AN EXPLORATION OF PSYCHOPATHY AS A NEUROSCIENCE CONSTRUCT

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ABSTRACT

Hare's psychopathy construct as defined by the Psychopathy Checklist- Revised has been utilized internationally as a risk assessment instrument for quite some time. Despite this, since its inception it has and continues to raise criticism from the academic community. There is ongoing debate over what the construct entails and how it should be used. Most recent developments in the construct revolve around it being defined as a neurological manifestation. To explore the psychopathy construct's connection with neuroscience, this thesis focusses on one foundational experiment by the most prominent team of researchers in the field. The exploration borrows from Science and Technology Studies, more specifically Actor-Network Theory and the semiotic of scientific texts. The goal of this analysis is not to criticize nor defend the psychopathy construct, but rather explore the facticity of psychopathy as a neuroscientific fact. Considering the widespread use of the construct across criminal justice systems and mental health practices, understanding the facticity of psychopathy is imperative. Our contention is that psychopathy as defined by neuroscience was not merely a pre-discovered fact of nature, but rather it is a fact that is hybrid; it is both built by researchers and a part of our natural world, social and real. Our findings reveal that the facticity of psychopathy as a neuroscience construct is reliant on it being a Boundary Object: a scientific object that is able to intersect multiple social worlds through its adaptability (Star & Griesemer, 1989). We show how the construct is a boundary object by detailing the many translations it undergoes while it connects with a variety of heterogeneous actors. For each translation, the construct is rendered qualitatively different, yet it proves to be robust enough to maintain the identity of psychopathy and transform it into a neuroscientific fact.

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THE PAST AND FUTURE OF PSYCHOPATHY

Both the popular sphere and the social sciences are experiencing a "neuro" turn. Indeed, neuroscience assumptions, facts and practices are penetrating the realm of the "social" and promulgating a discourse that unites brain, mind and behaviour (Ortega & Vidal, 2011; Rose & Abi-Rached, 2013). This turn affects criminology to the extent that some propose that neurocriminology is the new paradigm in the discipline (Walsh & Beaver, 2009). An overarching question is, how is this neuro turn concretized in criminology and what is its significance? Inspired by Science and Technology Studies (STS), and more specifically Actor-Network Theory (ANT), our research is a case study of the way neurosciences contribute to the development of the psychopathic personality construct. We propose to use ANT to describe the way neuroscience is redefining psychopathy and thus forming a new network of knowledge, practice and scientific facts that try to solve the controversies surrounding this construct. Empirically, the focus of our study is facticity (how facts are produced); the way neuroscience assertions about psychopathy become facts. We start this introduction with a brief history that begins with the modern conception of the construct as described by Hervey M. Cleckley, whose work embarked a line of research that resulted in the current psychopathy construct. Next we present the contemporary definition of psychopathy that Robert Hare builds from Cleckley's original work. We then briefly touch on the controversies surrounding psychopathy as a personality disorder. Finally, we finish the chapter by discussing how neuroscience has permeated the research and discussion on psychopathy, and we narrow down on our study and its pertinence.

The following historical summary is simply a means of informing the reader of what psychopathy is and how it came to be. It is important for the reader to be aware of the history of the construct, so that they can better appreciate the extent of its most recent adaptations. The research object in this thesis, contemporary psychopathy as a neuroscience construct, is delineated in the last section of this chapter. The chapter ends with a description of our research object, and an explanation of how this STS/ ANT study contributes to knowledge on psychopathy.

A Brief History of Modern Psychopathy

Hervey Cleckley, MD worked in a psychiatric facility where he observed patients that did not display the usual signs and symptoms of mental illness, and after an initial assessment seemed relatively normal (Cleckley, 1976; Babiak & Hare, 2006). Over time, Cleckley observed these patients charm, manipulate and con other patients, family members, and hospital staff (Babiak & Hare, 2006). He came to understand and classify these patients as psychopaths. Eventually, Cleckley's work would mark the beginning of the modern clinical construct of psychopathy, which has maintained relative stability to this present day (Arrigo & Shipley, 2001). Cleckley would go on to publish 5 editions of *The mask of Sanity* (1941, 1950, 1955, 1964, 1976), which is considered a classic textbook on psychopathy, and has greatly influenced the development of the most up to date diagnostic criteria (Babiak & Hare, 2006; Hare, 2004, Hart & Hare 1997; Lykken, 2006). He was also the first to recognize that the psychopaths who were able to evade a life of crime would often succeed as businessmen where they could hone in on the benefits of their abnormal characteristics and find material success.

Based on his observations of white middle-classed males in a psychiatric hospital, Cleckley devised a set of 16 features to describe his conception of psychopathy (see Appendix: Table 1). The current conception of psychopathy and its associated characteristic traits cohere, for the most part, with Cleckley's original description; many of the items in the Psychopathy Checklist- Revised (PCL-R) diagnostic instrument (current standard practice for diagnosis) were based on Cleckley's work (Hare, 2004). Cleckley's list of features is better interpreted as a summative description and grouping of his observations (Cleckley, 1976). Cleckley's work on psychopathy consists of an in-depth analysis on a relatively small sample of clients. His 16 feature description of the psychopath is therefore a summary of his observations. His description was by no means intended for diagnosing the disorder (Babiak & Hare, 2006). At that point in time the ability to diagnose Checkley's construct was limited to Cleckley himself and his close medical associates. Nevertheless, Cleckley can be attributed to providing the first description of the current construct, a description that is still referred to today, and used as a foundation with regards to discussions around how the construct should be defined.

The first edition of the American Psychological Association's (APA) *Diagnosic and Statistics Manual of Mental Disorders* (DSM) was published in 1952, just over a decade after Cleckley's first edition of *The Mask of Sanity*. The DSM[1] renames the construct from Psychopathic Personality Disorder to Sociopathic Personality Disorder. This change in terminology reflected a change in the etiological assumptions about the disorder, which suggested that it should be attributed to the effects of cultural and social factors on the individual (Arrigo & Shipley, 2001). In regards to the age-old Nature-Nurture debate, while the psychopath is considered a product of nature (a biological abnormality), the sociopath is the product of their social surroundings (Lykken, 2006). Regardless of etiology, the DSM maintained symptom criteria that was similar to Cleckley's psychopath description for the diagnosis of Sociopathic Personality Disorder. That is, the focus was on internal processes and personality traits. However, these psychosocial causal attributions would later shift the emphasis on diagnostic criteria from internal personality traits to external behavioral symptoms.

The DSM-III (1980), DSM-III-R (1987), and the DSM-IV (1994) replaced the Sociopathic Personality Disorder with Antisocial Personality Disorder (ASPD). The DSM-IV claimed that ASPD "has also been referred to as psychopathy, sociopathy, or dissocial personality disorder" (Frances, 1994, p. 645). The disorder was completely revitalized to focus on external behavioural characteristics. The behavioural characteristics, which were originally explained as caused by the personality traits, were deemed more reliable for diagnostic purposes (Hare, 1996). The controversy that lies behind ASPD is that the behavioural characteristics used for diagnostic criteria are said to be the consequences of the true underlying disorder, personality (Hare, 1996; Millon, 1981). Thus, in increasing diagnostic reliability, some argue that the construct's validity has been sacrificed (Hart & Hare, 1997; Hare, 1998).

The Development of the Current Construct

In 1980 Robert D. Hare published the *Psychopathy Checklist* (PCL). Hare fully ascribed to Cleckley's work on psychopathy, while discounting the APA's conception of the ASPD

[1] This paper amalgamates the DSM and the DSM-II, because in regards to the sociopathy disorder, the differences between editions are scarce. The only notable distinction is that the DSM-II eliminates the dissocial/ antisocial distinction for the diagnosis (Arrigo & Shipley, 2001).

disorder as invalid (Babiak & Hare, 2006; Hart & Hare, 1997; Hare, 2004; Hare & Hart, 1993; Hare & Neumann, 2008). However, he considered Cleckley's work unfinished and proposed that "confirmation of his observations and development of scientific methods for assessment, therefore, was left to others" (Babiak & Hare, 2006, p. 22). Thus, the PCL was an attempt to operationalize Cleckley's concept of psychopathy, which involved moving beyond mere description and into a scientifically verifiable, reliable and valid diagnostic instrument. After publishing the original checklist in 1980, Hare collected more data and created a more statistically sound PCL-R (Hare, 2000).

The PCL-R is a 20-item instrument (See Appendix: Table 2) on a 0-40-point scale; each item can be scored as either 0- does not exhibit the trait or behaviour in question, 1- item applies to a certain extent but not to the degree required for a score of 2, or 2- item applies to the individual (Hare, 2004). The scoring on the PCL-R is conducted using both a semi-structured interview, and a review of collateral data (i.e. previous psychological assessments, testaments from family/ friends/ past employers, information gathered from the offender's file/ Criminal Justice System [CJS] interactions, etc.) (Forth, 2000; Hare, 1991, 2004, 1996).

Although still a subject of ongoing debate and inquiry, the PCL-R items have been organized according to two separate factors, which can then be divided again into four facets (see Appendix: Table 4). Factor 1 (facets 1 and 2) represents the constellation of interpersonal and affective traits that are considered to be the core features of the construct. The assessment of factor one traits depends on clinical inferences about affective processes, verbal and interpersonal style (Hare, 2004). The second factor (facets 3 and 4) describes a lifestyle defined by chronic instability, antisocialism, and deviance (Hare, 2004). Naturally, then, factor one is primarily concerned with personality characteristics, and would rely more heavily on structured clinical judgement, while factor two is primarily concerned with behavioral characteristics that can be discerned from collateral data. Although the PCL-R is intended to be a clinical diagnostic instrument, it has mainly been utilized for other purposes. First, it is often used as a representation of the psychopathy construct for research purposes (Cooke, Michie, & Hart, 2006; Edens, Skeem, Cruise, & Cauffman, 2001; Meloy, 2000; Seagrave & Grisso, 2002). Second, it

has been employed internationally as a risk assessment instrument in corrections (Webster & Hucker, 2007).

Research has discovered a remarkable correspondence between violent recidivism and a psychopathic personality diagnosis, as offenders who are psychopathic have higher rates of reoffending than non-psychopathic offenders (Webster & Hucker, 2007). Risk in corrections is defined as the probability that an offender will reoffend either within the institution or upon release (Webster & Hucker, 2007). At this point in time there has been no effective evidence-based treatments for psychopathy (Andrews, 2000; Hare, 1995; Kiehl, Hare, McDonald, & Brink, 1999; Webster & Hucker, 2007). As such, a psychopathy diagnosis is considered a static risk factor (one that cannot be changed), rather than a dynamic risk factor (one that can change over time) that can be targeted for rehabilitative intervention (Andrews & Bonta, 2010). Thus, being diagnosed with psychopathy is very debilitating for an offender, as they do not have the ability to change a risk factor that informs the decision-making of Criminal Justice System (CJS) authorities on how to process them through the system (e.g. security level of institution, parole eligibility, dangerous offender designation, access to rehabilitative interventions, long-term supervision orders, and stricter sentencing).

At this point in time the construct validity of psychopathy has only been validated on forensic patients: clients with recorded criminal or delinquent histories (Walters, 2004; Webster & Hucker, 2007). Thus, its ability to identify psychopathic personalities is limited to populations that have been in contact with the CJS, where diagnosticians can access necessary historical client information. Hare has developed the PCL-R- Screening Version (SV) and the PCL-R-Youth Version (YV) for non-forensic community populations, but these instruments are deemed less accurate and not as empirically validated as the PCL-R (Babiak & Hare, 2006; Hare & Neumann, 2008). Hare is clearly attempting to broaden the scope of the psychopathy construct to non-forensic populations with the PCL-SV and the PCL-YV.

Survey of Controversies Related to Psychopathy

Present day etiological research on psychopathy has created an excess of conflicting assertions both between and within paradigms. The confusion over etiology is a multilayered entanglement with conflicting and unresolved theories that are each supported or refuted with a vast array of conflicting hypotheses. What is meant by multilayered is that the controversy starts by trying to determine symptomology, followed by an attempt to explain the cognitive and affective deficits that cause the symptoms, followed by an attempt to explain the causes of the cognitive and affective deficits, and then followed by attempts to determine prognosis and best practice treatments. At each level of exploration there is controversy, and so it becomes problematic when these unresolved assertions begin to build off one another, because the premises from which they build their hypotheses are not always confirmed. Although these controversies are discussed in detail in the following chapter of this paper, consider this brief overview for an idea of what to expect.

In terms of symptomology, there is still ongoing debate between APA/DSM ASPD and PCL-R psychopathy (Arrigo & Shipley, 2001; Hart & Hare, 1997; Hare, 1996, 1998; Millon, 1981; Widiger, 2006); there is controversy in regards to the homogeneity of psychopathy and whether or not the disorder needs to be divided into primary and secondary subtypes (Fowles & Dindo, 2006; Hall & Benning, 2006; Poythress & Skeem, 2006); there is controversy over theoretical concerns (Jalava, 2007; Ronson, 2011) and the tautological nature of the construct (Cooke, Michie, & Hart, 2006; Cooke, Michie, & Skeem, 2007; Ellard, 1988); and there are also reliability/ validity concerns with the PCL-R (Cooke, Michie, & Hart, 2006; Edens, 2006; Miller, Rufino, Boccaccini, Jackson, & Murrie, 2011). It is true that research on psychopathy has been hampered with the methodological limitations caused by the use of multiple and problematic operationalisations of the construct (Seto & Quinsey, 2006).

The literature that describes symptomology by referring to potential cognitive and affective deficits is equivocal, and includes assertions related to low fear, low anxiety, behavioural activation systems, startle responses, linguistic and emotional processing, poor affective appraisals, high stimulation thresholds, response modulation difficulties, hypersensitivity to reward and hyposensitivity to punishment, passive vs. active avoidance of punishment etc. (Blackburn, 2006; Fowles & Dindo, 2006; Hare & Jutai, 1988; Hare 1984;

Kiehl, hare, McDonald, & Brink, 1999). Many of these competing theories are in direct conflict with each other, while others maintain the possibility of convergence. Additionally, some of the theories focus more on specific diagnostic items, and while these theories lend support to certain aspects of the construct, they simultaneously conflict other aspects (e.g. work supporting notions of behavioural impulsivity simultaneously contradict the idea of cold, calculated and manipulated interpersonal styles). What is important to consider here is that the theories surrounding the potential cognitive and affective deficits of psychopaths have implications for other layers of research; it produces backward controversy in regards to the diagnostic criteria, and it produces lines of inquiry for research on the causes of the cognitive and affective deficits that could be based on unaccepted assertions.

There are many competing discourses in relation to the causes of cognitive and affective deficits in psychopaths. These include social learning/ environmental attributions (APA, 2013; Farrington, 2006; Krischer & Sevecke, 2008; Salekin, Rosenbaum, Lee, & Lester, 2009); biological/ neurobiological/ neurochemical/ genetic attributions (Blair, 2001, 2003, 2006; Decety & Ickes, 2011; Glenn and Raine, 2014; Hare, 1984, 2004; Hare & Jutai, 1988; Kiehl, Smith, Hare, Mendrek, Forster, Brink, & Liddle, 2001; Minzenberg & Seiver, 2006; Raine & Yang, 2006; Waldman & Rhee, 2006); and a combination of the two (Barnow & Freyberger, 2010; Taylor, Loney, Bobadilla, Iacono, & McGue, 2003; Waldman & Rhee, 2006). It is important to note that both between and within social, biological, and combination paradigms there is controversy. While some researchers are willing to make large sweeping assertions based on their research, others are humbler in their approach and suggest that the knowledge is at an extremely formative stage (Rogers, 2006). At this point in time a reasonable stance on the understanding of the psychopathy construct is, “much work remains to be done before we can claim to understand the “Cleckley Psychopath” per se, or distinguish it from antisociality more generally in terms of its etiology” (Macdonald & Iacono, 2006, p. 375).

Neuroscience and psychopathy: Narrowing in on the Agenda

This introduction has provided a historical account for the psychopathic construct, and in doing so has provided clear evidence of its complexity, multifaceted nature, and vulnerability to

conceptual instability and ambiguity. To reiterate, Macdonald and Iacono (2006) recognize that at every layer of research discussed above, in the "different method[s] for understanding psychopathy, there is little convergence between the substantive hypotheses; each method leads to divergent conclusions about the nature of psychopathy… this has to be recognized as an uncomfortable state of affairs" (p. 375). Despite this, research seems to be running rampant in all directions, building off of produced "facts" that have unanswered counterfactual evidence.

What is interesting, and a particularly important point for this thesis, is recognizing that present day research has been primarily focused on the neuro-biology/chemistry/anatomy of psychopathy (Walsh & Beaver, 2009). There are thousands of research articles that allude to the neuroscience explanation of the disorder. Importantly, the creator of the modern-day construct, Robert Hare, is himself focused on explaining the neuro aspects of psychopathy; his own biography from the Darkstone Research Group Ltd. (for which Hare is the president) explains that his current research on psychopathy is attempting to identify and explain neurobiological correlates.

Despite momentous controversy, the PCL-R itself has become regarded by many as "the construct and measurement of psychopathy" (Seagrave & Grisso, 2002), the "standard of practice instrument" (Meloy, 2000, p. 43), and "the gold standard" (Edens et al., 2001, p.74). Cooke et al., (2006) caution researchers, however, "we must be alert to the danger that mono-operation bias may lead us into a conceptual cul-de-sac" (p. 103). In the wake of current research on psychopathy that has been largely unidirectional (applying neuroscience practices to psychopathy as it is defined by the PCL-R), this paper appreciates this word of caution. As the neurocriminology field continues to produce neuroscientific facts about psychopathy at alarming rates, it is important to consider how this construct is being redefined, and what the implications of this definition could entail. Exploring the development of the psychopathic construct in relation to neuroscience is an important contribution to social sciences, as this development will indeed entail clinical and forensic implications. For example, neurological causation begets a likely trajectory of pre-emptive 'screening and intervening' (e.g. preventative detention) on risky brains in adults and delinquent youth (Eastman & Campbell, 2006; Rose, 2010; Salekin, Rosenbaum, Lee, & Lester, 2009; Sevecke, 2011; Slough, & McMahon, 2008); increased

psychotropic and genetic interventions (Leifer, 2000; Rose, 2000; Rose, 2010); and ultimately, a CJS that reduces violent criminality to individual neurobiological factors, and fails to endorse policy that targets broader systemic societal factors. It is reasonable to ascertain that this redefining of psychopathy will be accompanied by a restructuring of clinical (surgical and psychopharmacological), forensic, correctional, and risk management practices (Leifer, 2000; Rose, 2000; Rose, 2010; Salekin, Rosenbaum, Lee, & Lester, 2009; Sevecke, 2011; Slough, & McMahon, 2008). Considering how the redefining of psychopathy as a neurological disorder entails a broad scope of plausible implications, it is undeniably important to understand what this definition will be, and how the research is creating it.

The scope of this paper will be limited to exploring the controversy surrounding a series of neuroscientific assertions about psychopathy; it is impossible to investigate the breadth of all the research on the topic in this paper. The data is limited to a selected academic article, "Limbic Abnormalities in Affective Processing by Criminal Psychopaths as Revealed by Functional Magnetic Resonance Imaging" by Kent A. Kiehl, Andra M. Smith, Robert D. Hare, Adrianna Mendrek, Bruce B. Forster, Johann Brink, and Peter F. Liddle (2001). Hereafter referred to as "the Kiehl (2001) article", as Kiehl is the main author. Despite this limited data set, this exploration may enlighten readers on the scientific processes that are at work to produce the plethora of facts around psychopathy and neuroscience. Our contention is that the analytical method employed, and the results obtained, may be applicable to much more than what is under the close analysis of this investigation.

We explore the facticity of neuroscience and psychopathy by utilizing Science in Action (1987) by Actor-Network Theorist, Bruno Latour. Latour (1987) provides an understanding of fact-making that appreciates the utility of both laboratories and texts in the process. Latour's (1987) method allows us to explore the Kiehl (2001) article itself, the preceding research (i.e. references) in the Kiehl (2001) article, as well as how Kiehl (2001) is later utilized (i.e. cited) by proceeding researchers. Let us imagine that the Kiehl (2001) article is placed in the middle of a flowing river, if we move upstream, we analyze how the Kiehl (2001) article utilized previous texts through referencing. If we move downstream, we analyze how the Kiehl article has been cited by others. Our thesis starts in the middle of things, with the Kiehl (2001) article and its

laboratory, and moves up and down stream in the analysis to get a holistic exploration of the facticity of the neuroscientific facts on psychopathy produced by Kiehl (2001). The case study of the Kiehl (2001) article presented in this paper attempts to illuminate the inner-workings of the neurocriminology field and provide a unique understanding of neuroscience and psychopathy.

Our contention is that the Kiehl (2001) facts are not merely pre-existing facts of nature that were discovered in a laboratory. Rather, they are created and built through work that involves building associations with other actors. That is, these facts of nature are products of scientific work that builds a reality, a concretized construct. We assert that psychopathy as a neuroscientific construct is built through a series of translations that requires many implicit techniques utilized by researchers. Our thesis shows how the nature of psychopathy is constantly changing to qualitatively different forms as a means of becoming an entity that is defined by the neurosciences.

We show how researchers utilize technical writing techniques, as well as each other's previous works, and instruments to accomplish these translations. We show how heterogeneous entities come together to translate psychopathy, and how psychopathy exercises flexibility as a construct. Psychopathy is made to change its composition many times over, and the many forms it takes provides it with the many qualities necessary to be a neuroscientific construct. In exercising flexibility as a means of connecting with the neurosciences, psychopathy can be defined as a "boundary object"; a scientific object that inhabits several intersecting social worlds, which is plastic enough to adapt to local needs, yet robust enough to maintain a common identity across sites (Star and Griesemer, 1989, p. 393). Psychopathy is a boundary object, in that it is flexible and adaptable to the many social worlds it intersects (i.e. neuroscience, social science, corrections, mental health, business, academics, etc.). This is the primary contention of our thesis, that psychopathy is a boundary object. In the following chapters we show how psychopathy meets the three criteria of a boundary object outlined by Star (2010); (1) the object resides between social worlds where it is ill-structured; (2) the object is worked on by local groups who maintain its vaguer identity, while making it more specific and tailored for their

local use; and (3) the heterogeneous social groups cooperate without consensus and tack back-and-forth between the forms of the object.

Now, to localize this thesis in relation to other STS research on psychopathy we consider the contributions of Pickersgill (2009) for it is the study that is the closest to our own. With a similar theoretical positioning as our own (STS), Pickersgill (2009) interviews prominent researchers who study neuroscience and psychopathy. Pickersgill's (2009) study looks at psychopathy as a clinical disorder, and explores how neuroscience literature pervades the social realm of clinical psychiatry. His study essentially explores how neuroscience assertions impact the psycho-social realm, and discusses the implications of neuroscience on clinical practise (i.e. discussion on biological reductionism, determinism, techniques of governance and intervention). While Pickersgill (2009) takes neuroscience assertions and moves forward to discuss their consequence in the psycho-social realm of clinical psychiatry, we do the opposite, we move backward. This thesis takes neuroscience assertions and unfolds them, unpacks their contents so that the readers of past literature can better understand the material that composes the facts. We present science as a craft by elucidating the processes and materials used to produce neuroscience facts on psychopathy. That is our contribution to the knowledge of psychopathy; we explore psychopathy as a scientific construct (whereas Pickersgill (2009) explores psychopathy as a clinical disorder), and unfold the work that has been neatly packed into academic articles. Essentially, while Pickersgill (2009) studied discourses on neuroscience colonization in the mental health domain, this thesis studies the material semiotic of psychopathy as a neurofact, how it comes to be.

To support our assertions we start by reviewingthe pertinent literature on neuroscience and psychopathy; it is followed by a chapter on our theoretical background and research methods; then a chapter is dedicated to an analysis of the Kiehl (2001) laboratory; another chapter present the upstream analysis of the Kiehl (2001) referencing techniques; and a last analysis chapter pertain to the downstream analysis of the works that cite the Kiehl (2001) article. Finally, the conclusion will argue that the neuroscience construct of psychopathy is a Boundary Object characterised by an inherent and productive fuzziness.

PSYCHOPATHY AS AN INDICATOR OF SUCCESS, A MEASURE OF RISK, A CLINICAL PRODUCT AND A SCIENTIFIC CONSTRUCT

In this chapter a detailed, but by no means exhaustive review of the literature regarding psychopathy is presented. The chapter is organized along four problematizations of psychopathy. First, we discuss psychopathy as a set of personality characteristics exhibited by successful business executives. Second, we present psychopathy as a correctional tool for risk management purposes. Third, we discuss psychopathy as a clinical instrument for diagnosing and treating psychiatric patients. Finally, this chapter closes with the problematization that we will adopt: psychopathy as a scientific construct, a construct where controversy and uncertainty proliferate. Approaching psychopathy as a scientific construct, will lead us to use Science and Technology Studies (STS), and more specifically, Actor Network Theory (ANT) as a theoretical framework for analyzing the neuroscience research on psychopathy. Although the first three problematizations of psychopathy presented in this chapter are not the ones adopted for this thesis, it is important that they are detailed so that the reader can appreciate the many social worlds that the construct, and consequently the neuroscience, affects. Reviewing the literature according to the first three problematizations is a means of sharing with the reader what psychopathy is outside of the world of research and science. This information allows for a better understanding of how the fourth problematization- psychopathy as a scientific construct, the topic of this thesis, can impact the other problematizations.

Psychopathy as a Trait Disposition for the Successful

A psychopath is often thought of in sublime manifestations including the most hardened and sadistic criminal cases, such as serial killers, rapists, cannibals, animal abusers/ killers. However,

> these cases that come to the public's attention are only the tip of the iceberg… the rest of the iceberg is to be found everywhere- in business, the home, the professions, the military, the arts, the entertainment industry, the news media, academe, and the blue-collar world. Millions of men, women, and children, daily suffer terror, anxiety, pain, and humiliation at the hands of psychopaths" (Hare, 1995, p. 115).

Although the PCL-R has only been validated on offender populations, and includes items that focus on criminality, Robert Hare (1995) attests that there are indeed psychopaths within the general population who are able to evade the Criminal Justice System. Researchers assert that there is a subtype of psychopath that differs from criminal psychopaths, as they are able to abstain from criminal behaviors, or at least avoid being caught (Babiak & Hare, 2006; Hall & Benning, 2006; Hare, 1995).

This subtype is often referred to as a "successful psychopath", and they are able to appear as high functioning members of society- as business executives, lawyers, academics, mercenaries, police officers, artists, doctors etc. (Babiak, 1995; Babiak & Hare, 2006; Hare, 1995). Despite their ability to achieve socioeconomic status, Hare (1995) refuses to use the term "successful". Instead, he labels the subtype *subcriminal psychopaths*, because their success is nothing more than a façade; achievements made from illusory and unethical methods at the expense of others. That is, although this population is able to build and maintain personal success, it comes at a cost; lives are ruined and corporations internally destroyed as the subcriminal psychopath ruthlessly and greedily lies, cheats and manipulates their way up the corporate ladder (Babiak, 1995, 1996, 2000; Babiak & Hare, 2006; Babiak &Neumann, 2010; Hare, 1995). These psychopaths either operate just within the parameters of the law (although certainly not within any sort of ethical standards), or are able to transgress the law without detection (Hare, 1995). There is much speculation and research on what differentiates this subtype from the criminal psychopath. This section considers three prevailing theories on what constitutes the successful/ subcriminal psychopath, and finishes with a brief discussion on the nature of successful psychopathy research.

The first conceptualization of the noncriminal psychopath originates with the founder of the modern depiction of psychopathy, Hervey Cleckley (1982), who describes them as incomplete or subclinical manifestations of the disorder. This conceptualization is premised on the notion that the core personality traits of the psychopath (reflective of PCL-R factor 1 items) are directly responsible for the behavioral manifestations of the disorder (factor 2 items). Thus, subcriminal psychopaths have subclinical personality characteristics, which results in less severe social transgressions at a lower frequency (Cleckley 1982; Hall & Benning, 2006). This conceptualization also aligns well with Gustafson and Ritzer's (1995) "Aberrant Self-Promotion

Theory" of psychopathy, which labels subcriminal psychopaths as Aberrant Self Promoters (ASP). They describe ASPs as those who commit crimes sporadically for narcissistic purposes, and are of the same kind as psychopaths but to a lesser degree.

The second conceptualization of noncriminal psychopaths considers it a 'moderated expression' of the disorder (Hall & Benning, 2006). That is, like the first conceptualization, noncriminal and criminal psychopaths share a common etiology (behavioral/ factor 2 items are consequent of the core personality characteristics/ factor 1 items). However, unlike the first conceptualization, the moderated expression conceptualization asserts that the noncriminal psychopath and the criminal psychopath share equivalent severity of the core personality characteristics. The difference between the criminal and non-criminal subtypes is that moderating factors (e.g. competent parenting, high socio-economic status, prosocial peers, effective socialization, high intelligence, etc.) subvert the non-criminal psychopath from acting out. Their underling trait disposition (genotype) can be channeled into socially sanctioned avenues, such as politics, business, athletics, extreme sporting, etc. The moderating factors determine how the trait disposition (genotype) will be manifested (phenotype), criminal or successful (Hall & Benning, 2006). This conceptualization is aligned with Lykken's (1995) model discussed later in this chapter, as he explains how society's heroes and leaders often come from the psychopathic genotype, avoid criminality, and attain greatness through the presence of the appropriate moderating factors (highly effective parenting and socialization, and high socioeconomic status).

