Ventromedial Frontal Lobe Plays a Critical Role in Facial Emotion Recognition

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Abstract

■ The ventromedial prefrontal cortex has been implicated in a variety of emotion processes. However, findings regarding the role of this region specifically in emotion recognition have been mixed. We used a sensitive facial emotion recognition task to compare the emotion recognition performance of 7 subjects with lesions confined to ventromedial prefrontal regions, 8 subjects with lesions elsewhere in prefrontal cortex, and 16 healthy control subjects. We found that emotion recognition was impaired following ventromedial, but not dorsal or lateral, prefrontal damage. This impairment appeared to be quite general, with lower overall ratings or more confusion between all six emotions examined. We also explored the relationship between emotion recognition performance and the ability of the same patients to experience transient happiness and sadness during a laboratory mood induction. We found some support for a relationship between sadness recognition and experience. Taken together, our results indicate that the ventromedial frontal lobe plays a crucial role in facial emotion recognition, and suggest that this deficit may be related to the subjective experience of emotion. ■

INTRODUCTION

The neurobiology of emotion is a topic of growing interest. Recent neuroscientific studies have investigated emotional memory (reviewed in LaBar & Cabeza, 2006; Phelps & Anderson, 1997; LeDoux, 1993), emotional experience and regulation (reviewed in Ochsner & Gross, 2005; Calder, Lawrence, & Young, 2001), emotion recognition (reviewed in Adolphs, 2002), and the relationships between these processes (e.g., Heberlein & Adolphs, 2007; Calder et al., 2001). This work has implicated several brain regions in the processing of emotional material in general. The subset of these regions which may be critical for emotion recognition in particular include the amygdala, insula, right somatosensory regions, basal ganglia, and various sectors of the prefrontal cortex (PFC).

Prefrontal Cortex Involvement in Emotion Recognition

Although PFC has frequently been implicated in the processing of emotional faces, the details have not been consistent: Studies have focused on different regions of PFC, and there has been little consensus regarding the generality of impairment across emotions. Inconsistencies across studies may be due to differences in task requirements, stimuli used, specific emotions tested, differential involvement of PFC regions in the processing of specific emotions and, especially in the case of lesion studies with small sample sizes, premorbid individual differences.

Functional neuroimaging studies examining emotional face processing have not typically required subjects to label or rate emotions while being scanned, and thus, one cannot conclude that the activations observed are associated with recognition processes per se. Nonetheless, various frontal lobe regions have been active during a range of tasks involving emotional faces: the right anterior cingulate cortex (ACC) and bilateral inferior frontal gyri during facial expression matching (George et al., 1993); the lateral orbito-frontal cortex (OFC) and ACC when matching fearful and neutral faces, but the medial OFC when viewing *but not attending* fearful faces (Vuilleumier, Armony, Driver, & Dolan, 2001); the left ventral PFC and left ACC when holding in memory happy, as compared to neutral, faces (Dolan et al., 1996); the OFC and ACC when making gender discriminations between angry, but not sad, faces (Blair, Morris, Frith, Perrett, & Dolan, 1999); and the bilateral inferior frontal gyri (near the frontal operculum) during both observation and imitation of emotional faces (individual emotions not examined separately; Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003).

The involvement of OFC and ACC regions in processing angry and fearful faces, but not sad or neutral faces, may relate to the roles these areas play in the regulation of arousal (Öngür & Price, 2000): Anger and fear (and to a lesser extent happiness) are considered to involve

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higher arousal states than sadness or ''neutral'' emotion (Adolphs, 2002). Thus, OFC and/or ACC activation in emotional expression processing may reflect the subject's own arousal (presumably elicited either in response to or in a mirroring of the emotion expressed in the stimulus). In contrast, the involvement of frontal opercular areas in emotional face processing has been attributed to ''mirror-neuron''-like activity in these regions (Dapretto et al., 2006; Carr et al., 2003)—viz., a specific mechanism of simulation whereby the same neurons participate in the representation both of an observed action and of the same, self-generated one.

Loss-of-function methods, such as studies of people with focal brain injury or relatively focal neurodegenerative conditions, or following transient focal cortical inactivation with transcranial magnetic stimulation, can more directly address the question of the necessity of neural structures for specific processes. Existing studies examining facial emotion recognition after focal brain damage have implicated a range of frontal lobe regions. Table 1 summarizes specific regions of PFC damage associated with impairment on emotion recognition from facial expressions. Ventral and medial regions are implicated in most of the studies summarized. Comparisons across studies are made difficult by the range of emotion tasks used. In addition, some tasks that require emotion labeling without adjusting for difficulty or normal performance may find apparent impairments

Table 1. Summary of Lesion Studies Finding Effects of PFC Damage on Facial Emotion Recognition

Publication	*Task/Stimuli*	*PFC Regions Implicated*	*Specific Emotions Impaired*
Hornak, Rolls, & Wade, 1996	forced-choice (F-C) labeling emotional face photographs	OFC	not specified
Adolphs et al., 2000	F-C labeling emotional face photographs	left frontal operculum	not specified
Marinkovic, Trebon, Chauvel, & Halgren, 2000	F-C labeling emotional face photographs	right ventrolateral (change in performance measured pre- and postsurgery)	primarily fear; also sadness, disgust, neutral
Blair & Cipolotti, 2000	F-C labeling morphed emotional face photographs (morphs between emotions)	right ventral including the OFC	anger, disgust
Harmer, Thilo, Rothwell, & Goodwin, 2001	F-C labeling (emotion vs. neutral) morphed emotional faces (morphs between neutral & emotion)	dorsomedial via transcranial magnetic stimulation	anger (only anger and happiness tested)
Keane, Calder, Hodges, & Young, 2002	F-C labeling emotional face photographs	frontal variant FTD (ventromedial)	all emotions tested
Beer et al., 2003	free-response labeling emotional face photographs	OFC	self-conscious emotions; basic emotion recognition spared
Hornak et al., 2003	F-C labeling morphed emotional face photographs (morphs between emotions)	none in groupwise analysis; some individuals w/bilateral OFC or dorsomedial damage impaired	no clear pattern
Shamay-Tsoory, Tomer, Berger, & Aharon-Peretz, 2003	F-C labeling emotional face photographs	right dorsolateral and ventromedial	not specified
Fernandez-Duque & Black, 2005	F-C labeling emotional face photographs	frontal variant FTD (ventromedial)	negative emotions
Mah, Arnold, & Grafman, 2005	matching cartoon faces and gestures	ventromedial	not specified
Shaw et al., 2005	''eyes'' task, labeling social emotions and cognitive states from photographs of eye region of face	right dorsolateral and ventromedial	both social and cognitive mental states (basic emotions not included)

FTD = frontotemporal dementia.

on specific emotions because these expressions are inherently harder to recognize—for example, normal subjects generally have more difficulty recognizing fear and sadness, whereas happiness and disgust generally are easy to recognize (Russell, 1994).

