Beyond Depression: Mothers with Comorbidity Differ in Neural Response to Infants' Cry

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Mothers without any added disorder often feel overwhelmed and stressed at times, but that normal, expected stress increases with a major depressive disorder. Normative stress and fears of parenting inefficacy and child safety may be further compounded by an anxiety disorder, especially because of the common overlap between the two. An fMRI study by Heidemarie Laurent and Jennifer Ablow published in 2012 showed blunted responses in subcortical motivational systems as well as the action-oriented, planning, and emotion-regulating areas of the prefrontal cortex of mothers with depression when listening to recordings of their own infant's cry, a response that differed from that of healthy controls. Greater relative right frontal EEG alpha asymmetry has been found in patients with major depression and anxiety as compared to either those with depression only or the controls (Bruder et al., 1997). That result is in contrast to greater relative left frontal asymmetry associated with approach behaviors in mothers without this comorbid condition, as noted in studies by Field and colleagues (2003) and Killeen and Teti (2012). These differences highlight the possibility that a comorbid anxiety disorder

could be related to differential neural responses in those suffering from a depressive disorder.

Although this literature sheds some light on the neural basis for difficulties associated with both maternal depression and anxiety, it does little to address the ubiquitous issue of comorbid depression and anxiety, especially as those conditions may relate to parenting. Thus, the current study addresses the need for research on maternal brain response in mothers who are suffering from both depression and anxiety.

LITERATURE REVIEW

Comorbidity of anxiety and depression and relevance to mother-infant well-being

Anxiety and depression co-occur quite often in the clinical setting, despite their separate diagnoses (Grohol, 2013). Meta-analysis suggests that adults with both depression and anxiety show a global deficit in emotion recognition (Demenescu, Kortekaas, den Boer, & Aleman, 2010) that may partially explain the relationship depression and anxiety have with empathy and its role in infant attachment. Both classifications of disorders contribute to attachment problems between parents and infants by affecting the neural circuitry for regulating emotions, motivation, attention, and empathy. These areas are regulated by specific subcortical and cortical (especially prefrontal) circuits that are, in turn, activated by infant stimuli (Swain, Lorberbaum, Kose, & Strathearn, 2007). According to attachment theory, maternal behavioral response directly impacts infant attachment, which, when adversely affected by aberrant parenting, is related to neurotic symptoms and personality disorders (Bowlby, 1977).

Problems in affective response related to maternal depression and anxiety

Mothers with depression exhibit systematic errors in recognizing and

interpreting affective stimuli. For example, postpartum women with major depression have been found less responsive to negative emotion-eliciting stimuli compared to healthy controls (Gollan, Hoxha, Getch, Sankin, & Michon, 2013). Some researchers have related generalized anxiety disorder to an anxiety-related bias in negative information processing (Bradley, Mogg, Millar, & White, 1995), and mothers specifically show this bias in interpreting their children's behavior (Youngstrom, Izard, & Ackerman, 1999).

Depressed mothers have reported difficulty in differentiating the types of cries from their infants, an important aspect of communication between mothers and their infants (Frizzo, Vivian, Piccinini, & Lopes, 2013). The difficulties discriminating and responding effectively to negative emotion signals may, in turn, be explained by deficits in neural response.

People with depression tend to exhibit an attentional bias towards sad faces, but because of the high occurrence of co-morbid generalized anxiety disorder in studied samples, it has not been determined if this attentional bias stems from depression or anxiety (Joormann & Gotlib, 2007). Negative bias in maternal interpretation of children's behaviors in Youngstrom, Izard, and Ackerman's study (1999) support further investigation into the role of comorbid anxiety.