Finally, the third conceptualization, the duel-process model, changes direction from the former conceptualizations by attesting that the non-criminal and criminal psychopaths have differing aetiologies (Patrick, 2007). This conceptualization draws from the research on the two-factor model of psychopathy, as detailed by the PCL-R factor analysis (Hare, 2004). In the duel-process model conceptualization, it is believed that the factor 1 and 2 features of psychopathy are etiologically distinct. That is, the interpersonal/ affective traits have different etiological mechanisms than the antisocial/ deviant traits. From this belief, researchers suggest that the successful psychopath would have a genotype that reflected the interpersonal/ affective traits, while the criminal psychopath genotype would reflect the antisocial/ deviant traits.

It must be noted that this research is purely exploratory and speculative. Researchers have hardly grasped the nature of the disorder outside of correctional settings. This is expected, considering that the PCL-R is the only validated instrument for diagnosing psychopathy, and its validation is intended for, and limited to, correctional populations. There are many methodological problems surrounding the study of psychopathy in a community setting that have yet to be solved (Hall & Benning, 2006). First, there are items on the PCL-R (e.g. criminal versatility) that rely on access to formal legal documents, and these are either nonexistent or inaccessible within community samples. Second, and similarly, the PCL-R was designed for criminal populations, so most of the items relate to deviant behaviors, or are detected through patterns of deviance and criminality. This not only makes psychopaths difficult to detect without historical data, but also makes it impossible to detect the topic of this section, the non-criminal psychopath; the non-criminal or successful subtype may not even be the same species as that which is detected by the PCL-R. Finally, the PCL-R is not researcher friendly when applied to community samples. The PCL-R requires a lengthy semi-structured interview, which would be costly and time consuming to administer on a sample where the base rate of psychopathy is only an estimated 1%. Thus, before we are able to study the nature of the successful psychopath further, we must develop instruments that are better able to detect them. Researchers have been working toward the development of such instruments, such as the PCL-SV, Psychopathy Q-Sort (Reise and Oliver, 1994), Psychopathy-Scan (Hare & Herve, 1999), the Busines-Scan (Babiak & Hare, 2012), Self-Report Psychopathy-II (Hare, 1991), Levenson's Self-Report of Psychopathy (Levenson et al, 1995), and the Psychopathic Personality Inventory (Lilienfeld & Andrews, 1996). However, these assessment/ diagnostic instruments are also exploratory, as they have not yet been able to achieve similar levels of confidence in diagnosing the disorder as the PCL-R (Hare, 2004, Hall & Benning, 2006).

Psychopathy as a Correctional Tool for Risk Management

Rose (2010) defines a risk society as a society obsessed with the fantasy of security and minimizing the risk of harms. In this context, the public demands protection from criminals, those who are perceived as outsiders, violent predators that compromise public safety and well-being. The public demands that their risk of victimization be reduced. Risk in relation to mental disorder is a high priority for many, because of the perception that violence is rooted in mental

pathology (Monahan et al., 2001; Rose, 2010). This drives the public demand for mechanisms capable to identify, categorize, and arbitrate the psychopaths who are believed to have a high propensity for violently offending.

In accordance with this notion of a risk society, Criminal Justice Systems (CJS) are operating under a "New Penology". Operating under the 'new penology', risk assessment and management of offenders is the paramount objective of the CJS instead of proportionality of crime and punishment/ retribution or rehabilitation (Feeley & Simon, 1992). Since the psychopathy diagnosis is positively correlated with violent recidivism (Dolan & Doyle, 2000; Hare, 2004), the PCL-R is used in corrections as a risk assessment instrument, and so those diagnosed with psychopathy are subject to being managed accordingly (e.g. indeterminate sentencing, dangerous offender designations, long-term supervision orders etc.). This section of the chapter details how the CJS defines psychopathy as a tool for risk management through its ability to predict violent recidivism.

The Conceptual Link.

After multiple studies established the position that mental health professionals were unable to predict violence or recidivism through traditional structured clinical judgements (Monahan et al., 1981), there was a push for finding an alternative means to predict violent recidivism. Naturally then, there was a prevailing view that psychopathy as a clinical diagnosis had little utility in understanding and predicting criminality (Hare, 2004). That is, since psychopathy is construed as a mental disorder by clinical practitioners, and their structured clinical judgements were deemed unreliable, it was ousted from discussions of criminal risk assessment. However, researchers have attested that even a preliminary look at the clinical diagnosis suggests a conceptual association with criminality; the conceptual link is straightforward considering that callousness, impulsivity, egocentricity, grandiosity, irresponsibility, lack of empathy/ guilt/ remorse, and criminal versatility are core characteristics of the disorder (Hare, 2004; Silver, Mulvey, & Monahan, 1999). Many researchers indicate that psychopathy is the most important clinical diagnosis in the CJS as it is, in itself, a criminogenic personality trait (Hare, 1996, 1998; Wilson & Herrnstein, 1998). This conceptual link has steered researchers to assert that psychopathy is indeed an important clinical diagnosis for policy

and practical criminal justice purposes (Edens, Petrila et al., 2001; Harris, Skilling, et. al., 2001; Webster & Hucker, 2007).

Despite the clear conceptual link between psychopathy and criminality, the majority of research on psychopathy's prediction of recidivism is atheoretical (Douglas, Vincent, & Edens, 2006). However, there is some research that explores how variance in criminality and recidivism is related to the differences between primary and secondary subtypes of psychopathy. It is theorized that the primary psychopaths are more instrumentally aggressive in that they plan for material gain and can achieve their goals through cunning manipulation instead of physical violence, while secondary psychopaths (factor 2) would engage in more reactive, angry, emotionally aroused physical violence, (Cornell et al., 1996; Patrick & Zempolich, 1999; Porter & Woodsworth, 2006; Skeem et al., 2003; Williamson, Hare, Wong, 1987). This theoretical conceptualization of how the different subtypes of psychopathy have separate pathways to criminality (i.e. instrumental vs. reactive) could prove useful for predicting what types of crime a given psychopath would commit (Douglas, Vincent, Edens, 2006). For example, a primary psychopath is more likely to commit instrumental offences like fraud or robbery (Cornell et al., 1996; Molto, Poy, &Tourrubia, 2000), while secondary psychopaths will act more reactively aggressive (e.g. assault and/or murder committed in the heat of passion).

Finally, in regards to a theoretical link between psychopathy and criminality/ recidivism, some researchers take issue with the tautological nature of the factor 2 traits and the prediction of future deviance. It is tautological to use items detailing past deviance to predict future deviance (Cooke et al., 2004); the flawed reasoning can be broken down as follows: the psychopath is a psychopath because they committed deviant acts, and, because the person is a psychopath they will commit deviant acts. Really, all that this conceptualization is able to say is a person with a history of deviance is at risk of committing future deviance. Because of this: "PCL-R recidivism research cannot answer the question whether psychopathy is a causal risk factor or whether its connection to future antisocial acts simply reflects the inclusion of items relating to past criminal behavior" (Douglas, Vincent, & Edens, 2006, p. 544). Cook and colleagues (2004) conceptualize all of the behavioral traits included in the PCL-R as consequent of the actual personality characteristics. However, this position is not without controversy; another family of researchers directly protest this conceptualization by referring to a complex system of statistical

analysis (Neumann, Vitacco, Hare, & Wupperman, 2005). That is, Neumann et al. (2005, p. 625) explain that using structural equation modelling, the Cook et al. (2004) conceptualization of psychopathy results in statistically untenable parameters. Additionally, Neumann et al. (2005) argue that theoretically and empirically, the antisocial component of psychopathy that involves externalized behaviors is not merely consequent of the personality characteristics, but rather independent aspects of personality itself; "personality traits involve purposive direction of behavior including sociability and antisocial tendencies" (p. 626). Cook and Colleagues (2004) believe that a "pure" measure of psychopathy (i.e. a measure that excludes historical behavioral items) would benefit its utility as a risk assessment tool, as it would increase specificity by advancing the understanding of the many pathways to the different forms of deviance (e.g. sexual violence, spousal violence, parasitic non-aggressive white-collar crimes, etc.). Clearly, there is controversy when contrasting these two incompatible conceptualizations of the diagnosis (i.e. Cook et al., 2004 and Neumann et al., 2005).

These debates on the conceptual nature of the diagnosis are important to consider when detailing how the PCL-R has been empirically validated as a violence risk assessment instrument. As is explained in the following discussion, statistically valid results in psychopathy studies can often only be attributed to factor 2 items; PCL-R total scores and factor 2 scores meet cut-off scores for validity of findings, but when factor 1 scores are isolated from factor 2 scores, the cut-off for validity is most often missed. This supports the notion that the tautological nature of the PCL-R is what renders it a valid risk assessment instrument, not the core personality characteristics.

The Empirical Evidence for Psychopathy as a Valid Risk Assessment.

The PCL-R was designed as a diagnostic tool, "[not] to predict criminal behavior or to assess risk for violence" (Hare, 2004, p. 145). However, there is an overwhelming amount of influential evidence that details a positive correlation between criminal behavior and PCL-R scores (Cooke et al., 2012; Gacono, 2015; Hare et al, 1999; Hart, 1998; Hart & Hare, 1997; Hemphill, Hare, & Wong, 1998; Hemphill & Hart, 2003; Millon et al, 2003; Raine & Sanmartin, 2001). Thus, the PCL-R has garnished robust predictive validity for its ability to predict criminal behavior across a variety of correctional populations and contexts, which has influenced CJS practitioners to employ it internationally as a risk management tool (Hare, 2004; Webster &

Hucker, 2007). That is, while other risk assessment instruments are designed to predict a specific type of criminal behavior in a specific offender population, the PCL-R is able to predict recidivism over a wide range of criminal behaviors and offender populations; if the diagnosis can be applied to an offender convicted of any given offence (i.e. high PCL-R scores), then it can be used, and is relevant to the offenders' risk profile. The diagnosis must be relevant to the case at hand (i.e. the offender must be a psychopathic) in order to contribute in predicting recidivism, but this relevance spans across all sorts of criminal behaviors and correctional demographics (e.g. young, old, violent, sexual, fraud, robbery, etc.). As such, a low PCL-R score does not indicate low risk like other risk assessment instruments, rather, it indicates that the psychopathic disorder is inapplicable to the offender being assessed.

Practically, the PCL-R has an enormous impact on offender's lives, "parole boards take into account an offender's PCL-R score in arriving at a release decision. Those with a high PCL-R score are less likely to receive conditional release than are other offenders" (Hare, 2004, p. 147). Authors from the Correctional Services of Canada (CSC) research branch also support Hare`s (2004) assertion, and state that the PCL-R assists decision makers in making custodial decisions due to its good predictive power for general and violent recidivism (Serin, Mailloux, Hucker, 2000). Serin et al. (2000) of the CSC research branch explain that "the Psychopathy Checklist-Revised (PCL-R; Hare, 1991) correctly classifies 77% of violent men offenders (Harris, Rice, & Cormier, 1991) and is equally efficient at predicting violence among individuals with psychiatric disorders, such as schizophrenia (Rice & Harris, 1992)" (p. 1). CSC supports Hare's (2004) assertion on the use of the PCL-R in corrections; the PCL-R is used by CSC to predict risk for recidivism (especially violent recidivism), and based on an offenders' risk profile the Parole Board of Canada may deny day or full parole, or the courts may even subject offenders to indeterminate sentencing, which overrides their statutory right to being released from prison after serving 2/3 of their sentence (Bonta & Motiuk, 2015; Correctional Services Canada, 2015; Public Safety Canada, 2015). Additionally, the PCL-R is often utilized in court hearings to determine if an offender should be assigned a Dangerous Offender designation, which also impacts offender sentencing and release decisions (John Howard Society of Alberta, 2000). The following paragraphs some of the empirical literature that supports the use of the PCL-R as a risk assessment instrument.

One of the most prominent ways psychopathy is tested for predictive validity in determining future criminality is to compare the PCL-R with other risk assessment instruments. With samples sizes ranging from n= 93 to 248, researchers have found that their PCL-R scores significantly correlated with three separate empirically validated risk instruments: the General Statistical Information of Recidivism (GSIR), the Salient Factor Score (SFS), and the Base Expectancy Score (BES) (Glover et al., 2002; Hemphill, 1992; Serin, 1996; Serin, Peters, and Barbaree, 1990). However, it is important to note that while overall PCL-R scores correlated with the GSIR, BES, and SFS in these studies, the correlation with factor 2 items was highest, and the correlations with factor 1 scores were insignificant (remember the tautology issue discussed earlier). Regardless, this evidence (and other supporting studies) is the basis for using the PCL-R as a risk assessment instrument.

It is stated that the Violence Risk Appraisal Guide (VRAG) is one of the most effective and heavily employed risk assessment instruments for violent crime (Correctional Service Canada, 2015; Harris, Rice, & Quinsey, 1993; Quinsey et al., 1998). A PCL-R score is one of the 12 items used in the VRAG. Through a series of separate studies with large sample sizes ranging from n=212-618 male offenders, researchers have supporting evidence for the notion that PCL-R scores are correlated with VRAG scores (.59-.77) (Barbaree, Seto, Langton, & Peakcock, 2001; Loza & Dhaliwal, 1997; Hemphill, Hare, et al., 1998; Glover et al., 2002). However, it should be noted that these scores are obviously inflated considering that the PCL-R is an item in the VRAG. Additionally, when the PCL-R item was removed from the VRAG, Glover and colleagues (2002) found that factor 1 was insignificantly correlated with the VRAG, and factor 2 was highly correlated (more so than the correlation when the PCL-R was included in the VRAG) with the VRAG. This suggests that factor 1 actually reduces the strength of the correlation between the PCL-R and the VRAG. Again, refer to the earlier discussion on tautological reasoning in regards to factor 2 items.

The HCR-20 is another risk assessment instrument utilized in corrections to predict future violence (Webster, 1997). One of the 20 items (H7), includes psychopathy as defined by the PCL-R, as it adds the the HCR-20's predictive validity for future violence (Webster, Douglas, Eaves, & Hart, 1997). Studies have shown that when the H7 item is removed from the HCR-20, the HCR-20 and the PCL-R/ PCL-SV are significantly correlated (Hare, 2004). Again, like the

VRAG, when looking at the strength of correlations between the HCR-20 and the PCL-R, the HCR-20 is more strongly correlated with PCL-R factor 2 items than it is with factor 1 items (Hare, 2004).

The Level of Service Inventory- Revised (LSI-R) is another risk management instrument that utilizes the PCL-R score in its assessment procedures. The LSI-R is used to predict risk of recidivism and determine offender management based on an assessment of offender risks/ needs (Andrews & Bonta, 2016). Many researchers have found a significant positive correlation between the LSI-R and PCL-R (Hemphill, Hare, et al., 1998; Loza & Simourd, 1994; Simourd & Malcom, 1998). However, like the VRAG and HCR, the LSI-R has consistently shown to have a much stronger correlation with PCL-R factor 2 scores than factor 1, which inflates the correlation between PCL-R total scores and LSI-R scores.

There is evidence that the PCL-R is also correlated, albeit to a lesser degree, with many sex offender risk assessment instruments, such as the Sex Offender Risk Appraisal Guide (SORAG), the Rapid Risk Assessment of Sexual Offence Recidivism (RRASOR), the Minnesota Sex Offender Screening Tool-Revised (MnSOST-R), the Multifactorial Assessment of Sex Offender Risk for Recidivism (MASSOR), and the Static 99 (Barbaree, Seto, Langton et al., 2001). Unlike the other studies mentioned above, these analyses did not compare and contrast the differences between factor 1 and 2 correlations. Additionally, Kropp and colleagues (1999) have found a correlation between the Spousal Assault Risk Assessment Guide (SARA) and the PCL-R. In contrast to other correlational evidence on the PCL-R and risk assessment instruments, Kropp and colleagues (1999) found that the SARA was more significantly correlated with PCL-R factor 1 than 2. This suggests that the risk associated with spousal violence recidivism is more related to the core personality features of psychopathy. Thus, this evidence suggests that while the PCL-R is able to assist in predicting violent recidivism primarily through factor 2 items, it is the factor 1/ personality items that are better able to predict spousal violence.

Finally, in regards to the support for the predictive validity of psychopathy as a risk assessment instrument, there are several meta-analyses that provide quantitative evaluations of empirical research on the topic (Dolan & Doyle, 2000; Hemphill, Hare, & Wong, 1998; Salekin, et al., 1996). These meta-analysts consulted a vast array of empirical research on the topic that

accumulates to large sample sizes of thousands of participants. The meta-analyses purport that psychopathy as defined by the PCL-R holds predictive validity for both general and violent recidivism. The meta-analysis conducted by Salekin and colleagues (1996) found a significant correlation between sexual recidivism and PCL-R scores. Hemphill, Hare, and Wong (1998) consulted five studies on general recidivism (N=1021) and four studies on violent recidivism (N=1089), and found that relative to non-psychopaths, psychopaths were three times more likely to recidivate, and four times more likely to violently recidivate. However, consistent with the trend shown in the research consulted above, factor 2 was more strongly correlated with general recidivism than factor one.

In contrast to the meta-analyses discussed thus far, Gendreau and colleagues (2002) challenge the use of the PCL-R as a valid risk assessment instrument. They conduct their own meta-analysis that contradicts the findings of the two meta-analyses discussed above. This analysis both consults different articles, and uses different statistical analyses to produce results. Additionally, they highlight issues with research on the PCL-R that supports its use as a risk assessment instrument. Gendreau and colleagues (2002) not only refute the effectiveness and predictive validity of the PCL-R as a risk assessment instrument through a statistical analysis of empirical research, but also through a discussion of the conceptual and ethical limitations; they recognize that the PCL-R's predictive validity is limited to the Factor 2 items, and argue that it is both tautological and irresponsible for criminal justice practitioners to rely on static factor file data that cannot be targeted for rehabilitation.

Hemphill and Hare (2004) were quick to respond to the Gendreau et al. (2002) article and defend their position. Their defense consisted of an extensively technical document that refuted the Gendreau et al. (2002) article's techniques for statistical analysis (e.g. dichotomizing continuous variables, setting extremely high base rates for recidivism, using unpublished studies as data, using a biased selection of articles as data, consulting data with inappropriate samples, etc.). In their critique Hemphill and Hare (2004) also argue for why their meta-analytic techniques are far superior to those used by Gendreau et al. (2002).

Thus, while there is indeed a wide array of empirical and meta-analytic research that supports the use of psychopathy as a risk assessment instrument, it is not without dissenters.

However, despite prevailing controversy, those who support the utility of the PCL-R in corrections have been able to accumulate enough support to overthrow the dissenters and facilitate partnerships with criminal justice systems internationally. Psychopathy, as defined by the PCL-R is utilized as a risk assessment instrument in corrections internationally.

Hare (2004) openly admits that other risk instruments are superior at predicting risk, but he explains that this is expected, considering how those instruments were built for the sole purpose of risk assessment/ management. He explains,

> it would be odd-even embarrassing- if an instrument whose raison d'être is to assess risk did not perform better than the PCL-R… The PCL-R certainly holds its own with these instruments, but its robustness as a risk factor lies in its ability to predict recidivism and violence across diverse groups, including male and female offenders, sex offenders, forensic psychiatric patients, civil psychiatric patients, and spousal assaulters" (Hare, 2004, p. 147).

Hare (2004) explains that other risk assessment instruments are minimal in that they depend on the particular context, population, and type of criminal activity they were designed for, while the PCL-R was made to measure a personality disorder, and so its importance in risk assessment is dependent on whether or not psychopathy is relevant to the behavior being predicted. Finally, Hare (2004) advises that the strength of the PCL-R lies within its ability to measure a personality disorder, which is why it is able to measure a wide variety of criminal behaviors across a variety of contexts and correctional populations. Hare has developed the PCL- Screening Version (SV) and the PCL- Youth Version (YV) for other criminal and non-forensic community populations as an attempt to broaden the scope of the diagnosis. Since the PCL-R, PCL-SV, and PCL-YV are applicable to such variable conditions, they are particularly useful in predicting future criminality in youth, and in areas where crime is sparse or covert, such as white-collar crime, law enforcement, military, and politics (Babiak, 1995, 1996, 2000; Babiak & Hare, 2006; Babiak, Neumann, & Hare, 2010).

At this point in time the construct validity of psychopathy has only been validated on forensic patients: clients with recorded criminal or delinquent histories (Horley, 2014; Walters, 2004; Webster & Hucker, 2007). Thus, its ability to identify psychopaths is limited to

populations that have been in contact with the criminal justice system (CJS), where diagnosticians can access necessary historical client information. This limitation has fostered criticism of the PCL-R, and has lead researchers to call for a diagnostic instrument that focuses on personality characteristics and is uncontaminated by the circular reasoning associated with behavioral items (Farrington, 2006). Hare has developed the PCL-R- Screening Version (SV) and the PCL-R- Youth Version (YV) for non-forensic community populations, but these instruments are less accurate, not as empirically validated as the PCL-R, and still focus on behavioral characteristics (Babiak & Hare, 2006; Hare & Neumann, 2008). In attempting to diagnose the disorder without relying on historical data on behaviour, researchers dive deeper into understanding the disorder's etiology. There is a breadth of research on the etiology of the disorder, which inspires research on new diagnostic methods and treatments. This is the topic of the following section of this chapter.

Psychopathy as a Clinical Instrument

Although the PCL-R has been regarded as the best, if not only instrument for diagnosing psychopathy (Edens et. al., 2001; Meloy, 2000; Seagrave & Grisso, 2002), the clinical disorder itself has not garnished as much consensus. There is indeed ongoing exploration and debate on what defines the clinical disorder, and what etiological mechanisms cause the disorder. Future findings will likely have diagnostic implications. The following subsections of this chapter provide a brief summary of the etiological literature on psychopathy as a means of better understanding what the clinical disorder entails beyond its diagnostic definition. This section begins with a discussion on the differences between psychopathy and sociopathy; then introduces topics surrounding the duel-deficit model of psychopathy (i.e. fearlessness and impulsivity); the neuroscientific assertions that attribute the disorder to neurological dysfunction; and finally, current and prospective treatments for the disorder. It should be noted that while this section reviews some of the key components of psychopathy as a clinical personality disorder, a holistic review of the literature is beyond the scope of this thesis. A major focus of this section is on neuroscientific assertions related to psychopathy as a personality disorder, but the etiological considerations could be taken another step back; there is literature surrounding the possible genetic, environmental, and developmental causes of the psychopaths' neurological

abnormalities (Waldman & Rhee, 2006). However, this thesis is focused on the role of neuroscience in defining psychopathy, and so that is the focus of this section.

Socialization practices and a decline in adequate parenting: Differentiating the Psychopath from the Sociopath.

Davit T. Lykken's (1995, and 2006) work on psychopathy is essential literature for understanding the foundations of psychopathy as a clinical instrument. Lykken (1995) suggests that deviance is the behavioral manifestation of psychological disorder, and he identifies different etiological pathways that produce heterogeneous disorders. Lykken (1995) presents a theoretical depiction of psychopathy by differentiating between subtypes of antisocial and deviant personalities. His theoretical depiction of the antisocial personality acts as a foundation from which further research has attempted to support and build from. He ascertains that antisocial personalities should be treated as a family of diverse disorders that differ in both symptomology and etiology. Lykken (1995) proposes that antisocial personalities fall under two main genera, the psychopath and the sociopath. First, he considers the true psychopaths to be those who are inherently antisocial due to genetic peculiarities. According to Lykken (2006), these genetic peculiarities are rare, and therefore psychopaths are of less concern in comparison to their sociopathic counterparts in regards to their impact on society. He believes that the sociopath is an average person in terms of genetics, but has become antisocial due to inadequate socialization. Lykken (2006) suggests that while the psychopaths' prevalence will remain constant over time due to the nature of genetic variability, the prevalence of sociopaths is increasing at an alarming rate.

Socialization is the key ingredient to Lykken's (1995, 2006) theory on antisocial personality, and is used to explain why the incidence rates of sociopathy is on the rise. Lykken (2006) explains that we do not have innate, well-formed prosocial inclinations, but rather, like an inborn capacity for language, it must be elicited, shaped, and reinforced through socialization practices. He explains further that if we are not socialized as children, our ability may all together wither and fail to develop, thereby producing sociopaths.

While Lykken (1995, 2006) emphasizes the effects of socialization practices on personality, he also recognizes that there are three distinct genotypes that can be attributed to the development of antisocial personalities. He explains how the socialization differently impacts each genotype. At one end of the genotype spectrum, the *easily socialized* genotype has a temperamental disposition that will likely render the individual as prosocial/ socialized even with incompetent parenting. The *average genotype* has a temperamental disposition that is dependent on parental competence for socialization; with competent parenting the individual will become socialized, but with incompetent parenting the individual is likely to become a sociopath. Now, the *hard to socialize* genotype is where Lykken (1995, 2006) recognizes the psychopath, which he describes as a heterogeneous population. Lykken (1995, 2006) asserts that there are different species of psychopaths with different etiology that lead to similar personalities; he describes the overly impulsive species, whose strong impulses override any forces of restraint (e.g. fear or guilt), and a species with an inherent inability to develop conscience or empathy at all, and thereby unable to feel guilt all together.

Lykken then explains how these species (i.e. hard to socialize genotypes) are likely to become full psychopaths unless the parental competence is unusually high. If the parental competence is high for the hard to socialize genotype, depending on the species of psychopath, a variety of outcomes are possible. For the impulsive species, unusually high parental competence could produce individuals with a "talent" for psychopathy who become heroic. For the species without conscience or empathy, the disorder is more likely to produce highly successful, noncriminal people with psychopathic traits. Lykken (2006) describes this species in accordance with Hare's (1993) and Babiak's and Hare's (2006) successful psychopath[2]; the socialization process produces a psychopath that "can avoid petty crime and misdemeanors (or at least avoid getting caught) while boldly cultivating his innate charm and other talents to win success and status in legitimate society" (Lykken, 2006, p. 11).

[2] It is important to note that Hare does not differentiate between species of psychopath in the same way that Lykken does. Hare sees psychopaths as a homogeneous population that all share inherent neurological abnormalities.

The psychopath that is reared by incompetent parents that fail to adequately socialize their child is a double liability, and will likely account for the most criminal, wicked, and deranged subtype of the species. Finally, Lykken relates to Cleckley (1941, 1955, and 1982) and explains how the psychopaths he describes are not how the media describes them as implacably evil. Their antisocialism is "not deeply vicious, [they] carry disaster lightly in each hand" (1955, p. 33). This means that the psychopath is not implacably evil in and of itself, what creates the wicked individuals depicted in the media is a combination of traits and circumstances (Lykken, 2006).

The duel-deficit model of the psychopathic personality.

This model borrows from and advances the early claims made above by Lykken in regards to the heterogeneous nature of psychopathy. Many researchers have studied how fearlessness and impulsivity act as etiological mechanisms for psychopathy (Fowles & Dindo, 2006). This research has led to a growing consensus that psychopathy is indeed a heterogeneous disorder that can be differentiated into subtypes (Hicks et al., 2004). This differentiation has been largely focused on creating subgroups according to the PCL-R factor structure. That is, psychopaths can be differentiated according to what group of traits are more applicable to them, factor 1 (facets 1 and 2) or 2 (facets 3 and 4). The duel-deficit model of psychopathy proposes that there are two distinct etiological pathways to developing the disorder, which in turn determine what subtype of psychopathy will develop in an individual. The psychopath with a deficit in fear conditioning will associate more strongly with a factor 1 trait disposition, while the psychopath that is overly impulsive will associate more strongly with the factor 2 trait disposition (Patrick, 2004). Although the duel-deficit model borrows from Lykken's work, there are indeed some differences. In Lykken's species the impulsive/ fearless traits were used to describe one species and a lack of consciousness/ empathy was used to describe another. The duel-deficit model asserts that impulsivity is the etiological mechanism for one species, while fearlessness is an etiological mechanism for another.

Fearlessness. Lykken (1957) set the foundation for the duel-deficit model of psychopathy by conducting a study that measured fearlessness in psychopaths. He recruited psychopaths from a correctional facility and separated them into two groups based on their core

traits; “primary psychopaths” were delineated by grouping participants that most closely resembled Cleckley’s prototype, and “neurotic psychopaths” (secondary psychopaths) were delineated by grouping those who displayed psychopathic tendencies but did not resemble Cleckley’s prototype. Lykken (1957) then subjected the groups to a battery of tasks which were used as a means of testing levels of fearlessness. The results supported the assertion that primary psychopaths have a greater propensity for fearlessness than do secondary psychopaths. This was the beginning of differentiating between subtypes of psychopathy, and defining the disorder as a product of either fearlessness or impulsivity.

