

Facial expressions, their communicatory functions and neuro-cognitive substrates

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Human emotional expressions serve a crucial communicatory role allowing the rapid transmission of valence information from one individual to another. This paper will review the literature on the neural mechanisms necessary for this communication: both the mechanisms involved in the production of emotional expressions and those involved in the interpretation of the emotional expressions of others. Finally, reference to the neuro-psychiatric disorders of autism, psychopathy and acquired sociopathy will be made. In these conditions, the appropriate processing of emotional expressions is impaired. In autism, it is argued that the basic response to emotional expressions remains intact but that there is impaired ability to represent the referent of the individual displaying the emotion. In psychopathy, the response to fearful and sad expressions is attenuated and this interferes with socialization resulting in an individual who fails to learn to avoid actions that result in harm to others. In acquired sociopathy, the response to angry expressions in particular is attenuated resulting in reduced regulation of social behaviour.

Keywords: facial expressions; amygdala; communication; psychopath; autism

1. INTRODUCTION

Facial expressions are a crucial component of human emotional and social behaviour and are believed to represent innate and automatic behaviour patterns (Darwin 1872). The purpose of this paper is to consider facial expressions: the stimuli that elicit their presentation, the neuro-cognitive systems necessary for their production, the neuro-cognitive systems that interpret the expressions produced by others and the conditions under which the interpreter may respond to the emoter thus closing the communicatory loop. To do this, I will make one fundamental assumption: that facial expressions of emotion do indeed have a communicatory function, and that they impart specific information to the observer. Thus, the suggestion will be that expressions of fearfulness, sadness and happiness are reinforcers that modulate the probability that a particular behaviour will be performed in the future. Indeed, fearful faces have been seen as aversive unconditioned stimuli that rapidly convey information to others that a novel stimulus is aversive and should be avoided (Mineka & Cook 1993). Similarly, it has been suggested that sad facial expressions also act as aversive unconditioned stimuli discouraging actions that caused the display of sadness in another individual and motivating reparatory behaviours (Blair 1995). Happy expressions, in contrast, are appetitive unconditioned stimuli which increase the probability of actions to which they appear causally related (Matthews & Wells 1999). Disgusted expressions are also reinforcers but are used most fre-

quently to provide information about foods (Rozin *et al.* 1993). Displays of anger or embarrassment, it is argued, do not act as unconditioned stimuli for aversive conditioning or instrumental learning. Instead, they are important signals to modulate current behavioural responding, particularly in situations involving hierarchy interactions (Blair & Cipolotti 2000; Keltner & Anderson 2000).

In contrast to the communicatory function assumption, there have been suggestions that emotional expressions are automatic displays that occur as a function of the emotional experience of the individual (Darwin 1872; Buck 1984; Izard & Malatesta 1987; Ekman 1997). According to these authors, although the expression may impart information to observers, the transmission of information is not their function. Instead, the expression is an automatic consequence of the individual's experience (Ekman 1997). However, the empirical literature does not indicate that individuals display emotional expressions automatically as a function of the degree to which they feel a particular emotion (Fridlund 1991; Camras 1994). Instead social context predicts probability of emotional expression in humans as it does probability of non-verbal displays in non-human species (Cheney & Seyfarth 1980; Hinde 1985). Thus, participants smile more at a humorous video or show greater distress to the sound of an individual in distress if they are together with another rather than if they are alone (Chovil 1991; Fridlund 1991). Similarly, infant smiling from the age of 10 months is almost entirely dependent on visual contact with the caregiver: without such contact the infant is very unlikely to smile (Jones & Raag 1989; Jones *et al.* 1991).

Importantly, the argument here is not that the display of an emotional expression implies intent to convey a specific message to the observer. The argument is simply that

One contribution of 15 to a Theme Issue 'Decoding, imitating and influencing the actions of others: the mechanisms of social interaction'.

emotional expressions serve a communicatory function that they have evolved so that information on the valence of objects/situations can be transmitted rapidly between conspecifics. Thus, important triggers for an emotional display include both an emotional event and also a potential observer. If there is no observer, the emotional display will either not occur or be considerably muted.

A particularly clear illustration of the communicatory function of emotional expressions can be seen after an infant's discovery of a novel object. The infant will look towards the primary caregiver and their behaviour will be determined by the caregiver's emotional display. If the caregiver displays an expression of fear or disgust, the child will avoid the novel object. If the caregiver displays a happy expression, the child will approach the novel object. This process is known as social referencing and is seen in children from the age of eight to ten months (Klunnert *et al.* 1983, 1987; Walker-Andrews 1998). Interestingly, comparable social referencing is seen in chimpanzees (Russell *et al.* 1997) and a very similar process has been shown in other monkeys and labelled observational fear (Mineka & Cook 1993).

