Dissociable neural responses to facial expressions of sadness and anger

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Summary
Previous neuroimaging and neuropsychological studies have investigated the neural substrates which mediate responses to fearful, disgusted and happy expressions. No previous studies have investigated the neural substrates which mediate responses to sad and angry expressions. Using functional neuroimaging, we tested two hypotheses. First, we tested whether the amygdala has a neural response to sad and/or angry facial expressions. Secondly, we tested whether the orbitofrontal cortex has a specific neural response to angry facial expressions. Volunteer subjects were scanned, using PET, while they performed a sex discrimination task involving static grey-scale images of faces expressing varying degrees of sadness and anger. We found that increasing intensity of sad facial expression was associated with enhanced activity in the left amygdala and right temporal pole. In addition, we found that increasing intensity of angry facial expression was associated with enhanced activity in the orbitofrontal and anterior cingulate cortex. We found no support for the suggestion that angry expressions generate a signal in the amygdala. The results provide evidence for dissociable, but interlocking, systems for the processing of distinct categories of negative facial expression.

Keywords: amygdala; orbitofrontal cortex; sadness; anger; facial expression

Abbreviations: ANCOVA = analysis of covariance; rCBF = regional cerebral blood flow; SPM = statistical parametric mapping

Introduction
Facial expressions are non-verbal communicative displays which are crucial in social cognition, allowing rapid transmission of valence information to conspecifics concerning novel objects or environments (e.g. Klinnert et al., 1987; Kling and Brothers, 1992). Thus, infant monkeys and humans show fear of novel objects to which their mothers have expressed fear and disgust (e.g. Klinnert et al., 1987; Mineka and Cook, 1993). Sad and angry facial expressions also regulate social interactions. The display of sad expressions has long been linked to the inhibition of aggression and the elicitation of prosocial behaviour (e.g. Miller and Eisenberg, 1988; Eisenberg et al., 1989). Anger is displayed to curtail the behaviour of others in situations where they have broken the social rules or social expectations (e.g. Averill, 1982).

There is now reasonable agreement concerning the anatomical structures involved in processing expressions of fear and disgust. Thus, the amygdala has been implicated in the processing of fearful expressions both in neuropsychological and functional imaging studies. Patients with lesions to the temporal lobe, including the amygdala, often fail to recognize fearful expressions (e.g. Adolphs et al., 1994; Calder et al., 1996; Broks et al., 1998), though not always (Hamann et al., 1996). Functional imaging studies consistently report increased activation in the left amygdala on presentation of fearful expressions (Breiter et al., 1996; Morris et al., 1996; Phillips et al., 1997; Whalen et al., 1998). As regards disgust expressions, both basal ganglia and insula have been implicated (Sprengelmeyer et al., 1996; Gray et al., 1997; Phillips et al., 1997). Patients with Huntington’s disease, which initially affects the basal ganglia and caudate nucleus, show impaired recognition of the expression of disgust (Sprengelmeyer et al., 1996; Gray et al., 1997). However, a functional MRI study of subjects exposed to expressions of disgust failed to identify basal ganglia activation though it did reveal activation of the anterior insula (Phillips et al., 1997). The anterior insula has been implicated in responding to offensive tastes (e.g. Yaxley et al., 1988; Kinomura et al., 1994; Zald et al., 1998).

