Understanding the development of the psychopathic individual:

An affective cognitive neuroscience approach

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Introduction

Psychopathy is characterized by callousness, a diminished capacity for remorse, superficial charm, proneness to boredom and poor behavioral control (Cleckley, 1967; Hare, 1991). Psychopathic individuals commit a disproportionate amount of crime, habitually fail to fulfill societal obligations and are unperturbed when confronted with the destructive consequences of their behavior.

The purpose of this chapter is to provide a model of the development of psychopathy from the perspective of affective cognitive neuroscience. The fundamental suggestion of this chapter is that psychopathy is linked to early amygdala dysfunction (Blair, 2001, 2002; Blair, Morris, Frith, Perrett, & Dolan, 1999; Patrick, 1994). In line with this suggestion, relative to comparison individuals, psychopathic individuals have been found to present with reduced amygdaloid volume (Tiihonen et al., 2000) and reduced amygdala activation during emotional memory (Kiehl et al., 2001) and aversive conditioning tasks (Veit et al., 2002). Moreover, functions that recruit the amygdala such as aversive conditioning and instrumental learning, the augmentation of startle reflex by visual threat primes and arousal to the anticipation of punishment, are all impaired in psychopathic individuals (Blair, 2001); see also below. Of course, it should be noted that other structures, such as orbital frontal cortex, which are interconnected with the amygdala, may also be affected (Damasio, 1994; LaPierre, Braun, & Hodgins, 1995; Mitchell, Colledge, Leonard, & Blair, 2002; Raine, 2002). In this chapter, I will attempt to specify the details of a model of psychopathy as tightly as is currently possible.

The amygdala and aversive conditioning

Aversive conditioning involves the individual acquiring negative valence information with respect to a novel stimulus. Thus, the novel stimulus (the conditioned stimulus; CS) is paired with a loud noise (the aversive unconditioned stimulus; US) so that the CS comes to elicit an unconditioned response (UR), for example, freezing behavior/ autonomic activity. The amygdala, and particularly the central nucleus of the amygdala, are known to be implicated in aversive conditioning (Davis, 2000; Killcross, Robbins, & Everitt, 1997; LeDoux, 1998). Animals and humans with lesions to the amygdala present with impairment in aversive conditioning (Bechara et al., 1995; Davis, 2000; Killcross et al., 1997; LaBar, LeDoux, Spencer, & Phelps, 1995; LeDoux, 1998).

INSERT FIGURE 1 ABOUT HERE

Figure 1 depicts a simplified version of a model of aversive conditioning (Armony, Servan-Schreiber, Romanski, Cohen, & LeDoux, 1997). In this model, two modules of non-linear, identical computational units are depicted, with one module representing the amygdala and one model representing sensory regions (e.g., auditory, visual and temporal cortex). In contrast to the original model (Armony et al., 1997), the connections between the units in the different modules are reciprocal, reflecting the interconnections of the amygdala with cortical regions (Amaral, Price, Pitkanen, & Carmichael, 1992). Units within a module are mutually inhibitory. The strength of the connections between units in the different modules increase as a function of the number of times that these units are simultaneously active through a process called

Hebbian learning (Hebb, 1949). Thus, for example, every time unit 1 is active in the amygdala module at the same time as unit 3 in the sensory cortex module, the strength of the connection between these units will increase as a function of the level of activation of the two units (Hebb, 1949). Recent data at the cellular level confirms this characterization of learning within the amygdala as Hebbian (Blair, Schafe, Bauer, Rodrigues, & LeDoux, 2001).

There are at least three possible explanations of the amygdala dysfunction shown by psychopathic individuals. First, there could be reduced nociceptive (US) input to the amygdala. This would result in reduced activation of the amygdala neurons and thus prevent learning. Secondly, the amygdala neurons of psychopathic individuals may be hypo-responsive; i.e., less likely to fire than the amygdala neurons of comparison individuals to a given level of input. This would again interfere with learning. Thirdly, there might be some cellular property of the amygdala neurons of psychopathic individuals such that they are less capable of Hebbian learning irrespective of their level of activation. While these explanations give rise to slightly different predictions, they are unlikely to be disentangled in the near future. However, functionally their impact would be similar. The amygdala's capacity to perform aversive conditioning would be detrimentally affected.

