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The Neurobiological Basis for Psychopathy: Why Current Treatment is Inadequate

Ian G. R. Jones*

Research surrounding psychopathy has progressed greatly in the last two decades firmly establishing psychopathy as a psychological condition. However, clinical practice has not altered to keep pace with current research. This paper reviews evidence from imaging, behavioural, and clinical studies of psychopathy focusing on its etiology and treatment. A neurobiological basis for psychopathy is established from this evidence, and the Somatic Marker Hypothesis, Violence Inhibition Mechanism Model, and Integrated Emotional System Model are considered. This neurobiological basis is then used as a foundation for a discussion on how clinical practice should proceed. Clinical strategies such as cognitive behavioural therapy and behavioural therapy are reviewed, and current treatments are found to be lacking in effectiveness. Finally new clinical directions such as drug treatments, targeted neuropsychological interventions based on an individual's specific neurological deficits, and targeting youth populations are suggested in order to improve the clinical prognosis and reduce incarceration levels for psychopathic individuals.

Psychopathy is often defined based on Robert Hare's Psychopathy Checklist (PCL and subsequent revised version PCL-R; 1991). Hare's (1991) operationalization of psychopathy describes callous, unemotional, and affectively detached individuals who frequently engage in antisocial behaviours. This lack of emotional attachment is associated with atypical interactions with others and failure to recognize the emotional impact of the individual's actions on others. (Hare, 1991).

The PCL-R measure allowed an explosion of research surrounding the concept of psychopathy, and is still widely used in research, the criminal justice system, and clinical practice. However, the etiology of psychopathy is still unclear. Recent advances in neuroimaging and cognitive research methods have allowed insight into the biological and cognitive basis of psychopathy. As a result, traditional treatments, including various types of behaviour therapy, have generally been ineffective. The aim of this paper is to describe the current theories of psychopathy and how they relate to a clinical conception of this

dimension. The current literature on the biological basis for psychopathy will also be discussed. A review of the evidence from neuroimaging will then be used to demonstrate that based on the current neurobiological understanding of psychopathy, the majority of previous treatment programs are misdirected, and that novel treatment programs should target individualized behavioural modification and skill learning to compensate for the specific neurological deficits of individual psychopaths.

Theories of Psychopathy

When discussing theories of psychopathy, it is most useful to consider it as a special subtype of a personality disorder. Psychopathy shares many traits similar to other personality disorders; including antisocial personality disorder (ASPD), and impulse control disorders such as conduct disorder (CD). On this basis, psychopathy is often mistaken as being analogous to these disorders (Hare, 1996). While psychopathy shares many of the antisocial and criminal traits with ASPD and CD, psychopathy is characterized by profound emotional impairments not seen in individuals

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with ASPD or CD. This is supported by Hare's PCL, where incarcerated offenders displaying antisocial-personality traits typically score highly on factor two of the PCL (factor two is related to antisocial behaviours), but only psychopathic individuals score highly on the first factor which measures emotional detachment (Weber, Habel, Amuts, & Schneider, 2008). It is therefore theorized that psychopathy is a subset of antisocial personality disorder.

Psychopathy is also unique in that it has a largely biological basis. It has been previously theorized that emotional trauma in early life, or environmental insult; defined as injury due to external circumstances, such as birth defects, may lead to psychopathy. However, there is not enough evidence to support this (Blair, Peschardt, Budhani, Mitchell, & Pine, 2006). Blair et al. (2006) argue that the emotional deficiencies seen in psychopathic individuals are a protective factor from early social causes. This is contrasted with CD and ASPD individuals who have a strong relationship between childhood trauma and emotional reactivity. Blair et al. (2006) also hold that birth problems and environmental insults are closely linked to reactive aggression, which is aggression in response to provocation. However, these explanations do not account for the unique expression of calculated, cold, instrumental aggression (pre-meditated aggression to achieve a gain) seen in psychopathy. Among these theories of psychopathy, Weber et al. (2008) describe two theoretical models that have informed current research.

Somatic Marker Hypothesis

The somatic marker hypothesis (SMH) devised by Damasio (1994) attributes psychopathy and its associated behaviours to prefrontal brain damage, which results in impaired decision-making abilities. This damage also inhibits activation of somatic states linked to reward and punishment. The

disruption of this somatic system inhibits learning of negative outcomes, and leads to difficulty in the application of appropriate social behaviours. Based on this theory, the ventromedial pre-frontal cortex may be involved in psychopathy, as it is the brain area linked to decision making processes. The biological basis for this theory will be elaborated upon in subsequent sections relating to the neural correlates of psychopathy. The SMH has been used to provide a model of psychopathy that has informed research on psychopathy and conceptualized the cognitive processes involved in psychopathy. However, this theory does not provide reasoning for how psychopathy emerges.

