Frontal and Posterior Asymmetry in Female Youth With Borderline Personality Disorder

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Abstract

Objective: This study aimed to identify pathophysiological markers of borderline personality disorder (BPD) using electroencephalogram (EEG). More specifically, it examined whether female youth diagnosed with BPD exhibited more frontal or posterior alpha asymmetry relative to healthy youth. Presently, there is limited EEG data on alpha asymmetry in youth with BPD. This study hypothesized that female youth diagnosed with BPD would exhibit greater frontal asymmetry compared to healthy youth. In keeping with this initial hypothesis, it was expected that greater frontal alpha asymmetry in youth with BPD would be associated with greater BPD severity and rumination. Finally, this study also explored posterior asymmetry in the BPD and healthy samples.

Method: Female youth diagnosed with BPD (n = 33) were recruited from McLean Hospital’s 3East residential program and healthy controls (n=35) were recruited from the greater Boston community. Both samples were aged 13 to 23 years. Data regarding psychiatric conditions were obtained through clinical interviews and self-report measures, and resting eyes closed EEG data were acquired using a 128-channel high density net. Analyses focused on total alpha (8-13 Hz), alpha 1 (8.5-10 Hz), and alpha 2 (10.5-12 Hz). Correlations among all study variables were analyzed, and t-tests comparing differences among alpha asymmetry between BPD and healthy youth were conducted.

Results: In contrast to the hypothesis, there were no significant frontal alpha asymmetry differences among healthy and BPD youth. Exploratory analyses, however, found that posterior asymmetry in the alpha 2 band was greater in the BPD relative to the healthy adolescents \(t(65) = 2.04, p = .046\).
Conclusions: These findings suggest that alpha posterior asymmetry may be a potential pathophysiological marker of BPD.
Acknowledgements

This thesis was possible in large part because of Randy Auerbach’s outstanding mentorship. As Director of the Child and Adolescent Mood Disorders Laboratory at McLean Hospital-Harvard Medical School, Randy welcomed me into his laboratory as a student visitor in May 2014. Under his kind guidance, I was introduced to several members affiliated with his laboratory and from whom I learned different research methods. First, Randy introduced me to Alexis Whitton, who devotedly taught me how to administer the Structured Clinical Interview for DSM-IV and greatly expanded my growing understanding of the discipline of clinical psychology. Randy then graciously agreed to be my advisor for this master’s thesis and taught me the fundamentals of EEG as a method for data acquisition. His support never wavered despite the challenges I faced in learning this new technique. He remained patient and gracious despite my initial errors. He was always attentive to my needs as a student and I felt tremendously fortunate in knowing that I could always turn to him for any assistance. I grew to rely on quite a few of his team members, especially, Erin Bondy, Erika Esposito, Angela Pisoni and Naomi Tarlow, at different times during my training. Each of them kindly entertained my many questions and was always approachable and willing to help.

At the Harvard Extension School, I benefitted appreciably from the thoughtful feedback I received from Dante Spetter on my thesis proposal. Her detailed comments pushed me to think deeper and improve upon my work. While I learned much from many faculty members during the coursework stage, two professors in particular, William Milberg and Shelley Carson, greatly enhanced my knowledge and understanding of
psychology. Milberg’s course on the neuroanatomy of psychological function remains the most stimulating and engrossing course I have ever taken. His passion and expertise as a professor in the classroom was rivaled only by his generosity and constant encouragement to question and think critically. Similarly, Carson’s lectures proved to be some of the most engaging ones I had the privilege of taking and significantly enhanced my interest in the field of psychology.

Finally, I would like to acknowledge the role my spouse, Sachin Agarwal, has played in supporting me tirelessly throughout my academic endeavors. Without him, none of this would have been possible.

To all of you, thank you.
# Table of Contents

Acknowledgements .............................................................................................................v

List of Tables ........................................................................................................................ix

List of Figures ........................................................................................................................x

I. Introduction ........................................................................................................................1

  Borderline Personality Disorder .........................................................................................3

  Assessment ...........................................................................................................................9

  Electroencephalogram .......................................................................................................9

  Frontal and Posterior Asymmetry .......................................................................................12

  Study Aims & Hypotheses ..................................................................................................14

  Significance .......................................................................................................................15

II. Method ................................................................................................................................17

  Participants ........................................................................................................................17

  Procedure ...........................................................................................................................17

  Instruments ........................................................................................................................20

  Electroencephalogram ........................................................................................................21

  Data Analytic Overview ....................................................................................................22

III. Results ................................................................................................................................23

  Preliminary Analyses .........................................................................................................23

  Frontal Asymmetry ............................................................................................................23

  Posterior Asymmetry ..........................................................................................................24

IV. Discussion ........................................................................................................................29
Limitations ....................................................................................................................................31
Implications and Future Directions ..........................................................................................31
References ...................................................................................................................................34
List of Tables

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Demographic characteristics for the study sample</th>
<th>18</th>
</tr>
</thead>
<tbody>
<tr>
<td>Table 2</td>
<td>Comorbidity for youth with borderline personality disorder</td>
<td>19</td>
</tr>
<tr>
<td>Table 3</td>
<td>Correlations among alpha asymmetry, BPD symptoms, and rumination in healthy adolescents</td>
<td>25</td>
</tr>
<tr>
<td>Table 4</td>
<td>Correlations among alpha asymmetries, BPD symptoms and rumination in youth with BPD</td>
<td>26</td>
</tr>
</tbody>
</table>
List of Figures

Figure 1  Layout illustrating the approximate 10-10 equivalent on the 128-channel Hydrocel GSN……………………………………………………………10

Figure 2  Mean Frontal Asymmetry……………………………………………27

Figure 3  Mean Posterior Asymmetry…………………………………………28
Borderline personality disorder (BPD) is a mental disorder characterized by emotional lability, unstable relationships, impulsivity, and self-destructive behavior. The prevalence rate within the United States is 1% (Lenzenweger, Lane, Loranger, & Kessler, 2007) with typical onset in late adolescence (Paris, 2014). Approximately 11% of outpatients (Chanen, Jackson, McGorry, Allot, Clarkson, et al., 2004) and 43-49% of inpatients (Levy, Becker, Grilo, Mattanah, Garnet, et al., 1999) reported a BPD diagnosis (Sharp & Fonagy, 2015), and comorbidity with other mental disorders is common (Zimmerman & Mattia, 1999; Yoshimatsu & Palmer, 2014; Hooley, Cole, & Gironde, 2012).

