Review article

Brain activation during processing of genuine facial emotion in depression: Preliminary findings

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A B S T R A C T

Objective: The current study aimed to examine the neural correlates of processing genuine compared with posed emotional expressions, in depressed and healthy subjects using a novel functional magnetic resonance imaging (fMRI) paradigm

Method: During fMRI scanning, sixteen depressed patients and ten healthy controls performed an Emotion Categorisation Task, whereby participants were asked to distinguish between genuine and non-genuine (posed or neutral) facial displays of happiness and sadness.

Results: Compared to controls, the depressed group showed greater activation whilst processing genuine versus posed facial displays of sadness, in the left medial orbitofrontal cortex, caudate and putamen. The depressed group also showed greater activation whilst processing genuine facial displays of sadness relative to neutral displays, in the bilateral medial frontal/orbitofrontal cortex, left dorsolateral prefrontal cortex, right dorsal anterior cingulate, bilateral posterior cingulate, right superior parietal lobe, left lingual gyrus and cuneus. No differences were found between the two groups for happy facial displays.

Limitations: Relatively small sample sizes and due to the exploratory nature of the study, no correction was made for multiple comparisons.

Conclusion: The findings of this exploratory study suggest that depressed individuals may show a different pattern of brain activation in response to genuine versus posed facial displays of sadness, compared to healthy individuals. This may have important implications for future studies that wish to examine the neural correlates of facial emotion processing in depression.

1. Introduction

Depression is characterized by negative biases in emotional information processing and it is believed that this may play a critical role in the development and maintenance of the disorder (Roiser and Sahakian, 2013). As such, there has been considerable interest in examining the neural correlates of emotion processing in individuals with depression.

Neural models of depression posit that negative affect and mood congruent biases arise from, or are related to, dysfunction within fronto-limbic circuits (Malhi et al., 2015). Frontal brain regions such as

the dorsolateral prefrontal cortex (DLPFC) and dorsal anterior cingulate cortex (dorsal ACC) are thought to be hypoactive, whereas regions such as the ventral/rostral anterior cingulate cortex (ventral/rostral ACC) and amygdala are thought to be hyperactive; particularly in the context of mood congruent stimuli (Hamilton et al., 2013; Mayberg, 1997).

A number of functional magnetic resonance imaging (fMRI) studies have examined neural activity in depression using paradigms that involve facial emotion processing (Stuhrmann et al., 2011). Some studies have found evidence of decreased frontal activity in depressed individuals relative to controls (Fu et al., 2004; Siegle et al., 2007a, 2007b). However, others have found evidence of increased frontal

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activity during the processing of negative facial expressions (Anand et al., 2005; Keedwell et al., 2005; Lawrence et al., 2004; Rosenblau et al., 2012). There are similarly diverse findings with regards to limbic activation in response to negative stimuli, with a number of studies reporting increased limbic activity in depressed individuals versus controls (Anand et al., 2005; Siegle et al., 2002, 2007b), in contrast to others that have found no such differences (Lee et al., 2008; Scheuerecker et al., 2010). Inconsistencies between studies likely stem from heterogeneity among patient samples, the use of varying fMRI paradigms and stimuli, the differential effects of psychotropic medication and the use of differing neural models to interpret imaging findings.

A key issue of interest that has not been examined in detail, is whether there are differences in the way the depressed brain responds to *genuine* compared with *posed* displays of emotion. Genuine expressions are spontaneously generated as part of an emotional experience. In contrast, posed expressions are not coupled with their respective emotion and are used as a means to fake, mask or suppress emotional experience (Ekman and Friesen, 1982; Ekman and Rosenberg, 1997). Determining whether facial information specifies emotion or not, is crucial for effective social functioning. For instance, mistaking posed displays for genuine displays can result in negative outcomes for the social perceiver (Miles and Johnston, 2007). Any compromise in the ability to distinguish posed from genuine expressions of emotion might help explain why depressed individuals often find it difficult to engage socially. Indeed, using an Emotion Categorisation Task to assess sensitivity to genuine versus posed facial displays (McLellan et al., 2010), Douglas et al. (2012) have shown that depressed patients are less able than healthy controls, to differentiate between posed and genuine expressions of sadness. Interestingly, in an fMRI experiment in healthy subjects, this same task activated different brain regions when viewing and judging emotional veracity of genuine versus posed emotions (McLellan et al., 2012). However, whether this holds for depressed patients is unknown.

