Blaming the Parts Instead of the Person: Understanding and Applying Neurobiological Factors Associated with Psychopathy[1]

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Dans l'article, on examine l'impact de nombreuses recherches indiquant que les psychopathes auraient des irrégularités neurobiologiques, qui se manifestent par des troubles d'apprentissage et du traitement de la peur, ainsi que des anomalies des neurotransmetteurs. Même si la présente recherche suggère que les psychopathes pourraient avoir de nombreuses irrégularités neurobiologiques, l'article porte plutôt sur les anomalies liées aux amygdales et au cortex orbitofrontal du cerveau, en plus de celles liées aux neurotransmetteurs. Ces irrégularités influeraient sur le comportement des psychopathes et contribueraient à expliquer leur tendance à adopter des comportements antisociaux. C'est pourquoi on soutient que ces facteurs devraient réduire le degré de responsabilité criminelle attribué aux actions des contrevenants psychopathes.

Mots clés : psychopathie, neurobiologie, responsabilité criminelle

This article examines the implications of the body of research that asserts that psychopaths have neurobiological irregularities that are manifested by learning and fear-processing deficits as well as neurotransmitter abnormalities. While this research suggests that psychopaths may have many neurobiological irregularities, the present article focuses on abnormalities related to the amygdala and orbitofrontal cortex of the brain, in addition to those related to neurotransmitters. It is argued that these irregularities influence the conduct of psychopaths and help to explain their propensity to engage in antisocial behaviour. Further, it is argued that these factors should mitigate the degree of criminal responsibility that is attributed to the actions of psychopathic offenders.

Keywords: psychopathy, neurobiology, criminal responsibility

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Introduction

Psychopathy is currently understood as a cluster of behaviours and personality traits that are typically viewed in a negative light (Hare 1993). Psychopaths are described as callous individuals who are aware of their wrongdoings but lack remorse. They are individuals who fail to accept responsibility for their actions, while priding themselves on having the skill to avoid sanctions (Cleckley 1982). They possess superficial charm and are able to convey the impression that they are agreeable individuals; however, they also have the ability to lie with remarkable conviction. They have average-to-above-average intelligence and are typically unreliable (Cleckley 1982).

Based on the premise that psychopaths do not have a distorted sense of reality and appear to be both rational and aware of their actions, the mental health community classifies psychopaths as sane (Hare 1993). Furthermore, the mental health community regards the conduct of psychopaths as being derived from a combination of cold rationality and an inability to view others as sensitive beings (Arrigo and Griffin 2004). As a result, psychopathy continues to be understood as a set of traits and behaviours that exist independently from any mental disorder.

However, if psychopathy is not associated with a mental disorder, the question arises as to what exactly does spur the development of these traits and behaviours? There tends to be a natural inclination to presume that psychopathy is related to upbringing and, more specifically, that individuals who experience difficult childhoods are more prone to psychopathy (Hare 1993; Petrunik and Weisman 2005; Marshall and Cooke 1999). It has been argued, however, that many people who experience troubled childhoods do not grow up to become psychopaths and, more importantly, there are a great number of psychopaths who were raised in loving and nurturing environments (Hare 1993). While it is true that the presence of antisocial parents, parental alcoholism, inconsistent discipline, and lack of supervision are related to psychopathy, it seems more apparent that these factors exacerbate the antisocial behavioural patterns of psychopaths rather than explain the behavioural deficits common to psychopaths (Blair, Mitchell, and Blair 2005). It seems clear, therefore, that psychopathy cannot be attributed solely to environmental factors and that there must be other factors that can more satisfactorily explain this set of traits and behaviours.

While environmental factors fail to provide a compelling explanation for the development of psychopathy, these factors are clearly connected to the development of antisocial personality disorder (APD). In a recent study of the relationship between APD and psychopathy, it was found that the two conditions share a common genetic factor (Larsson, Tuvblad, Rijsdijk, Andershed, Grann, and Lichtenstein 2007). Significantly, it was concluded that psychopaths are not sensitive to environmental stimuli in the development of their behavioural patterns, whereas a greater proportion of those who were subjected to environmental strains developed APD than those who were not exposed to such stressors (Larsson et al. 2007). Therefore, it appears that while environmental factors influence the development of APD, the same is not true for psychopathy. This suggests that APD may be characteristic of behavioural adaptations, whereas psychopathy is more likely to be innate to the individual. Thus APD and psychopathy are bound by a common genetic factor but they differ insofar as APD is susceptible to external triggers, while psychopathy is not.

Similarly, Blair (2006) has acknowledged the possibility that there is a genetic factor that predisposes individuals with psychopathy to develop antisocial behaviour. While he argues that it is unlikely that a genetic factor *causes* antisocial behaviour, genetics are recognized as a possible moderating factor in the expression of antisocial behaviour. More specifically, it is argued that when individuals with certain genetic predispositions are placed in stressful environments, genetics may play a role in determining the probability that those individuals will learn an antisocial, rather than socially acceptable, method of achieving their goals (Blair 2006).