Brain response to infant emotion cues related to depression, anxiety

Lorberbaum and colleagues (1999) established the feasibility of using fMRI to measure the neural responses of mothers to the sound of infant cries and found statistically significant activity extending from the right subgenual anterior cingulate cortex to the right medial prefrontal cortex. Laurent and Ablow (2012, 2013), in their fMRI studies, found that many areas were associated with a mother's response to the cry of her own infant, especially the para/limbic and prefrontal regions that showed activation

in mothers with no history of psychopathology but showed no signs of activation in mothers with a history of perinatal depression. Also, non-depressed mothers showed greater activation than depressed mothers in medial thalamic and ventral striatal areas. More severe current depression related to hyporesponse in the anterior cingulate cortex, a region that, for depressed mothers, showed less activation in response to their infant's distress faces (Laurent & Ablow, 2012, 2013). Together, these findings suggest mothers suffering from depression fail to show responses to their infants' distress cues in neural circuits supporting emotion regulation and motivated approach behavior.

Although researchers had not previously addressed anxiety in this sample, increased stress reactivity (indexed by hypothalamic-pituitary-adrenal response) related to decreased activation to their infant's cry in periaqueductal gray, right insula, bilateral orbitofrontal cortex, and anterior cingulate-medial prefrontal cortex (Laurent, Stevens, & Ablow, 2011). In a sample of mothers studied early in the postnatal period, anxious thoughts correlated with activation to infant cry in subcortical areas associated with obsessive-compulsive behaviors (Swain et al., 2008). Finally, EEG studies show that a shift toward greater relative right frontal activation in response to infant emotional stimuli, which may support empathic processes is associated with lower maternal anxiety (Killeen & Teti, 2012). Still, inconsistent reports support the need for further investigation into maternal anxiety and associated brain region activation.

Why we need to examine maternal response that differs with a comorbid anxiety diagnosis

Donna Karl (1995) observed high-risk mothers in natural environments and measured infant cues and the maternal behavioral response as well as observed affect. She characterized maternal response as 1) under-

responsive 2) good enough or 3) over responsive. Interestingly, none of the mothers in the sample who placed in the "good enough" category reported anxiety, although most of the mothers in that category suffered from depression. Anxiety, however, was associated with under-responsive mothers, and the only mother who was over-responsive was both depressed and anxious. While only one mother met criteria for the over-responsive category, 80% of intrusive maternal responses were depressed. The study does not show the proportion of anxious mothers who make up the intrusive category of responses. Although Karl notes that the small sample size does not allow for a comparison of depression and anxiety in relation to maternal response, the pattern noted above indicates further research may find a difference in maternal responsiveness from mothers who are co-morbidly diagnosed with depression and anxiety (Karl, 1995).

Given evidence that depression and anxiety differentially affect the ability of mothers to respond to their infants, as well as the lack of available research on neural mechanisms, the current study was designed to address possible differences in neural response to infant cues related to comorbid anxiety and depression vs. depression alone.

THE CURRENT STUDY

This study consisted of a reanalysis of existing data collected as part of a larger research endeavor by Laurent and Ablow (2012). These data were collected in a sample of low-income mothers at risk for parenting difficulties. Half of the mothers were diagnosed with major depressive disorder, and half were healthy controls. All of the mothers in this data set who were categorized as having depression also had a comorbid condition; a subset of mothers had anxiety disorders, allowing a direct comparison of depression/anxiety vs. depression vs. control mothers. We hypothesized that the neural response to "own infant cry" of mothers with depression

and anxiety would differ significantly from the response of both mothers with depression without anxiety and healthy control mothers. Areas of differential activation were suspected to include subcortical motivational systems, as well as cortical regions involved in self-regulation, as defined by previous research.

METHODOLOGY

All methods for data collection are presented below as reported by Laurent and Ablow (2012).

Participants

A community sample of primiparous mothers of 15- to 18-month-old infants was recruited from the Women, Infants, and Children (WIC) program. Mothers who indicated interest by responding to a flier were contacted for further screening. After consenting to participate (per IRB-approved guidelines), they were screened for MRI contraindications and psychopathology using the Structured Clinical Interview for the DSM-IV (SCID). To be in the "depressed group," a mother had to meet criteria for a major depressive episode (MDE) during the perinatal period and report ongoing symptoms at least at the level of minor depression. To be in the "non-depressed group," a mother could not meet criteria for any axis I disorder. All mothers who met the criteria agreed to participate, but the final sample ($n = 22$, 11 per group) represents the subset of those screened into the study ($n = 34$) who were eligible to complete the study. Reasons for discontinuation included new pregnancies, failure to complete a well-baby visit, or the lack of an infant cry at the visit.

