
</tr>
</tbody>
</table>

*Multiple diagnoses make the total >100%.

BPD=borderline personality disorder.


### TABLE 2. MEAN AND STANDARD DEVIATIONS FOR SELF-REPORT TOTAL AND SUBSCALE MEASURES

<table>
<thead>
<tr>
<th></th>
<th>ALS Mean</th>
<th>ALS SD</th>
<th>AIM Mean</th>
<th>AIM SD</th>
<th>CTQ EA Mean</th>
<th>CTQ EA SD</th>
<th>CTQ Total Mean</th>
<th>CTQ Total SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sample</td>
<td>1.29</td>
<td>0.62</td>
<td>3.52</td>
<td>0.58</td>
<td>14.4</td>
<td>5.4</td>
<td>55.6</td>
<td>16.7</td>
</tr>
<tr>
<td>OPD</td>
<td>1.12</td>
<td>0.58</td>
<td>3.43</td>
<td>0.62</td>
<td>13.5</td>
<td>5.4</td>
<td>53.3</td>
<td>16.5</td>
</tr>
<tr>
<td>BPD</td>
<td>1.61</td>
<td>0.56</td>
<td>3.70</td>
<td>0.46</td>
<td>15.9</td>
<td>5.01</td>
<td>59.5</td>
<td>16.4</td>
</tr>
<tr>
<td>Females</td>
<td>1.34</td>
<td>0.70</td>
<td>3.73</td>
<td>0.58</td>
<td>16.4</td>
<td>4.8</td>
<td>62.4</td>
<td>17.9</td>
</tr>
<tr>
<td>Males</td>
<td>1.26</td>
<td>0.56</td>
<td>3.41</td>
<td>0.55</td>
<td>13.2</td>
<td>5.3</td>
<td>51.9</td>
<td>14.8</td>
</tr>
<tr>
<td>Female BPD</td>
<td>1.68</td>
<td>0.55</td>
<td>3.79</td>
<td>0.45</td>
<td>16.8</td>
<td>4.36</td>
<td>61.7</td>
<td>16.7</td>
</tr>
<tr>
<td>Male BPD</td>
<td>1.55</td>
<td>0.57</td>
<td>3.62</td>
<td>0.46</td>
<td>15.1</td>
<td>5.43</td>
<td>57.8</td>
<td>16.2</td>
</tr>
</tbody>
</table>

ALS=Affective Lability Scale; AIM=Affective Intensity Measure; CTQ=Childhood Trauma Questionnaire; EA=emotional abuse subscale; OPD=other personality disorder; BPD=borderline personality disorder.

### TABLE 3A. CORRELATION AMONG KEY VARIABLES—TOTAL SAMPLE (N=174, n=168 FOR CTQ EA)

<table>
<thead>
<tr>
<th></th>
<th>ALS</th>
<th>AIM</th>
<th>CTQ EA</th>
<th>Diagnosis</th>
<th>Gender</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALS</td>
<td>—</td>
<td>—</td>
<td>.449†</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>AIM</td>
<td>.209†</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CTQ EA</td>
<td>.386†</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>.054 NS</td>
<td>.274†</td>
<td>.207†</td>
<td>.138*</td>
<td>—</td>
</tr>
<tr>
<td>Gender</td>
<td>.292†</td>
<td>.223†</td>
<td>.282†</td>
<td>—</td>
<td>.148*</td>
</tr>
</tbody>
</table>

CTQ=Childhood Trauma Questionnaire; EA=emotional abuse subscale; ALS=Affective Lability Scale; AIM=Affective Intensity; NS=non-significant.


### TABLE 3B. CORRELATION AMONG KEY VARIABLES—MALES ONLY (n=111)

<table>
<thead>
<tr>
<th></th>
<th>ALS</th>
<th>AIM</th>
<th>CTQ EA</th>
<th>Diagnosis</th>
<th>Gender</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALS</td>
<td>—</td>
<td>—</td>
<td>.517†</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>AIM</td>
<td>.216*</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CTQ EA</td>
<td>.337†</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>—</td>
<td>—</td>
<td>.236†</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Gender</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

ALS=Affective Lability Scale; AIM=Affective Intensity Measure; CTQ=Childhood Trauma Questionnaire; EA=emotional abuse subscale.


