BRIEF REPORT

Cognitive Vulnerability and Frontal Brain Asymmetry: Common Predictors of First Prospective Depressive Episode

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The hopelessness theory of depression proposes that individuals with a depressogenic cognitive style are more likely to become hopeless and experience depression following negative life events. Although the neurophysiological underpinnings of cognitive style remain speculative, research indicates that decreased relative left frontal brain electrical activity holds promise as a traitlike marker of depression. This begs the question: Do measures of depressogenic cognitive style and resting frontal brain asymmetry index a common vulnerability? The present study provides preliminary support for this hypothesis. At baseline assessment, increased cognitive vulnerability to depression was associated with decreased relative left frontal brain activity at rest in individuals with no prior history of, or current, depression. Following baseline assessment, participants were followed prospectively an average of 3 years with structured diagnostic interviews at 4-month intervals. Both cognitive vulnerability and asymmetric frontal cortical activity prospectively predicted onset of first depressive episode in separate univariate analyses. Furthermore, multivariate analyses indicated that cognitive vulnerability and frontal asymmetry represented shared, rather than independent, predictors of first depression onset.

Keywords: cognitive vulnerability, frontal EEG, depression

Supplemental materials: http://dx.doi.org/10.1037/a0022940.supp

What leads some individuals to be vulnerable to depression? Over the past 30 years, investigators have addressed this question from both cognitive and neurophysiological perspectives. To date, however, these two lines of research have proceeded independently. The current study begins to bridge researchers' knowledge of cognitive and neurophysiological vulnerabilities, with the objective of generating a more integrative biocognitive understanding of depression risk.

Cognitive vulnerability models of depression posit that individuals with maladaptive cognitive styles are more vulnerable to depression when they encounter negative events. According to the hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989), individuals who make stable, global attributions, infer negative self-characteristics, and anticipate negative consequences when negative events occur are more likely to develop depression than individuals who do not exhibit this negative cognitive style. Prospective and retrospective tests of the hopelessness theory indicate that individuals with negative cognitive styles are more vulnerable to depression (Abramson et al., 2002).

Asymmetries in resting electroencephalographic (EEG) activity recorded over the frontal cortex may suggest important clues about how cognitive vulnerability to depression is instantiated neurally. The approach–withdrawal motivational model of frontal brain asymmetry posits that increased relative left frontal activity indicates a propensity to approach or engage a stimulus, whereas decreased relative left frontal activity indicates a propensity toward reduced approach-related motivation or increased withdrawal motivation (Coan & Allen, 2004). Consistent with this view, increased relative left frontal activity has been associated with

heightened self-reported approach system sensitivity (Harmon-Jones & Allen, 1997), anger (Harmon-Jones, 2003), and positive activation (Coan & Allen, 2004). Furthermore, stimuli intended to elicit approach-oriented responses (e.g., reward cues, anger-evoking stimuli) are associated with increased relative left frontal activity (Coan & Allen, 2004).

By contrast, individuals with depression show decreased relative left frontal activity at rest during both depressive and euthymic states (Thibodeau, Jorgensen, & Kim, 2006). These data have been interpreted in the context of a vulnerability–stress framework in which resting frontal asymmetry reflects a state-independent risk factor for depression (Coan & Allen, 2004). In line with this view, abnormal regional hemispheric asymmetries have been observed in offspring of depressed individuals who have yet to experience a depressive episode themselves (Bruder et al., 2005; Dawson, Frey, Panagiotides, Osterling, & Hessl, 1997).

Abramson et al. (2002) proposed a conceptual integration of the hopelessness theory and approach–withdrawal model of depression, arguing that "hopelessness, the expectation to which cognitively vulnerable individuals are predisposed, may represent the cognitive, affective, and behavioral manifestations of an inactive approach system" (p. 287). From this perspective, both heightened cognitive vulnerability and decreased relative left frontal activity serve as shared or common risk factors for experiencing an excessive decrease in approach-related affect or behavior (i.e., depression) following negative events. From the cognitive perspective, when vulnerable individuals encounter stressful events, they generate negative inferences about their future and self-worth. These inferences lead to hopelessness about achieving current and future goals, which leads to a disengagement from approach-oriented action and symptoms of depression. Consistent with this view, cognitive vulnerability interacts with life stress to predict a reduction in goal-directed behavior and a concomitant increase in withdrawal or depressive symptoms (Haeffel, Abramson, Brazy, & Shah, 2008). Cognitively vulnerable individuals are also more likely to disengage from approach-oriented behaviors during laboratory stressors (Alloy, Peterson, Abramson, & Seligman, 1984).

