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Research report

Emotional arousal modulation of right temporoparietal cortex in depression depends on parental depression status in women: First evidence

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ABSTRACT

Background: Structural and Electroencephalography (EEG) abnormalities in right temporoparietal cortex have been associated with family history of depression (FH). Here we investigate if functional abnormalities in this area, indexed by attenuated responses to emotionally arousing stimuli, are also family-history-dependent.
Methods: Neuromagnetic activity for emotional and neutral complex scenes was recorded by Magnetoencephalography (MEG) in 20 depressed patients without, 8 depressed patients with FH, and 15 healthy controls. Emotion-sensitive neuronal steady state responses were cortical source localized and tested for group-by-emotion interactions.
Results: The group-by-emotion interaction ($F(4, 80)=4.4$, $p=0.004$) was explained by a significant modulation of right temporoparietal cortex activity by emotional arousal in controls and patients without FH. This effect was reduced in FH positive patients. The difference between patient groups remained when clinical variables such as symptom severity were accounted for.
Limitations: All patients were medicated, but differences between patient groups remained after accounting for medication dosage. Further, the sample size was limited, but data-driven resampling statistics showed the robustness of our effects. Finally, the sample consists of female patients only and we cannot generalize our results to male samples.
Conclusions: Patients with FH show impaired recruitment of attention-relevant cortical circuitry by emotional stimuli. The neuroanatomical locus of this effect accords with previous reports on structural abnormalities and electrophysiological deficits at rest in individuals with FH. Our results speak to the relevance of right temporoparietal dysfunction in emotional information processing as a potential endophenotype for depression with FH.

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1. Introduction

The familial nature of depression has been consistently shown and is thought to reflect a genetic component of this disease (Gershon et al., 1982; Sullivan et al., 2000). Individuals with parental depression status are considered likely to present endophenotypes of depression (Talati et al., 2013). Depression is regarded as an affective disorder, characterized by disturbed emotional information processing (Forbes et al., 2005; Gehricke and Shapiro, 2000) resulting in high levels of anhedonia, and blunted affect (Loas et al., 1994), related to low emotional arousal (Heller and Nitschke, 1997). Therefore, abnormalities in brain function underlying these processes and associated with family history of depression represent good candidates for identifying endophenotypes of depression.

There is accumulating evidence that electrophysiological measures of brain asymmetry in depression may be the expression of such endophenotypes. Right posterior cortical hypo-activation as indexed by increased EEG alpha band activity at corresponding electrode sites may be under genetic influences, as it depends on family load of depression (Bruder et al., 2005, 2007). Individuals at high risk for depression due to parental depression status show decreased cortical gray matter volumes in right temporoparietal

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cortex (Peterson et al., 2009) associated with increased right posterior EEG alpha band power (Bruder et al., 2012). Importantly, these changes have been observed in healthy and depressed individuals with high family load of depression, and likely represent an endophenotype of depression (Talati et al., 2013).

However, little is known about how these functional and structural deviations are related to blunted affect associated with low emotional arousal, one of the core symptoms of depression. Posterior oscillatory neuromagnetic and electrophysiologic brain responses to emotional pictures recorded with Magnetoencephalography (MEG) or Electroencephalography (EEG) are reliably modulated by emotional arousal (Keil et al., 2003, 2009, 2008; Moratti and Keil, 2009; Moratti et al., 2006, 2004, 2008). Critically, magnetocortical enhanced responses to high arousing emotional stimuli have been localized to fronto-parietal cortical areas (Moratti et al., 2004, 2011) that spatially overlap with attention-relevant cortical networks (Corbetta, 1998; Corbetta et al., 2008; Corbetta and Shulman, 2002). This is in line with the concept of motivated attention in emotion research (Lang et al., 1997) that emphasizes that behavioral, autonomic, and central nervous system responses that indicate enhanced orientating and allocation of attentional resources are modulated by emotional arousal (Bradley, 2009). Therefore, it is believed that emotional arousal drives attention-relevant brain circuits thus facilitating the processing of emotional stimuli (Vuilleumier and Driver, 2007). Critically, depressed patients do not show a modulation of right parietal cortex activity by emotional arousal (Deldin et al., 2000; Kayser et al., 2000; Moratti et al., 2008) and a recent report found this also in individuals at high risk for depression (Kayser et al., 2014). This accords with observed behavioral deficits in depressed patients in perceptual asymmetry tasks that require intact right posterior brain functioning (Heller et al., 1995; Keller et al., 2000). Further, slowing of reaction times to left hemi-field stimuli in depressed patients has been associated with an impaired right hemisphere arousal–vigilance system (Liotti et al., 1991; Liotti and Tucker, 1992). Taken together, these findings indicate a deficient engagement of right hemisphere attention relevant brain circuits by emotional arousal in depression.