1.1. Aims of the study

The current study aimed to examine the neural correlates of processing genuine and posed facial expressions in depression, using the Emotion Categorisation Task. Based on extant neural models of depression, it was hypothesised that there would be a discernible difference in regional brain activity in response to genuine versus posed facial expressions of emotion, in the depressed group compared with controls.

2. Method

2.1. Participants

Nineteen right-handed depressed participants (7 male: 12 female;

22–57 years of age) were recruited from a randomised outpatient psychotherapy trial of Cognitive Behaviour Therapy (CBT) and Metacognitive Therapy (MCT) for depression (Jordan et al., 2014). Inclusion criteria for the clinical trial included a current primary DSM-IV diagnosis of major depressive disorder or bipolar II disorder-depressed phase, an age of 18 years or older and the ability to converse and answer questionnaires in the English language, and provide informed consent. Exclusion criteria included bipolar I disorder, schizophrenia, current severe substance misuse, an adequate course of CBT or MCT in the past year, use of psychotropic medication (other than intermittent short term hypnotic use), or severe physical illness. Participants of the clinical trial were required to be drug free for a minimum of two weeks or five drug half-lives. A research nurse screened referrals for the clinical trial's inclusion/exclusion criteria and potential participants were contacted by the next available therapist and booked for a clinical interview. Informed consent was obtained from eligible participants. Clinician-rated diagnostic assessments of mood were conducted using the Structured Clinical Interview (SCID I and II) for the Diagnostic and Statistical Manual of Mental Disorders-IV (American Psychiatric Association, 2000) and clinician ratings of current mood severity were made using the 16 item Quick Inventory of Depressive Symptomology (QIDS16-C) (Rush et al., 2003), and the Montgomery Asberg Depression Rating Scale (MADRS) (Montgomery and Asberg, 1979). The nineteen depressed participants taking part in the present fMRI study were a consecutively recruited subgroup of the larger clinical trial sample. Further inclusion criteria included a willingness to participate in the fMRI scanning component of the study. Thirteen right-handed healthy controls (7 male: 6 female) with no history of depression, were also recruited. Control participants were a convenience sample and were matched for age bands (+/− 5 years) with the depressed outpatients. The healthy controls had no history of depression, which was assessed using the SCID for DSM-IV and symptoms were assessed using the Depression, Anxiety, Stress Scales (DASS; (Lovibond and Lovibond, 1995)). Exclusion criteria for both patient and control participants in the fMRI study were left-handedness and any medical conditions that might interfere with MRI scanning (e.g. pacemaker, metal implants). Informed consent was obtained from all participants prior to being scanned. Ethical approval was received from the Upper South B Regional Ethics Committee, New Zealand (URB 09/03/012).

2.2. Emotion categorisation task

The stimuli used during the fMRI paradigm were photographs depicting a female target (five different targets were used). The target displayed either posed or genuine facial expressions (happy or sad) or a neutral expression (see Fig. 1 for example). The facial displays used were taken from an established behavioural task (McLellan et al., 2010) and met the FACS (Ekman and Friesen, 1975) criteria as being indicative of their respective emotions.

fMRI data was collected in four 7-min runs, with images presented

Fig. 1. Examples of a neutral, posed sad and genuine sad expressions from the Emotion Categorisation Task.

one at a time through adjustable binocular glasses (Avotec SV-7021, Stuart, Fl.) from a Pentium 4 computer. Each run consisted of a set of 21 facial images presented in a pseudo-random order, with each followed by a fixation cross. The facial images were seven each of posed and genuine expressions of the target emotion (happy and sad) as well as seven neutral expressions. Facial images were presented for 2 s and fixation crosses were presented for 4, 4.5 or 5 s (also pseudo random, seven of each duration). Each set of facial images and fixation durations were repeated three times within a single run. An additional 10.5 s fixation cross was presented at the end of each run. The four runs were presented as follows: *SHOW Happiness*, *SHOW Sadness*, *FEEL Happiness* and *FEEL Sadness*. For the SHOW runs, the participants were asked to judge whether the face was showing the target emotion. For the FEEL runs, the participants were asked to judge whether the face was feeling the target emotion. Participants responded yes or no by pressing a two key-response box held in the right hand. Because of the complexity of the analysis and the small size of the sample, we made the decision only to analyse the contrasts for the FEEL conditions.