An impairment in aversive conditioning was one of the original impairments shown by psychopathic individuals in the earliest empirical work on the disorder and has demonstrated again several times since (Flor, Birbaumer, Hermann, Ziegler, & Patrick, 2002; Hare & Quinn, 1971; Lykken, 1957). Moreover, recent neuro-imaging work has demonstrated reduced amygdala activation in psychopathic individuals during aversive conditioning (Veit et al., 2002). Interestingly, Raine has reported a relationship between ability to perform aversive conditioning and the probability of a

healthy prognosis in boys at high risk for antisocial behavior (Raine, Venables, & Williams, 1996).

The architecture depicted in Figure 1 can also be used to account for data obtained through the anticipation of punishment paradigm. There are different forms of this paradigm where electro-dermal or neural activity may be related to stimuli that anticipate shock (Hare, 1965, 1982; Hare, Frazelle, & Cox, 1978; Ogloff & Wong, 1990; Phelps et al., 2001). Neuro-imaging work has shown that the amygdala responds to stimuli that the participants have been told anticipate shock (Phelps et al., 2001). Moreover, in studies where the participant has the seconds to the shock counted down to them, non-psychopathic individuals show greater autonomic responses as the shock becomes more imminent. However, in striking contrast, psychopathic offenders show smaller electro-dermal responses than non-psychopathic offenders and these occur much closer to the shock than those of the non-psychopathic offenders (Hare, 1965, 1982; Hare et al., 1978; Ogloff & Wong, 1990). Within the framework developed in Figure 1, stimuli that the participant learns, or is told, anticipate shock are CSs represented by the sensory cortical regions that activate the amygdala. In the countdown paradigms, the suggestion would be that as the countdown and the shock becomes more imminent, the CS becomes more salient (i.e., is activated more strongly) and correspondingly there is greater amygdala, brainstem and therefore autonomic activity.

As mentioned above, CSs come to activate the brainstem through the amygdala. Effectively, aversive CSs can prime (activate) brainstem neurons such that later nociceptive (US) input will give rise to greater firing of these brainstem neurons than would have occurred without the original aversive CS activation. Alternatively, appetitive CSs can actually suppress the activity of the brainstem neurons mediating

the response to threat. It is this priming of the brainstem neurons as a consequence of amygdala activation that is implicated in the augmentation of the startle response by threat primes (Davis 2000). Patients with amygdala lesions show reduced augmentation by threat primes (Angrilli et al. 1996) as do psychopathic individuals (Patrick et al. 1993; Levenston et al. 2000).

Words, as any other stimuli, can become aversive and appetitive CSs if they are paired with either aversive or appetitive USs. Thus, "murder" is an aversive CS and "love" is an appetitive CS. Such affective word stimuli generate a neural response within the amygdala (Hamann & Mao, 2002). In line with the amygdala dysfunction hypothesis developed here, while non-psychopathic individuals show autonomic responses to unpleasant or fearful experiences they have been asked to imagine, psychopathic individuals do not (Patrick, Cuthbert, & Lang, 1994).

As mentioned above, and illustrated in Figure 1, the interconnections of the amygdala with cortical regions are reciprocal (Amaral et al., 1992). Thus, activation of the amygdala by a linguistic CS such as the word "murder" will subsequently result in increased activation of the representation of this linguistic CS because of reciprocal activation from the amygdala. In short, emotional words are more salient than neutral words *ceterus paribus* because of these reciprocal connections and are thus likely to be processed more rapidly. It is likely that this drives the emotion effect in lexical decision tasks; non-psychopathic participants are faster to state that an emotional word is a word than a neutral word (Graves, Landis, & Goodglass, 1981; Strauss, 1983). Moreover, they show larger evoked related potentials (ERPs) over central and parietal cortical sites to emotional words (Begleiter, Gross, & Kissin, 1967). In contrast, but again in line with the hypothesis developed here, psychopathic individuals fail to show any reaction time or ERP differences between neutral and

emotional words (Day & Wong, 1996; Lorenz & Newman, 2002; Williamson, Harpur, & Hare, 1991).

The amygdala and instrumental learning

Instrumental learning involves the individual learning to perform an action to a stimulus if this action results in reward and to withhold from performing an action to a stimulus if this action results in punishment (the second form of learning is referred to as passive avoidance learning). The amygdala, and particularly the basolateral nucleus of the amygdala, are known to be implicated in instrumental learning, including passive avoidance learning (Ambrogi Lorenzini, Baldi, Bucherelli, Sacchetti, & Tassoni, 1999; Everitt, Cardinal, Hall, Parkinson, & Robbins, 2000; Killcross et al., 1997; LeDoux, 2000).