The most detailed lesion study to date (Hornak et al., 2003) compared emotion recognition from face and vocal stimuli in groups with damage to the bilateral OFC, unilateral OFC, unilateral dorsomedial PFC or ACC, and dorsolateral PFC. Facial emotion recognition was tested using a task that required subjects to label emotional faces, including morphs between ''adjacent'' emotions (happiness and surprise; surprise and fear; fear and sadness; sadness and disgust; disgust and anger; anger and happiness). This study found no groupwise impairment on facial emotion identification, although some individuals were impaired. Those individuals who were impaired had either bilateral OFC or unilateral dorsomedial lesions, and did not show any clear pattern in the specific emotions affected. In contrast, there were detectable groupwise deficits in recognizing emotions from vocal stimuli in those with OFC or dorsomedial PFC damage. This dissociation is difficult to interpret: Is the OFC important only in voice emotion recognition? Was the facial emotion recognition task somehow less sensitive?

In summary, in loss-of-function and in functional imaging studies, ventral and medial regions of PFC are repeatedly implicated in facial emotion recognition. However, the wide variability in both methods and results leaves considerable uncertainty as to the specific role of this region.

Relationships between Recognition and Experience

How do we recognize emotions? Recent research suggests that at least part of the process involves representing the observed emotion expression in one's own emotion-experience and/or emotion-expression neural substrates, a process known as *simulation*, which is a feature of *shared-substrates* models of emotion recognition. In support of such a process, many of the structures that are known to be important for emotion recognition also appear to be involved in other aspects of emotion processing (Heberlein & Adolphs, 2007; Morrison, 2007; Adolphs, 2002, 2006; Goldman & Sripada, 2005; Calder et al., 2001). For example, the insula and basal ganglia play important roles in both the experience and the recognition of disgust (Calder, Keane, Manes, Antoun, & Young, 2000). Simulation models propose that the neural substrates for emotional experience and for the recognition of emotion in others are at least partially overlapping: Internal representations of others' emotional behavior and of our own behavior and feelings are posited as critical for recognizing the emotional significance of others' nonverbal emotional expressions.

The insula, amygdala, and right somatosensory cortices have figured prominently in simulation models of emotion recognition. However, there is some suggestive evidence that regions of PFC are also involved in both emotion recognition and emotional experience, suggesting a role in simulation-based emotion recognition: Some functional imaging studies have implicated regions of PFC, particularly the ventromedial and/or OFC, in emotional experience (Ochsner et al., 2004; Phan, Wager, Taylor, & Liberzon, 2002; Schaefer et al., 2002; Damasio et al., 2000), and, as reviewed above, separate work has implicated similar areas in emotion recognition. Interestingly, a recent paper has also provided evidence for ventromedial PFC involvement in simulation-based models of mental state attribution (Mitchell, Banaji, & Macrae, 2005).

Although findings of similar patterns of activation during the experience and recognition of emotion are consistent with simulation models, they are far from conclusive. Loss-of-function studies can test whether a given neural region is necessary for recognition processes, experience processes, both, or neither. If the ventral medial PFC (VMF) is involved in emotion recognition by virtue of its role in simulating emotional experience, then damage to that region should lead to deficits in both the recognition and experience of a given emotion. (However, existing models do not specify the form these deficits might take. For example, should emotion recognition correlate with changes in transient emotional experience, mood, or affective traits?) Existing lesion studies have provided support for both parts of this prediction, albeit of a piecemeal and somewhat inconsistent nature, and using a variety of experience measures (Roberts et al., 2004; Beer, Heerey, Keltner, Scabini, & Knight, 2003; Hornak et al., 2003; Rule, Shimamura, & Knight, 2002). However, there is only preliminary support for the conjunction of these two, and this is in the medial, but not the ventral, PFC: Hornak et al. (2003) found both changes in self-reported sad mood experience and deficits recognizing sadness from vocal expressions consequent to anterior cingulate and/or dorsomedial PFC damage. Their experience measure consisted of a questionnaire asking whether participants had noticed changes in their experience of each of several emotions since their brain damage was acquired. Damage to other PFC regions, including the OFC, did not result in the same overlap in deficits, nor in other conjunctions of experience and recognition performance.

The Current Study

In the present study, our primary goal was to provide a clear characterization of the role of human ventromedial PFC in facial emotion recognition. To that end, we used a sensitive facial emotion-recognition task to examine the performance of subjects with ventromedial frontal brain damage, compared to a group with dorsal and/or

lateral frontal brain damage, and to demographically matched healthy control subjects. The task we used features morphs between an emotionally neutral face and an emotional expression posed by the same individual (Adolphs & Tranel, 2004; Adolphs, Jansari, & Tranel, 2001; Jansari, Tranel, & Adolphs, 2000). These morphs are significantly more difficult to identify than fully posed emotional expressions (Jansari et al., 2000); further, the specific morphs we used were chosen because they are recognized at levels between floor and ceiling by normal subjects. In addition, this task requires participants to rate each face on scales for the presence of all of the basic emotions tested. This rating system has been shown to identify impairments that are not seen in forced-choice labeling tasks (Adolphs et al., 1999). Furthermore, the inclusion of ratings on multiple emotions for each stimulus provides a broader picture of the emotion perception ''space'' of a subject than do labeling tasks, in that it allows for examination of the relationships between emotion categories.