### TABLE 3C. CORRELATION AMONG KEY VARIABLES—FEMALES ONLY (n=63)

<table>
<thead>
<tr>
<th></th>
<th>ALS</th>
<th>AIM</th>
<th>CTQ EA</th>
<th>Diagnosis</th>
<th>Gender</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALS</td>
<td>—</td>
<td>—</td>
<td>.363†</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>AIM</td>
<td>—</td>
<td>—</td>
<td>.189 NS</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CTQ EA</td>
<td>.446†</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>—</td>
<td>—</td>
<td>.209 NS</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Gender</td>
<td>—</td>
<td>—</td>
<td>.259†</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

ALS=Affective Lability Scale; AIM=Affective Intensity Measure; CTQ=Childhood Trauma Questionnaire; EA=emotional abuse subscale; NS=non-significant.


### TABLE 3D. CORRELATION AMONG KEY VARIABLES—BPD ONLY (n=61)

<table>
<thead>
<tr>
<th></th>
<th>ALS</th>
<th>AIM</th>
<th>CTQ EA</th>
<th>Diagnosis</th>
<th>Gender</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALS</td>
<td>—</td>
<td>—</td>
<td>.102 NS</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>AIM</td>
<td>.164 NS</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CTQ EA</td>
<td>.102 NS</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>—</td>
<td>—</td>
<td>.129 NS</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Gender</td>
<td>—</td>
<td>—</td>
<td>.200 NS</td>
<td>.162 NS</td>
<td>—</td>
</tr>
</tbody>
</table>

BPD=borderline personality disorder; ALS=Affective Lability Scale; AIM=Affective Intensity Measure; NS=non-significant; CTQ=Childhood Trauma Questionnaire; EA=emotional abuse subscale.


### TABLE 3E. CORRELATION AMONG KEY VARIABLES—OPD SUBJECTS ONLY (n=113)

<table>
<thead>
<tr>
<th></th>
<th>ALS</th>
<th>AIM</th>
<th>CTQ EA</th>
<th>Diagnosis</th>
<th>Gender</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALS</td>
<td>—</td>
<td>—</td>
<td>.301*</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>AIM</td>
<td>.164†</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>CTQ EA</td>
<td>.074 NS</td>
<td>.280*</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Gender</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

OPD=other personality disorder; ALS=Affective Lability Scale; AIM=Affective Intensity Measure; CTQ=Childhood Trauma Questionnaire; EA=emotional abuse subscale; NS=non-significant.

provide "invalidating" environments for the affectively unstable child who later develops BPD. In such environments, the vulnerable individual is not supported in efforts to label emotions, modulate arousal, or tolerate distress. Abuse—physical, sexual, or emotional—in childhood is an extreme form of an invalidating environment.

The vulnerability of the individual with BPD is not yet well understood. Affective instability has been proposed to be a core psychobiologic predisposition and likely involves alterations in the circuitry of emotional regulation that includes regions of the prefrontal cortex, amygdala, hypothalamus, anterior cingulate cortex, hippocampus, insular cortex, and ventral striatum. There exist individual differences in the asymmetry of activation in anterior structures of this pathway, and in particular, the prefrontal cortex, that have been linked to aspects of emotional responsivity, including the return to baseline after an emotional challenge. Prefrontal cortex fibers help dampen negative affect through an inhibitory influence in the amygdala. Whether these observed effects are from genetic parameter settings, environmental influences on the parameter settings, or a combination of these factors is currently unknown and will be difficult to delineate. It may well be that genetic factors are implicated in the impaired capacity to regulate emotional experience, or it may be that experiential and/or environmental factors, such as trauma, impact basic biologic mechanisms implicated in emotional experience, or both. The latter could occur early in a child's life when neural pathways are being established, or it could occur later when developmental changes that typically require the reorganization of neural pathways occur. In either case, such longstanding alterations may be the mechanism by which affective instability continues to characterize and distress those with personality disorder, and especially those with BPD. Further investigation of the role of environmental influences, especially traumatic influences, is a necessary path of inquiry.

