The emergence of psychopathy: Implications for the neuropsychological approach to developmental disorders

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Abstract

In this paper, I am going to examine the disorder of psychopathy and consider how genetic anomalies could give rise to the relatively specific neuro-cognitive impairments seen in individuals with this disorder. I will argue that genetic anomalies in psychopathy reduce the salience of punishment information (perhaps as a function of noradrenergic disturbance). I will argue that the ability of the amygdala to form the stimulus–punishment associations necessary for successful socialization is disrupted and that because of this, individuals with psychopathy do not learn to avoid actions that will harm others. It is noted that this model follows the neuropsychological approach to the study of developmental disorders, an approach that has been recently criticized. I will argue that these criticisms are less applicable to psychopathy. Indeed, animal work on the development of the neural systems necessary for emotion, does not support a constructivist approach with respect to affect. Importantly, such work indicates that while environmental effects can alter the responsiveness of the basic neural architecture mediating emotion, environmental effects do not construct this architecture. However, caveats to the neuropsychological approach with reference to this disorder are noted.

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1. Introduction

The intellectual roots of psychopathy can be traced to Pritchard (1835); see (Pichot, 1978). Pritchard developed the concept of “moral insanity” to account for socially damaging or irresponsible behavior that was not associated with known forms of mental disorder. He attributed morally objectionable behavior to be a consequence of a diseased “moral faculty”. Psychiatric and legal classifications have followed this tradition; Antisocial Personality Disorder is a classification within the American Psychiatric Association’s DSM-IV.

Unfortunately, the psychiatric diagnoses of Conduct Disorder (CD) and Antisocial Personality Disorder (APD: American Psychiatric Association, 1994) are seriously flawed. These diagnoses of CD and APD focus on the presence of antisocial behavior and tend to identify a highly heterogeneous sample. Indeed, DSM-IV acknowledges this heterogeneity by specifying two forms of CD: childhood- and adolescent-onset types. In childhood-onset type, onset of at least one criterion characteristic of CD must have occurred prior to 10 years of age. In adolescent-onset type there should be not be any criteria characteristic of CD prior to 10 years of age. 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 (Hart & Hare, 1996). Unsurprisingly, therefore, diagnoses of CD and APD are relatively uninformative regarding an individual’s prognosis.

In sharp contrast, the classification of psychopathy, introduced by Hare (1980, 1991), is highly informative. The classification is a very useful predictor of a patient’s future behavior (Hare, 1991) and is at the basis of many recidivism risk assessments. The classification of psychopathy involves affective-interpersonal (e.g., such as lack of empathy and guilt) and behavioral components (e.g., criminal activity and poor behavioral controls); (Frick, O’Brien, Wootton, & McBurnett, 1994; Hare, 1980, 1991; Harpur, Hare, & Hakstian, 1989). It is a developmental disorder (Harpur & Hare, 1994). 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 (Forth, Kosson, & Hare, 2003; Kosson, Cyterski, Steuerwald, Neumann, & Walker-Matthews, 2002). In adulthood, psychopathy is identified though use of the Psychopathy Checklist – Revised (Hare, 1991).

A remarkable feature of the behavioral profile of individuals with psychopathy is their excessive displays of instrumental aggression (Cornell et al., 1996; Williamson, Hare, & Wong, 1987). The term instrumental aggression (also referred to as proactive aggression) is used to distinguish aggression that is purposeful and goal directed. The aggression is used instrumentally to achieve a specific desired goal such as obtaining the victim’s possessions or to increase status within a group hierarchy (Berkowitz, 1993). Bullying is an example of instrumental aggression and, unsurprisingly, individuals who engage in bullying behaviors, frequently engage in other forms of instrumental antisocial behavior in other contexts (Roland & Idsoe, 2001). In contrast, reactive aggression (also referred to as affective aggression) occurs when...
a frustrating or threatening event triggers the aggressive act and frequently also induces anger (Barratt, Stanford, Dowdy, Liebman, & Kent, 1999; Barratt, Stanford, Kent, & Felthous, 1997; Berkowitz, 1993; Crick & Dodge, 1996; Linnoila et al., 1983). Importantly, the aggression is initiated without regard for any potential goal. Elevated levels of reactive aggression are found in many disorders; e.g., psychopathy, depression, anxiety, bipolar disorder and post traumatic stress disorder (see, for a review, Blair, 2003a). However, only individuals with psychopathy show elevated levels of instrumental aggression. Instrumental aggression is, of course, a form of instrumental behavior. Whether an individual displays a particular instrumental behavior is a product of their developmental learning experiences and decision-making regarding the current context. The suggestion that will be made here is that the impairment seen in individuals with psychopathy disrupts their capacity for normal socialization such that antisocial behavior is more likely to be displayed (Blair, 1995; Lykken, 1957; Trasler, 1973).

The disorder of psychopathy should be of interest to developmental cognitive neuroscientists for at least two main reasons. First, psychopathy allows us to understand the cognitive mechanisms necessary for the development of morality, an issue that is of clear intrinsic interest. Second, and more importantly, psychopathy represents a developmental disorder which affects a relatively specific set of neuro-cognitive systems. The pathology seen in individuals with psychopathy does not lead to global deficits in the development of a variety of neuro-cognitive systems. Instead, the effects of the pathology influence a relatively specific series of systems. In this paper, I will consider why this is. However, first I will describe the functional impairment seen in individuals with psychopathy.

1.1. What is the functional impairment shown by individuals with psychopathy?

Psychopathy is associated with a core set of empirically demonstrated behavioral difficulties. There are clear difficulties with emotional processing (Blair, 1995; Blair, Jones, Clark, & Smith, 1997; Cleckley, 1976; Eysenck, 1964; Fowles, 1988; Gray, 1987; Lykken, 1995; Mealey, 1995; Patrick, 1994; Pichot, 1978; Trasler, 1978, 1973) and some indications of difficulty with some forms of attentional processing (Jutai & Hare, 1983; Raine & Venables, 1988). I will concentrate on the difficulties with emotional processing as these have the clearest developmental implications.