Research shows a significant correlation between self-injury and BPD (Dulit, Fyer, Leon, Brodsky, & Frances, 1994; Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006). BPD also has been identified as a major risk factor for suicide, with patients reporting more than 3 lifetime attempts on average (Soloff, Lis, Kelly, Cornelius, & Ulrich, 1994). A longitudinal study revealed that a BPD diagnosis prospectively predicted suicide attempts in adults (Yen, Shea, Pagano, Sanislow, Grilo, et al., 2003). Compared to other mental disorders, adolescents with BPD report suicide ideation at earlier ages and with greater frequency (Venta, Ross, Schatte, & Sharp, 2012).

Presently, the pathophysiology of BPD is not well understood, and electroencephalogram (EEG) offers a non-invasive approach to study neural activity in
relation to mental illness. EEG records scalp-recorded brain electrical activity in the form of wave oscillations. It provides excellent temporal resolution, as data reflect ongoing neural activity in the milliseconds (ms) range but poor spatial resolution (Luck, 2014). Temporal resolution means that EEG allows for the recording of neural activity continuously as it occurs. In contrast, other neural data recording methods, such as functional magnetic resonance imaging (fMRI), is slower relative to EEG as it depends on blood flow and may take 3 to 5 seconds to record neural activity (Glover, 2011). On the other hand, fMRI can tell us what parts of the brain are differentially activated in response to stimuli, that is, it has better spatial resolution, whereas EEG measures neural activity across the scalp and so the source of neural activity is poor. However, different wave patterns may emerge in different disorders so EEG may allow us to objectively determine whether a particular disorder, such as, BPD, may be characterized by a wave pattern that is distinct from other mental disorders.

One of the frequency bands recorded by EEG is the alpha band (8-13Hz), a slow wave usually generated at the back of the head and typically associated with drowsiness, fatigue, or closed eyes. When greater alpha is observed in one hemisphere in relation to the other in the frontal region, it is referred to as frontal asymmetry (Davidson, 1993). Frontal asymmetry is associated with neural deficits related to approach or avoidance tendencies. Paradoxically, greater alpha activity is believed to reflect reduced neural activation, such that when generated in the left frontal lobe it indicates reduced positive affect. Research on frontal asymmetry and emotion shows that positive affect and approach related behavior is associated with greater activation (i.e. lower alpha power) of the left frontal lobe (Pizzagalli, Sherwood, Henriques, & Davidson, 2005), whereas
negative affect and avoidance related behavior is associated with greater activation of the right frontal lobe (i.e., higher alpha power) (Davidson, Schwartz, Saron, Bennett, & Goleman, 1979). Therefore, greater alpha power in the left frontal lobe would be indicative of reduced cortical activity, and past research has shown that relatively reduced left frontal asymmetry is present in individuals at-risk for depression, currently depressed individuals, and adults with remitted depression (Auerbach, Stewart, Stanton, Mueller, & Pizzagalli, 2015; Schaffer, Davidson, & Saron, 1983; Davidson, 1993; Gotlib, Ranganath, & Rosenfeld, 1998; Tomarken et al., 2004). Although frontal asymmetry is well explored in the context of depression, less research has tested whether frontal asymmetry is observed in people with BPD.

Along with frontal asymmetry, posterior asymmetry has also emerged as a potential pathophysiological marker of depression, such that subjects with current or remitted depressive disorder have shown right posterior hypoactivation (indicated by greater alpha power) (Bruder et al., 1997; Henriques & Davidson, 1990; Kentgen et al., 2000; Stewart et al., 2011). However, there is lack of research examining posterior asymmetry in BPD. Therefore, both frontal and posterior asymmetries in BPD remain to be investigated. Towards addressing this important research gap, this study tests whether female youth diagnosed with BPD exhibit frontal or posterior alpha asymmetry relative to healthy youth.

Borderline Personality Disorder (BPD)

In 1980, personality disorder (PD) was formally recognized as a distinct category of mental illness (Cloninger, 2007). A personality disorder refers to a pervasive,
inflexible pattern of behavior that endures over a period of time and causes considerable
distress or functional impairment in cognitive, affective, or interpersonal domains
(Butcher, Hooley, Mineka, 2014). In the DSM-5 (American Psychiatric Association,
2013), personality disorders are divided into three clusters: Cluster A includes three
personality disorders that are characterized by odd or unusual behavior; Cluster B
includes 4 personality disorders that involve emotional, dramatic or erratic behavioral
tendencies; and finally, Cluster C includes 3 personality disorders that are marked by
fearfulness or anxiety. Borderline personality disorder is subsumed in Cluster B.

In comparison to other personality disorders, borderline personality disorder is
more prevalent, however, important questions remain about its etiology and treatment
(Bradley, Conklin & Western, 2007). The term “borderline” was first coined by Adolph
Stern (1938) to denote a state between neurosis and psychosis. Many believe that the
term “borderline” is unclear and problematic because it does not operationalize the
specific “borders” (Hooley, Cole & Gironde, 2012). In contrast, the World Health
Organization’s (WHO) *International Classification of Diseases (ICD-10)* refers to BPD
as “emotionally unstable disorder”, which, some contend, more accurately conveys the

Otto Kernberg has emerged as one of the most influential figures in the
construction of borderline personality (Kernberg, 1967). He held that the behavior of
people with borderline personality organization (BPO) was characterized by impulsivity
and emotional instability. Although this differed from psychosis, people with BPO
displayed more cognitive disorganization, especially when under duress. He suggested
that BPO individuals exhibited unstable views of the self and of others and developed
dichotomous perceptions of people as *absolutely good* or *absolutely bad*. Similarly, Gunderson and Singer (1975) identified six features that characterize BPD: intense negative affect, impulsive behavior, some degree of social adaptation (e.g., manipulative), psychotic episodes, odd responses to unstructured tests, and unstable interpersonal relationships. As the construct of BPD evolved over the decades, diagnostic criteria were set and BPD was incorporated as an Axis II personality disorder in DSM-III (American Psychiatric Association, 1980; Bradley, Conklin & Western, 2007).