Lykken’s (1957) results were subsequently replicated over the years by numerous researchers who studied fearlessness and psychopathy through tests that employed a variety of techniques, such as go/no-go tasks, response-contingent punishment/ aversive conditioning, card-playing tasks, electro dermal and cardiac response measurement, and Multidimensional Personality Questionnaire (MPQ) scoring (Fowles, 1993; Fowles and Missel, 1994; Newman & Kosson, 1986; Newman Patterson & Kosson, 1978; Newman, Widom, & Nathan, 1985; Scerbo et al., 1990; Schachter &Latante, 1964; Schmauk, 1970; Siddle & Trasler, 1981; Siegel, 1978; Zahn, Schooler, & Murphy, 1986). Furthermore, as this line of inquiry gained momentum, a desire to trace back the etiological mechanisms of psychopathy even further was established; researchers applied the Behavioral Inhibition System (BIS) (Gray, 1970; Pickering & Gray, 1999) to explain the etiology of fearlessness, and in turn, psychopathy (Fowles & Dindo, 2006).

The BIS proposed by Gray (1970) was originally derived from theories on animal learning and motivation, but was later supplemented and solidified with neurobiological findings (Fowles & Dindo, 2006). The BIS is a theory that is in accordance with classical and operant conditioning. Behaviors are activated (Behavioral Activation System- BAS) or inhibited (Behavioral Inhibition System- BIS) in response to stimuli; in theory, a person will learn to inhibit behaviors that have either adverse consequences (punishment) or a failure to produce favored consequences (reward). Thus, a person with a weak BIS would be more inclined to engage in conflict situations (fail to avoid aversive stimuli), and slow to stop responding when favored consequences are absent (extinction). A neurobiological substrate for the BIS was identified by McNaughton and Gray (2000). They suggest that the septohippocampal system is responsible for the BIS, and in turn, fear conditioning. The psychopath is then the product of the

following chain of events: abnormal/ dysfunctional septohippocampal system causes a weak BIS, which causes fearlessness, which produces behaviors unconstrained by the possibility of negative consequences (Fowles & Dindo, 2006).

Defining psychopathy as caused by a dysfunctional septohippocampal system did not last long, as conflicting research was quick to respond to this assertion. Researchers interested in identifying the neural network associated with classical aversive fear conditioning have found that the amygdala is responsible for initiating the behavioral and physiological expression of fear (Davis, Walker, & Lee, 1997; Lang et. al., 2000; Ledoux, 1995, 2000, 2003). After being confronted by such a large body of conflicting research, McNaughton and Gray (2000) modified their BIS theory to include the amygdala as an active cortical structure in the BIS theory of fear conditioning. Leading researchers today still uphold that the amygdala is the most prominent cortical structure involved in causing psychopathy, along with other prominent structures within the paralimbic system (Birbaumer, Veit, Lotze, Erb, Grodd, Flor, 2005; Blair, 2003; Glenn and Raine, 2014; Hare, 2004; Jones, Lauren, Herba, Barker, Viding, 2009; Kiehl, Smith, Hare, Mendrek, Forester, Brink, & Liddele, 2001; Vollm, Taylor, Richardson, Corcoran, Stirling, Mckie, Elliott, 2006), which solidifies the theoretical notion that psychopathy is a product of fearlessness.

However, although Lykken (1995) believes that the core features (factor 1 traits) of psychopathy reflect low fear, there is currently no literature on the theoretical link between fearlessness and factor 1 traits (Fowles and Dindo, 2006). The assertions that disruptions in paralimbic system (i.e. the amygdala, the hippocampus, and other surrounding/ related structures) are responsible for fearlessness and the core features of psychopathy (i.e. factor 1 items) have been derived separately and are unable to explain how or even if the factor 1 traits are direct manifestations of fearlessness. Fowles and Dindo (2006) suggest that the dual-deficit model may never be able to explain the manifestations of factor 1 traits, and perhaps looking for a conceptualization of the disorder that includes other (e.g. temperamental) deficits would prove fruitful as a third etiological factor that could bridge the gap between fearlessness and the core features.

Impulsivity. The behavioral/ antisocial dimension of the disorder entails the PCL-R factor 2 items and reflects observable behaviors, while the interpersonal/ affective dimension entails PCL-R factor 1 items and reflects interpersonal style and affective deficits. The impulsivity deficit[3] as an etiological mechanism for psychopathy is best applied to the factor 2 (or as some researchers call *secondary)* traits of the disorder. That is, impulsivity/ disinhibition is more strongly associated with the behavioral/ antisocial dimension of the disorder, not the interpersonal/ affective dimension (Patrick & Lang, 1999; Patrick 2004; Fowles and Dindo, 2006). As such, this deficit is able to extend to a variety of behavioral/ externalizing disorders (e.g. ADHD, alcoholism, sociopathy/ APD/ conduct disorder).

While there is indeed debate as to whether or not psychopathy should be differentiated into subtypes based on associations with factor 1 or 2 traits, the literature has been fairly consistent in regards to factor 2 traits being a manifestation of an impulsivity deficit. This impulsivity deficit is most notably attributed to damage/ dysfunction in the orbitofrontal cortex of the brain; both human and animal patients with a damaged/ dysfunctional orbitofrontal cortex display behavioral characteristics that are akin to those of factor 2 psychopathy traits (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Blair, 2001; Blumer & Benson, 1975; Damasio, Grabowski, Frank, Galaburda, Damasio, 1994; Elliott, 1978; Hare, 1984; Hornak, Branham, Rolls, Morris, O'Doherty, Bullock, Polkey, 2003; Kiehl, 2006; Mitchell, Colledge, Leonard, Blair, 2002).

Psychopathy as a Neurological Manifestation: Key Findings

First, it is necessary to understand the two ways neuroscience is able to provide information on the psychopathic personality disorder. One way to gain information from the neurosciences is to compare the behaviors and personality characteristics of patients with damage to specific brain regions to those who display psychopathic traits. When there are similarities between those with psychopathy and those with localized brain damage, neuroscientists infer that the psychopath also has damage/ dysfunction in the same region of the

[3] Often referred to in the literature as an "impulsivity deficit", this phrasing may be a source of confusion. The Psychopaths do not lack the ability to be impulsive, rather it is the opposite, they are said to be overly impulsive due to their cognitive/ emotional deficits.

brain. The second way to gain information from neurosciences is to administer psychological tests to individuals with psychopathic traits. Performance on these tests can be measured in two ways. The tests are designed to reflect a specific brain structure/ region/ system, and so measurement of performance levels on the test would indicate how the area of the brain is functioning. Neuroscientists can either (1) develop these tests to measure the participants' performance itself, and/or, because of modern neuroimaging techniques (e.g. MRI, fMRI, CT, etc.), (2) the actual neurological response to the test can be recorded and analyzed while participants perform the tasks. This second way of measuring the psychological testing has revolutionized the neurosciences, because even if the control and experimental groups don't differ on task performance, a difference can be found in how the participants process the tasks in the brain. It is important to mention, especially in the context of this thesis, that the research on the neuroscience of psychopathy is rife with differing, conflicting and contrasting findings. As such, we are presenting a range of possibilities that have been supported, not confirmed, by exploratory research studies.

The Amygdala.

With regards to research that compares task performance of patients with amygdala damage to individuals with psychopathic traits, there are similarities between subject groups. Brain damaged patients and psychopathic individuals were both worse than the control groups at recognizing fearful facial expressions (Adolphs et, al., 1999; Blair, 2001). Additionally, psychopaths and amygdala damaged patients were worse than controls at recognizing and differentiating fearful, angry, or surprised vocalizations (Blair, Budhani, et. al., 2005; Dellacherie et al., 2011). When compared to a control group, Newman and Kosson (1986) found that psychopaths performed poorer on tasks intended to measure their ability to learn to approach stimulus associated with reward and avoid those associated with punishment.

The neuropsychological testing discussed above was designed to measure the functioning of psychopaths' amygdala during related tasks, and their performance indicated amygdala dysfunction. There are also a number of studies that actually measured amygdala activity during psychological testing using neuroimaging techniques. Yang, Raine, and Colletti (2009) found that psychopaths have reduced amygdala volume in comparison to control subjects. Compared to

a control group, a reduction in amygdala activity was found in psychopaths during emotional processing tasks (Kiehl, et. al., 2001; Gordon, Baird, & End, 2004), aversive fear conditioning tasks (Birbaumer, et. al., 2005), a social cooperation game (Rilling, et. al., 2007), moral decision making tasks (Glenn, Raine, & Schug, 2009), while viewing fearful faces (Jones, et. al., 2009), and during an affective-memory task (Kiehl, et al., 2001).

There have been many assertions made that are based on the findings of the studies noted above (and other similar studies that support their assertions), which include: psychopaths are unable to learn that some behaviors are bad, and will thereby be more inclined to use antisocial strategies to achieve their goals (Blair, 2008); they are unable to associate their actions to the pain of others, which results in a lack of empathy for their victims (Blair 2006); and the amygdala is responsible for producing and experiencing emotional states (Phillips et al., 2003; Moll et al., 2002), judgements during moral dilemmas (Berthoz et al., 2006; Glenn, Raine, & Schug, 2009), and enhancing attention to emotional stimuli (Glenn & Raine, 2014). Thus, brain imaging and neuropsychological testing have supported the hypothesis that the amygdala could be a key structure that can account for a number of psychopathic features.

The Hippocampus.

Through studies that use MRI and fMRI neuroimaging techniques while participants perform tasks that are made to reflect specific cognitive functions, leading researchers suggest that the hippocampus could be responsible for the retrieval of emotional memories and fear conditioning (Glenn & Raine, 2014). M.S. Faneslow (2000) produced the study from which this assertion is derived, which includes the use of laboratory rats; the rats are subjected to hippocampal lesions and made to complete tasks that simulate classical fear-conditioning. Additionally, neuroimaging studies on 18 alcoholic subjects with antisocial tendencies found that they have reduced volume in the posterior section of their hippocampus in comparison to pro-social controls (Laakso et al., 2001). This study on a sample of antisocial alcoholics was used to substantiate the assertion that psychopaths[4] have morphometric differences in the hippocampus in comparison to controls (Glenn & Raine, 2014; Laakso et al., 2001). Boccardi and colleagues (2010) used MRI images and compared 26 habitually violent offenders who scored high on the

[4] The authors generalized the antisocial alcoholic as being comparable to the psychopath.

PCL-R with 25 controls and found that specific neurons in the hippocampus had either abnormal enlargements or depressions in the psychopath group. The regions that Boccardi et al. (2010) found abnormalities in a said to be associated with autonomic responding, the processing of emotional input, and contextual fear conditioning. Also, the most cited fMRI study on psychopathy and the limbic system (i.e. the empirical material of this thesis) found that during an affective memory/ emotional processing task, a sample of psychopaths showed reduced hippocampal functioning (Kiehl et al., 2001).

Based on this type of supporting evidence, researchers have come to the understanding that psychopaths could have abnormal hippocampal formation and functioning, which could cause affective abnormalities (Glenn & Raine, 2014). These assertions align with the dual-deficit model discussed above, because it is theorized that the deficiency in emotional processing and fear conditioning disrupts learning in social contexts and reduces the psychopaths' ability to process cues of future punishment (Glenn & Raine, 2014). That is, fearlessness is possibly a product of an inability to be classically conditioned to fear. Researchers have also suggested that there is a possible difference between successful (uncaught) and unsuccessful psychopaths in regards to hippocampal abnormality, which suggests that successful psychopaths may be more sensitive to environmental cues that predict capture (Raine et al., 2004).

The Prefrontal Cortex (PFC)[5].

While the previously discussed amygdala and hippocampus are both subcortical brain structures, the PFC is a part of the cortex and is said to be responsible for higher order executive functioning. Researchers believe that the PFC is responsible for the ability to shift behaviors when rewards change, moral judgement, processing of emotional and social stimuli, self-reflection, guilt and embarrassment, cognitive appraisal of emotions, and emotion regulation (Glenn & Raine, 2014). These functions do indeed overlap with the proposed functions of the amygdala and hippocampus, as all three structures are said to be involved in the processing of emotional information. However, the PFC is considered to be more associated with the actual cognitive appraisals of the emotional information and the corresponding decision making/

[5] The PFC contains many cortical regions (e.g. orbitofrontal cortex, ventromedial PFC, Dorsolateral PFC, etc.) that all have unique functions. However, these regions also share many functions, and so for the purpose of this thesis they will be conflated and discussed as a single entity, the PFC.

behavioral response. The amygdala and hippocampus are limbic structures that are believed to respond more instinctively to emotional information, thereby creating a neurochemical reaction that the PFC would respond to cognitively, which is manifested behaviorally. Despite the understanding that PFC and limbic structures are interrelated, researchers are confronted with determining the source of psychopaths' neural dysfunction; the PFC dysfunction could be attributed to abnormal inputs from the amygdala and/or hippocampus, alternatively, the PFC may be the only source of dysfunction, or the PFC could cause amygdala and hippocampal dysfunction by providing abnormal feedback input (Finger et al., 2008; Glenn & Raine, 2014). Different studies on how the functions of the PFC were identified by neuroscientists, and how the PFC is said to be related to psychopathy are discussed below.

Neuropsychological testing and neuroimaging techniques were used to explore if the PFC is involved with operant conditioning. First, neuropsychological tests were used to assess whether or not subjects had the ability to shift/ extinguish responses when a previously rewarded behavior no longer provided a reward (i.e. reversal learning abilities) (Fellows & Farah, 2005). Researchers assert that subjects with damage to the PFC were unable to perform the task as well as healthy controls (Fellows & Farah, 2005), nor were those with psychopathic tendencies (Blair, 2006; Blair, Colledge, & Mitchell 2001; Mitchell et al., 2002). Similar findings on the impairment of psychopaths in reversal learning have been made with other neuropsychological tasks (Budhani & Blair, 2005; Glenn and Raine, 2014). Also, other lesion studies with both human and animal subjects have supported the notion that the PFC is responsible for reverse learning (Rolls et al., 1994; Dias, Robbins, & Roberts 1996). Therefore, there are studies that support the assertion that psychopaths are unable to shift behaviors when rewards change (reversal learning), because they have dysfunctional PFCs.

Researchers theorize that this reversal learning/ response preservation deficiency in psychopaths is related to the lifestyle-antisocial Factor 2 features of psychopathy (Mahmut, Homewood, & Stevenson 2008, Mitchell et al, 2002). This reversal learning assertion aligns well with the dual-deficit model of psychopathy, as the inability to learn to switch unrewarded behaviors is linked to the impulsivity deficit (i.e. the psychopath will impulsively maintain the previously rewarded behavior), which is also linked to secondary/ factor 2 subtype of psychopathy.

The PFC is also said to be a primary cortical structure involved in making moral judgements. Research has supported this assertion through the use of neuropsychological tests where subjects are asked to make decisions when presented with hypothetical moral dilemmas (Greene et al., 2001), or morally transgressive pictures (Harenski & Hamann, 2006). PFC damaged subjects would consistently make decisions that erred toward emotionless utilitarianism (e.g. kill one man to save many) in comparison to the control group (Ciaramelli et al., 2007; Koenigs et al., 2007; Mendez, Anderson, & Shapira, 2005). Subjects with psychopathic traits would respond similarly to the brain damaged subjects (Bartels & Pizarro, 2011), and showed less PFC activity than a control group on an fMRI (Harenski et al., 2010). This supports the assertion that psychopaths may have dysfunctional PFCs and thereby deficits in moral reasoning and emotional input processing. Additionally, in conjunction with the reversal learning deficit, the deficit in moral judgement in psychopaths is related to the factor 2 features of the disorder; impairment in judgement processes leads to impulsivity, risk taking and antisocial behavior (Glenn & Raine 2014), which are all factor 2 features of psychopathy. Many researchers even claim that secondary psychopathy (i.e. impulsive and reactively aggressive subtype) is more strongly related to PFC abnormality, while primary psychopathy (i.e. fearless, instrumentally aggressive subtype) is related to subcortical limbic system abnormalities (Davidson et al., 2000; Raine, Meloy et al., 1998; Scarpa & Raine, 2000). However, these assertions are merely speculative, and would benefit from further research that would delineate empirical subtypes of psychopathy (Raine & Yang, 2006).

There is also evidence that PFC dysfunction is related to factor 1 affective-interpersonal traits as well. With regards to emotion processing/ appraisal/ regulation, functional neuroimaging studies have suggested that psychopaths show reduced PFC neural activity during cognitive and emotional tasks; the items on the interpersonal facet (factor 1) of the PCL-R have a negative correlation with PFC perfusion (i.e. blood flow); reduced PFC activity during fear conditioning (recall that fearlessness is associated with primary psychopathy according to the duel-deficit model); and reduced activity during socially interactive games (Glenn & Raine, 2014). There is also a large amount of fMRI studies that have shown how samples of psychopaths have displayed reduced PFC activation when exposed to emotion arousing pictures (Gordon, Baird, & End, 2004; Muller et al., 2003). Thus, there is evidence which suggests that

an abnormal/ dysfunctional PFC is related to many of the psychopathic traits from both factors on the PCL-R.

Interconnectivity and the Multiplicity of Brain Regions in any Given Function.

The focus of this section has discussed how specific cortical structures are responsible for specific cognitive and emotional processes. However, it must be noted that in reality the cognitive/ emotional processes can be attributed to the function of multiple brain regions (Glenn & Raine, 2014). This review of etiological findings (i.e. brain dysfunction that causes psychopathy) has limited its focus to the amygdala, the hippocampus, and the PFC, because these are the most prominently dysfunctional and studied structures in regards to psychopathic traits. However, there is evidence that suggests many more brain structures and pathways could be involved in producing psychopathic traits, including the angular/ superior temporal gyrus (Kiehl et al, 2004; Muller et al, 2008; Oliverira-Souza et al, 2008), the anterior temporal cortex (Harenski et al., 2010; Oliverira-Souza et al, 2008), the anterior and posterior cingulate (Birbaumer et al, 2005; Glen, Yang, et al, 2010; Kiehl, et al, 2001; Muller et al, 2008; Rilling et al, 2007; Yang, Raine, Colletti, et al, 2009), the ventral striatum (Glenn, Raine, et al, 2010; Glenn & Yang 2012; Kiehl et al, 2001), the parahippocampal gyrus (Kiehl et al, 2001), the insula (Birbaumer, et al., 2005), and the corpus callosum (Raine et al, 2003; Raine & Yang, 2006). There is clearly evidence that suggests dysfunction in many regions of the psychopathic brain. However, it is difficult for researchers to pinpoint if certain structures are dysfunctional in and of themselves, or as a result of dysfunctional structures that are connected to it (i.e. dysfunctional amygdala and PFC could cause disruptions in other areas of the brain) (Glenn & Raine, 2014).

Also in regards to interconnectivity, some researchers have proposed that the dysfunction actually lies within the connecting tracts between cortical structures. For example, it is hypothesized that if there was a communication failure between an otherwise functional amygdala and PFC, then the PFC would be operating without receiving emotion-related information from the amygdala, and therefore result in the callousness, lack of empathy, poor inhibitions, risk taking, and instrumental aggression displayed by psychopaths (Glenn & Raine, 2014). Researchers have found evidence to support this "connectivity dysfunction" hypothesis through neuroimaging studies that use either experimental-control group comparison (Craig et

al., 2009), or picture viewing tasks (Marsh et al., 2008). In sum, there are multiple brain regions and pathways that could be involved in producing psychopathic traits, and researchers are still in the early stages of research where controversy and debate are commonplace.

Treatments: Current and Prospective.

The hard and fast truth about empirically supported treatments for psychopathy is that there is no effective treatment of the disorder to date (Hare, 2004; Harris & Rice, 2006; Glenn & Raine, 2014). Most researchers are pessimistic about using traditional treatment methods[6] with psychopathic offenders (Andrews, 2000; Dolan & Coid, 1993; Hare, 1999; Kiehl, Hare, McDonald, & Brink, 1999; Losel, 1998; Quinsey et al, 1998; Suedfeld & Landon 1978; Webster & Hucker, 2007; Wong & Hare, 2001; Young et al, 2000), because "unlike most offenders, psychopaths suffer little personal distress, see little wrong with their attitudes and behavior, and seek (and remain in) treatment only when it is in their best interests to do so, such as when applying for probation or parole" (Hare, 2004, p. 158). For this reason, it is believed that correctional treatments only exacerbate their destructive paths, because they learn the ins and outs of correctional risk management. Some researchers have found that offenders who scored high on psychopathy and did better than others in treatment were actually the most likely to reoffend (Rice, Harris, & Cormier, 1992; Seto & Barbaree, 1999). It is hypothesized that traditional treatments are unable to instill remorse, guilt, empathy, responsibility etc., because there are biological manifestations that render psychopaths physically incapable (Seto & Quinsey, 2006). However, it should be noted that these assertions on the effectiveness of treatments are not without controversy, as some researchers have found conflicting evidence that suggests psychopathy does not impact the effects of traditional treatment methods (Salekin, 2002; Salekin, Worley, & Grimes, 2010; Skeem, Monahan, & Mulvey, 2002).

With regards to untraditional treatment methods, there is some preliminary research that has fostered confidence in finding either effective preventative measures or treatments for psychopathy. There have been a variety of suggestions on how to prevent psychopathy,

[6] Traditional treatment methods include correctional programming that attempts to instil empathy, consciousness, and interpersonal skills into offenders (Hare, 2004).

including increasing levels of nutrition[7] during the prenatal period (Glenn & Raine, 2014); avoidance of post-partum depression because it causes the mother to be less warm and responsive to their babies, thereby increasing the babies stress hormones (i.e. cortisol) (Halligan et al., 2004); and increasing parental sensitivity, competence, and bonding through training programs (Liu, 2011; Lykken, 1995; McDonald et al., 2011). These preventative measures ascribe to the theory that psychopathy is developmental in nature, and can thereby be avoided given the right environmental circumstances.

There has also been preliminary research on identifying biological targets for the treatment of psychopathy. There is evidence that cortisol, a stress hormone, is a promising target for intervention. In samples of youth who exhibit both disrupted cortisol activity and aggressive or antisocial behaviors, psychosocial interventions have been shown to reduce their aggression and restore the cortisol disruption (Brotman et al., 2007; Fisher et al., 2007; O'Neal et al., 2010). These studies have inspired researchers to investigate the effects of cortisol on the expression of psychopathic traits for the purpose of developing a treatment (Glenn & Raine, 2014).

Oxytocin is another hormone that has been targeted for treating psychopathy. M. M. Lim and L. J. Young (2006) observed that oxytocin was involved in forming social bonds, recognizing social stimuli, and facilitating social affiliation/ attachment in samples of voles, sheep, and mice. They suggest that oxytocin is a potential therapeutic target for psychiatric disorders associated with disruptions in social behavior. Additionally, Beitchman and colleagues (2012) found in a sample of 162 youths a "significant association between callous-unemotional traits in children and adolescents with extreme, persistent pervasive aggression and a polymorphism on the oxytocin receptor" (p. 125). Dadds and Rhodes (2008) speculate that oxytocin manipulation could be used as a therapy to alleviate deficits in communication and emotion perception in youth with callous-unemotional traits[8]. Considering that psychopaths suffer from all of the affective deficits noted above, researchers are hopeful that more research

[7] Protein, zinc, iron, vitamin B, and Omega-3 fatty acids have all been identified as nutrients that impact violence, aggression, emotional responsivity, other antisocial behaviours, and brain development (Liu et al, 2004; Golub, Hogrefe, & Germann, 2007; Hibbeln, 2001; Kitajka et al., 2004; Kohlboeck et, al., 2011; Rosen et al., 1985)

[8] Note that Dadds and Rhodes (2008) research is speculative, and is geared toward a synergy, where traditional psychosocial interventions are used in conjunction with biological intervention.

on the effects of oxytocin could lead to a future treatment for psychopathy (Glenn & Raine, 2014).

Finally, research on the use of repetitive transcranial magnetic stimulation (rTMS) for treating depression (Peng at al., 2012), has interested those who are trying to find a treatment for psychopathy. S. O. Tassy and colleagues (2011) conducted a study where 24 men were grouped in either an experimental or control group. The experimental group received rTMS to their prefrontal cortex (PFC), which caused a disruption in cortical activity. The group with a disrupted prefrontal cortex displayed more utilitarian responses to moral dilemma questions in comparison to the control group. This study not only sustains the assertion that the PFC is involved in moral decision making, but suggests that rTMS technology has the potential to alter psychopathic behavior through direct manipulation of the brain. Additionally, rTMS stimulation to the PFC can actually improve functioning of the orbitofrontal cortex (Knoch et al., 2006). Considering that psychopaths have a dysfunctional orbitofrontal cortex, some researchers believe that this kind of cranial manipulation could act as a treatment for psychopathy (Glenn & Raine, 2014).

In terms of developing a treatment for psychopathy, the current trend has moved away from a purely psychosocial intervention and toward finding a treatment that targets biological factors through the use of neuroscience (Harris & Rice, 2006). In sum, "major progress in the near future is likely to be dominated by advances in neuroscience associated with better neuroimaging technologies, [and] a better understanding of how neurotransmitters work" (Seto & Quinsey, 2006, p. 598). Although neuroscientists have expressed optimism in finding a neurological treatment, and have created a trend that defines the disorder in terms of biology, other researchers have expressed concern with such a reductionist model. For example, neurological causation begets a likely trajectory of pre-emptive 'screening and intervening' (e.g. preventative detention) on risky brains in adults and delinquent youth (Rose, 2010; Salekin, Rosenbaum, Lee, & Lester, 2009; Sevecke, 2011; Slough, & McMahon, 2008); increased psychotropic and genetic interventions (Leifer, 2000; Rose, 2000; Rose, 2010); and ultimately, a criminal justice system (CJS) that reduces violent criminality to individual neurobiological factors, and fails to endorse policy that targets broader systemic societal factors. However, while developing an effective treatment lingers in its exploratory stages, psychopaths are currently

being treated by a correctional system, which is limited to managing the risk of their antisocial behaviors primarily through incapacitation.

Psychopathy as a Scientific Construct

It is important to note that the research on psychopathy is still in the exploratory stages, and so contrasting/ conflicting evidence and theoretical models are common place in the literature. Despite decades of research, the construct is not as solidified or universally accepted as other scientific facts. For example, consider the double helix structure of the DNA molecule, this is a concretized fact that is black boxed and no longer the subject of controversy. With psychopathy, the only aspect of the construct that is close to concretization and undisputed acceptance is the PCL-R as definition and diagnostic instrument. However, it seems as though there has been a push from neuroscientists to develop a more comprehensive diagnostic instrument that differentiates between subtypes of psychopathy (Patrick, 2006). This could explain the most recent advancement in the PCL-R that have split the items into two factors and then again into four correlated facets (Hare, 2004; Hare and Neumann, 2005, 2006); to provide neuroscientists with the ability to determine how their findings relate to specific clusters of psychopathic traits[9]. Further diagnostic advancements of this kind will likely help clarify the discrepancies within the literature, and render neurobiological research on psychopathy more sophisticated, because researchers are currently limited to targeting specific psychopathic traits in subjects who rarely meet the criteria for psychopathy (e.g. delinquent youth, APD patients, violent criminals, variable PCL-R cut off scores, etc.) (Hare, 2004; Patrick, Venables, & Skeem, 2012).

Indeed, we can see how the involvement of neuroscience is influencing the career of psychopathy as a scientific construct. This literature review has shown how the PCL-R has undergone revisions; research on a superordinate factor and correlations between items is coupled with and compared to neuroscientific findings; samples of patients with behavioral and

[9] Hare may accept that different aspects of psychopathy can be attributed to underlying deficits in impulsivity fearlessness, or temperament, and that different brain structures/ circuits can be attributed to specific traits. However, he does not ascribe to the position that there are and should be subtypes of psychopathy. His work in looking for a "super-factor" that connects all of the facets (Hare, 2004; Neumann, Hare, & Newman, 2007) is proof of his position.

neural abnormalities (but not necessarily PCL-R diagnosable) are used to represent psychopaths in neuroscience research; and ultimately, the construct is often being defined by, and referred to as a neurological dysfunction. What we want to emphasize is that psychopathy is first and foremost a scientific construct, and so everything we have presented in this literature review is subject to controversy. We have shown that the uncertainties concerning this construct are extensive, rendering it malleable; the construct is subject to change as new areas of research and new technologies contribute to the fact building process. Despite these constitutional uncertainties, we have shown that psychopathy is a prevalent construct across a variety of fields (e.g. psychology, criminology, psychopharmacology, business, etc.). Our interest resides in the way the construct is built and made real through the use of science and technology, which consequently affects the way psychopathy is conceptualized in the first three problematizations of this chapter. To explore how psychopathy as a scientific construct is being reconstructed through neuroscience, we analyze a neuroscience article as a case study through the lens of Science and Technology Studies (STS).