There is a considerable body of literature revealing that individuals with psychopathy are impaired in the processing of fear-related stimuli. Thus, individuals with psychopathy show impairment in: (1) aversive conditioning (Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Lykken, 1957); (2) generating autonomic responses to anticipated threat (Hare, 1982; Ogloff & Wong, 1990); (3) the augmentation of the startle reflex to visual threat primes (Herrertz et al., 2001; Levenston, Patrick, Bradley, & Lang, 2000); (4) passive avoidance learning (Lykken, 1957; Newman & Kosson, 1986); and (5) response reversal (Mitchell, Colledge, Leonard, & Blair, 2002; Newman, Patterson, & Kosson, 1987). In addition, individuals with psychopathy show clear difficulties in empathic responding. Thus, individuals with psychopathy show impaired processing of the sadness and fear of others (Aniskiewicz, 1979; Blair,
In addition, individuals with psychopathy show impaired recognition of/autonomic responding to sad and fearful facial and vocal expressions (Aniskiewicz, 1979; Blair et al., 2002; Blair, Colledge, & Mitchell, 2001; Blair, Colledge, Murray, & Mitchell, 2001; Blair, Monson, & Frederickson, 2001; Blair et al., 1997). The fear-related data have led to repeated suggestions that individuals with psychopathy show impairment in the neuro-physiological systems modulating fear behavior (Cleckley, 1976; Eysenck, 1964; Fowles, 1988; Gray, 1987; Lykken, 1995; Mealey, 1995; Patrick, 1994; Pichot, 1978; Trasler, 1978, 1973). Developmentally, these positions all assume that moral socialization is achieved through the use of punishment (Eysenck & Gudjonsson, 1989; Trasler, 1978). In essence, they assume that the healthy individual is frightened by punishment and associates this fear with the action that resulted in the punishment thus making the individual less likely to engage in the action in the future. They suggest that individuals with psychopathy, because they are less aversively aroused by punishment, make weaker associations and thus are more likely to engage in the punished action in the future than healthy individuals. However, several researchers have questioned the assumption that conditioned fear responses play a crucial role in moral socialization (Blackburn, 1988; Blair & Morton, 1995). Thus, the developmental literature indicates that moral socialization is not achieved through the formation of conditioned fear responses but rather through the induction and fostering of empathy (Hoffman, 1984). Studies have shown, for example, that moral socialization is better achieved through the use of induction (reasoning that draws children’s attention to the effects of their misdemeanors on others and increases empathy) than through harsh authoritarian or power assertive parenting practices which rely on the use of punishment (Baumrind, 1971; Baumrind, 1983; Hoffman & Saltzstein, 1967). Indeed, several researchers have suggested that while empathy may facilitate moral socialization, fear may actually obstruct it (Brody & Shaffer, 1982; Hoffman, 1994).

On a related note, if healthy individuals learn to avoid antisocial behavior because of fear of punishment, it must be assumed that the healthy child judge all rules/transgressions in a similar way. In other words, if we learn to avoid talking in class and hitting other individuals because we are punished when we commit these actions, there is no reason for us to distinguish between these two transgressions. However, healthy developing children make a distinction between moral (victim-based) and conventional (social order based) transgressions from the age of 36 months (Smetana, 1981, 1985, 1993). In other words, children do not judge all transgressions in an identical fashion. Instead, they differentiate between those transgressions that result in harm to another from those that simply cause social disorder.

The importance of empathy for moral socialization was one of the reasons for the development of the Violence Inhibition Mechanism model of psychopathy and moral development (Blair, 1995; Blair et al., 1997). At its simplest, I argued that the Violence Inhibition Mechanism (VIM) was a system that when activated by distress cues (the sad and fearful expressions of others) results in increased autonomic activity, attention and activation of the brainstem threat response system (usually resulting in freezing); (Blair, 1995); see Fig. 1. I considered the VIM to be activated whenever
distress cues are displayed rather than being reliant upon contextual information about ongoing violence for activation. In line with the model, the display of distress cues has been found to result in the inhibition of not only aggression (Perry & Perry, 1974) but also non-violent disputes over property ownership (Camras, 1977) and sexual activity (Chaplin, Rice, & Harris, 1995).

The main focus of the model was to describe the cognitive prerequisites for moral development. According to the model, moral socialization occurs through the pairing of the activation of the mechanism by distress cues with representations of the act that caused the distress cues (Blair, 1995). Through association these representations of moral transgressions become triggers for the mechanism. The appropriately developing child thus initially finds the pain of others aversive and then, through socialization, the thoughts of acts that cause pain to others aversive. I proposed that individuals with psychopathy have had disruption to this system such that representations of acts that cause harm to others do not become triggers for the VIM (Blair, 1995).

One early index of appropriate moral socialization, and thus the developmental integrity of the VIM, is the demonstration by the child of the moral/conventional
distinction mentioned above. Children with psychopathic tendencies and adults with psychopathy show impairment in distinguishing moral and conventional transgressions (Blair, 1995, 1997; Blair, Jones, Clark, & Smith, 1995; Blair, Sellers, et al., 1995; Blair, Colledge et al., 2001; Blair, Colledge, Murray et al., 2001; Blair, Monson et al., 2001); see, for related work with children with Behavior Disorder and CD (Arsenio & Fleiss, 1996; Nucci & Herman, 1982). In addition, and in line with the VIM position, psychopathic adults show reduced comprehension of situations likely to induce guilt although they show appropriate comprehension of happiness, sadness and even complex social emotions such as embarrassment (Blair, Jones et al., 1995; Blair, Sellers, et al., 1995). Moreover, and a direct prediction of the model, children and adults with psychopathy show pronounced impairment in processing sad and fearful facial and vocal expressions (Aniskiewicz, 1979; Blair et al., 2002; Blair, 1999; Blair, Colledge et al., 2001; Blair, Colledge, Murray et al., 2001; Blair, Monson et al., 2001; Blair et al., 1997; House & Milligan, 1976).

However, while the original VIM model could provide an account of the emergence of instrumental antisocial behavior in individuals with psychopathy and while it did generate a variety of predictions that have been empirically confirmed, it faced a serious difficulty; it could not account for the data associated with the fear hypotheses. Moreover, it could not account for data on the interaction of temperament and socialization practice on the development of moral development/conscience. Kochanska has stressed the role of fearfulness as the important temperamental factor (Kochanska, 1993, 1997). Indeed, she and others have found fearful children to show higher levels of moral development/conscience using a variety of measures (Asendorpf & Nunner-Winkler, 1992; Kochanska, 1997; Kochanska, De Vet, Goldman, Murray, & Putman, 1994; Rothbart, Ahadi, & Hershey, 1994). In addition, Kochanska has stressed that different socialization practices may promote moral development in children with different temperaments (Kochanska, 1993, 1997). In line with this, she found that for fearful children, maternal gentle discipline promoted moral/conscience development. In contrast, for “fearless” children, alternative socialization practices, presumably capitalizing on mother-child positive orientation (secure attachment, maternal responsiveness), promoted the development of conscience (Kochanska, 1997). Because of these difficulties for the VIM model, I recently expanded it at both the cognitive and neural levels (Blair, 2003a, 2003b, 2003c, 2004; Blair & Charney, 2003); see below.

