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Part I

‘Dysempathy’ in psychiatric samples

Empathic dysfunction in psychopathic individuals

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1.1 Introduction

Psychopathy can be considered one of the prototypical disorders associated with empathic dysfunction. Reference to empathic dysfunction is part of the diagnostic criteria of psychopathy (Hare, 1991). The very ability to inflict serious harm to others repeatedly can be, and is (Hare, 1991), an indicator of a profound disturbance in an appropriate ‘empathic’ response to the suffering of another. The goal of this chapter will be to consider the nature of the empathic impairment in psychopathy.

First, I will consider the disorder of psychopathy and the definition of empathy. Second, I will consider whether individuals with psychopathy are impaired in ‘cognitive empathy’ or Theory of Mind. Third, I will consider the cognitive and neural architecture mediating ‘emotional empathy’. Fourth, I will consider whether individuals with psychopathy are impaired in ‘emotional empathy’.

1.1.1 The disorder of psychopathy

The origins of the concept of psychopathy probably originate in the writings of Pritchard (1837); see Pichot (1978). Pritchard developed the concept of ‘moral insanity’ to account for socially damaging or irresponsible behaviour that was not associated with known forms of mental disorder. He attributed morally objectionable behaviour to be a consequence of a diseased ‘moral faculty’. While the notion of a ‘moral faculty’ has been dropped, modern psychiatric classifications such as the *American Psychiatric Association’s Diagnostic and Statistical Manual* (currently, DSM-IV) make reference to syndromes associated with high levels of antisocial behaviour: conduct disorder (CD) in children and antisocial personality disorder (APD) in adults.

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Unfortunately, the psychiatric diagnoses of CD and APD are flawed. Partly because they only focus on the presence of antisocial behaviour, these diagnoses tend to identify highly heterogeneous samples. This heterogeneity is even acknowledged in DSM-IV where two forms of CD are specified: childhood- and adolescent-onset types. Because of their lack of precision, the diagnostic rate of CD can reach 16% of boys in mainstream education (American Psychiatric Association, 1994) while the diagnostic rate of APD can reach over 80% in adult forensic institutions (Hare, 1991). Unsurprisingly, therefore, diagnoses of CD and APD are relatively uninformative regarding an individual's prognosis.

The classification of psychopathy, in contrast, is informative. This classification was introduced by Hare (1980; 1991) and has proved to be a useful predictor of future risk (Hare, 1991). The classification involves both affective-interpersonal (e.g. such as lack of empathy and guilt) and behavioural components (e.g. criminal activity and poor behavioural controls) (Frick & Hare, 2001; Hare, 1991). Psychopathy represents a developmental disorder. In childhood and adolescence, psychopathic tendencies are identified principally by either the use of the Antisocial Process Screening Device (Frick & Hare, 2001) or by the Psychopathy Checklist: Youth Version. In adulthood, psychopathy is identified through use of the Psychopathy Checklist – Revised (Hare, 1991).

As noted above, psychopathy can be considered one of the prototypical disorders associated with empathic dysfunction. In this chapter, I will consider the nature of the empathic impairment in psychopathy.

1.1.2 Defining empathy

Empathy has been defined as ‘an affective response more appropriate to someone else’s situation than to one’s own’ (Hoffman, 1987; p. 48); it is an emotional reaction in an observer to the affective state of another individual. This form of definition of empathy will underpin this paper. Unfortunately, however, the term empathy has been used in a variety of ways by a variety of authors (Hoffman, 1987). At least three different types of empathy can be considered. The differences between these types are important to identify as they must implicate notably different cognitive architectures. The three types of empathy are: (1) motor empathy where the individual mirrors the motor responses of the observed actor; (2) ‘cognitive’ empathy where the individual represents the internal mental state of the other (effectively Theory of Mind); (3) an emotional response to another individual that is congruent with the other’s emotional reaction. In this chapter, I will briefly consider ‘cognitive empathy’, from here onwards referred to only as Theory of Mind, and emotional empathy with respect to psychopathy (a distinction will be made between the two forms outlined above later in the paper). I will not consider motor empathy in this chapter.

