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Resting posterior alpha power and adolescent major depressive disorder

Akina Umemoto [a], Lidia Y.X. Panier [b], Sally L. Cole [a], Jürgen Kayser [a,b], Diego A. Pizzagalli [c,d], Randy P. Auerbach [a,b,e,*]

[a] Department of Psychiatry, Columbia University, New York, NY, USA
[b] New York State Psychiatric Institute, New York, NY, USA
[c] Department of Psychiatry, Harvard Medical School, USA
[d] Center for Depression, Anxiety and Stress Research, McLean Hospital, Belmont, MA, USA
[e] Division of Clinical Developmental Neuroscience, Sackler Institute, USA

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ABSTRACT

For several decades, resting electroencephalogram (EEG) alpha oscillations have been used to characterize neurophysiological alterations related to major depressive disorder. Prior research has generally focused on frontal alpha power and asymmetry despite resting alpha being maximal over posterior electrode sites. Research in depressed adults has shown evidence of hemispheric asymmetry for posterior alpha power, however, the resting posterior alpha-depression link among adolescents remains unclear. To clarify the role of posterior alpha among depressed adolescents, the current study acquired eyes-closed 128-channel resting EEG data from 13 to 18 year-old depressed ($n = 31$) and healthy ($n = 35$) female adolescents. Results indicated a significant group by hemisphere interaction, as depressed adolescents exhibited significantly larger posterior alpha (i.e., lower brain activity) over the right versus left hemisphere, whereas healthy adolescents showed no hemispheric differences. Relatively greater alpha over the right versus left hemisphere correlated with depression symptoms, anhedonia symptoms, rumination, and self-criticism. Further, depressed adolescents had reduced overall posterior alpha compared to healthy youth; though, no associations with symptoms and related traits emerged. Resting posterior alpha may be a promising neurophysiological index of adolescent depression, and more broadly, may relate to risk factors characterized by enhanced perseveration.

1. Introduction

Major depressive disorder (MDD) is a leading cause of disability worldwide with onset peaking during adolescence (Avenevoli et al., 2015). For several decades, research has used the electroencephalogram (EEG) to investigate alpha oscillations as a neurobiological marker of MDD (Coan and Allen, 2004; Davidson et al., 1985; Fingelkurts and Fingelkurts, 2015). Typically measured between 8 and 13 Hz, scalp-recorded resting alpha power is observed during a relaxed, wakeful state, is most prominent during eyes closed (Fingelkurts and Fingelkurts, 2015), and inversely relates to cortical activity (Oakes et al., 2004). The majority of this research has focused on frontal asymmetry, showing that depression is generally characterized by relatively greater alpha over the left versus right hemisphere (Allen et al., 2004; Davidson, 1998; Thibodeau et al., 2006; van der Vinne et al., 2017). Markedly less research has tested resting posterior alpha, but there is evidence of resting alpha alterations over posterior regions in MDD. However, the opposite effect emerges—namely, relatively greater alpha over the right versus left hemisphere among depressed individuals (Blackhart et al., 2006; Bruder et al., 1997; Stewart et al., 2011). Research also has shown increased overall posterior alpha, indicating reduced cortical activity, in depressed adults (Flor-Henry et al., 2004; Grin-Yatsenko et al., 2010; Henriques and Davidson, 1990; Pollock and Schneider, 1990). Despite these findings in adults, the resting posterior alpha-depression link in youth is less clear. Given that resting alpha is maximal over posterior regions, particularly when participants have their eyes closed (Debener et al., 2000; Stewart et al., 2014), and studies have demonstrated strong temporal stability for overall posterior alpha (Tenke et al., 2018) and acceptable stability for posterior alpha asymmetry (Debener et al., 2000; Tenke et al., 2018), we sought to clarify the role of posterior alpha in adolescent MDD.

* Corresponding author. 1051 Riverside Drive, Pardes 2407, New York, NY, 10032, USA.
E-mail address: rpa2009@cumc.columbia.edu (R.P. Auerbach).

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1.1. Posterior alpha power

Posterior Alpha Asymmetry. Resting posterior asymmetry—relatively greater alpha over the right versus left hemisphere—has been observed in depressed adults (Blackhart et al., 2006; Bruder et al., 1997), depressed adolescents (Kentgen et al., 2000), and high-risk youth and adult offspring of depressed parents (Bruder et al, 2005, 2007, 2012). Resting posterior alpha asymmetry is heritable (Bruder et al, 2005, 2007), persists into remission (Henriques and Davidson, 1990), and predicts antidepressant treatment response (Bruder et al, 2008, 2013; Ulrich et al., 1986). Collectively, these findings suggest that resting posterior alpha asymmetry may provide important insight into adolescent MDD (Bruder et al, 2005, 2007, 2012; Davidson, 1992; Henriques and Davidson, 1990).