Presently, the DSM-5 (American Psychiatric Association, 2013) stipulates that BPD is diagnosed if a person has at least 5 of the following 9 symptoms: (i) frantic efforts to avoid real or imagined abandonment; (ii) a pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation; (iii) identity disturbance: markedly and persistently unstable self-image or sense of self; (iv) impulsivity in at least 2 areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating); (v) recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior; (vi) 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); (vii) chronic feelings of emptiness; (viii) inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights); (ix) transient, stress-related paranoid ideation or severe dissociative symptoms (American Psychiatric Association, 2013).

The DSM-5 (American Psychiatric Association, 2013) and the proposed *International Classification of Diseases, 11th Revision*, recently extended the diagnosis of
BPD to adolescent populations (Kaess et al., 2014). Historically, mental health professionals have been reluctant to diagnose BPD in adolescents for fear of stigmatization and because of the challenge in distinguishing BPD symptoms from normal developmental features common to adolescence (Meijer, Goedhart, & Treffers, 1998; Miller, Muehlenkamp, & Jacobson, 2008; Sharp & Tackett, 2014). At the same time, and perhaps unintentionally, the difficulty of disentangling BPD symptoms from commonly observed adolescent characteristics has hampered efforts for early detection, prevention, and timely intervention (Miller, Muehlenkamp, & Jacobson, 2008; Kaess et al., 2014; Sharp & Tackett, 2014; Chanen, 2015).

Part of the problem in diagnosing adolescents with BPD also emerges from establishing a stable BPD diagnosis in a population that is experiencing ongoing developmental changes. For example, within a Dutch sample, a diagnosis of BPD among adolescents was found not to be stable; in fact, only 2 of the 14 adolescents initially diagnosed with BPD using the diagnostic interview for borderline patients (DIB) met BPD criteria again after 3 years (Meijer, Goedhart, & Treffers, 1998). However, studies also have shown that a diagnosis of BPD can remain stable over time (Miller, Muehlenkamp, & Jacobson, 2008) and support for the construct validity of BPD in adolescents is compelling (Bondurant, Greenfield, & Tse, 2004).

Longitudinal studies suggest that BPD emerges in adolescence, peaks in young adulthood, and diminishes across the lifespan (Kaess et al., 2014). Prevalence rates among adolescents and adults are comparable. Cumulative data suggest that for young people, 1.4% will meet diagnostic criteria for BPD by age 16 years, and by age 22, the prevalence rate will increase to 3.2%. Among adults, between 0.7% and 2.7% will meet
diagnostic criteria for BPD (Kaess et al., 2014). In clinical settings, the gender ratio for females to males with BPD is 3:1, but no significant differences are observed in the general population (Kaess et al., 2014).

Both genetic and psychosocial environmental factors have been implicated in the etiology of BPD (Hooley, Cole & Gironde, 2012). Some contend that personality traits, such as, instability and impulsivity, that are hallmarks of BPD, are heritable themselves, which may explain to some degree the heritability of BPD (Butcher, Hooley & Mineka, 2014; Hooley, Cole & Gironde, 2012; Paris, 2007). A recent Dutch-based extended twin study provided strong evidence for genetic rather than cultural transmission of BPD (Distel, Rebollo-Mesa, Willemsen, Derom, Trull, et al., 2009). A sample of twins \( n=5017 \) and their parents, siblings and spouses were recruited for the study and BPD features were measured using a Dutch translation of the Personality Assessment Inventory-Borderline Features scale (PAI-BOR). Following genetic modeling, strong evidence for genetic transmission emerged, while no evidence for shared environmental influences was found. In contrast, retrospective studies have shown that reported adverse childhood experiences, especially neglect from caregivers, constitute a significant risk factor for the development of BPD (Zanarini, Williams, Lewis, Reich, Vera, et al., 1997; Butcher, Hooley & Mineka, 2014).

At present, there is no evidence of effective pharmacological treatment for BPD (Stoffers, Völlm, Rücker, Timmer, Huband, et al., 2010; Kaess et al., 2014). Randomized control trials have provided evidence for the effectiveness of four psychosocial treatments, namely: cognitive analytic therapy (CAT) (Ryle & Kerr, 2002); emotion regulation training (ERT) (Schuppert, Giesen-Bloo, van Gemert, Wiersema, Minderaa,
Emmelkamp, et al., 2009); mentalization-based treatment for adolescents (MBT-A) (Bateman & Fonagy, 2010); and, dialectical behavior therapy for adolescents (DBT-A) (Rathus & Miller, 2002). Based on Marsha Linehan’s (1993) DBT for adults, DBT-A is the most commonly used psychotherapeutic treatment for adolescents with BPD (Kaess et al., 2014).