Our secondary goal was to perform an exploratory analysis of the relationship between this sensitive emotion-recognition task and an on-line, laboratory measure of transient emotional experience. The same PFC-lesioned subjects participated in a study of emotional experience and regulation, in which self-report and objective measures of emotion experience were collected during a mood induction. In addition, they completed an index of dispositional affect. This design permitted assessment of the effects of frontal lobe damage on emotion recognition and two measures of emotional experience in the same subjects, allowing us to test whether regions of PFC are shared substrates for these two aspects of emotion. Here we report correlations between the emotion recognition and experience measures; details of the emotion induction second study are reported separately (Gillihan et al., submitted).

METHODS

Participants

Participants included 15 subjects with damage to PFC, divided a priori into two subgroups approximately following the boundaries laid out in Stuss and Levine (2002): 7 subjects with damage primarily involving the medial orbito-frontal and/or VMF and 8 with frontal lobe damage primarily involving dorsal and/or lateral prefrontal cortices (D/LF). In addition, we tested 16 age- and education-matched control subjects (CTRL). Subjects with prefrontal damage were recruited through the University of Pennsylvania Center for Cognitive Neuroscience patient database. All patients with focal damage principally involving the cortex anterior to the precentral sulcus (based on the most recent clinical computed tomography or magnetic resonance imaging available) were eligible to participate. As can be seen in Figure 1, the D/LF group was primarily composed of subjects with damage to the inferior and/or middle frontal gyrus. In two D/LF cases, damage extended into the adjacent anterior insula.

VMF damage was due to rupture of anterior communicating artery aneurysm in six cases, and bilateral anterior cerebral artery stroke in one. D/LF damage followed ischemic or hemorrhagic stroke in seven subjects, and resection of a low-grade glioma in one. Three

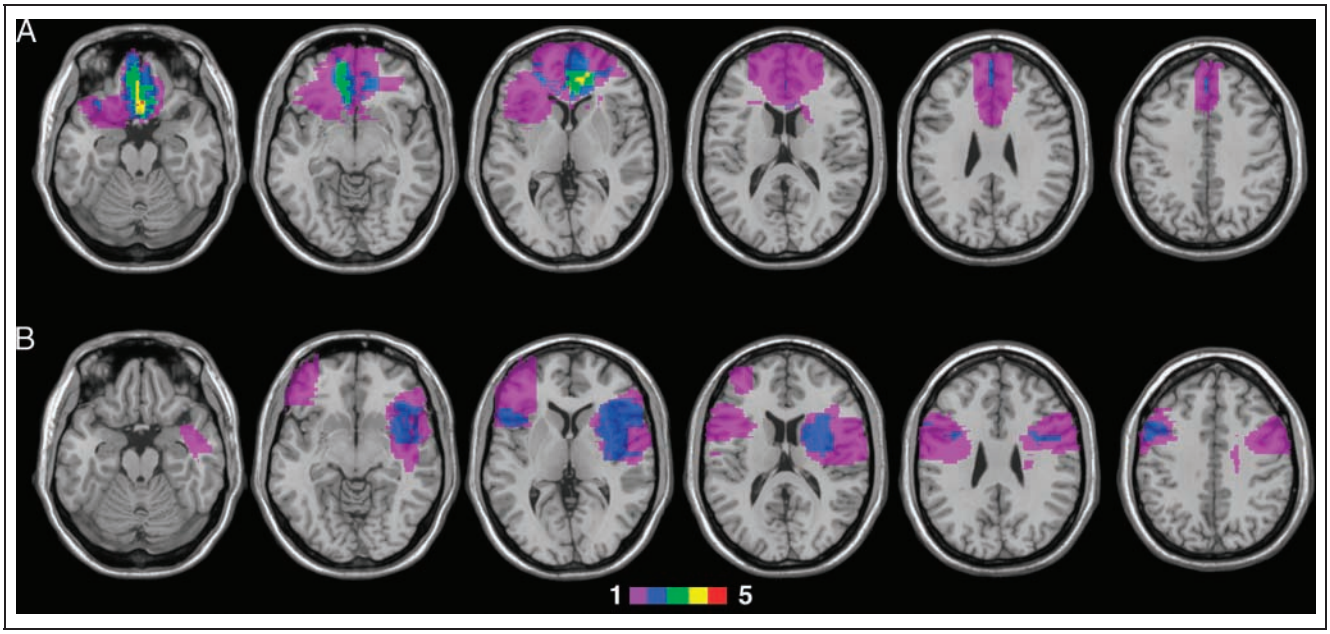

Figure 1. Overlaps of lesions for (A) the seven subjects with ventromedial prefrontal lesions (VMF group), and (B) the eight subjects with non-ventromedial prefrontal lesions (D/LF group). The number of subjects with lesions at each location is indicated by the color bar at bottom.

Table 2. Background and Demographic Information for All Three Groups

Group	*Age*	*Education*	*Sex (M:F)*	*IQ*	*BDI*	*Lesion Volume (cm^3)*
CTRL (n = 16)	56 (7)	14.8 (2.1)	4:12	125 (7.5)*	5.9 (4.2)*	–
VMF (n = 7)	53 (11)	13.0 (2.1)	3:4	110 (6)	15.7 (9.7)	22.1 (24.8)
D/LF (n = 8)	59 (10)	14.9 (2.5)	3:5	118 (11)	11.6 (8.3)	42 (38.2)

Numbers given are mean (*SD*).

*Indicates significant ($p < .05$) differences based on an ANOVA.

subjects in the D/LF group and one control were taking SSRIs at the time of testing. One subject in VMF group was taking methylphenidate and donepezil.

Healthy control subjects matched for age and education with the patient group were drawn from a database of individuals recruited via advertisements in the community. Controls had no history of major neurological or psychiatric disorders and scored >27 on the Folstein Mini-Mental status examination and <15 on the Beck Depression Inventory (BDI). All subjects provided written informed consent prior to participation in accordance with the Declaration of Helsinki, and were paid a nominal fee for their time. The Institutional Review Board of the University of Pennsylvania approved the study protocol. Demographic information for all participants is summarized in Table 2; results of selected neuropsychological screening tests administered to the patients are shown in Table 3. Lesion overlaps for both groups are shown in Figure 1.