One model for the longstanding and detrimental effects of early environmental stress on developing biological processes is provided by the work of Francis and Meaney. Studying rats exposed to early environmental stressors of changed frequency of maternal licking and grooming, andarched back nursing, significant neurophysiologic, neurohormonal, and neuroendocrine changes down to the level of gene expression occurred in the offspring as a direct consequence of environmental influences. These changes affected stress responsivity, as well as components of the circuitry of emotional processing; the amygdala, hippocampus, and prefrontal cortex.

Empirical research from the last 15 years has noted high rates of childhood physical and sexual abuse in patients with BPD compared with other comparison groups. For BPD, the presence of some type of childhood abusive experience was almost ubiquitous. Zanarini and colleagues found a 91% abuse rate and a 92% rate of emotional or physical neglect in their cohort of BPD subjects. Estimates of the overall rate of childhood sexual abuse in those with BPD range from 40% to 70% compared with an overall rate of childhood sexual abuse for other Axis II patients to be between 19% and 26%. Severity of sexual abuse and neglect was significantly related to affective symptoms of BPD. This prompted some to consider the possibility that BPD was a form of posttraumatic stress disorder (PTSD). A meta-analytic study of 21 studies comprising 2,414 subjects that examined the relationship between BPD and childhood sexual abuse yielded only a moderate pooled effect size of $r=0.28$. Diagnostic criteria and gender did not have a predictive role on effect size. A negative relationship between sample size and correlation coefficients ($r=-0.75$) was present, suggestive that smaller, less representative studies were associated with larger effect sizes, and consequently weakening the validity of the finding.

There still exists the possibility that childhood sexual abuse is related to only some features of the BPD condition, and since the diagnosis is polythetic, the relationship may not be consistently manifested at the BPD diagnosis level. For example, in BPD, there are several studies that support a relationship between dissociative phenomena and childhood trauma. Despite these conceptual suggestions, minimal empirical data exist that examines the relationship of childhood trauma and the core BPD dimension of affective instability. The study of affective instability has also been hampered by the complexity of the construct and the difficulty of developing measures that validly assess it. The absence of a wide variety of scales to measure the construct has led to reluctance to study the phenomenon empirically. There exist two validated self-report scales that address two of the several aspects of affective instability: affective lability, measured by the Affective Lability Scale (ALS) and affect intensity, assessed by the Affective Intensity Measure (AIM). We sought to examine the relationship between childhood trauma and affective instability, as indexed by these two scales, in patients with personality disorder diagnoses.

**METHODS**

**Participants**

The sample consisted of 174 patients with personality disorder (62 females, 112 males) between 18 and 60 years of age (mean±SD=38.4±9.9), meeting criteria for an average of 2.3±1.5 personality disorder diagnoses, 61 of whom met criteria for BPD. Participants were recruited from the Bronx Veterans Affairs Medical Center’s and the Mount Sinai Medical Center’s Psychiatric Outpatient Clinics in New York City or from referrals from outside mental health professionals or from paid advertisement for a comprehensive study of which we report data from only a part. All subjects received a medical evaluation, which included history, physical, and neurologic examination, and laboratory testing.

Patients with evidence of serious systemic illness that might affect central nervous system functioning, such as diabetes,
hypertension, autoimmune illness, and renal, liver, or cardiac disorders, were excluded. Patients with neurologic impairment or a history of severe head trauma with loss of consciousness were excluded as well. Potential participants were excluded if they met the Diagnostic and Statistical Manual of Mental Disorders, Third Edition–Revised (DSM-III-R) criteria for schizophrenia or any schizophrenia-related psychotic disorder or for bipolar I affective disorder. Meeting criteria for past major depressive disorder did not exclude potential participants because this is a common comorbidity in cluster B personality disorders. Patients were free from substance abuse for at least 6 months and did not currently take medication for medical or psychiatric reasons. All participants gave written informed consent after receiving a complete description of the study.