Linehan (1993) has conceptualized BPD as a disorder of emotion regulation. Consistent with the diathesis-stress model (e.g., Monroe & Simons, 1991), this formulation posits that BPD is a combination of emotional vulnerability and an invalidating environment that fails to promote the development of emotion regulation skills. Dialectical behavior therapy (DBT) emphasizes finding a balance between acceptance and change, and developing validation, problem-solving, and communication strategies. Suicidal tendencies are understood to be maladaptive ways of coping with overwhelming emotional distress. Treatment is structured to address a hierarchy of behaviors: life-threatening behaviors, therapy-interfering behaviors, behaviors that negatively impact the quality of one’s life, and increasing effective use of behavioral skills. DBT for adolescents (DBT-A) (Rathus & Miller, 2002) is an adaptation of Linehan’s proposed treatment for adults with the following modifications: reducing the number of therapy sessions to 12 weeks to ensure that adolescents perceive therapy completion as an achievable goal; including family members in therapy sessions for skills training and family problem solving; limiting the number of skills being taught to set realistic targets; and, simplifying the language used in sessions. A quasi-experimental study showed promising results with noteworthy reductions in psychiatric symptoms (Rathus & Miller, 2002).
Assessment

Currently, the most widely accepted diagnostic measure for assessing BPD is the Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II). Two major advantages of this diagnostic measure are: first, it directly addresses each DSM criteria necessary for diagnosis; and second, it has very good psychometric properties (Huprich, Paggeot & Samuel, 2015; Bradley, Conklin, & Western, 2007). Other diagnostic tools, such as, the Clinical Diagnostic Interview (CDI), that rely on the clinician’s judgment of patients’ detailed narratives, have also been developed. The reliance on clinical judgment is a limitation to any interview-based approach, and thus, developing a psychophysiological approach to assess BPD may provide a more objective, empirically verifiable diagnostic measure of BPD. EEG may provide a promising means of identifying neural markers that underlie BPD. In other words, it may help identify whether specific wave patterns characterize BPD as well as differentiate it from other disorders.

Electroencephalogram (EEG)

EEG reflects scalp-recorded electrical brain activity and was first studied by Hans Berger in 1929 (Luck, 2014). The basic principle of EEG involves measuring brain electrical activity by placing electrodes on the scalp, securing each electrode connection using a gel or liquid, and recording the changing voltage over a period by amplifying the signal received. The naming convention for the electrodes is based on the placement of each electrode denoted by an alphanumeric code, as shown in the map. The letters signify
Figure 1. Layout illustrating the approximate 10 – 10 equivalent on the 128-channel HydroCel GSN.
different lobes of the brain: F=frontal, P=parietal, T=temporal, O=occipital. There are
different electrode placement systems that ensure that electrodes placed across the scalp
are always positioned on the same cranial landmark relative to other electrodes (Jurcak,
Tsuzuki & Dan, 2007). The 10-10 system with 128 electrodes placed across the scalp
(Figure 1) was used for this research. Data are continuously recorded from each
electrode. In the current study, however, data analyses are restricted to F3/F4 and P3/P4,
which is consistent with frontal asymmetry approaches (Beeney, Levy, Gatzke-Kopp, &
Hallquist, 2014).

Compared to other neuroimaging research methods, EEG is non-invasive and less expensive. One noteworthy advantage of EEG is its temporal sensitivity, as data are
recorded in the milliseconds range. This means that the data gathered will be reflective of
continuous neural activity—particularly important for the study of human emotion. Brain
electrical activity occurs in oscillations, and the alpha wave oscillates approximately 10
times per second (Luck, 2014). This means that each cycle of the alpha wave lasts for
approximately 100 milliseconds. As a recording tool, EEG has the capacity to measure
such a frequency. This is important because it will allow us to determine whether alpha asymmetry is present in the BPD sample compared to the healthy controls.

Given the time resolution afforded by EEG, it is a valuable tool to probe neural
processes that characterize BPD. Resting EEG activity—probing alpha asymmetry—has
been used to identify potential biomarkers of mental illness (Coan & Allen, 2004). EEG studies of emotion and cerebral asymmetry suggest that positive affect is associated with
greater activation of the left frontal lobe and approach related behaviors (Pizzagalli,
Sherwood, Henriques, & Davidson, 2005). Conversely, increased negative affect and
avoidance behaviors are associated with greater activation of the right frontal lobe (Davidson, Schwartz, Saron, Bennett, & Goleman, 1979). In other words, research suggests that emotion is lateralized in the brain. One of the early EEG experiments examining this pattern involved seventeen right-handed participants who were presented with emotionally varying stimuli and were asked to press down on a knob when they disliked what they were shown and let up on the knob when they liked the presented stimulus. The registered pressure changes on the knob were correlated with simultaneously gathered frontal and parietal EEG asymmetry scores, and it was found that positive affect was associated with relatively greater activation of the left frontal hemisphere as compared with negative affect. In contrast, negative affect was associated with relatively greater activation of the right frontal hemisphere compared with positive affect. No emotion related parietal asymmetry was observed (Davidson, Schwartz, Saron, Bennett, & Goleman, 1979).

Frontal and Posterior Asymmetry

Past research examining hemispheric asymmetry has often measured the alpha wave band whereby greater alpha power is indicative of less cortical activity (Tomarken, Dichter, Garber, & Simien, 2004). Therefore, greater alpha power in the left frontal hemisphere would reflect less neural activity in this region. Left frontal hypoactivity is considered a neurophysiological marker for vulnerability to depression (Schaffer, Davidson, & Saron, 1983; Davidson, 1993; Gotlib, Ranganath, & Rosenfeld, 1998), and adolescents at high risk for depression have demonstrated relative left frontal hypoactivity on alpha band measures (Tomarken et al., 2004).
Building on this research, a recent study (Beeney, Levy, Gatzke-Kopp, & Hallquist, 2014) compared frontal asymmetry and behavioral tendencies following rejection in female adults with BPD. No baseline asymmetry differences were reported between the healthy adults and those with BPD. However, following a rejection mood induction, adults with BPD showed left frontal asymmetry. While promising, little is known about pathophysiological markers in youth with BPD. Given profound neurodevelopmental differences in younger versus older individuals, further research is warranted.