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1.1.3 Theory of Mind and psychopathy

Theory of Mind refers to the ability to represent the mental states of others, i.e. their thoughts, desires, beliefs, intentions and knowledge (Frith, 1989). Theory of Mind allows the attribution of mental states to self and others in order to explain and predict behaviour.

The classic measure of Theory of Mind is the Sally-Anne task (Wimmer & Perner, 1983). In this task, the participant is shown two dolls, Sally and Anne, and a basket and a box. The participant watches as Sally places her marble in the basket and then leaves the room. While Sally is out, naughty Anne moves Sally's marble from the basket to the box. Then she, too, leaves the room. Now Sally comes back into the room. The participant is asked the test question: 'Where will Sally look for her marble?'. In order to pass this task, the participant must represent Sally's mental state, her belief that the marble is in the basket. Without this representation, the participant will answer on the basis of the marble's real location, i.e. the box. Most healthy developing individuals from the age of 4 years pass this task (Wimmer & Perner, 1983).

In addition to being considered a form of empathy in its own right, the ability to represent the mental states of others has been considered to be necessary for 'emotional empathy' to occur (Batson *et al.*, 1987; Feshbach, 1987). Within these positions, representations of the internal mental state of another are assumed to act as stimuli for the activation of the affective, empathic response (Batson *et al.*, 1987). Feshbach (1987), for example, viewed empathy to be a function of three processes: first, the cognitive ability to discriminate affective cues in others; second, the more mature cognitive skills entailed in assuming the perspective and role of another person; third, emotional responsiveness (i.e. the ability to experience emotions) (Feshbach, 1987). According to Feshbach (1987), 'empathy is conceived to be the outcome of cognitive and affective processes that operate conjointly' (p. 273).

There are no indications of Theory of Mind impairment in individuals with psychopathy. Three out of four studies assessing the ability of individuals with psychopathy on Theory of Mind measures have reported no impairment (Blair *et al.*, 1996; Richell *et al.*, 2003; Widom, 1978). Only one study has reported impairment and this used a rating scale that is not a typical measure of Theory of Mind (Widom, 1976).

Blair *et al.* (1996) assessed the ability of individuals with psychopathy to perform the Advanced Theory of Mind test (Happé, 1994). This is a story comprehension measure that assesses understanding of mental states. Individuals with autism, a population with known Theory of Mind impairment (Frith, 1989), are impaired on this measure (Happé, 1994). However, the individuals with psychopathy were not (Blair *et al.*, 1996).

Richell *et al.* (2003) examined the ability of individuals with psychopathy to perform the ‘Reading the Mind in the Eyes’ task. In this task, participants must judge the complex social emotion being displayed by an individual based on information only from the eye region (Baron-Cohen *et al.*, 1997). Individuals with autism are impaired on this task (Baron-Cohen *et al.*, 2001). However, again, the individuals with psychopathy were not (Richell *et al.*, 2003).

In addition to the above work with individuals with psychopathy, it is important to note that even in the broader spectrum of antisocial individuals, there are few data suggesting any link between Theory of Mind impairment and antisocial behaviour. Hughes and colleagues did find some indication of Theory of Mind impairment in their ‘hard-to-manage’ preschoolers relative to the comparison group (Hughes *et al.*, 1998). However, Happé and Frith found no impairment in their children with emotional and behavioural difficulties (Happé & Frith, 1996). Similarly a study of school bullies found no indications of Theory of Mind impairment (Sutton *et al.*, 1999). In addition, Sutton and colleagues also found no relationship between Theory of Mind performance on the advanced Eyes task and ‘disruptive behaviour disorder’ symptoms in children aged 11–13 years (Sutton *et al.*, 2000).

