

Borderline personality disorder features in adolescent girls: P300 evidence of altered brain maturation

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Abstract

Objective: To examine brain maturation in adolescent girls with borderline personality disorder (BPD) features using the P300 event-related potential.

Methods: One hundred twenty-three girls, aged 14–19 yrs, were assigned to one of 4 groups formed by the crossing of BPD features (< vs. ≥ 5 BPD criteria) and median age (< vs. > 16.5 yrs). P300 responses were measured while subjects performed a complex visual oddball task.

Results: ANCOVAs of P300 amplitude—adjusting for variability associated with comorbid conduct disorder and depression symptoms—revealed a significant interaction. Among subjects without BPD features, aging was associated with the normal reduction in visual P300 amplitude. Among subjects with BPD features, there were no age-related changes. Additional analyses, which tested the effects of BPD features across the full age range, supported these findings.

Conclusions: The present findings suggest abnormal brain maturation among adolescent girls exhibiting features of BPD.

Significance: These results support a hypothesis of altered brain maturation in adolescents exhibiting BPD features at an early age. It is suggested that measures of brain maturation obtained during adolescence may improve our ability to predict BPD and comorbid disorders in adulthood.

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Adolescence is a period of significant neurological development. Alterations in brain maturation during the adolescent years may result in behavioral changes, particularly with regard to social development and risk-taking (Spear, 2000). Adolescence is also a developmental period in which psychiatric symptoms may emerge. Importantly, several studies have implicated altered brain maturation in a variety of clinical disorders (Kaufman and Charney, 2003; Siever et al., 2003).

The externalizing disorders are a subset of the clinical disorders frequently linked to altered brain development (Mezzacappa et al., 1999; Bauer and Hesselbrock, 1999, 2003; Tarter et al., 2004). Although the cardinal features of

these disorders—namely impulsivity and risk-taking—are considered a normal part of adolescent development, they can promote delinquency, suicidality, and substance abuse disorders if the behaviors are serious and persistent (Vitacco and Rogers, 2001; Chambers et al., 2003). As a result, there is a growing clinical and research interest in the role of brain development in individuals regularly exhibiting behavioral undercontrol.

A number of recent studies have utilized the P300 event-related electroencephalographic potential (ERP) as a means for examining brain development (Bauer and Hesselbrock, 1999, 2003; Hill et al., 1999; Hill and Shen, 2002). The P300 is a positive deflection in electrocortical activity that occurs approximately 300 ms after stimulus onset. The amplitude of the P300 is thought to reflect the allocation of attentional resources toward the processing of a rare event, whereas P300 latency is interpreted as an indication of

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stimulus evaluation time (Hillyard and Kutas, 1983; Donchin and Coles, 1988). The P300 is also sensitive to the effects of aging, although these effects are specific to the modality of the eliciting stimuli. Generally, studies of P300 amplitude in adolescents have demonstrated that auditory P300 amplitude increases with age (Ladish and Polich 1989; Polich et al., 1990; Hill et al., 1999; Bauer and Hesselbrock, 2003) while visual P300 amplitude decreases with age (Courchesne 1978; Katsanis et al., 1996; Bauer and Hesselbrock, 1999; Hill et al., 1999). Studies of P300 latency and aging suggest a general increase in latency with advanced age (Polich, 1996). Reports of latency changes during adolescence are sparse and less consistent (Zenker and Barajas, 1999).

In two previous studies, Bauer and Hesselbrock (1999, 2003) used a cross-sectional approach to examine the effects of brain maturation on P300 in adolescents with or without conduct disorder (CD). In both studies, subjects with a history of CD failed to exhibit normal maturational changes in P300 amplitude. Similarly, Hill and Shen (2002) reported differential developmental trajectories of P300 amplitude, with a slower rate of age-related change in P300 amplitude associated with a combination of childhood psychopathology (externalizing and internalizing) and a family history of alcoholism. The effect was particularly robust in girls.