In addition to frontal asymmetry, links between posterior asymmetry and depression have also been investigated but findings have been mixed (Schaffer, Davidson & Saron, 1983; Henriques & Davidson, 1990; Henriques & Davidson, 1991; Bruder, Fong, Tenke, Leite, et al., 1997; Kentgen, Tenke, Pine, Fong, Klein, et al., 2000; Debener, Beauducel, Nessler, Brocke, Heilemann & Kayser, 2000; Metzger, Paige, Carson, Lasko, Paulus, et al., 2004; Stewart, Towers, Coan & Allen, 2011) For instance, in adult samples, right posterior hypoactivation (i.e. greater alpha power) has been reported among those with current and remitted depressive disorder (Bruder et al., 1997; Henriques & Davidson, 1990; Stewart et al., 2011). On the other hand, significant posterior asymmetry differences have not been found between some samples of depressed patients and healthy controls (Henriques & Davidson, 1991; Debener et al., 2000). Furthermore, in cases of depression with comorbid anxiety, posterior asymmetry findings have shown to be reversed, such that right posterior hyperactivation (as opposed to hypoactivation in depression alone) has been noted (Metzger et al., 2004).

Similarly, youth focused studies with regard to posterior asymmetry have also
demonstrated conflicting results. While alpha asymmetry indicative of right posterior hypoactivation, as noted among depressed adults, has been found in a sample of female adolescents with depression (Kentgen et al., 2000), another youth sample revealed no such posterior asymmetry (Schaffer, Davidson & Saron, 1983). One study examining alpha asymmetry in high-risk adolescents, who had attempted suicide, suggested that attempters without a depressive disorder displayed greater alpha power (reduced activity) in the left posterior hemisphere, whereas attempters with major depressive disorder did not (Graae, Tenke, Bruder, Rotheram, Piacentini, et al., 1996). This study was the first to examine EEG alpha asymmetry among high-risk adolescents. The sample consisted of Hispanic females high school students from lower range socioeconomic status. The study showed that greater alpha (less activation) in the left posterior hemisphere was related more to suicidality rather than depression. No significant correlation was found between alpha asymmetry and depression severity.

At present, there is no EEG research on posterior alpha asymmetry in BPD. By examining frontal and posterior alpha asymmetry in youth with BPD, this study seeks to identify potential neural markers of BPD.

Study Aim & Hypotheses

This study examines whether female youth diagnosed with BPD exhibit more frontal or posterior alpha asymmetry relative to healthy youth. Specifically, three aims and hypotheses were tested:
Aim 1

I hypothesized that female youth diagnosed with BPD will exhibit greater frontal alpha asymmetry (i.e., less cortical activity in the left compared to the right hemisphere) than females without BPD. This hypothesis is based on previous studies that have shown a correlation between left frontal hypoactivation and depression (Schaffer, Davidson, & Saron, 1983; Davidson, 1993; Gotlib, Ranganath, & Rosenfeld, 1998).

Aim 2

I expected that greater frontal alpha asymmetry in youth with BPD will be associated with greater BPD severity and rumination. If frontal asymmetry emerges as a potential correlate of BPD, as it has in the case of depression, then it may be extrapolated that the degree of asymmetry may be correlated to the severity of BPD.

Aim 3

I also explored posterior alpha asymmetry to determine whether there is greater asymmetry in BPD relative to healthy youth. Given the mixed findings with regards to posterior alpha asymmetry and depression in youth (Schaffer, Davidson & Saron, 1983; Kentgen et al., 2000), this aim was largely exploratory.

Significance

Testing frontal and posterior alpha asymmetry in BPD may reveal important neurophysiological markers of BPD and help determine if there is a biological basis for this disorder (Sharp & Tackett, 2014). Identifying promising biological markers may lead
to early identification of BPD and could provide promising targets in the development of new interventions. Examining neural markers in youth is especially important as BPD is often manifested in adolescence (Kaess et al., 2014). Therefore, testing frontal and posterior asymmetry in youth is vital. In prior research, adults with BPD exhibited left frontal asymmetry following a rejection task (Beeney, Levy, Gatzke-Kopp, & Hallquist, 2014); however, no research has tested whether frontal or posterior asymmetry is present in youth diagnosed with BPD.
Chapter II
Method

Participants

The study included female youth (borderline personality disorder [BPD] = 33, healthy controls [HC] = 35) aged 13 to 23 years. Demographic data are summarized in Table 1. There were no between-group differences for age or ethnicity; however, BPD participants reported a higher family income. BPD participants reported a wide range of comorbid mental illnesses, including mood and anxiety disorders (see Table 2). Inclusion criteria for youth included English fluency, female, and right-handedness. Exclusion criteria for healthy participants included history of any psychiatric illness, psychotropic medication use, organic brain syndrome, neurological disorders, and history of seizures. BPD participants had the same exclusion criteria with the exception of psychiatric history and psychotropic medication use.

Procedure

The Partners Institutional Review Board provided approval for the study. Assent was obtained from female participants aged 13-17 years, and signed consent was obtained from participants 18 years and older as well as legal guardians of minors. The BPD sample was recruited from McLean Hospital’s 3East residential program, which provides intensive dialectical behavioral therapy (DBT). Healthy adolescents were recruited from the greater Boston community through flyers and online advertisements.
Table 1  
Demographic characteristics for the study sample

<table>
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<th>Healthy</th>
<th>BPD</th>
<th>Statistics</th>
<th>p-value</th>
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<td></td>
<td>(n = 35)</td>
<td>(n = 33)</td>
<td>t/t²</td>
<td></td>
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<tr>
<td>Age (SD)</td>
<td>18.14 (3.44)</td>
<td>17.21 (1.82)</td>
<td>1.38</td>
<td>0.17</td>
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<td>BPD symptoms (SD)</td>
<td>0.63 (0.91)</td>
<td>15.36 (7.49)</td>
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</tr>
<tr>
<td>Ethnicity, n (%)</td>
<td></td>
<td></td>
<td>2.5</td>
<td>0.64</td>
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<tr>
<td>White</td>
<td>24 (68.57)</td>
<td>25 (75.76)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>6 (17.14)</td>
<td>4 (12.12)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiple Races</td>
<td>3 (8.57)</td>
<td>4 (12.12)</td>
<td></td>
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</tr>
<tr>
<td>Black</td>
<td>1 (2.86)</td>
<td>0 (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>1 (2.86)</td>
<td>0 (0)</td>
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<tr>
<td>Household Income, n (%)</td>
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<td>18.09</td>
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<td>≥ $100,000</td>
<td>16 (45.7)</td>
<td>29 (87.9)</td>
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<tr>
<td>$75,000 - $100,000</td>
<td>2 (5.7)</td>
<td>1 (3.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$50,000 - $75,000</td>
<td>6 (17.1)</td>
<td>0 (0)</td>
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</tr>
<tr>
<td>$25,000 - $50,000</td>
<td>3 (8.6)</td>
<td>0 (0)</td>
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</tr>
<tr>
<td>$10,000 - $25,000</td>
<td>0 (0)</td>
<td>1 (3.0)</td>
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</tr>
<tr>
<td>&lt;$10,000</td>
<td>4 (11.4)</td>
<td>0 (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unknown/Unreported</td>
<td>4 (11.4)</td>
<td>2 (6.1)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note.* BPD = Borderline Personality Disorder
Table 2
Comorbidity for youth with borderline personality disorder (n = 33)