2.3. Image acquisition and analysis

Images were acquired on a 3 T General Electric HDxt scanner (GE Healthcare, Waukesha, WI) with an 8-channel head coil at Hagley Radiology, Christchurch, New Zealand. T1-weighted structural images were acquired with a three-dimensional spoiled gradient recalled echo sequence (echo time [TE] = 2.8 ms, repetition time [TR] = 6.7 ms, inversion time = 400 ms, flip angle = 15°, acquisition matrix 256 × 256 × 158, axial acquisition, field of view = 250 mm, slice thickness = 1 mm, voxel size $0.98 \times 0.98 \times 1$ mm^3). BOLD functional images were acquired with a T2*- weighted single shot, echo planar imaging (EPI) sequence (echo time = 35 ms, repetition time = 2500 ms, flip angle = 90°, acquisition matrix = 64 × 64 × 37, field of view = 240 mm, slice thickness = 4 mm, voxel size = $3.7 \times 3.7 \times 4$ mm^3). A Gradient echo field map acquisition acquired at two different echo times (TE1 = 5.3 ms, TE2 = 7.6 ms, TR = 600 ms, acquisition matrix = 96 × 96 × 37, field of view = 240 mm) was used to correct distortion. Structural and functional data were analysed with SPM12b (v5581, www.fil.ion.ucl.ac.uk/spm) running in MATLAB (R2010a; The Mathworks Inc, 2010) and FSL v.5.0.7 (www.fmrib.ox.ac.uk/fsl). Structural T1-weighted images were intensity bias corrected and tissue classified into grey matter and white matter segments using the VBM8 toolbox (http://dbm.neuro.uni-jena.de/vbm/). DARTEL (existing template) was then run using the DARTEL template provided with VBM8 to warp grey matter images into standardized space and smooth with an 8 mm^3 isotropic Gaussian kernel. Functional images were spatially realigned and unwarped using the FieldMAP utility in SPM. Realignment produced estimates of motion during the task, which were then used to reduce movement artefacts. The unwarp function was used to minimise susceptibility distortions. Slice timing correction was then conducted and a mean functional image for the four different conditions was produced. The mean functional images were co-registered to their corresponding structural images and applied to all functional images, which were then normalised (using the parameters derived from the structural procedure) and smoothed (8 mm^3).

To compare grey matter volumes between the depressed and control groups, the smoothed, normalised grey matter images were analysed in SPM12b using an independent sample *t*-test, with age and gender as covariates. The statistical threshold was set at $p \leq 0.001$ uncorrected for multiple comparisons, with an extent threshold of > 10 voxels.

The pre-processed functional images were analysed in SPM12b using the general linear model in a two-staged approach. Individual analysis of both the FEEL HAPPY and FEEL SAD conditions was modelled with the stimulus onset times and durations per item. First level general linear modelling included six regressors: one regressor each for correct responses to the three types of facial stimuli—genuine, posed, and neutral—and one regressor each for the incorrect responses to each

of the three facial stimuli. Regressors were convolved with a canonical haemodynamic response function (Friston et al., 1998). Six rigid-body motion correction parameters (three translation and three rotation) were included as nuisance covariates. Contrast images were extracted for individuals and entered into a second level whole-brain analysis. The contrasts of interest in the current study were: Genuine vs. Neutral, Posed vs. Neutral and Genuine vs. Posed for the *FEEL SAD* and *FEEL HAPPY* conditions. The 'second-level' analyses were conducted using the contrast images generated at the first level for each participant. Independent sample *t*-tests were used to compare differences in brain activation between the depressed outpatients and controls, with age and gender entered as covariates. As with the structural comparison, the statistical threshold was set at $p \leq 0.001$ uncorrected for multiple comparisons, with an extent threshold of > 10 voxels.

3. Results

3.1. Excluded data

Functional MRI data from three of the control subjects could not be used because field maps were not acquired. Additionally, data from three of the depressed participants was unable to be used as the participants had difficulty in fully understanding and performing, the posed versus genuine task.

3.2. Demographic and clinical data

Demographic and depression severity data are reported in Table 1. The mean age of the patients and control samples were comparable. According to both depression measures, the depressed group had a mean depression severity that was in the moderate range. None of the patients had been prescribed antidepressants in the six weeks prior to recruitment. Fifteen of the sixteen participants in the depressed sample had a primary diagnosis of MDD, and one had a primary diagnosis of Bipolar II Disorder, depressed phase.

3.3. Structural data

Structural data were compared between the depressed (n = 16) and healthy control group (n = 10) in order to reduce the possibility that differences in brain activation were a result of structural differences. Whole brain analyses revealed no significant differences between groups.

Table 1
Demographic data for depressed and healthy controls.