From the approach–withdrawal perspective, decreased relative left frontal activity at rest reflects a propensity to experience an excessive decrease in approach-related affect (i.e., depression) or increase in withdrawal-related affect following negative events (Coan & Allen, 2004). Thus, both the hopelessness and approach–withdrawal models outline a framework in which vulnerable individuals are prone to an excessive decrease in appetitive motivation, which is reflected in depressive symptoms. Given the conceptual overlap between these two models, an important and untested hypothesis is that there is a meaningful relationship between cognitive vulnerability, as defined by hopelessness theory (Abramson et al., 1989), and frontal asymmetry. The present study provided the first test of this hypothesis.

First, we examined the relationship between resting frontal asymmetry and individual differences in cognitive vulnerability to depression at baseline assessment in individuals with no history of depression. Participants with no prior depression were sampled to examine the relationship between frontal asymmetry and cognitive vulnerability among individuals whose neural and cognitive profiles were unaffected by previous depressive episodes (as in the scar hypothesis; Lewinsohn, Steinmetz, Larson, & Franklin, 1981).

We predicted that increased cognitive vulnerability to depression would correlate with decreased relative left frontal activity at rest.

Second, we examined whether cognitive vulnerability and resting frontal asymmetry prospectively predicted onset of first depressive episode over a 3-year follow-up period. At the univariate level, we predicted that both heightened cognitive vulnerability and decreased relative left frontal activity at rest would prospectively predict a greater likelihood of a first depressive episode. Multivariate modeling was then employed to test the hypothesis proposed by Abramson et al. (2002) that cognitive vulnerability and frontal asymmetry represent common, as opposed to independent, predictors of depression onset.

Given Abramson et al.'s (2002) proposed conceptual integration of the approach–withdrawal and hopelessness models, hypothesis testing focused on the cognitive vulnerability outlined in hopelessness theory (Abramson et al., 1989) and indexed by the Cognitive Style Questionnaire negative events composite (CSQ-N; Alloy et al., 2000). The CSQ-N assesses an individual's tendency to make negative inferences for cause, consequence, and self, in response to negative events, as specified in the hopelessness theory. However, to assess the specificity between frontal asymmetry and the cognitive vulnerability outlined in hopelessness theory, exploratory analyses were conducted examining relationships between frontal asymmetry and other relevant indices of cognitive style and temperament. Of particular interest is whether frontal asymmetry will be specifically related to CSQ-N scores with respect to the prospective onset of first depressive episode, or whether it will also be related to inferential style for positive life events, as indexed by the CSQ positive events composite (CSQ-P; Alloy et al., 2000). Given that inferential style for positive events has been associated more with time to recover from a depressive episode (Needles & Abramson, 1990), as opposed to depression onset, we predicted this relationship would be specific to CSQ-N scores. Moreover, the CSQ-P assesses postgoal responses and not the pregoal, appetitive responsiveness tapped by asymmetric frontal cortical activity (Harmon-Jones, Harmon-Jones, Fearn, Sigelman, & Johnson, 2008); thus, it should not be related to asymmetric frontal cortical activity.

Method

Participants

Participants were a subgroup of healthy control participants in the Longitudinal Investigation of Bipolar Spectrum (LIBS) Project. At recruitment into the LIBS Project, healthy control participants were University of Wisconsin students ($M_{age} = 20.32$ years, $SD = 1.25$) and required to have no history of affective psychopathology, as indexed by both the General Behavior Inventory (GBI; Depue et al., 1981; GBI–Hypomania-Biphasic subscale score < 13 and GBI–Depression subscale score < 11) and a Schedule for Affective Disorders and Schizophrenia–Lifetime (SADS-L; Endicott & Spitzer, 1978) interview.