Violence Inhibition Mechanism Model

A violence inhibition mechanism model (VIMM; Blair, 1995) places emphasis on the role of empathy in the development of moral social skills (social behaviours that are morally based; e.g. honesty) and rule acquisition (acquisition of normative societal rules). This model theorizes the malfunction of a "violence inhibition mechanism" in animals that in normal functioning leads to halting aggression when subjects display submission cues (Blair, 1995). This theory implicates the amygdala as the primary neurological structure related to psychopathy due to its role in emotion and fear, with malfunction leading to a lack of empathy, and social rule acquisition. This theory is supported by cognitive and biological evidence. For example, psychopathic individuals demonstrated deficits in recognizing sad and fearful facial expressions. (Blair, Colledge, Murray, & Mitchell, 2001).

Theoretical Summary

There is evidence for both the VIMM and the SMH in current literature (Blair et al., 2001; Chow, 2000; Damasio, 1991; Koenigs, Kruepke, & Newman, 2010). On this basis, a combination of these may be warranted in future research. This combined theory of psychopathy is referred to as the Integrated Emotional

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System (IES) model, and is the most well-supported model of psychopathy (Blair, 2006). Psychopathic individuals show increases in reactive aggression that may be linked to abnormalities in the ventromedial prefrontal cortex (Mitchell et al., 2006). Deficiencies in empathy and emotional capacity may be associated with amygdalar dysfunction. Research on these brain structures indicates a high degree of connectivity between them in normal individuals (Banks et al., 2007). This connected system supports a theory of psychopathy that most likely displays abnormalities in these systems.

Neurobiological Basis of Psychopathy

Extensive research has been conducted into the biological and cognitive aspects of the amygdala and prefrontal cortex in psychopathic individuals. In a neuroimaging study, Tiihonen et al. (2000) used volumetric Magnetic Resonance Imaging (MRI) analysis on violent offenders classified into high and low psychopathic trait conditions based on scores of Hare's PCL-R. They found that individuals with high levels of psychopathic personality traits on the PCL-R demonstrated a decrease of approximately 20% in right hemisphere amygdala volume compared to healthy controls (Tiihonen et al., 2000). These results were obtained after adjusting for age, intelligence, and level of previous alcohol abuse, which are confounding factors in psychopathy research. The significant differences in amygdalar volume is evidence for the implication of amygdalar dysfunction in psychopaths, as well as the violence inhibition mechanism model of psychopathy.

In another study conducted by Mitchell et al. (2006), the acquisition of stimulus-reinforcement associations was examined. These stimulus-reinforcement associations were previously shown to require involvement of the amygdala, while the pre-frontal cortex is necessary for reversal learning, which is changing a previous behaviour in response to a

change in the stimulus-response pattern (Mitchell et al., 2006). An instrumental learning task and reversal learning task was administered to psychopathic individuals, community individuals, criminal-non-psychopathic individuals and to individuals with lesions to either the amygdala, right, or left prefrontal cortex. It was found that psychopathic individuals performed significantly worse than community or criminal controls on the task, and scored similarly to an individual suffering a lesion to the amygdala on the stimulus reinforcement association task. Psychopathic individuals also showed similar results to prefrontal cortex lesion individuals on the reverse learning task (Mitchell et al., 2006). This is further evidence for the roles of the amygdala and prefrontal cortex in psychopathy.

Further supporting the role of biology in psychopathy, in a neuroimaging study conducted by Vieira et al. (2015), psychopathic traits in normal healthy individuals were associated with volume alterations in the amygdala. Using the Triarchic Psychopathy Measure, 25 healthy individuals were assessed for psychopathic traits (Vieira et al., 2009), and voxel-based morphometry was used to determine structural correlates. It was found that psychopathy was negatively correlated with grey matter volume in the left putamen and amygdala, while some aspects of psychopathy such as meanness were correlated with increases in volume of the orbital frontal cortex (OFC; Vieira et al., 2009). This further demonstrates a link between the amygdala and prefrontal cortex in psychopathic individuals, while suggesting that changes in different areas are associated with various traits of psychopathy.