Much less is known about the anatomical structures implicated in processing expressions of sadness and anger.
The suggestion that the amygdala might be implicated in processing sad expressions comes from work with psychopathic patients. Several studies have observed selective reduced autonomic responses to sad expressions, relative to controls, in psychopathic patients (e.g. House and Milligan, 1976; Aniskiewicz, 1979; Chaplin et al., 1995; Blair et al., 1997). Psychopathy has been linked to amygdala dysfunction (e.g. Patrick, 1994; Blair and Frith, 1999); psychopathic individuals show impoverished aversive conditioning and reduced augmented startle reflexes similar to those seen in patients with amygdala lesions (e.g. Hare et al., 1978; Davis, 1986; Patrick et al., 1993; Bechara et al., 1995; LaBar et al., 1995; Angrilli et al., 1996; LeDoux, 1998). Given that psychopathic patients are hyporesponsive to sad expressions and the indications of amygdala dysfunction in this population, it is plausible to suggest amygdala involvement in processing of sad expressions. The suggestion that the amygdala is implicated in processing sad expressions has received recent support from two studies by Phelps and colleagues. In the first, patients with right, but not left, amygdaloid lesions were impaired in the ratings of sad, fearful and disgusted expressions (Anderson and Phelps, 1997). In the second, a patient with bilateral amygdaloid lesions due to left amygdaloid gliosis and right temporal lobectomy showed a similarly selective impairment for sad, fearful and disgusted expressions (Anderson and Phelps, 1998).

There is a paucity of work focusing on the neural substrates which process angry expressions. Two candidate structures are the amygdala and orbitofrontal cortex. The suggestion of amygdala involvement is prompted by knowledge that this structure is crucial for processing threatening stimuli (e.g. Davis, 1986; LeDoux, 1998). Angry expressions can be thought of as threatening stimuli. In addition, at least some patients with amygdala lesions have been reported to show impaired anger recognition (Calder et al., 1996; Scott et al., 1997). Thus, Calder and colleagues (Calder et al., 1996) report that patient D.R. showed a recognition impairment for angry and fearful (and disgusted) expressions. Similarly, Scott et al. (1997) report that the same patient was impaired in the recognition of the sounds of anger and fear and angry and fearful (and sad) tones of voice when words were being spoken. However, in this context, it should be noted that two further patients with amygdala lesions, S.E. and S.M., did not show impairment in the recognition of angry expressions (Adolphs et al., 1995; Calder et al., 1996). Similarly, three out of four post-encephalitic patients with amygdala lesions also did not show impairment in the recognition of angry expressions. In addition, the patient who did show an impairment in the recognition of anger expressions was also impaired in the recognition of happiness and sadness expressions (Broks et al., 1998).

A second suggestion is that the orbitofrontal cortex is involved in the neural response to anger expressions. The orbitofrontal cortex has been implicated in the recognition of emotional expressions (Hornak et al., 1996). In addition, the orbitofrontal cortex has been implicated in both animal and human lesion studies in behavioural extinction and reversal learning (e.g. Dias et al., 1996; Rolls, 1996). Angry expressions curtail the behaviour of others in situations where social rules or expectations have been violated and, in this sense, they are effectively used to terminate the on-going behaviour of others (e.g. Averill, 1982). Angry expressions may thus serve as a cue for behavioural extinction/reversal learning. On this basis, it is plausible that the orbitofrontal cortex may mediate the neural response to angry expressions.

In the present study, volunteer subjects were scanned using PET while they performed a sex discrimination task involving static grey-scale images of faces expressing varying degrees of sadness and anger (see Fig. 1). We tested two hypotheses: first, whether the amygdala has a neural response to sad and/or angry facial expressions; and secondly, whether the orbitofrontal cortex has a specific neural response to angry facial expressions.

Methods

Subjects

Thirteen male subjects (mean age 25 years 3 months) took part in the study which was approved by the ethics committee of the National Hospital for Neurology and Neurosurgery and ARSAC (UK). All subjects were healthy, with no past history of psychiatric or neurological illness, and were not on any medication. All subjects gave informed consent to participate in the study.

PET scan acquisition and analysis

Scans of the distribution of H15O were obtained using a Siemens/CPS ECAT EXACT HR+ PET Scanner operated in high sensitivity three-dimensional mode. Subjects received a total of 350 Mbq of H15O over 20 s through a forearm cannula. Images were reconstructed into 63 places, using a Hanning filter, resulting in a 6.4 mm transaxial and 5.7 mm axial resolution (full width was half the maximum). Each scanning window was of 90 s duration.