Of the 110 healthy control participants recruited at the Wisconsin site, 56 completed baseline EEG recording and cognitive measures for the present study (baseline data collection for the present study occurred an average 14 months following initial recruitment into the LIBS Project). Participants were further excluded if they (a) were not right-handed (>32; Chapman & Chap-

man, 1987), (b) met Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978) for a major or minor depressive episode during the 14-month period from initial recruitment into the LIBS Project to baseline EEG or cognitive data collection for the present study, (c) had unusable data in frontal EEG electrodes, or (d) failed to provide at least 1 year of diagnostic data during the follow-up period. Together these criteria yielded 40 (17 female) participants who provided data for the present study. None were taking psychotropic medications or had a comorbid anxiety or alcohol or substance use disorder at baseline assessment. There were no differences in age, gender, CSQ-N, CSQ-P, or Behavioral Inhibition System/Behavioral Activation System (BIS/BAS; Carver & White, 1994) scale scores between healthy control LIBS participants who did and did not complete data collection for the present study ($ps > .24$). Informed written consent was obtained at the EEG session.

Procedure

At baseline, participants completed EEG recordings, the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979), the CSQ (Alloy et al., 2000), and other relevant measures of cognitive style and temperament. Participants then were followed prospectively for an average of 3 years with diagnostic interview assessments every 4 months via the Schedule for Affective Disorders and Schizophrenia–Change (SADS-C; Spitzer & Endicott, 1978) interview.

Measures

CSQ (Alloy et al., 2000). The CSQ consists of 12 hypothetical negative and 12 hypothetical positive events. Respondents write down one cause for each event and rate the degree to which the cause of the event is stable and global. In addition, they rate the likelihood that further negative consequences will result from the occurrence of the negative event (e.g., "How likely is it that the other person no longer wanting a romantic relationship with you will lead to other negative things happening to you?"), or positive consequences from the occurrence of the positive event, and the implication of the event for their self-worth (e.g., "To what degree does your receiving a negative evaluation of your job performance mean to you that you are flawed in some way?"). We computed composite scores for negative events (CSQ-N) and positive events (CSQ-P) based on a sum of stability, globality, consequences, and self dimensions for each event type. Higher CSQ-N and CSQ-P scores indicate more negative and positive cognitive styles, respectively. Mean and Cronbach's alpha in the present study were 3.7 ($SD = 0.70$) and .87 for CSQ-N and 5.2 ($SD = 0.70$) and .93 for CSQ-P, respectively. As expected, CSQ-P scores were higher than CSQ-N scores in the present study, $t(38) = 9.65$, $p < .001$.

GBI (Depue et al., 1981). The GBI contains 73 items assessing affective experiences and their intensity, duration, and frequency and is composed of Depression and Hypomania-Biphasic scores. Mean and Cronbach's alpha at initial recruitment were 1.49 ($SD = 2.06$) and .90 for the Depression subscale and 2.49 ($SD = 2.92$) and .92 for the Hypomania-Biphasic subscale, respectively.

BDI (Beck et al., 1979). The BDI is a 21-item self-report inventory used to assess initial levels of depressive symptoms. Mean and Cronbach's alpha in the present study were 2.51 ($SD = 2.40$) and .81, respectively.

SADS-L (Endicott & Spitzer, 1978). The SADS-L is a semistructured diagnostic interview that assesses current and lifetime history of Axis I diagnoses. Interviewers were blind to GBI scores. For both SADS-L and SADS-C, consensus *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) and RDC diagnoses were determined by a three-tiered standardized review procedure involving project interviewers, senior diagnosticians, and an expert psychiatric diagnostic consultant. An interrater reliability study based on 57 jointly rated SADS-L interviews on healthy control participants from the LIBS Project yielded kappas greater than .96 for both *DSM–IV* major and RDC major and minor depressive episodes.

SADS-C (Spitzer & Endicott, 1978). The SADS-C was administered at 4-month intervals during prospective follow-up to assess onset of Axis I psychopathology. The present study used RDC diagnoses to assess both major and minor depressive episodes. Major depressive episodes were defined by (a) depressed mood or loss of interest greater than or equal to 90% of waking hours, (b) four or more additional major depressive symptoms, and (c) significant distress or impairment. A definite major depressive episode met these criteria for at least 2 weeks and a probable major depressive episode for at least 1 week. Minor depressive episodes were defined by (a) depressed mood or loss of interest greater than or equal to 50% of waking hours, (b) two or more additional depressive symptoms, and (c) significant distress or impairment for at least 1 week. In the present study, both major and minor episodes were considered evidence of first-time depression. An interrater reliability study based on 52 jointly rated SADS-C interviews on healthy control participants from the LIBS Project yielded kappas greater than .92 for both *DSM–IV* major and RDC major and minor depressive episodes.