INSERT FIGURE 2 ABOUT HERE

Figure 2 develops the model depicted in Figure 1 to provide an account of instrumental learning. In this model, three additional modules of non-linear, identical computational units are depicted. The first of these corresponds to units coding motor responses and includes premotor cortex. The second corresponds to units coding expectation of reward (medial orbital frontal cortex). Within the model, the amygdala acts to modulate the Hebbian learning occurring as a function of mutual activation of representations of the sensory stimuli and premotor response output neurons. If the response has elicited reward, the Hebbian learning will be augmented. If the response has elicited punishment, the Hebbian learning will be suppressed.

As regards the second module, corresponding to medial orbital frontal cortex, here a claim is being made about a commonality of function of orbital frontal cortex with other regions of frontal cortex. There have been several recent suggestions that left dorsolateral prefrontal cortex, in particular, is involved in the selection of a verbal response option when more than one is in competition (Frith, 2000; Robinson, Blair, & Cipolotti, 1998). These have been elegantly modeled computationally (Usher & Cohen, 1999). Very briefly, the Usher and Cohen (1999) model assumes the existence of modality specific posterior units that are limited by temporal decay while anterior units use active reverberations which can sustain themselves and which are limited by displacement from competing new information. The anterior units, by being self excitatory, but mutually inhibitory allow rapid selection between competing, multiple active posterior response options (Usher & Cohen, 1999). The suggestion here is that units in orbital frontal cortex may serve a similar function over units in premotor cortex that mediate motor responses. The units in orbital frontal cortex would receive information in order to solve response competition on the basis of not only the activation of premotor units but also expectations of reinforcement provided by the amygdala. In addition, they would receive input from units from the third module, possibly involving anterior cingulate, representing desired goal states. The suggestion would be that reinforcer devaluation (Baxter, Parker, Lindner, Izquierdo, & Murray, 2000; Gallagher, McMahan, & Schoenbaum, 1999) would reduce potential activation of the corresponding units in orbital frontal cortex. This would reduce the probability that a response associated with these units would be chosen; the units involved would be less likely to win out in competition with other units that had not associated with reinforcer devaluation.

Amygdala lesions should impair instrumental learning, including passive avoidance learning and, at least in animals, it is known that they do (Ambrogi Lorenzini et al., 1999; Everitt et al., 2000; Killcross et al., 1997; LeDoux, 2000). In line with the suggestion that psychopathic individuals present with amygdala dysfunction, psychopathic individuals present with marked impairment on measures of instrumental (Fine et al., submitted) and, in particular, passive avoidance learning (Budhani, Johnston, & Blair, under revision; Newman & Kosson, 1986; Newman & Schmitt, 1998; Thornquist & Zuckerman, 1995).

Orbital frontal cortex, aversive conditioning and instrumental learning

There have been repeated suggestions that psychopathy is due to dysfunction within either frontal cortex more generally or orbital frontal cortex in particular (Damasio, 1994; Damasio, Tranel, & Damasio, 1990; Gorenstein & Newman, 1980; Raine, 1997, 2002). While the general frontal cortex position has been largely discredited (Kandel & Freed, 1989; Pennington & Ozonoff, 1996), there is reason to believe that there may be pathology in orbital frontal cortex in this population (LaPierre et al., 1995; Mitchell et al., 2002; Raine, Lencz, Bihrle, LaCasse, & Colletti, 2000). Could such pathology account for the impairments seen in psychopathic individuals in measures of aversive conditioning and instrumental learning?

There are data that neurons in orbital frontal cortex respond differentially to stimuli during aversive conditioning and instrumental and passive avoidance learning (Garcia, Vouimba, Baudry, & Thompson, 1999; Schoenbaum, Chiba, & Gallagher, 1999; Tremblay & Schultz, 1999). According to the model developed in Figure 2, we believe that these are crucial when the individual has to choose between two or more

behavioral responses. However, there is no reason to believe that these neurons are crucial for aversive conditioning and instrumental learning *per se*. If only one stimulus is present in the environment to respond to, orbital frontal cortex involvement should not be necessary. In line with this position, lesions of orbital frontal cortex do not impair aversive conditioning (Bechara, Damasio, Damasio, & Lee, 1999; Quirk, Russo, Barron, & Lebron, 2000) or instrumental learning/ passive avoidance (Schoenbaum, Nugent, Saddoris, & Setlow, submitted). This strongly suggests that orbital frontal cortex, unlike the amygdala, is not necessary for either function. Thus, while there may be orbital frontal cortex pathology in adult psychopathic individuals, this cannot be the explanation of the results discussed above.