**Psychiatric Assessments**

Patients completed extensive psychiatric and diagnostic assessment by trained raters, utilizing the Structured Clinical Interview for Axis I disorders and the Structured Interview for DSM III-R Personality Disorder for Axis II disorders (κ=.81 for BPD). A consensus was obtained in consultation with a clinical psychologist not otherwise involved in the study.

**Measures**

Childhood abuse experience was measured using the Childhood Trauma Questionnaire (CTQ), short form, a 28-item self-report questionnaire that assess a broad range of abusive experiences, including childhood sexual, physical and emotional abuse, and physical and emotional neglect. The response format of the items is a five-point Likert scale with options ranging from "never true" to "very often true." The 70-item measure has been shown to have good reliability (Cronbach's α=.79-.94), and good test-retest reliability (0.80-.83). It yields five subscales: Sexual Abuse (SA), Emotional Abuse (EA), Physical Abuse (PA), Emotional Neglect (EN), and Physical Neglect (PN), as well as a total score. In our sample, coefficient α for the total score was 0.68.

Each participant completed the ALS. ALS items are rated on a four-point scale (0–3) ranging from "very undescrptive" to "very descriptive." The ALS can be scored for six subscales which demonstrate high internal consistency, but we used only the total score. The ALS has been shown to have good internal consistency, suitable test-retest reliability and not to vary substantially by gender. Previously reported data from a subset of those reported here lend support to the construct validity of the measure. Coefficient α in the present sample was 0.97.

Participants also completed the AIM. The AIM is a 40-item self-report measure, with good internal consistency and test-retest reliability and yields a single Total score. It has been validated by a study comparing subjects' AIM scores with ratings by informants as well as a study of the intensity of subjects' emotional reactions to actual life events and self-assessed characteristic reactions to a set of standardized descriptions of life events. In the present sample, coefficient α was 0.91.

**Missing Data**

Seven subjects had incomplete responses on the CTQ. Our repair strategy was as follows: If at least 80% of the questions were answered for a particular subscale, the mean value of the remaining items was imputed for the missing value, rounded to the closest integer, and scale score computed since the scales are based on sums. Six subjects (two female BPD, two male BPD, and two females with other personality disorder [OPD]), were eliminated from the analyses on the CTQ subscales of EA, PN, and PA since their responses fell below our threshold for imputation.

**Data Analysis**

Mean differences in continuous variables, such as CTQ, AIM, and ALS scores, were analyzed using student's t-test. Pearson correlations of ALS and AIM were performed, with each other and by diagnosis, gender, and CTQ subscales. Lastly, hierarchical regression analyses using ALS, AIM, and CTQ were conducted to test the interaction of gender and diagnosis in the relationships between the affect variables and the trauma variables.

**FINDINGS**

The personality disorder diagnoses of the 174 participants were distributed as shown in Table 1. Since our focus is on affective instability in BPD, we compared those with BPD (35%; n=61) to the remaining 112, whom we refer to as OPD.

The results for the CTQ were somewhat unexpected. The distribution of SA was markedly skewed (55% had the equivalent of a zero) and, therefore, unusable to correlate with the measures of affect. It also suggested that in this sample, at least, sexual abuse was not a pervasive factor for affective instability. The remaining four subscales had appropriate distributions, and each was correlated with ALS and AIM.

We adopted a decision rule, which in addition to being statistically significant, dictated that the subscale must account for at least 4% of the variance—that is r needed to be >0.20. Only EA and Total score met this criterion and was carried forward for further analysis. The correlation between EA and ALS was .20 (P=.009); for EA and AIM it was .31 (P<.000).