Overall Posterior Alpha. A growing body of research also has highlighted a relationship between overall resting posterior alpha power and MDD. Overall resting posterior alpha has shown strong reliability (Debener et al., 2000; Smith et al., 2020), temporal stability in healthy (Enoch et al., 2008; Smit et al., 2005; Tenke et al, 2017a, 2018) and depressed adults (Bruder et al., 2008), and intergenerational heritability (Enoch et al., 2008; Smit et al., 2005). Increased resting overall posterior alpha power (i.e., cortical deactivation) characterizes adults with depression (Flor-Henry et al., 2004; Grin-Yatsenko et al., 2010; Pollock and Schneider, 1990), however, several studies have found the opposite pattern of effects—decreased overall resting posterior alpha power (i.e., increased cortical activity (Jiang et al., 2016; Volf and Passynkova, 2002; Zoon et al., 2013)). In light of these mixed findings and limited research in adolescents, further research is warranted to investigate the relationship between resting posterior alpha and depression among adolescents.

1.2. Posterior alpha and associated psychological constructs

Although a large body of research has shown abnormalities in posterior alpha asymmetry (Bruder et al., 1997; Henriques and Davidson, 1990; Kentgen et al., 2000) and overall posterior alpha (Flor-Henry et al., 2004; Grin-Yatsenko et al., 2010; Pollock and Schneider, 1990) among depressed individuals, it is plausible alpha alterations may imp act core psychological processes that lead to the emergence of a wide range of mental disorders, including general anxiety disorders (Bruder et al., 1997), panic disorder (Heller et al., 1997), and post-traumatic stress disorder (Kemp et al., 2010; Metzger et al., 2004). For example, resting frontal alpha asymmetry is believed to reflect approach-avoida nce behaviors (Coan and Allen, 2004; Davidson, 1998; Harmon-Jones, 2003), with relatively reduced left frontal activity indexing reduced motivation, anhedonia, and blunted reward sensitivity (Coan and Allen, 2004; Davidson, 1998; Harmon-Jones, 2003; Nusslock et al., 2018; Shankman and Klein, 2003). The associated constructs related to posterior alpha at rest are less clear, but there is some preliminary evidence showing that relatively reduced posterior alpha power over the right versus left hemisphere may be associated with increased anxious arousal (Bruder et al., 1997; Heller et al., 1997; Metzger et al., 2004; Nitschke et al., 1999), whereas reduced left versus right alpha power was associated with worry and rumination (Heller et al., 1997; Nitschke et al., 1999; Schmidtke and Heller, 2004). Building on this prior research, we sought to clarify whether posterior alpha—asymmetry and overall posterior alpha—in depressed female adolescents associated with symptom severity (e.g., anhedonia, anxiety) and traits related to perseverative patterns of thinking, including self-criticism (i.e., overly critical self-view; (Kopala-Sibley et al., 2015), dependency (i.e., tendency to repetitively think about and rely on close relationships; (Adams et al., 2009), and rumination (i.e., tendency to perseverate about depressed mood; (Abela and Hankin, 2011; Sarin et al., 2005). Clarifying the relationships among symptoms, traits, and posterior alpha is critical, as it may inform the development of transdiagnostic interventions that target symptoms and traits that cut across traditional diagnostic boundaries.

1.3. Current study

Presently, there is limited research elucidating the relationship between resting posterior alpha and depression relationship among adolescents. To address this gap, the following hypotheses were tested among healthy and depressed female adolescents. First, compared to healthy adolescents, depressed youth will exhibit relatively greater right versus left posterior alpha asymmetry as well as increased overall posterior alpha power. Second, given that research has demonstrated resting posterior alpha power relationships with both arousal symptoms and perseverative traits, we tested whether resting posterior alpha power asymmetry and overall power were associated with core symptoms (i.e., anhedonia, anxiety) and traits (i.e., self-criticism, dependency, rumination). Third, research has demonstrated that alpha power is related to suicidal thoughts and behaviors (Benschop et al., 2019; Graae et al., 1996). Graae et al. (1996) found that depressed female adolescent suicide attempters exhibited increased posterior alpha over the right relative to the left hemisphere compared to non-attempters. Similarly, Benschop and colleagues (Benschop et al., 2019) found greater increased overall resting posterior alpha power among adult suicide ideators relative to depressed individuals with no history of suicidal thoughts and behaviors. Consequently, we explored whether resting posterior asymmetry and overall posterior alpha were related to suicidal thoughts and behaviors among adolescents.

2. Material and methods

2.1. Participants

Healthy control (HC = 43) and depressed (MDD = 38) female adolescents ages 13–18 years ($M = 15.5$, SD = 1.69) were recruited through online advertisements, posted fliers, and direct mailing from the greater Boston area. All participants were right-handed (based on self-report assessment (Chapman and Chapman, 1987), and fluent in English, and were excluded if they reported a seizure disorder, and/or a head injury resulting in loss of consciousness (>5 min). HC participants were excluded if they reported a lifetime mental disorder or were currently using psychotropic medication. All MDD participants reported a current major depressive episode, and 13 participants reported a secondary anxiety diagnosis (panic disorder and agoraphobia = 1; general anxiety disorder = 12). Approximately one-third of the MDD participants (36.8%) were medicated (Selective Serotonin Reuptake Inhibitor = 13 [Citalopram ($n = 1$), Escitalopram ($n = 3$), Fluoxetine ($n = 5$), Sertraline ($n = 4$)]; Bupropion = 1); however, only a smaller subset of these medicated participants ($n = 9$) was retained in the final analyses.