The data were analysed with statistical parametric mapping (SPM 96) software from the Wellcome Department of Cognitive Neurology, London. After initial realignment, mean PET images from each subject were scalp-edited and used as a template to edit all 12 individual PET images. Structural MRIs from each subject were co-registered into the same space. The scans were then transformed into standard stereotactic space (Friston et al., 1995a). The scans were smoothed using a Gaussian filter set at 12 mm full width at half maximum. The rCBF (regional cerebral blood flow) measurements were adjusted to a global mean of 50 ml/dl/min. A blocked (by subject) ANCOVA (analysis of covariance) model was fitted to the data at each voxel, with a condition effect for each level of emotional intensity, and global CBF as a confounding covariant. Predetermined...
contrasts of the condition effects at each voxel were assessed using a $t$-statistic, giving a statistic image for each contrast. The SPM approach brings together two well-established bodies of theory (the general linear model and the theory of Gaussian fields) to provide a complete and simple framework for the analysis of imaging data. The method of SPM data analysis is described in more detail elsewhere (Friston et al., 1995b).

**Experimental design**

Subjects viewed static grey-scale images of emotionally expressive faces taken from a standard set of pictures of facial affect (Ekman and Friesen, 1978). The faces depicted either sad or angry expressions. For each emotional category and individual face, a range of six intensity levels was produced by computer graphical manipulation (see Fig. 1). The 20, 40, 60 and 80% faces were interpolations created using computer morphing procedures (Perrett et al., 1994). These involved the shifting of the shape and pigmentation of the 0% (neutral) face towards the sad or angry prototype (100%). For each subject, separate scans were acquired for each intensity level, in a $2 \times 6$ (category by intensity) factorial experimental design.

For each presented face, subjects were simply required to make a sex classification (male or female) by pressing left or right response buttons. No explicit recognition or categorization of emotional expression was required during the scans. Post-scan debriefing confirmed that subjects were not aware that the implicit emotional variable was crucial in the experimental design.

During each scan, 10 photographs of faces were presented, one at a time, on a computer monitor screen. Each presentation lasted 3 s, followed by a 2 s interval in which the screen was blank. The 10 faces were of different individuals (five males and five females), but all had the same category and intensity of emotional expression. The faces of the same 10 individuals were used in all 12 scans, in a randomized order. The emotional category and intensity of the faces were varied systematically across scans. The order of presentation of sad and angry conditions was counterbalanced across subjects. The six different intensity levels were given in a counterbalanced order within and across subjects.

**Results**

**Behavioural tests**

The validity of the different emotional categories and ‘morphed’ intensity levels was confirmed using explicit rating and psychophysiological response. Thirty subjects (mean age 20 years 7 months) blind to, and independent of, the main study were asked to classify the expression category (sad, angry, neutral or other). The subject’s response was calculated as correct if it matched the dominant affect in the image. Thus, the correct response to the 0, 20 and 40% sad and angry expressions faces was neutral. The correct response to the 60, 80 and 100% sad and angry expressions was sad and angry, respectively. Ninety-six percent of the subjects'...
responses were correct. Subjects then rated the intensity of expression of the face on a seven point scale. These ratings correlated highly with the proportion of the sad or angry prototype in the morphed face (correlation coefficients $r = 0.93$ for sad, $r = 0.88$ for angry). The skin conductance responses of 15 separate subjects (mean age 21 years 2 months) were recorded as they viewed the facial affects. The amplitude of skin conductance responses in the 1–3 s interval following stimulus onset were recorded. These responses significantly correlated with the proportion of the sad or angry prototype in the morphed face (correlation coefficients: $r = 0.43$ for sad, $r = 0.38$ for angry; $P < 0.05$).