The focus of this article is to argue that individuals with psychopathy are neurobiologically different from their non-psychopathic counterparts and that these differences affect their propensity to violate the law. The article provides an in-depth analysis of neurobiological research relating to selected cognitive and affective deficits common to psychopaths. It commences with an examination of the fear-processing deficits associated with psychopathy and continues with a discussion of learning deficits and neurotransmitter abnormalities. The implications of each of these various neurobiological factors are then analysed in the context of their relationship to criminal behaviour, and it is asserted that individuals with psychopathy should not be held fully responsible for their antisocial behaviour.

Neurobiological irregularities

Fear-processing deficits

One of the most striking characteristics common to psychopaths is their general failure to express fear. A great deal of research has been conducted to determine whether psychopaths are, in fact, devoid of fear and, if so, why this might be the case. While there is overwhelming agreement that psychopaths have fear-processing deficits (Birbaumer, Veit, Lotze, Erb, Hermann, Grodd, and Flor 2005; Lorenz and Newman 2002; Patrick, Bradley, and Lang 1993; Dolan and Fullam 2006; Dolan and Fullam 2006), the cause of these deficits is less clear.

Nearly 15 years ago, it was revealed that the startle reflexes of psychopaths differ from those of their non-psychopathic counterparts. One notable study indicated that, unlike ''normal'' individuals, when psychopaths are shown pleasant, unpleasant, and neutral pictures, their blinking patterns and heart rates remain constant (Patrick et al. 1993). This finding suggests that psychopaths are not emotionally affected by unpleasant images and do not exhibit signs of fear. More than a decade later, similar experiments were conducted, with a new focus on measuring the brain activity of psychopaths during the presentation of images that ranged through various degrees of unpleasantness and were accompanied by the application of painful pressure to the subject. The findings of these studies indicate that psychopaths have highly unusual brain activation patterns, as some parts of the emotion-related brain circuit were found to be significantly overactive, while other parts of the same circuit were severely underactive (Birbaumer et al. 2005; Müller, Sommer, Wagner, Lange, Taschler, Röder, Schuiererc, Kleina, and Hajaka 2003). Thus, it would appear that psychopaths do not exhibit normal physiological responses to fear.

Psychopaths also show abnormal physiological responses to frightening sentences. Upon listening to sentences designed to elicit fear, the muscular responses of psychopaths remain largely unchanged. It is common for people to unconsciously tense their muscles when they are afraid, yet psychopaths failed to show significant muscular responses to frightening sentences (Patrick, Cuthbert, and Lang 1994). This suggests that psychopaths have difficulty interpreting such sentences at an emotional level. This research further supports the finding

that psychopaths do not respond to fear in the same way as do most other individuals.

In addition to exhibiting diminished physiological responses to fear, there is evidence that psychopaths have difficulty identifying fear expressed by others. Research has indicated that psychopaths have difficulty recognizing fear and sadness in facial expressions, typically misinterpreting them as neutral (Dolan and Fullam 2006). This is particularly noteworthy in light of the fact that psychopaths have no difficulty identifying happy facial expressions (Dolan and Fullam 2006). Thus, while psychopaths are capable of experiencing happiness and recognizing it in others, they appear to have a fear-processing deficit that prevents them from experiencing fear themselves and from recognizing fear in the facial expressions of others.

Despite the fear-processing deficits common among psychopaths, there is evidence to suggest that they do have some conception of the emotion of fear. It might be expected that because psychopaths have difficulty experiencing fear or recognizing it in facial expressions, they would also have difficulty identifying frightening words; yet, this is not the case (Lorenz and Newman 2002). Research shows that psychopaths are able to differentiate between a word that may elicit fear and one that would not. This counter-intuitive ability introduces a central paradox of psychopathy: namely, psychopaths are able to identify emotional cues, but those cues are meaningless to the psychopath and cannot be used to guide their personal judgements or behaviours (Lorenz and Newman 2002).

It has been suggested that the atypical physiological responses of psychopaths to fear may be associated with dysfunction of the amygdala and/or orbitofrontal cortex (OFC) of the brain (Müller et al. 2003). The amygdala, which is located in the temporal lobe of the brain, is associated with impaired startle reflexes and poor recognition of facial expressions, particularly that of fear (Blair 2006). In contrast, the OFC, which is part of the frontal lobe, is associated with the anticipation of punishment and reward (Birbaumer et al. 2005). The anticipation of negative responses plays an integral role in fear, as fear is typically premised upon the realization that something negative may happen. This results in anxiety until the negative event occurs or until an intervention takes place to alleviate the anxiety (i.e., it becomes clear that the negative event will not occur). As a result, dysfunction in the OFC, which is associated with the anticipation of negative responses, could logically result in failure to experience fear. Moreover, OFC

dysfunction is associated with increased reactive aggression, which is common among psychopaths (Blair 2006) – further evidence to support the hypothesis that OFC dysfunction is characteristic of psychopathic individuals.