2.2. Procedure

The Partners Human Research Committee Institutional Review Board approved all study procedures. Following an initial screening to determine eligibility, parental consent and adolescent assent or consent were obtained. At baseline, study staff administered clinical interviews assessing lifetime mental disorders, and adolescents completed self-report measures. Approximately 7–10 days later, 8-min of high-density resting EEG (4-min eyes closed and 4-min eyes open) were acquired.

2.3. Clinical characterization

Adolescents were administered the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present (K-SADS; Kaufman et al., 1997) to assess lifetime mental disorders based on DSM-IV-TR criteria, which is the gold-standard diagnostic interview in adolescents. Adolescents also were administered the Columbia Suicide

Severity Rating Scale - Children's Lifetime/Recent Version (C-SSRS-L/R; Posner et al., 2011). The C-SSRS-L/R is a structured clinical interview assessing suicidal thoughts and behaviors. In the current study, we focused on lifetime suicidal thoughts and suicidal behaviors with each question scored dichotomously (Yes = 1; No = 0). Lifetime suicidal thoughts reflected the sum of the 5 ideation items whereas suicidal behaviors reflected the sum of the 5 behavioral-oriented questions.

Adolescents also completed several symptom and trait self-report measures. Specifically, depressive symptoms were assessed with the Beck Depression Inventory-II (BDI-II; Beck et al., 1996), which is a 21-item inventory assessing depression symptoms over the past 2 weeks. Item-scores ranged from 0 to 3, and higher scores indicated more depression severity. Additionally, the Snaith-Hamilton Pleasure Scale (SHAPS; Snaith et al., 1995) is a 14-item measure that assessed anhedonia symptoms. Item values ranged from 1 to 4, with higher scores indicating greater anhedonia severity. Anxiety symptom severity was assessed using the Multidimensional Anxiety Scale for Children (MASC; March et al., 1997). The MASC is a 39-item measure, which assessed anxiety symptoms. Items ranged from 0 to 3, and higher scores reflected greater anxiety symptom severity.

To determine whether resting state processes were associated with perseverative traits, participants completed measures assessing rumination, dependency, and self-criticism. The Children's Response Styles Questionnaire (CRSQ; Abela et al., 2000) includes a 13-item subscale assessing rumination, with item values ranging from 1 to 4 and higher scores indicating a greater tendency to ruminate. The Children's Depressive Experiences Questionnaire (CDEQ; Abela and Taylor, 2003) is a 20-item measure that assesses dependency (e.g., "I very much want most people I know to like me.") and self-criticism (e.g., "I only feel that I am a good person when I do things well."). Item values ranged from 1 to 3, and higher scores on each subscale indicated greater dependency and self-criticism, respectively.

2.4. Resting EEG data acquisition

Continuous EEG data were recorded using a 128-channel HydroCel geodesic sensor net (Electrical Geodesics, Inc., EGI) with a Cz reference (which was re-referenced offline to the average of all channels; see Supplement) and digitized at 250 Hz. Impedances were maintained below 75 kΩ. Data were continuously recorded while participants had their eyes open (EO) or closed (EC) for a total of 8 min (i.e., 4 min EO and 4 min EC). During the EO condition, a central white fixation cross was presented on a black background of a computer screen, and participants were instructed to fixate their eyes while minimizing blinking. Within the EC condition, participants were instructed to close their eyes. For both conditions, participants were instructed to remain as still as possible to reduce motor artifacts. EO and EC conditions were collected contiguously within 1-min segments. The condition order was counterbalanced across participants.

2.5. Data Reduction and Analysis

Post-processing and data visualization were performed in MATLAB 2018a (MathWorks, Natick, USA) using EEGLAB (Delorme and Makeig, 2004) and ERPLAB (Lopez-calderon and Luck, 2014) toolboxes (see Supplement for data processing pipeline). Given that the primary focus of our analyses was on posterior alpha, spectral power in the range of 8–13 Hz (Bruder et al., 2013; Smith et al., 2020; Stewart et al., 2011) was extracted from posterior electrode locations based on a visual inspection of the topographical map across all participants (Fig. S1), which is similar to the selected electrodes for posterior alpha in our previous work (Smith et al., 2020; Tenke et al., 2017a) and were averaged across frequency for all analyses (see Supplement for alpha peak analysis). Consistent with prior posterior alpha power asymmetry research (Bruder et al, 1997, 2005), three electrodes from the left hemisphere (channels 60 (P3), 59 (P7), and 70 (O1)) and right hemisphere (channels 85 (P4), 91 (P8), and 83 (O2); Fig. S1) were separately averaged. Posterior log alpha power asymmetry scores were calculated for each participant by subtracting posterior alpha power over the right hemisphere from that over the left hemisphere; positive scores indicate increased posterior alpha power over the left hemisphere compared to the right hemisphere, whereas negative scores indicate increased posterior alpha power over the right hemisphere compared to the left hemisphere (Allen et al., 2004). Consistent with our prior studies in depression (Auerbach et al., 2015; Pizzagalli et al, 2001, 2006), only results from EC data are reported (see Supplement for results averaged across conditions). Although the primary focus of the current study was resting posterior alpha, for completeness, we also include analyses relating to resting frontal alpha asymmetry (see Supplement).