**Parametric analyses of brain activity**

The different conditions were weighted according to the proportion of the sad, angry and neutral prototypes in the presented faces. Contrasts were performed to reveal brain areas sensitive to the emotional intensity of the facial expressions. First, regions where there was a differential response to sad and angry faces were identified. Subsequently, regions within this subset of voxels were identified where activity increased as a function of the degree of sadness or anger expressed in the stimuli. With respect to sad faces, this analysis revealed significant activations within the left amygdala, right temporal pole, right inferior temporal gyrus and right middle temporal gyrus (see Table 1 and Fig. 2; $P < 0.001$, uncorrected). In other words, the enhanced neural responses in these regions correlated with the increasing intensity of the sad expressions. With respect to angry faces, the analysis revealed significant activations within the right orbitofrontal cortex and the anterior cingulate cortex bilaterally (see Table 1 and Fig. 3). Again the enhanced neural responses in these regions correlated with the increasing intensity of the angry expressions ($P < 0.001$, uncorrected).

The weighted contrasts described above could have obscured an amygdala response to angry expressions if the amygdala response to sad expressions was stronger than that to angry expressions. Consequently, we directly contrasted the two highest intensities of anger expression (80 and 100%) with the corresponding neutral conditions (0 and 20%). This contrast again revealed activity in the right orbitofrontal cortex and anterior cingulate cortex bilaterally (see Table 1; $P < 0.001$, uncorrected). However, there were no indications of significantly greater amygdala activity to the anger expressions than to the neutral expressions. Thus, the results of the direct contrast analysis were in accordance with those of the earlier weighted contrasts.

A final analysis focused on which neural regions conjointly responded as a function of increasing emotional intensity of both sad and angry expressions. In other words, this analysis identified regions with increasing activity to the intensity of emotional expression regardless of type. The analysis revealed significant activations of the right temporal pole and anterior cingulate cortex (see Table 1 and Fig. 4; $P < 0.001$, uncorrected).

**Discussion**

To our knowledge, this study is the first investigation that has described neural responses to sad and angry expressions. We found that the left amygdala and right inferior and middle temporal gyri responded to sad, but not angry, expressions. In contrast, the right orbitofrontal cortex responded to angry, but not sad, expressions. The anterior cingulate cortex and right temporal pole responded to both sad and angry expressions. Our data thus indicated dissociable neural substrates that differentially responded to these distinct emotional expressions. These dissociable neural patterns are in keeping with the suggestion that there are distinct neural systems which respond to basic emotion expressions (e.g.
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Fig. 2 A statistical parametric map (SPM) showing activation of the left amygdala and temporal pole in response to increasing intensity of sad expression. An uncorrected \( P \)-value of 0.001 was used as the threshold for the contrast of the sad with the angry conditions, which was also weighted for degree of stimulus intensity. Views of the brain are shown for orthogonal slices at the pixel of maximal activation within (A) the left amygdala \((x = -20, y = -10, z = -18)\) and (B) the right temporal pole \((x = 44, y = 22, z = -32)\). The significant areas of activation are displayed on mean MRIs produced from the co-registered structural MRIs from all 13 subjects.

Adolphs et al. (1996). However, the conjoint neural activation by both sad and angry expressions implies a degree of commonality in the processes engaged by both expressions. The possible significance of the neural structures identified in the processing of the expressions is discussed in detail below.

The design of the experiment involving presentation of faces and a sex discrimination decision is likely to engage numerous cognitive processes. These processes include knowledge retrieval: both knowledge required for the explicit task, the recognition of the face’s sex, and knowledge of the expression being displayed. In addition, the stimuli are likely to be encoded into declarative memory (allowing subsequent recall of the items displayed). Also, since we know that both sad and angry expressions evoke autonomic activity (e.g. Eisenberg et al., 1989; Blair et al., 1997), the systems that mediate these responses must be activated. However, it is important to emphasize that these processes are common across the two classes of stimuli and thus are unlikely to be confounds in any direct comparisons of the conditions. More critically, the contrasts utilized in the statistical analyses sought to identify regions which selectively respond to either increasing intensity of sad or angry expressions or the degree of expression intensity irrespective of emotion.