The goal of the present study was to extend previous research by examining P300 indicators of maturation in another disorder characterized by impulsivity and risk-taking—borderline personality disorder (BPD). BPD is characterized by a pervasive pattern of instability in emotion regulation, interpersonal relationships, self-image, and impulse control (Skodol et al., 2002a,b). BPD is often the most common personality disorder found in clinical settings (Loranger et al., 1994), and is frequently associated with other disorders of clinical significance, including mood (Zimmerman and Mattia, 1999; Benazzi, 2000; Skodol et al., 2002a,b) and substance use disorders (Grilo et al., 1997; Zimmerman and Mattia, 1999).

Recent research has begun to recognize the importance of biological factors in the development, manifestation, and treatment of this disorder (Skodol et al., 2002a,b; Posner et al., 2003). Several studies have focused on altered brain function in adult BPD patients through a variety of electrophysiological and neuroimaging techniques (Blackwood et al., 1986; Kutcher et al., 1989; Drake et al., 1991; Soloff et al., 2000; Herpertz et al., 2001; Tebartz van Elst et al., 2001; Norra et al., 2003; Soloff et al., 2003). For example, previous research has demonstrated reduced P300 amplitude in adult BPD patients (Blackwood et al., 1986; Kutcher et al., 1989; Drake et al., 1991) as well as deficits in executive attention (Posner et al., 2003).

Few studies, however, have focused on the expression of BPD characteristics during adolescence or specifically in relation to neurodevelopment. This is likely due, in part, to the fact that the diagnosis of BPD during childhood and adolescence is a controversial topic (Bleiburg, 2000).

Yet, it is clear that borderline features do emerge during these developmental periods (Bleiburg, 2000; Paris, 2000; Crawford et al., 2001) and are significantly related to adult psychopathology (Becker et al., 2000). Recently, data from our laboratory have suggested that adolescents characterized with BPD features (i.e. endorsing 5 or more BPD characteristics on a screening instrument) exhibit decrements in P300 amplitude (Houston et al., 2004). In the present study, we hoped to expand upon this finding by evaluating potential maturation effects. Specifically, it was hypothesized that girls characterized with BPD features would not exhibit the same pattern of maturational change in visual P300 amplitude (a decrease in amplitude with age) as compared to girls in the non-BPD group. Furthermore, we examined the topography of these effects using high spatial resolution recording and analysis (i.e. current source densities (CSD) calculated via realistic head-shape boundary element method) techniques for the neuroanatomic localization of maturational differences in P300. Because previous research has suggested frontal brain deficits in relation to impulsivity, a hallmark feature of BPD, a frontal brain focus for group differences in P300 topography was also expected.

1. Method

1.1. Subjects

The subjects were 123 girls aged 14–19 years. They were recruited as part of a larger study of conduct problems and familial risk factors for alcoholism and drug abuse. They were not initially recruited for borderline personality disorder characteristics. Most subjects were recruited from the greater Hartford area via advertisements (e.g. are you the child of a substance abuser?) or presentations directed toward a parent enrolled in a substance abuse treatment program. Additional subjects were recruited through presentations before high school classes or guidance counselors within the inner-city, YMCA/YWCA organizations, police athletic leagues or similar venues geared toward children with a history of conduct problems.

A prospective subject or parent(s) were invited to telephone a research assistant for additional information about the study. Each subject and at least one biological parent were interviewed individually at the University of Connecticut Health Center. At the beginning of the session, a consent form agreement, approved by the Health Center's Institutional Review Board, was reviewed and signed by each parent and offspring. The subjects included in the present analysis were those girls who completed both the ERP assessment and the structured clinical interview for the DSM-III-R (SCID-II) personality disorders questionnaire (Spitzer et al., 1987).