Implications of amygdala dysfunction: Moral socialization

Socialization is the name given to the process by which caregivers, and others, reinforce behaviors that they wish to encourage and punish behaviors that they wish to discourage. Socialization involves aversive conditioning and instrumental learning. The unconditioned stimulus (US; the punisher) that best achieves socialization as regards instrumental antisocial behavior is not physical pain (Hoffman, 1994). Physical pain is rarely contiguous with the antisocial behavior and only occurs when the individual willing to use force is available. In addition, according to conditioning theory and data, the conditioned stimulus (CS) that is associated with the US is the CS that most consistently predicts the US (Dickinson, 1980). Indeed, in households using physical punishment, the CS predicting the US is rarely the antisocial behavior but rather the individual who delivers the US. Thus, in these households, aversive

conditioning may occur but the US-CS association will be physical pain & a particular parent, rather than physical pain & antisocial behavior (Hoffman, 1994).

A US that is present whenever antisocial behavior is committed, particularly in childhood, is the distress of the victim. The suggestion is that the sadness and fearfulness of the victim act as USs eliciting aversive conditioning and instrumental learning. Thus, in order to learn that hitting another individual is bad, a representation of this action must be associated with an aversive unconditioned stimulus (i.e., the distress of the victim). Similarly, learning to avoid committing moral transgressions involves either personally committing or viewing another commit a moral transgression and then being 'punished' by the aversive response of the victim's distress (Blair, 1995).

Sad and fearful expressions are thought to act as aversive unconditioned stimuli and an appropriate response to these stimuli is crucial for socialization (Blair, 1995). Functional imaging studies have shown, with a few exceptions (Kesler/West et al., 2001), that fearful and sad expressions all modulate amygdala activity (Baird et al., 1999; Blair et al., 1999; Breiter et al., 1996; Drevets, Lowry, Gautier, Perrett, & Kupfer, 2000; Morris et al., 1996; Phillips et al., 1998; Phillips et al., 1997; Schneider, Gur, Gur, & Muenz, 1994). In line with the amygdala dysfunction hypothesis, psychopathic individuals show pronounced impairment in processing sad and fearful expressions. They show reduced autonomic responses to these expressions (Aniskiewicz, 1979; Blair, Jones, Clark, & Smith, 1997) and, particularly in childhood, impaired ability to recognize these expressions (Blair, Colledge, Murray, & Mitchell, 2001).

One early index of appropriate moral socialization is the demonstration by the child of the moral/conventional distinction. From the age of 3.5 years, children

distinguish in their judgments between moral (victim-based) and conventional (social disorder-based) transgressions (Smetana, 1993; Turiel, Killen, & Helwig, 1987). Crucially, normally developing children best discriminate in their judgments between the two types of transgressions when they are asked to imagine situations where there are no rules prohibiting the transgressions. In contrast, adults with psychopathy and children with psychopathic tendencies are least likely to make a discrimination under these conditions (Blair, 1995, 1997; Blair, Jones, Clark, & Smith, 1995; Blair, Monson, & Frederickson, 2001). Moreover, similar difficulties have been observed with more general populations of children presenting with antisocial behavior (Arsenio & Fleiss, 1996; Dunn & Hughes, 2001; Hughes & Dunn, 2000; Nucci & Herman, 1982). In addition, psychopathic adults show reduced comprehension of situations likely to induce guilt although they show appropriate comprehension of happiness, sadness and even complex emotions such as embarrassment (Blair, Sellars et al., 1995).

There is also good direct evidence that the impairment shown by psychopathic individuals interferes with socialization. Thus, while it has been repeatedly shown that the use of empathy inducing positive parenting strategies by caregivers decreases the probability of antisocial behavior in healthy developing children, it does not decrease the probability of antisocial behavior in children who present with the emotional dysfunction of psychopathy (Wootton, Frick, Shelton, & Silverthorn, 1997).

Orbital frontal cortex and response reversal

Response reversal involves 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 reversal is the crucial component here; the individual must reverse their response to a stimulus. Response reversal is thus not involved in the passive avoidance task (Newman & Kosson, 1986) where the individual simply learns to respond to some stimuli and withhold responses to others but never to reverse their response to a stimulus. There is a considerable neuropsychological and neuro-imaging literature demonstrating that orbital frontal cortex is crucially involved in response reversal (Cools, Clark, Owen, & Robbins, 2002; Rahman, Sahakian, Hodges, Rogers, & Robbins, 1999; Rolls, Hornak, Wade, & McGrath, 1994).