	Depressed (n = 16)		Controls (n = 10)		Statistic[a]	p
	Mean/%	SD	Mean/%	SD		
Age (years)	35.4	12.7	35.1	12.1	− 0.07	0.95
Gender (male %: female %)	37:63	–	60:40	–	1.25	0.26
Age of Onset (years)	20.1	11.6				
Number of Prior MDEs	4.1	3.8				
QIDS	14.7	5.0	–	–	–	–
MADRS	22.1	9.3	–	–	–	–
DASS (Depression subscale)	–	–	1.9	4.0	–	–

MDE, major depressive episode; QIDS, Quick Inventory of Depressive Symptomology; MADRS, Montgomery Asberg Depression Rating Scale; DASS, Depression, Anxiety, Stress Scales.

[a] T-tests for continuous variables and chi square for dichotomous variables.

Table 2
Brain regions showing greater activation in the depressed group (n = 16) relative to controls (n = 10) during the processing of genuine displays of sadness.

Cluster Location	Coordinates			Cluster Size	t value	p
	x	y	z			
Genuine > Posed						
L. Medial orbital frontal cortex	−15	50	−6	55	5.22	< 0.001
L. Putamen	−18	20	−3	26	4.50	< 0.001
L. Caudate	−16	8	11	18	3.89	< 0.001
Genuine > Neutral						
R. Medial frontal/ orbitofrontal cortex	23	53	−3	19	3.77	0.001
L. Medial frontal/ orbitofrontal cortex	−31	50	−11	28	4.57	< 0.001
L. Dorsolateral prefrontal cortex	−34	42	17	29	4.18	< 0.001
R. Anterior cingulate cortex (dorsal)	2	9	24	36	4.02	< 0.001
R. Posterior cingulate cortex	3	−19	29	74	3.91	< 0.001
L. Posterior cingulate cortex	−22	−57	9	11	3.98	< 0.001
R. Superior parietal lobe	21	−54	56	97	4.27	< 0.001
L. Lingual gyrus	−30	−60	−3	40	4.49	< 0.001
	−27	−69	−2		3.79	0.001
L. Occipital lobe - cuneus	−21	−84	33	19	4.49	< 0.001
	−7	−82	18	56	4.14	< 0.001
	−4	−79	26		3.78	0.001

L, Left; R, Right.
Results of whole brain analyses, $p \leq 0.001$ uncorrected, extent threshold of > 10 voxels per cluster.

3.4. Functional data

Whole brain analyses for the FEEL SAD condition revealed a number of significant differences in brain activation between depressed and healthy controls (Table 2). In comparison to the healthy control group, the depressed group showed greater activation whilst processing genuine versus posed facial displays of sadness, in the left medial orbitofrontal cortex and the left caudate and putamen (Fig. 2). The depressed group also showed greater activation during genuine facial displays of sadness relative to neutral displays, in the right medial frontal/orbitofrontal cortex, left medial frontal/orbitofrontal cortex (medial aspect), left DLFPC, right dorsal ACC, bilateral posterior cingulate cortex, right superior parietal lobe, left lingual gyrus and the cuneus of the left occipital lobe (Fig. 3). No differences were found for the posed versus neutral comparison.

Whole brain analyses for the FEEL HAPPY condition revealed no significant differences in brain activity between the depressed and healthy control group. This applied to the processing of genuine displays of happiness versus neutral faces, posed displays of happiness versus neutral faces and genuine versus posed displays of happiness.
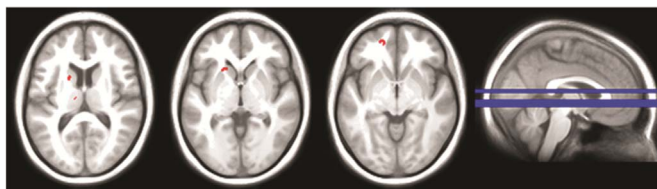

Fig. 2. Increased activation of depressed patients compared to healthy controls when processing genuine versus posed displays of sadness. Red clusters indicate greater activation in the depressed group, $p \leq 0.001$ uncorrected, extent threshold of > 10 voxels per cluster. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article).

4. Discussion

To date, no previous neuroimaging studies have examined differences in brain activation in response to genuine versus posed facial expressions in individuals with depression, and only one has examined this in healthy adults (McLellan et al., 2012). Our small, exploratory study revealed intriguing differences in brain activity between the depressed and healthy control groups during the processing of genuine sad facial expressions. No group differences were found in relation to happy facial expressions. In comparison to healthy controls, depressed patients showed greater activation within the medial orbitofrontal cortex (OFC), caudate and putamen, whilst processing genuine versus posed facial displays of sadness. Additionally, depressed patients also showed greater activation within prefrontal regions, the dorsal ACC, posterior cingulate cortex, occipital lobe and extra-striate regions, during the processing of genuine facial displays of sadness, relative to neutral displays.