1.1.1. Summary

Individuals with psychopathy are profoundly impaired in the processing of fear-related stimuli and in the processing of the fearful and sad expressions of others. The Violence Inhibition Mechanism (VIM) model provided an account of the impairment in the processing of sad and fearful expressions in individuals with psychopathy and the implications of this impairment for moral development in these individuals. However, the VIM model cannot be considered a complete model of psychopathy; notably, it could not account for the impairments in the processing of fear-related stimuli in individuals with psychopathy.
In the following sections of this paper, I will consider a series of issues. First, I will consider whether there is a genetic basis to this disorder. Second, I will consider the neural and neuro-chemical systems on which I believe this genetic contribution may be operating (the systems which mediate the basic response to threat). Two options will be considered: (a) the genetic anomalies disrupt the functioning of the amygdala; and (b) the genetic anomalies disrupt the functioning of specific neurotransmitter(s) which are involved in specific aspects of amygdala functioning, in particular coding punishment information. Third, I will consider other neural systems that may be dysfunctional in psychopathy, in particular, the extent of orbital frontal cortex dysfunction in psychopathy. Within these second and third issues, I will develop a neurocognitive account of psychopathy, the Integrated Emotion Systems model. Fourth, I will consider the dissimilarities between psychopathy and another disorder linked to impairment in social cognition, autism. Fifth, I will consider psychopathy with respect to the neuropsychological approach to developmental disorders. I will point out the considerable benefit the study of psychopathy has gained from the neuropsychological approach. However, I will also point out the limits of the neuro-psychological approach; specifically, the pathology associated with psychopathy is not equivalent to that seen subsequent to an amygdala lesion.

1.2. What is the ultimate cause of psychopathy?

Growing evidence suggests a genetic contribution to psychopathy. Early twin, adoption, and family studies indicated that antisocial behavior was heritable (Rhee & Waldman, 2002). However, such studies are difficult to interpret. Much antisocial behavior is goal directed: the individual mugs the victim for their wallet, the individual steals the bag to obtain its contents, the individual engages in an elaborate sting operation to gain another person’s money. It is extremely unlikely that there is a direct genetic contribution to these behaviors or at least it is as likely as there is a direct genetic contribution to the use of a light switch to enable the navigation of a dark room. An individual might learn to use a light switch and under particular conditions, alternatively he/she might learn to mug people for their wallets. However, where genetics are likely to play a role is in determining the probability that the individual will learn an antisocial strategy to gain money (mugging other people) as opposed to a strategy sanctioned by society (using an ATM machine at the end of the workday). Many individuals have argued that the emotional dysfunction shown by individuals with psychopathy makes them more likely to learn antisocial strategies to reach goals (Blair, 1995; Eysenck, 1964; Lykken, 1995; Trasler, 1973). The argument developed here is that there may be a genetic contribution to the emotional dysfunction and that it is this which results in an apparent genetic contribution to antisocial behavior. Recent data suggests that this is indeed the case.

Blonigen, Carlson, Krueger, and Patrick (2003) collected data from 353 adult male twins using the self-report Psychopathic Personality Inventory [PPI] (Lilienfeld & Andrews, 1996). The PPI includes 163 items and forms a global index of psychopathy with eight sub-scales. Most of these subscales showed moderate heritability ($h^2 = 0.29–0.56$) and negligible shared environmental influence (Blonigen et al.,
Moreover, in a considerably larger study, examining almost 3500 twin pairs within the Twins Early Development Study (TEDS), the callous and unemotional component of psychopathic tendencies was indexed at age 7 (Viding, Blair, Moffitt, & Plomin, 2005). This study revealed a significant group heritability of $h^2_g = .67$ and no shared environmental influence on the callous-unemotional component; i.e., genetic factors account for two thirds of the difference between the callous-unemotional probands and the population.

As yet, the roles of environmental influences on the emergence of psychopathy remain unclear. The genetics studies above suggested relatively little influence. However, there is a considerable literature indicating a relationship between socio-economic status (SES) and antisocial behavior (Raine, 1993). With respect to psychopathy, it would be surprising if social variables did not impact on the probability of antisocial behavior; SES, for example, is likely to constrain the possibility of alternative behavioral choices to antisocial behavior as well as increase the salience of the money contained in a potential victim’s wallet. Indeed, in line with this, a relationship between SES and the antisocial behavior component of psychopathy has been reported (Hare, 2003). Thus, genetics would determine the level of emotional dysfunction while the environment would influence how this genetically determined emotion dysfunction was expressed.

1.2.1. Summary

It is likely that there is a genetic contribution to psychopathy. I argue that this contributes to the emotional dysfunction that is the basis of the disorder. The emotional dysfunction, in turn, puts the individuals for learning antisocial behaviors to solve goals. Social environmental variables are likely to have an influence, most importantly constraining the possibility of alternative behavioral choices to antisocial behavior as well as potentially increasing the salience of the rewards the might accrue through antisocial behavior. In the following section, I will consider the neural and neuro-chemical architecture upon which this genetic contribution may be operating.

1.3. The basic threat response

Three basic responses to threat are freezing, escape behaviors and reactive aggression (a rage attack at the threat); (Blanchard, Blanchard, & Takahashi, 1977). Which behavioral response is initiated is determined by the threat’s intensity and proximity. At low levels of intensity, from a distant threat, the animal will freeze. At higher levels, from a closer threat, the animal will attempt to escape the environment. At higher levels still, when the threat is very close and escape is impossible, the animal will display reactive aggression (Blanchard et al., 1977).

These responses to threat are mediated by a neural circuit that is common to many mammalian species (Gregg & Siegel, 2001; Panksepp, 1998). It runs from medial amygdaloidal areas downward, largely via the stria terminalis to the medial hypothalamus, and from there to the dorsal half of the periaqueductal gray (PAG). With respect to the basic response to threat, these systems function as a unitary
entity. Stimulation of any of these systems may induce the basic threat responses. Which one is elicited is determined by the level of stimulation.

An important neuro-chemical response to threat involves the noradrenergic system (Charney, 2003; Francis & Meaney, 1999). When specific neurons in the central nucleus of the amygdala are activated by threat, they activate, in turn, the locus coeruleus, leading to an increase in noradrenaline release. Rogers and others have argued that noradrenaline influences the salience of aversive cues (Rogers, Lancaster, Wakeley, & Bhagwager, 2004); greater noradrenaline levels should allow faster learning about information associated with the aversive cues.