Mineka characterizes the process of observational fear within an aversive conditioning framework (Mineka & Cook 1993). The US is the mother macaque's expression of fear, which she shows to the CS, the novel object. This maternal fearful expression, the US, elicits an unconditioned response, a fearful reaction, in the infant monkey. Pairing of the US with the CS, the novel object, allows the CS to elicit a conditioned response; the infant monkey comes to show a fearful reaction to the novel object.

A simple conditioning approach is, however, unlikely to be appropriate in humans. In humans, the representation of the emoter's intent has been shown to be crucial. Indeed, the learning of valences for novel objects can be thought of similarly to the learning of names for novel objects. When hearing a new word, children do not automatically associate this word with whatever novel object is in their immediate field of view. Instead, they turn towards the speaker, calculate the object that they are attending to, and associate the new word with this novel object (Baldwin *et al.* 1996; Bloom 2002). Similarly, during social referencing, if the child is attending to one object when the caregiver displays an emotional response to another, the child will look at the caregiver to determine the direction of their attention. The child will then form the appropriate association between the information communicated by the caregiver's expression and the object to which the caregiver had been attending (Moses *et al.* 2001). Thus, the communication of valence to objects, like the communication of names to objects, involves association of the affective information with a CS that corresponds to the communicator's referent.

2. THE PRODUCTION OF EMOTIONAL EXPRESSIONS

The suggestion developed above is that emotional expressions are communicatory signals that function to convey valence information rapidly to conspecifics. Specifically, they are particularly likely to be elicited under conditions when there is an emotional stimulus in the environment and there is an audience to perceive the

expression. But emotional expressions are not automatically elicited under these conditions. Individuals are capable of intentionally manipulating their emotional displays, they may follow 'display rules', societal proscriptions as to what emotion should be displayed in given circumstances and how intensely it should be displayed (Ekman & Friesen 1969). Indeed, one major task faced by the child in middle childhood is to learn the culture's display rules governing the conditions that are appropriate for the display of specific emotions. In a classic study of the development of display rules and control over emotional expressions, age-related changes were demonstrated in the ability of children to cover their disappointment at the discovery that their gift for helping out an adult was much less interesting than the gift they had been expecting; the disappointment of the younger children was far easier to detect (Saarni 1984).

There is thus a suggestion of spontaneous or over-learned emotional expressions to emotional stimuli in the presence of observers as well as controlled or posed emotional expressions as a function of display rules. It has been argued that the neuropsychological data about the production of emotional expressions echo this dichotomy (Rinn 1984; Hopf *et al.* 1992). Thus, it has been claimed that sub-cortical regions are necessary for spontaneous emotional displays but not controlled ones, whereas cortical regions are necessary for controlled emotional displays but not automatic emotional displays (Rinn 1984). However, this strict dichotomy overstates the empirical picture. Thus, investigations of patients with Parkinson's disease and other patients with damage to the basal ganglia report marked reductions in the production of spontaneous emotional expressions; such patients show reduced displays of emotional expressions when watching emotionally arousing videos relative to comparison individuals (Borod *et al.* 1990; Pitcairn *et al.* 1990; Weddell 1994; Smith *et al.* 1996). However, such patients also show some impairment in the production of posed emotional displays, though to a lesser degree (Borod *et al.* 1990; Weddell 1994; Smith *et al.* 1996). Similarly, there have been reports that lesions of frontal cortex impair the ability of the patient to pose emotional expressions but spare the production of spontaneous emotional expressions (Hopf *et al.* 1992). However, other studies find significant impairment in the production of both posed and spontaneous emotional expressions in patients with frontal cortex lesions (Weddell *et al.* 1988, 1990; Weddell 1994).

The data therefore suggest that sub-cortical regions, in particular basal ganglia, and cortical regions, particularly frontal cortex, are involved in both the production of spontaneous and controlled emotional displays. A schematic of regions known to be involved is presented in figure 1. Basal ganglia and frontal cortex are represented as reciprocally interconnected such that damage to either structure impairs the production of emotional expressions. The greater output from the frontal cortex represents the fact that while frontal cortical lesions cause significant impairment to both the production of spontaneous and controlled expressions (Weddell *et al.* 1988, 1990; Weddell 1994), lesions to the basal ganglia disproportionately affect the production of spontaneous expressions (Borod *et al.* 1990; Weddell 1994; Smith *et al.* 1996). Frontal

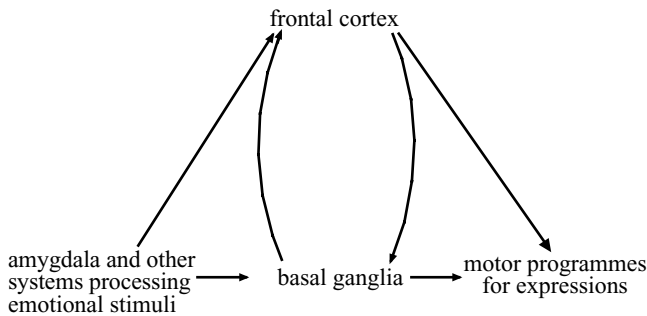


Figure 1. A schematic of regions known to be involved in the production of emotional expressions.

cortex is likely to be crucial for representing goals to either show or suppress an emotional expression. The basal ganglia receives inputs from both the amygdala and other structures processing emotional information. Although amygdala lesions do reduce the display of spontaneous fearful displays to novel objects (Prather *et al.* 2001), they do not affect the production of controlled fearful or other emotional displays (Anderson & Phelps 2000).