Exploratory Measures

For brevity, we provide an abbreviated description of the cognitive/temperamental measures used in exploratory analyses. See supplemental materials for detailed description.

Sociotropy–Autonomy Scale (Beck, Epstein, Harrison, & Emery, 1983). The Sociotropy–Autonomy Scale includes an Autonomy scale measuring valuing achievement, mobility, and freedom and a Sociotropy scale measuring valuing of attachment and fears of abandonment.

Dysfunctional Attitude Scale (Weissman & Beck, 1978). The Dysfunctional Attitude Scale assesses dysfunctional beliefs regarding concerns about others' approval and performance expectations.

Depressive Experiences Questionnaire (Blatt, D'Afflitti, & Quinlan, 1976). The Depressive Experiences Questionnaire measures the three depressive personality styles: Dependency, Self-Criticism, and Efficacy. We used the Dependency and Self-Criticism subscales.

Response Style Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991). The RSQ consists of Rumination and Distraction scales, each designed to assess characteristic styles of responding to or regulating dysphoria.

BIS/BAS (Carver & White, 1994). The BIS/BAS scale assesses self-reported sensitivity of the BAS and the BIS.

EEG recording and reduction. Eight 60-s eyes-open/eyes-closed trials were collected in a counterbalanced sequence with 16 electrodes (A1/A2, F3/F4, F7/F8, C3/C4, T3/T4, T5/T6, P3/P4, Cz, Pz) grounded at Fz. The online reference was the left earlobe (A1), and data were recorded from the right earlobe (A2), enabling computation of an offline averaged-ears reference (impedances < 5kΩ; homologs ± 1kΩ). Data were filtered (0.1–100 Hz; 60 Hz notch-filter enabled), amplified, and digitized (500 Hz).

The EEG and electrooculogram signals were visually scored, and portions of data containing aberrant eye, muscle movements, or other sources of artifact were removed (data from all channels were removed at that point). Vertical electrooculogram was then used in a regression-based artifact correction of the EEG (Semlitsch, Anderer, Schuster, & Presslich, 1986; another visual inspection ensured that no aberrations remained). We used only the regression-based artifact correction for removing clearly defined eyeblinks. All nonblink horizontal and vertical eye movement, as well as data containing aberrant muscle movement, was manually removed based on visual inspection of the data. Derived averaged-ears reference data were used for further data reduction. Artifact-free epochs (1.024 s) were Hamming windowed (75% overlap), and power spectral density (μV^2/Hz) was computed for the alpha band (8–13 Hz) across eyes-open/eyes-closed for each channel. Power densities were log-transformed and asymmetry indices [ln(right)–ln(left) alpha power] computed. Because alpha power is inversely related to cortical activity (Coan & Allen, 2004), higher scores indicate greater relative left-hemisphere activity. Hypothesis testing focused on frontal sites, consistent with prior research (Thibodeau et al., 2006).

We averaged alpha power in F3/F7 into a "left frontal region" and F4/F8 into a "right frontal region" and computed a composite frontal asymmetry index as follows: [ln(mean of F4/F8)–ln(mean of F3/F7)]. We used a composite frontal asymmetry index (a) because we did not have separate hypotheses for mid- (F3/F4) and lateral-frontal (F7/F8) electrodes, (b) because of the high correlation between mid- and lateral-frontal regions (rs > .97), and (c) to minimize Type I error by reducing number of statistical analyses. Across electrode pairs, mean Cronbach's alpha for eight 1-min recordings was .93.

Results

Relations Between Cognitive Vulnerability and Frontal Asymmetry

In line with prediction, individuals with greater CSQ-N scores (i.e., greater cognitive vulnerability) had decreased relative left frontal EEG activity at baseline, $r(38) = -.41$, $p = .01$ (see Table 1 and Figure 1A).[1] This relationship was maintained after controlling for baseline BDI scores, $r(37) = -.42$, $p = .01$ (see table in supplemental materials). As predicted, the relationship between CSQ-N scores and hemispheric asymmetry was specific to the composite frontal region (for nonfrontal sites, ps > .26; see Figure 1B).