During the session, the semi-structured assessment for the genetics of alcoholism (SSAGA; Bucholz et al., 1994;

Hesselbrock et al., 1999) was used to obtain a personal psychiatric history, which surveyed the major axis I psychiatric disorders defined in DSM-III-R. The SSAGA employs DSM-III-R, Feighner, DSM-IV and ICD-10 criteria to define alcoholism and has been shown to be a highly reliable and valid instrument in evaluating a variety of psychiatric conditions (Bucholz et al., 1994; Hesselbrock et al., 1999). This interview also elicited pertinent demographic and medical history data. Prospective subjects were excluded if they reported a history of head injury (e.g. loss of consciousness greater than five minutes), seizures, life-threatening disease, regular psychoactive medication use, alcohol or other drug dependence (excluding nicotine), schizophrenia or bipolar disorder, neurosurgery, major medical disorders (including diabetes, hypertension, renal disease, lupus, cardiac disorders, etc.). Visual acuity, color vision, and audiometric testing was performed to detect and exclude subjects with uncorrected visual or auditory deficits.

The assignment of a subject to the BPD-feature-positive (BPDf+) group ($n=61$) was defined as having 5 or more DSM-III-R BPD criteria as indicated by the structured clinical interview for the DSM-III-R (SCID-II) personality disorders questionnaire (Spitzer et al., 1987). Subjects endorsing less than 5 BPD criteria constituted the BPD-feature-negative (BPDf-) group ($n=62$). The cutoff of 5 BPD criteria was based on the number of criteria necessary for a DSM diagnosis of BPD.

Subjects were also divided into two groups based upon the median age of the entire sample (16 years). The younger group ($n=62$) was comprised of subjects aged 14–16 years, while those subjects between the ages of 17–19 constituted the older group ($n=61$).

1.2. P300 Task

The task used to elicit the P300 waveform was identical to a visual ‘oddball’ task previously shown to differentiate subjects with or without antisocial personality disorder (O’Connor et al., 1994), depression (Houston et al., 2003), or a family history of alcoholism (Begleiter et al., 1984). The task consisted of one block of 300 stimuli presented with an interstimulus interval of 1.5 s. Each stimulus was presented for 0.1 s on a computer screen located 1 m distant from the subject. A frequently ($n=220$) presented oval figure, 3.75 in. in diameter, served as the non-target stimulus. Two types of target stimuli, which resembled an aerial view of a human head, were presented less frequently ($n=40$ each) and pseudo-randomly interleaved among the non-target stimuli. Each target stimulus consisted of an oval (3.75 in. in diameter) and two smaller triangular symbols (0.25 in. wide) representing the ‘nose’ and one ‘ear’ of a human head. For one target stimulus type (easy discrimination), the ‘nose’ was positioned to point upward; for the other (difficult discrimination), the ‘nose’ pointed downward. For both types of target stimuli, the ‘ear’ occurred

with equal probability on either side of the head. The subject’s task was to press one of two response keys, positioned under right and left index fingers, to indicate whether the depicted symbol represented the left or right ‘ear’ of the head. The subject was required to practice the task for at least 5 min to ensure adequate comprehension of the instructions.

The target stimulus drawn with an upward-pointing nose permits a more rapid and accurate decision regarding right versus left because no mental rotation of the stimulus is needed—its right versus left orientation is consistent with that of the viewer. It therefore represents an easier condition than the target stimulus with the downward-pointing nose. In a previous study, we (Bauer and Hesselbrock, 1999) found that the more difficult, downward-pointing nose condition was optimal for revealing significant effects of conduct problems on P300 amplitude. Analyses of the present data were therefore limited to this condition.

1.3. ERP Recording

Subjects were seated in a sound-attenuated chamber and wore a fitted cap (Electro-Cap International, Inc., Eaton, OH) consisting of 31 electrodes. A single electrode placed on the bridge of the subject’s nose served as the reference. A mid-forehead electrode was the ground. For the detection of eyeblink and eye movement artifacts, a pair of electrodes was placed diagonally above and below the left eye. The 31 channels of the EEG and 1 channel of eye movement (EOG) activity were appropriately amplified (EEG gain=20K, EOG gain=2K) and filtered (bandpass=0.01–30 Hz) using a Grass Instrument Co. Neurodata Acquisition System (Model 12). Along with markers indicating stimulus and response onsets, the EEG and EOG channels were routed to an A/D converter, and sampled at a rate of 250 Hz for 50 ms preceding and 950 ms following the onset of each stimulus. During off-line computations, single trial data were sorted by electrode and stimulus type. Before averaging, eye movement artifacts were removed using the algorithm described by Semlitsch et al. (1986). Time-point averaged waveforms were then created, omitting trials with A/D converter overflow and incorrect responses. The P300 waveform was then identified as the peak voltage in the target stimulus waveform, between 250 and 900 ms following target stimulus onset. P300 amplitude was expressed in microvolts as the voltage difference between this peak and the average voltage during the 50 ms prestimulus period.