3. RESPONDING TO THE EMOTIONAL EXPRESSIONS OF OTHERS

Two dissociable routes have been shown to be involved in processing fear conditioning (Armony *et al.* 1997; LeDoux 2000). Thus, information on conditioned stimuli during auditory fear conditioning can be mediated by projections to the amygdala from either the auditory thalamus or auditory cortex (LeDoux *et al.* 1984; Romanski & LeDoux 1992*a,b*; Campeau & Davis 1995). Analogously, there have been suggestions that information on the emotional expressions of others can be conveyed either by a sub-cortical pathway (retinocollicular–pulvinar–amygdalar) or by a cortical pathway (retinogeniculostriate–extrastriate–fusiform) (de Gelder *et al.* 1999; Morris *et al.* 1999; Adolphs 2002).

The suggestion is that the sub-cortical pathway is fast and allows immediate automatic access of information on emotional expressions to the amygdala that can then modulate the processing of information through the cortical pathway (Pizzagalli *et al.* 1999; Adolphs 2002). In support of a sub-cortical pathway, positive covariations of cerebral blood flow (as measured by positron emission tomography imaging) have been demonstrated in the pulvinar, superior colliculus and amygdala in response to masked facial expressions of anger that had been previously associated with an aversive stimulus (Morris *et al.* 1999). Visual masking is assumed to be a result of interference between the induction of neural activity by the stimulus and the mask, which occurs within the relatively slow response time of primary visual cortex neurons (Macknik & Livingstone 1998). Neurons in the superior colliculus are capable of responding to much more rapid changes in visual input and hence produce quite distinct responses to the facial expression and neutral mask. However, such responses fail to elicit conscious experience. Additional support for the suggestion of a sub-cortical pathway has been provided by work with G.Y., a patient with a long-standing right-sided hemianopia after occipital

lobe damage at the age of 8 years (de Gelder *et al.* 1999). This 'blindsight' patient showed some ability to discriminate (by guessing) between different facial expressions in his blind hemifield. Later neuro-imaging work with G.Y. demonstrated differential amygdala responses to fearful versus happy expressions when these were presented to both the blind and seeing hemifields. However, striate and fusiform activity only occurred in response to stimuli presented to the seeing hemifield. In addition, amygdala responses to fear conditioned faces exhibit condition-specific covariations with neural activity in the posterior thalamus and superior colliculus (Morris *et al.* 2001).

The cortical route involves regions of occipital and posterior temporal visual cortex (Haxby *et al.* 2000, 2002). In particular, neuro-imaging studies have indicated that three specific areas are involved in face processing: the lateral occipital gyri, bilateral regions in the lateral fusiform gyrus and the posterior superior temporal sulcus (Kanwisher *et al.* 1997, 2000; Haxby *et al.* 1999). Moreover, there are strong suggestions of a dissociation in function between the fusiform gyrus and superior temporal sulcus (Hasselmo *et al.* 1989; Hoffman & Haxby 2000). The suggestion is that the fusiform gyrus is more involved in the processing of facial identity whereas the superior temporal sulcus is more involved in the processing of social communication (Haxby *et al.* 2002).

Recent event-related potential and magnetoencephalography studies have allowed considerable specification of the time-course for the processing of emotional expressions (Pizzagalli *et al.* 1999, 2002; Streit *et al.* 1999; Halgren *et al.* 2000). The earliest activity that discriminates between emotional facial expressions is seen in mid-line occipital cortex from between 80 to 110 ms post-stimulus (Pizzagalli *et al.* 1999; Halgren *et al.* 2000). From *ca.* 160 ms, activity is seen in the fusiform gyrus and superior temporal sulcus (Streit *et al.* 1999; Halgren *et al.* 2000; Pizzagalli *et al.* 2002). This literature has yet to find evidence of early amygdala activity that the sub-cortical route should predict. Indeed, the earliest activity seen is at *ca.* 220 ms in the right amygdala (Streit *et al.* 1999). However, there has been a report of neuronal discrimination, as single unit responses, between the emotions of fear and happiness after only 120 ms in the orbital frontal cortex of a patient (Kawasaki *et al.* 2001). This would suggest a sub-cortical route to orbital frontal cortex.