Amygdala

The left amygdala showed an enhanced response to sad expressions that correlated with expression intensity. This unilateral response in the left amygdala to sad expressions parallels earlier functional imaging studies on the neural response to fearful expressions (Breiter et al., 1996; Morris et al., 1996, 1998a; Phillips et al., 1997; Whalen et al., 1998). The fact that sad expressions activate the amygdala might be considered surprising in that patients with amygdala lesions have been found to be unimpaired in recognizing sad facial expressions (e.g. Adolphs et al., 1994; Calder et al., 1996). However, it should be noted that our task did not involve explicit expression recognition. It thus appears that left amygdala activation is part of a neural response to sad expressions even if it is not a prerequisite for naming sad expressions. Moreover, it should be noted that other emotional expressions have produced apparently contradictory results in neuroimaging and neuropsychological studies. For example, functional imaging studies have found a differential amygdala response to happy expressions (Breiter et al., 1996; Morris et al., 1996; Whalen et al., 1998). Yet patients with amygdala lesions are unimpaired in the recognition of happy expressions. In addition, two recent reports describe patients with amygdala lesions who have shown impairments in rating the intensities of sad, fearful and disgusted, but not other, expressions (Anderson and Phelps, 1997, 1998). The present findings therefore suggest that the amygdala might not simply be involved in the recognition of fearful expressions. The present findings are compatible with the notion that the amygdala responds to, at a minimum, sad and fearful expressions. Although an intact amygdala may not be a prerequisite for the recognition of sad expressions, we would predict that it was necessary for the activation of autonomic responses to this expression. By contrast, we would predict that patients with amygdala lesions should be able to generate autonomic responses to angry expressions.

Why might sad expressions activate the amygdala? Indeed, more generally, why might there be a differential amygdala response to sad and fearful expressions? It is now generally accepted that the amygdala has a crucial role in emotional learning (e.g. Davis, 1986; Bechara et al., 1995; LaBar et al., 1995; Buchel et al., 1998; LeDoux, 1998). One possibility is that sad and fearful expressions act as unconditioned stimuli for behaviour in primates. This would explain why...
Fig. 3 A statistical parametric map (SPM) showing the right orbitofrontal cortex and anterior cingulate cortex which were increasingly active in response to increasing intensity of anger expression. An uncorrected $P$-value of 0.001 was used as the threshold for the contrast of the angry with the sad conditions, which was also weighted for degree of stimulus intensity. Views of the brain are shown for orthogonal slices at the pixel of maximal activation within (A) the right orbitofrontal cortex ($x = 42, y = 42, z = -16$) and (B) the anterior cingulate ($x = 0, y = 38, z = 36$). The significant areas of activation are displayed on mean MRIs produced from the co-registered structural MRIs from all 13 subjects.

Infant monkeys and humans show fear of novel objects to which their mothers have expressed fear, a transfer of valence information thought to occur through fear conditioning (e.g. Klinnert et al., 1987; Mineka and Cook, 1993). Similarly, there have been suggestions that moral socialization is achieved through aversive conditioning where sad expressions serve as the unconditioned stimulus (Blair, 1995).

The initial hypothesis that the amygdala is involved in the response to sad expressions was generated through work with patients with psychopathy. Psychopathic individuals show reduced autonomic responses to sad expressions (e.g. House and Milligan, 1976; Aniskiewicz, 1979; Chaplin et al., 1995; Blair et al., 1997). Psychopathy may reflect early amygdala damage (Patrick, 1994; Blair and Frith, 1999). This is because psychopathic individuals show impairments in both fear conditioning and augmentation of startle reflex by visual
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Fig. 4 A statistical parametric map (SPM) showing the anterior cingulate and right temporal pole which were increasingly active in response to increasing intensity of both sad and anger expressions. An uncorrected P-value of 0.001 was used as the threshold for the weighted contrast of intensity for both the angry and sad conditions. Views of the brain are shown for orthogonal slices at the pixel of maximal activation within (A) the right temporal pole ($x = 40, y = 16, z = -34$) and (B) the anterior cingulate cortex ($x = 4, y = 54, z = 20$). The significant areas of activation are displayed on mean MRIs produced from the co-registered structural MRIs from all 13 subjects.