THE MAKING OF PSYCHOPATHY AS A NEURO FACT: SCIENCE AND TECHNOLOGY STUDIES, AND ACTOR-NETWORK THEORY.

The preceding chapter provided an overview of what is known, theorized, and being explored in regards to the psychopathy construct. Elements of a different nature (i.e. clinical, etiological, practical) were discussed in trying to detail what exactly psychopathy is, and how current researchers have been attempting to define the construct. However, this paper has yet to comment on the importance of the processes involved in these scientific endeavours, or how researchers have been able to produce the widely-acknowledged assertions surrounding the psychopathy construct. That is, although the preceding chapter has detailed assertions around the construct, we have yet to explore how psychopathy as a scientific fact is produced. Our thesis focuses on some neuroscience facts of psychopathy by analyzing one key research article composed by a group of prominent researchers. By exploring how these facts are produced, we hope to support our contention that psychopathy as a scientific construct is not merely discovered as a pre-existing natural entity, rather it is built by a series of decisions and judgments. Our thesis is that psychopathy is a *boundary object*, a flexible, construct that is adaptable to local sites across heterogeneous social worlds (Star & Griesemer, 1989, p. 410). It is important to note that in exploring the facticity of this construct, like other STS researchers, the intention is not to question or appraise the truthfulness of the facts, but rather explore how the world adjudicates reality, how facts come into being (Pickersgill, 2010).

To help guide the exploration of facticity, this paper engages with the work of prominent STS and Actor Network Theory (ANT) researchers. Both STS and ANT provide a theoretical background from which this paper can participate in a guided exploration on the facticity of the neuroscientific specificities of psychopathy. This chapter details the theoretical background used for this thesis, and includes sections that discuss the building versus the discovery of facts; where to start a STS/ ANT exploration; the nature of truth and facticity in science from and ANT perspective; the process of translation; and criticisms of ANT. The following discussions provide the foundation from which we can introduce specific concepts in the following analysis chapters as building blocks for our methodology.

The Building vs. Discovery of facts

The conventional sociologist of science would ascertain that science and the social should maintain separation in order to ensure the purity of scientific discoveries. This conventional supposition asserts that if science and scientists were to succumb to social pressures, the legitimacy and purity of their discoveries would inevitably be contaminated and unable to reflect nature (Latour, 2005). This thesis adopts an opposing STS framework that does not limit the relationship between science and the social to determinism and pollution. That is, we suggest that science and technology are "integrally connected to the context within which they are developed and used; culture is made up of such connections; and that [science] and technologies arise within these connections as part of them and as effective within them (Slack and Wise, 2007, p. 112). Most STS researchers agree that science and technology (taken together "technoscience") are "social constructs" … like all forms of knowledge, [technoscience] is generated through social processes made possible by a wide variety of practices and institutions that include political decision-making, funding bodies, experiments, collaborations, dissemination, and contestation (Pickersgill, 2010, p. 383). However, this theoretical positioning should not be conflated with radical social constructivism where theorists rigidly oppose the relevance of material reality. This positioning is inclusive of both construction/ fabrication and reality/ truth.

As Bruno Latour (2005) explains in his book *Reassembling the Social*, this theoretical positioning is often misunderstood as implying that science can be reduced to dust by "associating the artificiality of the construction with a deficit in reality" (p. 92). Latour (2005) explains how the word construction is often misconstrued by researchers due to the connotations of the prevailing social constructivist discourse. Researchers are often limited to the duality of realism and social constructivism, which limits their thinking to "the strange idea that you had to submit to this rather unlikely choice: either something was real and not constructed, or it was constructed and artificial, contrived and invented, made up and false" (Latour, 2005, p. 90). Latour (2005) negates this duality, and explains how the construction of facts is actually a function of reality and artificiality coinciding.

In regards to science, this version of constructivism (both real and constructed) is well suited; scientists build facts in their laboratories, fabricating them out of artificial situations, and they take pride in the quality of their constructions and data (Latour, 2005). This version of construction simply accounts for the solid, objective reality where various entities are mobilized and articulated into an assemblage, a web of associations, which constitute facts (Latour, 2005; Slack and Wise, 2007, p. 113). For example, a house is constructed, and it is real. Using the term socially constructed loses all meaning, as this 'social' entity is a lost concept of 'stuff' that cannot be grasped or directly observed. However, if we replace the blanket word 'social' with 'association' (Latour, 2005), we are able to identity all of the entities, observe all of the actors (social and material) that are mobilized and associate to participate in constructing the reality that is the concrete, observable house. These actors are both human and non-human: the construction workers, engineers, investors, concrete, earth beneath the house, mortgage interest rate, wood, gravity, nuts and bolts, drywall, property as a sign of success, architects, buyers, etc. Although this example seems trivial in a paper about personality and neuroscience, it is meant to paint a picture of all the parts and processes involved in building a reality. The following section of this chapter elaborates on this idea.

Where to Start the Exploration

"[Psychopathy] classification and diagnosis involves the construction of representations of aspects of the patient in terms of a presumed underlying reality, constructed as part of biological, medical, or social science" (Manning, 2000, p. 624). This means that aspects (i.e. behaviours and personality characteristics) of patients are represented through constructs (i.e. items on the PCL-R), which are then presented as part of scientific knowledge that is able to capture a presumed underlying reality. In the case of psychopathy, it is clear from the information presented in the preceding chapter that the constructed representations of an underlying reality are plagued with uncertainties; much of the scientific knowledge and technological applications surrounding this construct are uncertain.

Many scientific assertions are taken for granted as factual and are used as starting points for further research despite controversial histories, complex inner workings, or large commercial/ academic networks that hold them in place (ex: DNA as foundation of life). These taken for

granted assertions are called *black boxes* (Latour, 1987, p. 3). Scientific facts that have been *black boxed* are less easily studied by ANT researchers because the assertion has fostered certainty, the work is done, history, the unproblematic black box leaves a cold trail where the researcher is stunted by a lack of activity (Latour, 1987, p. 4; Venturini, 2010, p. 264). If the controversy were hot, there were open debates, uncertainties, and the assertions were actively debated by scientists, then the work, decision making, and competition among disagreeing scientists would be available for the sociologist to see the action, the science in the making (Latour, 1987, p. 4; Venturini, 2010, p. 264). In other words, uncertainty promulgates activity, which is where the raw observable data presents itself to the sociologist.

As a scientific and practical uncertainty, some aspects of psychopathy are more easily examined than others in regards to facticity, because sociologists are able to study and observe the *science in action* as it is built (Latour, 1987; Manning, 2000). However, other aspects of psychopathy have maintained relative stability over the years, and can perhaps be considered black boxed, so the line of inquiry for sociologists has gone stale. For instance, the PCL-R could very well be considered a black box. Despite a controversial history in regards to the PCL-R representing a valid clinical and empirical construct , complex and debated inner workings (Gendreau, Goggin, & Smith, 2002; Hemphill & Hare, 2004)[10], and large commercial/ academic networks that hold the PCL-R in place (e.g. Darkstone research group, Aftermath: Surviving Psychopathy, support from a variety of academics, etc.), the PCL-R has been taken for granted as the gold standard construct and measurement of psychopathy (Edens et al., 2001; Meloy 2000; Seagrave and Grisso, 2002). The PCL-R has been black boxed, as the overwhelming majority of influential and active psychopathy researchers use it as a taken for granted fact, case closed, use and move forward. This is why our paper focuses on a more active chapter in the life of psychopathy, an episode at this very point in time that is rife with ongoing controversy, where "scientists and engineers are busy at work… our entry into science and technology will be through the back door of science in the making, not through the more grandiose entrance of readymade science" (Latour, 1987, p. 4). We enter into and explore the point where psychopathy

[10] These citations mark an unsolved debate between research groups, where Gendreau et al., 2002 discuss the inner workings and complexities of the PCL-R that may be problematic in terms of its construct validity and its practical utility as a risk measure. Hemphill and Hare, 2004 defend the construct with an alternative description of the inner workings and complexities of the PCL-R.

intersects with neuroscience, because this is the most current and active area of study where the most prominent researchers are invested, science is in the making, and scientists are at work exploring the field for the right pieces to build facts that will hold. Scientists are occupied with the neurosciences in their attempt to build facts around construct definitions, etiological mechanisms, diagnostic measures, and treatments (Glenn & Raine, 2014; Patrick, 2006).

Truth, Associations, and Actor-Networks

Marx states that "an objective truth is not a theoretical but a practical question" (as cited in Latour & Woolgar, 1979; Manning, 2000). The strength of Marx's statement is evident with regards to psychopathy, as the construct has prospered in practical application despite theoretical uncertainty. It seems as though a statement's transition from proposition or hypothesis to fact is its ability to fit within the world to which it applies. Bruno Latour (1987) explains how a common conception of truth is that "when things are true they hold", but he challenges this notion with his conception of truth, "when things hold they start becoming true" (p. 12). This aligns with the notion of fittedness, because the idea is that assertions are not factual within and of themselves, but rather because they have been able to connect, associate, coincide, support, and hold within the area and context to which they apply. This also has implications for what constitutes 'nature'; nature, or, reality, is not the cause that allows controversies to be settled, but rather the consequence of settlement (Latour, 1987, pp. 12-13). For example, in regards to psychopathy, Manning (2000) explains how the construct has garnished truth due to its ability to fit and link within a network of seemingly heterogeneous actors, such as doctors, patients, home secretaries, drugs, drug companies, insurance schemes, prison, surveillance, and risk technology (p.627). Thus, psychopathy is a fact of nature, the construct is real, as a consequence of its construction and settlement of its existence among associated actors.

Until now this discussion has referred to the quality of facticity in terms of fittingness. However, this term does not do justice to the complex processes involved in making something fit. Latour (2005) discusses his framework of the 'sociology of associations' in contrast to the more practiced 'sociology of the social'. The latter relies on the word 'social' as an explanation of the world, but Latour (2005) rejects the notion of a social dimension: "there is no social

dimension of any sort, no 'social context', no distinct domain of reality to which the label 'social' or 'society' could be attributed… no 'social force' is available to 'explain' residual features other domains cannot account for… There is no such thing as a society" (pp. 4-5). Latour (2005) explains how sociologists of the social are limited in their ability to explain the objective/ natural sciences, because by definition, the natural sciences are supposed to be separate and protected from the social domain, they are supposed to reflect pure, untouched, discoverable nature. Thus, sociology of the social is limited to a tautological explanation of how "'social factors' could explain 'social aspects' of non-social phenomenon" (Latour, 2005, p. 3), essentially, the social explains the social.

This has lead Latour (2005) to suggest that social theory must be reworked, as "the social has never explained anything; the social has to be explained instead" (p. 97). Latour (2005), and this paper, negate this undefinable and mysterious 'social' entity, and instead replace the term with *associations:* the sociology of associations accounts for nature, or a reality, that can be identified by tracing a trail of associations between heterogeneous elements. This sociology of associations is best defined as Actor-Network theory (ANT). These heterogeneous elements can be referred to as actors, and tracing how the seemingly disconnected actors articulate together and build a network of associations is the work of an ANT theorist. The analyst must follow the actors themselves, let the actors define their own activity, assembly, and reassembly. It is imperative that the analyst refrain from projecting their own explanations onto the actors, and instead let them explain for themselves. In ANT, the definition of an actor is more inclusive than other social theories, as it includes anything that has agency, both human and non-human, breathing and non-breathing/ inanimate objects (Callon 1986; Latour 1987, 2005; Manning 2000). Taking psychopathy as an example, the PCL-R, Robert Hare, news media, academic journals, CJS practitioners, forensic psychologists, neuroscientists, fMRI machines, etc. could all be considered actors that associate together to make psychopathy exist.

Now, this all-encompassing idea of an actor-network where associations are documented without limitation is obviously impractical, or even impossible for any given case study. Also, the usefulness of such an all-encompassing tracing of associations is questionable. What actors

and associations are included or excluded from a network are therefore fundamental to the way scientific issues are tackled and how stories unfold (Manning, 2000).

Latour (2005) explains how an actor-network is not a thing or an entity, but rather a concept, it is a tool to describe something, not the thing being described. The actor-network is a tool to identify and describe the fluid, ever moving, dynamic flow of actors and associations that are in flux throughout scientific controversy. For ANT, a good textual account of a given topic is one that is able to trace a network of actors where all actors actually do something; they impact the movement, organization, associations, and processes of *translation* (inducing two or more actors into coexisting) [11] between actors (Latour, 2005). Determining what actors are actually doing something at a given moment depends on the ANT researchers' entry point into the topic for which they are providing an account for. Due to the constant flux of associations, actors included in the researchers' account are dependent on where, when, and what they are providing an account for. Key actors in a good ANT account mediate associations between all interested actors, and actively interest and recruit other actors that bolster their own interests (Manning, 2000). Thus, in a good ANT account the researcher must carefully observe, recognize, and delimit their account to the actors that are actually doing something, making a difference.. In regards to this paper, the framework on how to provide a good ANT account of a case is essential. As detailed in the preceding chapter, psychopathy is a construct that has associations spanning across a plethora of domains (e.g. criminal justice, psychology, psychiatry, pharmaceuticals, genetics, victimology, neuroscience, psychometrics, databases, academic journals and articles, business corporations, media, etc.) each with an uncountable number of associated actors. Thus, it is essential for us to clearly define the episode in the career of psychopathy that we are attempting to trace, and then identify the actors who are actually doing something accordingly.

The ability to identify the actors who are actually doing something within the network of relationships is a task that could benefit from the use of some tools, or, theoretical guidance. Latour (2005) asserts that the actors who are actually doing something are *mediators*, in contrast to their less important counterparts, *intermediaries.* An intermediary is an actor that merely

[11] The process of translation is described and detailed at a later point in this chapter.

transports meaning, movement, effects and/or force across and between actors without any form of transformation along the way; when a force or factor is moved by an intermediary actor without change, then the force/ factor is what carries significance, not the intermediary. In contrast, a mediator is an actor that has impact, it *translates* (transforms) the force or factor that passes through, it changes the organization of associations. For example, in this thesis a few actors that we would consider mediators are the fMRI machine, the PCL-R, and the Handbook of Semantic Norms (Toglia and Battig, 1978) used in the Kiehl et al. (2001) experiment. These actors are mediators because they transform the force that passes through them; the fMRI machine transforms neural activity into a computer-generated image based on changes in magnetic fields; the PCL-R transforms observable personality characteristics into a calculated numerical score out of 30; and the Handbook of Semantic Norms (Toglia and Battig, 1978) transforms real life emotionally arousing material into a list of words with calculated affective ratings. Thus, an actor that is actually doing something is a mediator, and a good ANT account should thereby focus more on mediators than intermediaries (Latour, 2005).

Making Nature in the Laboratory

In STS, social constructivism explains that science and technology do not provide direct access to the natural world, as the products of science and technology are not themselves natural (Sismondo, 2010, p. 57). Conversely, ANT is considered a 'relational materialism' as it reduces the natural world/ actors to matter (abandons the 'social' stuff), and defines the objects in relation to their places and roles within networks, not in isolation; "both the social and material worlds [are] the products of networks" (Sismondo, 2010, p. 86-87). It is understood that science and technology translates nature from one form to another; the original object (ex: running around constantly) is embodied in another material form (ex: diagnostic of attention deficit disorder), by way of material manipulation, and these referents to the object, are just as much material and real as the original object.

Laboratories are places where instruments are available to researchers to engage in such manipulations, and create accessible material forms that are embodiments of their research objects. As scientists translate the original object into an expression of that object, it is made

accessible to the rest of the world, and able to relate, or, articulate with other entities. Moving a step further, material forms that embody the object of interest can be manipulated to make forms that are embodiments of the embodiments as a means of making them more general and applicable within a network (Latour, 1999). That is, there may be multiple layers of embodiment. For example, Latour (1999) followed a team of researchers who took an object, soil in the amazon rainforest, manipulated it by taking samples meant to embody differences of soils in the entire rainforest, and then manipulated it again by translating the samples into a data set where the soils are embodied by a uniform colour code. The translation of material embodiments into new forms allows them to aggregate more actors; the data set is an embodied material form of the amazon rainforest soil, and can articulate with other entities and produce universal scientific knowledge. The material referents that allowed for the production of knowledge should be able to be systematically traced back to local interactions and hands on manipulations; there should be no gap between steps, the minute transformations should be traceable (Sismondo, 2010, p. 83). That is, the representativeness of the translated material must be accurate in order for a fact to hold (Callon, 1986). Facticity depends on those who are represented to agree to their transformations, to their enrolment in the network (Callon, 1986). Relational materialism (ANT) is so valuable as a methodological framework, because it allows us to trace scientific knowledge, the product, to its conditions of production, the local accounts/ objects from which it was derived.

The laboratory is a place where researchers collect and organize instruments that give them the power to harness material and produce nature. The natural world is made accessable and communicable through charts, graphs, tables, and written word. In this state, nature can be articulated with other entities, form a stable network, and produce scientific knowledge about our world. This is why we analyze how the Kiehl (2001) article used instruments in the laboratory to produce inscriptions, or referents, to the objects of their investigation. Although our study is limited to analyzing the aspects of the Kiehl team's laboratory from traces that are elucidated in their 2001 article, a brief analysis of the laboratory equipment and processes is valuable in understanding how their work translates into facts about nature.

Criticisms of ANT

Prior to detailing our methodology and the analysis of the Kiehl (2001) article, it is necessary to address the criticisms of ANT and justify its use within this thesis. First and foremost, ANT has been criticized for being "culturally- flat" (Sismondo, 2010, p. 89), as it fails to "bring society ready-made to its studies" (Matthewman, 2011, p. 121). As we have shown above, Latour (2005, p. 97) purposefully rejects the notion of a society, a social entity, or a social force that pre-exists and can be used to explain phenomena. While rejecting the social is one of the main tenets of ANT, it is also a main source of criticism. Critics argue that a sociological theory which fails to account for socio-economic conditions (e.g. gender and racial inequalities, class, capitalism, etc.) and established cultural practices cannot provide a holistic account of events (Matthewman, 2011, p. 121; Sismondo, 2010, pp 87-89). Critics argue that ANT fails to account for the conditions that set the context for technological and scientific advances; ANT "can tell you how two people conduct a conversation, but cannot tell you why they would be in a room together in the first place" (Matthewman, 2011, p. 121). Matthewman (2011) provides an example of how ANT fails to illuminate determining factors by explaining how disregarding culturally embedded discrimination like racism renders the theory ineffectual in accounting for slavery, as slavery absolutely did not rely on the free movement of people through obligatory passage points.

Considering the scope of this thesis and our research question, the above noted ANT criticism is not overly detrimental. Our work intends to show how constructing neuroscientific facts around the psychopathy is both made and real, social and natural, a hybrid. We analyze an episode in the career of the psychopathy construct that relates to fact construction through scientific texts. This thesis analyses one academic article on neuroscience and psychopathy (i.e. the Kiehl 2001 article) to explore how academic procedures, rules and writing is able to harness the world and produce facts about psychopathy. This thesis explores how the scientific activity and results in the Kiehl (2001) article are both social and natural, and produces facts that are simultaneously constructed and real. Relating back to Matthewman's (2011, p. 121) quote expressed above, our intention is to tell you how people conduct a conversation, not to tell you why they would be in a room together in the first place. That is, this thesis discusses how

authors create facts in a laboratory, present their assertions through texts, and communicate through referencing techniques (i.e. conduct a conversation). This thesis does not attempt to explore why the authors in the Kiehl (2001) article came together as writers, what the social or economic consequences of the research are, or what cultural preconditions or consequences are implicated with the Kiehl (2001) publication.

Another criticism with ANT is with the concept of actor agency. Following from the criticism of being culturally flat and failing to acknowledge social forces; ANT has been criticized for ignoring social structures that either allow or prevent actors from participating in networks, which results in the narrow focusing on central actors and missing non-central actors (Sismondo, 2010, pp. 71-72). With regards to this thesis, we are indeed focusing on the central actors, and mainly, the work of academic writers. Any marginal activity performed by non-central actors will be missed in this thesis. For example, edits from peer reviewers, pressures on the authors from their respective faculties, business transactions with equipment suppliers, perspectives from laboratory technicians and volunteer students will all be neglected in this thesis. Focusing on the central actors is a decision that is due, primarily, to our research interest. Broadening the horizons and considering the role and perspectives of non-central actors is beyond the scope of this paper. Perhaps we may miss some mediators in our exploration, but we are analyzing an article where the scientific work performed in and prior to the laboratory has already occurred and been lost. Thus, a suggestion for future research would be to start the exploration earlier by following a team of researchers from the point of their inception.

The following chapters present our analysis of the Kiehl (2001) article. Our analysis is divided into three chapters: an analysis of the Kiehl (2001) laboratory, an upstream analysis of their referencing techniques, and a downstream analysis of how Kiehl (2001) is cited and mobilized by proceeding researchers. Each chapter follows a similar structure whereby the relevant concepts and methods pertinent to the subjects being discussed are introduced first and the empirical analysis proceeds afterwards. In the following analysis chapters we show how the Kiehl (2001) assertions became facts by producing nature through the building of associations. This supports our contention that psychopathy is being redefined as a neuroscience fact by researches who purposefully built fortified assertions. The researchers' techniques allowed for

them to collect an array of previously heterogeneous entities, and organize them into a buttressed fortress. We assert that psychopathy as a neuroscience fact was not simply a pre-existing fact of nature waiting to be discovered, rather the facticity- the quality of being a fact, lies in the quality of the work researchers did to build psychopathy as a neuroscience construct. Psychopathy is a hybrid fact, it is social/ created and therefore a real part of our natural world. Part of the quality/ condition of the construct as it is built by researchers is its flexibility. We ascertain that psychopathy is indeed a fluid construct that is translated into many qualitatively different forms so that it is amenable to connecting with other entities. Our thesis is that psychopathy is a *boundary object*, an adaptable construct that can be abstracted from by multiple heterogeneous domains, and thereby inhabit several intersecting worlds in its qualitatively different abstracted forms (Star & Griesmer, 1989, p. 410). As a *boundary object*, psychopathy is "plastic enough to adapt to local needs and constraints of the several parties employing [it], yet robust enough to maintain a common identity across sites. Through its fluidity, its chameleon like properties, it has been able to connect with the neurosciences and be defined as a neuroscientific construct.

INSIDE THE KIEHL (2001) LABORATORY

As we have stated earlier in this thesis, it is our contention that rather than being discovered, psychopathy as a known scientific fact is built, created, made through a series of judgments, decisions, and evolutions. This thesis focuses on the psychopathy construct's turn toward neuroscience to solidify its existence as a fact of nature. Rather than simply interpreting the neuroscience literature on psychopathy as evidence that psychopathy has been discovered in the brain, we recognize the work that is involved in constructing the facts. In this chapter, we analyze information pertaining to the Kiehl (2001) article's laboratory, and detail the evidence of an enormous quantity of decisions and judgments that were made in producing the facts on neuroscience and psychopathy. Now, although we are alluding to the social work that is performed in constructing these facts, we are by no means suggesting that the facts are constructed, and thereby not true or real. Rather, we assert that the more work put into construction makes it more real, more solid, effective and powerful. Therefore, the facts produced in the Kiehl (2001) article can be considered hybrids, both social and natural.

We begin with a brief summary of the Kiehl (2001) article. We then move into a discussion on how Latour (1987) conceptualizes the nature of laboratories, which informs our own methodology for analyzing the Kiehl (2001) article. Finally, we detail our own ANT inspired analysis of the Kiehl (2001) laboratory, which effectively acts as evidence for our contention that psychopathy as a neuroscientific construct is a hybrid (social/ constructed, and natural/ real) fact produced by researchers, and the quality of being a fact, its facticity, lies in the quality of the work done to build their assertions. We will direct your attention to five main translations that are taking place in Kiehl's lab to create the neuroscience reality of psychopathy: name the 5 translations.

A Summary of the Article used as Data for our Empirical Exploration

As was mentioned in the preceding chapter, this empirical exploration is limited to the analysis of one peer reviewed article published in 2001 by a team of leading researchers: Kiehl, K. A., Smith, A. M., Hare, R. D., Mendrek, A., Forster, B. B., Brink, J., & Liddle, P. F. We refer to the article as the Kiehl (2001) article. The Kiehl (2001) article reports on the findings of a

laboratory experiment conducted in attempt to determine the neurological causes for psychopaths' difficulties/ abnormalities in processing emotions, or, affective stimuli. Their study is intended to identify the neural networks underlying the abnormal emotional processing in psychopaths by utilizing an fMRI machines' ability to produce images of the brain with a greater resolution than other previously used imaging machines.

The experiment consisted of n=8 criminal psychopaths and n=8 criminal non-psychopaths from a Canadian maximum-security prison. The psychopath designation was according to high PCL-R scores for the psychopathic group, and low PCL-R scores for the non-psychopathic group. Additionally, n=8 non-criminal non-psychopathic (measured using the PCL-SV) participants were recruited from the general population. All of the participants, N=24 were males selected according to strict screening criteria in order to limit the effects of confounding variables (i.e. substance abuse, brain trauma, age, intelligence). The experimenters subjected all of the participants to 8 rounds of an affective memory task. As a means of measuring neural activity during affective processing, the participants were to perform the task while having their brains scanned and imaged in an fMRI machine.

The task consisted of three phases: encoding, rehearsal, and recognition. In half (4) of the 8 rounds participants were presented with words rated as neutral in affect (e.g. chair) during the encoding phase. In the other half of the 8 rounds, during the encoding phase, participants were presented with 12 words rated as negative in affect (e.g. death). Participants were then given time to remember the 12 words (the rehearsal phase). Finally, in the recognition phase, participants were presented with some novel and some rehearsed words. Participants were asked to indicate whether or not they recognized each word from the encoding phase by using their index and middle fingers to answer yes or no on a custom switchboard.

The experimenters were not interested in what type of word (negative or neutral) was more easily recognized by the participants, but rather how their brains processed the exposure to negative versus neutral words. After subjecting the participants to 8 rounds of the three-phase task, the researchers took the fMRI results and meticulously cropped, smoothed, normalized, and reconstructed the data to produce average images of the participants' brains across trials. The

researchers determined the statistical significance of the differences between images of the three participant groups through ANOVA- fixed effects and random effects analyses. Compared to the criminal non-psychopaths and non-criminal participants, criminal psychopaths showed less affect-related neural activity when processing the negative affect words in 6 of the 10 brain zones. However, the criminal psychopaths showed more brain activity in 2 cortical brain zones that are situated outside of the limbic system. The researchers concluded that their hypothesis was confirmed, and previous research was supported by their results: "[T]he results support the hypothesis that criminal psychopathy is associated with abnormalities in the function of structures in the limbic system [reduced activity] and frontal cortex [increased activity] while engaged in processing of affective stimuli" (Kiehl et al., 2001, p. 682). The researchers assert that the heightened activity in the frontal cortex indicates that psychopaths use more cognitive resources when processing affective stimuli than non-psychopathic persons, who rely more on limbic structures to process the affective stimuli. That is, psychopaths use alternative neural systems to process affective stimuli.

Finally, the researchers support their claims with both their experiment results, and by linking their findings to other researchers' corresponding experimental results and theories. The article is modest in discussing the strength of their findings. The researchers openly state that the limitations of their experiment (i.e. sample size, confound variables, and the presentation of affective stimuli was limited to verbal material) reduces the ability to generalize to other populations, and express how the research was intended to be exploratory, not confirmatory.

Creating Nature in Laboratories

The academic article that is analyzed in our study began in the laboratory. We are going to introduce concepts that delineate how we understand scientific work.. The following quote from Latour (1987) is a highlight summary of how we conceptualize assertions, and the kinds of questions we ask when analyzing claims within a text:

> What is behind the claims? Texts. And behind the texts? More texts, becoming more and more technical because they bring in more and more papers. Behind these articles?

> Graphs, inscriptions, labels, tables, maps, arrayed in tiers. Behind these inscriptions? Instruments, whatever their shape, age, and cost that end up scribbling, registering and jotting down various traces. Behind the instruments? Mouthpieces of all sorts and manners commenting on the graphs and 'simply' saying what they mean. Behind them? Arrays of instruments. Behind those? Trials of strength to evaluate the resistance of the ties that link the representatives to what they speak for (Latour, 1987, p. 79).

This section of the chapter will elaborate on and detail the concepts Latour (1987) has developed to support his theoretical framework on science and fact making.

Researchers often test their hypotheses in their laboratories, which Latour (1987) has defined as the place where scientists work, whether a wet laboratory, a dry laboratory or a dark corner with a computer.

In the case before us, a laboratory is comprised of many instruments that the scientist(s) have meticulously recruited, organized, calibrated, and mobilized to create an entire system that is designed to measure the object under investigation. Latour (1987) looks to "shake up the complicated set-up that provides graphs and traces in the author's laboratory in order to see how resistant the array is which has been mobilized in order to convince everyone" (p. 75). It is common for a peer reviewed text to contain images, charts, and/or graphs that act as a visual representation, and display of the findings, object that was discovered, effects, etc. For example, in the neuroscience article on psychopathy under study, there are digitized images of the brain, and charted statistical analyses of the average differences between images according to different participant groups (Kiehl et al., 2001).