The majority of mothers (77%) were Caucasian, but 14% were African American and 9% were Latina. Most had experienced a vaginal delivery, although 18% had babies by caesarian section. Although 64% had some form of post-high school education, only 18% had completed college.

Mothers tended to be young ($M = 24.1$ years, $SD = 4.1$) and low SES (32% reporting household income <$10,000 per year, 36% $20,000–$40,000, 32% > $40,000). A minority (36%) were married. Non-depressed mothers did not differ from depressed mothers on any demographic variable, including marital status, but non-depressed mothers were more likely to report being in a stable married or cohabiting relationship with the biological father of their infant [$n = 8$ vs. 3 of the depressed group, $X^2(1) = 4.54$, $p = 0.03$].

Mothers in the depressed group all reported the onset of major depressive disorder (MDD) prior to the perinatal period ($M = 14.3$ years, $SD = 2.9$); thus, they represented a group prone to recurrent depression, and symptoms were not unique to perinatal events. The range of chronicity/severity varied from two fairly limited MDEs (two mothers) to too many occurrences to count (3 mothers). Six mothers reported significant (past) suicidality (four attempted, two planned). During the perinatal period, three subjects had a MDE only during pregnancy, and three others only postpartum. The remaining five mothers reported being depressed both during pregnancy and postpartum. In keeping with the less controlled but ecologically valid community sampling approach, depressed mothers were allowed to have comorbidities, although MDD had to be the dominant current complaint. Of the mothers who had MDD, 7 were also diagnosed with an anxiety disorder, while the other 4 did not qualify for an anxiety diagnosis.

Three of the depressed mothers reported having begun antidepressant (SSRI) treatment (duration 2–4 weeks). Dropping these cases did not alter the pattern of results in the previous study; therefore, in the interests of retaining balanced groups and reflecting a range of treated/untreated depression syndromes, they were kept in analyses.

Stimulus collection and presentation

Researchers attended the participants' 18-month well-baby visits and recorded infant cry sounds following injections. To create the cry stimulus, researchers recorded twenty-one seconds of crying (maximum amplitude set to 1 dB) from the beginning of each infant's cry. Comparison of depressed and non-depressed group cries revealed non-significant differences in fundamental frequencies. In addition to the participant-specific stimulus, a cry sound from an unfamiliar infant was collected (using the same procedures) and presented to all participants. A non-cry control sound was developed by editing a rising and falling tone to have a fundamental frequency within the range of a normal infant's cry, 400–600 Hz and 1 dB maximum amplitude (Zeskind & Lester, 1978).

The stimulus protocol was created as a block design and presented with the Presentation 10.0 program. The protocol consisted of two 9-minute runs. Each run contained six repetitions of own infant cry, other infant cry, control sound, and rest. Sound blocks were 23 seconds (2 seconds pause, 21 seconds sound), and rest blocks of 21 seconds. Multiple presentation input files dictating different orders of blocks within runs counterbalanced stimulus presentation within and across experimental and control groups. Participants were simply instructed to listen to the sounds to allow the most natural range of response to cry. Sounds were presented via earphones in the scanner, and a sound check carried out before each scan to ensure audibility.

Scanning

Magnetic resonance imaging was carried out with a 3T Siemens Allegra 3 magnet. A standard birdcage coil was used to acquire data from the whole brain. Sessions began with a shimming routine to optimize signal-to-noise ratio, followed by a fast localizer scan (FISP) and Siemens Autoalign routine, then the two functional runs and anatomical scan.

Functional

T2*-weighted gradient echo sequence, 64 X 64 voxel matrix, TE = 30 ms, TR = 2000 ms, flip angle = 80°, 32 contiguous slices thickness = 4 mm; 273 volumes per run.

Structural

T1-weighted 3D MP-RAGE sequence, TI = 1100 ms, TR = 2500 ms, TE = 4.4 ms, 176 transverse slices 1.0mm thick, 256 X 176 matrix FOV = 256 mm.