Summary

The profound empathic dysfunction reported in the clinical description of psychopathy (Hare, 1991) does not involve Theory of Mind impairment. Individuals with psychopathy are unimpaired on measures of Theory of Mind. Indeed, there are no indications that any populations who show heightened levels of antisocial behaviour are associated with Theory of Mind impairment.

1.2 Emotional empathy

Figure 1.1 represents a simple schematic of the cognitive processes that I consider to underpin empathy. Here empathy is being defined as the emotional response to another individual’s visual or vocal expression of emotion. This schematic assumes that there may be at least two routes to the generation of an emotional empathic response: one which relies on the ‘semantic processing’ of the expression and one which does not. This follows 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) (Adolphs, 2002).

These two routes for expression processing mirror those previously suggested to be involved in aversive conditioning (LeDoux, 2000). The sub-cortical route is

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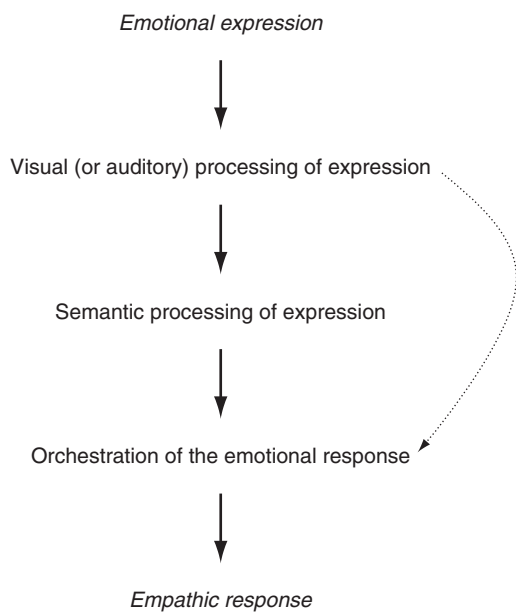


Figure 1.1. A schematic of the cognitive processes thought to underpin empathy. The dotted line refers to the suggested (sub-cortical) route that bypasses the semantic processing of the expression

thought to provide coarse stimulus processing while the cortical route is thought to allow more precise stimulus encoding and allow discrimination learning. The cortical route would underpin the ‘semantic processing’ of the expression; i.e. it would allow the expression to be named and would allow goal-directed behaviour to be initiated in response to the expression (e.g. initiate helping behaviour to a crying individual).

In Figure 1.1, there is reference to the systems involved in ‘the orchestration of the emotional response’. I have stressed elsewhere that the facial expressions of emotion each have a communicatory function, that they impart specific information to the observer (Blair, 2003a). The systems involved in ‘the orchestration of the emotional response’ are those systems which respond automatically to the communicatory value of the expression. In short, an empathic response is a translation of a non-verbal communicatory signal. Because of the different implications of these communicatory signals, I have argued that they are translated in several separable systems (Blair, 2003a). I will consider this communication and the systems that orchestrate the response to this communication below.

I have suggested that fearfulness, sadness and happiness are reinforcers that modulate the probability that a particular behaviour will be performed in the

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future (Blair, 2003a). 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, I have 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. Happy expressions, in contrast, are appetitive unconditioned stimuli which increase the probability of actions to which they appear causally related.

The amygdala has been implicated in aversive and appetitive conditioning including instrumental learning (LeDoux, 2000). It is thus unsurprising, given the suggested role of fearful, sad and happy expressions as reinforcers, that neuroimaging studies, with a few exceptions, have generally found that fearful, sad and happy expressions all modulate amygdala activity (see, for a review, Blair, 2003a). The neuropsychological literature supports the neuroimaging literature as regards 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, 2002; Blair, 2003a). Impairment in the processing of sad expressions is not uncommonly found in patients with amygdala lesions (Blair, 2003a). However, amygdala lesions rarely result in impairment in the recognition of happy expressions although this may reflect the ease with which happy expressions are recognized (Blair, 2003a).