The fear deficits common to psychopaths have also been explained through the application of the integrated emotions systems (IES) model. The IES model utilizes research that implicates amygdala dysfunction in psychopathy, while providing an alternative explanation as to why this dysfunction results in fear deficits. The IES model suggests that an amygdala-based impairment hinders the ability of the individual to be successfully conditioned. While fearful and sad expressions are unconditioned responses to negative stimuli, the association between those expressions and the actions that result in them (e.g., violence) is a conditioned association. As a result, proponents of the IES model postulate that individuals with amygdala dysfunction fail to learn, and subsequently recognize, the link between fearful and sad experiences and expressions related to those emotions (Dolan and Fullam 2006).

Although the role of OFC and amygdala dysfunctions is debatable, there is relative agreement that the OFC and/or amygdala are related to the fear-processing deficits associated with psychopathy. Regardless of their precise causes, these deficits have serious implications. The first relates to the presumed relationship between fear and moral socialization, since there is some belief that moral socialization is achieved through the use of anticipated punishment, which most people fear, with the result that the prohibited behaviour is inhibited (Hare 1993). In fact, fear and anxiety have been referred to as the ''springboards of conscience'' (Hare 1993: 76) because, for most people, early childhood punishments produce life-long associations between wrong-doing and fear or anxiety that is associated with potential punishment (Hare 1993). However, because psychopaths are less aversively aroused by punishment (as a result of their lack of fear), they make only weak connections between misconduct and punishment and are, therefore, more likely to engage in punishable acts than are individuals without fear deficits (Blair 2006).

Another important characteristic associated with the lack of fear experienced by psychopaths relates to empathy. Given that psychopaths present with fear-processing deficits, it is not surprising that they do not experience empathy; after all, it is impossible for someone

to experience the emotions of others vicariously when he or she does not acknowledge those emotions or finds them foreign. This finding is of great importance because there is a counter-argument to the proposition that punishment is necessary for moral socialization which claims that moral socialization is achieved, instead, through empathy. The approach is based on research showing that children tend to learn more effectively how to avoid punishable offences when they understand the effects that their actions have on others than when they are simply punished (Hoffman 2000). However, for this method of moral education to be effective, it is necessary that the learner be capable of empathy, a necessity that clearly presents a problem for the moral socialization of psychopaths.

However, another theory suggests that the emotional deficits of psychopaths can be explained by a malfunctioning of the right hemisphere. Using functional MRI, it was determined that psychopaths have difficulty activating various regions of the right hemisphere while processing abstract words, performing abstract categorization tasks, or interpreting abstract ideas as conveyed through metaphors and that, as a result of this difficulty, they are prevented from completing the tasks (Kiehl, Smith, Mendrek, Forster, Hare, and Liddle 2004). In light of this finding, it is suggested that some of the emotional deficits associated with psychopathy may be related to a difficulty with processing abstract notions, including emotions such as love (Kiehl et al. 2004). While empathy is not an emotion but rather a state of mind, it is premised upon abstract concepts that are linked to an awareness of the emotions of others and thus requires the processing of abstract ideas. For this reason, it is possible that the absence of empathy among individuals with psychopathy may be a result of the right hemisphere malfunctioning.

Regardless of the model adopted to explain the lack of empathy manifested by psychopaths, the failure to experience empathy has grave implications. A study conducted by Blair, Jones, Clark, and Smith (1995) indicates that psychopaths present with an impaired ability to differentiate between moral and conventional rules, and it is possible that this impairment can be linked to the lack of empathy. Before discussing this research, it is useful to note that, at a very basic level, there are two types of rules: *moral rules*, which are defined as governing consequences for the rights and welfare of others, and *conventional rules*, which are defined as the behavioural principles that govern social interactions within social systems (Blair et al. 1995).

To test the capacity of psychopaths to differentiate between moral and conventional rules, Blair et al. (1995) conducted an experiment involving the use of stories describing various transgressions. At the conclusion of each story, participants were asked if the events described were wrong, and if so, why they were wrong (Blair et al. 1995). In general, participants with psychopathy scored the moral transgressions as being more serious than the conventional transgressions. However, this was perceived to be linked to the fact that many of those participants had been incarcerated for committing similar transgressions and thus it was possible that their perception of seriousness was related to the likelihood of imprisonment and not to morality (Blair et al. 1995). Furthermore, unlike ''normal'' participants, those with psychopathy were not able to distinguish which of the transgressions described was moral and which conventional (Blair et al. 1995). Finally, participants with psychopathy were much less likely than their non-psychopathic counterparts to reference the welfare of others in explaining why the transgression was wrong (Blair et al. 1995). The experiment suggested that psychopaths are aware of societal rules but that they do not fully understand the nature of moral transgressions (Blair et al. 1995).

It is not entirely clear why psychopaths are unable to distinguish between moral and conventional transgressions as this is an ability that most children have acquired by the age of four (Blair et al. 1995). It is possible that this inability is linked to emotional affects associated with psychopathy. The failure of psychopathic participants to cite the welfare of others when discussing transgressions may be linked to their incapacity to feel empathy, which can in turn be linked to the fear-processing deficits that may be rooted in the abnormal functioning of the brains of individuals with psychopathy. The inability of individuals with psychopathy to recognize moral standards can also be linked directly to fear-processing deficits, as there is some belief that moral socialization is achieved through the use of anticipated punishment, which is an outcome that arouses fear in most people (Hare 1993).