The correlations between AIM and the other CTQ subscales were: r=.19 (P=.012) for PA, r=.12 (NS) for PN; r=.07 (NS) for EN and r=.23 (P=.002) for total score. The correlation between BPD and the other CTQ subscales were: r=.01 (NS) for PA, r=.14 (NS) for PN; r=.05 (NS) for EN and r=.15 (NS) for total score. SA had the lowest correlation (r=.43) and EA the highest correlation (r=.71) with CTQ Total item score.

In Table 2, the means and standard deviations are presented for the total sample and separately for diagnosis, gender, and gender by diagnosis for the affect variables and CTQ EA and Total. The results of the t-tests comparing means
showed that BPD patients had a significantly higher mean ALS than the OPD group (t[172] = -5.3, P<.000, ES = .38) as well as AIM (approximation to t[156] = -3.3, P<.001, ES = .25).

When differences in the affect measures were examined within gender and diagnosis, a slightly different picture emerged. In males, the difference in mean AIM total scores was significantly larger for BPD than OPD subjects, with an ES of .33 (t[109] = -3.7, P<.000). In females, however, mean AIM total score for BPD and OPD subjects was not significantly different ES=-.07 (t[61]=0.65, NS). There were significant differences in ALS mean score for BPD and OPD subjects in both male (t[109]=-3.7, P<.000, ES=.35) and female (t[61]=-2.7, P<.000, ES=.47) groups.

The further investigation of the differential pattern of results yielded by gender and diagnosis considered separately and in combination, are presented in Tables 3A–3E which present the Pearson correlations among the affect, trauma, and categorical variables. Patterns of relationship differ across groups with BPD and females demonstrating the most variability. Moreover, the relationship of ALS and AIM to CTQ EA is different as a function of both gender and diagnosis.

ALS in the total sample correlated significantly with CTQ EA (r=.20, P<.01) and diagnosis (r=.39, P<.01) but not gender (r=.05, NS). In the BPD and female subgroups, the association of CTQ EA and ALS was no longer significant (r=.10, NS and r=.19, NS, respectively). AIM correlated significantly with CTQ EA in the total (r=.31, P<.01), OPD (r=.30, P<.01) and male (r=.25, P<.01) subgroups but not for the female (r=.21, NS) or BPD (r=.15, NS) subgroups.

The correlation of AIM and ALS with each other ranged from 0.16–0.52 depending upon subgroup studied. BPD subjects demonstrated the lowest correlation (r=.16, NS) compared to a significant correlation in OPD subjects (r=.52, P<.01). We performed a Z test of the difference between two independent correlations and found that the two coefficients are significantly different (Z=2.43, P<.02).

The results of the series of hierarchical regression analyses using the dependent variables of ALS and AIM and variables of gender, diagnosis, CTQ EA score, and their interactions did not show evidence of a statistically significant interaction of gender, diagnosis, or their combination in the total sample. Similar analyses conducted solely within males, females, BPD patients, and OPD patients, similarly failed to reveal statistically significant interactions.

**DISCUSSION**

The magnitude of affective intensity as measured by the total AIM score in our BPD sample is somewhat lower than the level of 3.9 of another report examining hospitalized BPD patients.1 The complete sample total AIM score is consistent with a value of 3.5 found in an overlapping sample of outpatient subjects with personality disorder2 and suggests that moderate levels of affective symptomatology are present in these individuals. The participants in this study were part of a larger study examining biological parameters of personality disorder which required subjects to be free of potential confounds of medication, current substance abuse, or medical illness. For this reason, the sample may be somewhat unrepresentative of those with current active strong affective symptomatology, for example for those whose current BPD symptomatology would require hospitalization.