Cognitive Vulnerability, Frontal Asymmetry, and First Depressive Episode Onset

All prospective analyses involving onset of first depressive episode controlled for BDI scores at baseline assessment. Thirteen of the 40 participants developed a first-ever depressive episode over the 3-year follow-up (three participants had an RDC major depressive episode, and 10 had an RDC minor depressive episode). Consistent with prediction, logistic regression indicated that decreased relative left frontal activity at baseline was associated with a greater probability of a first prospective depressive episode during the follow-up period ($p = .02$; see Table 2).[2] Furthermore, a separate logistic regression analysis indicated that increased CSQ-N scores were associated with a greater probability of having a first depressive episode during the follow-up period ($p = .04$; see Table 2).

To examine whether frontal asymmetry and cognitive vulnerability represented common or independent predictors of first depressive episode, we conducted a third logistic regression with both frontal asymmetry and CSQ-N scores entered simultaneously as predictors. In line with prediction, the omnibus model was significant ($p = .02$; see Table 2), indicating that frontal EEG and CSQ-N scores collectively predicted first depressive episode. However, neither frontal asymmetry ($p = .21$) nor CSQ-N scores ($p = .14$) remained independent predictors of first depressive episode. Furthermore, the interaction between frontal asymmetry and CSQ-N scores in predicting depression onset was nonsignificant ($p = .10$). Collectively, this suggests that resting frontal asymmetry and cognitive vulnerability may represent common, rather than independent, predictors of depression onset.[3]

Exploratory Analyses of Relationships Between Frontal Asymmetry and Other Indices of Cognitive Style and Temperament

No relation was observed between frontal asymmetry and inferential style for positive events, as indexed by the CSQ-P ($p = .94$; see Table 1). Moreover, the correlation between frontal asymmetry and CSQ-N scores was significantly different from the correlation between frontal asymmetry and CSQ-P scores ($Z = 1.96$, $p < .05$), suggesting that the relationship between cognitive vulnerability and frontal asymmetry was specific to people's inferences for negative life events. Individuals with greater RSQ–Rumination scores had decreased relative left frontal activity ($p = .02$) and increased CSQ-N scores ($p = .05$). However, there was no relationship between RSQ–Rumination scores and first depressive episode onset. All exploratory analyses were replicated after controlling for BDI scores at baseline assessment (see table in supplemental

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[1] Comparable effects were observed for the relationship between CSQ-N scores and frontal asymmetry separately at both the mid-, $r(38) = -.38$, $p = .02$, and lateral-frontal region, $r(38) = -.36$, $p = .02$.

[2] Relations between hemispheric asymmetry and first depressive episode onset was specific to the frontal region (for nonfrontal sites, ps > .38).

[3] We conducted separate logistic regression analyses of cognitive vulnerability, frontal asymmetry, and first depressive episode onset at both the mid- and lateral-frontal region. Results for these analyses at the midfrontal region were equivalent to the composite frontal asymmetry index. By contrast, there was no significant relationship between lateral-frontal asymmetry and first depressive episode ($B = -2.59$, $\chi^2 = 1.42$, $p = .23$).

Table 1
Correlations Among Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. FEA	—															
2. CSQ-N	−.41**	—														
3. CSQ-P	.02	.05	—													
4. DEP[a]	−.39*	.35*	−.03	—												
5. DEQ-DEP	−.04	.21	.21	.30	—											
6. DEP-SC	−.05	.31*	.02	.04	−.30	—										
7. RSQ-R	−.36*	.32*	−.06	.23	.29	.20	—									
8. RSQ-D	−.09	.04	.24	−.15	−.04	−.24	−.18	—								
9. DAS-T	−.14	.37*	.16	.31	.37*	.47**	.26	−.31	—							
10. DAS-PE	−.17	.34*	.15	.08	−.12	.55**	.05	−.14	.79**	—						
11. DAS-AO	−.10	.27	.11	.35*	.38*	.15	.24	.39*	.72**	.25	—					
12. SAS-SOC	−.16	.29	−.03	.21	.44**	.30	.34*	−.18	.68**	.42**	.51**	—				
13. SAS-AUT	−.19	−.07	.12	−.15	−.51**	.20	.03	.36*	−.22	.04	−.38*	−.20	—			
14. BAS	.10	.11	.32*	.11	.01	−.09	−.14	.28	−.22	−.14	−.27	−.27	−.29	—		
15. BIS	−.21	.29	.13	.15	.43**	−.02	.23	.03	.27	.13	.25	.38*	−.14	.13	—	
16. BDI	.08	.13	.12	.05	.19	.33*	.23	.08	.15	.23	−.14	.20	.20	−.03	−.02	—