1.4. Topographical analysis

The topographic localization of P300 utilized a three-compartment head model computed using the boundary element method (BEM). This method has been shown to be superior to the three-shell spherical model because it is constrained by realistic head shape geometry and by

the thickness of each compartment. Indeed, when the current source resides in either frontal or temporal brain regions, the BEM model is highly preferred (Fuchs et al., 1998).

Ideally, a separate BEM calculation should occur for each subject to minimize interindividual variation in the thickness of the compartments. However, it was not possible to obtain magnetic resonance imaging (MRI) scans from the entire sample of 130 study subjects. Rather, group-averaged data were fitted to a single MRI obtained from a single, 17 year old female subject. The following compartment parameters were used to constrain the BEM model: 3-mm skin thickness, 9-mm skull thickness and 2-mm cortical thickness. The transformation of electrode locations and MRI data into the same coordinate system was accomplished using the CURRY software package (Neuroscan, Inc., Herndon, VA).

The source reconstruction method (Minimum Norm, maximum χ^2 regularization, equal location weighting) was applied to the difference waveform obtained by subtracting the group averaged ERP recorded on all trials for the two age groups (<16.5 vs. >16.5). This data reduction method is similar to procedures commonly used in the analysis of fMRI data. It yields a single topographic map that illustrates the difference between the two groups. Through this process, the reliability of the source reconstruction is improved relative to the alternate method of calculating a separate topographic map for each subject and subsequently averaging the maps over subjects.

Localization of the P300 difference occurred by converting scalp potential data to current source density (CSD) estimates (Hjorth, 1980). The calculation of a CSD map is roughly equivalent to the application of a high-pass spatial filter that, when applied to common-referenced data, markedly reduces the electrical distortions produced by the skull and scalp. For the present analysis, CSD measurements were obtained for each of the 3394 triangles that estimated the cortical surface. These measurements were converted to z scores for analysis. Z statistics exceeding a value of 3 SD units ($P < 0.005$) were deemed significant.

1.5. Statistical analyses

1.5.1. Demographic data

For the analysis of age and education differences, a one-way ANOVA was conducted with BPDF as the grouping variable. Chi-square analyses were used to examine the distribution of race among the experimental groups.

1.5.2. Behavioral performance data

Separate univariate ANOVAs were conducted on the number of correct responses and reaction times. BPDF and Age groups were the independent variables.

1.5.3. P300 Data

Principal components analysis (PCA) was used to reduce the number of independent analyses performed on

interrelated data and the overall probability of Type I error. The input to the PCA was the correlation matrix of P300 amplitudes or latencies measured at each of the 31 scalp locations. Through the PCA, scalp regions containing inter-correlated activity were identified and separated into functional units by orthogonal rotation. Each functionally distinct region was analyzed separately. The PCA yielded two orthogonal factors. The electrode sites loading highest on the first factor included 17 sites located adjacent or posterior to the central sulcus: C₃, CP₁, C₄, CP₂, P_z, P₃, PO₁, P₄, PO₂, CP₅, T₇, CP₆, T₈, P₇, P₈, O₁ and O₂. The second factor included the remaining anterior sites: F₃, F₇, FC₁, FC₅, F₄, FC₂, F₈, FC₆, FP₁, AF₁, FP₂, AF₂, F_z and C_z.