With regards to psychopathy, I suggest that the genetic anomalies potentially present in individuals with psychopathy (Blonigen et al., 2003; Viding et al., 2005), affect particularly a component of this circuit; the amygdala. I argue that this leads to an individual who is less responsive to aversive stimuli. I will consider here two possibilities regarding how this might occur. First, the genetic anomalies disrupt the functioning of the amygdala (Blair, 2001, 2002; Blair, Morris, Frith, Perrett, & Dolan, 1999; Patrick, 1994). Second, the genetic anomalies disrupt the functioning of specific neurotransmitter(s) which are involved in specific aspects of amygdala functioning, in particular coding punishment information (Blair, Leonard, Morton, & Blair, 2006a; Blair et al., 2006b).

1.3.1. Summary

The mammalian basic response to threat is mediated by a circuit that runs from the amygdala to the hypothalamus and from there to the peri-aqueductal gray. An important neuro-chemical response to threat involves the noradrenergic system. In the following sections we will consider whether the genetic contribution to psychopathy disrupts the functioning of the amygdala or, perhaps, disrupts the functioning of specific neurotransmitter(s) which are involved in specific aspects of amygdala functioning.

1.4. Psychopathy and amygdala dysfunction

The suggestion that psychopathy might be due to early amygdala dysfunction has generated considerable research. In line with the suggestion, individuals with psychopathy show reduced amygdaloid volume relative to comparison individuals (Tiibonen et al., 2000) and reduced amygdala activation during emotional memory (Kiehl et al., 2001) and aversive conditioning tasks (Veit et al., 2002). Moreover, the position’s origins in the neuropsychological approach to developmental disorders (Baron-Cohen, 1998; Frith, 2002; Leslie, 1992) has allowed the generation of a variety of predictions regarding the functional impairment. Thus, human and animal neuropsychological work has informed us that the effects of amygdala lesions include impairment in: (1) aversive conditioning (Bechara, Damasio, Damasio, & Lee, 1999; Bechara et al., 1995; LaBar, Gatenby, Gore, LeDoux, & Phelps, 1998); (2) the augmentation of the startle reflex to visual threat primes (Angrilli et al., 1996; Funayama, Grillon, Davis, & Phelps, 2001); (3) passive avoidance learning (Ambrogi Lorenzini, Baldi, Bucherelli, Sacchetti, & Tassoni, 1999); and (4)
expression recognition that is particularly marked for fearful expressions (Adolphs, 2002; Blair, 2003a). If psychopathy is associated with amygdala dysfunction, the neuropsychological approach would predict that individuals with psychopathy are impaired in the above tasks. Considerable data shows that they are (Blair, Colledge et al., 2001; Blair, Colledge, Murray et al., 2001; Blair, Monson et al., 2001; Flor et al., 2002; Herpertz et al., 2001; Levenston et al., 2000; Lykken, 1957; Newman & Kosson, 1986).

Not only has the amygdala dysfunction position generated a host of predictions it has also allowed the reconciliation of fear (Lykken, 1995; Patrick, 1994) and empathy (Blair, 1995) dysfunction positions. Many of the findings associated with both positions are direct results of amygdala dysfunction (see above). Moreover, it becomes easier to understand findings indicating the importance of “fearfulness” as an important temperamental factor in socialization (Kochanska, 1993, 1997). The temperamental variable “fearfulness” can be understood as an index of the integrity of the amygdala (Blair, 2003a). While fear conditioning is not considered to be important in socialization (Brody & Shaffer, 1982; Hoffman, 1994), I argue that the amygdala’s response to the fear and sadness of victims, during empathy induction, is crucial for socialization. Murray and colleagues argue that the amygdala is crucially involved in the formation of stimulus–reward and stimulus–punishment associations (Baxter & Murray, 2002). I argue that the associations required for appropriate socialization are associations between moral transgressions and victim’s distress. Empathy induction and other positive parenting techniques should aid the formation of associations between the moral transgression and the victim’s distress (empathy induction focuses the child’s attention on the aversive stimulus that is the victim’s distress). A considerable literature shows that these parenting techniques do reduce antisocial behavior in children (Brody & Shaffer, 1982). Individuals with psychopathy are less likely to form these associations and so should be more likely to engage in actions that will harm others. Indeed, interestingly, while positive parenting techniques are associated with reduced antisocial behavior levels in healthy children, they have no impact on the level of antisocial behavior expressed by children who present with the emotional dysfunction associated with psychopathy (Wootton, Frick, Shelton, & Silverthorn, 1997).

Despite the success of the amygdala dysfunction position, two caveats should be considered here. First, very few patients with amygdala lesions acquired in adulthood present with instrumental aggression [though this may not be the case if the lesion occurs early in life (Fine, Lumsden, & Blair, 2001)]. Of course, this should be expected. My argument is that the amygdala is required for the formation of moral transgression–victim’s distress associations. However, typically, goal directed behavior does not require the accessing of these associations. Instead, these moral transgression–victim’s distress associations resulted in the individual failing to learn antisocial behaviors to achieve their goals. The behaviors the individual does use to achieve their goals will be a function of their learning history. While the individual with psychopathy might learn that a solution to a lack of money was to mug another (rewarded by the money but not punished by the victim’s distress), the individual with an acquired amygdala lesion would have learnt, before their lesion, that this
was not appropriate (such actions were punished by the victim’s distress). When considering how to rectify a lack of money, the individual with an acquired amygdala lesion would activate previously successful motor programs for achieving this goal (a visit to an ATM).

The second caveat is slightly more problematic. As noted above, the amygdala is crucially involved in the formation of stimulus–reward and stimulus–punishment associations; animals with amygdala lesions show impairment in both reward and punishment related behavior (Baxter & Murray, 2002). Yet, the impairment in individuals with psychopathy is far more marked for processing dependent on stimulus–punishment associations than for stimulus–reward associations (Levenston et al., 2000; Patrick, 1994; Blair et al., 2006a, 2006b). Thus, whereas individuals with psychopathy do not show augmentation of the startle reflex following a negative visual prime relative to comparison individuals, they do show a comparable reduction in startle reflex following a positive visual prime relative to comparison individuals (Levenston et al., 2000; Patrick, 1994). Moreover, in an affective priming study, Peczschhardt et al. found that whereas individuals with psychopathy showed no congruence facilitation for negative target words, they did show congruence facilitation for positive target words (Blair et al., 2006a). In other words, while a positive prime primed the output response associated with positive valence in individuals with psychopathy (albeit less so than in comparison individuals), a negative prime did not prime the output response associated with negative valence. Finally, in a decision making study, individuals with psychopathy showed particular difficulty, relative to controls, when choosing between stimuli associated with different levels of punishment. Their impairment in choosing between stimuli associated with different levels of reward was far less marked (Blair et al., 2006a).