It is necessary to look at the laboratory as a means of understanding where and how these images/ results are produced, as this will help the reader understand what the results actually entail; the facticity of the scientist's assertions. Latour (1987) explains that a text's results are the product of a long laboratory process that involved extracting images from instruments in a laboratory, cleaning and redrawing the images, and transcribing them into something that can be read and understood by a reader. For example, in the neuroscience experiment under study this

would include selecting participants according to a specific demographic; calibrating and recalibrating an fMRI machine; taking the fMRI results and attaching it to a computer with graphics software; cleaning the images with the software by comparing it to a baseline and removing visual 'noise'; statistically analyzing the multiple images and producing a chart that displays the numerical significance of the images; and then writing the results into a language for the reader to understand. In this example, we see a series of moves that the scientist makes from subjecting participants to stimuli to producing a text that comments on their neural activity. Latour (1987) explains that paying attention to the instruments, or, *inscription devices* used in laboratories allows us to follow the scientists' moves (i.e. their move from nature, to referent, to yet another referent of nature. The reader, or even the scientists, are not looking directly at the brain of any subjects (nature), but rather inscriptions (i.e. referents) of the neural activity (i.e. nature) produced by the complex set up of instruments that the scientists designed.

The author collects and assembles a complex system of instruments that produces a visual display of their research results (Latour, 1987). Returning to our fMRI study, an isolated spectator would only see a participant being subjected to stimuli in an fMRI machine, a computer displaying neon coloured blotchy images, the researcher meticulously calibrating the instruments and cropping the produced images through computer software, and a table of numbers that differentiates the images. The only way a non-initiated spectator, or sometimes even a specialist, can make sense of any of it is through the researchers' verbal explanation. The researcher must act as a *spokesperson* to give the whole setup meaning (Latour, 1987). The researcher describes what is happening at each stage of the experiment by explaining what the stimuli represents in the real world: that the participants' brains are responding to the stimuli, the fMRI machine is measuring changes in brain activity detected through changes in blood flow, the computer images are cropped to reduce the brain activity that is not associated with the stimuli, and the images are compared to one another to determine the significance of the change in brain activity.

Latour (1987) explains that an inscription, is nature that has been translated from its natural form by an instrument. The inscription is nature in a more accessible, communicable form. For example, an fMRI image is an inscription of neural activity, a form of neural activity that is accessible and readable for us. The strength of the laboratory does not lie in the

instruments' inscriptions themselves, but rather the representative of the inscriptions; the researcher who represents the inscriptions acts as a mouthpiece for the silent inscriptions that do not speak for themselves. A person who visits the laboratory sees a complicated set up that seems to speak for itself through the representatives' voice; "the mouthpiece [researcher] does not 'really talk'… he or she is just commenting on what you yourself directly see, 'simply' providing you with the words you would have used anyway" (Latour, 1987, p. 73). However, Latour (1987) explains that this is a major weakness in how facts are created in the laboratory in the sense that it is impossible to discern who is speaking. The things/ instruments (nature that comes up in the form of inscriptions) and the people (subjects) are given a voice through the speaker, but it is impossible to know whether or not the speaker is accurately representing the things and people in the laboratory. By the very nature of the laboratory, such direct communication is impossible, the things and people being represented cannot talk directly to someone who visits a scientists' workplace. Thus, what the visitor of a laboratory sees is indeed different from what the spokesperson (researcher) says about the visual display, and this indirect communication is subject to error. The laboratory is a carefully constructed, convincing stage that provides the illusion of nature directly communicating. The researcher builds the complicated set-up that produces graphs, images, and traces which are mobilized to convince others of their assertions (Latour, 1987). The researcher may be an objective representative of the things it speaks for, but this can only be determined through imposing *trials of strength* on the researcher and their laboratory (Latour, 1987). That is, the ties that connect the represented and their spokesmen should be tried and tested to determine whether the spokesperson is an objective individual, or a subjective individual who "speaks for him or herself, who represents only him or herself, his or her wishes and fancies (Latour, 1987, p. 78).

The fact making process has its roots in the laboratory. The methods and results noted in the scientific articles are not taken for granted. When analyzing our data (articles), using Latour's (1987) conceptualization of fact making in the laboratory facilitates a more holistic understanding, and a keen eye for determining what aspects of an article should be dissected and explored. The following section of this chapter details our analysis of the remnants of the Kiehl (2001) laboratory, which we were able to identify from their article.

In sum, nature is produced in the lab using instruments. Those instruments are speaking in tongues. They produce inscriptions and only a skilled spokesperson can translate what those inscriptions mean. The scientist speaks for nature through his instruments and their inscriptions. Each time matter goes through an instrument and generates an inscription, there is a translation, a qualitative jump, being accomplished. Nature is made of this series of translations (jumps), between the physical world (matter) and signs (the inscriptions). Matter is not nature. There is no direct correspondence between matter and nature. It is through necessary translations that nature is "fabricated". No translation from the physical world to the symbolic world: no nature. In the following section, we show you five key translations that happened in K laboratory to produce psychopathy. These include 1. The translation of the subject- from sample to population; 2. The translation of affective material and processing- from life events to operationalized stimuli; 3. The translation of neural activity- from blood-flow to a responding brain; 4. The translation of brain anatomy- from coordinates to neural structures; and 5. The translation of images- from raw to reconstructed. These translations are evidence that psychopathy as a neuroscientific construct is built in the laboratory, that the raw substance of psychopathy, the matter, is not what defines the nature of the construct, but rather the qualitatively different inscriptions.

Analysis of the Kiehl (2001) Laboratory: Inscriptions, Referents, and Embodiments of the Brain

1. The Translation of the Subject- from Sample to Population

Starting with what is the most recognizable information to social scientists, a sample is a small part or quantity that is intended to show what the whole is like. In the Kiehl (2001) article the researchers sample from three populations: non-criminal participants (n=8), non-psychopathic criminals (n=8), and criminal psychopaths (n=8). This can be seen as the first transformation, the first movement from the natural world to a representation/ referent of it. The physical world where 1% of the global population is a psychopath is transformed into a representation, a sign of that world; there are eight incarcerated participants chosen from a super-max prison. It does not take any special insight to understand how scientists harness nature through sampling techniques, and the researchers are explicit in their article on the limits of generalizing the results from their sample to the rest of the population. However, we will see

that later in time when this research is borrowed and taken downstream (cited and used in later research articles), translations such as these are often lost.

Additionally, the samples used in the Kiehl (2001) study have shown us clear instances of subjective decision-making by the researchers whereby the entities, or actors, enrolled in their study were made to negotiate their constitution. For instance, the diagnosis of psychopathy requires a score of at least 30/40 on the PCL-R, any less than 30 and the person is not a diagnosed psychopath (Hare, 1991). However, in the Kiehl (2001) study the experimenters chose to include those with a score below 30 in the psychopath sample group. The researchers explain "inmates with a score above the mean psychopathy score (23.6, SD 7.9) for the 1192 inmates presented in the PCL-R manual were defined as psychopaths" (Kiehl et al., 2001, p. 679). Here we see an entity in the experiment, psychopathy as defined by the PCL-R, forced to negotiate its definition to include those with a score below 30. Thus, the facticity of the Kiehl (2001) assertions rests on the notion that psychopathy can be defined by a score of 28.

2. The Translation of Affective Material and Processing- from Life Events to Operationalized Stimuli

Knowing the researchers are attempting to access the nature of the neural systems that mediate the affective abnormalities in psychopaths, it is imperative to consider how these abnormalities are recreated and accessed within the laboratory. First, in the Kiehl (2001) article the researchers iterate that according to the PCL-R, glibness, superficiality, and a lack of guilt, empathy and remorse are features central to the psychopathy disorder. They follow up by explaining how the symptoms are associated with "difficulties or anomalies in the processing and production of affective material" (Cleckley, 1976; Hare 1993: as cited in Kiehl et al, 2001). By tracing the quotation back to the cited authors, we can see more translations with regards to what represents "affective material" and the "processing of affective material". Kiehl (2001) moves from detailed real-life observations of affective processing abnormalities in psychopaths (i.e. Cleckley, 1976, Hare, 1993), to the PCL-R as a valid instrument that measures and operationalizes real-life characteristics. Kiehl (2001) also moves from real-life affective stimuli, to four-letter stimulus words. Both Cleckley's (1976) *The Mask of Sanity* and Hare's (1993) *Without conscience: The Disturbing World of the Psychopaths Among Us* are books that detail

the disorder through a multitude of case studies. Cleckley (1976) and Hare (1993) deliberate on the affective processing of psychopaths by way of in-depth case analyses where the researchers contemplate psychopaths' behaviours, decision making patterns, and affective processing in real life situations. For example, Hare (1993) recounts his interactions with the psychopath "Ray" (one of many case studies), which provides a detailed explanation of Ray's life history, interpersonal style, examples of social interactions, decisions Ray makes, and his behaviours in the context of complex social situations. Based on observations from case analyses like these, Hare (1993) asserts that psychopaths are abnormal in their processing of affective stimuli, and thereby abnormal in their behavioural responses to such stimuli. While case studies like these indeed illuminate the presence of a so called personality type within the criminal population, the data is too raw, too localized, to try and harness and connect it with other actors (i.e. neuroscience) in an attempt to build knowledge. As such, nature, the real life affective processing and affective stimuli, is translated, manipulated, extracted from its original setting, and made real, or, fabricated within the Kiehl (2001) laboratory. The detail of how a psychopath processes complex affective material in the material world is translated into a score out of 40, an operationalized definition of a set of human characteristics. There is indeed a qualitative difference between the person/ participant as a disordered individual and a numerical score out of 40. With regards to facticity, here we see no direct connection between matter- the psychopath and nature. Nature is fabricated through the inscription- the numerical score out of 40, produced by the PCL-R- the inscription device/ instrument.

Instead of discussing the real life affective stimuli that is complicated, messy, and unique to each participants' experiences, the laboratory harnesses these natural circumstances by employing an instrument, *The Handbook of Semantic Word Norms* (Toglia & Battig, 1978), to represent the affective stimuli. The semantic word norm handbook included a *pleasantness* dimension in which university students rated thousands of 4-5 letter words along a 7-point scale from negative, neutral, to pleasant. The Kiehl (2001) experimenters decided to operationally define negative affective stimuli as "words [from the handbook of semantic norms] rated as more than 1.3 standard deviations below the mean pleasantness rating" (Kiehl et al, 2001, p. 679). The real life affective stimuli that made researchers (Cleckley, 1976, and Hare, 1993) proclaim psychopaths have abnormal processing of affective material based on their behaviours, decision-

making patterns, and self-reported rationale for their behaviours, has been replaced by a 7 point pleasantness scale. This is how the Kiehl (2001) article used an instrument to make the nature of affective stimuli accessible (i.e. measurable and relational) to researchers; by translating the real-life experiences of affective material into semantic stimuli. Again, the quality of the nature of affect is not connected to matter, to the substance of real lived experience, but rather to an inscription of it, an operationalized form where the quality is transformed into a statistically valid set of 4-letter words.

3. The Translation of Neural Activity- from Blood-Flow to a Responding Brain

Moving forward from the translations used in the Kiehl (2001) study to define their independent variables (i.e. participant groups and affective stimuli), we now consider how the researchers define and measure their dependent variable, affective processing. Premised on the notion that psychopathy symptomology is attributable to affective processing abnormalities, the experimenters attempt to locate the disorder at the source, neural mechanisms responsible for affective processing. Considering that the brain is enclosed in skull, is composed of approximately 1 billion neurons that make up a vast array of interrelated structures, and is the most complex and least understood organ in the body, researchers are forced to be creative when accessing it as an object of study. The Kiehl (2001) experimenters are the first to employ functional magnetic resonance imaging (fMRI) to capture the neural activity of psychopaths as they process affective material. They explain how their fMRI machine is superior to earlier imaging techniques (e.g. Single Photon Emission Computer Tomography [SPECT], and electroencephalography [EEG]), because the fMRI produces images with greater spatial resolution, which renders it better equipped to identify the neural systems underlying the abnormal affective processing in psychopaths.

While the participants performed the experimental task described in the Kiehl (2001) article, the fMRI machine detects, and captures images based on blood-oxygen-level-dependent (BOLD) contrast, which is essentially contrasting an image captured at baseline (no activity) with an image captured while the participants perform the task (i.e. subtract the baseline image from the task performance image to get an image that reflects neural activity specific to the task performance). The premise to this technique is that oxygenated blood will transfer its oxygen to

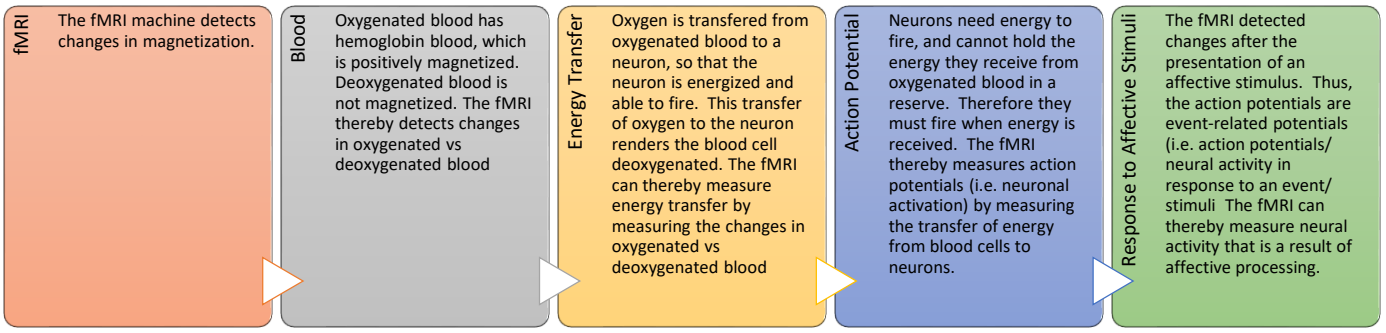
areas of the brain that are active; the more oxygen transferred from blood to an area, the more 'activity' in that area of the brain. The areas of the brain that have changes in oxygenated blood levels during the performance of the task are said to be activated.

The Kiehl (2001) article does indeed provide a thorough description of the laboratory procedures and an explanation of the results. However, we divulge and offer a description of their laboratory that considers the implications of their procedures with regards to facticity. Using Latour's (1987) framework we consider how nature is made in the laboratory, going from matter to signs. As described above, the actual activity of the human brain is not accessed directly in the Kiehl (2001) experiment. Instead, neural activity is accessed according to a series of premises on the nature of the neural activity, and based on said premises, the fMRI instrument produces inscriptions that represent neural activity:

> -The fMRI machine detects changes (from baseline) in magnetization of the brain when affective stimuli (4 letter words) are being processed by contrasting baseline and task performance magnetization levels.
> -This change in magnetization is said to be the result of the difference in magnetization between oxygenated, and deoxygenated blood. Blood contains hemoglobin, which is an iron (Fe) that contains magnetic properties.
> -The premise with fMRI imaging is that neurons do not have their own energy reserves, so when they fire (i.e. activate), more energy is brought in through a process called the *hemodynamic response*: blood releases oxygen to active neurons at a noticeably greater rate than inactive neurons. The change in oxygen levels changes the structure of the hemoglobin molecule (deoxyhemoglobin or oxyhemoglobin), which changes its magnetization, which is detectable by fMRI (Poser, 2009).

Therefore, the fMRI is actually detecting changes in magnetization in different parts of the brain that result from changes in levels of oxygenated blood (Poser, 2009). That is, the fMRI does not detect neural activity (i.e. action potentials) directly, or even the transfer of energy that signifies activity. Rather, the fMRI detects changes in magnetization, which represents changes in

oxygenated blood levels, which represents a transfer of oxygen from blood to neuron, which indicates that the neuron is firing since it needs oxygen (Poser, 2009).



The above explanation contains the facts which act as premises for the legitimacy of fMRI imaging techniques and applications, and already we see many degrees of separation from the actual object under investigation. In this explanation of neuroimaging using the fMRI and the BOLD response alone we see four clear translations. The quality of the natural world is translated four times over by the time we reach "response to affective stimuli". The matter, change in magnetization, has no direct link to what Kiehl (2001) refers to when they discuss the natural world. There are three translation, three qualitatively different forms in between: (de)oxygenated blood, energy transfer, and action potentials. Here we see the original form of affective processing/ neural activity negotiate its constitution whereby it is defined by a set of layered premises that can ultimately be reduced to changes in magnetization. Changes in magnetization is how we are able to translate the quality of neural activity into a communicable form, it is how the Kiehl (2001) article defines neural activity. Again, the facticity of their assertions is attributable to their inscriptions, and thereby the notion that our neural activity can be signified by changes in magnetization. Here we have shown how matter, the active human brain, has no direct link to the natural world according to Kiehl (2001). Rather, through the fMRI we measure magnetization levels, the brain is transformed into a qualitatively different entity, magnetization.

4. The Translation of Brain Anatomy- from Coordinates to Neural Structures

The Kiehl (2001) researchers are tasked with more than just accessing the brain as a whole as it responds to affective stimuli. They must identify and locate the structures that are active due to the affective component of the stimuli. This involves a meticulous process of neurological mapping that entails the translation of matter, neural structures, into coordinates.

While the participants completed 8 trials of the 3-phase task meant to represent and contrast the processing of neutral versus affective stimuli, they laid down inside of a "standard clinical General Electric 1.5 T whole body system fitted with a Horizon echo-speed upgrade" (Kiehl et al, 2001, p. 679). Their heads were secured in a "custom head holder and external references were used to position the anterior commissure- posterior commissure (AC-PC) line at right angles to the slice-select gradient" (Kiehl et al, 2001, p. 679). The accurate positioning of the head, so that the fMRI could capture the appropriate images, was confirmed using "conventional echo T1 weighted sagittal localizers", which simultaneously "prescribed a subsequent 3D SPGR (TR/TE 11.2/21 msec, flip angle 60°, FOV 26 _ 26 cm, 256 _ 256 matrix, slice thickness 1.5 mm) volume acquisition" (Kiehl et al, 2001, p. 679). Then, while the participants performed the memory task "functional image volumes were collected with a gradient-echo sequence (TR/TE 2500/50 msec, flip angle 90°, FOV 24 _ 24 cm, 64 _ 64 matrix, 62.5 kHz bandwidth, 3.75 _3.75 mm in plane resolution, 4 mm slice thickness, 23 slices)" (Kiehl et al, 2001, p. 679).

The work performed by the Kiehl (2001) researchers described above involved a calibration process prior to taking the images. This entails trying to capture and notice the minutest differences in activity while simultaneously maintaining enough resolution to produce a readable image of the intended structures. This calculation involves understanding the differences in magnetization of oxygenated versus deoxygenated blood; neural activity that is not a result of the experimental task (i.e. *noise*); neural activity/ noise that is a result of the task but not the activity that is of interest to the researchers (e.g. the section of the motor cortex that activates when responding by moving the index and middle finger, the occipital lobe and surrounding areas that activate in response to seeing the word stimulus, and/ or the language processing area (Wernicke's) that activates when we process language); a multitude of other

anatomical and functional properties of the brain; and of course, advanced knowledge of the technical specifications and properties of the fMRI machine (Ulmer & Jansen, 2013).

Now in acknowledging this calibration process we are acknowledging the carefully staged situation specific to the Kiehl (2001) laboratory. We can see traces of black boxes stacked on top of one another to create the conditions necessary for accessing affective processing in the brains of psychopaths. The black boxed landmarking techniques and instruments allowed the researchers to fabricate nature by creating inscriptions of neural structures. We show how the brain is translated into coordinates. The experimenters onerously manufactured a custom headrest that would be able to hold the participants' heads in the correct position for the fMRI to capture the areas of the brain the experimenters presumed specific structures were located. They also confirmed their landmarking through a black boxed fMRI landmarking technique (i.e. acquiring "conventional spin echo T1 weighted sagittal localizers" and using "Talairach coordinates"). This landmarking technique uses functional localizers to locate the structure of interest in each individual subject, which accounts for the differences in brain size, shape, folding patterns and the locations of areas across subjects. The "sagittal localizers" are essentially low-resolution high-field-of-view sagittal-plane brain slice fMRI images that act as scout views of individuals' brains. The localizer images are then used by the researchers to plot/ landmark the region of interest in each individual participant. Due to individual differences, a one landmark fits all approach to imaging would not work. This landmarking technique ensures that the appropriate area of the brain is captured in each individual participant. Thus, in addition to the BOLD black-box that is the central premise to fMRI imaging, we see two more black-boxes stacked into the Kiehl (2001) experiment; a custom headrest that positions the head properly, and a black-boxed landmarking/ localization technique used to capture the areas of interest.

This realization provides us with another translation; coordinates obtained from landmarking techniques represent the regions of the brain that are alluded to in the Kiehl (2001) article. The researchers do not explicitly see a neural structure, rather they see a section of the brain marked by coordinates that represent a structure. That is, the images in the experiment are so zoomed-in to an area of the brain, that the specific region of the brain in the image cannot be

identified in relation to other regions within the image. The only region included in the image, is the region of interest. The only way the experimenters know they are actually looking at the area of interest is by referring to the coordinates obtained from the landmarking techniques described above.

To make this more understandable, consider this example: you are looking through a powerful magnifying glass to view a small superficial flesh wound. While looking through the magnifying glass you can only see the wound. You cannot see anything else through the magnifying glass that would identify where the wound is in relation to other points of reference within the field of view. It is only when you zoom out, that you see a thumb, a wrist, a forearm, and can thereby identify that the wound is on the palm of a right hand. This is the same with fMRI images, while zoomed in, they cannot tell what is being viewed by relation to other regions. However, the researchers are unable to simply zoom out at will. Instead, they must locate where the area of interest is, landmark it (i.e. get the coordinates), and trust that their landmark is correct and that they are indeed imaging what they intend to. The structure that is imaged (e.g. the amygdala) is represented by coordinates obtained from the landmarking technique. Talairach coordinates, from the Talairach atlas, are used to position the zoomed-in image in relation to the rest of the brain. That is, based on a structural probability map, the coordinates of the zoomed in image of a region can be used to map and create a zoomed-out image of the most probable stereotaxic brain.

In other words, knowing that one set of coordinates localizes a structure (e.g. amygdala), based on previously mapped Talairach coordinates of the entire human brain, the rest of the brain coordinates can be inferred, and a zoomed-out picture of the most probable brain can be generated. Matter, the brain, is not what we are presented as nature in the Kiehl (2001) article. Instead we are presented with a series of translations that leave us with a qualitatively different entity, an inscription of neural structures. With these coordinates we have a fabricated nature; the physical neural structures are actually coordinates. The Kiehl (2001) laboratory fabricated the natural world by using black boxed Talairach coordinates that can map out the brain symbolically through statistical probabilities.

5. The Translation of Images- from Raw to Reconstructed

The calibration/ landmarking process described in the previous section allowed for the raw images of the participants' brains to be captured while they observed the affective stimuli. Once the images (raw data) were acquired by the experimenters, an arduous process of data mapping (Statistical Parametric Mapping [SPM]) was completed; "we performed repeated measures group…analyses of variance (ANOVAs) on the accuracy data… Functional images were reconstructed offline and the two runs were separately realigned using the procedure by Friston et al (1996) as implemented in Statistical Parametric Mapping" (Kiehl et al, 2001, p. 679). Next, from the realigned image volumes, mean/ average image volumes (i.e. averages across the 8 trials) were constructed. Finally,

> Mean image volume was then used to determine parameters for spatial normalization into the modified Talairach space employed in SPM99 using both affine and nonlinear components (Friston et al 1995a). In this space, coordinates are expressed relative to a rectangular coordinate frame with the origin at the midpoint of the anterior commissure and the y axis passing through the posterior and anterior commissures. The normalization parameters determined for the mean functional volume were then applied to the corresponding functional image volumes for each participant. The normalized images were then smoothed with an 8 mm FWHM Gaussian filter. (Kiehl et al, 2001, pp. 679-680).

The above technical writing means that after each participant completes the 8 trials and the raw data is obtained by the experimenters from the fMRI, image reconstruction begins. Reconstructing the raw images into coherent, informative material is another arduous procedure guided by black-boxed SPM. SPM essentially uses parametric statistical analyses coupled with fMRI raw image data to produce mean image volumes, and set the image parameters to localized areas of interest. That is, the researchers take all of the individual fMRI images collected across experimental trials and combine them to produce a mean image volume (i.e. an image that represents the average of all images by combining them into one image). The reason it is called mean image 'volumes' is because the image is detecting the volume (i.e. amount) of oxygenated vs. deoxygenated blood in the area of the brain being observed during the experimental task. Thus, the mean image volume is the average amount of blood levels in an area of the brain across trials and participants. It is noteworthy that the experimenters noticed a "relatively slow

onset of the hemodynamic response" (Kiehl et al., 2001, p. 680), and so they made the decision to delay the mean image computation by six seconds. This is another example of an instance where the experimenters' judgment and decisions were clearly a part of the fact making process. Also, setting the image parameters refers to the process by which the researchers identify the locations of the brain they wish to capture in their image, and focus in on those locations so as to produce quality images that focus the resolution on the area of interest. A much simpler example of setting image parameters that might make the processes more understandable is in photography; a photographer may have captured an image containing a large landscape, but only be interested in a small section of the landscape. The photographer would then adjust their lens, angles, and aperture to focus in on and set the scope of their image (i.e. set the parameter).

Once the parameters had been identified, experimenters were able to spatially normalize (still using SPM) and smooth (using an 8mm FWHM Gaussian filter) the images to produce the final product. Spatial normalization is required to normalize the mean volume images because the raw images from which the mean images were obtained were taken from multiple participants with different sized and shaped brains. The differences in size, shape, and general location of brain structures across participants' causes an unreadable mean volume image when the raw images are collapsed into a single image. Spatial normalization deforms the brain scans so one location and size of a structure in one subject's brain scan corresponds to the same location and size of a structure in another subject's brain scan. Finally, image smoothing refers to a statistical procedure in fMRI image processing whereby important patterns in the image data are made to be more pronounced by modifying the image so that noise data is eliminated from the image. The resulting modified image is one that is focused in on the brain regions of interest to the researchers, is in the clearest resolution possible, and is limited to the data that is of interest to the researchers (i.e. neural activity that is a result of affective processing).

Clearly, the Kiehl (2001) laboratory is a thoughtful and complicated design of many inscription devices that have been aggregated to make a coherent whole; the ability to produce the inscriptions that support their claims relies on a finely tuned set-up of elements necessary to make up the whole experiment (Latour, 1987, pp.74-75). As a result of such efforts, the

researchers are able to provide the reader with a neatly packaged image that provides a visual display of their results:

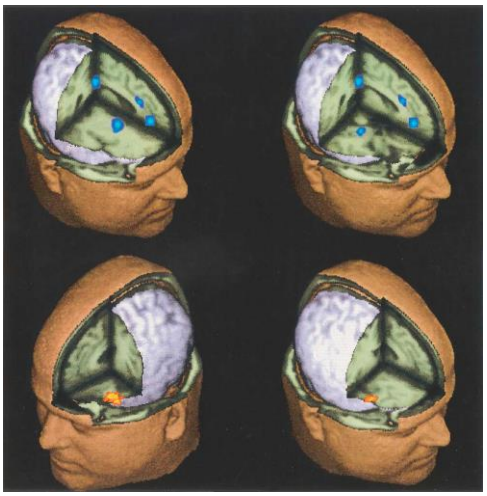


Figure 1. Rendering of the neural areas in which criminal psychopaths showed significantly less affect-related activity than noncriminal control subjects for the comparison of all affective phases versus all neutral phases from the random-effects analysis (top panels; depicted in blue color scheme, all voxels illuminated are statistically significant at the $p < .05$ level). Regions include (top left) posterior cingulate, caudal and rostral anterior cingulate, and ventral striatum (top right), right amygdala/hippocampus. The Talairach coordinates for these results are listed in Table 1. Also shown are the regions in which criminal psychopaths showed greater affect-related activity than noncriminal control subjects and criminal nonpsychopaths (bottom panels; depicted in orange, all voxels illuminated have a probability level < .05). These regions include bilateral inferior frontal gyrus (see text for details).

(Kiehl et al., 2001, p. 681)

This neatly packed image is the product of another translation, from raw data/ images to reconstructed mean images. The experimenters took the raw material produced by the fMRI and surrounding instruments and reconstructed them with black-boxed SPM methods, which included prescribed ranges for image translation and rotation corrections[12], production of mean image volumes, spatial normalization parameters, and image smoothing (Friston et, al, 1995abc; as cited in Kiehl et al, 2001). We see layers of representations in this process that create many degrees of separation from the actual object of study. First the brain, the physical matter, is translated into a qualitatively different form, a reading of magnetization levels. Second, regions of the brain alluded to in the article are translated into landmarking coordinates. Third, the raw fMRI images are translated into a reconstructed mean image, which were then subject to a series of SPM informed image reconstructions. In the Kiehl (2001) article we do not only move once from the physical (matter) to the signs (inscriptions), but rather multiple times where we have layered inscriptions of inscriptions.