A 2 (BPDF+/BPDF-) \times 2 (<16.5/>16.5 years) \times 2 (anterior/posterior) repeated measures ANCOVA design was used to analyze P300 amplitudes and latencies. Scalp region served as the within-subjects variable while BPDF and Age groups were the between-subjects factors. Because recent work has demonstrated significant effects of conduct disorder (Bauer and Hesselbrock, 1999) and depression (Houston et al., 2003) on P300, and these disorders could confound the effects of BPDF, the number of lifetime DSM-III-R conduct disorder and depression symptoms were entered as covariates. In an attempt to validate the relationship between age and P300, a separate set of linear regression analyses were performed. P300 amplitude and latency values for the anterior and posterior scalp regions were each regressed against age for the BPDF- and BPDF+ groups separately.

2. Results

2.1. Demographic data

Demographic characteristics are summarized in Table 1. Analyses of the demographic variables indicated no significant difference between the BPDF groups in age. There was also no difference between the BPDF groups in educational level. Chi-square analysis for ethnic distribution across the 4 experimental groups indicated no significant differences ($\chi^2 = 2.82$, $P = 0.420$).

2.2. Behavioral performance data

Univariate analysis of the number of correct key press responses for the difficult condition indicated a significant main effect of BPDF [$F(1,117) = 8.76$, $P = 0.004$] (Table 2). BPDF- subjects correctly responded to more of the target stimuli than their BPDF+ peers. Analysis of reaction times indicated no significant effects ($P_s > 0.259$).

2.3. P300 Amplitude

Repeated measures analysis of P300 amplitude revealed a significant interaction of BPDF and Age [$F(1,117) = 6.33$,

Table 1
Demographic data [mean (SD)]

	BPDf+ <16.5 (n=34)	BPDf+ >16.5 (n=27)	BPDf– <16.5 (n=28)	BPDf– >16.5 (n=34)
<i>Demographics</i>				
Age	15.4 (0.6)	17.7 (0.7)	15.5 (0.6)	18.1 (0.9)
Yrs Education	9.1 (0.9)	11.5 (1.0)	9.4 (1.0)	11.9 (1.1)
BPD symptoms ^a	7.3 (1.9)	8.0 (2.4)	2.6 (1.3)	2.4 (1.4)
% Caucasian	55.9	81.4	71.4	64.7
<i>SSAGA</i>				
# Conduct disorder symptoms	2.4 (2.3)	2.3 (1.7)	0.8 (1.2)	0.9 (1.1)
# Depressive symptoms	2.5 (2.6)	3.4 (2.7)	1.1 (1.9)	1.3 (2.3)

BPDf+ = Borderline Personality Disorder feature positive; BPDf– = Borderline Personality Disorder feature negative; SSAGA = semi-structured assessment for the genetics of alcoholism.

^a # of BPD symptoms on the SCID-II personality disorders questionnaire.

$P=0.013$]. Analyses of simple effects revealed that older, BPDf– subjects exhibited smaller P300 amplitudes than younger, BPDf– subjects [$F(1,58)=10.03$, $P=0.002$] (Fig. 1; Table 2). The effect of age did not replicate within the BPDf+ group.

To further explore the effects of age, a linear regression analysis was employed in which P300 amplitude values were regressed against age for each BPDf group. The regression analysis indicated a significant linear decline in P300 amplitude across the entire age range for BPDf– subjects (Fig. 2). The decline in P300 amplitude was significant for both anterior ($r=0.336$, $\beta=-0.336$, $t=-2.763$, $P=0.008$) and posterior ($r=0.307$, $\beta=-0.307$, $t=-2.498$, $P=0.015$) scalp regions. However, this decline was not found in the BPDf+ group (anterior $r=0.025$; posterior $r=0.030$; $P_s>0.81$).

In addition to the BPDf×age interaction, the analysis of P300 amplitude also revealed a significant main effect for scalp region [$F(1,113)=87.98$, $P<0.001$]. In general, P300 amplitudes were larger over the posterior versus anterior

scalp. The difference between posterior and anterior P300 amplitudes did not vary as a function of the other independent variables.

Finally, symptoms of attention-deficit hyperactivity disorder (ADHD) were also explored as a potential confound in this sample. However, the prevalence of ADHD symptoms, obtained via SSAGA, in the present sample was quite low with only 16% of BPDf+ girls and 11% of BPDf– girls reporting at least one ADHD symptom. In addition, correlations between ADHD symptoms and P300 amplitude and latency were non-significant with r -values ranging from -0.17 to 0.07 ($P_s>0.13$).