Note. FEA = composite frontal electroencephalographic asymmetry [(mean of F4/F8)–ln(mean of F3/F7)]; CSQ-N = negative composite of the Cognitive Style Questionnaire; CSQ-P = Cognitive Style Questionnaire positive composite; DEP = prospective depressive episode; DEQ-DEP = Dependency subscale from the Depressive Experiences Questionnaire; DEQ-SC = Self-Criticism subscale from the Depressive Experiences Questionnaire; RSQ-R = Rumination subscale from the Response Style Questionnaire; RSQ-D = Distraction subscale from the Response Style Questionnaire; DAS-T = Total scale from the Dysfunctional Attitude Scale; DAS-PE = Performance Evaluation subscale from Dysfunctional Attitude Scale; DAS-AO = Approval by Others subscale from the Dysfunctional Attitude Scale; SAS-SOC = Sociotropy subscale from the Sociotropy–Autonomy Scale; SOC-AUT = Autonomy subscale from the Sociotropy–Autonomy Scale; BAS = Total scale from the Behavioral Approach System Sensitivity scale; BIS = Total scale from the Behavioral Inhibition System Sensitivity scale; BDI = Beck Depression Inventory.
[a] 1 = yes, 0 = no.
* $p < .05$. ** $p < .01$.

materials). As in past research, there was a positive relationship between frontal asymmetry and BAS–Total scores. However, in the present sample, this relationship was not significant.

Discussion

Consistent with prediction, decreased relative left frontal activity at rest was associated with increased cognitive vulnerability to depression (i.e., heightened CSQ-N scores) at baseline assessment among euthymic individuals with no prior history of depression, ruling out the possibility that this relation is a by-product of previous depressive episodes (i.e., scar hypothesis; Lewinsohn et al., 1981). The relationship between cognitive vulnerability and frontal asymmetry was specific to people's inferences for negative life events, as no relation was observed between frontal asymmetry and inferential style for positive events (CSQ-P). Also consistent
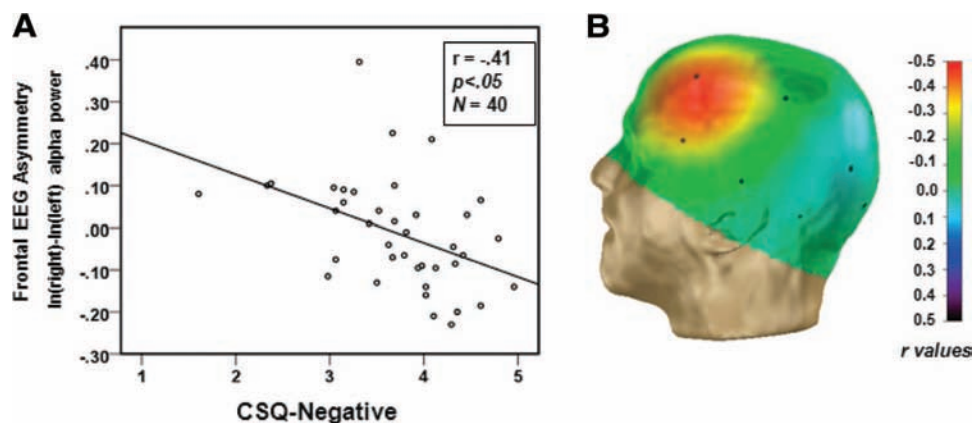

Figure 1. (A) Scatterplot of the correlation between resting frontal asymmetry [ln(mean of F4/F8)–ln(mean of F3/F7)] and Cognitive Style Questionnaire negative events composite (CSQ-N) scores. Lower electroencephalographic (EEG) asymmetry scores reflect reduced relative left frontal activity. Higher CSQ-N scores reflect greater cognitive vulnerability for depression. (B) Topographic map of the distribution of correlations between frontal asymmetry and CSQ-N scores. As predicted, the relation between frontal asymmetry and CSQ-N scores is specific to the frontal region (values for nonfrontal indices, $r > .26$).