INSERT FIGURE 3 ABOUT HERE

In figure 3, the model is developed further. In particular, it is suggested that there are comparator units in lateral orbital that would detect mismatches between expectations of reinforcement (provided by the amygdala units) and actual reinforcement (the nociceptive input). When activated these would disrupt the connections (weights) between amygdala units and orbital frontal cortex units as a function of the degree of the previous strength of these connection weights. Thus, under conditions where reinforcement had been a certainty and the connection weights were high, there would be considerable disruption. Under conditions where the reinforcement contingency was less obvious and the connection weights were lower, there would be less disruption. This disruption process would allow another

unit to develop the new expectation of reinforcement associated with the changed contingency to the stimulus and thus allow faster response reversal.

Within the model, the known role of orbital frontal cortex in response reversal (Cools et al., 2002; Dias, Robbins, & Roberts, 1996; Rolls et al., 1994) is seen as a function of the degree to which there is a mismatch between the expectation of reinforcement, provided by the amygdala to orbital frontal cortex, and the presence of reinforcement. This suggests that if there is dysfunction in either the amygdala or orbital frontal cortex or the connections between the amygdala and orbital frontal cortex, response reversal will be detrimentally affected. Moreover, the greater the degree of dysfunction, the more difficult it will be for the individual to identify the contingency change.

Children with psychopathic tendencies and adult psychopathic individuals show comparably impaired performance on measures of amygdala functioning such as passive avoidance (Newman & Kosson, 1986; Newman, Widom, & Nathan, 1985), the processing of fearful expressions (R. J. Blair, E. Colledge, L. Murray et al., 2001) and aversive conditioning (Lykken, 1957; Raine et al., 1996). However, there is less clear evidence that children with psychopathic tendencies show comparably impaired performance on measures requiring orbital frontal cortex such as response reversal. Newman's card playing task (Newman, Patterson, & Kosson, 1987) involves response reversal; the participant learns to play the card for reward but then must extinguish this response as, proceeding through the pack of cards, the probability of reward decreases successively. Both children with psychopathic tendencies and adult psychopathic individuals do show marked impairment on this task (Fisher & Blair, 1998; Newman et al., 1987; O'Brien & Frick, 1996). However, the ID-ED paradigm also includes response reversal; the participant must reverse their responding from the

object that, when responded to, had elicited rewarded but which now elicits punishment. While adult psychopathic individuals show notable impairment in response reversal on this task (Mitchell et al., 2002), children with psychopathic tendencies do not (Blair, Colledge, & Mitchell, 2001). 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 psychopathic individuals are impaired in the detection of contingency change, this impairment is markedly greater in the adult psychopathic individuals. 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. For each pair, one of the stimuli was rewarded more often than the other. The probability of reward was different across pairs (i.e., for pair 1, stimulus 1 was rewarded 100% of the time, for pair 2, stimulus 3 was rewarded 90% of the time etc). Following a set number of trials the contingency was reversed (i.e., for pair 1, stimulus 2 was rewarded 100% of the time, for pair 2, stimulus 4 was rewarded 90% of the time). While the children with psychopathic tendencies showed no difficulty reversing their responses for salient contingency changes, they did show significant difficulty as the salience of the contingency change decreased (Budhani et al., in preparation).

Orbital frontal cortex and response control

According to the model, orbital frontal cortex in crucially involved in resolving motor response conflict. If two or more motor responses are activated by stimuli, orbital frontal cortex resolves the conflict and allows one motor response to be initiated. According to the model, this is achieved by mappings of orbital frontal cortex units to premotor cortex units. The orbital frontal cortex units, by being self-excitatory, but mutually inhibitory allow rapid selection between competing, multiply active premotor response options (see also, Usher & Cohen, 1999). According to the model depicted in Figure 3, the initial activation of the orbital frontal cortex units can be a function of activation of expected reward as transmitted from the amygdala, premotor response units and the goal units. We have discussed above situations where decision-making is a function of expectations of reward/ punishment and violations of these expectations. However, orbital frontal cortex is also involved in resolving response competition in tasks where there are no clear expectations of reward and thus presumably little amygdala involvement. We refer to these tasks as response control tasks.