2.4. P300 Latency

Repeated measures ANCOVA of P300 latency revealed a significant main effect for scalp region [$F(1,117)=4.85$, $P=0.030$]. P300 latency was longer over the posterior scalp. There were no significant effects of BPDf or Age.

Table 2
Behavioral performance and P300 data [mean (SD)]

	BPDf+ <16.5 (n=34)	BPDf+ >16.5 (n=27)	BPDf– <16.5 (n=28)	BPDf– >16.5 (n=34)
<i>Task performance</i>				
# Correct responses ^a	11.7 (13.4)	11.1 (10.9)	19.5 (13.1)	17.4 (13.4)
Reaction time (ms)	865 (36)	778 (41)	912 (25)	882 (39)
<i>P300</i>				
Anterior amplitude (μV) ^b	13.3 (5.4)	12.8 (4.7)	16.8 (5.8)	12.0 (4.7)
Posterior amplitude (μV) ^b	18.8 (6.9)	19.8 (6.0)	23.3 (7.8)	19.3 (6.0)
Anterior latency (ms)	426.1 (54.6)	443.7 (63.0)	432.7 (68.4)	416.4 (39.5)
Posterior latency (ms)	438.2 (53.0)	453.6 (55.7)	437.5 (66.6)	437.6 (43.5)

BPDf+ = Borderline Personality Disorder feature positive; BPDf– = Borderline Personality Disorder feature negative.

^a Significant difference between BPDf groups ($P<0.05$).

^b Significant BPDf×Age interaction ($P<0.05$).

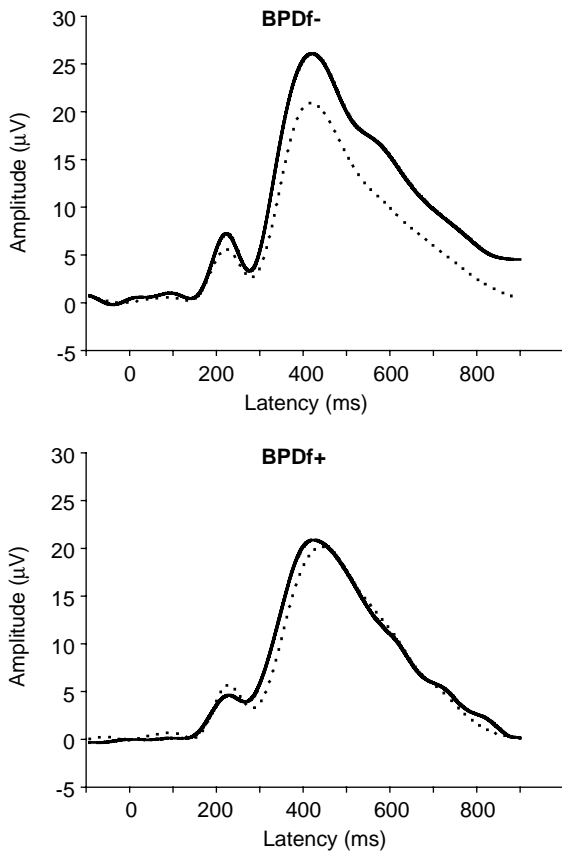


Fig. 1. Group averaged event-related potential waveforms at representative electrode C_z . The top and bottom panels represent the BPDf– and BPDf+ groups, respectively. The solid line represents the younger subjects (<16.5). The dotted line represents the older subjects (>16.5).

2.5. P300 Topography

The topographic map of the P300 current source density difference between younger and older adolescent girls for each BPDf group is presented in Fig. 3. Areas rendered in yellow or green denote areas where age significantly affected CSD. As can be seen in the left panel of Fig. 3, aging among girls in the BPDf– group was associated with changes in the right prefrontal region. In contrast, the effects of age on CSD within the BPDf+ group are negligible (right panel).