threat primes (e.g. Hare et al., 1978; Patrick et al., 1993). Both these impairments are associated with amygdala dysfunction (Davis, 1986; Bechara et al., 1995; LaBar et al., 1995; Angrilli et al., 1996; LeDoux, 1998). Thus, the present study not only provides data indicating that the amygdala responds to sad expressions, but also these findings are compatible with the suggestion that the psychopathic personality reflects early amygdala damage (Patrick, 1994; Blair and Frith, 1999).

In this study, we found no evidence that the amygdala responds to angry expressions. Yet two patients with amygdala lesions (D.R. and J.C.) have been reported as showing impairments in the recognition of anger (Calder et al., 1996; Scott et al., 1997; Broks et al., 1998). However, it should be noted that these two patients presented more generalized emotion recognition impairments: D.R. was also impaired in the recognition of sadness in vocal tone and disgust expressions, while J.C. was impaired in the recognition of happy and sad expressions. As far as we are aware, no
patients with selective impairments for anger recognition have been reported.

One final consideration is the observation that the amygdala response to sad expressions was lateralized to the left. Lateralized amygdala responses to facial expressions of emotion, specifically fear, have been described previously in a study where the task requirements were identical to those of the present study (Morris et al., 1996). The interpretation proposed for this lateralized response was that it might reflect the modulation of amygdala activity by left hemisphere systems such as those mediating language. Support for this conjecture was provided by a further study whereby the lateralization of the amygdala response was contingent upon reportability of the fear-inducing conditioned stimuli (Morris et al., 1998b). In this light, we would suggest that the lateralized response seen in the present study reflects the modulation of left hemisphere systems such as language on an amygdala response to sad facial expressions.

Inferior and middle temporal gyri

The observation that the right inferior and middle temporal gyri showed enhanced responses to sad expressions which correlated with expression intensity was of interest. Neuropsychological studies consistently have reported a right hemisphere dominance in the processing of facial emotion (e.g. DeKosky et al., 1980; Bowers et al., 1985; Kapcsak et al., 1989; Gur et al., 1994; Adolphs et al., 1996). Only very rarely have patients with lesions restricted to the left hemisphere shown expression recognition impairments (e.g. Young et al., 1993). It remains unclear which regions in the right hemisphere are particularly implicated in processing emotion expressions. However, the lesion analysis conducted by Adolphs and colleagues revealed ‘damage which includes the right inferior parietal cortex results in expression recognition impairments that correlate for most negative emotions, especially fear and sadness’ (Adolphs et al., 1996; p. 7682). The present study did not observe right inferior parietal activation. However, there were clear indications that right inferior and middle temporal gyri were implicated in mediating the response to sad expressions. This activity may reflect the functioning of a more extended right hemisphere neural system which Adolphs et al. (1996) propose is important in processing facial expressions.

The right inferior temporal gyrus activity found in the present study was proximal to face-related activity reported in two previous studies (Dolan et al., 1997; Morris et al., 1998a). In the Dolan et al. (1997) study, it was shown that perceptual learning of faces enhanced activity of the right inferior temporal regions. They suggested that this activity may reflect learning-related tuning of neural activity similar to that seen in monkeys (e.g. Li et al., 1993; Tovee et al., 1996). In the Morris et al. (1998a) study, this area and the right middle temporal gyrus were found to receive a greater contribution from the amygdala during the processing of fearful expressions. This led to the suggestion that the amygdala itself was modulating this activity, potentially as part of its role in emotional learning (see above). These results are of interest in the context of the present study given the putative functional similarities suggested above between the responses initiated by sad and fearful expressions.