Two examples of response control tasks are the go/ no-go task and the Stop task. In the go/ no-go task, the participant is told to respond to one set of stimuli but not to respond to another set of stimuli; for example, the participant is told to press a button whenever any letter is on the screen other than an X. Imaging work has shown that if there are relatively few no-go stimuli relative to the number of go stimuli (i.e., there is a prepotent response to respond), lateral orbital frontal cortex is recruited and is involved in the resolution of the conflict between the prepotent respond response and a goal to withhold from responding on the basis of the task instructions (Casey et

al., 2001). In the Stop task, the participant is presented with a stimulus and instructed to respond to these stimuli as rapidly as possible unless a stop signal is presented in which case they are to stop their response (Logan, Cowan, & Davis, 1984).

Response control tasks are interesting because, although they involve orbital frontal cortex (Casey et al., 2001), unlike response reversal tasks they do not appear to require the amygdala. Response control tasks thus allow a direct test of whether there is dysfunction to regions external to the amygdala that could not be a result of the amygdala dysfunction. There have been relatively few investigations of the ability of individuals to perform response control tasks (Kiehl, Smith, Hare, & Liddle, 2000; LaPierre et al., 1995; Roussy & Toupin, 2000). However, two out of three studies using the go/no-go task, did report impairment in individuals with psychopathy (LaPierre et al., 1995; Roussy & Toupin, 2000). The third study did not but did find an atypical ERP response in the individuals with psychopathy to the no-go trials (Kiehl et al., 2000). The only study using the Stop task also reported that the psychopathic individuals were less successful than comparison individuals in withholding their response following the stop signal (Roussy & Toupin, 2000).

Orbital frontal cortex and the development of psychopathy

Earlier, I discussed data from aversive conditioning and instrumental learning tasks which strongly indicated amygdala dysfunction in individuals with psychopathy. Crucially, amygdala but not orbital frontal cortex lesions, result in impairment in both functions (Ambrogi Lorenzini et al., 1999; Bechara et al., 1999; Davis, 2000; Killcross et al., 1997; Quirk et al., 2000; Schoenbaum et al., submitted). However, the data from the response control tasks reviewed above would suggest that there are

indications of orbital frontal cortex pathology in individuals with psychopathy that are not a consequence of amygdala dysfunction. Moreover, the findings with the response reversal paradigms suggest that the orbital frontal cortex dysfunction may be greater in adults with psychopathy relative to children with the disorder. Thus, while both adults and children with psychopathy are insensitive, relative to comparison individuals, to subtle changes in reinforcement contingency (Fisher & Blair, 1998; Newman et al., 1987; O'Brien & Frick, 1996), only adults are insensitive to obvious changes in reinforcement contingency (Blair, Colledge, & Mitchell, 2001; Mitchell et al., 2002).

Given the evidence of amygdala dysfunction discussed above, there are several possibilities regarding the origins of the orbital frontal cortex pathology found in individuals with psychopathy. First, the orbital frontal cortex pathology could be developmentally independent of the amygdala pathology. For example, there might be genetic influences that affect the development of the amygdala and orbital frontal cortex independently of one another. Secondly, 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 present with impaired performance 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 lifestyle chosen by psychopathic individuals may be the cause of their orbital frontal cortex pathology.

Conclusions

In this chapter, I have developed a neuro-cognitive model of the development of psychopathy. At its heart is the suggestion of amygdala dysfunction in individuals with this disorder. This amygdala dysfunction gives rise to impairment in aversive conditioning, instrumental learning and the processing of fearful and sad expressions. These impairments interfere with socialization such that the individual does not learn to avoid actions that cause others harm. If such an individual has a reason to offend, because their other opportunities for the financial resources or respect are limited, they will be more likely to offend.

In addition, there are also indications that individuals with psychopathy present with orbital frontal cortex dysfunction. One aspect of this impairment, impairment on reversal learning tasks, may be related to the amygdala pathology. However, a second aspect of this impairment, impairment on response control tasks, cannot easily be related to amygdala pathology. This suggests that there is orbital frontal cortex pathology that is additional to the amygdala pathology. As yet, the degree to which the amygdala and orbital frontal cortex pathology have similar developmental origins remains unclear.