Table 2
Logistic Regression Analyses Predicting Onset of First Depressive Episode

Predictor	B	SE	χ^2	p
Analysis 1: Frontal asymmetry predicting onset of first depressive episode				
BDI	0.13	0.17	0.56	.45
FEA	−8.40	3.6	5.34	.02
Analysis 2: Cognitive vulnerability predicting onset of first depressive episode				
BDI	0.041	0.16	0.07	.79
CSQ-N	1.46	0.70	4.38	.04
Analysis 3: Cognitive vulnerability and frontal asymmetry predicting onset of first depressive episode				
Omnibus			12.06	.02
BDI	0.09	0.18	0.23	.63
CSQ-N	1.16	0.79	2.17	.14
FEA	−5.11	4.06	1.58	.21
CSQ-N × FEA	19.08	11.36	2.82	.10

Note. BDI = Beck Depression Inventory; FEA = composite frontal electroencephalographic asymmetry score [ln(mean of F4/F8)–ln(mean of F3/F7)]; CSQ-N = negative composite of the Cognitive Style Questionnaire.

with prediction, both decreased left frontal activity and heightened CSQ-N scores, in separate regression analyses, prospectively predicted a greater likelihood of having a first-ever depressive episode during the follow-up period. This is the first study demonstrating that decreased relative left frontal activity serves as a risk factor for the prospective onset of first depressive episode. Finally, we found evidence that resting frontal asymmetry and cognitive vulnerability serve as common, as opposed to independent, predictors of first depressive episode. When both frontal EEG and CSQ-N scores were simultaneously entered into a regression model, the overall model predicted depression onset; however, neither frontal asymmetry nor CSQ-N scores remained significant as independent predictors.

An overarching objective of this study was to facilitate more dialogue between cognitive and biological models of psychopathology. Abramson et al. (2002) conceptually initiated this integration by speculating that cognitive styles that predispose an individual to hopelessness, as specified in the hopelessness theory (Abramson et al., 1989), should be related to biological vulnerabilities associated with deficits in approach system activity. The present study provides support for this perspective, suggesting that increased cognitive vulnerability and frontal asymmetry may reflect common predictors of depression onset. Exploratory analyses indicating that heightened rumination scores were associated with both decreased relative left frontal activity and increased CSQ-N scores are also in line with a conceptual integration of the hopelessness theory and approach–withdrawal model. As noted by Abramson et al. (2002), high levels of rumination or perseverative attention may cause a person to have difficulty disengaging from negative events, resulting in a decrease in approach-related motivation and a concomitant increase in symptoms of hopelessness and depression.

Three limitations of the current study represent challenges for future research. First, both the hopelessness (Abramson et al., 1989) and approach–withdrawal models (Coan & Allen, 2004) take a vulnerability–stress framework in which vulnerable individuals are at higher risk for depression following negative events. Future studies should examine the role of life events in the rela-

tionship between cognitive vulnerability, frontal asymmetry, and depression onset.

Second, the cognitive vulnerability specified in the hopelessness theory puts an individual at risk for a particular subtype of depression, referred to as hopelessness depression (Abramson et al., 1989). Symptoms of hopelessness depression include sadness, decreased initiation of responses, low energy, apathy, and psychomotor retardation, all of which reflect decreased approach motivation (Abramson et al., 2002; Haeffel et al., 2008). Because we did not assess these depressive symptoms prospectively, we were unable to examine whether frontal asymmetry predicted the onset of hopelessness depression specifically. Future studies should examine this in tests of the integration of the hopelessness theory and approach–withdrawal model.

Finally, the majority of depressive episodes observed in this study were minor episodes. Further research is needed to examine the relationship between frontal asymmetry and cognitive vulnerability in the context of severe depression onset.

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