Data were excluded if there was more than: (a) 25% of channels bridged or visually identified as bad and/or (b) 25% of epochs removed due to motor and other artifacts (Tenke et al., 2017a, 2017b). Based on these criteria, data from 11 subjects were removed: bridged channels = 10 (HC = 5, MDD = 5) and artifacts = 1 (MDD). Additionally, data from two participants were removed as a result of low-quality raw EEG data (HC = 1, MDD = 1), and data from two HC participants were not recorded due to a technical issue. Thus, data were analyzed for the EC condition in 66 participants (HC = 35, MDD = 31). There were no group differences in the number of bridged channels (HC = 1.0 ± 4.14, MDD = 2.06 ± 4.69; $t(64) = -0.98$, $p = 33$) and corrupted channels (HC = 2.4 ± 2.59, MDD = 2.45 ± 3.57; $t(64) = -0.07$, $p = 0.95$).

2.6. Statistical analysis

All statistical analyses were performed in SPSS 24 (IBM). The MDD group was older than the HC (Table 1) and was included as a covariate in all models. Notably, posterior alpha asymmetry scores ($t(29) = -0.37$, $p = 0.72$) and posterior alpha ($t(29) = 0.16$, $p = 0.88$) did not differ among medicated ($n = 9$) and unmedicated ($n = 22$) depressed participants. Thus, medication status was not included as a covariate. Given the distribution of self-report measures across all participants, Spearman's rank-order correlations across all participants and within the MDD group were examined among posterior alpha power asymmetry scores, posterior alpha power, and self-report questionnaire scores. A repeated measures ANOVA was conducted on posterior alpha power asymmetry with *Group* (HC, MDD) as a between-subject factor, *Hemisphere* (Right, Left) as a within-subject factor, and *Age* as a covariate. We also conducted a binomial test separately for each group to evaluate whether participants were more likely to exhibit posterior alpha power asymmetry in the MDD group compared to in the HC group. For this analysis, the pattern of alpha power asymmetry was coded categorically for each participant; participants with a negative asymmetry score (i.e., increased posterior alpha power over the right compare to the left hemisphere) were coded as 1, whereas participants with a positive asymmetry score (i.e., increased posterior alpha power over the left compare to the right hemisphere) were coded as 0. We then conducted a chi-square test to examine whether MDD participants were more likely to exhibit posterior alpha power asymmetry compared to HC participants. Univariate analysis of variance (ANOVA) was conducted separately on overall posterior alpha power with *Group* as a between-subject factor and *Age* as a covariate. Given that the suicide-related scores were count variables, exploratory negative binomial regression analyses tested whether posterior alpha power asymmetry and overall posterior alpha power scores were associated with suicidal thoughts or behaviors over and above depression severity. All models included age as an offset variable.

3. Results

3.1. Descriptive data

Descriptive data comparing between HC and MDD are summarized

Table 1
Descriptive statistics for the sample stratified by group.

	HC ($n = 35$)	MDD ($n = 31$)	*t* or $\chi 2$ (*df*)	*p*	*d* or *Cramer's V*
Age *M* (*SD*)	15.20 (1.62)	16.16 (1.59)	−2.42 (64)	0.02	0.60
Ethnicity					
White (*n*)	27	24	2.37 (3)	0.50	0.19
Asian (*n*)	5	2			
Black or African American (*n*)	0	1			
More than one race (*n*)	3	4			
Unknown or Not Reported (*n*)					
Family Income					
$25,000 or less (*n*)	0	1	4.20 (3)	0.24	0.25
$25,000-$75,000 (*n*)	2	6			
$75,000 or more (*n*)	25	18			
Unknown or Not Reported (*n*)	8	6			
Self-report Questionnaires					
Depression Symptoms *M* (*SD*)	1.92 (3.19)	30.84 (9.78)	−16.54 (64)	<0.01	3.98
Anhedonia Symptoms *M* (*SD*)	17.23 (3.69)	30.91 (6.18)	−11.06 (64)	<0.01	2.69
Anxiety Symptoms *M* (*SD*)	39.16 (16.10)	63.84 (19.58)	−5.62 (64)	<0.01	1.38
Rumination *M* (*SD*)	22.46 (8.65)	40.21 (6.37)	−9.39 64)	<0.01	2.34
Self-Criticism *M* (*SD*)	16.51 (4.02)	21.67 (3.74)	−5.36 (64)	<0.01	1.33
Dependency *M* (*SD*)	19.45 (3.68)	23.89 (3.79)	−4.83 (64)	<0.01	1.19
Clinical Interview (HC: n = 25; MDD: n = 28)					
Suicidal thoughts *M* (*SD*)	–	2.61 (1.71)			
Range		0–5			
Suicidal behaviors *M* (*SD*)	–	0.71 (0.94)			
Range		0–3			