ANALYSIS

All analyses were conducted using FSL 4.1. At the within-participant level, response to own infant cry was tested with the contrast of activation during own cry > control sound blocks. At the between-participant level, contrasts based on psychopathology group were tested (i.e. control > depression only and depression + anxiety, depression + anxiety > or < depression only). Because of the limited number of women within each group, fixed-effects analysis methods were used. Although results cannot be generalized to the wider population, the fixed-effects analysis does allow for a description of patterns within this sample.

Preprocessing steps included motion correction with MCFLIRT, non-brain structure removal with BET, spatial smoothing using Gaussian kernel 5-mm FWHM, intensity normalization using grand mean scaling and high-pass temporal filtering (sigma ¼ 65 s). Data for the within-subject time series were analyzed using FILM with local autocorrelation correction, and boxcar models describing onset/offset of each sound stimulus were convolved with a double-gamma basis function. Functional data were registered to the participant's own high-resolution structural image (6 df) and to a standard brain (Montreal Neurological Institute template; 12 df)

using FLIRT. All data were checked for excessive motion (>1mm) and artifacts (Laurent & Ablow, 2012).

Within-participant and group-level analyses were carried out using FEAT v.5.98. For each participant, two explanatory variables (EVs) modeled signal associated with own infant cry and control sound; zero for both stimuli EVs corresponded to rest. Contrasts of parameter estimates (COPEs) for own cry > control sound tested primary hypotheses regarding response to own infant cry. First-level COPE images were averaged across runs using fixed-effects analysis. These served as inputs to higher level group analyses, conducted using FLAME to model random effects components of mixed-effects variance. AlphaSim was used to determine cluster size needed, in conjunction with intensity threshold $P < 0.005$, to achieve a false discovery rate (FDR) of 0.05 for whole-brain analyses (Cox, 1996 as cited in Laurent and Ablow, 2012). Using these criteria, activation clusters exceeding 16 voxels, or 615 mm3, were considered significant in group analyses. (Laurent & Ablow, 2012).

For the group-level analyses, tests directly compared response between groups (depressed > depressed + anxiety, depressed + anxiety > depressed, control > depressed + anxiety, and control > depressed).

RESULTS

Depression > Depression + Anxiety

Group differences when the response to own infant cry was greater than to the control sound revealed that mothers in the depressed-only group activated more to their infant's cry than comorbidly diagnosed mothers in two cortical clusters: one included the lingual gyrus, temporal occipital fusiform cortex, and parahippocampal gyrus; the other was located at the frontal pole, concentrated in the right hemisphere (see Figure 1). Refer to Table 1 for a complete listing of activation area differences by group comparison.

Depression + Anxiety > Depression

The reverse comparison, when the response of the comorbid group was greater than the depression group showed widespread differences in activation. Comorbid mothers activated more in several prefrontal and temporal cortical areas, such as the inferior frontal gyrus, precentral gyrus and supplementary motor area, and Heschl's gyrus in both the left and right cerebral cortex (see Figure 2). Greater activation was also seen in the insula and in subcortical areas such as the thalamus and putamen.

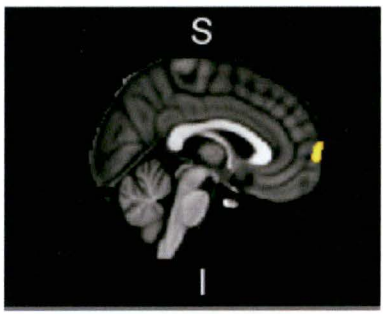

Figure 1. Frontal pole activation in depression group > depression + anxiety group as seen in the right hemisphere, sagittal plane.