It may not be clear why individuals with psychopathy are unable to distinguish between moral and conventional transgressions, but one thing is clear: They simply cannot make the distinction. This means that psychopaths are not able to judge their actions by the moral standards of ordinary individuals because they are unaware of those standards. Hare (1998) explained that the consciences of psychopaths are only partially formed, consisting only of an intellectual awareness

of the rules of society and that, as a result, ''their rule books are pale, abridged versions of those that direct the conduct of other individuals'' (205).

Learning deficits

Prior to discussing the learning deficits of psychopaths, it is useful to provide some basic definitions to facilitate greater understanding of the concepts that will be discussed. It is necessary to provide definitions of *stimulus-reinforcement* and *stimulus-response* associations, as these are the bases of a great deal of research relating to the learning patterns of psychopaths. Both stimulus-reinforcement and stimulus-response associations are learned when an individual's behaviour is consistently reinforced with the use of punishment or reward. However, stimulus-reinforcement associations are dependent upon changes in reinforcement and thus, as the nature of the reinforcement changes, so too does the behaviour of the individual (Baxter and Murray 2002). In contrast, stimulus-response associations are insensitive to changes in reinforcement and, therefore, regardless of how the reinforcement is altered, the behaviour of the individual will remain consistent (Baxter and Murray 2002). The two differ further in that the neural processing underlying stimulus-reinforcement involves the amygdala, whereas this neuroanatomical structure is not necessary for the successful acquisition of stimulus-response associations (Baxter and Murray 2002).

Stimulus-reinforcement is an umbrella term that encompasses several forms of learning, including passive avoidance. Passive-avoidance learning is a form of stimulus-reinforcement whereby a punishment is applied each time a particular behaviour is performed. The goal of the punishment is to inhibit the behaviour by creating an association in the individual's mind between the punishment and that particular behaviour. As the association that is formed through this method is ultimately one of stimulus-reinforcement, the amygdala is implicated (Blair, Mitchell, Leonard, Budhani, Peschardt, and Newman 2004). Given the large body of research that has suggested a link between amygdala dysfunction and psychopathy, it is not surprising to learn that psychopaths present with deficiencies related to stimulus-reinforcement learning. This can be explained by the deficiencies in relation to learning through passive-avoidance tasks that psychopaths present with. Research has shown that as the level of punishment for a particular behaviour increases, most people modulate their behaviour to

avoid punishment; however, this is not the case for individuals with psychopathy (Blair et al. 2004).

In contrast to their failure to modulate behaviour when faced with a passive-avoidance learning task, individuals with psychopathy present with no difficulty altering their behaviour when faced with stimulus-response learning tasks (Blair et al. 2004). There is an important distinction to be made here with reference to the learning deficits associated with psychopathy. Although individuals with psychopathy do not have the cognitive ability to modulate their behaviour as the nature of the reinforcement is changed, when faced with a stimulus-reinforcement task, they are able to learn the initial rule that is taught through the basic stimulus-reinforcement association. This can be explained by referencing back to the abilities of individuals with amygdala dysfunction, who are capable of learning through stimulus-response associations, but are not able to modulate their behaviour accordingly as the nature of the reinforcement changes (Baxter and Murray 2002). As a result, individuals with amygdala dysfunction are able to grasp initial stimulus-response associations but are unable to change their behaviour as reinforcements are modified – a step that is necessary for properly developing stimulus-reinforcement associations.

Both psychopaths and individuals with amygdala dysfunction perform similarly when faced with learning tasks, suggesting that the learning deficits of psychopaths can be explained by amygdala dysfunction. To test this assertion, Mitchell, Fine, Richell, Newman, Lumsden, Blair, and Blair (2006) performed an experiment in which individuals with known amygdala dysfunction were asked to complete a series of learning tasks that were also completed by individuals with psychopathy. When faced with tasks that required the individuals to learn behaviours through the use of stimulus-reinforcement association, both groups performed poorly, presenting evidence of similar difficulties in the development of these associations (Mitchell et al. 2006). These findings have been interpreted as confirming that insofar as learning capabilities are concerned, there are parallels between individuals with amygdala dysfunction and those with psychopathy.

There is another important question that arises with respect to the link between psychopathy and amygdala dysfunction: If it is true that amygdala dysfunction can explain several of the behaviours associated with psychopathy, why, then, are there some individuals who

suffer from amygdala dysfunction but are not psychopaths? This is a logical question, but the answer does not affect the strength of the hypothesis that amygdala dysfunction may help to explain the behaviours of psychopaths, for, while it is true that the amygdala is required for several forms of learning, an injury sustained in the amygdala will not wipe out information that had been acquired prior to that injury (Blair 2006). As a result, individuals who sustain injuries to the amygdala later in life typically have already formed the associations necessary to inhibit antisocial behaviour. Thus, amygdala dysfunction can help to explain the behaviours of individuals with psychopathy; however, amygdala dysfunction is not synonymous with psychopathy.