Note. *d* = Cohen's *d*;
Beck Depression Inventory II = Depression Symptoms; Snaith-Hamilton Pleasure Scale = Anhedonia Symptoms; Multidimensional Anxiety Scale for Children = Anxious Symptoms; Children's Response Styles Questionnaire = Rumination; Children's Depressive Experiences Questionnaire = Self-Criticism and Dependency.

in Table 1. Depressed adolescents were slightly older than healthy youth, but there were no differences in ethnicity or family income. Internal consistency of each self-report measure and correlations among these measures are provided in Table 2. All self-report instruments showed excellent internal consistency (Cronbach's alphas>0.8). Split-half reliability using Spearman-Brown Coefficient was excellent for posterior alpha asymmetry and overall posterior alpha (Table 2).

3.2. Posterior alpha power

Posterior Alpha Asymmetry. The repeated measures ANOVA for posterior alpha power asymmetry with *Group* as a between-subject factor, *Hemisphere* as a within-subject factor, and age as a covariate revealed a non-significant effect of *Group*, $F(1,63) = 3.19$, $p = 0.08$, $\eta_p^2 = 0.05$ (MDD: $M = 2.96\ \mu V^2$, SD = $0.47\ \mu V^2$; HC: $M = 3.14\ \mu V^2$, SD = $0.31\ \mu V^2$), and a significant *Group* × *Hemisphere* interaction, $F(1,63) = 5.94$, $p = 0.02$, $\eta_p^2 = 0.09$ (Fig. 1A and B), indicating that groups differed in their relative posterior alpha asymmetry.[1] Bonferroni-corrected post-hoc tests revealed that MDD adolescents exhibited relatively reduced posterior alpha power over the left hemisphere ($M = 2.91\ \mu V^2$, SD = $0.45\ \mu V^2$) compared to the right hemisphere ($M = 3.00\ \mu V^2$, SD = $0.49\ \mu V^2$; $p < 0.01$, $\eta_p^2 = 0.16$). Conversely, HC showed no hemispheric differences ($p = 0.99$, $\eta_p^2 < 0.01$). A binomial test revealed that the proportion of MDD participants exhibiting a posterior alpha asymmetry (0.81; $n = 25$ vs. 6) was significantly higher than the null hypothesized proportion (0.50), $p < 0.01$. By contrast, the proportion of HC participants exhibiting a posterior alpha asymmetry (0.51; $n = 18$ vs. 17) was not significantly higher than the null hypothesized proportion (0.50), $p = 1.00$. A chi-square test further indicated that MDD participants were significantly more likely to exhibit posterior alpha asymmetry compared to HC participants, $X^2(1, N = 66) = 6.18$, $p = 0.01$. Cramer's $V = 0.31$.

Overall Posterior Alpha. The univariate ANOVA on overall posterior alpha power with *Group* as a between-subject factor and age as a covariate revealed a significant main effect of *Group*, $F(1,63) = 4.15$, $p = 0.046$, $\eta_p^2 = 0.06$, such that MDD adolescents ($M = 2.94\ \mu V^2$, SD = $0.43\ \mu V^2$) showed significantly reduced posterior alpha power compared to HC adolescents ($M = 3.13\ \mu V^2$, SD = $0.30\ \mu V^2$) (Fig. 1C and D). Age was not a significant predictor of posterior alpha power ($p = 0.98$).

3.3. Clinical associations with posterior alpha power

Across all participants posterior alpha power asymmetry was negatively correlated with depression symptoms, anhedonia severity, rumination, and self-criticism (i.e., greater severity was associated with increased posterior alpha power over right electrode sites) (Table 2). Overall posterior alpha power was not significantly correlated with clinical variables (Table 2; Figs. 2 and 3). Associations with alpha asymmetry did not remain significant when restricted to adolescents with MDD, suggesting that these correlations may be driven by group differences in symptom measures.

Exploratory analyses tested whether posterior alpha power asymmetry scores related to suicidal thoughts and behaviors. Across all participants posterior alpha power asymmetry scores were associated with suicidal thoughts (overall model: $\chi^2(1, n = 53) = 6.04$, $p = 0.01$), $B = -4.62$, $SE = 1.58$, $\chi^2(1, n = 53) = 8.59$, $p < 0.01$. This effect remained when controlling for depressive symptoms (overall model: $\chi^2(2, n = 53) = 21.03$, $p < 0.01$), with both increased alpha asymmetry (i.e., greater alpha power over the right than the left hemisphere), $B = -4.27$, $SE = 1.99$, $\chi^2(1, n = 53) = 4.60$, $p = 0.03$, and depression symptom severity, $B = 0.10$, $SE = 0.04$, $\chi^2(1, n = 53) = 7.87$, $p = 0.01$, related to more suicidal thoughts. The relation between resting posterior alpha asymmetry and suicidal thoughts trended toward significance when the analysis was restricted to adolescents with MDD (overall model: $\chi^2(1, n = 28) = 3.46$, $p = 0.06$; Fig. 2D). By contrast, posterior alpha power asymmetry scores were not related to suicidal behaviors, $\chi^2(1, n = 53) = 0.99$, $p = 0.32$), and overall posterior alpha was neither related to suicidal thoughts nor behaviors (overall model: $\chi^2(1, n = 53) = 0.10$, $p = 0.76$) nor behaviors (overall model: $\chi^2(1, n = 53) = 2.10$, $p = 0.15$).