Findings of a differential pattern of neural activity between the depressed and healthy control group in response to genuine facial emotion, is in line with our prior assumptions. During the processing of genuine versus posed facial expressions of sadness, the depressed group showed greater activation within the medial OFC, caudate and putamen, in comparison to healthy controls. One possible explanation for finding greater medial OFC activity in the depressed group, is that it may reflect an increase in attention towards mood congruent stimuli. A study by Bhanji and Beer (2012) found that medial OFC activity may be associated with emotion-congruent judgement in healthy individuals, suggesting that the medial OFC may direct attentional resources towards information that is congruent with emotional-state (Bhanji and Beer, 2012). Additionally, Wang et al. (2006) have found evidence that neural activity within the OFC may be elevated in response to sad distractors during sad mood induction (Wang et al., 2006). Based on these findings, it is possible that greater activation within the medial OFC of the depressed group, reflects an increase in attention towards mood-congruent information, since genuine sad facial displays may be more congruent with mood than posed displays. This idea is partly supported by the fact that no differences were found between the depressed and healthy control groups whilst processing happy facial expressions.

The current study also found greater activity within the ventral striatum (caudate and putamen) of the depressed group, in response to genuine displays of sadness, relative to posed displays. A number of studies have shown the ventral striatum to be hyperactive in depressed individuals (Fu et al., 2004; Lawrence et al., 2004; Scheuerecker et al., 2010). Furthermore, the striatum (particularly the caudate) has been shown to produce strong responses in relation to negative pictures, with activity increasing as a function of arousal (Carretie et al., 2009). This finding further supports the notion that individuals with depression may be more responsive to genuine facial displays of sadness compared with posed displays.

Findings of greater neural activation in the depressed group, during the processing of genuine versus posed displays of sadness are important. This raises the possibility that differing findings from some neuroimaging studies investigating facial emotion processing in depression, may be a consequence of using posed facial displays, which elicit a significantly different emotional experience.

The current study also found differences between the depressed and healthy control groups during the processing of genuine sad facial displays versus neutral displays. The depressed group showed greater activation within numerous regions of the brain, including prefrontal areas and the dorsal ACC, in response to genuine sad facial expressions. These findings are contrary to extant neural models of depression, which posit that the disorder is usually associated with decreased frontal activity and increased limbic activity (Mayberg, 1997). Furthermore, many neuroimaging studies have found evidence in support of these models, especially in the context of negative affective stimuli
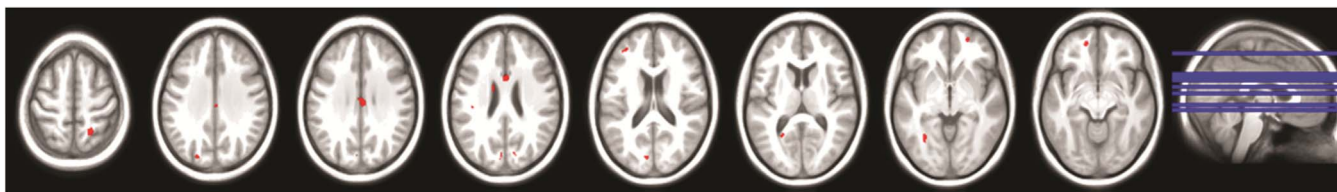
Fig. 3. Increased activation of depressed patients compared to healthy controls when processing genuine sad versus neutral faces. Red clusters indicate greater activation in the depressed group, $p \le 0.001$ uncorrected, extent threshold of > 10 voxels per cluster. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article).