In addition to the above, the amygdala has also been considered to play a role in certain aspects of social cognition, at least in humans and possibly primates; in particular, affect-related judgments based on facial stimuli (Adolphs, 2003; Baron-Cohen et al., 2000). Thus, in one paradigm, participants were shown pictures of individuals in natural poses and asked to judge the trustworthiness of these individuals. While healthy individuals typically judge some individual’s faces less trustworthy than others, patients with amygdala lesions present with atypical judgment patterns (Adolphs, Tranel, & Damasio, 1998). Moreover, recent neuro-imaging work has indicated that healthy individuals show greater amygdala activation to faces judged to be untrustworthy relative to faces judged to be trustworthy (Winston, Strange, O’Doherty, & Dolan, 2002). In a second paradigm, participants must judge the complex social emotion being displayed by an individual based on information only from the eye region (Baron-Cohen, Wheelwright, & Joliffe, 1997). Individuals with amygdala lesions show impairment on this task (Adolphs, Baron-Cohen, & Tranel, 2002; Stone, Baron-Cohen, Calder, Keane, & Young, 2003) while neuro-imaging work has indicated the involvement of the amygdala whilst performing this task (Baron-Cohen et al., 1999). However, despite this apparent role of the amygdala in these two aspects of social cognition, individuals with psychopathy are not impaired either in making trustworthiness judgments (Richell et al., 2005) or in judging complex social emotions from the eyes (Richell et al., 2003).
1.4.1. Summary

Individuals with psychopathy are impaired on many tasks that require the functional integrity of the amygdala. Both the impairments in fear-related and “empathic” responding found in individuals with psychopathy can be attributed to amygdala dysfunction. In short, predictions developed from human and animal neuropsychological work with respect to amygdala dysfunction in individuals with psychopathy have been useful with respect to the impairment in emotional learning and basic emotional responding seen in individuals with psychopathy. However, individuals with psychopathy are not equivalent to patients with amygdala lesions; functions that appear to require the amygdala such as the formation of stimulus–reward associations and certain aspects of social cognition are instead only mildly, or not at all, impaired in individuals with psychopathy. This suggests that the genetic anomalies present in individuals with psychopathy do not lead to the development of the disorder through a global disruption of the functioning of the amygdala. Instead, they may have a more selective effect, disrupting the functioning of specific neurotransmitter(s) which are involved in specific aspects of amygdala functioning. This will be considered in the following section.

1.5. Psychopathy, neurotransmitter dysfunction and developing the neuro-cognitive model

Polymorphisms of particular genes can alter the functioning of specific neurotransmitter systems (Lichter et al., 1993; Shih, Chen, & Ridd, 1999; Vandenbergh et al., 1992). Currently, it remains unclear which neurotransmitter systems might be dysfunctional in individuals with psychopathy. However, one possibility is that the noradrenergic response to stress/threat stimuli, described above, is disturbed in individuals with psychopathy (Blair, 2003a; Blair et al., 2006b). Interestingly, there have been recent suggestions that noradrenaline is involved in mediating the impact of aversive cues in human choice (Rogers et al., 2004). Moreover, recent pharmacological data imply that noradrenergic manipulations selectively impact on the processing of sad expressions (Harmer, Perrett, Cowen, & Goodwin, 2001; Sustrik, Coupland, & Blair, in preparation). Further support for this suggestion comes from studies linking NA abnormalities to antisocial behavior/Conduct Disorder (Raine, 1993; Rogeness, Cepeda, Macedo, Fischer, & Harris, 1990; Rogeness, Javors, Mass, & Macedo, 1990). In this regard it is interesting to note that NA function appears to be increased in a range of anxiety disorders (Charney, Heninger, & Breier, 1984); i.e., it is increased in populations that present with a heightened responsiveness to aversive cues, the opposite of the emotional impairment seen in psychopathy. I therefore argue that the genetic anomalies considered to be present in individuals with psychopathy disrupt the functioning of the noradrenergic system such that the impact of aversive stimuli is muted.

The integrated emotion systems (IES) model represents a neuro-cognitive model of the interactions of the systems involved in emotional processing (Blair, 2004). Initial components of this model, those most related to the earlier VIM model, are depicted in Fig. 2. Three systems are depicted. One allows sensory representations;
e.g., the representation of objects currently in the environment, including moral transgressions. At the neural level, this system is primarily implemented by temporal cortex. The second contains “valence representations”; i.e., representations that can be activated by unconditioned stimuli (e.g., loud noises but also distress cues). At the neural level, this system is implemented by the amygdala. The third will be discussed below.

The “valence representations” can represent both appetitive and aversive stimuli following claims that the amygdala is crucially involved in the formation of stimulus–reward and stimulus–punishment associations (Baxter & Murray, 2002). The strength of the connections between representations in the different systems increase through Hebbian learning (Hebb, 1949); if two representations in different systems are simultaneously active the connection between these two representations will strengthen. In short, activation of a “valence representation” by an observed distress cue will increase the connection between this “valence representation” and currently active sensory representations (perhaps a representation of the moral transgression that has caused this distress cue). This computation was also the basis of the VIM model (see above and Fig. 1). The connections between the representations in the different systems are reciprocal, reflecting the interconnections of the amygdala with
cortical regions (Amaral, Price, Pitkanen, & Carmichael, 1992). These reciprocal connections allow the valence representations to bias the processing of emotional sensory representations (an emotional sensory representation will activate the valence representations, leading to greater activation of the emotional sensory representation). In short, activation of the valence representations can increase attention to the sensory representation which activated them.

If we assume that the genetic anomalies considered to be present in individuals with psychopathy disrupt the functioning of the noradrenergic system such that negative valence representations are less activated by aversive stimuli, we can predict many of the behavioral phenomena associated with psychopathy. Aversive conditioning will be impaired, and passive avoidance errors will be more common, because sensory representations will be more weakly associated with negative valence (Flor et al., 2002; Lykken, 1957; Newman & Kosson, 1986). Reduced activation of negative valence representations by sad and, particularly, fearful expressions will result in reduced autonomic responses to these expressions and may impair the naming of these expressions (Aniskiewicz, 1979; Blair, 1999; Blair et al., 1997; House & Milligan, 1976; Stevens, Charman, & Blair, 2001).

The third system depicted in Fig. 2 mediates the production of motor responses (and, at the neural level, is implemented by basal ganglia and motor cortex). The valence representations are not involved in the formation of stimulus–response associations (the linking of sensory representations to motor responses). At the neural level, it is known that the amygdala is not necessary for the formation of stimulus–response associations (Baxter & Murray, 2002). Instrumental learning tasks reliant on stimulus–response associations, such as object discrimination, are not impaired by amygdala lesions while instrumental learning tasks that are reliant on stimulus–reinforcement associations (within the IES, the linking of sensory representations with valence representations), such as passive avoidance are (Baxter & Murray, 2002). Interestingly, and in line with the suggestion of disruption of the valence representations, individuals with psychopathy are not impaired on object discrimination tasks (Mitchell et al., 2002) but are impaired on passive avoidance learning (Newman & Kosson, 1986).