4. Discussion

Resting alpha neural oscillations have been extensively studied as a neurobiological marker of MDD (Bruder et al., 2013; Coan and Allen, 2004; Davidson et al., 1985; Fingelkurts and Fingelkurts, 2015). A major focus of this research has described the role of frontal alpha asymmetry, but prior evidence also implicates resting posterior alpha. Consistent with this latter body of work, we found that female adolescents with

[1] This effect also was found when EO and EC conditions were averaged (see Supplement).

Table 2
Spearman's rank-order correlations among symptoms, and electrophysiological components, and internal consistency for self-reports and split-half reliability for posterior alpha.

	1.	2.	3.	4.	5.	6.	7.	α/SB
1. Depression Symptoms	–							.97
2. Anhedonia Symptoms	.79***	–						.94
3. Anxious Symptoms	.71***	.52***	–					.94
4. Rumination	.80***	.63**	.71***	–				.95
5. Self-Criticism	.68***	.56***	.64***	.73***	–			.86
6. Dependency	.58***	.39***	.68***	.62***	.45***	–		.82
7. Posterior Alpha Power	-.23*	-.20	-.17	-.10	-.03	-.22*	–	.98
8. Posterior Alpha Asymmetry Scores	-.31**	-.32**	-.16	-.28**	-.25**	-.22*	-.06	.98

Note. ***$p < 0.01$; **$p < 0.05$; *$p < 0.10$.
Beck Depression Inventory II = Depression Symptoms; Snaith-Hamilton Pleasure Scale = Anhedonia Symptoms; Multidimensional Anxiety Scale for Children = Anxious Symptoms; Children's Response Styles Questionnaire = Rumination; Children's Depressive Experiences Questionnaire = Self-Criticism and Dependency. α = Cronbach's alpha for 1–6. SB = Spearman-Brown Coefficient for split-half reliability for 7–8.

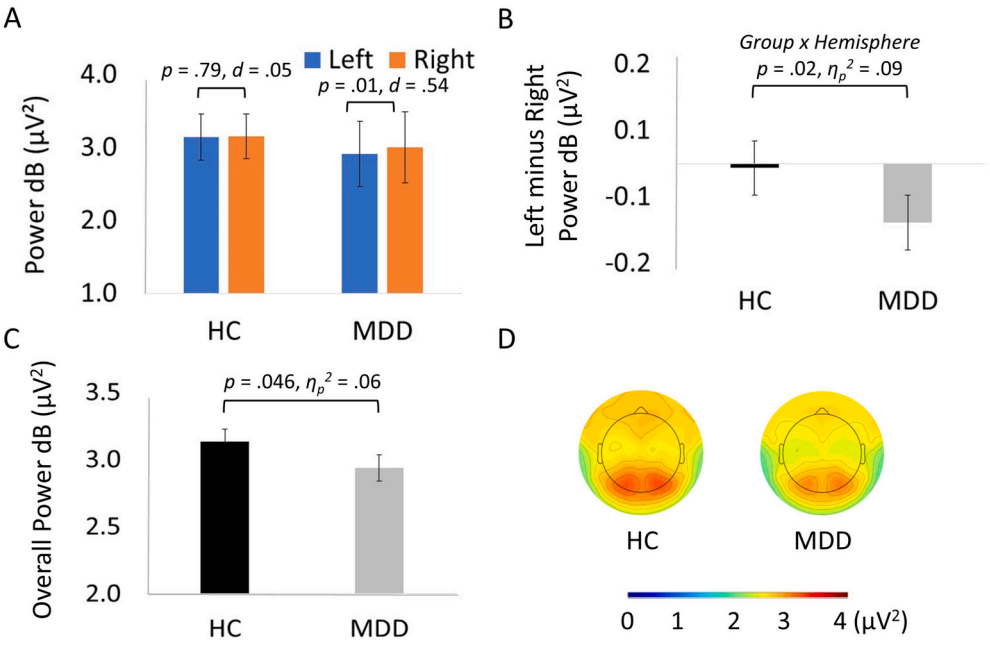

Fig. 1. Comparison of Alpha among Healthy and Depressed Adolescents. Results of the Fast Fourier Transform (FFT) focusing on the posterior alpha power averaged across the 8–13 Hz frequency between healthy controls (HC) and major depressive disorder (MDD) group. (**A**) Posterior alpha power separately for the right and left hemisphere; (**B**) Posterior alpha power asymmetry (left minus right posterior alpha); (**C**) Overall posterior alpha power separately for the two groups. Error bars indicate standard error of mean. (**D**) Topographical maps depicting the posterior alpha power between groups. Note that alpha power in (A), (B), and (C) are derived from different sets of electrodes (see **Data Reduction and Analysis** section).