<table>
<thead>
<tr>
<th>Comorbidity</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Depressive Disorder/Dysthymia</td>
<td>24 (72.7)</td>
</tr>
<tr>
<td>Social Phobia</td>
<td>10 (30.3)</td>
</tr>
<tr>
<td>Attention Deficit Hyperactivity Disorder</td>
<td>9 (27.3)</td>
</tr>
<tr>
<td>Posttraumatic Stress Disorder</td>
<td>7 (21.2)</td>
</tr>
<tr>
<td>Bipolar II Disorder/ Bipolar Disorder NOS</td>
<td>7 (21.2)</td>
</tr>
<tr>
<td>Generalized Anxiety Disorder</td>
<td>6 (18.2)</td>
</tr>
<tr>
<td>Panic Disorder</td>
<td>6 (18.2)</td>
</tr>
<tr>
<td>Obsessive Compulsive Disorder</td>
<td>5 (15.2)</td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>4 (12.1)</td>
</tr>
<tr>
<td>Specific Phobia</td>
<td>2 (6.1)</td>
</tr>
<tr>
<td>Psychotic Disorder</td>
<td>1 (3)</td>
</tr>
</tbody>
</table>
The study included two visits. On the first study visit, participants were administered clinical interviews assessing psychiatric diagnoses, and additionally, they completed self-report measures assessing symptom severity. For the second study visit, 8 minutes of resting EEG data were acquired using a 128-channel HydroCel GSN Electrical Geodesics, Inc. (EGI) net. Each participant was remunerated $50.

**Instruments**

*Mini International Neuropsychiatric Interview for Children and Adolescents* (MINI-KID; Sheehan, Sheehan, Shytle, Janavs, Bannon, Rogers, et al., 2010). Participants were administered the MINI-KID to assess Axis I Disorders. The MINI-KID is a brief, structured diagnostic interview that assesses current Axis I psychopathology in youth. It has demonstrated good reliability and validity in past research (Sheehan et al., 2010; Auerbach, Millner, Stewart, Esposito, 2015).

*Structured Clinical Interview for DSM-IV Axis II Personality Disorders, BPD module* (SCID-II; First et al., 1994). The SCID-II is a semi-structured clinical interview assessing personality disorders. For this study, the module assessing borderline personality psychopathology was administered. Past research has shown that the BPD module of the SCID-II possesses strong psychometric properties (Huprich, Paggeot & Samuel, 2015; Bradley, Conklin, & Western, 2007).

*Zanarini Rating Scale for Borderline Personality Disorder* (ZAN-BPD; Zanarini, Vujanovic, Parachini, Boulanger, Frankenburg, & Hennen, 2003). The ZAN-BPD is a 9-item self-report instrument that assesses borderline personality disorder symptom
severity. Item scores range from 0 to 4 with greater scores indicating greater BPD severity. Exemplar items include anger, moodiness, emptiness, identity disturbance, self-destructive behavior, impulsivity, suspiciousness, fear of abandonment, and unstable relationships. In the current study, the Cronbach’s alpha was 0.93, indicating strong internal consistency.

_Ruminative Response Scale (RRS; Treynor, Gonzalez, & Nolen-Hoeksema 2003)._ The RRS is a 22-item self-report instrument that assesses participants’ response to depressed mood regarding thoughts about fatigue, difficulty concentrating, motivation, personal shortcomings, self-analysis and self-criticism, reasons for depressed mood, preoccupation with recent events, and so on. Item scores range from 1 (almost never) to 4 (almost always). In the current study, the Cronbach’s alpha was 0.97, highlighting excellent internal consistency.

_Electroencephalogram (EEG)_

EEG data were acquired using a 128-channel HydroCel GSN Electrical Geodesics, Inc. (EGI) net. Continuous EEG data was sampled at 250 Hz and referenced online to Cz. Resting EEG data consisted of eight contiguous, 1-min segments (four eyes open, four eyes closed). These eight segments were randomly sequenced in each participant and the order of the segments was counterbalanced across participants. Consistent with past research, this study included only the eyes closed data to ensure that visual stimuli would not negatively impact findings (Auerbach et al., 2015).

The four 1-minute segments were then concatenated using Matlab 8.1 (MathWorks, Natick, USA). BrainVision Analyzer 2.04 software (Brain Products,
Germany) was used to perform independent component analysis to remove eye movement artifacts and eye blinks. The acquired EEG data was analyzed using SPSS to assess frontal asymmetry. Frontal asymmetry scores were computed by subtracting the alpha power of left frontal electrodes (F3) from the alpha power of right frontal electrodes (F4). Similarly, posterior alpha asymmetry scores were computed by subtracting alpha power of P3 (left) from P4 (right). Furthermore, the alpha wave band was divided into two ranges: alpha 1 from 8.5 to 10 Hz and alpha 2 from 10.5 to 12 Hz. Alpha asymmetry scores were computed for alpha 1, alpha 2 and the total alpha range.