Task and Stimuli

We used stimuli provided by Adolphs and Tranel (2004), described in previous studies from this group (Adolphs & Tranel, 2004; Jansari et al., 2000), which comprise series of morphs of a single individual's posed emotional faces from the Ekman and Friesen (1976) stimulus set. The stimuli were selected from a series of 19 linear morphs between a neutral expression and a fully posed emotional expression, for each of six basic emotions (anger, disgust, fear, happiness, sadness, surprise; see Figure 2 for examples). The specific stimuli chosen were selected to avoid floor and ceiling effects, based on a sample of normal subjects tested for this purpose (Jansari et al., 2000). Twenty-five faces were shown in total: two afraid, five angry, three disgusted, five happy, five sad, two surprised, and three neutral.

Subjects were presented with one face at a time and rated each in series, using 10-point (1–10) Likert scales. First, all 25 faces were rated on one emotion, then on the next, and so on for each of the six emotions. Thus, each subject saw the entire stimulus set six times. Emotions were always rated in the same order: fear, happiness, anger, surprise, disgust, and sadness. No time limit was imposed.

Data Analysis

Following Adolphs and Tranel (2004), we used two primary measures of emotion recognition performance. First, we calculated the difference between ratings given by each subject to each stimulus and that same subject's average rating of the three neutral stimuli. This procedure was intended to correct for any baseline biases in subjects' rating tendencies. The mean difference score was then calculated for each set of predefined emotional faces, collapsing across morph degree. This provided a simple summary measure of the detected intensity of each emotion, in each subset of emotional faces.

Second, we examined the ratings' sensitivity to the parametric increases in difficulty afforded by the incrementally morphed stimuli. We compared each brain-damaged subject's difference scores across all stimuli with the healthy control mean difference scores using a Pearson's correlation, thus quantifying how close a given subject's difference scores are to the difference scores given by healthy controls. This correlation is a global measure of correctness (i.e., agreement with the normal control group), which is affected both by ratings of the

Table 3. Results of Selected Neuropsychological Screening Tests for All Three Groups

Group	*Digit Span Forward*	*Animal Fluency*	*F Fluency*	*Trails B Errors*	*Reversal Learning Errors*	*Verbal Recall (5 min Delay)*
VMF	5.0 (0)	14.3 (5.3)	9.5 (3.3)	4.2 (3.6)*	9.2 (3.5)	3.6 (1.5)
D/LF	4.9 (0.4)	16.8 (3.5)	10.5 (3.0)	0.6 (0.5)	6.9 (2.2)	3.9 (0.9)

Not all subjects completed all tests.

*Indicates significant ($p < .05$) differences based on t tests.

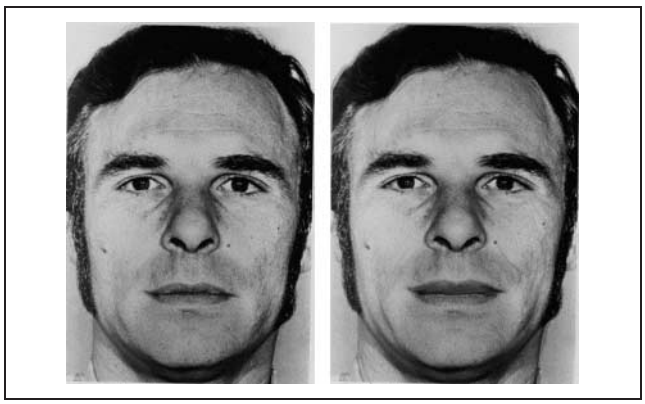

Figure 2. Examples of the morphed face stimuli. The figure on the left is a morph at step 2 of 19 steps between neutral and happy; the figure on the right is at step 5. Original images copyright Paul Ekman; morphs courtesy of Ralph Adolphs.

target emotion and by ratings of all five of the other emotions. It allows for the normal phenomenon of the confusability of certain emotions: For example, normal subjects detect some degree of sadness in the experimenter-defined ''angry'' stimuli. The correlation measure captures the degree to which those with frontal damage detect a given emotion across all stimuli, compared to the control group. These correlations were Fisher *z*-transformed, and all groupwise comparisons were carried out on the transformed correlations. We compared the average correlations for each group (VMF and D/LF), both across all stimulus ratings and for each emotion stimulus category separately.

Correlations with Experience Measures (Happiness and Sadness)

To the extent that regions within PFC are involved in simulation-based emotion recognition, we would expect PFC damage that affected one process to also affect the other. Thus, there should be a relationship between lesion-induced deficits in emotion experience and emotion recognition. We tested for such a relationship with two different emotion experience measures, one based on a laboratory mood induction, and the other based on subjects' dispositional affect, as measured by the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). We calculated correlations between the above ''normalness'' correlation scores and ratings derived from an emotion induction experiment carried out on the same day (described in detail in Gillihan et al., submitted). Briefly, subjects watched a 3-min movie clip intended to induce either happiness or sadness, and then rated their emotional state on a visual analog scale. Subjects next listened to music excerpts selected to induce either happiness or sadness, while focusing on a previously selected happy or sad autobiographical memory, and rated their emotional state on a visual analog scale after approximately 3 min of this recall. We calculated the averages of the two subjective ratings for each induction type (happiness and sadness) and subtracted from these averages the baseline (preinduction) mood ratings, as a measure of the ability to experience transient sadness and happiness. Because of the nonnormal distribution of these data, we calculated Spearman's rho to compare the emotion recognition and emotion induction measures. These correlations were calculated across the combined VMF and D/LF sample. For comparison, both of the above measures were also calculated for the healthy control subjects. For this purpose, ''normalness'' ratings were also calculated for each CTRL subject, by comparing individual emotional face ratings against those given by the rest of the CTRL group. Finally, to test for a relationship between dispositional affect and emotion recognition, we calculated Spearman's rho between the emotion recognition measure just described and PA and NA subscales of the PANAS. Participants rated the extent to which they generally experience these emotions; individuals with lesions were instructed to base their ratings on their emotional experience subsequent to their brain injury (for details, see Gillihan et al., submitted).

RESULTS

Each group's mean ratings of each emotion, for each stimulus face category, at each morph level included, are shown in Figure 3. (Mean ratings shown, as described in the Methods, are based on difference scores between the ratings given to each emotional stimulus face and the mean ratings of the three neutral faces, for each subject.) Two separate aspects of these data, one based on comparison of means and the other based on correlations, are considered in the following analyses.