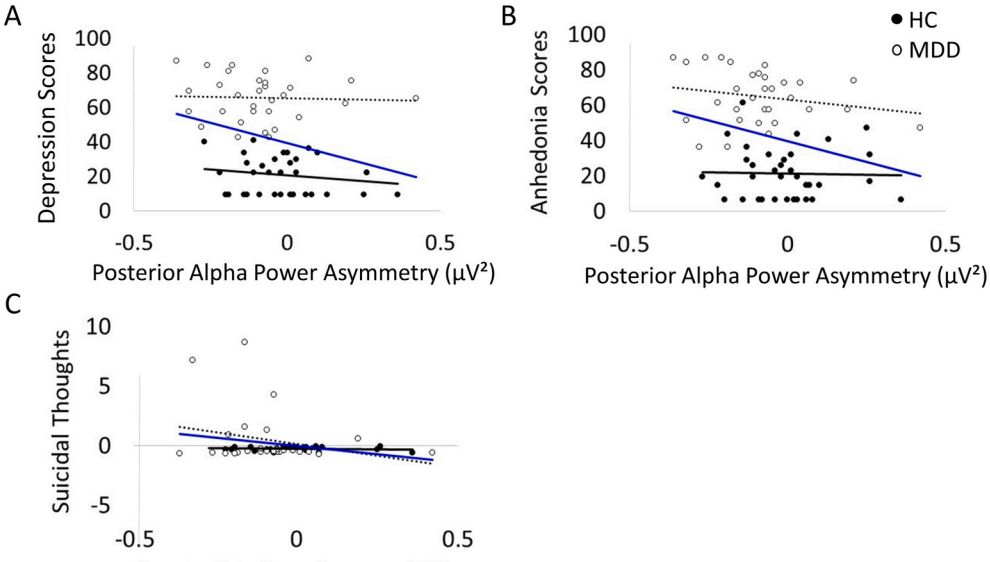

Fig. 2. Correlations between Posterior Alpha Power Asymmetry and Clinical Variables. Nonparametric Spearman rank-order correlations across healthy control (HC) and adolescents with major depressive disorder (MDD) between Posterior Alpha Power Asymmetry scores and (**A**) Depression Scores (Beck Depression Inventory II), (**B**) Anhedonia Scores (Snaith-Hamilton Pleasure Scale), and (**C**) Suicidal Thoughts. Suicidal thoughts are the standardized residual scores controlling for depression scores. Goodness-of-Fit lines are provided for HC (black line), MDD (dotted black line), and All (blue line) adolescents. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

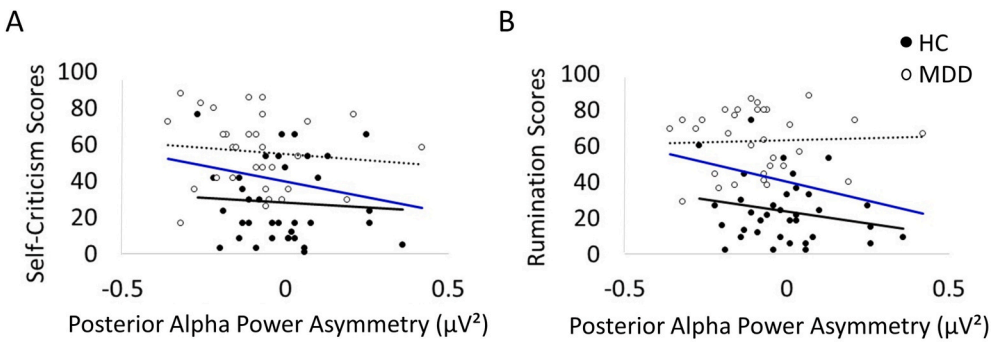

Fig. 3. Spearman's Rank-Order Correlations between Posterior Alpha Asymmetry and Perseverative Traits. Spearman's rank-order correlations across healthy control (HC) and adolescents with major depressive disorder (MDD) between Posterior Alpha Power Asymmetry scores and (**A**) Self-Criticism Scores (Children's Depressive Experiences Questionnaire), and (**B**) Rumination Scores. Goodness-of-Fit lines are provided for HC (black line), MDD (dotted black line), and All (blue line) adolescents. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

MDD exhibited greater posterior alpha power over the right relative to the left hemisphere whereas healthy female adolescents displayed no hemispheric asymmetry. This is consistent with past findings in adults (Bruder et al., 1997; Heller et al., 1997; Henriques and Davidson, 1990; Kemp et al., 2010) and female adolescents with MDD (Kentgen et al., 2000), suggesting that this abnormality emerges early in the course of MDD. Interestingly, resting posterior alpha asymmetry also was associated with core symptoms (e.g., depression, anhedonia) and traits (rumination, self-criticism), but no associations emerged with overall posterior alpha.