There appear to be further activations of superior temporal cortex after the amygdala activation (Streit *et al.* 1999), perhaps as a consequence of the amygdala activity. Indeed, a recent study examining single unit activity in the temporal visual cortex in monkeys found that information sufficient to distinguish different emotional expressions occurred *ca.* 50 ms after information sufficient to distinguish faces from other objects was available (Sugase *et al.* 1999). This again suggests the possibility that response to emotional stimuli in the temporal cortex is modulated by feedback from structures such as the amygdala (Adolphs 2002). Moreover, many imaging studies investigating the neural response to emotional expressions have reported greater superior temporal sulcus and fusiform gyrus activity to emotional expressions relative to neutral expressions (Phillips *et al.* 1998; Critchley *et al.* 2000; Iidaka *et al.* 2001). In addition, task conditions that increase attention to emotional expressions result in

increased superior temporal sulcus and fusiform gyrus activity (Narumoto *et al.* 2001; Vuilleumier *et al.* 2001; Pessoa *et al.* 2002).

Two additional cortical areas that have been linked to the processing of emotional expressions are bilateral regions of inferior frontal cortex and inferior parietal cortex. Three neuro-imaging studies have observed inferior frontal cortex activity to emotional expressions (George *et al.* 1993; Nakamura *et al.* 1999; Gorno-Tempini *et al.* 2001) although, it should be noted, many other studies have not. Activity in the inferior parietal cortex, or at least the proximal region of superior temporal sulcus, is frequently implicated in the processing of face stimuli (Haxby *et al.* 2000) and expression processing (Phillips *et al.* 1997; Streit *et al.* 1999; Halgren *et al.* 2000; Kesler-West *et al.* 2001; Pizzagalli *et al.* 2002). Moreover, two studies investigating which cortical regions, when damaged, most affected expression recognition stressed the importance of the inferior parietal cortex (Adolphs *et al.* 1996, 2000). These areas are of potential interest as proximal areas are activated when either an individual is initiating a movement or when they are observing another initiate the same movement (Iacoboni *et al.* 1999). This has prompted suggestions that responding to another individual's expression relies on the activation of motor programmes that the individual uses for the production of expressions (Preston & de Waal 2003).

As stated in the beginning of this paper, a fundamental assumption of this paper is that emotional expressions are communicatory signals that serve specific purposes. The claim is that this perspective allows an understanding into specific patterns of activation seen for specific emotions. Importantly, fearful, sad and happy expressions can all be viewed as reinforcers that modulate the probability that a particular behaviour will be performed in the future. The amygdala has been implicated in aversive and appetitive conditioning including instrumental learning (Killcross *et al.* 1997; Everitt *et al.* 2000; LeDoux 2000). It is thus unsurprising, given the suggested role of fearful, sad and happy expressions as reinforcers, that neuro-imaging studies, with a few exceptions (Kesler-West *et al.* 2001), have generally found that fearful, sad and happy expressions all modulate amygdala activity (Schneider *et al.* 1994; Breiter *et al.* 1996; Morris *et al.* 1996; Phillips *et al.* 1997, 1998; Baird *et al.* 1999; Blair *et al.* 1999; Drevets *et al.* 2000), though it should be noted that happy expressions have been reported to both increase and decrease amygdala activity (Breiter *et al.* 1996; Morris *et al.* 1996). The neuropsychological literature supports the neuro-imaging literature about the importance of the amygdala in the processing of fearful expressions. There have been occasional suggestions that amygdala damage leads to general expression recognition impairment but these reports are typically from patients whose lesions extend considerably beyond the amygdala (Rapcsak *et al.* 2000). Instead, amygdala lesions have been consistently associated with impairment in the recognition of fearful expressions (Adolphs *et al.* 1994, 1999; Calder *et al.* 1996; Schmolck & Squire 2001). Impairment in the processing of sad expressions is not uncommonly found in patients with amygdala lesions (Adolphs *et al.* 1999; Schmolck & Squire 2001). Indeed, a recent review of patient performance across studies, reported that *ca.* 50% of patients with

amygdala damage present with impairment for the recognition of sad expressions (Fine & Blair 2000). Amygdala lesions rarely result in impairment in the recognition of happy expressions (Adolphs *et al.* 1999; Fine & Blair 2000). However, this may reflect the ease with which happy expressions are recognized (Ekman & Friesen 1976).