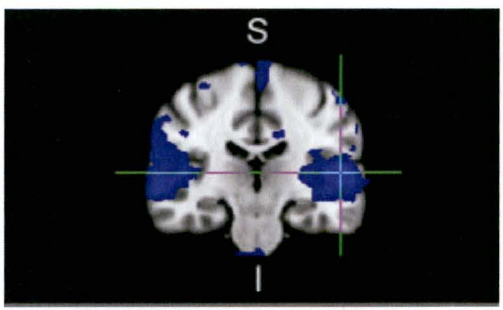

Figure 2. Difference in activation by depression + anxiety > depression. Crosshairs located at Heschl's Gyrus in the Temporal Lobe, left hemisphere, and coronal plane.

Control > Depression + Anxiety

The control group activated more than the depression + anxiety group in key subcortical motivation and prefrontal regulatory areas. The inferior frontal gyrus activated more for the control group, as did both the left and right nucleus accumbens (see Figure 3). The anterior cingulate gyrus also activated more for the control group than the comorbid group.

Control > Depression

As expected, based on previous comparisons, the control group activated more than the depressed group in Heschl's gyrus, the pre- and post-central gyri, the frontal pole, and the cingulate gyrus. Cortical peak activation extended into subcortical areas, including the thalamus, putamen, and nucleus accumbens (see Figures 4 and 5).

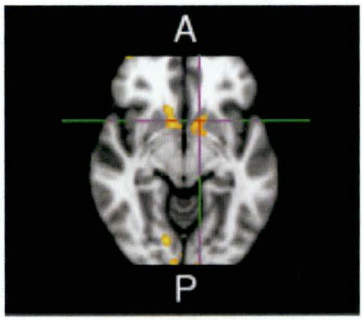

Figure 3. Activation in the left and right accumbens for the group comparison of control > depression +anxiety, as seen in the axial plane.

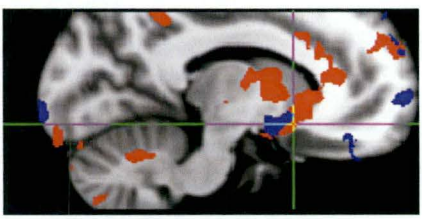

Figure 4. Red color shows activation of control > depression; blue shows activation of control > depression + anxiety. Crosshairs located at the left accumbens where both comparisons showed significant activation. Sagittal plane, right hemisphere.

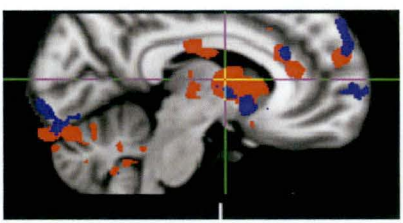

Figure 5. Red color shows activation of control > depression; blue shows activation of control > depression + anxiety. Crosshairs located at the right thalamus. Activation extends into the caudate. Sagittal plane, right hemisphere.

DISCUSSION

In the current study, mothers with a comorbid anxiety disorder showed differential activation to their infants' cry when compared to mothers with depression and no anxiety disorder. Overall, the comorbid group exhibited a greater neural response than the depression-only group, but they also showed a few areas of lesser response. As predicted, the control group

activated more than both of the other groups. These findings suggest that the effect of an additional anxiety diagnosis may attenuate the effects of depression on the mother's brain response to her infant.

Mothers with depression alone showed more activation in the lingual gyrus and the temporal occipital fusiform cortex than comorbid mothers. These areas are associated with object and facial recognition, suggesting that mothers with anxiety do not associate their babies' crying with their babies' faces to the same degree as mothers without anxiety. Furthermore, activation in the parahippocampal gyrus suggests that mothers may be thinking more about how the babies' cries fit in a social context, although further research is needed to determine if these findings are consistent with behavioral or subjective thought differences.

When the contrast was reversed, mothers with depression and anxiety showed significantly more activation in several prefrontal, temporal, and subcortical areas than mothers with depression only. Heschl's Gyrus in the temporal lobe is associated with auditory processing of speech. This result may suggest that the cry is being appropriately interpreted not simply as than noise but as speech. Even at 18 months when verbal communication has typically begun to develop, a cry may express more than the child is able to communicate with speech. For the mothers with depression and anxiety, this speech auditory processing appeared to be more prevalent than for the mothers with depression alone. Activation in Heschl's gyrus also spread to the Planum Temporale, which is involved in locating sounds in space. This may indicate that the mothers are asking, "Where is my baby, and what is my baby trying to tell me?"