Despite the existence of research indicating a strong link between psychopathy and amygdala dysfunction, there is reason to believe that the learning deficits associated with psychopathy are not caused solely by this dysfunction. In an attempt to better understand the behaviours of psychopaths, a study was carried out that incorporated two experiments. The first experiment tested the ability of psychopaths to complete a learning task that is known to implicate the OFC of the brain, while the second tested their ability to complete a learning task that is thought to implicate either the amygdala or OFC (Mitchell, Colledge, Leonard, and Blair 2002). The objective of this experiment was to determine whether OFC dysfunction might be a factor associated with psychopathy. The central hypothesis was that if the psychopathic participants were unable to complete a task known to involve the OFC, then a dysfunction of this structure might help to explain some of the deficits associated with psychopathy that were previously thought to be associated with the amygdala – despite our knowledge that some deficits could implicate either of these neuroanatomical structures (Mitchell et al. 2002). Subjects with psychopathy did have difficulty completing the task involving the OFC and, therefore, there is reason to believe that some of the learning deficits associated with psychopathy may be linked to dysfunction of the OFC (Mitchell et al. 2002).

The second experiment is of particular interest, as it tested the ability of psychopaths to learn stimulus-response associations and subsequently apply the information learned from the associations in order avoid making risky decisions (Mitchell et al. 2002). The experiment required individuals with and without psychopathy to select cards from one of four decks, each of which was associated with a set of potential consequences and rewards. The experiment was designed

so that it would become clear which decks were most advantageous through the process of trial and error, thus facilitating stimulus-response associations (Mitchell et al. 2002). Though psychopaths are able to acquire information from stimulus-response associations, this experiment illustrated that they are less likely than those without psychopathy to learn and apply the associations necessary to avoid making risky decisions (Mitchell et al. 2002). Thus, despite their awareness that some of the decks were advantageous, psychopaths would continue to select cards from the less advantageous decks.

Contrary to the typical assumption that their stimulus-reinforcement deficit is caused by a dysfunction of the amygdala, Mitchell et al. (2002) interpreted the propensity of psychopathic participants to make high-risk decisions as indicating a dysfunction either of the amygdala or the OFC or of the connections between the amygdala and the OFC (Mitchell et al. 2002). This interpretation was based on the first finding of the study, which indicated that the OFC may have a role in explaining some of the deficits associated with psychopathy (Mitchell et al. 2002). This suggests that the roots of the learning deficits common to psychopaths may be more complex than other interpretations have suggested. The amygdala and OFC are part of a neural circuit that is believed to play a crucial role in the interpretation and neurological imprinting of information regarding learned associations and their motivational value (Mitchell et al. 2002). As a result, difficulty with stimulus-reinforcement associations could indicate a dysfunction within any part of this neural circuit. Furthermore, it is possible that both the amygdala and the OFC dysfunctions are caused by a third abnormality, which may be the true source of the learning deficits common to psychopaths (Mitchell et al. 2002).

There is still another theory that offers an explanation as to why psychopaths present with learning deficits. The response-modulation hypothesis (RMH) has been used to explain these deficits in terms of deficient emotional processes (Lorenz and Newman 2002). Response modulation is a brief, automatic shift of attention from one focal point to another. This shift of attention allows individuals to monitor, and, if relevant, use information that is outside their deliberate focus of attention (Lorenz and Newman 2002). According to the RMH, in addition to the learning and emotion-processing deficits of psychopaths, many of their behavioural problems may be related to a failure to meaningfully process peripheral information (Lorenz and Newman 2002). This implies that many deficiencies associated with psychopathy may actually be caused by an inability to shift readily

from one focus of attention to another. This can easily be applied to stimulus-reinforcement associations; for example, according to the RMH, it may be possible for psychopaths to learn to avoid prohibited behaviour through passive avoidance if the *goal* of the psychopath is to avoid punishment while completing the task. However, if the psychopath is focusing on an alternative goal during completion of the task, the negative association between the punishment and behaviour will remain a peripheral focus and the inability to shift attention will prevent the formation of a meaningful association (Lorenz and Newman 2002).

It is noteworthy that psychopaths have difficulty using learned associations to guide their behaviour. One of the experiments discussed previously was designed to measure the likelihood that psychopaths would use learned associations in order to avoid risky behaviour (Mitchell et al. 2002). This experiment, which was based on performance of the gambling task, can be seen as being somewhat analogous to dealing with the legal system. The decks used in the experiment can be understood as representing the two paths available to each individual: the path of law-abiding behaviour and that of misconduct. Individuals who select the path of legal obedience are rewarded with continued liberty; those who select the path of misconduct are subject to legal sanctions. However, psychopaths have difficulty internalizing these associations in a meaningful way and thus engage in risky behaviours that are sometimes punishable. This inability to learn to avoid risky decisions presents a conundrum in relation to psychopaths and the criminal justice system.

In sum, just as it is unclear exactly what causes the fear-processing deficits of psychopaths, the precise cause of the learning deficits associated with psychopathy is also uncertain. Nevertheless, the implications of these learning deficits, like those of the fear-processing deficits, are serious. Findings as to learning deficits further support the view that psychopaths are neurobiologically different from their non-psychopathic counterparts. One thing that is clear from this discussion is that the brains of psychopaths work differently and are comparable to those of individuals with brain damage that results in amygdala and/or OFC dysfunction.