Our BPD sample experienced lower levels of sexual abuse than other BPD cohorts studied in the literature. Forty-five percent of the total sample and 51% of BPD subjects (female BPD 61% and male BPD 42%) endorsed any SA item. This contrasts with 71% of outpatient female BPD subjects3 and 48% of outpatient male BPD subjects4 endorsing histories of childhood SA. For BPD inpatients of mixed gender, rates as high as 62% to 75%5,6 have been reported. Our inclusion criteria may also have had an impact on this factor as well, leading to a selection bias against those with a history of sexual abuse, since they may be unable to do without medication and/or remain substance-free for 6 months.

This study yielded several important findings. First, in the total sample, ALS and AIM total scores did not appear to be significantly associated with childhood abuse or neglect except for emotional abuse.

In the OPD group, childhood EA is a significant predictor for AIM. In the BPD group, EA and gender are NS predictors of both ALS and AIM. When the BPD sample was examined by gender, we found no significant correlations for female or male BPD subjects with ALS, AIM, and CTQ EA.

These findings suggest that non-childhood variables, such as an inherited affective instability trait may be more important in the etiology of affective dyscontrol in BPD subjects. Whether this is influenced by the relative absence of childhood sexual trauma in this sample of subjects awaits further study. It is possible that childhood trauma is an unfortunate consequence of inherited affect instability traits in an infant with a caregiver whose own impulse control is already compromised and this combination evokes abuse, as it is possible that abuse negatively influences emotional regulation because of the affect trauma and stress have on the neurobiology of affect. Further study is definitely required.

The complicated differences between groups suggest that the relationship of ALS and AIM vary as a function of both gender and diagnosis and highlight the need to examine affective instability and trauma variables in future analyses by subgroup, even though in the present study none of the tests of the interaction terms were significant. Studies with additional BPD subjects and adequate power to analyze female and male OPD and BPD groups, could further delineate the importance of the subgroup differences we found here.

Our empirical findings using self-report measures of childhood abuse offer limited support to the theoretical notion that much of the psychopathology in BPD can be linked to emotional abuse rather than frank physical or sexual abuse.2,7 The special nature of the selection of the sample...
may have predisposed toward this finding and samples of patients with BPD and OPD with less current control over symptoms than the current sample may not confirm this implication. Studies examining the relationship of childhood trauma with other BPD characteristics, such as dissociation, also found correlations with BPD diagnosis\(^5\) and not with indices of childhood trauma.\(^3\)\(^6\)

The relationship of asymmetrical activation patterns in the prefrontal cortex and affect duration\(^4\)\(^6\) and the prefrontal cortex's inhibitory role of the amygdala\(^8\) highlight the importance of this structure in emotional regulation. Recent findings of diminished serotonin responsivity in the prefrontal cortex of borderline subjects\(^8\)\(^9\) and recent report of increased activation of the amygdala in BPD\(^3\) potentially link disturbed affect regulation with defining aspects of BPD phenomenology, impulsive aggression and behavioral dyscontrol.

Heritability for personality disorder in general is estimated at 0.60 and for BPD 0.69.\(^6\)\(^8\) Statistically significant heritability estimates from twin studies\(^7\)\(^8\) exist for aggression, impulsivity,\(^7\)\(^8\) and irritability,\(^7\)\(^8\) all traits that are inclusion criteria for the diagnosis of BPD. Whereas irritability is theorized to be the affective state that predisposes an individual to aggressive acts,\(^9\) it may overlap conceptually with aspects of emotional instability. Heritability for affective liability has been estimated at 0.45\(^8\) and for a higher order factor labeled emotional dysregulation, multivariate genetic analyses examining correlations between multiple traits within this construct, estimates heritability at 47%.\(^8\) Such data highlight the need of continuing investigation into the genetic underpinnings of affective instability.