[12] The translation and rotation corrections involves realigning each of the images in a functional time series so that they are all in the same orientation, which is necessary to correct for disorientation due to participant head movement and time differences between image slice acquisition.

The Significance of Laboratory Translations

The Kiehl (2001) researchers were able to access their object of study by fabricating the nature of it. The physical brain, the human psychopath, the affective material were all created by translating the physical into the symbolic. The experimenters used black boxed instruments to create the inscriptions of their objects of study, and in doing so rendered them qualitatively different. The fact is, facts are built on a foundation of inscriptions with more inscriptions as the materials. Thus, when traced back to the local interactions, what constitutes the fact that psychopaths process affective stimuli abnormally is as follows: in comparison to n=16 non-psychopathic participants, n=8 psychopathic participants (as defined in this experiment) showed statistically significant differences in neural magnetization while processing four letter affective words. What we see is representations of psychopathic and non-psychopathic populations through samples; representations of real life affective stimuli through 4-letter words rated on a 7-point pleasantness scale; representations of the brain through a statistically significant set of amalgamated and reconstructed images, that represent multiple raw images, that represent magnetization levels, that represent blood flow, that represent energy transfer, that represent neural activity.

In determining what constitutes facticity, the answer is not simple, harnessing nature involves transforming it and working with the accessible referents to it. And often, by the time a referent to nature is made accessible, it is so far removed from the original object, there are so many layers of transformation, that it takes a well-articulated spokesperson to explain how the referent represents its original form.

Latour (1987, pp. 71-73) explains that the inscriptions created in the laboratory say little on their own accord, and that it takes a spokesperson to speak for them; direct communication with the objects under investigation is impossible, and so we are provided with inscriptions (i.e. a visual spectacle in the form of a 3D image of the human head/brain and raw fMRI images), but even the inscriptions are meaningless to an outside observer without the spokesperson (i.e. the researcher) to explain their meaning and represent them. Thus, we as readers/ observers are not only forced to believe that the multiple layers of inscriptions (i.e. inscriptions of inscriptions:

original fMRI image, reconstructed images, 3D image) represent the actual object, but also that their spokesperson accurately represents the inscriptions. Knowing that the inscriptions and the spokespeople are reliable representations of the objects in question requires that the carefully staged situation, the calibrated instruments, and the spokespersons' claims be subjected to trials of strength (Latour, 1987).

Subjecting the Kiehl (2001) article to trials of strength would require visiting the laboratory, shaking up the complicated set-up of instruments, and questioning the spokespersons' explanations of the visual spectacle (Latour, 1987). This is beyond the scope of this thesis, as it would require the replication of their laboratory. We did not have the opportunity to alter the elements in the Kiehl (2001) laboratory, or question the experimenters' explanations of the inscriptions. Latour (1987) deliberated that the setup in a laboratory is a "coherent whole… an aggregate of many elements that could be induced to go in many different directions" (p. 75). What if we shook up the elements? What if we subjected the results to different statistical testing; change the words rated on a pleasantness scale from 1.3 standard deviations (SD) below the mean to 1.2 SD; used a different model of fMRI machine; present the words with something other than the VAPP; change the positioning of the head by using a different custom headrest; use image reconstructing where translation and rotation corrections did not exceed 2mm and 2 degrees as opposed to their 3mm and 3 degrees; normalize the image with a different type of Gaussian filter; define a region of the brain as something other than a cube of volume $1000mm_3$ (i.e. a voxel); or, when adjusting mean images, change the temporal delay from 6 seconds to 5 seconds? How do we know that the change in magnetization is an accurate representation of neurons firing; why was the mean PCL-R score 23.6- SD 7.9 for the psychopathic group when a diagnosis requires a score above 30 (Hare, 2004); why did the non-psychopathic group have participants with a psychopathy score of 23 included in it considering that is very close to the psychopathic group cut-off score? These possible questions for an outside observer would appear to be endless. Questions like these test the ties that make up the whole, and see how easily the array can be induced to go in different directions, to produce different results.

The questions presented above are examples of the kinds of decisions made by the researchers that create space for controversies. In this chapter, we have brought to the surface

the many conditions, decisions, negotiations and compromises that all had to come together to form the facticity of psychopathy as a neuroscientific construct. We see in the Kiehl (2001) laboratory what Callon (1986) calls enrolment "the group of multilateral negotiations, trials of strength and tricks that accompany the interessements and enable them to succeed" (p. 10). We have exhibited the negotiations and compromises the researchers made in this chapter (e.g. changing PCL-R cut-off scores, custom head rests, computation delays, etc.). Despite these complications, the Kiehl (2001) researchers were indeed able to enrol the necessary actors for their laboratory and produce the results that rendered psychopathy a neuroscientific construct. However, in detailing the conditions, compromises, and negotiations, we have shown how there are many elements that make up the whole. In showing the many elements that had to come together to form the facticity of psychopathy as a neuroscientific construct, we have shown how psychopathy is a sophisticated, yet fragile neuroscientific construct that offers many handles/ grounds for controversies.

In all likelihood, if we had the opportunity to question the experimenters, our inquiry would be stunted at every step. The array would likely resist all modifications we attempt, or we would be scoffed at for our absurd questioning. The reality of contemporary science, of the Kiehl (2001) laboratory, is that scientists do not stand alone in their endeavours; they are supported by thousands of contemporary instruments used in their laboratory, and classic claims in neuroscience, fMRI methodology, statistical analysis, sampling and research methodologies, etc. The researchers are supported against any kind of criticism in their representation of nature, as their constructions included other facts, sciences and black boxes, which come to their rescue against any dissenter (Latour, 1987). To question the ties that make up the Kiehl (2001) laboratory would be to question all of the allies they have mobilised to convince others of their results. We would have to question Friston's (1995abc) SPM, the VAPP, the fMRI imaging machine, etc., which are all black boxed facts beyond the scope and resources of this thesis.

We have shown how Kiehl (2001) built the facts, created the reality through their ability to harness nature, and translate it into more accessible (symbolic) forms that represent the original element (matter); there is no direct connection between matter and reality in the Kiehl (2001) article. The referents to matter were made via the manipulations of local accounts, and

can systematically be traced back to those local interactions (Sismondo, 2010). To a degree, we have shown how the Kiehl (2001) researchers logically trace their referents of nature back to the local accounts (e.g. sample of psychopaths as representation of all psychopaths; 4-letter words back to real life affective material; 3D image of the human head/ brain back to raw fMRI data, etc.). These laboratory processes outlined in this chapter support our contention that the psychopathy construct is a boundary object, a scientific object that is adaptable and malleable so that it can inhabit many intersecting worlds. We have shown how psychopathy is translated into an affective and neurological abnormality in the Kiehl (2001) article. However, this is where supporting and justifying their fabrication of nature goes beyond what is done in the laboratory. To truly fortify their assertions, they must bring in friends (i.e. other renowned researchers) to support and legitimize the premises (i.e. supporting facts) from which their claims are based on (Latour, 1987). This part of the fact-building process is the topic of the following chapters.

UPSTREAM ANALYSIS- HOW KIEHL (2001) BUILDS PSYCHOPATHY AS A NEURO FACT THROUGH TECHNICAL WRITING

In this chapter, we dive further into the exploration of the facticity of the assertions made within the Kiehl (2001) article. As was mentioned in the closing of the previous chapter, having their assertions qualified as factual goes beyond the work that was done within the laboratory. In this chapter, we analyze the technical writing used by the Kiehl (2001) authors, and explore how it is used as a means to substantiate their claims. Latour (1987, p. 62) supports our position on the hybrid nature of the Kiehl (2001) facts by stating that "the more technical and specialized a literature is, the more 'social' it becomes". That is, a fact is built, and is able to become a reality through its ability to be social (i.e. form associations).

This chapter begins with a description of Latour's (1987) framework (used as our methodology), which details the 'social' work performed by academic authors to produce facts/ nature/ reality. We then move into an 'upstream analysis' of the Kiehl (2001) article. By 'upstream' we mean that we move backward analyze the references they used, as opposed to 'downstream' (the following chapter), which analyzes how the Kiehl (2001) article has been enrolled and cited by proceeding authors.

The first section of the analysis details how an agenda is set by researchers, whereby the construct is described in such a way that renders its many definitions necessary to the entire plot. Psychopathy is defined as an affective abnormality, a neurological disorder, and it is associated with neighbouring entities that render it a flexible construct capable of gaining strength from those entities with which it connects. The second section of the analysis shows how psychopathy is a neuroscience construct because it is a construct that is supported by a fortress of corroborating allies. Psychopathy is embedded in instruments that create inscriptions, which are ultimately the very substance of the construct. Also, psychopathy is a construct that is the final product, the whole of many incomplete translations that come together. Finally, the last section of the analysis explains how psychopathy is a construct that has gained veracity through the logical stacking of many heterogeneous truths. The construct is not a physical entity that stands alone, but rather a complex positioning of multiple truths together to logically form the overarching truth that psychopathy is a neuroscientific construct. Together, these three

subsections support our overarching position that psychopathy as a hybrid, built neuroscientific fact is indeed a malleable construct, a boundary object.

Literature: Technical Writing is the 'Social Work' in Fact Making

Empirically, the focus of our study is facticity; the way neuroscience assertions about psychopathy become seen as facts. Our data consists of a series of empirical neurocriminological studies about psychopathy. The framework developed by Latour (1987) is used to analyze the data. Therefore, in moving to Latour's (1987) chapter on literature, this section details our methodology for analyzing the technical writing used in the Kiehl (2001) article. This section on the literature introduces concepts and tools, which are used directly as a method for analyzing the data. However, these tools only make sense in the context of the theoretical positioning from which they were created, and so the theoretical background related to fact making and academic literature is explored in this section of the chapter as well.

To begin, it is necessary to understand how we conceptualize fact making in relation to academic literature. Latour (1987) explains that "the status of a statement depends on later statements" (p. 27). That is, once an article with a researcher's findings is published and disseminated to the public, its fate lies in how others receive and mobilize the article. For instance, consider a researcher who discovers a revolutionary fact about the human brain, who used up to date laboratory equipment, sound methodology, and was able to quash all controversy surrounding the topic with their finding. After publishing their well written and edited academic article that presents their findings, no one in the academic community or greater public even reads the paper, it is ignored. The finding is ignored, the article unread, and the fate of the assertion is inevitable; it simply cannot be turned into a fact (Latour, 1987). Latour (1987) explains how there is a collective fate of fact making; an assertion requires the help of others to be mobilized and made into a fact. A given statement (or article) does not possess the ability to be qualified as fact or fiction by its own accord, rather, its fate is determined by how others qualify it at a later point in time; statements cannot survive without being taken up by proceeding authors (Latour, 1987). Unfortunately, the first, and most difficult step is interesting others enough to read your article, as most articles are never read at all (Latour, 1987). This involves

carefully constructing an article so that it interests others and gains readers, which involves techniques that are discussed later in this chapter.

Now we will introduce concepts that are used in our method for analyzing academic articles. Latour (1987) explains how statements that modify, or qualify, previous statements (i.e. one author refers to, and qualifies the statement of a predecessor) are called *modalities*. There are both positive and negative modalities. A positive modality is a statement about a previous author's statement that "lead [the original] statement away from its conditions of production, making it solid enough to render other consequences necessary" (Latour, 1987, p. 23). A positive modality is essentially when an author refers to another authors' previous statement, qualifies the statement as factual, and moves forward from, or, builds from the statement that was qualified/ modified as factual. Negative modalities are just the opposite, they are "sentences that lead a statement in the other direction toward its conditions of production and that explain in detail why it is solid or weak instead of using it to render some other consequences more necessary" (Latour, 1987, p. 23). A negative modality modifies, or, qualifies, the statement it is referring to by moving backward and commenting on its conditions of production (e.g. the laboratory, research methodology, theoretical positioning, author's biases, etc.). For example:

Original statement (A): We raced a Labrador Retriever, a Golden Retriever, a Jack Russell Terrier, and a Yorkshire Terrier. The Labrador Retriever and Golden Retriever came in 1st and 2nd place. Therefore, Retriever breeds are faster than Terrier breeds.

Positive Modality (B): Knowing that Retriever breeds are faster than Terrier breeds (*A*), we propose that Retriever breeds should be the work dogs used in emergency rescue teams.

Negative Modality (C): Statement *A* only raced four dogs, of which we do not know if the Retrievers were younger and healthier than the Terriers used in the race. Also, the Terrier breeds chosen for the race (i.e. Jack Russell and Yorkshire) are the two smallest terrier breeds, perhaps if other Terrier breeds, such as the Pitbull or Staffordshire Terrier were raced, the outcome would be different.

In the above example, we see an assertion made based on a single race of four dogs (A). In statement (B), statement (A) is positively modalized: taken as fact, led away from its condition of production (i.e. a single race between four dogs where the breeds selected may not be representative of the dog type), and further consequences were made necessary (i.e. use the Retriever as a work dog). Alternatively, Statement (C) negatively modalizes statement (A): the conditions of production were alluded to (i.e. single race, non-representative sample of dog types, and issues with sample age and health), and the statement was qualified as weak. Remember that it was noted above how an assertion cannot become factual if it is ignored, or, not utilized by future researchers. This concept of modalities introduces how statements that are not ignored can be utilized, and whether or not they will be modified into fact or fiction.

This study uses the concept of positive and negative modalities as a tool for analyzing the data. As we analyze the academic articles we follow the line of references and identify the positive and negative modalities. That is, we start with the main article being analyzed, but then trace back the references upstream to see how the article *modalized* (i.e. modified) the previous literature. Additionally, in the following chapter we move downstream and explore how the following generations of articles have modalized the article we chose to analyze. By following the references, and exploring how they are modalized up and downstream, we should be able to provide insight into the facticity of neuroscience and psychopathy. We support the notion that the work put into building the real neuroscientific psychopathy construct did indeed require referencing, decision making, judgments, and modifications on the part of the researchers. However, knowing that references in a given article are positively or negatively modalized is not enough. We must analyze the intricacies of the modifications to better elucidate the decision making, judgments, compromises, and modifications the researchers made in building their facts.

Now when an author modalizes another authors' statement, it is transformed, and thereby qualitatively different from its original form. Latour (1987) explains how authors engage in politics, and organize their papers as rhetorical devices for supporting their claims. One of their main strategies is to "do whatever they need to the former literature to render it as helpful as possible for the claims [they] are going to make" (p. 37). They adapt the literature to suit the needs of their own text. Authors are well aware that the fate of their articles are dependent on

how or if they are modalized, and so they carefully construct and fortify their articles based on the estimated forecast of proceeding articles (Latour, 1987). That is, authors organize their articles, and the references in the article in such a way that they can pre-emptively defend themselves from what they predict readers and future authors will take issue with. The authors are effectively organizing their articles like pieces on a chess board or troops on the battlefield, where they fortify their defences and plan their attacks. Another useful analogy is the similarity between technical writing and combat sports; the boxer calculates their opponents' possible attacks and prepares for them many moves in advance so that they can be ready with an effective defence and/or counter attack.

It is a rare instance for a given statement to become a fact. Even when a statement does reach factual status, it does not maintain its original form; it becomes *eroded*, *stylized*, polished, and transformed by each successive author (Latour, 1987). This means that as each successive generation[13] of authors cite the original statement, it undergoes generations of transformations. The original statement is first transformed by the following generation of citing authors, then again by the generation following them, etc. When the first generation cites the original statement, they may black box the assertion, accept it as fact and move forward (i.e. positively modalize the assertion). As each successive generation cites the previous generation, the origin of the statement, its conditions of production, are lost and forgotten; the original statement undergoes more and more transformations, until it is hardly recognizable. This is how facts are produced, "every new paper getting into the fray pushes it one step further, adding its little force to the force of the already established fact, rather than reversing the trend" (Latour, 1987, p. 42). Eventually, the original statement, in its new form, after numerous transformations, becomes *tacit knowledge*: a fact so well known it is unnecessary to provide citations when talking about it, the reference becomes redundant, the assertion, reality (Latour, 1987).

It is clear that there is no qualitative difference between an assertion that is accepted as fact, versus one that remains or is deemed fictional (Latour, 1987). The difference between fact

[13] Latour (1987) notes a 'generation' is delineated by the span of time it takes for another round of papers to be published that would be citing the former generation. He explains that this time span is between two and five years.

and fiction lies within the collective action that is imposed on the statement. We have already discussed how the author refers to resources outside of the paper; the author has carefully calculated how to utilize and organize a mass of referees, allies, and thousands of articles that assist in supporting their claims (Latour, 1987). Thus, the reader cannot simply confront the article they take issue with, instead they are directed to a stockpile of absent articles and events that they are forced to consult if they wish to form a substantial opposition.

In addition to external allies (articles), authors also fortify their articles with rhetorical techniques that are present within the texts themselves. Latour (1987) explains that the internal anatomy of academic articles is arranged in such a way that they are more technical, or, *stratified,* than other types of texts. That is, the text is arranged in layers that make it "more difficult to read, just as a fortress is shielded and buttressed; not for fun, but to avoid being sacked" (Latour, 1987, p. 46). The text is stratified, or layered, in that:

> Each claim is interrupted by references outside the texts or inside the texts to other parts, to figures, to columns, tables, legends, graphs. Each of these in turn may send you back to other parts of the same text or to more outside references… no part of the paper stands by itself but each is linked by many references to other layers (Latour, 1987, p. 48).

Therefore, the reader is forced to navigate a well-articulated maze of cross referencing, and layered information. Of the few people who read a given article, most will be overwhelmed by these 'technical details' and simply go along with the author, succumb to the rhetoric, and accept the claims (Latour, 1987). However, our study utilizes Latour's (1987) insight and attempts to deconstruct the author's construction by unpacking the internal and external references of a given article, in hopes of gaining a better understanding of the facticity of the neuroscience of psychopathy.

Latour (1987) advises that skilled authors use *positioning tactics* as a means of organizing and arranging their mass accumulation of references. The first tactic is *stacking,* for which he uses the example of a study on mammal kidney structure:

Low Induction ← - → **High Induction**

Slices of Flesh→ Three Hamster Kidneys→ Hamster Kidneys→ Rodent Kidney Structure→ Mammal Kidney Structure

(Latour, 1987, p. 51)

Latour (1987) explains that through strategically stacking layers of internal and external references, authors are able to take advantage of inductive reasoning, and convince the reader that their laboratory study findings are evidence of wider reaching claims. In the mammal kidney structure example, the scientist cut a slice of kidney flesh from three hamsters, via induction, this translated to three full hamster's kidneys, which translates to all hamster kidneys, which translates to all rodent kidneys, which then translates to all mammal kidneys. Through induction, the findings of three slices of kidney flesh in the laboratory are translated, and extended into all mammal kidneys.

Latour (1987) explains that for stacking to be successful, authors should never stack two layers, or, two episodes of translation on top of/ next to one another; the author should never go straight form the first to the last layer; and the author should attempt to prove as much as they can with as little as possible. This way, the author can extend their results as far as possible, and the reader cannot easily link their inductions/ translations to call the authors' bluff (Latour, 1987). This is where the careful articulation of internal and external references comes into play, as authors make audacious claims about nature, they must curtail the reader from recognizing what their claims could be reduced to. Of course, this is not foul play on the side of the author, it is a necessary trick of the trade; they must rely on inductive reasoning if they wish to make any claims about nature at all. The neuroscientist must induce that one brain represents all brains, the pedologist must induce that one sample of soil represents all the soil in the area, the chemist must induce that one type of reactant represents all reactants etc.

This stacking through induction within the text is similar to what positive modalities do to articles; the end claim is accepted and pushed downstream, making other consequences necessary. For example, if the author within the text induced that three slices of kidney flesh represents all hamster kidney structures, and then stopped there. The next generation author could positively modalize the statement and use it to make claim about all mammal kidney

structures. In this example provided by Latour (1987), the author stacks the layers themselves to make the claim about all mammals, thereby avoiding redundancy; the author should claim as much as possible for just that reason, to avoid being made redundant by the next generation.

Similar to this idea, and the ideas discussed in the 'Laboratories' section of this chapter, Latour (1999) explains how scientists paint reality through successive translations. Latour (1999) followed a group of pedologists on an expedition, and observed the long chain of references necessary to transport the natural world into a text. He observed how the material world, the Amazon rainforest soil, underwent successive translations, where each stage of transformation replaced the original matter. Latour (1999) explains that the Amazon rainforest soil was translated to mapping, field walking, drawing sections, photographs, samples, measurements of samples, narrations of observations, etc. As the researchers navigated the rainforest and recorded their observations, reality was successively being replaced by references to reality. Along the way Latour (1999) recognized a chain of discontinuous transformations as a result of using referents, and explains that is how the researchers linked themselves to the rainforest. "Through successive stages [scientists] link us to an aligned, transformed, constructed world" (Latour, 1999, p. 79). We can study this construction of reality, and trace the chain of references back to the original material world.

The references are treated as mediators, as they translate reality- the force that passes through them, by reorganizing the chain of associations at each stage (Latour, 1999, 2006). The process of using referents to represent reality can be traced back to its origin, to reality, the process is reversible, it circulates. This is why we use Latour's (1987, 1999) conception of science and reality in our study, we can trace the references to reality back to their conditions of production (or as close as possible within our means), and get a better understanding of how the Kiehl (2001) researchers constructed their fact- the reality that psychopathy is a neuroscientific construct.

Other positioning tactics described by Latour (1987) are *Captation*, and S*taging/ Framing.* This involves incorporating a rhetorical writing style that is able to subtly control their readers' moves. These strategies involve subtlety because the reader must be made to believe

that they are free to navigate the paper and come to conclusions on their own (Latour, 1987). The author carefully chooses their words and phrases to captivate their ideal reader. Then, the author calculates their phrasing, and arrangement of internal and external references so that the reader, while free to navigate the terrain, is guided toward the authors chosen path. This involves anticipating what the reader will object to, and how they could be lead to believe; exercising caution and understatement when treading on thin ice, while being audacious when a black box fact is available. Ultimately, authors are creating an intricate maze of references and black boxes mixed with arguable positions, disguised as open terrain so the reader does not recognize that the author is herding them (Latour, 1987).

In summary, the above explanation of academic literature explained by Latour (1987) is what he calls *fact-writing.* The layman, and even the academic is meant to feel isolated when reading a peer reviewed article, while the author carries with them an army of allies. Articles are designed like fortresses that are able to withstand hostile assaults. However, it is rare that an article succeeds and builds a fact, or, creates a reality. For this to happen a multitude of conditions must be met, and as was said earlier, success or failure has little to do with qualitative characteristics. Rather, it must be built to withstand a hostile environment in order to be believed, and more importantly, it must interest readers in order to be considered.

Recall from the previous chapter on the Kiehl (2001) laboratory, we understand how the Kiehl (2001) article is able to access affective processing in psychopaths by creating representations of many heterogeneous elements. We have discussed how the laboratory is a place where a complicated and sophisticated set up of instruments allows for these representations to be created. However, what gives weight to these representations? How is the world convinced that their experiment created the conditions able to truly represent the objects in their study? It is not only a sophisticated experimental design that is necessary for the building of facts, but also a sophisticated system of referencing. The work done with a sample of 24 participants in a laboratory transcends the meaning it would have on its own by connecting with other researchers, other laboratories, other articles, which all lend a deeper, more enhanced meaning to the Kiehl (2001) experiment. By strategically designing the article to support their claims with referencing techniques, a skeptical reader is not only tasked with questioning the

Kiehl (2001) article, but also the 51 cited articles and their corresponding claims. "Attacking a paper heavy with footnotes means that the dissenter has to weaken each of the other papers, or will at least be threatened with having to do so… you might have to engage with all of these papers and go back in time as many years as necessary" (Latour, 1987, p. 33). This chapter includes the upstream analysis: what we found using Latour's (1987) framework to analyze how references were used to support the building of facts around psychopathy and affective processing in the Kiehl (2001) article. It must be noted that this is not a critique on the Kiehl (2001) article, but rather an analysis of how they effectively gather, enroll, and mobilize available resources to build a fact. Our analysis in no way takes away from the veracity of the Kiehl (2001) assertions, it simply comments on how science is performed and truths about our world are manufactured.

Setting the Agenda: Psychopathy as a Neuro, Flexible, and Necessary Construct

In this subsection, we show how the Kiehl (2001) authors build allies with other authors through their referencing techniques as a means of justifying and legitimizing their research. In building their alliances, the Kiehl (2001) authors negotiate the identity of their own psychopathy construct by associating it with other constructs. The researchers ground their own research by associating with previous research on similar topics, while simultaneously rendering their own research as a necessary development in the knowledge of psychopathy as a neuroscientific construct. The following subsections detail the technical writing techniques used by the Kiehl (2001) authors to set the stage for their research, and ultimately define psychopathy as a neuroscientific construct. In setting the stage, we show how psychopathy is construed as an affective abnormality, a neurological disorder, as well as a flexible/ fluid construct.

Psychopathy as an Affective Abnormality.

What is immediately prevalent, is a self-referential process that highlights and aligns the Kiehl (2001) team's earlier publications over 40 years. In this article, they positively modalize their past works when they cite themselves. That is, they confirm the veracity of their previous statements, thereby strengthening them, and assert that further action is necessary (i.e. locating the source of affective abnormalities in the brain). Indeed, positively modalizing their past

results benefits the work they had published earlier and supports the black boxing of their global proposition according to which psychopathy is a distinct and solid construct. The Kiehl (2001) article opens with a paragraph in which every sentence cites works by Robert Hare, the leading researcher in psychopathy, and co-author of the article. The paragraph effectively serves to introduce the scope of the psychopathy problem, delineate the core characteristics of the disorder, and attribute such characteristics to abnormalities in affective processing:

> Psychopathy is a personality disorder believed to affect approximately 1% of the general population and approximately 15%–25% of incarcerated offenders (Hare 1991). Compared with other inmates, psychopathic offenders commit a disproportionate amount of repetitive, often violent, criminal acts (Hare and McPherson 1984; Hart et al 1994). Central to the disorder is a complex of features—glibness; superficiality; and lack of empathy, guilt, or remorse—that appear to be associated with difficulties or anomalies in the processing and production of affective material (Cleckley 1976; Hare 1993). Although the clinical symptomology of criminal psychopathy is well characterized (Hare 1991), relatively little is known regarding the neural systems mediating its affective abnormalities. (Kiehl et al., 2001, p. 677)

This series of statements is weaved to form a coherent program, a program for which the Kiehl (2001) study becomes a logical, if not necessary development. Aside from self-promotion, the opening paragraphs also ascribe a necessary agenda for future research. After the opening paragraph the Kiehl (2001) article asserts, and reasserts that psychopathy is related to affective abnormalities. They support this premise to their research by citing multiple authors:

> One of the most consistent findings from these studies is that criminal psychopaths fail to experience or appreciate the emotional significance of stimuli in the way that nonpsychopaths do (Christianson et al 1996; Day and Wong 1996; Kiehl et al 1999; Louth et al 1998; Patrick et al 1993; Patrick et al 1994; Williamson et al 1991). For example, data from our laboratory has shown that criminal psychopaths fail to show normal behavioral facilitation and event-related potential (ERP) differentiation between emotional and neutral words (Williamson et al 1991). Subsequent research has confirmed the presence of affective abnormalities in criminal psychopaths (Kiehl et al., 1999). These deficits appear to be most prominent in response to negatively valanced emotional stimuli (Day and Wong 1996; Patrick et al 1993; Patrick et al 1994) (Kiehl et al., 2001, p. 677).

The above quoted section of the Kiehl (2001) article is an example of how they support one of the premises to their research by referring to multiple outside sources. The Kiehl (2001)

researchers attempt to locate the source of psychopaths' affective abnormalities in the brain. However, the necessity of this research rests on the notion that psychopaths do indeed have affective abnormalities. Considering that their entire study relies on this premise, it is paramount that its legitimacy is well established. By bringing in powerful allies (i.e. other well established research articles) the Kiehl (2001) article does just that, fortifies their premise by connecting it with other entities. Now, if a dissenter attempts to refute said assertion, they are not only refuting the Kiehl (2001) authors, but all of those authors cited in the above quotation (Latour, 1987).

Psychopathy as a Neurological Disorder

Following from the discussion above, the Kiehl (2001) researchers then explain how ERP studies are limited in their ability to characterize neural sources of psychopathic affective abnormalities due to limited spatial resolution (p. 677). This statement suggests that a.) there is a neural source to psychopathic behaviour, and b.) there is a need for a research design that is able to identify said neural sources. The authors then directly follow up with a statement referring to a functional imaging study (i.e. Intrator et al., 1997) that uses Single Photon Emission Computed Tomography (SPECT). The Kiehl (2001) article suggests that this functional imaging study is more valuable than ERP studies, and quotes their findings:

> psychopathic individuals show greater activation for affective than for neutral stimuli bilaterally in temporofrontal cortex (Intrator et al 1997). These latter data have been interpreted as supporting the notion that psychopathic individuals require more cognitive resources to process and evaluate affective stimuli than do comparison subjects. We note however that this study was limited in that it only assessed function in a 13.5 mm axial of cortex (Kiehl et al., 2001, p. 677).