Data Analytic Overview

All data analyses were completed using SPSS. Correlations among all study variables were tested. Additionally, t-tests compared differences in alpha asymmetry in BPD and healthy youth. Outliers were removed for each alpha band (alpha 1, alpha 2 and total alpha) prior to all analysis. Hence, the results reflect different degrees of freedom.
Chapter III

Results

Preliminary Analyses

Within the BPD group, BPD symptoms were associated with rumination ($r = .43, p < .05$) and reflection ($r = .36, p < .05$). Contrary to my hypothesis, there was no correlation between frontal or posterior asymmetry and BPD symptoms. Within the healthy group, there was no correlation between BPD symptoms and rumination or reflection ($p > .05$), however, there was a modest correlation between BPD symptoms and depression ($r = .35, p < .05$) (see Tables 3 and 4).

Frontal Asymmetry

When comparing HC and BPD participants, there was no significant difference for the alpha 1, $t(66) = -.27, p = .79$, alpha 2, $t(62) = -.45, p = .65$, or total alpha $t(64) = -.10, p = .92$ (see Figure 1). Thus, the results obtained did not support our hypothesis on frontal asymmetry.
Posterior Asymmetry

BPD participants exhibited greater alpha 2 asymmetry relative to HC youth, $t(65) = 2.04$, $p = .046$ (see Figure 2). At the same time, between group comparisons were non-significant for alpha 1, $t(65) = 1.14$, $p = .26$ and total alpha, $t(65) = 1.37$, $p = .17$. This finding was unexpected as it was not part of my a priori hypothesis.
Table 3

*Correlations Among Alpha Asymmetry, BPD Symptoms, and Rumination*

*in Healthy Adolescents (n=35)*

<table>
<thead>
<tr>
<th>Healthy Adolescents</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Frontal Alpha 1</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Frontal Alpha 2</td>
<td>.79**</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Frontal Alpha Total</td>
<td>.95**</td>
<td>.91**</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Posterior Alpha 1</td>
<td>-0.08</td>
<td>-0.09</td>
<td>-0.1</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Posterior Alpha 2</td>
<td>0.18</td>
<td>-0.02</td>
<td>0.1</td>
<td>.49**</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>6. Posterior Alpha Total</td>
<td>0.09</td>
<td>-0.05</td>
<td>0</td>
<td>.87**</td>
<td>.79**</td>
<td>--</td>
<td></td>
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</tr>
<tr>
<td>7. BPD Symptoms</td>
<td>-0.01</td>
<td>0.02</td>
<td>0.01</td>
<td>-0.11</td>
<td>0.31</td>
<td>0.05</td>
<td>--</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>8. Rumination</td>
<td>0.24</td>
<td>0.03</td>
<td>0.18</td>
<td>0.18</td>
<td>0.29</td>
<td>0.31</td>
<td>0.29</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Reflection</td>
<td>0.23</td>
<td>0.02</td>
<td>0.16</td>
<td>0.24</td>
<td>0.24</td>
<td>.36*</td>
<td>0.18</td>
<td>.92**</td>
<td>--</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. Brooding</td>
<td>0.19</td>
<td>0.05</td>
<td>0.17</td>
<td>0.21</td>
<td>0.15</td>
<td>0.24</td>
<td>0.18</td>
<td>.86**</td>
<td>.82**</td>
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</tr>
<tr>
<td>11. Depression</td>
<td>0.22</td>
<td>0.34</td>
<td>0.18</td>
<td>0.1</td>
<td>0.32</td>
<td>0.26</td>
<td>.35*</td>
<td>.95**</td>
<td>.76**</td>
<td>.70**</td>
<td>--</td>
</tr>
</tbody>
</table>

Note: *p < 0.05; **p < 0.01
Table 4

Correlations Among Alpha Asymmetries, BPD Symptoms and Rumination in Youth with Borderline Personality Disorder (n=33)

<table>
<thead>
<tr>
<th>Borderline Personality Disorder</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Frontal Alpha 1</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Frontal Alpha 2</td>
<td>.73**</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Frontal Alpha Total</td>
<td>.93**</td>
<td>.87**</td>
<td>--</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Posterior Alpha 1</td>
<td>-.12</td>
<td>-.01</td>
<td>-.16</td>
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<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Posterior Alpha 2</td>
<td>-.04</td>
<td>.08</td>
<td>-.06</td>
<td>.83**</td>
<td>--</td>
<td></td>
<td></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>6. Posterior Alpha Total</td>
<td>-.15</td>
<td>-.04</td>
<td>-.19</td>
<td>.96**</td>
<td>.93**</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. BPD Symptoms</td>
<td>-.17</td>
<td>-.01</td>
<td>-.13</td>
<td>.11</td>
<td>.21</td>
<td>.12</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Rumination</td>
<td>-.22</td>
<td>-.31</td>
<td>-.27</td>
<td>-.1</td>
<td>-.13</td>
<td>-.11</td>
<td>.43*</td>
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</tr>
<tr>
<td>9. Reflection</td>
<td>.05</td>
<td>-.16</td>
<td>-.02</td>
<td>-.22</td>
<td>-.24</td>
<td>-.26</td>
<td>.36*</td>
<td>.75**</td>
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<tr>
<td>10. Brooding</td>
<td>-.21</td>
<td>-.34</td>
<td>-.28</td>
<td>-.08</td>
<td>-.17</td>
<td>-.12</td>
<td>.32</td>
<td>.85**</td>
<td>.56**</td>
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</tr>
<tr>
<td>11. Depression</td>
<td>-.22</td>
<td>-.24</td>
<td>-.25</td>
<td>-.02</td>
<td>-.01</td>
<td>.01</td>
<td>.39*</td>
<td>.92**</td>
<td>.48**</td>
<td>.68**</td>
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</tbody>
</table>

Note: *p < 0.05; **p < 0.01
Figure 2

Mean Frontal Asymmetry
Note. *$p < 0.05$
Chapter IV
Discussion

Borderline personality disorder is a mental disorder characterized by a pervasive pattern of instability in emotions, relationships, and self-image (Kaess, Brunner & Chanen, 2014). Symptoms may include a sense of emptiness, fear of real or imagined abandonment, impulsive, reckless, self-injurious or suicidal behavior, and psychotic or dissociative symptoms (DSM-5, American Psychiatric Association, 2013). Onset usually occurs in late adolescence or early adulthood and may cause significant psychosocial impairment (Keass et al., 2014; Paris, 2014). The noted correlation between self-injury and BPD (Dulit, Fyer, Leon, Brodsky, & Frances, 1994; Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006) and the identification of BPD as a risk factor for suicide (Soloff, Lis, Kelly, Cornelius, & Ulrich, 1994), make it necessary to further investigate the underlying neural correlates of this disorder.