Recognition of Different Emotions

To examine overall differences in emotional face ratings between the three subject groups across the six emotions, we performed a mixed-model analysis of variance (ANOVA) on the difference scores (rating of emotional stimulus − mean rating of neutral stimuli), with subject group (VMF, D/LF, CTRL) and emotion (anger, fear, happiness, disgust, surprise, and sadness) as factors. There were significant effects of group [$F(2, 28) = 6.75$, $p < .005$] and of emotion [$F(5, 28) = 2.29$, $p < .05$]; the interaction was not significant ($p > .15$). As shown by the generally lower intensity ratings along the entire bottom row of Figure 3, this analysis indicates that VMF subjects generally provided lower difference scores across all emotions. Because there was a significant effect of emotion in this first analysis, we next performed ANOVAs on ratings of each of the six face categories separately, an analysis that captures both the

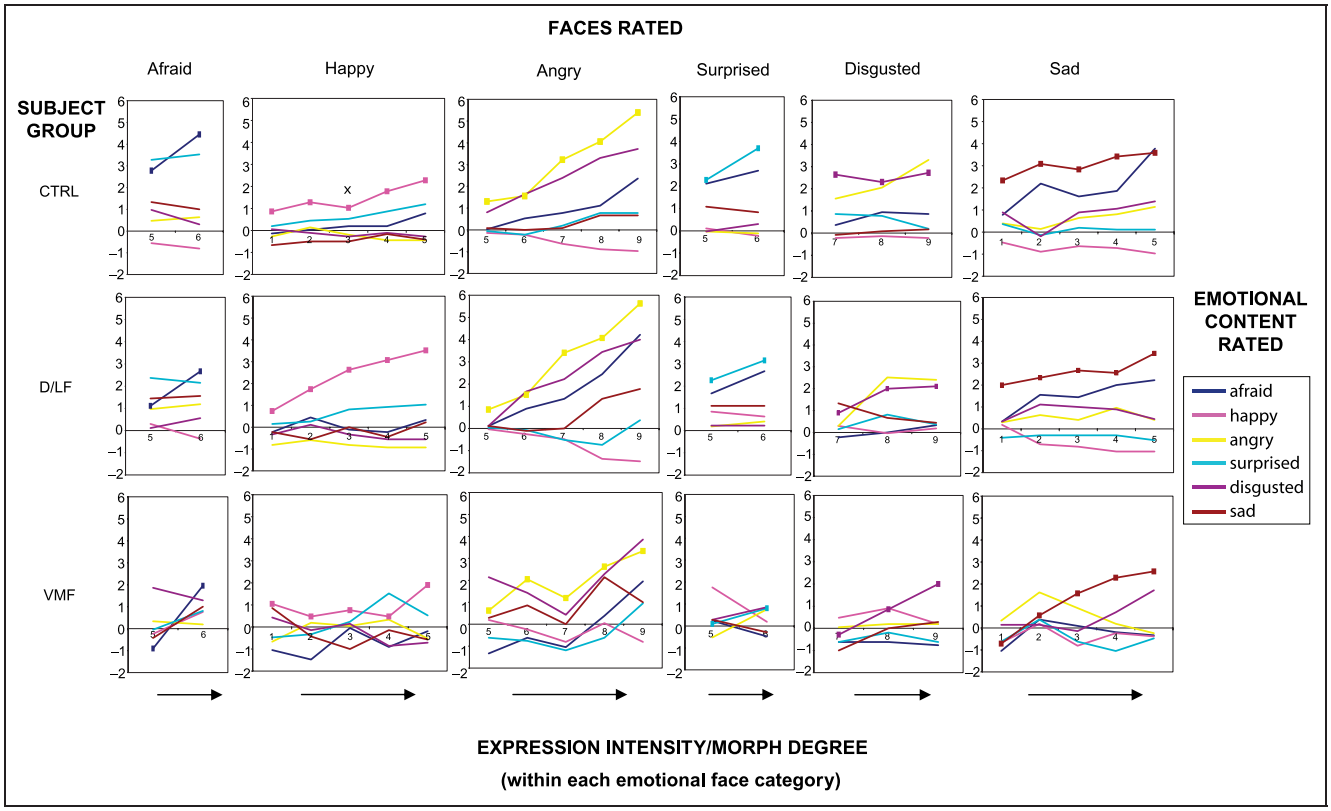

Figure 3. Ratings for each stimulus category. Each column corresponds to one stimulus face category, and each row corresponds to a subject group (CTRL, D/LF, VMF). In each graph, the *x*-axis represents morph degree, from most neutral on the left to most intense emotion on the right; the *y*-axis represents the rating assigned on Likert rating scales. Each colored line corresponds to ratings on one emotion scale (e.g., ratings of disgust in each face type are in purple; see legend). Lines for target emotion ratings only have square markers. All values are differences from average rating of neutral stimuli (see Methods/Results).

ability to correctly detect an emotion when present, and to correctly rate it as absent when absent, again collapsed across morph degree. Significant effects of group were found for three emotions: fear [$F(3, 28) = 6.6$, $p < .005$], surprise [$F(3, 28) = 5.4$, $p = .01$], and happiness [$F(3, 28) = 3.7$, $p < .05$]. Post hoc Student's–Newman–Keuls tests indicated that individuals with VMF lesions rated both fear and surprise faces as less intense (i.e., as more similar to their neutral ratings) than either the CTRL group or the D/LF group, whereas the D/LF group did not differ from the CTRL group on any of these three emotions. These differences are captured in Figure 3: VMF group's mean ratings of afraid (i.e., fearful) and surprised faces were lower across most emotions rated, whereas the D/LF group's ratings were generally similar to the CTRL group.

We next compared subjects' ratings of the three neutral faces on each of the six emotions to ensure that differences in ratings of emotional faces were not driven by differences in baseline ratings. In separate ANOVAs for each emotion rating given, collapsed across the three neutral faces, there were no main effects of group [Anger: $F(2, 28) = 1.78$, $p = .19$; Fear: $F(2, 28) = 0.43$, $p = .67$; Happiness: $F(2, 28) = 0.12$, $p = .89$; Surprise: $F(2, 28) = 2.29$; $p = .12$; Disgust: $F(2, 28) = 1.24$, $p = .30$; Sadness: $F(2, 28) = 0.72$, $p = .50$]. This implies that the group differences discussed above are not attributable to baseline biases in ratings of faces in general. Thus, VMF, but not D/LF, damage was associated with deficits in processing fearful and surprised faces.