Our findings suggest there may be developmental differences rega rding the posterior alpha-MDD relationship. Similar to adults with MDD, depressed female adolescents were characterized by increased posterior alpha asymmetry (Blackhart et al., 2006; Bruder et al., 1997; Kentgen et al., 2000). However, unlike prior studies with adults, overall posterior alpha power was reduced in depressed adolescents relative to healthy controls (Jiang et al., 2016; Volf and Passynkova, 2002; Zoon et al., 2013). One possibility for these differences may be that the observed alpha oscillations are more variable during key periods of neurodevelopment, and accordingly, may be impacted by a variety of developmental factors (e.g., puberty) (Segalowitz et al., 2010). Our sample precludes the ability to test sex differences. Prior research, however, suggests that posterior alpha asymmetry found in depressed women may be reduced or show an opposing pattern in depressed men (Stewart et al., 2011). Thus, it will be important to explore sex differences in future research with larger samples.

Although alpha has long been implicated in MDD (Bruder et al, 2005, 2012), an unanswered question is whether alpha alterations underlie MDD specifically, or rather, contribute to the emergence of core symptoms and traits that may lead to MDD. Our cross-sectional study shows preliminary evidence that resting posterior alpha asymmetry was related to anhedonia, rumination, and self-criticism, perhaps suggesting that alpha impacts core psychological processes that compromises well-being. Though the current study design precludes any conclusions about whether posterior alpha alterations are a cause or consequence of depression, alpha disturbances likely impact specific symptoms and cognitive processes that may confer increased risk for a wide range of mental disorders (Bruder et al., 1997; Heller et al., 1997; Kemp et al., 2010; Metzger et al., 2004). Nevertheless, results should be interpreted with caution, as correlations were only observed across, but not within, groups.

4.1. Posterior alpha and suicide

Exploratory analyses tested whether resting posterior alpha was associated with suicidal thoughts and behaviors given prior work showing an alpha-suicide link (Benschop et al., 2019; Graae et al., 1996). Across both groups, increased resting posterior asymmetry (but not overall posterior alpha) was associated with increased suicidal thoughts, but not suicidal behaviors, above and beyond depression symptom severity. That said, these results require further replication, as the alpha-suicidal thinking relationship only trended toward significance when analyses were conducted separately among the depressed adolescents.

4.2. Limitations

There are several limitations. First, the sample size was modest, predominantly Caucasian, and female-only, which limit the generalizability. Second, the study is cross-sectional, and consequently, we cannot determine whether posterior alpha effects were a cause or consequence of MDD. Third, we did not record what time of day EEG data were collected for each participant. Thus, we cannot account for the potential confound related to vigilance effects. Last, our assessment of suicidal thoughts and behaviors probed lifetime occurrence, and thus, it is plausible that the suicidal thoughts and behaviors occurred prior to the emergence of alpha alterations.

5. Conclusion

Posterior alpha is a promising neurophysiological indicator of MDD among adolescents. The study found increased posterior alpha power asymmetry and reduced overall posterior alpha for female adolescents with MDD, and posterior alpha asymmetry was associated with core symptoms and traits. Although our correlational design precludes any conclusions about the cause-effect relation, one possibility is that alpha alterations may exacerbate core traits (e.g., rumination), which then lead to the emergence of depressive symptoms. Further research is needed to parse the time course related to the sequela of alpha perturbations, traits, and associated symptom manifestation (Tenke et al., 2017b). If posterior alpha proves to a be transdiagnostic risk factor—reflecting symptoms (anhedonia) and core traits (repetitive thinking style)—it would be important to know whether existing treatments modulate posterior alpha and attenuate associated clinical targets.

Contributors

Akina Umemoto: Conceptualization, data analysis/processing, writing; **Lidia Panier**: Data analysis/processing; **Sally Cole**: Data processing, literature search; **Jurgen Kayser**: Data analysis/processing, writing;Diego A. Pizzagalli: Conceptualization, writing; **Randy P. Auerbach**: Conceptualization, data analysis/processing, writing.

Data sharing

The data that support the findings of this study are available from the corresponding author upon request.

Declaration of competing interest

Dr. Auerbach is an unpaid scientific advisor for Ksana Health. Over

the past 3 years, Dr. Pizzagalli has received consulting fees from Albright Stonebridge Group, BlackThorn Therapeutics, Boehringer Ingelheim, Compass Pathway, Concert Pharmaceuticals, Engrail Therapeutics, Neurocrine Biosciences, Otsuka Pharmaceuticals, and Takeda Pharmaceuticals; one honorarium from Alkermes, and research funding from NIMH, Dana Foundation, Brain and Behavior Research Foundation, and Millennium Pharmaceuticals. In addition, he has received stock options from BlackThorn Therapeutics. No funding from these entities was used to support the current work, and all views expressed are solely those of the authors. The other authors have no financial disclosures.

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Appendix A. Supplementary data

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