Towards this end, EEG offers a cost effective and non-invasive method to examine the neurophysiological markers of BPD. Since current EEG research on BPD is limited, this study is one of the early attempts to adopt such a methodological approach. Thus far, one study has shown left frontal asymmetry in adults with BPD following a rejection mood induction task (Beeney, Levy, Gatzke-Kopp, & Hallquist, 2014). Based on this finding, as well as previous research on left frontal alpha asymmetry in depression (Schaffer, Davidson, & Saron, 1983; Davidson, 1993; Gotlib, Ranganath, & Rosenfeld, 1998), I hypothesized that youth with BPD sample will exhibit greater frontal alpha asymmetry than females without BPD. However, the results obtained in this study do not
support this. The lack of significant difference in frontal asymmetry across all alpha bands between the BPD and healthy group is somewhat surprising. One potential explanation may be that compared to the frontal asymmetry findings among adults with BPD (Beeney, Levy, Gatzke-Kopp, & Hallquist, 2014), youth with BPD may not exhibit similar frontal asymmetry. In other words, differential frontal asymmetry patterns in BPD may be a result of age related neurodevelopmental differences.

As part of my second hypothesis, I expected to find a positive correlation between frontal alpha asymmetry and BPD severity and rumination in youth with BPD. However, no correlation between frontal asymmetry and BPD symptoms was found. An association between BPD symptoms and rumination and reflection was observed in the BPD sample. Interestingly, a modest correlation between BPD symptoms and depression was also found in the healthy group. This finding is somewhat puzzling, as the healthy group was not expected to display BPD or depressive symptoms, let alone a correlation between them.

The results with regard to posterior asymmetry were most striking. Of the three alpha bands, alpha 2 reached significance indicating greater alpha power in the left compared to the right posterior lobe in the BPD sample. In other words, the BPD group exhibited left posterior hypoactivation. This finding is consistent with a previous study in which high-risk adolescents, who had attempted suicide and did not have a depressive disorder, displayed greater alpha power (reduced activity) in the left posterior hemisphere, whereas attempters with major depressive disorder did not (Graae, Tenke, Bruder, Rotheram, Piacentini, et al., 1996).
Limitations

This study has a few noteworthy limitations. First, since EEG data was gathered during rest, that is, when eyes are closed, the EEG asymmetry findings are not applicable to when responding to specific types of stimuli (e.g., affective-based words, images). Second, since EEG detects scalp-recorded activity, it cannot provide insight regarding neural activity in subcortical structures. Third, the generalizability of the study is limited. As the study focuses only on female youth within a specific age group, the findings may not apply to males or older populations. Last, comorbidity limits the ability to determine whether effects are unique to youth with BPD. Since some of the youth in the BPD sample had comorbid depression, anxiety disorders, and other mental illnesses, it is difficult to ascertain whether the observed posterior asymmetry is primarily due to BPD. However, youth with BPD and comorbid depression could not be excluded to maintain external validity.

Implications and Future Directions

This study is one of the early attempts to identify neural markers of BPD in youth using EEG. It was based on the premise that the proposed correlation between left frontal hypoactivation and depression (Schaffer, Davidson, & Saron, 1983; Davidson, 1993; Gotlib, Ranganath, & Rosenfeld, 1998) may also be displayed in BPD. However, the lack of significance in frontal alpha asymmetry in the BPD group implies that BPD and depression may not have similar neural markers with regard to frontal alpha asymmetry. However, more studies are needed to corroborate or contradict these findings.

The left posterior hypoactivation displayed in the BPD sample in this study poses
an interesting point of comparison to the right posterior hypoactivation noted in depressed samples (Bruder et al., 1997; Henriques & Davidson, 1990). Again, this may suggest that BPD and depression may actually have different pathophysiology, in contrast to what was initially hypothesized.

It is striking that left posterior hypoactivation displayed in this BPD sample was also observed in high-risk female adolescents who had attempted suicide (Graae, Tenke, Bruder, Rotheram, Piacentini, et al., 1996). As noted earlier, BPD has been identified as a major risk factor for suicide (Soloff, Lis, Kelly, Cornelius, & Ulrich, 1994; Yen, Shea, Pagano, Sanislow, Grilo, et al., 2003), and adolescents diagnosed with BPD report suicide ideation at earlier ages and with greater frequency (Venta, Ross, Schatte, & Sharp, 2012). This may suggest that suicidal tendencies and BPD have similar pathophysiology in youth (rather than BPD and depression). Significantly, posterior alpha asymmetry was observed in those female suicide attempters who did not have depression; that is, those who had depression and had attempted suicide did not display reduced left posterior activation. Again, this points to the differences in pathophysiology between depression, suicide and BPD. That is, BPD and suicide appear to have common neural markers, rather than BPD and depression as initially hypothesized.

Future studies could further explore the pathophysiology of suicide and BPD to identify correlations and differences. For instance, they may investigate whether there are different neural markers for youth with BPD who may or may not have attempted suicide. In other words, is left posterior hypoactivation more indicative of suicidal tendencies rather than BPD?

In addition, future studies could also include more varied populations based on
gender and age to better understand the pathophysiology of BPD in a more general sense. For instance, it may be important to test whether boys and older populations with BPD also exhibit posterior asymmetry. Studies including more varied groups may provide a more holistic picture of the neural markers of BPD, thereby opening new lines of inquiry for better intervention methods.
References


Psychophysiology, 48, 82–95.


(ZAN-BPD): A continuous measure of DSM-IV borderline psychopathology. 