Confusions and Sensitivity to Graded Changes in Emotional Expressions

Because it is not possible to determine objective "correctness" for the ratings we obtained, we simply compared the correlation between the mean CTRL difference scores on all emotion ratings across all morph levels to the difference scores for each patient individually. This yielded a measure which took into account both changes with degree of morph (i.e., CTRL ratings for the target emotion tended to increase with increasing morph degree) as well as confusions (ratings on other emotions remained the same or decreased). This correlation can thus serve as a "normalness" score, an overall measure of how well a subject derived emotion information from the morphed face stimuli. This measure is more sensitive to changes in ratings with increasing intensity of expression (i.e., degree of morph) than the ANOVA analyses above, and captures the similarity between the slopes of the lines across groups, shown in Figure 3. Across all stimuli in all emotion categories

(i.e., across all columns within Figure 3), average *Z*-transformed correlations with the CTRL group were significantly lower for VMF group than for the D/LF group (*t* test, two-tailed, $p < .005$; Figure 4A). We performed a similar analysis within each stimulus category (i.e., within each column of Figure 3) to assess the consistency of the rating abnormality, examining correlations between difference scores for all ratings of sad faces, all ratings of happy faces, and so on. For each stimulus category, ratings given by VMF-lesioned subjects were less well correlated with CTRL ratings than were D/LF-lesioned subjects' ratings (Figure 4B; Table 4). Correlations in VMF group were lowest for ratings of disgusted and surprised faces, but were also low for ratings of afraid, happy, and sad faces. In both frontal groups, ratings of fearful faces were least like CTRL ratings. Because of high correlations with CTRL ratings in the D/LF group but relatively low correlations with CTRL ratings in VMF group, ratings of angry and happy faces showed the largest differences between the two frontal groups (Table 4).

Table 4. D/LF and VMF Pearson Correlations with CTRL Ratings for All 6 Emotional Face Categories (All Correlations are *Z*-Transformed)

	D/LF Average	*VMF Average*	*Effect Size*
Afraid	.14	−.09	−0.62
Happy	.52	.08	−1.87
Angry	.72	.24	−1.72
Surprised	.37	−.2	−1.33
Disgusted	.33	−.13	−1.09
Sad	.43	.07	−1.12

Qualitative Differences in Emotion Ratings

In addition to these quantitative measures, qualitative examination of the patterns of ratings assigned to each category of emotional faces as they vary with emotion intensity or morph degree (Figure 3) provides further insights. Healthy controls assigned the highest intensity ratings to the correct emotion for most of the stimuli (e.g., rated morphed happy faces as higher on the happiness scale than on any other emotion scale), with few exceptions. D/LF participants' ratings were generally similar to those of controls; all confusions, or cases in which another emotion was rated higher than the correct emotion, were similar between the two groups, and in some cases, D/LF subjects even showed a greater distinction in the rating of the correct emotion as compared to the other emotions than did controls. In

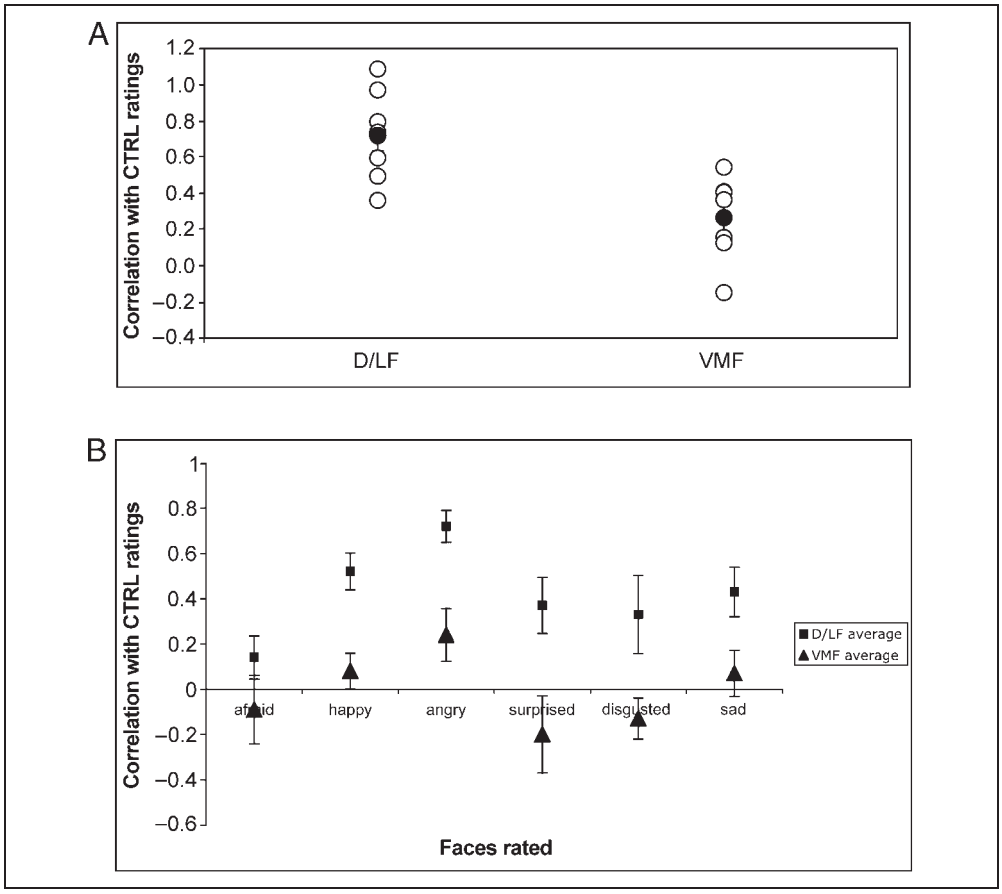

Figure 4. Pearson correlations with control ratings. (All correlations are *z*-transformed, leading to some values >1.0; see Methods.) (A) Individual (open circle) and mean (closed circle, ±standard error of the mean) correlations across all ratings of all stimuli. (B) VMF (triangles) and D/LF (squares) correlations for each category of stimulus faces.

contrast, VMF participants showed more general confusion for all six emotions tested, and showed different confusion patterns in some cases than did the other two groups. Nonetheless, at the highest morphed intensity level, four of the six emotions were rated most highly on the correct emotion by VMF group; such damage degrades, but does not obliterate, the ability to recognize emotion.