Whether this deficit varies with symptom severity (e. g. as reflected by increased symptom scores on depression rating scales) or a genetic pre-disposition for depression (e. g. family history of depression) is currently unknown. A link between family history of depression and reduced arousal modulation of right temporoparietal cortex activity independent from symptom severity would speak to a common ground of reduced oscillatory brain activity to emotions and family history dependent biological measures such as cortical thinning and attenuated tonic cortical activation. Therefore, we extend previous observations regarding reduced emotional activity modulation in depression (Moratti et al., 2008) by investigating its relationship with parental depression status. We hypothesize that variance in emotional arousal modulation of right temporoparietal cortex activity in depression is better explained by family history of depression than by symptom severity that may vary over time. This would add further functional relevance to the potential endophenotypes of right parietal structural and EEG abnormalities indicating a dysfunction of attention-relevant cortical structures normally engaged by the process of orientating towards emotional arousing stimuli.

2. Methods and materials

2.1. Participants

Twenty-eight female patients from the Servicios de Salud Mental Retiro, and Hospital 12 de Octubre, Madrid, Spain meeting Diagnostic and Statistical Manual for Mental Disorders (DSM)-IV criteria for Major Depressive Disorder (MDD) volunteered to participate. Diagnosis was made using the Structural Clinical Interview for DSM-IV. Patients meeting DSM-IV criteria for anxiety disorder, and current or past substance abuse were excluded. Fifteen out of 28 patients had been included in our previous study (Moratti et al., 2008). Twenty out of 28 patients suffered from chronic, five from recurrent, and three from double depression. Eight out of 28 patients had first-degree relatives with a history of depression (family history positive: FH+; family history negative FH−). Patients were considered as FH+ if at least one of their parents had suffered from a clinical depressive episode in their life. The FH+ status was determined by an interview of the patients and their family members and by evaluations of clinical records. Six of the FH+ patients' mothers suffered from recurrent MDD, one FH+ patient's mother was diagnosed with recurrent MDD and comorbid anxiety, and one FH+ patient's father suffered from recurrent MDD. Because of standard treatment protocol all patients were medicated (see Table 1 for clinical characteristics of patient groups).

Fifteen healthy female participants volunteered as controls and met all the following criteria: no family history of mental illness, no history of psychotherapy or substance abuse. Results with respect to cortical activity modulation by emotional arousal have been reported for these control subjects previously (Moratti et al., 2008). All participants had normal or corrected-to-normal visual acuity. All subjects gave written informed consent to participate in the study. The study had approval from the Complutense University of Madrid ethics committee. Table 1 summarizes the demographic data of all participants. Healthy controls, FH+, and FH− patients differed in terms of Age ($F(2, 40)=3.8$, $p=0.03$; single comparisons: FH+ *vs.* FH−: $t(26)=2.5$, $p=0.02$; FH+ *vs.* controls: $t(21)=2.6$, $p=0.02$; FH− *vs.* controls: not significant n. s.), Hamilton-Depression-Rating-Scale (HDRS) scores ($F(2, 40)=48.3$, $p<0.001$; single scores comparisons: FH+ *vs.* FH−: $t(26)=2.5$, $p=0.02$, FH+ *vs.* controls: $t(21)=10.4$, $p<0.001$, FH− *vs.* controls: $t(33)=8.7$, $p<0.001$), Hamilton-Anxiety-Rating-Scale (HARS) scores ($F(2, 40)=52.0$, $p<0.001$; single scores

Table 1
Demographic and clinical data.

Group	No.	Handedness	Age	HDRS	HARS	Episode duration (months)	Onset (age)	Education (age of graduation)	Imipramine equivalent dosage (mg)[a]	SSRI	Tricyc.	SNRI
FH−	20	18 right, 1 left, 1 ambidx	38.3 (2.0)	16.4 (1.5)	9.5 (0.5)	18.6 (4.1)	36.8 (1.9)	16.8 (0.5)	117.2 (8.8)	20	–	–
FH+	8	8 right	47.0 (2.0)	23.9 (2.9)	10.4 (0.7)	27 (5.4)	44.8 (2.3)	18.3 (1.3)	170.3 (29.9)	4	1	3
Controls	15	14 right, 1 left	36.4 (2.8)	0.93 (0.4)	2.8 (0.5)	–	–	19.9 (1.2)	–	–	–	–

SSRI: Selective serotonin reuptake inhibitor; Tricyc.: Tricyclic antidepressant; SNRI: Serotonin-noradrenaline reuptake inhibitor, ambidx: ambidextrous.

[a] 40 mg Fluoxetine and Paroxetine (SSRI), 150 mg Venlafaxine (SNRI), and 150 mg Clomipramine (Tricyc.) dosages were estimated to be equivalent to 150 mg of Imipramine.

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