Disgusted expressions are also reinforcers but are used most frequently to provide information about foods (Rozin *et al.* 1993). In particular, they allow the rapid transmission of taste aversions; the observer is warned not to approach the food that the emoter is displaying the disgust reaction to. Functional imaging studies have consistently shown that disgusted expressions engage the insula and putamen (Phillips *et al.* 1997, 1998; Sprengelmeyer *et al.* 1998) and patients with damage to the insula present with selective impairment for the recognition of disgusted expressions (Sprengelmeyer *et al.* 1996; Calder *et al.* 2000). Experimental investigations in macaques have shown that there is a primary taste cortical region in the anterior insula (Rolls 1997) and neuro-imaging studies in humans have also shown the insula to be involved in the representation of taste (O'Doherty *et al.* 2001*b*; Small *et al.* 2001). Crucially, insula lesions have been found to block the acquisition and expression of taste aversion learning (Cubero *et al.* 1999). Thus, the suggestion is that the disgusted expressions of others activate in particular the insula allowing taste aversion (disgust expression US–novel food CS associations) to occur.

In contrast to the expressions considered above, it is far less clear that the angry expression is a basic reinforcer. Angry expressions are known to curtail the behaviour of others in situations where social rules or expectations have been violated (Averill 1982). They appear to serve to inform the observer to stop the current behavioural action rather than to convey any information as to whether that action should be initiated in the future. In other words, angry expressions can be seen as triggers for response reversal (Blair *et al.* 1999; Blair & Cipolotti 2000). Orbital frontal cortex is crucially implicated in response reversal (Dias *et al.* 1996; O'Doherty *et al.* 2001*a*; Cools *et al.* 2002). Interestingly, similar areas of lateral orbital frontal cortex are activated by angry expressions and response reversal as a function of contingency change (Sprengelmeyer *et al.* 1998; Blair *et al.* 1999; Kesler-West *et al.* 2001). In addition, most neuro-imaging studies do not observe amygdala activation to angry expressions (Sprengelmeyer *et al.* 1998; Blair *et al.* 1999; Kesler-West *et al.* 2001). The only study, to my knowledge, that did observe amygdala activation by angry expressions found very weak activation that was significantly less than that seen to fearful expressions (Whalen *et al.* 2001).

4. NEUROTRANSMITTER INVOLVEMENT IN RESPONDING TO THE EXPRESSIONS OF OTHERS

There is a growing body of data indicating a degree of differential neurotransmitter involvement in systems responsible for the processing of emotional expressions. Thus, pharmacological interventions can alter the communicatory salience of emotional expressions. For example, serotonergic manipulations have been found to

differentially affect the processing of fearful and happy expressions (Harmer *et al.* 2001a), noradrenergic manipulations to differentially affect the processing of sad expressions (Harmer *et al.* 2001b) whereas dopaminergic and GABAergic manipulations differentially affect the processing of angry expressions (Borrill *et al.* 1987; Blair & Curran 1999; Zangara *et al.* 2002). Given these differential effects one might predict that the serotonergic and noradrenergic manipulations are differentially affecting the amygdala's role in responding to fearful, sad and happy expressions as unconditioned stimuli for aversive and appetitive conditioning and instrumental learning, whereas GABAergic manipulations impact the role of orbital frontal cortex in modulating the response to interpersonal signals of conflict such as anger. Certainly, it is known that there is considerable serotonergic and noradrenergic innervation of the amygdala (Amaral *et al.* 1992) and the impact of noradrenergic manipulations of the amygdala's role in the augmentation of episodic memory is well known (Cahill & McGaugh 1998; Cahill 2000). There are high concentrations of benzodiazepine receptor sites in both amygdala and the frontal cortex (Dennis *et al.* 1988; Bremner *et al.* 2000). However, although the central nucleus of the amygdala which projects to autonomic centres in the brain stem is densely innervated by GABA neurons, the basolateral nucleus of the amygdala, projecting to cortical regions, contains only scattered GABA neurons (Swanson & Petrovich 1998). It is plausible that the basolateral nucleus, as a function of its interconnections with cortical regions, is more involved in responding to fearful expressions and thus relatively unaffected by GABAergic manipulations.

At present only one study, to my knowledge, has examined the neural underpinnings of the effects of these pharmacological agents (Blair *et al.* 2003). This investigated the impact of diazepam on the neural response to morphed angry and fearful expressions. Interestingly, while diazepam abolished the increase in lateral orbital frontal cortex activity as a function of increased angry expression intensity, the increase in amygdala activity as a function of increased fearful expression intensity was not affected by diazepam. This study thus adds support to the suggestion that GABAergic manipulations impact the role of orbital frontal cortex in modulating the response to interpersonal signals of conflict such as anger.

5. ACKNOWLEDGING OTHER INDIVIDUALS' EXPRESSIONS: CLOSING THE COMMUNICATORY LOOP

In this paper the communicatory function of emotional expressions has been stressed. Reference was made to a crucial determinant of whether an expression will be elicited: the presence of others (Jones & Raag 1989; Chovil 1991; Fridlund 1991; Jones *et al.* 1991). Individuals typically display expressions when there is an audience to witness these expressions. This might suggest that individuals should stop displaying emotional expressions when the audience has demonstrated that they have registered the display of the emoter. Thus, for example, in the social referencing example provided above, the caregiver should stop to display fear when the infant demonstrates that they will now not approach the aversive novel object. However,

although this would intuitively appear to be the case, I know of no empirical literature demonstrating it to be so.