There are several limitations to this study. The ALS and AIM have not been well validated in personality disorder populations, and are relatively new measures attempting to assess a complex phenomenon. Other limitations include the validity of self-report measures in assessing past abusive experiences, particularly in a population of symptomatic individuals potentially prone to recall bias. There are data to support the verification of reported childhood events\(^10\)\(^11\) and stability of CTQ retrospective reporting in the context of differing levels of psychopathology.\(^12\) Under-reporting\(^12\) and over-reporting\(^12\) are still concerns. Structured childhood trauma interviews might yield more detailed responses. In a meta-analysis of 24 studies\(^13\) exploring the relationship between traumatic experiences and a dissociation scale, however, there was no difference in effect size between studies that assessed trauma through interview or questionnaire. The possibility exists that with affective instability, structured childhood trauma interviews might yield more detailed responses.

**CONCLUSION**

These data indicate measures of affective instability are related to indices of self-reported childhood trauma only for emotional abuse, and this relationship varies as a function of gender and personality diagnosis. Within BPD, nontrauma factors may be more predominant in the etiology of affective dyscontrol. CWS

### APPENDIX

**Childhood Trauma Questionnaire Emotional Abuse Subscale Individual Items**

15. When I was growing up, people in my family called me things like “stupid,” or “dumb,” or “ugly.”
13. When I was growing up, people in my family called me things like “fat,” “dumb,” or “ugly.”

73A. When I was growing up, I thought my parents wished I had never been born.
28A. When I was growing up, people in my family said hurtful or insulting things to me.

47A. When I was growing up, I felt that someone in my family hated me.
57A. When I was growing up, I felt that someone in my family hated me.

### REFERENCES


**ABILIFY™ (aripiprazole) Tablets**

**Brief Summary of Prescribing Information**

**Indications and Usage**

ABILIFY is indicated for the treatment of schizophrenia (including acutely agitated patients) and the treatment of schizoaffective disorder (including acutely agitated patients). ABILIFY is also indicated for the symptomatic treatment of the manic episode associated with bipolar I disorder. ABILIFY is also indicated for the treatment of major depressive disorder.

**Usage and Administration**

ABILIFY may be administered as a single daily dose, irrespective of the time of day. ABILIFY is not recommended for long-term use in patients with dementia-related psychosis.

**Dosage**

The recommended initial dosage of ABILIFY is 5 mg/day for adult patients and 3 mg/day for elderly patients. The dosage may be increased at weekly intervals up to a maximum daily dosage of 20 mg/day for adult patients and 10 mg/day for elderly patients, if necessary, to achieve the desired response.

**Contraindications**

ABILIFY is contraindicated in patients with a known hypersensitivity to aripiprazole or any of its excipients. ABILIFY is also contraindicated in patients with a history of seizures. ABILIFY should not be used in patients with a known history of seizure disorder.

**Warnings**

**Seizures**

Seizures have been reported in patients treated with antipsychotic drugs, including ABILIFY. Patients predisposed to seizures (e.g., patients with a history of seizures) are at increased risk for developing seizures while receiving antipsychotic therapy.

**Orthostatic Hypotension**

Orthostatic hypotension has been reported in patients treated with ABILIFY. ABILIFY should be used with caution in patients with hypovolemia, dehydration, or conditions that predispose to hypotension.

**Tardive Dyskinesia**

Tardive dyskinesia, a potentially irreversible syndrome characterized by orofacial dyskinesia, may occur in patients treated with antipsychotic drugs, including ABILIFY. The risk of tardive dyskinesia is increased in patients aged 65 years and over; a higher incidence of tardive dyskinesia has been observed in patients treated with antipsychotic drugs for longer periods of time.

**Neuroleptic Malignant Syndrome**

Neuroleptic malignant syndrome (NMS) has been reported in association with the use of antipsychotic drugs, including ABILIFY. NMS manifests with symptoms such as hyperpyrexia, muscle rigidity, altered mental status, and evidence of autonomic instability. The symptoms of NMS may evolve over several days and may not be reversible. NMS has been reported in association with the use of other antipsychotic drugs, including ABILIFY. Patients treated with antipsychotic drugs, including ABILIFY, should be observed for signs and symptoms of NMS.

**Hypotension**

Hypotension has been reported in patients treated with ABILIFY. Patients with a history of hypotension or dehydration should be monitored for signs of hypotension.