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 to which the emoter is displaying the disgust reaction. Thus, the suggestion is that the disgusted expressions of others activate in particular the insula allowing taste aversion [the disgust expression is the unconditioned stimulus (US) that is associated with the novel food conditioned stimulus (CS)] to occur (Blair, 2003a).

I have argued that displays of anger or embarrassment do not act as unconditioned stimuli for aversive conditioning or instrumental learning (Blair, 2003a). Angry expressions are known to curtail the behaviour of others in situations where social rules or expectations have been violated (Averill, 1982). Instead, they are important signals to modulate current behavioural responding, particularly in situations involving hierarchy interactions (Blair, 2003a). 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.

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In other words, angry expressions can be seen as triggers for response reversal. Orbital and ventrolateral frontal cortex is crucially implicated in response reversal (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 (Blair, 2003b).

Summary

In short, emotional expressions are non-verbal communications. Empathy is a prime component of the translation of this communication within the observer. This translation is potentially reliant on both cortical and sub-cortical routes. These routes convey the communication to regions of the brain involved in emotional processing (the amygdala, insula and orbital and ventrolateral frontal cortex). These regions orchestrate a response to this communication; mediating emotional learning about objects or food or initiating response reversal.

1.3 Psychopathy and emotional empathy

As noted in the introduction, there can be no doubt that psychopathy is associated with empathic dysfunction. However, the question remains regarding the form of this dysfunction. I outlined above that the empathic dysfunction in psychopathy does not include impairment in Theory of Mind. What about emotional empathy? In Section 1.2, I outlined a schematic of the empathic process. Currently, no data exist regarding the two routes to the systems that allow the orchestration of the emotional response. We do not know whether psychopathy is associated with dysfunction in systems involved in face processing. However, one reason to believe that there is no obvious general dysfunction in the systems involved in facial processing is that while individuals with psychopathy are impaired in expression processing, their impairment appears to be selective. Given this selectivity (see below), it is unlikely that there is notable dysfunction in the systems involved in face processing.

Two main forms of paradigm have been used to index empathy in individuals with psychopathy: skin conductance responses (SCRs) to empathy-inducing stimuli and the ability to recognize facial expressions. Three studies have examined vicarious conditioning in individuals with psychopathy; i.e. the extent to which the participant will learn an autonomic response to a stimulus associated with another individual's distress (Aniskiewicz, 1979; House & Milligan, 1976; Sutker, 1970). Two of these three studies reported reduced vicarious conditioning in the individuals with psychopathy, the third did not.

Two studies have examined SCRs to sad faces in individuals with psychopathic tendencies: one examined adults with psychopathy, the other children with psychopathic tendencies (Blair, 1999; Blair *et al.*, 1997). In these studies,

the participants were presented with images of sad faces, threatening stimuli (e.g. pointed guns but also including an angry face) or neutral stimuli (e.g. a book). Both the adults with psychopathy and the children with psychopathic tendencies showed reduced SCRs to the sad faces relative to their respective comparison populations. Interestingly, both adults with psychopathy and the children with psychopathic tendencies showed relatively appropriate SCRs to the angry face amidst the threatening stimuli. This was the first indication that the empathic impairment in individuals with psychopathy might be selective for particular expressions.

Studies have examined the ability of individuals with psychopathy to recognize the facial or vocal emotional expressions of others (Blair *et al.*, 2001, 2002, 2004, 2006a; Kosson *et al.*, 2002; Stevens *et al.*, 2001). In most of these studies, the children with psychopathic tendencies/adults with psychopathy have been impaired in the recognition of sad/fearful expressions. Typically, the children with psychopathic tendencies have shown impairment in the recognition of sad expressions (Blair *et al.*, 2001; Stevens *et al.*, 2001). However, this has not been found in the adults with psychopathy (with one exception; Dolan, personal communication). In all of the studies, except that of Kosson *et al.* (2002), the children with psychopathic tendencies and the adults with psychopathy have been impaired in the recognition of fearful expressions. Kosson *et al.* (2002) reported some difficulty with the recognition of disgusted expressions (but only when the participants were responding with the left hand). Blair *et al.* (2004) also found some impairment in the adults with psychopathy for the recognition of disgusted expressions, however this deficit was not present if the effect of intelligence quotient (IQ) was co-varied out.