(Fu et al., 2004; Siegle et al., 2002, 2007a, 2007b). Nevertheless, there are some neuroimaging studies that have found evidence of increased prefrontal activity (Anand et al., 2005; Keedwell et al., 2005; Lawrence et al., 2004; Rosenblau et al., 2012) and unaltered limbic activity (Lee et al., 2008; Scheuerecker et al., 2010), in individuals with depression. For example, Scheuerecker et al. (2010) found that patients with depression showed greater activation within the middle frontal cortex, caudate nucleus, precuneus and lingual gyrus, during a face matching task using sad and angry faces. Moreover, they found no differences in amygdala activation between the patients with depression and healthy controls. They suggested that due to the cognitive nature of the task, the hypothesised increase in limbic activation may have been suppressed by prefrontal regions. Similarly, this may explain the increase in prefrontal activity and lack of differences in limbic activation in the current study, as the emotional processing task required participants to make a judgement about whether the faces were *feeling* the emotion. A previous study has shown that individuals with depression have greater difficulty in determining the difference between "feeling" and "showing" emotions (Douglas et al., 2012). Thus, it is possible that this judgement component led to the activation and elaboration of more cognitive processes, as it required greater cognitive effort from the depressed patients.

Another possible explanation for finding increased prefrontal activity in the depressed group during the genuine versus neutral condition, is that it reflects an increase in attention towards mood-congruent stimuli. The prefrontal cortex is known for its role in attention (Miller and Cohen, 2001) and as mentioned above, there is some evidence that the OFC may direct attentional processes towards information that is congruent with emotional state. Moreover, whilst the depressed group displayed greater activation in these areas during the processing of sad facial expressions, no differences were found with happy expressions. In line with this theory, the depressed group showed greater activation within the dorsal ACC, which is thought to initiate the elaborative processing of emotional stimuli (Davidson et al., 2002), and the superior parietal cortex, which is known to activate during processes of attention (Behrmann et al., 2004). Greater activity was also detected within the posterior cingulate, occipital cortex and extra-striate area (lingual gyrus) of the depressed group. Evidence suggests that the posterior cingulate is activated during the processing of emotionally salient stimuli (Maddock et al., 2003) and studies have found increased activity in depressed individuals (Fu et al., 2004; Keedwell et al., 2005). The affective nature of stimuli can also influence the activation of both the primary and secondary visual cortices (Lang et al., 1998), so greater activity within the posterior cingulate and visual areas also suggests greater processing of mood congruent stimuli. It is also important to note that the depressed group only showed greater neural activation for the genuine versus neutral comparison, but not the posed versus neutral comparison; underscoring the importance of attentional engagement by mood congruent stimuli.

4.1. Strengths and limitations

The current study has a number of strengths. Firstly, the emotional processing task is novel and includes examination of the processing of genuine emotional expressions. This may have greater ecological validity in examining typical emotional processing and perception in depression. To date, no studies have examined differences in brain activity in response to processing *posed* versus *genuine* facial expressions in individuals with depression. Secondly, all the depressed patients were free of psychotropic medication. This is extremely important as many studies do not control for medication effects and even with compensation within analyses, it is difficult to parse out the effects on neural activity.

Equally, it is important to acknowledge the limitations of the present study. Most notably, its small sample size, which limits statistical power. Because of this and also the exploratory nature of the study, we presented the uncorrected results from our analyses, increasing the risk of Type I error. It should be noted that when a correction was made for multiple comparisons, nothing survived. However, we believe that the uncorrected results are of interest, as they help to inform other researchers of brain regions that may be of particular interest for further study. Our findings are preliminary and indeed require further replication. Another limitation was the use of a mixed patient sample, with one participant in the depressed group having a diagnosis of bipolar II disorder. It was decided that the participant should not be excluded from the analyses because while there is evidence of differences between bipolar I disorder and major depressive disorder (MDD) in terms of brain activation/function, there is little evidence of differences between bipolar II disorder and MDD (Malhi et al., 2015; Porter et al., 2015). For this reason, we felt it reasonable to include this patient in this preliminary study. Another limitation of the current study was that it only focussed on brain activation during the FEEL conditions of the task. We focussed on the FEEL conditions rather than the SHOW as our primary focus was how the depressed brain responds to genuine facial expressions of emotion. Moreover, we felt that given the small sample sizes, it would be better to limit the amount of analyses we performed. Future studies should also consider only examining the FEEL SAD condition and adding more genuine and posed sad facial expressions to enhance the robustness of the task.

5. Conclusion

This exploratory study found evidence suggesting that depressed individuals may show a differential pattern of brain activation in response to genuine versus posed facial displays of sadness, compared to healthy individuals. This may have important implications for future research on facial emotion processing in depression, as it raises the possibility that inconsistencies between studies may be a function of the 'genuineness' of the stimuli being used. Furthermore, the findings also suggest that the novel fMRI paradigm used in the current study, taps into distinct emotion processing circuits compared with previously employed emotion activation paradigms.

analysis and interpretation of the data; in the writing of the report; and in the decision to submit the paper for publication.

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