One particular case where there are clear indications that the audience demonstrates that they have registered the display of the emoter is seen during embarrassment displays. Embarrassment is associated with gaze aversion, shifting eye positions, speech disturbances, face touches, a nervous smile and a rigid, slouched posture (Goffman 1967; Asendorpf 1990; Lewis *et al.* 1991). More recent work has demonstrated that embarrassment display unfolds in the following reliable sequence. This involves gaze aversion; a smile control, which is a lower facial action that potentially inhibits the smile; a non-Duchenne smile, which only involves the zygomatic major muscle action that pulls the corners of the lips upwards; a second smile control; head movements down; and then face touching, which occurred *ca.* 25% of the time (Keltner 1995).

Leary & Meadows (1991), Leary *et al.* (1996) and others (Keltner 1995; Miller 1996; Gilbert 1997; Keltner & Buswell 1997) have suggested that embarrassment serves an important social function by signalling appeasement to others. When a person's untoward behaviour threatens his/her standing in an important social group, visible signs of embarrassment function as a non-verbal acknowledgement of shared social standards. Leary argues that embarrassment displays diffuse negative social evaluations and the likelihood of retaliation. The basic idea is that embarrassment serves to aid the restoration of relationships following social transgressions (Keltner & Buswell 1997). In other words, embarrassment displays may be initiated by an individual following an emoter's display of anger: if the individual's behaviour was unintentional or the angry observer is of high status.

There is a good deal of empirical evidence to support this 'appeasement' or remedial function of embarrassment from studies of both humans and non-human primates (Leary & Meadows 1991; Gilbert 1997; Keltner & Buswell 1997; Keltner & Anderson 2000). For example, Semin & Manstead (1982) found that people reacted more positively to others after a social transgression if the transgressors were visibly embarrassed. In addition, Leary *et al.* (1996) presented evidence that people are actually motivated to convey embarrassment to others as a way of repairing their social image.

6. PATHOLOGICAL EXPRESSION PROCESSING: THE CASES OF AUTISM, DEVELOPMENTAL PSYCHOPATHY AND ACQUIRED SOCIOPATHY

If emotional expressions serve a communicatory function, as I have been arguing, we might expect that atypical responding to the emotional expressions of others would adversely affect development. Three ways in which development can be affected will be discussed below with reference to the neuro-psychiatric conditions of autism, developmental psychopathy and acquired sociopathy.

Autism is a severe developmental disorder described by the American Psychiatric Association's diagnostic and statistical manual (DSM-IV) as 'the presence of markedly abnormal or impaired development in social interaction and communication and a markedly restricted repertoire of activities and interests' (American Psychiatric Associ-

ation 1994, p. 66). The main criteria for the diagnosis in DSM-IV can be summarized as qualitative impairment in social communication and restricted and repetitive patterns of behaviour and interests. These criteria must be evident before 3 years of age.

As long as autism has been recognized, the idea has existed that the main difficulty for people with autism is an inability to enter into emotional relationships. Thus, Kanner, the psychiatrist who originally described the disorder in 1943, wrote 'these children have come into the world with an innate inability to form the usual, biologically provided affective contact with other people, just as other children come into the world with innate physical or intellectual handicaps' (Kanner 1943, p. 250). More recently, it has been suggested that autism is due to an innate impairment in the ability to perceive and respond to the affective expressions of others, and that this deficit leads to their profound difficulties in social interaction (Hobson 1993).

Many studies have investigated the ability of individuals with autism to recognize the emotional expressions of others. Many have reported that children with autism have difficulty recognizing the emotional expressions of others (Hobson 1986; Bormann-Kischkel *et al.* 1995; Howard *et al.* 2000) with a recent claim suggesting that this is specific for fearful expressions (Howard *et al.* 2000). However, the above only applies to studies where the groups have not been matched on mental age. When they are, children with autism have usually been found to be unimpaired in facial affect recognition (Ozonoff *et al.* 1990; Prior *et al.* 1990; Baron-Cohen *et al.* 1997b; Adolphs *et al.* 2001). In addition, several studies have found the emotion processing impairment to be pronounced only when the emotion is a complex 'cognitive' emotion such as surprise or embarrassment (Capps *et al.* 1992; Baron-Cohen *et al.* 1993; Bormann-Kischkel *et al.* 1995).