Correlations between Recognition and Experience: Dispositional Affect

Using *Z*-transformed correlations with CTRL ratings as a normalness measure, we tested for relationships between emotion recognition performance and dispositional affect as measured by the PANAS. None of the correlations between emotion recognition performance and corresponding trait affect measures (i.e., PA with happiness recognition and NA with sadness recognition, tested separately within each of the three subject groups) were significant. The only correlation which approached significance was between PA and happiness recognition in the D/LF group (Spearman's $\rho = .65$, $p = .08$, two-tailed).

Correlations between Recognition and Experience: Laboratory Mood Induction

Again using *Z*-transformed correlations with CTRL ratings as a normalness measure, we tested for associations between emotion recognition performance and self-reported sadness and happiness intensity during mood induction, as predicted by simulation models of emotion recognition. We found the predicted relationship between the intensity of transient experienced sadness and the normalness of sad face ratings in the frontal group as a whole (Spearman's $\rho = .53$, $p = .04$; Figure 5). However, this overall pattern was not detectable within the individual subgroups (VMF Spearman's $\rho = .29$, $p = .53$; D/LF Spearman's $\rho = .17$, $p = .69$; CTRL Spearman's $\rho = .26$, $p = .32$), likely due to limited statistical power. There was no consistent relationship between experienced happiness and happiness recognition either in the frontal group as a whole (Spearman's $\rho = -.28$, $p = .31$), or in controls (Spearman's $\rho = .19$, $p = .46$).

Subjects with Left Inferior Frontal Damage

Because lesion overlap studies (Adolphs, Damasio, & Tranel, 2002; Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000) and functional imaging studies (Carr et al., 2003; George et al., 1993) have implicated frontal operculum damage, especially on the left, in emotion recognition, we examined the performance of individual subjects who had damage including this region. Three D/LF subjects had lesions affecting the left inferior frontal gyrus (IFG); two of these subjects showed high correlations with healthy control ratings (Fisher *Z*-transformed *r*s = 1.09 and.80, respectively), whereas the third had a much lower correlation (Fisher *Z*-transformed $r = .49$). One subject with damage to the right IFG gave ratings that were very poorly correlated with those given by healthy controls (Fisher *Z*-transformed $r = .36$).

In other words, two subjects, one with left and the other with right-sided damage, gave abnormal ratings to

Figure 5. Correlations between emotion induction and recognition measures. (A) The *x*-axis represents the correlation between each subject's ratings of all happy stimuli and the control ratings of the same stimuli; the *y*-axis represents the mean intensity of self-reported happiness after happy mood induction. (B) The *x*-axis represents the correlation between each subject's ratings of all sad stimuli and the control ratings of the same stimuli; the *y*-axis represents the mean intensity of self-reported sadness after sad mood induction.

emotional faces, but two others, both with left-sided damage, were normal. Because our study was not designed to address the role of this region, we are not able to draw definitive conclusions based on our data, beyond the observation that IFG damage does not play an obligatory role in emotion recognition from facial expressions.

DISCUSSION

Emotion Recognition Measures

As a group, VMF subjects, but not subjects with D/LF damage, were impaired at recognizing emotions from facial expressions, as measured by a sensitive test of facial emotion recognition. This deficit was observed both in groupwise analyses of mean ratings across different intensities of emotion expression, and in the correlations of ratings given by members of each patient group with the mean ratings of the healthy control group.

VMF damage affected recognition of all emotions studied, although the extent of detectable impairment varied by emotion. VMF subjects rated both afraid and surprised faces as less intense, across emotions rated, than did controls or those with D/LF damage. Comparisons of ratings given to neutral faces indicated that these results were not due to baseline biases, or to different general response sets between the groups. The more sensitive correlation measure, which takes into account the ability to detect parametric changes in emotional intensity as well as the extent to which confusion across emotions parallels that seen in normal controls, suggests that VMF deficits in emotion recognition are pervasive: D/LF ratings of the faces were better correlated with control ratings than were VMF ratings across all emotions. VMF group diverged from the controls most for disgusted and surprised faces. However, the D/LF group's comparatively lower scores on those stimuli and comparatively higher scores on angry and happy faces resulted in the greatest differences between D/LF and VMF scores on happy and angry faces.

Because the mean difference and correlation analyses addressed different aspects of subjects' ability to derive information from emotional faces, they both are informative: Subjects with VMF lesions perceive emotion abnormally when the cues are fearful, sad, disgusted, or surprised faces. VMF group had lower mean intensity ratings of fearful and surprised faces than did controls, and VMF group's ratings of disgusted and surprised faces were very poorly correlated with the ratings given by controls. In addition, their ability to detect happiness and anger was worse than that of individuals with D/LF damage.

Although the general pattern of impairment across multiple emotions is consistent with previous reports in the literature, two features are notable: First, despite hypotheses that VMF involvement in emotion recognition was related to processing of arousal information, VMF-lesioned subjects in the present study were impaired on two low-arousal emotions, sadness and disgust. And second, although Hornak et al. (2003) report that sadness was the only facial expression which *none* of their frontal lobe-lesioned subjects had trouble recognizing, the present VMF subjects' ratings of sad faces differed significantly from those of healthy controls. This discrepancy presumably is due to the difference in tasks: had we used a labeling task such as that used by Hornak and colleagues, VMF group may not have had detectable impairment on three of the five sad morphs, and thus, may not have differed statistically across this category of stimuli (extrapolated from the fact that the highest mean ratings across all rating scales were on the sadness scale for the 3 most emotionally intense morphs tested; see Figure 3). In addition, our morphs were between neutral faces and emotional ones, whereas in the task Hornak and colleagues used, the morphs were between two emotional faces (Calder, Young, Perrett, Hodges, & Etcoff, 1996; Sprengelmeyer et al., 1996). It may be the case that morphs that are primarily neutral are harder to recognize than morphs that are a blend of emotions, and thus, provide a more sensitive test of emotion recognition.