Neurotransmitter abnormalities

Prior to discussing the role of neurotransmitters in prompting the behaviours of individuals with psychopathy, it is beneficial to provide

some background information regarding the concept of neurotransmission. Neurotransmitters are chemicals that transmit impulses across synapses, which form a junction between nerve cells. They are secreted from the presynaptic site and act at the postsynaptic receptor (Lawrence 2005). Thus neurotransmitters make communication possible from one nerve cell to another. The message that is sent is dependent upon the type of neurotransmitter released into the synapse and the type of receptor at the postsynaptic site (i.e., the part of the synapse that the neurotransmitter acts upon). Neurotransmitters can send messages related to processes such as urges, emotions, and physical sensations (Lawrence 2005).

The research relating to neurotransmitters and psychopaths is still in its early stages. However, there is a body of research suggesting that psychopaths have reduced serotonin (5-hydroxytryptamine) activity (Dolan 1994). More recent studies, however, suggest that the behavioural problems characteristic of individuals with psychopathy are linked to both serotonin and dopamine levels (Soderstrom, Blennow, Manhem, and Forsman 2001; Soderstrom, Blennow, Sjodin, and Forsman 2003). It has been suggested that the aggressive behaviours associated with psychopathy are related to the ratio of cerebrospinal fluid homovanillic acid (HVA), a metabolite of the neurotransmitter dopamine, and 5-hydroxyindolacetic acid (5-HIAA), a metabolite of the neurotransmitter serotonin (Soderstrom et al. 2003). More specifically, psychopaths tend to have a higher ratio of HVA to 5-HIAA (Soderstrom et al. 2003). These findings may explain the outwardly directed aggressive behaviours that are often exhibited by psychopaths, in addition to the disorganized behaviours such as the need for stimulation, poor behavioural controls, impulsivity, and irresponsibility that are so common among individuals with psychopathy (Soderstrom et al. 2003).

Soderstrom et al. (2003) raise the possibility that the aggression associated with psychopathy may be related to high dopamine turnover in combination with relative serotonergic dysregulation. They suggest that the high dopamine turnover may be an adaptation to postsynaptic dysfunction or may be due to deficient serotonergic tonic regulation of the dopamine system, and that pharmacological interventions targeting the dopamine and serotonin systems may be beneficial in terms of reducing the aggression associated with psychopathy. Neurotransmitter abnormalities are of great significance because addressing them may form the basis for developing promising medical interventions to manage some of the traits associated with

psychopathy. However, more research will be required before this knowledge can be fully applied.

Aggression in psychopaths

Individuals with psychopathy have a heightened propensity, as compared to their non-psychopathic counterparts, to participate in both reactive and instrumentally aggressive behaviours. While these aggressive outbursts cannot be linked specifically to the deficits or abnormalities discussed previously, they are, nonetheless, related to neurobiological functioning and are certainly worthy of mention.

Reactive aggression

Reactive aggression, which is also referred to as *affective* or *impulsive aggression*, relates to aggressive behaviour that is, by its nature, responsive. Reactive aggression is most commonly triggered by a threatening or frustrating event that induces anger on the part of the aggressor (Blair 2007). This aggression is initiated without regard for a particular goal but is instead fuelled by hostility and rage (Blair 2007). Reactive aggression is typically targeted at victims who are familiar to the aggressor and who are perceived by the aggressor as having provoked the violence (Cornell, Warren, Hawk, Stafford, Oram, and Pine 1996).

Individuals who abstain from regular reactive outbursts of an aggressive nature typically have the capacity to select alternative responses to aggression when angered and can control if, and how, they will display their aggression (Blair 2007). For example, those who are not prone to reactive outbursts are less likely to show aggression towards a colleague of higher status than towards a colleague of lower status because displaying aggression towards the former carries greater potential consequences in the workplace (Blair 2007). However, those who are prone to reactive outbursts are typically indifferent to conventional rules and fail to modulate their behaviours according to the status of the individuals with whom they are interacting (Blair 2007).

Individuals with psychopathy are generally at pronounced risk of engaging in reactive aggression (Blair 2007). This is evidenced by their short tempers and tendency to respond to frustration, failure, discipline, and criticism with sudden outbursts of violence, threats,

and verbal abuse (Hare 1993). These outbursts are typically short-lived and, once they are over, psychopaths usually return to their usual behaviour, acting as though nothing out of the ordinary has taken place (Hare 1993).

The brain activity involved in aggressive outbursts of a reactive nature is not fully understood, but several regions of the frontal cortex have been implicated in the regulation of the basic threat system that mediates threat responses. In particular, the OFC, which is believed to be impaired in psychopaths, has been linked to this system (Blair 2007). This information is valuable because reactive aggression is linked to perceptions of threat and it is believed that modulation of the basic threat system is more difficult in the presence of dysfunctional frontal regulatory systems (Blair 2007). It has been hypothesized that when a threatening or frustrating stimulus is highly intense, the frontal regulatory systems may be unable to reduce the activity of the basic threat system and so reactive aggression is displayed (Blair 2007). A comparable overload of the basic threat system that resulted in aggressive behaviour on the part of an individual with a fully functional frontal cortex would be recognized as a ''crime of passion'' (Blair 2007).