Orbitofrontal cortex

The right orbitofrontal cortex showed an enhanced response to angry expressions which correlated with expression intensity. Previously, functional imaging studies have reported frontal cortex involvement in processing emotional expressions though none have implicated specifically the orbitofrontal cortex, nor specifically investigated responses to angry expressions (e.g. George et al., 1993; Phillips et al., 1997). Yet, studies of patients with orbitofrontal cortex damage have described impairments in emotion expression recognition (Hornak et al., 1996). Angry expressions are displayed to curtail the behaviour of others in situations where others have broken the social rules or social expectations. Anger is less likely to be displayed when the behaviour is novel and more likely to be displayed if the behaviour is repeated (Averill, 1982). Thus, anger expressions in others tend to elicit behavioural extinction/response reversal. The orbitofrontal cortex is known to be involved in the mediation of behavioural extinction and reversal learning (e.g. Dias et al., 1996; Rolls, 1996). We suggest that the orbitofrontal cortex response to angry expressions observed in this study is a reflection of a behavioural extinction/response reversal effect of these stimuli. We propose that the orbitofrontal cortex, when activated by anger expression stimuli, acts to suppress current behaviour either through an inhibition of current behaviour or by activation of alternative behavioural responses.

Anterior cingulate

Increasing sad and angry expressions co-activated the anterior cingulate cortex as well as producing differential responses in the amygdala and orbitofrontal cortex, respectively. Anterior cingulate cortex activity has been found in previous studies which have examined neural responses to emotional expressions and emotional stimuli more generally (George et al., 1993; Sergent et al., 1994; Lane et al., 1997a, b). In addition, anterior cingulate activity has been found to correlate significantly specifically with fearful expression intensity (Morris et al., 1998a). It has been suggested that this structure plays a crucial role in assessing the motivational content of internal and external stimuli and in regulating context-dependent behaviours (Devinsky et al., 1995). In addition, activations of the anterior cingulate cortex have been observed in functional imaging studies addressing a wide range of cognitive contexts including selective attention and memory (for a review, see Cabeza and Nyberg, 1997). Damage to the anterior cingulate may produce impairment in movement, emotion and attentiveness (e.g. Damasio and van Hoesen, 1983). Thus, the cognitive implications of this anterior
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Temporal pole

The other region engaged by both sad and angry expressions was the right temporal pole. While activation of this region was related to the degree of expression intensity, irrespective of emotion, it was also associated specifically with the degree of sadness intensity. Activation of this region has been associated with the recall of affect-laden autobiographical material (Fink et al., 1996) and also following administration of a major reinforcer, cocaine (Breiter et al., 1997). Thus, while the cognitive implications of this activation are unclear, it is possible that it may reflect the cueing of affect-laden autobiographical material. In line with this, there are behavioural data indicating that the induction of sadness influences autobiographical recall; specifically, facilitating the recall of ‘negative’ memories (Mineka and Cook, 1993).

Conclusion

The present study provides evidence of differential, but interlocking, neural responses to two ‘negative’ emotion expressions, sadness and anger. The neural response to sad facial expressions mirrors that to fearful expressions in that both activate the left amygdala. The neural response to angry facial expressions activates the right orbitofrontal cortex. Both sad and angry expressions activate the anterior cingulate and right temporal pole. The current evidence thus indicates that there is not a unitary system which responds to aversive stimuli but at least two systems which respond to different classes of aversive stimuli. One of these systems responds to facial stimuli (sad, fearful) involved in (social) aversive conditioning, while the second responds to anger and related stimuli which encourage behavioural extinction. Finally, the present finding that the left amygdala preferentially activates in response to sad expressions, coupled with previous findings of reduced responsiveness to sad expressions in psychopaths (in whom amygdala dysfunction has been linked), provides tentative support for the suggestion that the amygdala is a locus of dysfunction in those labelled psychopaths.

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