I would therefore argue that autism does not represent a disorder where there is atypical recognition of emotional expressions. However, autism is interesting because of the well-documented impairment in theory of mind shown by patients with this disorder (Frith 2001). Theory of mind refers to the ability to represent the mental states of others, i.e. their thoughts, desires, beliefs, intentions and knowledge (Premack & Woodruff 1978; Leslie 1987; Frith 1989). Impairment in theory of mind is interesting for the communicatory role of emotional expressions. Thus, a healthy individual, when witnessing the emotional display of another individual, will attempt to represent the intended cue that elicited the emoter's expression. So, for example, during social referencing, if the child is attending to one object when the caregiver displays an emotional response to another, the child will look at the caregiver to determine the direction of their attention (Moses *et al.* 2001). Theory of mind should be involved in the representation of the emoter's intention. If it is, we might predict anomalous behavioural reactions to the emotional displays of other individuals in children with autism given their theory-of-mind impairment. In particular, we should see a reduction in the usual orientation response to the emoter to calculate the eliciting stimulus. Indeed, this is exactly what is seen in children with autism. A series of studies has examined the behavioural reactions of individuals with autism when the child has been playing with the exper-

imenter and the experimenter has feigned an emotional reaction, usually distress (Sigman *et al.* 1992; Dissanayake *et al.* 1996; Bacon *et al.* 1998; Corona *et al.* 1998). All four of these studies have reported reduced orientation to the caregiver by the children with autism although this was only in the lower ability sample in the Bacon *et al.* (1998) study. However, this does not reflect a lack of responsiveness to other individuals' emotion. A child with autism presented with another individual in distress will show aversive autonomic arousal to the other's distress (Blair 1999) and, as has been argued above, children with autism present with no impairment in expression recognition (Ozonoff *et al.* 1990; Prior *et al.* 1990; Baron-Cohen *et al.* 1997b; Adolphs *et al.* 2001).

The above argument generates further predictions about emotion in autism. Social referencing, the learning of emotional valence for novel objects, should be impaired in children with autism. The child with autism should fail to use the emoter's gaze direction to calculate the correct object to associate the valence elicited by the emoter's display in the same way that they fail to use a speaker's gaze direction during novel word use to calculate the speaker's referent (Baron-Cohen *et al.* 1997a). This, in turn, predicts that children with autism may present with very unusual emotional reactions to objects. That is, without representing the emoter's referent they may associate valence to novel objects inappropriately or not at all.

Psychopathy is a developmental disorder characterized in part by callousness, a diminished capacity for remorse, impulsivity and poor behavioural control (Hare 1991). It is identified in children with the antisocial process screening device (Frick & Hare 2001) and in adults with the revised psychopathy checklist (Hare 1991). Importantly, this disorder is not equivalent to the psychiatric diagnoses of conduct disorder or antisocial personality disorder (American Psychiatric Association 1994). These psychiatric diagnoses are relatively poorly specified and concentrate almost entirely on the antisocial behaviour shown by the individual rather than any form of functional impairment. Because of this lack of specification, rates of diagnosis of conduct disorder reach up to 16% of boys in mainstream education (American Psychiatric Association 1994) and rates of diagnosis of antisocial personality disorder are over 80% in forensic institutions (Hart & Hare 1996). Because of these high rates of diagnosis, populations identified with these diagnostic tools are highly heterogeneous and also include many individuals with other disorders. Psychopathy, in contrast, is shown by less than 1% of individuals in mainstream education (Blair & Coles 2000) and less than 30% of individuals incarcerated in forensic institutions (Hart & Hare 1996).

One account of psychopathy has linked the disorder to early amygdala dysfunction and consequent impairment in processing fearful and sad expressions (Blair 1995, 2001; Blair *et al.* 1999). The basic suggestion is that psychopathic individuals represent the developmental case where sad and fearful expressions are not aversive unconditioned stimuli. As a consequence of this, the individual does not learn to avoid committing behaviours that cause harm to others and will commit them if, by doing them, he receives reward (Blair 1995). In line with this theory, psychopathic individuals have been found to present with reduced amygdaloid volume relative to comparison indi-

viduals (Tiihonen *et al.* 2000) and reduced amygdala activation, relative to comparison individuals, during an emotional memory task (Kiehl *et al.* 2001) and aversive conditioning tasks (Veit *et al.* 2002). Moreover, in functions that recruit the amygdala such as aversive conditioning and instrumental learning, the augmentation of startle reflex by visual threat primes or arousal to the anticipation of punishment are all impaired in psychopathic individuals (Blair 2001). Also in line with the theory, psychopathic individuals show pronounced impairment in processing sad and fearful expressions. They show reduced autonomic responses to these expressions (Aniskiewicz 1979; Blair *et al.* 1997) and, particularly in childhood, impaired ability to recognize these expressions (Blair *et al.* 2001). Finally, their socialization is markedly impaired. Thus, although it has been repeatedly shown that the use of empathy inducing positive parenting strategies by caregivers decreases the probability of antisocial behaviour in healthy developing children, it does not decrease the probability of antisocial behaviour in children who present with the emotional dysfunction of psychopathy (Wootton *et al.* 1997).