1.5.1. Summary
In this section, I have suggested that the genetic contribution to psychopathy may influence the functioning of the noradrenergic system such that the formation of stimulus–reinforcement, particularly stimulus–punishment, associations is impaired. I have also begun to develop the neuro-cognitive Integrated Emotion Systems model.

1.6. Psychopathy and orbital frontal cortex

Orbital frontal cortex is considerably involved in the regulation and mediation of emotional behavior. Patients with orbital frontal cortex lesions may present with emotional dys-regulation and antisocial behavior (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Blair & Cipolotti, 2000; Blumer & Benson, 1975; Damasio, 1994; Pennington & Bennetto, 1993). This has led to considerable claims that
psychopathy might be due to early orbital frontal cortex damage and that patients with orbital frontal cortex damage and psychopathy present similarly (Damasio, 1994). Such claims have received questionable empirical support however.

Orbital frontal cortex does play a role in the regulation of the brainstem systems involved that mediate the basic responses to threat (including reactive aggression); (Gregg & Siegel, 2001; Panksepp, 1998). Damage to orbital frontal cortex should thus lead to a dys-regulation of brainstem systems involved in mediating the basic responses to threat and potentially increase the risk of threat/ frustration-based reactive aggression. Patients with lesions of orbital frontal cortex do indeed display a heightened risk of reactive aggression (Anderson et al., 1999; Blair & Cipolotti, 2000; Pennington & Bennetto, 1993). However, patients with lesions of orbital frontal cortex do not display a heightened risk of goal-directed, instrumental aggression, even if their lesions occur in early childhood (Anderson et al., 1999; Pennington & Bennetto, 1993). Yet, individuals with psychopathy are particularly notable for their use of instrumental aggression (Cornell et al., 1996; Williamson et al., 1987). Moreover, empirically, while patients with orbital frontal cortex lesions display generally impaired expression recognition (Blair & Cipolotti, 2000; Hornak, Rolls, & Wade, 1996), individuals with psychopathy do not show such general impairment but rather a relatively more selective impairment for fearful, and to a lesser extent, sad expressions (Blair, Colledge et al., 2001; Blair, Colledge, Murray et al., 2001; Blair, Monson et al., 2001). Additionally, findings such as the impairment in aversive conditioning shown by individuals with psychopathy (Flor et al., 2002; Lykken, 1957) are not found in patients with orbital frontal cortex lesions (Bechara et al., 1999).

This is not to suggest that individuals with psychopathy are without impairment for functions in which orbital frontal cortex is involved. There is a considerable neuropsychological and neuro-imaging literature demonstrating that orbital frontal cortex is crucially involved in response reversal and extinction (Cools, Clark, Owen, & Robbins, 2002; Rahman, Sahakian, Hodges, Rogers, & Robbins, 1999; Rolls, Hornak, Wade, & McGrath, 1994). Response reversal and extinction involve changing a response to a stimulus as a function of a change in contingency; i.e., learning to withhold a response that is now punished though previously it had been rewarded (Rolls, 1997). The difference between response reversal and extinction is that in response reversal tasks, the participant learns to respond to a second stimulus rather than the first while in extinction tasks the participant learns to withhold responding all together.

In Fig. 3, the depiction of the IES model is extended to include two systems influenced by input from the valence representations and therefore likely to be disrupted in individuals with psychopathy. The first of these, the response selection system, represents expected level of reinforcement associated with the stimulus (and, potentially, the form of response to this stimulus), information that is provided by the valence representations. If two or more stimuli are present in the environment, each of which is associated with a different response, it is important that the system can rapidly select the stimulus that will elicit the greatest level of reward. The response selection system allows this to occur (the computational details of this process are described elsewhere, Blair, 2004). At the neural level, this system is implemented
The second, the response gating system, is particularly activated when expected reinforcement information, provided by the response selection system, differs from actually received reinforcement. In Fig. 3, I propose that this system alters stimulus–response associations (the connections between the sensory representation and motor response systems, that terminating in small circles represent the gating by the response gating system of responses activated by these connections. CS = Conditioned stimulus; US = Unconditioned stimulus; $S_1$ and $S_2$ are competing sensory representations; $R_1$ and $R_2$ are alternative motor responses. The computational details of response selection are described elsewhere (Blair, 2004).
reinforcing; if the participant plays the card he/she will win points or money. However, as the participant progresses through the pack of cards, their probability of reward decreases. The participant should terminate his/her responding before he/she receives greater levels of punishment than reward. Children with psychopathic tendencies, like adults with psychopathy, are impaired on this task (Fisher & Blair, 1998; Newman et al., 1987; O’Brien & Frick, 1996). In the ID-ED task (Dias, Robbins, & Roberts, 1996), the participant learns that responding to one of two stimuli gains reward while responding to the other is punished. This contingency is then reversed; i.e., responding to the first stimulus is no longer rewarded but punished while responding to the second is now rewarded. While adults with psychopathy are impaired on this task, children with psychopathic tendencies are not (Blair, Colledge et al., 2001; Blair, Colledge, Murray et al., 2001; Blair, Monson et al., 2001; Mitchell et al., 2002). A major difference between these two tasks is in the salience of the contingency change. In the card-playing task, the probability of reinforcement decreases by 10% over every ten trials. In the ID-ED task, the probability of reinforcement changes from 100% to 0% once the initial learning criterion has been achieved. This indicates that while both children with psychopathic tendencies and adult individuals with psychopathy are impaired in the detection of contingency change, this impairment is markedly greater in the adult individuals with psychopathy. Moreover, this suggests that if we reduce the salience of the contingency change, we should see impairment in the children with psychopathic tendencies and that the degree of impairment will be a function of the salience of the contingency change. This was tested using a probabilistic response reversal paradigm. Participants were presented with pairs of stimuli. The probability of reward was different across pairs (i.e., for pair 1, stimulus A was rewarded 100% of the time, for pair 2, stimulus C was rewarded 90% of the time etc). Following a set number of trials the contingency was reversed (i.e., for pair 1, stimulus B was rewarded 100% of the time, for pair 2, stimulus D was rewarded 90% of the time). While the children with psychopathic tendencies showed no difficulty reversing their responses for salient contingency changes [pair 1], they did show significant difficulty as the salience of the contingency change decreased [pair 2] (Budhani & Blair, 2005).