3. Discussion

The present study indicated that the girls in the BPDf+ group did not exhibit the expected age-related reduction in visual P300 amplitude. This pattern was evident, however, in the BPDf– control girls. These findings suggest a pattern of abnormal brain maturation related to features of borderline personality disorder. The absence of a maturational change in P300 amplitude is consistent with a previous demonstration of altered P300 maturation in boys with conduct disorder (Bauer and

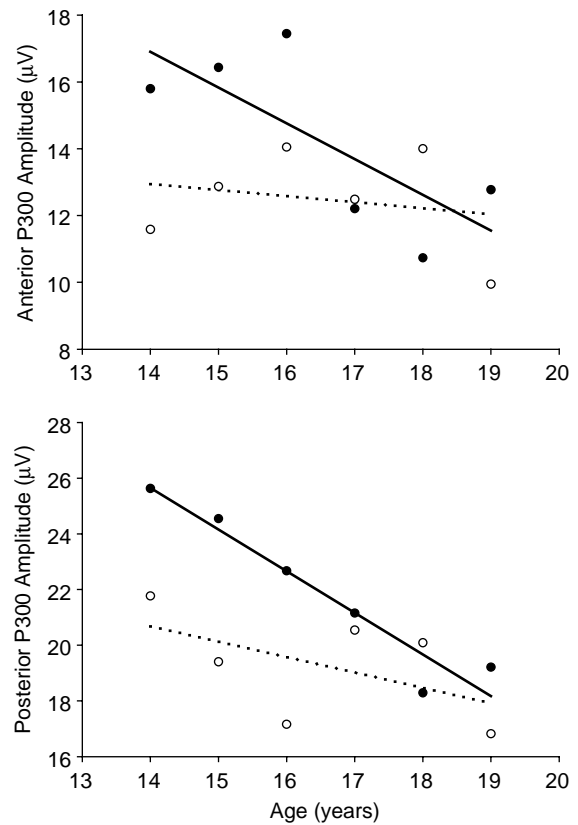


Fig. 2. Scatterplots of mean P300 amplitude across age for each scalp region. The top and bottom panels represent the anterior and posterior scalp locations, respectively. The solid lines/solid circles represent the BPDf– group. The dotted lines/open circles represent the BPDf+ group.

Hesselbrock, 2003). The present finding therefore provides further support for the notion that abnormal brain maturation during adolescence is a characteristic that may generalize across at least two of disorders characterized by externalizing behavior (Bauer and Hesselbrock, 1999, 2003; Mezzacappa et al., 1999; Tarter et al., 2004). It is important to note that the present findings were significant even when controlling for symptoms of conduct disorder and depression. There is evidence that abnormal brain maturation applies to other related disorders, such as attention deficit hyperactivity disorder (Rubia et al., 2000), as well. ADHD was not a significant problem in the current sample, but further investigation is warranted to determine the specific relationships between these factors.

The analysis of P300 topography via boundary element method mapping of current source density in this sample revealed an interesting result. It was found that the maturation of right frontal brain regions was specifically affected in adolescents with BPD features. Admittedly, this finding may be task-dependent. Because the task involves a mental rotation of a visual image, and does not have a prominent verbal component, it may challenge the function of the right hemisphere to a greater degree than the left