Comorbid mothers also showed more activation in paralimbic and subcortical areas important for social-emotional functioning. The frontal orbital cortex is involved in processing internal states, especially emotion. The insula is also implicated in subjective emotional responses and in

empathy, especially for pain. Empathy for pain is appropriate in this context because of the method used to obtain the cry sound from the baby. Therefore, the attentional bias of anxiety in these comorbid mothers may give mothers an increased awareness of the distress communicated in their babies' cries.

Additionally, comorbid mothers activated more in the primary motor cortex and supplementary motor area. Activation in these regions is consistent with planning or imagining movement. The specific motor area identified here has been associated with the arm and hand, possibly suggesting that these mothers were thinking more about holding or moving toward their baby. Further research examining behavioral maternal response in these populations could determine if the activation in the arm and hand regions is associated more with comforting or intrusive behaviors.

Mothers with anxiety also showed more activation to their infant's cry in the thalamus. This area activates as part of a basic parental response and had previously been shown to be more active in control compared to depressed mothers in this sample. While the mothers with anxiety typically activated more than mothers without anxiety, they still did not activate as much as the control group, a result that was expected, based on behavioral differences seen in mothers suffering from depression and the underlying neural activation as examined in the original study.

Control mothers showed widespread areas of greater activation than the depressed group but they only activated more in a few areas when compared with the comorbid mothers. These few areas, however, are crucial to effective and sensitive parenting. Compared to the mothers with depression and anxiety, control mothers activated more in the inferior frontal gyrus, an area associated with language processing. This difference suggests that control mothers are thinking more about what their baby is

trying to say than are the mothers with depression and anxiety. Indeed, the relative increased activation in the inferior frontal gyrus is associated with "mirroring" speech vocalization. This is a good illustration of the way in which the mothers with the comorbid anxiety diagnosis are activating more than the mothers with just depression, and in turn the control mothers are activating more than the mothers with the comorbidity. Paying attention to what the child is trying to communicate is part of sensitive parenting, a skill that is often negatively impacted by depression and anxiety.

Another important area involved in emotion and attention regulation processes, the cingulate gyrus, activated more for the control mothers than the comorbid mothers. While the mothers with depression and anxiety showed greater overall activation to their children's cry sounds as compared to the mothers with depression without anxiety, they did not show the same degree of activation as the control mothers. Monitoring salient cues and regulating one's own response is another important aspect of sensitive parenting. Mothers with depression and anxiety may appear to be paying more attention to their infant's cry than mothers with pure depression, but without adequate resources for regulating their attention or emotional reaction, these mothers may be displaying the intrusive behaviors such as those found in Karl's study (2005).

Differences in sensitive parenting between mothers with no psychiatric diagnoses and those with depression, including those with comorbid anxiety, may also be related to the differences seen in responsivity of the nucleus accumbens. Greater activation in the striatum—the nucleus accumbens, putamen, and caudate—imply a greater infant-related reward and approach motivation in the control mothers than in mothers with depression. Depression often results in a lack of motivation, and these findings are consistent with past research on the effects of depression. The fact that mothers with depression and anxiety showed no difference in this

area compared to depression alone suggests this component of maternal response is not as differentially maintained as the response interpretation components discussed above, and anxiety may be more universally blunted by the experience of depression.

Musser, Kaiser-Laurent, and Ablow (2012) examined the behavioral response of these mothers to their infants in an earlier study to determine the neural correlates related to sensitivity. Overlap between certain regions may help to explain why depressed mothers often show less sensitivity to their infants. For example, those mothers who showed more activation in the inferior frontal gyrus, an area that was found to be more responsive in the control group of the current study, were more sensitive to their infants' cues in the observed interactions. In fact, the left anterior insula and temporal pole associated with a trend in greater intrusiveness showed greater activation for mothers with depression and anxiety as compared to mothers with depression without anxiety in the current study. Future research will shed more light on neural correlates of behavioral differences between depressed vs. depressed and anxious mothers.