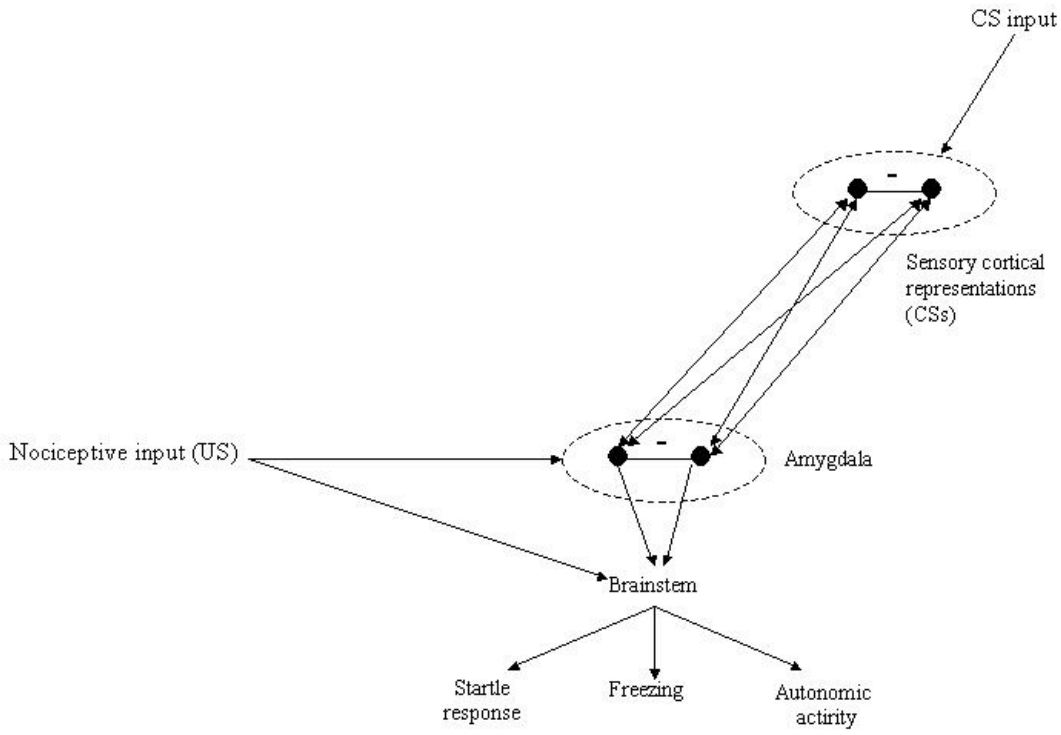

Figure 1: A simplified model of aversive conditioning. Sensory cortex (auditory, visual and temporal cortex) and the hippocampus allow the representation of conditioned stimuli. Contiguous activation of representations of conditioned stimuli in sensory cortex and amygdala activation by an unconditioned stimulus will increase the connections between the two representations through Hebbian learning allowing the CS to activate the brainstem even if the US is not present.