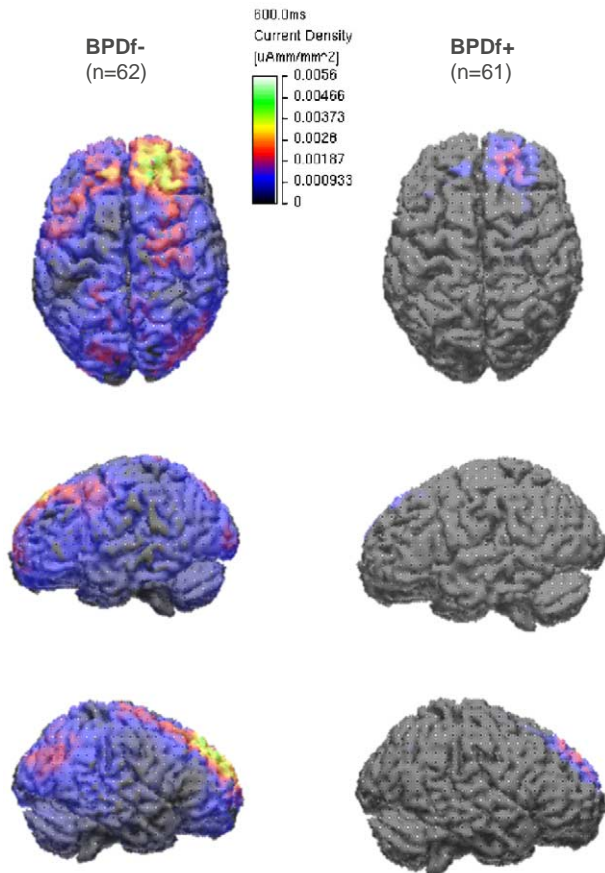


Fig. 3. Topographic map of the P300 current source density difference between younger and older adolescent girls. Areas rendered in green or yellow denote a significant effect of age. The left panel of the figure shows a significant effect of age on task-related activation of the right frontal cortex in the BPDf- group. This effect is absent in the BPDf+ group (right panel).

hemisphere. If we had employed a verbal working memory task which predominantly challenged left frontal areas of the brain, then the maturational deficit may have appeared on the opposite (left) side. Indeed, it seems unlikely that a neurodevelopmental disorder would be so selective and localized as to affect only the right frontal region. Other studies of adolescents with conduct disorder have detected altered function of both right and left frontal regions (Bauer and Hesselbrock, 1999, 2003).

The present study augments previous research on BPD in several additional ways. For example, the subjects were recruited from the community rather than from a clinical facility or practice group. As a result, the subjects did not possess a severe or acute level of psychopathology, lacked the complications of multiple co-occurring disorders, including substance dependence, psychosis or bipolar disorder, and had never been medicated for a psychiatric problem. Secondly, the present study employed a larger number of subjects than have been studied in comparable

ERP or neuroimaging studies. Thirdly, it employed a more complex cognitive activation than other studies and, therefore, offers greater power for detecting subtle deficits (Polich et al., 1994). Finally, the present study employed modern topographic mapping techniques, i.e. current source density calculated via realistic head-shape boundary element method, for identifying the anatomical location of the neurophysiological difference in brain maturation.

Despite the additions the present study may make to the current literature, there are a number of methodological limitations as well. It is anticipated that the present findings will serve primarily as an impetus to future work in this area. As a result, the current study was limited to female subjects in deference to the apparent gender bias in the diagnosis of BPD. Therefore the present results might not generalize to males with BPD features. Further, the subjects in the present study were not specifically recruited for their BPD symptoms and were not grouped according to a clinical diagnosis of BPD. Thus, unlike many studies that have examined BPD patients, the degree of symptom severity in the present sample was subthreshold. It is recognized that not every girl in the present BPDf+ group will go on to meet criteria for BPD as an adult. However, this feature of the study might also serve as a potential strength inasmuch as significant differences in brain maturation were demonstrated in individuals characterized by a mild level of BPD symptoms. Finally, the present study does not allow for an examination of the sample through the period of risk for BPD. We acknowledge that the BPDf grouping in the present study was based on a one-time assessment which does not allow for evaluation of the stability of the BPDf grouping over time. Ideally, future work will employ a longitudinal design which would be more useful for determining the true course of the disorder and the ability of the present measures to predict adult BPD or its axis I concomitants.

Finally, the diagnosis of BPD during adolescence is currently controversial (Bleiburg, 2000). Clinicians are often hesitant to diagnose adolescents with a personality disorder until young adulthood, when personality features are considered to be more stable. Yet, the present findings suggest that BPD features in adolescence are associated with a significant difference in P300 amplitude and its maturation. The measurement of P300 amplitude, and its maturation, in a larger sample of adolescents than were studied here may prove useful for resolving clinical controversies regarding the stability of the BPD diagnosis.

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