**Hypertension**

Hypertension has been reported in patients treated with ABILIFY. ABILIFY should be used with caution in patients with a history of hypertension or in patients with other cardiovascular risk factors.

**Hepatic Impairment**

Hepatic impairment has been reported in patients treated with ABILIFY. ABILIFY should be used with caution in patients with hepatic impairment.

**Other Adverse Events**

Other adverse events that have been reported in association with the use of ABILIFY include persistent somnolence; gingival hyperplasia; transient, reversible, involuntary, dyskinetic movements; hyperglycemia; hypothyroidism; hyperprolactinemia; dyslipidemia; angioedema; and increased appetite.

**Interactions**

ABILIFY is a substrate of CYP3A4 and CYP2D6 and is also a弱rennergic receptor antagonist. ABILIFY should be used with caution in patients taking drugs that may affect hepatic metabolism or cardiac function.

**Special Populations**

Patients with concomitant illness, elderly patients, and patients with dementia-related psychosis should be monitored closely for adverse events.

**Nursing Mothers**

ABILIFY is excreted in human milk. The decision to use ABILIFY in a nursing woman should be made after weighing the potential benefits and risks of treatment for the mother.

**Pediatric Use**

The safety and effectiveness of ABILIFY in children and adolescents have not been established.

**Geriatric Use**

Geriatric patients should be monitored closely for adverse events.

**Overdosage**

Symptoms of overdose may include somnolence, increased blood pressure, hypotension, tachycardia, agitation, tremor, dyskinesia, and vomiting. Treatment should be symptomatic and supportive.

**Clinical Pharmacology**

ABILIFY is a partial agonist at D₂ and 5-HT1A receptors. It has a low affinity for D₁ and 5-HT₂ receptors. ABILIFY has no significant affinity for M₁, H₁, α₁, β₁, or α₂ receptors.

**Pharmacokinetics**

ABILIFY is rapidly absorbed following oral administration. The peak plasma concentration is achieved within 1-4 hours. The elimination half-life is approximately 9-10 hours. ABILIFY is extensively metabolized by the liver and is eliminated primarily as metabolites in the urine. ABILIFY does not undergo significant enterohepatic recycling.

**Preclinical Pharmacology**

In preclinical studies, ABILIFY exhibited antipsychotic activity in animal models of schizophrenia.

**Clinical Studies**

ABILIFY has been studied in a variety of clinical trials, including acute and maintenance treatment of schizophrenia, schizoaffective disorder, and bipolar disorder. ABILIFY has also been studied in elderly patients with dementia-related psychosis and in patients with major depression.

**Post-Marketing Surveillance**

Since the marketing of ABILIFY, cases of adverse events have been reported, including arrhythmias, myocardial infarction, cerebrovascular disease, gastrointestinal perforation, and intracerebral hemorrhage. Patients treated with antipsychotics, including ABILIFY, should be monitored closely for signs and symptoms of adverse events, including cardiac, gastrointestinal, and cerebrovascular events.

**References**


Abilify is indicated for the treatment of schizophrenia.

As with all antipsychotic medications, a rare condition referred to as neuroleptic malignant syndrome (NMS) has been reported. As with all antipsychotic medications, prescribing should be consistent with the need to minimize the risk of tardive dyskinesia (TD). Abilify may be associated with orthostatic hypotension and should be used with caution in patients with known cardiovascular disease, cerebrovascular disease, or conditions which would predispose them to hypotension. Seizures occurred in 0.1% of Abilify-treated patients in short-term, placebo-controlled trials. As with other antipsychotic drugs, Abilify should be used with caution in patients with a history of seizures or with conditions that lower the seizure threshold.

Treatment-emergent adverse events reported at an incidence of ≥10% and greater than placebo include headache, anxiety, insomnia, nausea, vomiting, lightheadedness, somnolence, akathisia, and constipation.

Please see Brief Summary of Prescribing Information on adjacent page. For more information, visit our web site at www.abilify.com.