Acquired sociopathy represents an interesting counterpoint to developmental psychopathy. 'Acquired sociopathy' was a term introduced by Damasio *et al.* (1990) to characterize individuals who, following acquired lesions of the orbitofrontal cortex, fulfil the DSM-III diagnostic criteria for 'sociopathic disorder' (American Psychiatric Association 1980). Previously, Blumer & Benson (1975) had used the term 'pseudo-psychopathy' to refer to patients with frontal lobe lesions presenting in this manner. Although there have been suggestions that developmental psychopathy and acquired sociopathy might be different forms of the same disorder (Damasio 1994), this now appears unlikely (Blair 2001). Indeed, developmental psychopathy and acquired sociopathy present very differently. Psychopathic individuals present with pronounced levels of goal-directed instrumental aggression and antisocial behaviour, reflecting an impairment that interferes with their ability to be socialized (Cornell *et al.* 1996). In contrast, patients with acquired sociopathy present with frustration- or threat-induced reactive aggression whether their acquired lesion of the orbital frontal cortex occurs in childhood (Pennington & Bennetto 1993; Anderson *et al.* 1999) or adulthood (Grafman *et al.* 1996; Blair & Cipolotti 2000).

I have argued for the communicatory role of angry and embarrassment expressions in regulating social hierarchical interactions, in particular, the role of angry expressions in stopping the current behavioural action and the role of embarrassment displays in communicating a lack of intent to commit the action that has resulted in social disapproval. We might expect therefore that an individual whose response to angry/embarrassment expressions is dysfunctional should present with impaired modulation of their social behaviour. The orbital frontal cortex is implicated in the response to angry expressions (Sprengelmeyer *et al.* 1998; Blair *et al.* 1999; Kesler-West *et al.* 2001). Interestingly, then, patients with acquired sociopathy following lesions of the orbital frontal cortex present with generally impaired expression recognition but this impairment is particularly marked for angry expressions (Hornak *et al.* 1996; Blair & Cipolotti 2000).

The strong suggestion is therefore that this impairment underlies their socially inappropriate behaviour.

7. CONCLUSIONS

In this paper, I have stressed the communicatory function of emotional expressions. Importantly, the argument is not that the display of expressions implies that the emoter intended to convey a specific message to the observer, it is simply that emotional expressions serve a communicatory function. Crucially, the emoter's emotional displays are a function of the presence of observers and the observer will attempt to determine the referent of the emoter's display. Assuming the observer accomplishes this, appropriate information will have been transferred from the emoter to the observer.

Although emotional expressions are not intentional communications, their display can be intentionally manipulated. Children learn display rules; social rules that stipulate when it is, and when it is not, appropriate to display emotional expressions. Thus we can learn to intentionally mask or alter our expressions as a function of these display rules. Presumably, the emoter's intent modulates the frontal lobe-basal ganglia circuitry that has been implicated in the production of emotional expressions.

Although systems generally involved in processing facial stimuli, such as the occipital cortex, fusiform and the superior temporal sulcus process expressions, the communicatory function of emotional expressions is reflected in the partly dissociable neural systems that are additionally involved in processing emotional expressions. Thus, expressions that serve as positive or negative reinforcers preferentially activate the amygdala (fearfulness, sadness and happiness). Although disgusted expressions are also reinforcers, they are used most frequently to provide information about foods. As such they engage the insula, a region involved in taste aversion. Angry expressions initiate response reversal and activate regions of orbital frontal cortex that are involved in the modulation of behavioural responding.

If we assume that emotional expressions serve a communicatory function, we must predict that they will be more likely to be displayed when a potential observer is present. This is indeed the case. In addition, we must predict that the display of the expression will be terminated when the observer has shown clear indication that they have received the communication. This remains to be investigated.

The consequences of impairment in being able to adequately process the emotional displays of others can be severe. I have argued that although individuals with autism may be able to recognize the expressions of others, it is highly likely that they fail to adequately process the emoter's referent and that they therefore process the display incorrectly because of their impairment in theory of mind. In contrast, individuals with the developmental disorder of psychopathy and individuals with acquired sociopathy following lesions of the orbital frontal cortex fail to respond appropriately to specific expressions. In psychopathic individuals, the processing of other individuals' sadness and fear is particularly affected. This leads to a failure in socialization. The psychopathic individual does not learn to avoid actions that cause harm to others.

In acquired sociopathy, the processing of others' anger and probably embarrassment is particularly affected. This leads to a failure to adequately modulate behaviour according to the social context.

In short, emotional expressions allow the rapid communication of valence information between individuals. They allow the observer to rapidly learn which behaviours and objects (including foods) to approach or avoid, as well as information allowing rapid modification of behaviour according to the social environment and hierarchy. Impairment in systems that respond to the emotional expressions of others can have devastating effects.

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GLOSSARY

CS: conditioned stimulus

US: unconditioned stimulus