This difference between children and adults with psychopathy is of interest. Response reversal and extinction occur following the successful identification of violations between expected reward and received punishment (or absence of reward). Failure to perform response reversal and extinction could be due to impairment in systems necessary for: (1) detecting the violation; (2) changing the stimulus–reward associations; or (3) representing the punishment. We have argued that there is reduced punishment sensitivity in individuals with psychopathy. Children with psychopathic tendencies seem to be at least as insensitive to punishing stimuli as adults with psychopathy. They are comparably impaired on tasks such as passive avoidance learning (Newman & Kosson, 1986; Newman, Widom, & Nathan, 1985), the processing of fearful expressions (Blair, Colledge et al., 2001; Blair, Colledge, Murray et al., 2001; Blair, Monson et al., 2001) and aversive conditioning (Lykken, 1957;
However, the impairment of children with psychopathic tendencies for response reversal is far less marked. This suggests that adults with psychopathy are impaired in systems necessary for detecting the violation/changing the stimulus–reward associations; i.e., orbital/ventrolateral frontal cortex dysfunction.

Given the evidence of selective amygdala dysfunction discussed above, there are several possibilities regarding the origins of the orbital frontal cortex pathology found in adults with psychopathy. First, the orbital frontal cortex pathology could be developmentally independent of the amygdala pathology. For example, the genetic anomalies associated with psychopathy might affect the development of the amygdala and orbital frontal cortex independently of one another. Second, there are considerable interconnections between the amygdala and orbital frontal cortex (Amaral et al., 1992; Carmichael & Price, 1995). It is possible that a lack of afferent input from the amygdala to orbital frontal cortex could disrupt the development of orbital frontal cortex to an increasingly greater degree as development progresses. Thirdly, individuals with psychopathy present with higher levels of drug abuse, dependence, and poly-drug use than comparison individuals (Hemphill, Hart, & Hare, 1994; Smith & Newman, 1990). Alcohol and drug dependent individuals are impaired on measures assessing the functioning or orbital frontal cortex (Bechara et al., 2001; Grant, Contoreggi, & London, 2000; Rogers & Robbins, 2001). It is thus also possible that the apparent developmental effect seen here is simply a consequence of the lifestyle chosen by psychopathic individuals.

1.6.1. Summary

Damasio and others have suggested that psychopathy might be due to orbital frontal cortex damage. However, recent data suggest that the deficits expected from orbital frontal cortex damage do not mirror those seen in individuals with psychopathy. This is not to suggest that there is no orbital frontal cortex dysfunction in psychopathy. Children and adults with psychopathic tendencies show impairment in response reversal, a function known to recruit orbital frontal cortex. Interestingly, while the children and adults with psychopathic tendencies perform similarly with respect to their lack of guilt for their actions and on measures of, or based on, stimulus–reinforcement learning, the impairment in response reversal is far more marked in adults with psychopathy than children with psychopathic tendencies. This suggests a developmental progression to the orbital frontal cortex pathology found in psychopathy. The reasons for this progression remain unclear.

1.7. Psychopathy and autism

Psychopathy is not the only disorder linked to impairments in social cognition. Autism is a severe developmental disorder where there is marked neuro-cognitive impairment. It is described by the American Psychiatric Association’s Diagnostic and Statistical Manual (DSM-IV, American Psychiatric Association, 1994) as ‘the presence of markedly abnormal or impaired development in social interaction and
communication and a markedly restricted repertoire of activities and interests’ (p. 66). Like psychopathy, autism has also been linked to impairments in amygdala functioning (Baron-Cohen et al., 2000).

Individuals with autism may show structural amygdala abnormalities. However, these have been reported as an increase in grey matter volume in the amygdala/peri-amygdaloid cortex (Abell et al., 1999) rather than the decrease seen in individuals with psychopathy (Tiihonen et al., 2000). Functionally, the situation is complex. As discussed above, the amygdala is involved in the formation of stimulus–punishment and stimulus–reward associations as well as some affect laden components of social cognition. My colleagues and I argue that the sensitivity of individuals with psychopathy to punishment, and therefore the ability to form stimulus–punishment associations, is disproportionately impaired (Blair et al., 2006a). However, there is no reason to believe that this is the case in individuals with autism. Indeed, individuals with autism are at increased risk of presenting with anxiety (Gillott, Furniss, & Walter, 2001; Rumsey, Rapoport, & Sceery, 1985), rather than the decreased risk seen in individuals with psychopathy (Frick, Lilienfeld, Ellis, Loney, & Silverthorn, 1999; Patrick, 1994; Verona, Patrick, & Joiner, 2001). In contrast, there may be impairments in aspects of social cognition that involve the amygdala in individuals with autism. Thus, patients with autism, unlike individuals with psychopathy, are impaired on both the Eyes task and the face trustworthiness judgment tasks discussed earlier (Adolphs, Sears, & Piven, 2001; Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001). Of course, these impairments may not reflect amygdala dysfunction per se. It is possible that they are developmental consequences of impoverished face representations due to dysfunction in regions such as fusiform gyrus and superior temporal sulcus (Schultz et al., 2003).

1.7.1. Summary

Autism, like psychopathy, is associated with impairment in social cognition and, at the anatomical level, amygdala dysfunction. However, while psychopathy is associated with impairment in the formation of stimulus–reinforcement associations, a function that animal and human work has clearly indicated amygdala involvement in, there is little reason to suspect such impairment in autism. In contrast, several aspects of social cognition that have been related to amygdala functioning, though which may reflect the functioning of nearby neural systems, are impaired in autism but are not impaired in individuals with psychopathy.

1.8. Psychopathy and the neuropsychological approach to developmental disorders

The model that I have developed here follows a tradition of presupposing the existence of relatively independent neuro-cognitive systems that may be selectively impaired in developmental disorders (Baron-Cohen, 1998; Frith, 2002; Leslie, 1992). With respect to the predictions of the position, I have taken care taken to consider neuropsychological findings with adult lesions cases and, sometimes, to use tasks that have been previously used with these patients (Blair et al., 2002; Blair, Colledge et al., 2001; Blair, Colledge, Murray et al., 2001; Blair, Monson et al.,
Indeed, it was this neuropsychological approach which allowed the reconciliation of the fear-relevant and empathy-relevant data described above; most could be accounted for by assuming dysfunction in systems involved in emotional processing, in particular the amygdala. However, this approach has been recently criticized and sometimes considered to be not developmental (Karmiloff-Smith, Brown, Grice, & Paterson, 2003; Paterson, Brown, Gsodl, Johnson, & Karmiloff-Smith, 1999). In short, Karmiloff-Smith and colleagues argue that the existence of separable neuro-cognitive systems in the adult does not imply that these separable systems are present in the young child. They suggest that the separable systems seen in the adult may be constructed through development and may not be discrete systems at birth.