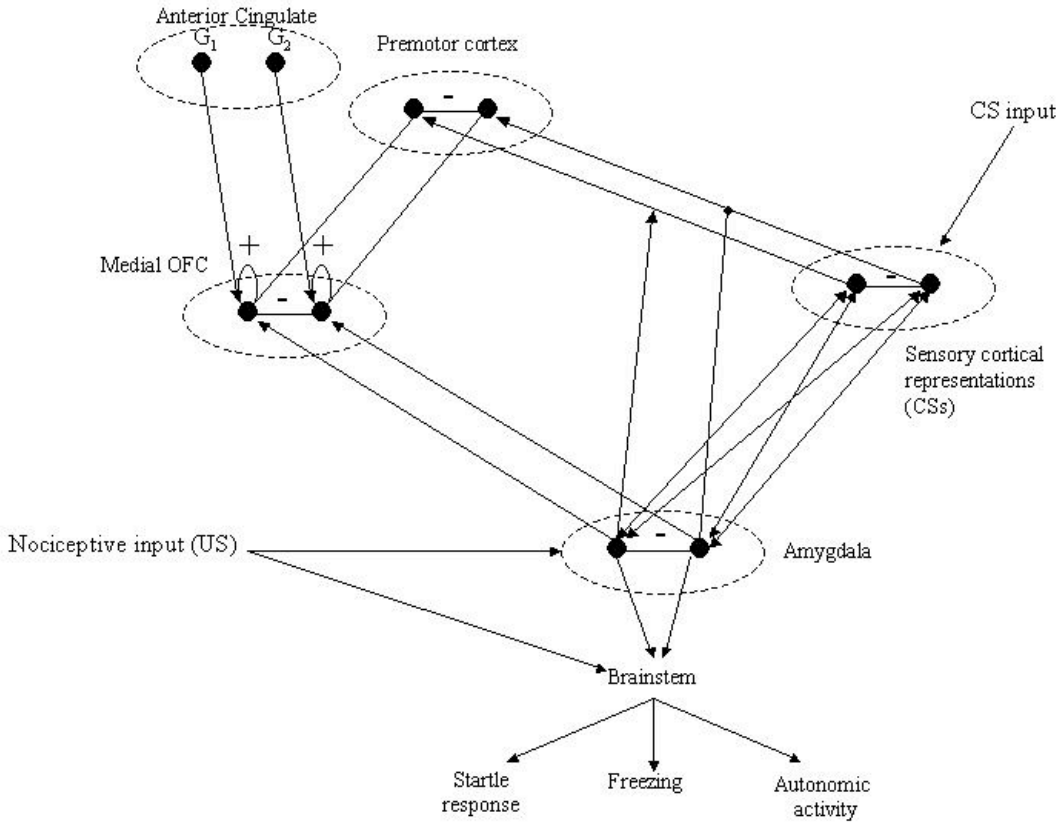

Figure 2: A development of the model to consider instrumental learning. The amygdala modulates the formation of associations between representations of the CS and motor responses mediated by premotor cortex. In addition, expectations of reinforcement/ punishment transmitted from the amygdala to orbital frontal cortex allow resolution if more than one motor response option has been activated. Goal representations also modulate this processing.

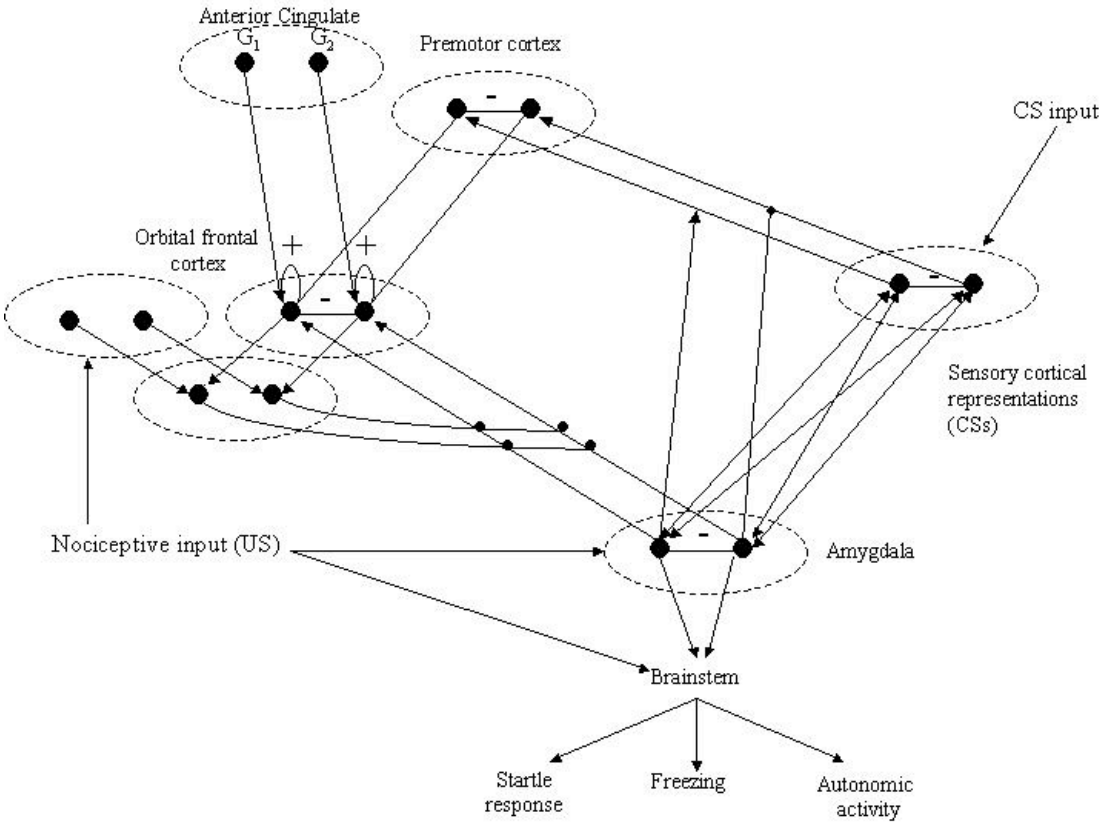

Figure 3: A development of the model to consider response reversal. It is suggested that there are comparator units in lateral orbital that would detect mismatches between expectations of reinforcement (provided by the amygdala units) and actual reinforcement (the nociceptive input). When activated these would disrupt the connections (weights) between amygdala units and orbital frontal cortex units as a function of the degree of the previous strength of these connection weights.

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