Since individuals with psychopathy have a reduced sensitivity to threat, which may be related to fear-possessing deficits, it is unlikely that the increased propensity for reactive outbursts is related to perceived threats (Blair 2007). It is more probable that the primary trigger for reactive aggression in psychopaths is frustration, which can be affected by an overload of the basic threat system (Blair 2007). In short, this means that reactive aggression can be virtually automatic for individuals with psychopathy when they are faced with intense frustration.

Instrumental aggression

In contrast to reactive aggression, instrumental, or proactive, aggression is purposeful and goal-oriented. It is aggression that is carried out as a means to a desired end, such as acquiring a victim's belongings (Blair 2007). As is the case for reactive aggression, individuals with psychopathy are more likely to engage in instrumentally aggressive behaviour. Research indicates that incarcerated instrumental offenders have higher PCL-R scores than reactive offenders, suggesting that higher levels of psychopathy are consistent with instrumental

offending (Cornell et al. 1996). Interestingly, the authors of this study hypothesize that instrumental aggression is characteristic of psychopathy and should be included in the list of behaviours and traits that are used to describe the disorder (Cornell et al. 1996).

Another study indicates that psychopaths are more likely to commit homicide for instrumental purposes than their non-psychopathic counterparts (Woodworth and Porter 2002). This may be related to emotional affects associated with psychopathy that allow psychopaths to carry out ruthless, cold-blooded, premeditated acts. For example, one offender with a high PCL-R score reported that he murdered his ex-girlfriend because he thought that she was interfering with his current relationship and he believed that murdering her would resolve this issue (Woodworth et al. 2002). It is likely that the inability to feel empathy played a significant role in this crime. Further, it is possible that the extreme lack of concern and respect for others, combined with the impulsivity that is characteristic of psychopathy, may well override any inhibitions psychopaths have and drive them to act aggressively in order to achieve instrumental goals (Cornell et al. 1996).

It is highly probable that instrumental aggression expressed by individuals with psychopathy is related to their neurobiological irregularities. While instrumental aggression is mediated by poor socialization (Cornell et al. 1996), psychopaths cannot readily be socialized (Blair 2007). The process of moral socialization requires individuals to find the distress of others aversive, as expressions of fear and sadness in others serve as important social reinforcements (Blair 2007). Moreover, moral socialization requires the ability to perform stimulus-reinforcement associations to learn that various actions are good or bad (Blair 2007). Yet owing to neurobiological irregularities, individuals with psychopathy do not have either of these capabilities and are, therefore, less able to absorb and apply lessons pertaining to moral socialization.

While reactive aggression can be automatic in psychopaths, individuals with psychopathy *choose* to engage in acts of instrumental aggression. However, the deliberateness of this choice is mitigated by the presence of a mind that features dysfunction in the neural regions necessary for successful moral socialization. When considered in these terms, instrumental aggression, to a psychopath, reflects a logical step towards achieving goals.

Conclusion

Accused persons in Canada who suffer from ''mental disorders'' may raise the special defence of not being criminally responsible on account of mental disorder (Verdun-Jones 2007). For this defence to be successfully asserted, two conditions must be satisfied: first, the accused person must have been suffering from a ''mental disorder'' (defined in section 2 of the Criminal Code as a ''disease of the mind''); second, the accused must have been incapable either of appreciating the nature and consequences of the act or omission in question or of understanding that it was legally or morally wrong (*Criminal Code* 1985, s. 16).

The Supreme Court of Canada has consistently emphasized that the question as to whether a mental condition, such as psychopathy, should be considered a ''disease of the mind'' must ultimately be decided by the trial judge. Medical evidence is undoubtedly highly relevant to the judicial decision-making process in relation to this issue. However, in *R. v. Stone*, the Supreme Court endorsed the view that the ''opinions of medical witnesses as to whether an abnormal mental state does or does not constitute a disease of the mind are not, however, determinative, since what is a disease of the mind is a legal question'' (*R. v. Stone* at para. 195). The Supreme Court of Canada has adopted a broad definition of ''disease of the mind.'' The classic definition was articulated by Dickson J. in *R. v. Cooper*: ''[I]n a legal sense 'disease of the mind' embraces any illness, disorder or abnormal condition which impairs the human mind and its functioning, excluding however, self-induced states caused by alcohol or drugs, as well as transitory mental states such as hysteria or concussion'' (*R. v. Cooper* at para. 144). In light of this expansive definition, the Supreme Court of Canada indicated, in *R. v. Kjeldsen*, that it would be prepared to accept that psychopathy meets the legal criteria for recognition as a ''disease of the mind.'' This decision was reached even though psychopathy is not included as a mental disorder in the *DSM-IV-TR* (American Psychiatric Association 2000). However, as McIntyre J. said, in *Kjeldsen*, the next – and more critical – issue that must be decided is whether psychopathy has one of the necessary attributes specified in section 16: ''either incapacity of appreciating the nature and quality of the act, or of knowing that the act was wrong'' (*R. v. Kjeldsen* at para 166).