Relationship between Recognition and Experience Measures

We found no significant relationship between postlesion dispositional affect measures and ratings of either sad or happy faces. We did, however, find a significant correlation across the combined PFC-damaged group between sadness recognition and the intensity of sadness experienced during a multimodal laboratory mood induction: People who were more impaired in their ratings of sad faces also experienced less intense induced sadness. This effect appeared to be driven by VMF group, whose sadness recognition was significantly lower than that of the D/LF group. No such correlation was found for the one other emotion for which we examined such a relationship, happiness, although this may be because of ceiling effects in ratings of experienced happiness. Although at face value, this finding would seem to support a role for VMF in simulation, clarification of the following two points is necessary: First, this general relationship was not detectable within *either* VMF or the D/LF subgroup. This may be due simply to a sample size limitation, or may reflect the challenges of precisely measuring emotional experience; nevertheless, it raises the possibility that the correlation is due to group effects rather than to a discernable relationship between emotion recognition and experience at the individual level. Hornak et al. (2003) observed a relationship between sadness recognition deficits from vocal cues and changes in global self-reported sadness experience in everyday life, but no similar relationships for other emotions, and also no relationship for facial emotion recognition

deficits. This relationship was evident only in their subjects with dorsomedial prefrontal/anterior cingulate damage; other patient groups showed effects on one or the other measure, but not both (demonstrating that recognition and experience deficits after frontal lobe damage are dissociable). This experience measure is closer to the PANAS scores that we measured, and differs considerably from our laboratory mood induction measure of emotional experience: Self-reported changes in sadness experience in everyday life presumably capture lesion-induced changes in dispositional (''tonic'') affect, as compared to the changes in short-term emotion dynamics, including reactivity to and recovery from readily identifiable emotional stimuli that were measured in our mood induction paradigm. Nonetheless, it is notable that both Hornak et al.'s work and ours found relationships between sadness experience and recognition of sadness in others. Further work will be required to determine whether VMF areas should be added to the list of brain regions implicated in simulation models, and if so, whether this role is emotion-specific.

A second important consideration is the directionality of any relationship between the recognition and experience of sadness. It may be the case that susceptibility to the subjective experience of sadness depends in part on the awareness of sadness in others (certainly, our movie clip emotion induction technique relied on empathy with the movie's characters to arouse sadness in the subject). If one is less sensitive to another person's sadness cues, one may be less susceptible to emotion induction—and one may have fewer episodes of sadness oneself. Note that this hypothesis differs from the more usual simulation models, in which impaired ability to model emotional states in one's own internal representations results in deficits in both experience and recognition. In this case, the ability to feel the emotion is unimpaired, but sensitivity to others' emotional cues is impaired, and it is the lack of empathic sensitivity that results in fewer elicitations of emotional response.

Such an account would predict that emotion induction that relies on one's own memories or imagining oneself in emotional situations should be relatively spared. Because we used two induction techniques (viewing movie clips and thinking about autobiographical episodes), we can examine this question directly. In support of a role for VMF in processing sadness cues and therefore in ''resistance'' to only certain kinds of mood induction, the correlation between sadness recognition and sad mood intensity in VMF-lesioned subjects was higher when sadness was induced via viewing a movie clip (Spearman's $\rho = .50$) than when it was induced via viewing autobiographical memory (Spearman's $\rho = .32$). This discrepancy indicates that subjects who are worse at recognizing sadness also get less sad when viewing movies of sad people. Thus, impairments in recognizing an emotion may, in fact, lead to reduced intensity of experienced emotion, when the induction depends on sensitivity to others' nonverbal behavior. Further studies examining the effect of the ''contagiousness'' of different emotions across different induction techniques in PFC-lesioned subjects would be useful to test this hypothesis.

It is interesting to compare this account with recent models of the role of VMF in decision making. It has been proposed that VMF is important in representing the ''emotional'' aspect of risky or moral decisions (Fellows, 2004, 2007; Greene & Haidt, 2002; Bechara, Damasio, & Damasio, 2000). Damage to VMF is thought to reduce the emotional response to such decision scenarios, in turn, altering the decision-making process. One interpretation of our data is that damage to VMF degrades the ability to appreciate the emotional content of explicitly emotional stimuli (i.e., emotional faces), in turn, influencing the experience of the observer. It remains to be seen whether these two roles for VMF reflect the same underlying process.

Conclusions

A group of seven subjects with VMF damage was impaired, relative to healthy control subjects, in a sensitive task of emotion recognition using faces morphed between neutral and posed emotional expressions. This effect was specific to VMF damage; a group of eight subjects with damage to dorsal and/or lateral prefrontal regions was not impaired. VMF-lesioned group appeared to have particular difficulty in judging fearful, surprised, sad, and disgusted faces, but different methods of comparing ratings identified different specific emotions, and such analyses are limited by the problem of multiple comparisons in small samples.

We also observed a correlation between the transient experience of sadness in response to a mood induction, and the ability to process sad faces across individual subjects with PFC damage, as would be predicted by a ''shared substrate'' role for regions within PFC for both processes. However, this association could equally be due to decreased susceptibility to emotional contagion from other people's sadness due to a lowered sensitivity to the relevant nonverbal cues. This exploratory examination of the relationship between emotion recognition and experience suggests directions in which simulation models may need to be further refined: In particular, we suggest that the particular aspect or aspects of emotional experience that are shared with recognition need to be more clearly defined.

Acknowledgments

This work was supported by NIH R21 NS045074, CIHR MOP-77583, and by a CIHR Clinician–Scientist award to L. K. F. A. S. H. was funded by NIH T32-NS07413. S. J. G. was funded by NIH F31-MH-073363-01A1. M. J. F. was funded by NIH R21-DA01586, R01-HD043078, R01-DA14129, and R01-DA18913. We thank Hilary Gerstein for assistance with data collection, Ralph

Adolphs for providing the morphed emotional face stimuli, and Marianna Stark for assistance recruiting and scheduling patients.

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