I do not propose to debate these issues fully here. The systems at which these attacks are made (systems for number and language processing) are usually cortically represented as opposed to the interaction of cortical and sub-cortical systems considered here. Whether or not this dichotomy is important, it is clear that such criticisms must be tempered when considering psychopathy. Work with animals allows considerable specification of the emotional systems that are dysfunctional in individuals with psychopathy as well as their development. Thus, let us consider the basic neural architecture for emotional processing; i.e., the amygdala, hypothalamus and periaqueductal gray. This neural architecture is present from birth, primarily genetically determined, and highly similar to that found in other mammalian species. In short, it is not constructed from environmental experience. Of course, this does not mean that this architecture functions equivalently in the child and adult or that environmental effects do not affect its functioning. Interestingly, animals during infancy do not demonstrate HPA axis responses to stress. However, infant animals exposed to stressors demonstrate increases in the expression of immediate early genes (e.g., c-fos and nerve growth factor inducible gene) in the paraventricular nucleus of the hypothalamus (Charney, 2003). In short, early postnatal adverse experiences increase the responsiveness of these stress response systems, effects which last throughout the lifetime of the individual (Heim, Owens, Plotsky, & Nemeroff, 1997; Liu et al., 1997; Plotsky & Meaney, 1993). Thus, the environment has an effect, by partially determining the responsiveness of these basic threat systems. However, it is important to again note here that this neural architecture is not constructed out of environmental experience [in contrast to processes proposed to underlie the development of some cortical neuro-cognitive systems (Karmiloff-Smith et al., 2003; Paterson et al., 1999)], it is present from birth.

But more generally, how appropriate is a neuropsychological approach when considering psychopathy? In many respects, the empirical literature on psychopathy demonstrates the clear advantages of the neuropsychological approach. However, at the same time, it identifies the need for caution. Following the neuropsychological approach, and assuming amygdala dysfunction, has allowed the generation of an array of predictions, many of which have been confirmed. The amygdala is involved in the formation of stimulus–punishment associations (Baxter & Murray, 2002) and individuals with psychopathy are impaired in a variety of tasks dependent on the formation of stimulus–punishment associations. However, the amygdala is involved in
the formation of stimulus–reward associations and certain affect-relevant aspects of social cognition. Individuals with psychopathy show far less impairment in these aspects of amygdala functioning. Thus, the impairment in psychopathy is not equivalent in impact to the effects of an amygdala lesion to a healthy adult.

However, it should be noted that even results where data obtained from individuals with psychopathy differs from that of patients with amygdala lesions are informative. Thus, the neuro-psychological and neuro-imaging literatures indicate a role of the amygdala in specific affect-relevant aspects of social cognition. Faces judged to be untrustworthy activate the amygdala (Winston et al., 2002) and patients with amygdala lesions are poor at judging a face’s trustworthiness (Adolphs et al., 1998). The neuropsychological approach might therefore predict problems in trustworthiness judgments in individuals with psychopathy but this prediction is not supported (Richell et al., 2003). This suggests either that the ability to make trustworthiness judgments involves neuro-cognitive systems independent of those involved in stimulus–punishment association formation (taking a refined neuro-psychological approach) or that individuals with psychopathy are making trustworthiness judgments using a neuro-cognitive architecture that is very different from that used by healthy individuals. The predictions of these contrasting positions are clear. The first suggests that individuals with psychopathy will show appropriate amygdala responses to untrustworthy faces even if their amygdala responses during aversive conditioning are reduced (Veit et al., 2002). The second suggests that the neural responses of individuals with psychopathy to untrustworthy faces will be very different from those of comparison individuals. The data to resolve these contrasting predictions should soon be available.

1.8.1. Summary

The study of psychopathy has gained considerably from the neuro-psychological approach. Predictions generated from following this approach have been systematically confirmed allowing a relatively tight specification of the impairment at the basis of this disorder. However, the neuropsychological approach cannot be followed slavishly. Psychopathy is not the equivalent to an amygdala lesion. The deficits in psychopathy are only a subset of those functions mediated by the amygdala.

1.9. Conclusions

The main goal of this paper was to consider how genetic anomalies could give rise to the relatively specific neuro-cognitive impairments seen in individuals with psychopathy. With respect to this goal, the suggestion is that genetic anomalies reduce the salience of punishment information (perhaps as a function of noradrenergic disturbance). This impairs various aspects of amygdala function, most importantly the ability to form stimulus–punishment associations. In order to achieve successful socialization, the child needs to form associations between representations of moral transgressions (acts which harm others) and the aversive ‘punishment’ caused by the victim’s distress. This allows the child to learn to avoid actions that will harm others. As individuals with psychopathy find the distress
of the victim significantly less aversive, they are less likely to learn to avoid actions that will harm others.

Why should the genetic effects operate so selectively, affecting primarily the salience of punishment information? It is likely that if a neurotransmitter such as noradrenaline is implicated in psychopathy, it may serve more functions than simply the coding of the salience of punishment information. This may mean that other systems are affected in individuals with psychopathy. Dysfunction in these other systems may have no causal role in the development of the core emotional aspects of psychopathy but may contribute to the full behavioral profile of the disorder. Certainly, by analogy, some individuals with autism show executive dysfunction in addition to their difficulties with Theory of Mind. While the executive dysfunction does not appear to be related to their social difficulties, it does appear to be related to other components of the behavioral profile such as their repetitive behavior (Turner, 1997). Individuals with psychopathy may show certain attentional abnormalities (Jutai & Hare, 1983; Raine & Venables, 1988). The role of these difficulties in the development of the emotional component of psychopathy remains unclear. However, it is possible that they reflect the more general influences of genetically mediated neurotransmitter anomalies. Alternatively, of course, the genetic anomalies may influence the functioning of one particular neurotransmitter receptor. Certain neurotransmitter receptors appear to be far more neural location specific than others. If this possibility is the case, the relatively selective nature of the deficits seen in individuals with psychopathy may reflect, for example, the impact of genetic anomalies on the density of particular neurotransmitter receptors in the amygdala crucial for punishment coding. At present, it is impossible to distinguish between these speculations. However, future work will allow the situation to be resolved.

The model that has been developed here follows a tradition of pre-supposing the existence of relatively independent neuro-cognitive systems that may be selectively impaired in developmental disorders. This neuropsychological approach has been recently criticized and sometimes considered to be not developmental. However, I would argue, at least with respect to psychopathy, and particularly given animal data on the development of the neural systems mediating emotion, that these criticisms are unfounded. I suggest that the neuropsychological approach will continue to be an invaluable tool with respect to the understanding, and ultimately the cure, of this pernicious disorder.

References