In *Kjeldsen*, the Court focused on the first of these two incapacities and ruled that Kjeldsen's psychopathy did not prevent him from

appreciating the physical nature and quality of his actions. McIntyre J. stated section 16 does not exempt from criminal responsibility an individual ''who has the necessary understanding of the nature, character and consequences of the act, but merely lacks appropriate feelings for the victim or lacks feelings of remorse or guilt for what he has done, even though such lack of feeling stems from disease of the mind'' (*R. v. Kjeldsen* at para. 168). Significantly, he added that there is no ''doubt the absence of such feelings is a common characteristic of many persons who engage in repeated and serious criminal conduct.'' The Court paid little attention to the second incapacity in section 16 because it was satisfied that a psychopath, such as Kjeldsen, had the capacity to know that his actions were *legally* wrong. At the time *Kjeldsen* was decided, the word ''wrong'' in section 16 was interpreted in the narrow sense of *legally*, rather than *morally* wrong. However, in the later case of *R. v. Chaulk*, the Supreme Court of Canada changed its position and held that ''wrong'' should be interpreted as meaning *morally* wrong. This is particularly important in light of the research suggesting that psychopaths lack the capacity to know that their actions are morally wrong.

Current case law suggests that Canadian courts are unlikely to find that a psychopath meets the criterion of incapacity to know that his or her actions are morally wrong. Indeed, the assumption appears to be that when this incapacity exists, it is generally a consequence of a psychotic delusion (*R. v. Chaulk*; *R. v. Landry*; *R. v. Oommen*; *R. v. Molodowic*). For example, in *R. v. Oommen*, the Supreme Court of Canada stated that the ''crux of the inquiry is whether the accused lacks the capacity to rationally decide whether the act is right or wrong and hence to make a rational choice about whether to do it or not'' (at para. 26). In this case, the psychiatric evidence was to the effect that a delusion had made ''the accused perceive an act which is wrong as right or justifiable'' and that he was deprived of ''the ability to rationally evaluate what he is doing'' (*R. v. Oommen* at para. 17). The Court took the opportunity to distinguish the situation in which a psychotic delusion causes an incapacity to know that one's actions are ''wrong by the standards of the ordinary person'' from the situation in which a psychopathic condition causes an accused person to adhere to a ''personal and deviant moral code'': In McLachlin J.'s view, the psychopath is ''capable of knowing that his or her acts are wrong in the eyes of society, and despite such knowledge, chooses to commit them'' (*R. v. Oommen* at para. 19). This assumption is now rendered questionable in light of the research evidence surveyed in this article, but it

remains to be seen whether the judicial approach will change course at any time in the foreseeable future.

Recent developments in the neurosciences are, perhaps, more likely to have an impact on the sentencing of psychopaths. Section 718.1 of the Criminal Code states that a ''sentence must be proportionate to the gravity of the offence and the degree of responsibility of the offender.'' If neurological abnormalities significantly contribute to the criminal behaviour of a convicted psychopath, his or her responsibility is diminished and that should be taken into consideration in the sentencing process. Indeed, section 718.2 provides that a sentence should be reduced in order to account for any relevant ''mitigating circumstances relating to ... the offender.'' However, at present, psychopathic offenders are typically subjected to longer, harsher sentences, since the courts routinely treat psychopathy as an *aggravating* rather than a *mitigating* factor (Hare 1998; Zinger and Forth 1998).

The intent of this article has been to focus on research that has identified the neurobiological abnormalities associated with psychopathy. A detailed examination of the implications of this analysis for the criminal law and criminal justice system must, of necessity, be undertaken elsewhere. However, it is appropriate to suggest that one of the more significant implications of the analysis is that the severe punishment of psychopathic offenders is unjustified on both practical grounds (it is ineffective) and moral grounds (owing to neurobiological deficits that are beyond their control, psychopaths lack the capacity to understand that their actions are immoral). Therefore, if restrictions on the liberty of such offenders are deemed to be necessary for community safety, such restrictions should be imposed on the basis that the individuals concerned engender an unacceptable level of risk of harm to others rather than on the basis of an escalating punitive response. Individuals subjected to preventive detention should be treated in a humane manner and subjected to the least restrictive controls commensurate with ensuring public safety. Where appropriate and likely to be effective, treatment should be provided as part of a *quid pro quo* for the loss of liberty that is imposed in the interests of society as a whole (Loving 2002; Reid and Gacono 2000; Vien and Beech 2006). What is clear is that imposing restrictions on psychopathic offenders for primarily punitive or retributive purposes is not acceptable in light of the rapidly emerging body of knowledge within the neurosciences.

Note

1. Part of this article was drawn from the first author's master's thesis, "A Cause for Compassion: Understanding and Applying Neurobiological Factors Associated with Psychopathy," Simon Fraser University, 2008.

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