Further neuroimaging evidence was found in a study by Contreras-Rodriguez et al. (2015), where the functional connectivity in the prefrontal cortex of psychopaths was examined. Functional Magnetic Resonance Imaging (fMRI) was used to analyze psychopathic individuals compared to healthy controls. Results demonstrated psychopathic individuals

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exhibited grey matter reduction in prefrontal, paralimbic, and limbic structures. Further analyses of functional connectivity revealed decreased connectivity between the pre-frontal cortex and the limbic system, while greater connectivity existed between the prefrontal and frontal cortex (Contreras-Rodriguez et al., 2015). Contreras-Rodriguez et al.'s results suggest that the link between emotional and cognitive domains in the psychopaths brain may be weakened, as well as suggesting that there are enhanced functional connections in frontal executive areas. This finding provides support that connectional differences may contribute to psychopathy.

The study conducted by Contreras-Rodriguez et al. (2015) supported the previous result of Craig et al. (2009) where diffusion tensor MRI imaging was used to analyze the white matter connectivity of the OFC and amygdala in psychopaths. Psychopathic individuals were selected from a group of violent offenders using the PCL-R. Results demonstrated decreases in the connections in the OFC-amygdala pathway, further implicating these structures, and demonstrating that a combination of dysfunction in both most likely is related to psychopathy (Contreras-Rodriguez et al., 2015).

In order to examine other biological areas thought to be involved in psychopathy, Boccardi et al. (2010) examined other aspects of the limbic system, and abnormal hippocampal shapes were found in psychopathic individuals. In this neuroimaging study, 26 criminal offenders were assessed for psychopathy using the PCL-R, and MRI was used to determine neurological features. This study revealed specific shape changes in the hippocampus of psychopathic individuals compared to individuals who scored low on the PCL-R and normal healthy controls. These changes were consistent within the psychopath group, and different than both control groups, suggesting that psychopathic individuals do indeed share similar morphological features of the brain,

implying that there is a common etiological aspect to the disorder (Boccardi et al., 2010).

These studies are a sampling of the research in a large body of evidence implicating neurobiology in psychopathy. It has become increasingly clear in the last 15 years that psychopathy is a biological disorder, with cognitive and psychological symptoms. Abnormalities in the amygdala and prefrontal cortex (specifically the OFC) have been heavily documented in individuals with psychopathy. This biological basis of psychopathy provides strong evidence for why current treatment protocols are ineffective, and need to be re-examined as will be discussed in the next section.

Current Treatment Strategies

Psychopathy is widely considered difficult to treat (Reidy, Kearns, & DeGue, 2013). As a result of the specific behavioural and cognitive symptoms of psychopathy, it is not amenable to psychotherapy (Salekin, Worley, & Grimes, 2010). Psychotherapy involves honest conversation with the therapist, in order for insight to be gained, and behavioural and cognitive changes to be made (Chi, M. 2016). Psychopaths have a tendency towards pathological lying, which makes treatment of this method very difficult. Psychopathy is also characterized by an unwillingness to change, whereas psychotherapy is most effective when the client has a strong desire to change (Wampold, 2015). These characteristics, along with the lack of deep or lasting emotion make psychopathy difficult to treat (Salekin, Worley, & Grimes, 2010).

The majority of current treatment studies for psychopathy involve behavioural therapy or cognitive behavioural therapy (Reidy, Kearns, & DeGue, 2013). In the review conducted by Reidy et al. (2013) however, few positive results were found. The studies reviewed showed lack of methodological rigor, often not including non-treatment groups. The results also indicate a

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low success rate for adults. The success rates for adolescents are only slightly higher than for adults. These results suggest that psychopaths are a population that is resistant to treatment, but that further research is necessary to support this conclusion due to the weak experimental designs used in previous studies.

In another study by Umbach, Berryessa, and Raine (2015), a link between treatment and neuropsychology was established. Similar neuroimaging implications were found by Umbach et al. (2015) as discussed in the previous section, with abnormalities in the amygdala and OFC of psychopathic individuals. In their review, Umbach et al. (2015) related the neurological findings into the cognitive traits of psychopathy by citing research that implicates the amygdalar-OFC system in affect processing, moral decision-making, fear conditioning, and executive functioning. Umbach et al. (2015) suggest that both adults and juveniles who exhibit psychopathic traits exhibit impulse control, empathy, guilt, and fear impairments. These impairments are risk factors that are highly associated with the prefrontal and amygdala changes exhibited in psychopaths. The study also discussed the possible uses of neuroimaging features for courts, and how psychopathy diagnoses should be applied to criminal responsibility. Ultimately, there is no current method for efficiently treating psychopathy.