The above quotation serves an important function in the Kiehl (2001) article. They cite the Intrator et al. (1997) study that asserts psychopaths show increased activation in the temporofrontal cortex, which suggests psychopaths are neurologically different from non-psychopaths. The Kiehl (2001) authors then negatively modalize the Intrator, et al. (1997) assertion by explaining how the study was limited to assessing the function of a mere 13.5mm axial slice of cortex. Negatively modalizing the Intrator et al. (1997) citation at the end of the

statement effectively makes the Kiehl (2001) study a necessary development in the plot; their study will make up for the Intrator et al. (1997) study by using a machine that captures images with a better resolution. Of course, in the Kiehl (2001) results, they were able to be more specific in their claims about heightened affect related activity, by identifying specific structures within the general temporofrontal cortex (p.681). The Intrator et al. (1997) citation served two simultaneous functions: 1. it provided supporting evidence of the affective and neurological abnormalities in psychopaths for the Kiehl (2001) article, and 2. It made the Kiehl (2001) study necessary by acting as an inferior study in need of support from the more advanced Kiehl (2001) imaging techniques.

Continuing with the analysis of how the Kiehl (2001) article's introduction sets the agenda for their study. The authors express how the neural sources of the affective abnormalities in psychopaths are poorly characterized due to the limited spatial resolution of neuroimaging techniques used in previous studies (Kiehl, 2001, p. 677). They follow up by explaining that:

> Researchers have suggested that a number of neural structures and systems may be implicated in psychopathic behavior. These regions include orbital frontal cortex (Damasio et al 1990), prefrontal cortex (Anderson et al 1999; Bechara et al 1994; Raine et al 2000), ventro-medial frontal cortex (Bechara et al 1999b), and limbic structures such as the amygdala (Bechara et al 1999b; Patrick et al 1993; Patrick et al 1994; Tranel and Damasio 1994) and cingulate (Dikman and Allen 2000; Tranel and Damasio 1994). (Kiehl et al., 2001, pp. 677-678).

In the above quotation, the Kiehl (2001) article reinforces the notion that it is possible to find the neural source of psychopathy. The calculated timing of the above statement is another staging technique employed by the authors. Up until this point, there was no clear connection between brain and psychopathy. This section of the article encourages the reader to believe that abnormal affective processing in psychopaths is indeed attributable to a neural source, the specific source(s) simply has to be identified. That is, the above quotation is used to show the reader that the Kiehl (2001) authors are not a group of mad scientists attempting to explore an implausible connection, as other well esteemed (and cited) authors have also been attempting to find the neural source of "psychopathic behaviour". By connecting to these other esteemed studies, the

Kiehl (2001) study ensures the reader that their aspirations are not too far reaching, and that their goal of localizing affective abnormalities in psychopaths' brains is indeed attainable.

Psychopathy as a Flexible Construct

Another common technical writing tactic can be seen in the above quotation. To analyze the above quotation, Latour (1987) suggested that we "trace each reference and probe its degree of attachment to the [authors'] claim" (p.33). By doing this we can see how the authors transform the former literature to their advantage through the *context of citation*, which shows us how the Kiehl (2001) article acts on others to make them more in keeping with their own claims (Latour, 1987, p. 35). First, let us consider the term "psychopathic behaviour" used in the above quotation. Robert Hare, one of the contributing authors in the Kiehl (2001) article has insistently and repeatedly asserted that the psychopathy construct cannot be defined by any one characteristic, rather the presence of the holistic constellation of characteristics is what defines the disorder (Hare, 2004, p.77). That is, there is a super-ordinate factor in psychopathy underpinned by correlated factors, which means psychopathy is a unitary construct that cannot be subdivided into multiple constructs; what makes psychopathy a construct is the homogeneity of the characteristics (Hare, 1996, p.30; Hare, 2004, p. 77; Neumann, Hare, & Newman, 2007).

The underlying point here is that the behavioral characteristics associated with psychopathy could be attributed to a higher-order factor specific to the psychopathy construct. Hare explains by way of an analogy:

> Many traits that define psychopathy- impulsivity, egocentricity, callousness, irresponsibility, and so forth- can be found either singly or in various combinations in other conditions or disorders… overlapping criteria are not uncommon… we have much the same situation in general medicine, where, for example, some cardiac and gastrointestinal disorders can present with similar symptoms" (2004, p.10)

Thus, non-psychopaths who share some of the psychopaths' defining characteristics (e.g. those with narcissistic personality disorder, antisocial personality disorder, brain trauma, borderline personality disorder, non-diagnosed persons who have some of the traits, etc.) would not share the same etiology, just as the patients with cardiac vs gastrointestinal disorders might share

symptoms, but with differing etiology. Now, with this in mind, returning to the Kiehl (2001) article, it seems as though this distinction does not preclude considering the homogeneity of neural etiological mechanisms between psychopaths and those who have displayed psychopathic behaviours/ characteristics. In the above Kiehl (2001) quotation, the cited studies on the neural structures implicated in psychopathic behaviour included participants with localized brain trauma (Anderson et al., 1999; Damasio et al., 1990; Bechara et al., 1994; Bechara et al., 1999; Tranel & Damasio, 1994), APD participants (Raine et al., 2000); and low-socialized individuals (Dikman & Allen, 2000). Although the Kiehl (2001) article follows up by saying "unfortunately, very little is known about the possible involvement of these structures in criminal psychopathy" (p.678), it is clear that the quote was intended to suggest the cited research on "psychopathic behaviour" could be generalizable to diagnosed psychopaths.

Considering the information presented above, we have found that depending on the context of citation, and how the comparison would suit the authors' claims, the exclusivity of psychopathy is flexible. When attesting to the legitimacy of the construct, Hare (1996, 2004) is assertive in how dissimilar and incompatible the psychopathy diagnosis is with other constructs. However, in the context of discussing the possibility of localizing the affective abnormalities associated with the disorder in the brain, similarity and comparison is accepted. The Kiehl (2001) article positively modalizes and modifies the studies they cite in the above quotation. The Kiehl (2001) authors move them from studies on samples of brain damaged, APD, and low-socialization participants, to studies on psychopathic behaviour. However, just as Latour (1987, p. 37-38) suggested they would, the Kiehl (2001) authors carefully close off the paragraph by explicitly stating they do not generalize the cited studies' results to criminal psychopathy, almost as if the entire paragraph were a meaningless digression. Latour (1987, p. 37-38) explains the technical writing technique employed here by the Kiehl (2001) authors: the authors have "carefully modified the status of all the other articles it puts to use" in the above quotation by linking them to their own object of study, diagnosed psychopaths, but are then "humble and understated" as they are "not sure of winning" (i.e. convincing their readers). That is, the above Kiehl (2001) quotation is efficient, strategic and tactical in its linking of the cited studies with their own through modalizations, where they also understate their statement to protect themselves from an argumentative audience. We see the Kiehl (2001) researchers carefully, and

meticulously constructing a reality through associations; decision making (i.e. who to enrol, and how to modalize those enrolled) and compromise (i.e. relating the psychopathy construct to other constructs) are clearly involved in the building of their facts.

This section of our analysis has shown us an example of how the Kiehl (2001) researchers were able to connect a collection of heterogenous entities in their fact building process. We have shown how psychopathy as an actor was forced to be flexible and accommodate as a means of achieving its own end. The Kiehl (2001) authors needed the psychopathy construct to accommodate its definition to fit with other studies on neural structures and behavioural abnormalities. Psychopathy was defined as an affective abnormality, a neurological disorder, and compared to other similar/ corresponding constructs and behaviour patterns.

The Kiehl (2001) article closes with a paragraph that logically follows those that precede it. The introduction intelligently collects, modalizes, and organizes allies to convince their readers why their research is necessary. The introduction discusses the severity of the disorders' impact on society, presents evidence that affective abnormalities are associated with psychopathic traits, presents evidence that many other researchers have been attempting to locate the neural source of said affective abnormalities, and explains that all research to date is limited by either inadequate neuroimaging techniques or samples that do not include PCL-R assessed criminal psychopaths. The Kiehl (2001) introduction then presents their own study, which includes the most refined neuroimaging technique available (fMRI), a criminal psychopath sample, a method for simulating affective stimuli, information based on a successful pilot study, and a hypothesis that fits with the evidence they previously presented: "psychopaths would show less activation than healthy controls and criminal nonpsychopaths when processing affective words compared with neutral words, at those cerebral sites where healthy controls had exhibited significant activation for affective words compared with neutral words in the pilot study" (Kiehl et al., 2001, p. 678). The stage is set, the actors aligned, and the reader anticipates a successful study based on the convincing array of evidence and support from other esteemed researchers. Through *captation* (Latour, 1987, p. 56), the authors have set up a logical article, where the reader is already convinced based on all of the allies the text has mobilized.

Psychopathy: a Neuro Construct Buttressed by a Fortress of Organized Allies

In this section we detail information on how the Kiehl (2001) authors build a scientific fact around psychopathy, affective processing, and neuroscience by mobilizing a multitude of heterogeneous actors. Here we show how psychopathy as a neuroscientific fact is embedded in multiple instruments and supported by many allies. We begin by showing how the Kiehl (2001) assertions are embedded in multiple instruments that are absent from the text, and thereby only accessible to those who look to outside sources for clarification. We then show how the Kiehl (2001) assertions are a product of many translations within the text that are incomplete by themselves, and rely on each other to make a whole. That is, we show how the claims about psychopathy are substantiated by a multilayered system of inscriptions.

Psychopathy is Embedded in Absent Instruments

Latour (1987, p. pp. 52-53) explains that authors are well aware of their readership, and actually target and entice them with the kinds of words they are using. By knowing who their readers are in advance, authors can anticipate their readers' objections in advance; they consider who their readers will be, what their likely objections are, and address them before the reader even gets a chance to read the text! This tactic is evident in the Kiehl (2001) article right at the opening of their 'methods and materials' section:

> All participants were free from any documented history of serious head injury (defined as a loss of consciousness for more than 1 hour) or psychotic illness (in self and first-degree relatives), were right-handed (Annett 1970), and spoke English as their first language. No participant met the DSM-IV criteria for substance abuse within the last 6 months. There were no group differences (criminal psychopaths: 5.5 (SD 3.2); criminal nonpsychopaths: 4.75 (SD 3.8) in the mean years of lifetime substance use (defined as self-reported use of any hard drug more than twice per week). There were no significant group differences in age (criminal psychopaths 33.9, SD 7.6; criminal nonpsychopaths 37.1, SD 7.1; controls 31.9, SD 8.4), parental socioeconomic status (based on the parental occupation section of the Hollingshead index of social position [Hollingshead and Redlich 1958]; criminal psychopaths 4.25, SD 1.4; criminal nonpsychopaths 4.25, SD 1.9; controls 3.1, SD 1.55), or IQ (measured with the National Adult Reading Test [Nelson and O'Connell 1978; Sharpe and O'Carroll 1991]; criminal psychopaths 111.2, SD 7.5; criminal nonpsychopaths 115.5, SD 5.9; controls 108.9, SD 11.5; and Quick Tests [Ammons and Ammons 1962];

> criminal psychopaths 102.7, SD 9.9; criminal nonpsychopaths 108.0, SD 5.86; controls 109.6, SD 17.5). (Kiehl et al., 2001, pp. 678-679)

Here we see multiple examples of the authors anticipating objections, and providing assurances of the comparability between groups on all dimensions except for the independent variables (i.e. psychopathy and criminality) before their readers have the opportunity to contest.

The Kiehl (2001) authors pre-emptively considered possible attacks from their privy readers, and resisted them before they could even come to formation. Knowing that the presence of confounding variables is a common critique in studies of this sort, the Kiehl (2001) researchers resisted such critique in advance; substance use, age, socioeconomic status, and intelligence levels were all controlled for in each participant group. What is noteworthy in the above quotation is again the evidence of the collective fate of fact-making. The researchers don't only anticipate and respond to potential dissent, they also recruit allies to support and lend strength to their defence. The Kiehl (2001) authors mobilize the DSM-IV, and consequently the American Psychological Association, the Hollingshead index of social position (Hollingshead & Redlich, 1958), the National Adult Reading Test (Nelson and O'Connell 1978; Sharpe and O'Carroll 1991), and Quick tests (Ammons & Ammons, 1962). The authors do not ask the reader to trust them, they divert the reader to well established measures of the potential confounding variables.

This technique does two important things to fortify the Kiehl (2001) article. First, consider how some of the above noted citations have an impressive history. The references to instruments enrolled by the Kiehl (2001) authors have been used/ cited in a multitude of other articles, and the citations are predominately positive modalizations where the instruments are accepted and used by the citing authors (e.g. Hollingshead and Redlich (1958) have been cited over 8000 times, Nelson and O'Connell (1978) have been cited over 1000 times). The authors embed their claims in well-established truths that are decades old, and so refuting their methods would require questioning the methods of thousands of researchers across decades of studies. Second, by linking themselves to referenced information that is absent from the article, the reader is forced to look outside the text and read other articles (Latour, 1987, p. 46). The reader would then likely be redirected again, as the cited articles also support their claims with a mass

of citations. Thus, a reader is sent into a multilayered maze of citations if they wish to question the Kiehl study's methods. Frankly, it is an unreasonable task (and beyond the scope and resources of this study) to trace each citation back in time across generations of articles to detail the essence of what makes up each truth for each of the 51 citations in the Kiehl (2001) article (Latour, 1987, p. 33).

We see much of the same as we move through the 'methods and materials' section of the Kiehl (2001) article. As was already discussed, the authors mobilize other actors including the PCL-R (Hare, 1991), the 'Handbook of Semantic Norms' (Toglia & Battig, 1978), SPM and other fMRI analysis techniques (Friston et al., 1999abc). Additionally, Latour (1987, p. 43) explains how over time, through a series of positive modalities, references to especially prolific facts become redundant, they become *tacit knowledge*. To explain this phenomenon, Latour (1987, p.43) refers to the fact that nobody cites Lavoisier when they refer to water as a molecule of 3 atoms, two hydrogen and one oxygen (H2O). In the Kiehl (2001) methods and materials section we see the same phenomenon, a fact so well accepted and understood in scientific texts that a reference is unnecessary. What we are speaking of is the Analysis of Variance (ANOVA)- the researchers describe how "difference images were entered into a random-effects one-way (three groups) ANOVA" (Kiehl et al., 2001, p. 680). The ANOVA is considered tacit knowledge, universally accepted, and so citing the statistician and evolutionary biologist, Ronald Fisher, who developed the model, would be redundant. This is an important idea to consider in the Kiehl (2001) article, and scientific claims in general. We have universally accepted some statistical testing- probabilities with specific confidence intervals, so much so that we have unquestionably mechanized the process into our methods for fact-building. To question the fact would require questioning the universally accepted and tested instrument that the fact is embedded in. In Building facts by embedding them within black boxed instruments such as the ANOVA, the Kiehl (2001) researchers lend strength and legitimacy to their assertions through association. Again, this is an example of how the reality of psychopathy as a neuroscientific construct is built through "social" work (i.e. association); the Kiehl (2001) assertions are concretized and made factual through the strength gained from the enrolled black boxes. Psychopathy as a neuroscience construct is dependent on the building blocks it uses (i.e. the

instruments). Its facticity is given its strength through the strength of the heterogeneous entities it mobilizes.

Psychopathy Pieced Together Through a Series of Incomplete Translations

Now we have considered in our analysis how the Kiehl (2001) article refers to articles and instruments that are absent from the text itself. However, Latour (1987, p. 46) brings our attention to references that are present within the text itself. There are several instances in the Kiehl (2001) article where the readers' attention is directed to tables or figures present elsewhere within the text. There are two tables, and one figure included in the Kiehl (2001) article. Table one is alluded to three times in the text. First the authors' claim that the affective memory task "revealed that affective stimuli elicit greater activation than do neutral stimuli in both limbic and neocortical brain regions, including the amygdala, hippocampal formation, and temporal and frontal cortex (see Table 1) (Kiehl et al., 1998)" (Kiehl et al., 2001, p. 678). Table 1 is then referred to again in explaining how the 10 brain regions of interest were identified in a pilot study, "the exact coordinates for the site of most significant activation in each of these regions are listed in Table 1" (Kiehl et al., 2001, p. 680). Directing the reader to Table 1 is how the authors support their statement with its source. The authors acknowledge that they are speaking for the data, translating it into a statement, and so they provide the reader with what they are referring to. The authors do not ask the reader to believe blindly in their word, they let the reader see for themselves what their statement consists of (Latour, 1987, pp. 47-48). However, Table 1 alone is not transparent enough for the reader, the authors have to add another layer of information, the legend on how to read the Table:

Table 1. Summary of the Results of the Functional Imaging Data for the Affective Memory Task

Regions of Interest	Talairach Coordinates x	y	z	Pilot study z score	Control vs Psychopath Fixed Effects t; e	Control vs Psychopath Random Effects t; e	Nonpsychopath vs Psychopath Fixed Effects t; e	Nonpsychopath vs Psychopath Random Effects t; e
Frontal lobe								
1. Rostral Anterior Cingulate	0	38	8	4.55	8.20; 27	3.92; 27	7.64; 6	3.61; 11
2. Caudal Anterior Cingulate	−8	22	20	5.35	11.85; 27	3.76; 27	7.30; 27	2.18; 7
3. L Inferior Frontal Gyrus Parietal Lobe	−38	41	−8	7.38	6.12; 18	3.41; 27	7.69; 17	2.67; 15
4. Posterior Cingulate Gyrus	−8	−38	16	8.30	12.56; 27	2.71; 27	11.19; 27	2.20; 27
Temporal Lobe								
5. R Amygdala/Hippocampus	34	−12	−20	7.57	9.22; 27	2.03; 6	4.15[a]	2.85; 7
6. L Amygdala/Hippocampus	−19	4	−24	4.30	7.48; 2	1.83; 3	11.06; 6	ns
7. L Parahippocampus	−38	−26	−20	7.37	11.01; 12	4.20; 27	7.63; 5	ns
8. R Anterior Superior Temporal Gyrus	49	19	−24	6.85	9.19; 18	1.94; 10	ns	ns
9. L Anterior Superior Temporal Gyrus	−49	12	−32	6.55	5.56; 2	1.97; 2	ns	ns
10. Ventral Striatum	4	−8	−8	5.45	7.95; 27	3.22; 27	5.13; 2	2.13; 3

Regions of interest, Talairach coordinates (x, y, and z), and z scores from the pilot study of healthy controls are listed in columns 2, 3, and 4, respectively. All z scores from the pilot study are statistically significant after correction for number of effective comparisons. The remaining columns contain the results of the fixed-effects and random-effects analyses for the comparisons of the difference images (all emotional phases minus neutral phases) where psychopaths showed less affect-related activity than did healthy controls (control vs psychopath; columns 4 and 5) and criminal nonpsychopaths (nonpsychopath vs psychopath; columns 6 and 7). The degrees of freedom associated with the fixed-effects analyses are 7872.

t values associated with the random effects analyses with 21 degrees of freedom of 1.72, 2.08, 2.52, 2.83, and 3.82 correspond to one-tailed probability levels of .05, .025, .01, .005 and .0005, respectively. t, t value; e, spatial extent of activation (i.e., the number of significant voxels contiguous with the peak voxel in the region of interest); ns, nonsignificant; L, left; R, right.

[a] All t values reported for the fixed effects analyses have a probability level less than .001 corrected for multiple comparisons, except the value for the right amygdala/hippocampus for the comparison of criminal nonpsychopaths with psychopathic criminals where p = 0.1 after correction.

(Kiehl et al., 2001, p.678)

The reader is now navigating a multitiered system, being diverted from the original statement, to a table that the statement represents. However, the table is not transparent or talkative enough on its own, it must be explained by the legend below. The authors once again must act as a spokesperson to tell the reader how the table represents their results. Then, if the reader wishes to understand how the results were produced, they are directed outside of the text to a pilot study (Kiehl et al., 1998). The reader cannot simply read through the text, rather, they are forced to navigate a multitiered system of internal and external references if they wish to understand the technical details of the study (Latour, 1987, p. 48).

Again, the third and final time table 1 is referred to in the Kiehl (2001) article shows how stratified their text is, and how the reader must navigate the linkages between instruments, figures, tables, and texts (Latour, 1987, p. 49). “Criminal psychopaths also showed less affect-related activity than did non-criminal controls in the left amygdala and parahippocampal gyrus, and bilateral anterior superior temporal gyrus (see Table 1 and Figure 1)” (Kiehl et al., 2001, p. 681). The reader is first directed to Table 1 to look at the data, then to Figure 1 to see a 3D image that displays how criminal psychopaths showed less affect-related activity than non-criminals. Figure 1 cannot be interpreted on its own, so it is accompanied by a legend explaining to the reader what they are looking at. Part of Figure 1’s legend refers to “Talairach coordinates… listed in Table 1” (Kiehl et al., 2001, p. 681), and so the reader is directed back to Table 1. Table 1 has columns with the Talairach coordinated, and table 1’s legend explains that

said coordinates were attained from the Kiehl et al. (1998) pilot study. The reader returns to the legend for Figure 1, and is told that the bottom panels of the figure show how psychopaths showed greater activity in the orange illuminated areas during the affective task than the non-criminal and non-psychopathic criminal participants. The legend then explains that the orange illuminated regions on the Figure 1 image include the bilateral inferior frontal gyrus, but the reader should "see text for details" (Kiehl et al., 2001, p. 681).

In analyzing the original statement (i.e. "Criminal psychopaths also showed less affect-related activity than did non-criminal controls in the left amygdala and parahippocampal gyrus, and bilateral anterior superior temporal gyrus (see Table 1 and Figure 1)" (Kiehl et al., 2001, p. 681), we can see how stratified the Kiehl (2001) text truly is. The number of redirections to other layers of the text is astounding: from Table 1 to Figure 1, from Figure 1 to the Figure 1 legend, from Figure 1 legend back to Table 1, from Table 1 to the Table 1 legend, from the Table 1 legend to the Kiehl et al. (1998) pilot study, then back to Figure 1 legend, and finally from the Figure 1 legend back to the text for details. All of these folded and stratified layers supporting each other, scattered across pages within and outside of the article, creates a thicket, the reader cannot get through an in-depth analysis of the article without strenuous effort (Latour, 1987, p. 49). This is a telltale sign of technical scientific writing: the information is not presented in the form of linear prose, but rather folded into an array of defence lines that are difficult to read and interpret (Latour, 1987, pp. 46- 48). This maze of translations clearly illustrates the social work that is necessary to construct nature and build a fact. The Kiehl (2001) researchers translate affect related neural activity so many times over that the qualitative nature of it is completely transformed. We went from actual neural activity to tables of statistics, to legends, back to tables, to figures, to legends of figures, back to tables etc. All of the translations are incomplete on their own, and require the reader to jump between the qualitatively different forms to piece together the product. The social work required to harness nature, transform it, and make it accessible is blatant; and conversely, the same amount of social work would be necessary for the reader to dissent and question the fact. This is another testament to our position that the more social the Kiehl (2001) facts are, the stronger they are, and the more they hold truth.

What is important to notice in all of this is how the authors observations and presentation of the results have been coded, and sifted for statistical significance; diagrams, tables, and legends support the statements they are represented by; no part of the paper stands by itself but each is linked by many references to other layers (Methods and Materials, Results, Discussion) (Latour, 1987, p. 49). Considering the depth of both external (absent) and internal (present within the text) references that are mobilized in the Kiehl (2001) article, we can see how their claims gain strength. They are buttressed by a well-organized system of supporters in the form of outside texts, figures, columns, tables, legends, graphs, and coded statistical results (Latour, 1987, p. 48). The statements presented in the Kiehl (2001) article are protected behind a fortress of allies, a dissenter is destined to get lost in the endless maze of stratified references that constantly divert them to other sections within the text, or other articles outside of it. Our analysis has shown how the Kiehl (2001) article builds facts by creating a system where endless new links tie instruments, figures, texts, and statements together. Psychopathy as a neuroscientific construct is built by the instruments it is embedded in, and is a final product of many incomplete and coupled translations.

Transforming Potentialities into Certainties, and Building Psychopathy as a Neuro Fact through Logic

In this section of the chapter we detail how the Kiehl (2001) authors present the results of their experiment. Here we show how psychopathy is built as a neuroscientific construct through three man techniques. First we explain how a series of previous assertions are mobilized and organized into a logical way that substantiates the Kiehl (2001) results. Then we discuss how those same assertions are translated from exploratory articles into confirmatory statements. Finally, we explain how inductive reasoning is used to substantiate the Kiehl (2001) assertions.

Psychopathy Logically Coordinated through a Series of Truths

Like the techniques used in the introduction of the Kiehl (2001) article, in the ‘discussion’ the authors carefully coordinate their citations to act as premises and supports for their own results. That is, they logically connect the interpretation of their own results with previous research that both coincides and compliments their own assertions. For example,

> The amydgala, ventral striatum, and hippocampal formation typically are associated with processes related to emotion and memory (Adolphs et al 1998; Bechara et al 1999a; Irwin et al 1996). In particular, studies have shown that the amydgala is likely to be involved in processes related to fear conditioning (LaBar et al 1995). Numerous studies have shown that psychopaths are insensitive to several types of fear and punishment contingencies (Hare 1965; Hare 1968; Hare 1982; Hare et al 1978; Hare and Quinn 1971; Patrick et al 1994). In addition, Patrick and colleagues have shown that criminal psychopaths do not show the same pattern of startle potentiation during viewing of negatively valenced stimuli as do nonpsychopathic criminals and healthy control participants (Patrick et al 1993; Levenston et al 2000). There is a large body of animal research indicating that startle potentiation to negatively valenced stimuli is mediated by circuits in the Limbic system, in particular, circuits in the amygdala (Patrick et al 1994). Taken together, our findings suggest that the neural systems associated with attentional processing of affective stimuli at both the limbic and paralimbic level are abnormal in criminal psychopaths. (Kiehl et al., 2001, p. 682)

When we break down the above quotation the new assertion being made is that "neural systems associated with attentional processing of affective stimuli at limbic and paralimbic levels are abnormal in criminal psychopaths" (Kiehl et al., 2001, p. 682). The Kiehl (2001) authors substantiate this assertion with an unquestionable, logical, and well-articulated series of citations. The premises for the above noted assertion, which are also present in the above noted quotation, are as follows:

1. Amygdala ventral striatum, and hippocampal formation are typically associated with emotion and memory processes (Adolphs et al., 1998; Bechara et al., 1999a; Irwin et al., 1996)
2. The amygdala is likely involved in fear conditioning processes (LaBar et al., 1995)
3. Psychopaths are insensitive to fear and punishment (Hare 1965, 1968, 1982; Hare, Frazelle, & Cox, 1978; Hare & Quinn, 1971; Patrick et al., 1994)
4. Psychopaths have reduced startle potentiation compared to nonpsychopaths when viewing negative stimuli (Patrick, Cuthbert, & Lang, 1993; Levenston et al, 2000)
5. Animal research shows that startle potentiation to negative stimuli is mediated by limbic system circuits, particularly the amygdala (Patrick, Bradley, & Lang, 1994)
6. The findings from the Kiehl (2001) study itself

Considering the above, statement (1) immediately connects to the new Kiehl (2001) assertion by identifying the same structures they did as being involved in affective processing. Statement (2) connects the amygdala to fear, and statement (3) connects fear deficits to psychopaths, thereby insinuating that psychopaths' fear deficit can be attributed to a dysfunctional amygdala. Then statement (4) states that psychopaths have a startle response deficit to negative stimuli, and (5) animal studies have shown that the startle response is mediated by the amygdala, thereby insinuating that the psychopaths' startle response deficit can also be attributed to a dysfunctional amygdala. Finally, statement (6) explains how all of the above supports and coincides with the Kiehl (2001) findings, and so deficits in affective processing in psychopaths is attributable to limbic and para limbic abnormalities.

Similar to the techniques used in the introduction paragraphs, the above quotation carefully coordinates its allies to support their proposition. However, while the introduction used a series of premises to justify and create a need for the the Kiehl (2001) study, the above quotation (and the other paragraphs in the 'discussion') uses the premises to prove their point and strengthen the interpretation of their results. The Kiehl (2001) 'discussion' also includes a paragraph that uses the same citation-based premises process to support their conclusions on the cingulate cortex (i.e. "combined with the results of the present data, these results suggest that some aspects of psychopathic behaviour [*sic*] may be related to abnormal function in the cingulate cortex" [Kiehl et al., 2001, p. 682]). Finally, the Kiehl (2001) article also supports their findings (and the interpretation of their findings) that criminal psychopaths showed greater activation for affective than neutral stimuli in some brain regions by linking their results with other complimentary studies.