The above data suggest a relative selectivity in the empathic dysfunction shown by individuals with psychopathy. Individuals with psychopathy are impaired when processing fearful, sad (in adulthood if responsiveness is indexed by SCRs, in childhood whether by SCR or recognition score) and possibly disgusted expressions. No study has yet reported that individuals with psychopathy show impairment in the processing of angry, happy or surprised expressions. The absence of impairment for angry expressions is particularly interesting. Neurological patients following lesions of orbital and ventral frontal cortex or psychiatric conditions which are thought to detrimentally affect orbital and ventrolateral regions, such as childhood bipolar disorder or intermittent explosive disorder, all show general difficulties with processing expressions but their difficulty is particularly marked for angry expressions (Best *et al.*, 2002; Blair & Cipolotti, 2000; Hornak *et al.*, 1996; McClure *et al.*, 2003).

In the Section 1.2, I suggested that there were at least three systems responsible for orchestrating responses to expressions; i.e. the core component of empathy

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(Blair, 2003a). One system responsive to the aversive and appetitive unconditioned stimuli of fearful, sad and happy expressions and consequently modulating the probability that any stimulus associated with these expressions will be avoided or approached in the future. A second system responsive to the aversive unconditioned stimulus of a disgusted expression will reduce the probability that any stimulus (particularly food) associated with this expression will be avoided in the future. A third system is particularly responsive to displays of anger and embarrassment which modulates on-going social interactions (eliciting responses according to the hierarchy level of the communicator amongst other factors).

I claim that individuals with psychopathy have dysfunction primarily in the first system (that responsive to fearful, sad and happy expressions). They may also have dysfunction in the second system (that responsive to disgusted expressions). However, in the absence of additional data that possibility will not be considered here.

As noted above, the primary neural system responsible for orchestrating an emotional response to fearful, sad and happy expressions is the amygdala (see Blair, 2003a). This suggests amygdala dysfunction in psychopathy. There are considerable data in line with this suggestion (Blair, 2003b). Thus, individuals with psychopathy show reduced amygdaloid volume relative to comparison individuals and reduced amygdala activation during emotional memory and aversive conditioning tasks. Human and animal neuropsychological work has informed us that the effects of amygdala lesions include impairment in: (1) aversive conditioning; (2) the augmentation of the startle reflex to visual threat primes; and (3) passive avoidance learning. If psychopathy is associated with amygdala dysfunction, the neuropsychological approach would predict that individuals with psychopathy are impaired in the above tasks. Considerable data show that they are (see, for a review, Blair, 2004).

While individuals with psychopathy are impaired in the processing of fearful and sad expressions, they show no impairment for the processing of happy expressions (Blair, 2003b; Blair *et al.*, 2001, 2002, 2006a; Kosson *et al.*, 2002; Stevens *et al.*, 2001). While this is consistent with the neuropsychological literature documenting the consequences of amygdala lesions, it is less consistent with the neuroimaging literature (see above) which suggests a role for the amygdala in the processing of happy expressions. Of course, the absence of impairment for happy expressions in individuals with psychopathy might reflect the ease with which they are recognized (i.e. an intact amygdala is not necessary for naming).

However, a more interesting possibility is that this absence of impairment reflects the selectivity of their impairment. The amygdala is involved in the formation of stimulus–reward and stimulus–punishment associations; animals with amygdala lesions show impairment in both reward- and punishment-related