A Novel Treatment Model

With the lack of evidence for effective treatment methods for psychopathy, there is much to be desired in terms of new treatments. Most promisingly, advances have been found in treatments directed at children. The current treatments for psychopathy use CBT, a model that is not designed for psychopathic offenders, and thus may contribute to the low rates of success.

The high societal costs of psychopathy, both in monetary, and emotional damages, make treatment an important goal. In a new treatment

model, one of the first considerations should always be whether a program is ethical, whether it is feasible, and whether it is beneficial for the target population. Historically, many psychopathic individuals have been incarcerated for justice purposes (Blair, 1995). Despite the practical appeal of having potentially unpredictable individuals away from society, incarceration of psychopaths for actions based on an involuntary psychological condition may present an ethical issue. A potential solution is to improve treatment for psychopathic offenders in order to reduce reoffending. Relevant ethical concerns include whether the use of drugs would be justified in treatment, the level of consent that must be attained in order for treatment to advance, and whether offenders should be incarcerated during treatment.

In a new model of psychopathic treatment, several factors should be considered. A primary factor should be its direction towards adolescents. While still being adaptable to adult offenders, perhaps the most effective way to prevent psychopathic incarceration, as supported by previous research, is targeting treatments at young offenders (Salekin et al., 2010). By increasing the time and resources devoted to this population increased results may be found. This method has already demonstrated promising results (Salekin et al., 2010), and further research should examine this approach to treatment.

Another strategy to improve treatment of psychopathic individuals would be to adopt an individually tailored treatment model. This treatment model could be devised using an individual's specific history, family circumstances, and symptoms. Based on the neurological evidence discussed, therapies could be individually tailored to address a patient's specific neurological abnormalities. According to Salekin et al. (2010), an emphasis on the developmental model of psychopathy, the theory of treatment, and the effects of treatment on individuals, are key components in an individually tailored treatment model. The

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current model being used follows CBT interventions such as promoting self-awareness, challenging cognitive distortions, and practicing positive behaviours (Reidy, Kearns, & DeGue, 2013). A new treatment method would begin with cognitive testing in neural areas where suspected neural deficiencies are located. Then, a specific treatment program would be designed to strengthen deficient areas (Reidy, Kearns, & DeGue, 2013). For example, it has been found that virtual reality programs can successfully improve social rule learning in autistic individuals through modelling role-playing behaviours (Parsons & Mitchell, 2002). Other similar inventions should be investigated to determine whether these improvements in social functioning could be transferred to psychopaths with a specific weakness in social rule learning.

It has been found that psychopaths display more reward-seeking behaviour than normal individuals (Hare, 1996). New methods of treatment should focus on improving healthy reward seeking, rather than using punishment which psychopaths have decreased sensitivity to. Treatment methods for psychopathy other than CBT may be helpful due to the lack of results from traditional methods found so far, due to the limited effectiveness of current treatment methods.

Another potential area of focus for the treatment of psychopathy is drug therapy. Due to the highly biological nature of psychopathy, drug therapies may be a more successful alternative to traditional psychotherapy. Future researchers may examine the potential effectiveness of stimulants on psychopaths. The deficits with executive function seen in psychopaths are also common to individuals with ADHD, who can be successfully treated with stimulants (Morgan & Lilienfeld, 2000). Another possible drug intervention could be the use of antidepressants to treat impulse control behaviours (Schreiber, Odlaug, & Grant, 2011). Since benefits have been found using these drugs in non-psychopathy related impulse control disorders with similar neurological

features, they may be useful if implemented in a future treatment method for psychopathy (Schreiber et al., 2011). The possibility for development of new genetic technologies that can target genetically causal sequences also presents a promising area for future research.

Summary

Previous research has supported a biological basis for psychopathy. Researchers continue to improve the scientific understanding of the mechanisms and underlying structural abnormalities involved in psychopathy. However, the treatment of psychopaths has not followed the advances in research, and remains, for the most part, ineffective. Future research should explore improved methods of treating psychopathy, using treatments that are adolescent-focused, individually-tailored, and strength-directed. Researchers developing new treatment should also investigate the potential of a drug component. Stemming from the existing evidence that psychopathy is neurobiologically based, the current treatment programs are ineffective, and new methods of treatment should be developed.

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