A limitation of this study is the restricted sample size and associated fixed effects analysis. Those factors precluded strong statements about application to clinical populations. Future research in larger and more varied samples is necessary to detect and interpret effects of depression comorbidity with specific types of anxiety disorders as well as other common comorbidities such as substance use and trauma-related disorders.

Despite these limitations, the current study contributes to research literature supporting a qualitative difference between depression and depression with anxiety. The neural mechanisms underlying maternal behavior are affected by the comorbid illness in several key frontal, temporal, and subcortical areas, suggesting differences in emotional information processing and affective regulation for those mothers

suffering from an anxiety disorder in conjunction with major depression. Considering the implications of these processes for parental sensitivity, parent child interactions, and child behavioral outcomes, understanding these differences may lead to more effective interventions.

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Table 1 Activation in mothers' response to infant cry by group differences

Group contrast	Region	L/R	X	Y	Z	Z_{max}	Volume (mm^3)
Depression > Depression + Anxiety	Frontal Pole	R	2	66	7	4.35	876
	Lingual Gyrus extending to Temporal and Occipital Fusiform Cortex, Parahippocampal Gyrus	L	-20	-43	-15	3.86	1539
Depression + Anxiety > Depression	Frontal Pole	L	-44	41	-7	4.77	4488
	Middle Frontal Gyrus extending to Inferior Frontal Gyrus	R	40	31	27	3.69	1437
		L	-43	31	27	4.54	1338
	Insular Cortex extending to Frontal Orbital Cortex, Putamen	R	27	21	-1	4.68	2592
	Precentral Gyrus extending to Inferior Frontal Gyrus	L	-36	12	22	4.08	822
	Supplementary Motor Cortex	L/R	1	0	66	4.82	1523
	Precentral Gyrus, Middle Frontal Gyrus	R	41	-1	55	4.85	3256
	Superior Frontal Gyrus	L	-16	-2	75	3.97	670
	Thalamus	L/R	-1	-8	2	4.43	1529
	Postcentral Gyrus	L	-57	-15	44	4.91	4530
	Precentral Gyrus	R	32	-16	68	4.3	724
	Heschl's Gyrus (includes H1 and H2),	R	49	-19	8	9.44	42469
	Planum Temporale	L	-54	-23	8	10.2	45589
	Precentral Gyrus	L	-6	-23	78	4.25	1508
	Superior Parietal Lobule	L	-29	-56	55	4.06	1831
	Frontal Pole	L	-13	45	47	4.51	2465

Table 1 Activation in mothers' response to infant cry by group differences

Group contrast	Region	L/R	X	Y	Z	Z_{max}	Volume (mm^3)
Control > Depression + Anxiety	Frontal Pole	L	-13	45	47	4.51	2465
		R	19	40	50	4.73	7706
	Frontal Medial Cortex	L	-5	43	-23	3.95	1223
	Anterior Cingulate Gyrus	R	6	29	23	3.81	891
	Inferior Frontal Gyrus	R	57	21	19	4.38	1173
	Superior Frontal Gyrus	R	10	19	66	3.97	2183
	Nucleus Accumbens, Putamen, Caudate	L	-11	11	-8	3.72	823
		R	9	10	-6	3.7	1769
	Supramarginal Gyrus, extending to Angular Gyrus	R	46	-46	45	4.18	781
	Occipital Pole	L	-11	-105	-7	4.54	2454
		L	-8	61	24	4.14	654
Control > Depression	Frontal Pole	R	45	54	-7	3.25	827
	Superior Frontal Gyrus	L	-4	29	59	3.67	1045
	Supplementary Motor Cortex	L/R	1	-1	70	5.33	4697
	Precentral Gyrus	L	-53	-13	45	5.17	3802
		R	33	-16	68	4.21	1005
	Heschl's Gyrus (includes H1 and H2)	R	49	-19	7	11.4	129550
	Postcentral Gyrus, extending to Superior Parietal Lobule	L	-30	-37	67	3.77	978
	Precuneous Cortex extending to Posterior Cingulate Gyrus	L	-13	-47	38	3.91	843