Exploratory to Confirmatory- Silencing the Limitations of Corroborating Evidence

An evident technique used by the Kiehl (2001) authors in the above quotation, and the paragraphs we just alluded to on the cingulate cortex and increased affect-related activity, is *Stylisation.* That is, all of the studies cited and positively modalized by the Kiehl (2001) authors are transformed from their original substance into one sentence. The details of all the cited

articles are lost in translation (Latour, 1987, p. 42). When we followed the citations (i.e. looked up the cited articles), we found that they were all exploratory, not confirmatory studies, just like this Kiehl (2001) article. They all have stated limitations, and provide evidence to support a notion, not confirm it. Yet this Kiehl (2001) article positively modalizes them, gives them strength that they did not have on their own, and stylises the cited texts so that they are "turned into a one-line long statement with only one simplified positive modality: 'X (the author) has shown that Y.'" (Latour, 1987, p. 42).

Again, we must stress that we are not criticizing the Kiehl (2001) authors' citation methods. What we have described above is commonplace to most scientific texts. *Stylisation* is a product of technical writing. In writing their article, the Kiehl (2001) authors could not possibly fit in a detailed explanation or justification of how they modalized and stylised each citation. The result then, is the closing of black boxes via their stylized positive modalizations; they cite uncertain and exploratory studies and transform such potentialities into certainties. The result of this technical writing is that the possibility for uncertainty and controversy is quashed; the conditions from which the assertions were generated are lost when the Kiehl (2001) authors positively modalize the citations within their own text. The Kiehl (2001) authors, exercised caution, stated their study's limitations, and stated that their study is exploratory not confirmatory. However, despite the stated limitations of their own research, mobilizing other articles via positive modalizations results in those cited assertions being black boxed. Considering, as was previously mentioned, that the Kiehl (2001) article constantly cites previous works by the authors, this black boxing effect promotes their global proposition that psychopathy is a concrete construct. Alternatively, considering that the research is presented by the authors as exploratory, a reader can also infer that their own reasoning and justifications made through citations are also exploratory. That is, by stating their studies exploratory nature, the authors could crutch on the notion that their positive modalizations are also exploratory, and so they are not closing black boxes or quashing controversy. This kind of technical writing offers both protection from dissenters' claims of overzealousness, as well as self-promotion through the proposition that their previous works can be mobilized as black boxes. Again, we see how the social work involved is what lends strength to the facticity of the Kiehl (2001) assertions;

stylisation, coupled with careful wording (i.e. exploratory study), allows for the authors to safely "prove as much as [they] can with as little as [they] can" (Latour, 1987, p. 51).

Psychopathy, a Result of Inductive Reasoning

Following from Latour's explanation of how researchers must "prove as much as [they] can with as little as [they] can considering the circumstances. If [they] are too timid, [their] paper will be lost, as it will if [they] are too audacious" (Latour, 1987, p. 51). We detail a technique used by the Kiehl (2001) authors to accomplish this calculated use of a study's resources. The technique is a *positioning tactic* called *stacking* (see the first section of this chapter for a detailed explanation of the stacking technique). The final statement of the Kiehl (2001) article is as follows:

> In summary, we have shown that processing of affective stimuli is associated with less limbic activation in criminal psychopaths than in criminal nonpsychopaths and noncriminal control participants. We have also shown that psychopathic offenders appear to use alternative neural systems to process affective stimuli. These findings support and extend previous lesion-based observations in psychopaths and provide in vivo visualization of the neural processes that may underlie the affective anomalies that clinicians have described in criminal psychopaths. (Kiehl et al., 2001, p. 683)

This closing statement is the most audacious the authors are throughout the entire paper. By presenting their findings in this way the use of inductive reasoning is evident. Throughout the entire article the details that support the quotation above have exercised varying degrees of timidity and caution. However, at the end of the article, the authors summarize their findings in such a way where all of the conditions that make up their assertion, and all of their cautions exercised are abandoned. The authors certainly followed the rules of inductive stacking; they did not stack two layers on top of one another, go straight from the first to the last layer, and asserted as much as they could, considering the circumstances of the study.

Consider how many instances of inductive reasoning we have in relation to the final summative statement quoted above:

-Words rated low on a pleasant scale from the Handbook of Semantic Norms→ all affective linguistic stimuli→ all affective stimuli
- varying degrees of statistically significant reduced activation in multiple limbic and non-limbic regions of the psychopathic brain→ less limbic activation
- 10 criminal psychopaths→ all criminal psychopaths
- 10 criminal nonpsychopaths→ all criminal nonpsychopaths
- 10 noncriminal control participants→ all noncriminal nonpsychopaths
- exploratory statistical analysis showed greater activation in multiple nonlimbic brain regions of participating psychopaths than the other two participating groups→ psychopaths use alternative systems to process affective stimuli→ psychopaths make up for affective deficits by relying on cognitive resources to process affective stimuli.

We fully acknowledge that the Kiehl (2001) article was humble in the presentation of their findings throughout the 'discussion' section. All of the instances of induction were actually discussed by the authors in regards to the potential limitations of their study. When reading through the Kiehl (2001) article, we see a balanced layering/ blending of the steps toward induction; at some points they refer to their sample, and at others to the population their sample represents; at some points they discuss neuronal structures individually with regards to the varying degrees of statistical significance of their results, and at others the discuss how they have shown decreased neural activity in psychopaths' limbic system; at some points they are humble in discussing their limitations, and at others they are more forward and pronounced in their findings. Despite the clear transparency in discussing the extent of their findings, the final statement focused on the biggest claims the authors could make from their study, and lacked any word of caution. Absolutely, the authors are protected against any critique of being overzealous in their conclusions, as the article does indeed include a humble presentation of their limitations, and the exploratory nature of the study. However, the article concludes with a less humble summarizing paragraph, and so they clearly would not refute being cited in such a fashion in future articles. And actually, this is the next step forward with inductive reasoning, being positively modalized by later texts, where the conditions of production, and the cautions and limitations provided by the Kiehl (2001) authors are lost. Only the sweeping summary provided at the end of the entire text is recognized and used. This is the topic of the next chapter, the point

in the Kiehl (2001) article's career where its fate no longer lies in its authors' hands, but in the hands of other researchers!

DOWNSTREAM ANALYSIS- HOW ACADEMIC WRITERS USE KIEHL (2001) TO BUILD PSYCHOPATHY AS A NEURO FACT THROUGH TECHNICAL WRITING

We have recognized the brilliantly articulated technical writing techniques that have helped strengthen and fortify the Kiehl (2001) article's assertions. We have indeed given credit to the researchers' skilled use of resources in both the laboratory and the corresponding text. They have done everything in their power to create a well-defended set of assertions. Those assertions being, "processing of affective stimuli is associated with less limbic activation in criminal psychopaths" and "psychopathic offenders appear to use alternative neural systems to process affective stimuli" (Kiehl et al., 2001, p. 683). However, once the article is published, the transformation from statement to fact no longer lies in the hands of the authors. It lies in the hands of others, who decide the fate of the above noted Kiehl (2001) statements by mobilizing them.

To date, the Scopus database notes that the Kiehl (2001) article has been cited in 470 other documents. This is the highest number of times a neuroscience of psychopathy article written by a research team that includes Robert Hare has ever been cited, which justified our decision to use the Kiehl (2001) article as our research material. This is an assuring sign that the Kiehl (2001) authors' statements have been translated into facts (Latour, 1987). In this chapter we analyze how the Kiehl (2001) statements have been translated into facts, the processes involved, and the transformative effects of the process.

Exploratory to Confirmatory

"The status of a statement depends on later statements" (Latour, 1987, p. 27). The Kiehl (2001) authors may have exercised caution, stated their limitations, and explained that their research is exploratory. However, once published, it is not for the authors to decide whether or not their results are a confirmation that psychopaths have reduced limbic activation when processing affective stimuli. It is for the authors who cite the Kiehl (2001) article to decide what their study explores or confirms. Remember how we mentioned that the end of the Kiehl (2001) article summarizes their findings with a less than cautionary or exploratory tone? Well the vast

majority of articles that cite the Kiehl (2001) article positively modalize the study, reiterate the summarized conclusion, and most importantly, ignore the stated limitations and exploratory nature of the study. For example,

> "In addition, they [psychopaths] showed reduced amygdala activation, relative to comparison individuals, during an emotional memory task (Kiehl et al., 2001)." (Blair et al., 2002, p. 685)

> "Interestingly, two recent neuroimaging studies have **confirmed** that amygdala dysfunction is associated with psychopathy (Tiihonen et al, 2000; Kiehl et al, 2001)." (Blair, 2003, p. 5 [emphasis added])

> "Corroborating this hypothesis, previous findings in pathologies associated with increased aggressive behavior (e.g., borderline personality disorder, psychopathy) revealed decreased functional activity and recued [sic] anatomical volume in dACC (Kiehl et al. 2001; van Elst et al. 2003; Birbaumer et al. 2005; Whittle et al. 2006; Enzi et al. 2013)." (Clemens et al., 2015, p. 1778)

> "Amygdala hypoactivation has been linked with psychopathy (Kiehl et al., 2001)" (Mier et al., 2014, p. 201)

> "Indeed, the amygdala has been shown to be a key player in mental and emotional health, with abnormal amygdala function identified in depression, anxiety, posttraumatic stress disorder, phobias, and panic disorders (Siegle et al., 2004; Siegle et al., 2007; Kiehl et al., 2001; Mervaala et al., 2000; Phan et al., 2006)" (Taren et al., 2013, p. 1)

> "Several functional neuroimaging studies have implicated the OFC (e.g., Birbaumer et al., 2005; Harenski, Harenski, Shane, & Kiehl, 2010) and ACC (e.g., Birbaumer et al., 2005; Kiehl et al., 2001; Muller et al., 2003) as being dysfunctional in psychopathy… Furthermore, across a range of cognitive and emotional tasks, functional neuroimaging has frequently identified these regions as abnormal in psychopathy: parahippocampal

> gyrus (Muller et al., 2003; Kiehl et al., 2001), amygdala (Harenski et al., 2010; Glenn et al., 2009; Birbaumer et al., 2005; Muller et al., 2003; Kiehl et al., 2001), hippocampus (Kiehl et al., 2001), and anterior temporal cortex (Harenski et al., 2010; Muller et al., 2003; Kiehl et al., 2001; Kiehl et al., 2004) … The PCC is important in emotional and moral processing and judgment (Kiehl et al., 2001; Glenn et al., 2009; Muller et al., 2003; Greene et al., 2004)." (Ermer, Cope, Calhoun, Nayalakanti, & Kiehl, 2012, pp. 650-656)

The six examples above are just a sample from the majority of the 470 documents that cite and positively modalize the Kiehl (2001) article. Through such positive modalizations, the Kiehl (2001) article is transformed from an exploratory study, to a confirmatory one. The limitations are ignored in both the citation itself, and the context surrounding the citation. Similar to how the Kiehl (2001) authors positively modalized the citations in their article, their assertions have been stylised, eroded, and used as premises to support another articles' assertions. The Kiehl (2001) assertions have been taken up and mobilized as facts, and thereby used to support the next generation of assertions.

Looking at the sixth example provided above, Kent A. Kiehl himself was one of the authors who positively modalized, and presented as confirmed evidence, what he had previously stated was exploratory 11 years prior. Remember how we saw the self-referential process in the Kiehl (2001) article, whereby the authors highlighted and strengthened their earlier work? Well fast forward 11 years from when the Kiehl (2001) article was published. We see a continuation of the process, as Kiehl participates in the transforming of his exploratory research into a confirmatory premise for his new research. What caused the change in tone? We can only speculate. Perhaps it was the plethora of positive feedback; the authors who supported, positively modalized, and put faith in the Kiehl (2001) assertions gave him confidence to do the same.

Also, looking at the fifth example provided above, we see how the Kiehl (2001) results were not only transformed from exploratory to confirmatory, but also generalized to a more inclusive population. In the fifth example presented above there is not clear connection to psychopathy, affective processing, or even any of the psychopathy symptoms or characteristics.

It must be assumed that the link between Kiehl (2001) and the fifth example provided above is 'emotional health'. Through the Taren et al., (2013) article, the Kiehl (2001) article transcends its pertinence to psychopathy, and is used as evidence for the amygdala's role in all emotional health.

From the examples above, we have shown how the Kiehl (2001) article's fate was decided by others. The collective fate of fact making (Latour, 1987, p. 26) is apparent with the Kiehl (2001) article. Their exploratory study was convincing, fit with other propositions, and was carried upstream where the collection of articles that borrowed from it stylised and eroded it into a confirmatory fact about psychopathy, affective processing, and the brain. However, a transition from exploratory to confirmatory is not the only process involved in rendering the Kiehl (2001) article as factual. Returning to the fifth example above, this broadened relevance of the Kiehl (2001) article shows us another process at work that strengthens and supports its rendition as factual. By connecting with neighbouring disciplines, the Kiehl (2011) article is able to gain strength from them.

The Neuroscience of Psychopathy becomes Relevant to, and Mobilized by Neighbouring Disciplines

The Kiehl (2001) article has indeed had a fulfilled career with its use in 470 publications, more than any other article on psychopathy and neuroscience. As one would expect, the Kiehl (2001) article, and the team of researchers' other works are well cited in the milieu of criminal psychopathy, and more precisely, criminal psychopathy and neuroscience. However, our analysis has revealed that the Kiehl (2001) article has been mobilized by authors in neighbouring disciplines, and has thereby broken the border of its topic, criminal psychopathy. The Kiehl (2001) article has been mobilized in many articles on a variety of topics and populations, including:

- people with narcissistic personality disorder (Baskin-Sommers et al., 2014; Ritter et al., 2011),
- people with borderline personality disorder (Buchheim et al., 2013),

- forensic patients (Müller et al., 2003),
- linking childhood diversity, glucocorticoid regulation, and later mental disorder (Struber et al., 2014),
- community participants with different degrees of psychopathy, from none to severe (Glenn, Raine, & Schug, 2009; Glenn, Raine, Schug, Young, & Hauser, 2009),
- children with psychopathic tendencies (Budhani & Blair, 2005),
- cognition, affect, and error-monitoring (Edwards, Calhoun, & Kiehl, 2012),
- people identified as suffering from antisocial personality disorder (Del-Ben, 2005),
- mindfulness and emotional health (Taren et al., 2013),
- un-medicated patients with major depressive disorders marked by anger attacks (Dougherty et al., 2004),
- a delusional patient who killed her children (Kalbe, Brand, Thiel, Kessler, & Markowitsch, 2008),
- cocaine-dependent men and women (Li, Kosten, & Sinha, 2006),
- behavioural genetics (Tancredi, 2009),
- aggressive or violent behaviors in general (Bobes, et al., 2013; Campbell & Eastman, 2012; Coccaro et al., 2011; Siever, 2008; Smith et al., 2016),
- untreated bipolar patients (Sassi et al., 2004),
- criminal responsibility (Farmer, 2011; Kinscherff, 2010),
- people with dementia (Cipriani et al., 2013),
- alcoholism and affective processing (Marinkovic et al., 2009),
- infants, children and adolescents with behavioural problems and associated DSM disorders (Fahim et al., 2011; Marsh et al., 2013; May & Beaver, 2014; Sarkar et al., 2013; Sterzer & Stadler, 2009; Viding & Jones, 2008),
- females with psychopathic traits (Cope et al., 2014),
- people with schizophrenia (Mier et al., 2014; Hoptman & Antonius, 2011; Tikasz et al., 2016),
- attention Deficit/ Hyperactivity disorder (Rubia, 2011),
- lie-detection techniques (Wolpe et al., 2005)

The Kiehl (2001) study did not mention any of the above noted topics, or refer to any of the above noted populations, yet their assertions have been used to strengthen others' assertions within those fields. In the articles referred to above, the authors positively modalized, and mobilized the Kiehl (2001) article as either a logical premise to their own assertions, or a fact that could elucidate information on their own topics by way of resemblance. By increasing the amount and variety of applications of the Kiehl (2001) article, the assertions within it gain strength in numbers. Now if anyone were to question or attempt to discredit their results, they are supported with allies that span far beyond the borders of criminal psychopathy. If the Kiehl (2001) article seemed fortified by being embedded with instruments and allies before it was taken downstream by other authors, look at it now. Now its fortification includes its upstream references, downstream citations, and their corresponding fields and populations. The facticity of assertions is built by other researchers that cite and refer to them. It is lucrative to diversify the portfolios of colleagues who participate in constructing a fact. Through this downstream activity, psychopathy as a neuroscientific fact is embedded within other fields, and can thereby borrow strength from those who borrow from it; psychopathy gains strength from both numbers and diversity. Defining the construct through neuroscience allows psychopathy as a boundary object to fit with/ adapt to a much greater variety of topics, and thereby gain strength as a fact.

Neural Interconnectivity and Multiplicity: A Resource and Source of Confusion for Psychopathy Research

In this subsection, we explain how the complexity of the brain and researchers' unfamiliarity with its precise inner-workings actually serves as a resource for neuroscientists. First we explain what is meant by multiplicity and interconnectivity of the neural system. Then we discuss how the zooming in and out of the brain is a tool that allows more varied set of researcher articles to connect and further develop the factual status of psychopathy as a neuroscientific construct. We also discuss how zooming in and out of the brain has given the Kiehl (2001) article the ability to remain relevant despite changing positions in neuroscience. Finally, we discuss how the collective nature of fact-making has helped support the Kiehl (2001) article by producing complimentary studies that quash a Kiehl (2001) study limitation.

As was discussed in the literature review of this study, the brain can be characterized by interconnectivity and multiplicity of functions. That is, despite the common trend of isolating structures associated with specific functions, it is more informative to acknowledge that interconnectivity between multiple structures is required to perform a function collectively. Also, it is important to remember that any given structure performs multiple, often unrelated, functions (Glenn & Raine, 2014). For this reason, it is difficult for neuroscientists to prescribe specific functions to individual structures. Neuroscientists more commonly associate functions to clusters of interconnected structures, or neural pathways that cross through multiple structures. For example, the limbic system is composed of many interrelated structures, but the dopaminergic pathway that is also composed of interrelated structures (and performs different functions) includes some limbic structures and not others. The borders, relatedness, and functions of structures is not clear. Rather, it is a maze, a thick web of connections and functions that is extremely difficult to delineate. When multiple structures are active and involved in a function, it is difficult to pinpoint where, or what structure(s) is responsible for what parts of the function. Especially when the function is complex and multidimensional (e.g. memory, empathy, affective processing, moral judgment, decision-making, etc.). When abnormalities occur, like psychopathy, it is difficult to attribute the affective/behavioural abnormalities to a specific neural source(s). When locating an abnormality in the brain, there are many possibilities: are the identified structures abnormal? Is the communication between them abnormal? Is one structural abnormality causing disruption across the whole interrelated circuit of structures, thereby making it look as if all structures are abnormal, when in reality the source is singular? These are questions that neuroscientists are confronted with to this day (Glenn & Raine, 2014), and is indeed a source of confusion among researchers.

This source of confusion has also proven to be a resource for the career of the Kiehl (2001) article. We have found that this fluidity of regional borders, and the interchangeability of attributing specific neural structures or entire regions to functions, is utilized by the Kiehl (2001) article and the articles that cite it. Consider how the Kiehl (2001) article is varied in its presentation of results, at one point they state that “compared with noncriminals, criminal psychopaths showed less affect-related activity during encoding and rehearsal in the right amygdala and bilateral anterior superior temporal gyrus” (Kiehl et al., 2001, p. 681). Then later

in the text they reassert, and partially translate their findings, by encapsulating the individual structures under the limbic system: “In summary, we have shown that processing of affective stimuli is associated with less limbic activation in criminal psychopaths than in criminal nonpsychopaths and noncriminal control participants” (Kiehl et al., 2001, p. 683). This flexibility of the study’s results has allowed for the Kiehl (2001) article to increase its applicability, and associate with more texts than it would with a more rigid presentation of the results. For example, some downstream articles (i.e. those that cite Kiehl et al., 2001) found significant differences in structural activation in psychopaths compared to controls that included structures unmentioned in the Kiehl (2001) results. However, when they cite the Kiehl (2001) article, they align their results by stating that both they and Kiehl (2001) found that the limbic/ paralimbic networks are implicated in psychopathy (e.g. Cope et al., 2013; Juarez et al., 2013). Other downstream articles cite the Kiehl (2001) article and align their results by alluding to specific structures that both studies found to be abnormal in psychopaths. For example, some texts that found significant abnormalities in the amygdala of psychopaths connect with the Kiehl (2001) article’s finding that psychopaths have reduced amygdala activity when processing affective stimuli. However, these citing articles fail to mention the other structures/ systems included in the Kiehl (2001) results (e.g. Gopal et al., 2012; Osumi et al., 2012). This zooming in and out of the brain when presenting results has made the Kiehl (2001) article more applicable to other articles, as it is able to connect with studies that allude to specific structures, as well as studies that allude to the entire limbic system. Even when the results do not perfectly align in terms of structural abnormalities in psychopaths, so long as both the Kiehl (2001) study and the study citing it include limbic structures, their results can be said to align. When studies focus solely on one neural structure, the Kiehl (2001) study is still applicable, because it also isolates structures in one version of their results. This has allowed for the Kiehl (2001) article to be taken up by more authors downstream, and thereby further solidify its status as factual.

Another important aspect of this zooming in and out effect, is how it has protected the Kiehl (2001) article from one controversial assertion. Shortly after the Kiehl (2001) article was published, a group of prominent neuroscientists asserted that the limbic system does not serve a specific function or have a discrete unity in the brain, and so neuroscientists should abandon the concept all together (LeDoux, 2003). This controversy could have very well threatened the life

and career of the Kiehl (2001) article, as the final presentation of their results refers to the limbic abnormalities in psychopaths. However, despite this conflict, the article was able to survive. Because the Kiehl (2001) article also presented their results with reference to individual structures that are considered to be part of the limbic system. Kiehl (2001) was able to rely on this version of the results when the other version was subjected to controversy. As was mentioned above, the Kiehl (2001) article was often cited in subsequent studies with reference to its findings pertaining to the amygdala (a structure that is said to be part of the limbic system), and not so much the limbic system. When the zoomed-out version (limbic system) of the results was subject to controversy, the zoomed-in version (amygdala) allowed the Kiehl (2001) article to maintain its facticity and resist becoming an artefact.

Finally, in the Kiehl (2001) article we saw how one of their stated limitations was in regards to the affective stimuli being restricted to verbal material. Considering the discussion pertaining to structural interconnectivity and multiplicity, this is an important detail of their study. They openly state that the observed effects could have been different with another form of affective material. We have observed another process involved in fact-building related to this Kiehl (2001) limitation. Another article cites and positively modalizes the Kiehl (2001) article, and also attempts to find the neural source of psychopaths' affective abnormalities. These authors conducted research on a similar sample (i.e. PCL-R psychopaths), and used a similar research design (i.e. control groups, fMRI, and affective task), but changed the affective stimuli to emotional facial expressions (Deeley et al., 2006). Additionally, there are many studies that correspond to the Kiehl (2001) article and use emotional facial expression stimuli, or 'witnessing others in pain' stimuli, to link the affective abnormalities associated with psychopathy to neural structures (e.g. Carre et al., 2013; Gordon et al., 2004; Hyde et al., 2014). These studies do not cite Kiehl (2001), but they still cite other works by Kent Kiehl and Robert Hare in their articles. By diversifying the type of affective stimuli, they are solidifying the fact that the neural structures/ regions identified as being responsible for the abnormal affective processing in psychopaths, are indeed responsible for psychopaths' affective abnormalities, and not just their processing of verbal material.

This is testament to the collective fate of fact-making. First, the citing authors either directly align themselves with the Kiehl (2001) paper by positively modalizing it in their citation, or they cite and positively modalize other well-aligned works by authors in the Kiehl (2001) article. The citing authors then attempt to build on the assertion by conducting a corroborating study that uses another type of affective stimuli, which can make up for the limitation of the Kiehl (2001) study. Authors who have supported the Kiehl (2001) assertions by citing them, also support the fact by conducting a study that makes up for the limitations. This helps to quash any controversy surrounding the Kiehl (2001) article's affective stimuli limitations. Any dissenter need only look at later research to be reassured that the verbal material as affective stimuli is not a viable alternative explanation for the results.

Being Ignored, and Apologizing for Conflict: How the Neuroscience of Psychopathy is Shaped by the Politics of Academic Writing

It is evident that the Kiehl (2001) authors have established an empire surrounding the topic of psychopathy and neuroscience. You would be hard pressed to find literature on psychopathy, or more precisely, psychopathy and neuroscience, where none of the Kiehl (2001) authors are cited. Especially Kent Kiehl and Robert Hare. It is in the best interest for authors to mobilize the Kiehl (2001) authors, because allying with such esteemed researchers gives strength to their own studies (Latour, 1987, p. 31). In a way, this constitutes an argument from authority, as their overwhelming presence in the field is close to attaining the status of a monopoly (especially in regards to Hare's delineation of the construct through the PCL-R). As such, producing literature that credits these authors is a better technique for positively influencing the career of their own articles than discrediting them is.

For example, we have found an article that negatively modalizes the Kiehl (2001) article by questioning the interpretation of their results:

> Given that research examining brain activity has concluded that psychopathic individuals use more cortical areas of the brain for processing affective stimuli (e.g., Kiehl et al., 2001; Munro et al., 2007), it may be that psychopaths are processing emotional stimuli in a more cognitive and rational manner as compared to nonpsychopathic individuals. In

> other words, rather than having a deficit, they may simply process the stimuli differently from nonpsychopaths. (Wheeler, Book, & Costello, 2009, p. 639).

This negative modalization of the Kiehl (2001) article suggests that their interpretation of the results as psychopaths having a deficit in affective processing may be better explained as psychopaths taking a different, more rational approach, to processing affective stimuli. Although their theory may have merit, and could have been a worthwhile line of reasoning to investigate, it has been widely ignored by any well-known research teams on psychopathy (including the Kiehl [2001] researchers). Despite the possibility of Wheeler et al.'s (2009) rational processing theory of psychopathy, fact-making is a collective process, and so being ignored by the academic community has destined their statement to remain an artefact. This example shows the politics of fact-making. When the Wheeler et al. (2009) authors failed to align with those in power, they were ostracized. There were no obvious qualitative differences between the Wheeler et al. (2009) article and others that saw a more successful career, except that they challenged one of the most prominent research teams in the field.

There are other studies that have produced results that are in conflict with the Kiehl (2001) results. However, to avoid being ostracized like the article noted in the above example, these studies dilute the contradiction, and phrase their results in such a way as to be inclusive of both their own results and those in the Kiehl (2001) article. For example, Deeley et al. (2006) did not find any significant differences in amygdala activation between control and psychopathic groups in response to affective stimuli. Despite this, they positively modalize the Kiehl (2001) article, and basically degrade, not just report, their conflicting findings. Deeley et al. (2006) exclaim that it was their failure to not find any differences between groups in amygdala activation, and then proceed to explain that said failure was a result of their study's limitations (i.e. small sample size, a lack of magnetic resonance acquisition parameters, and using a participant who engaged in cocaine use). The Deeley et al. (2006) authors are sure to include a statement saying they need better imaging acquisition to "increase the likelihood of detecting amygdala activation, given its central importance to theories of social cognition in general and psychopathy in particular" (p. 538). Here we see an article that like all others has a set of limitations, but since it does not align with predominant discourse and theory on psychopathy, is

discredited by its own authors. They do indeed report their findings as differing from the Kiehl (2001) findings, but are quick to explain that their study is faulty as opposed to the Kiehl (2001) study.

Muller et al. (2003) offer another example of how contrasting results are carefully reported and interpreted in such a way as to maintain an alliance with the powerful Kiehl (2001) team. Oppositely to the Kiehl (2001) study, the Muller et al. (2003) study found that psychopaths had an increased activation of the amygdala when processing affective stimuli. However, the Muller et al. (2003) article dilutes the contradiction with the Kiehl (2001) results by phrasing their findings in such a way that is inclusive of both: "Significantly increased activation, as well as reduced activation, have been found in different parts of the emotion-related brain circuit compared with healthy control subjects" (Müller et al., 2003, p. 160). In this example, the Muller et al. (2003) team stand by their findings, but also acknowledge the veracity of the Kiehl (2001) findings, and connect themselves with the Kiehl (2001) team. A few years later Kiehl (2006) addresses this discrepancy, negatively modalizes the Muller et al. (2003) study by stating its limitations, but also reciprocates the inclusiveness in his final statement: "in summary, cognitive neuroscience studies of affective processing have found that the neural circuits embracing the temporo-limbic system are either dysfunctional or hypofunctioning in psychopathy" (Kiehl, 2006, p. 122). We interpret this as an example of a technical writing technique. Despite an obvious contrast in results, both teams decide it is better to accompany each other and maintain an alliance than to engage in a dispute. Although the contents of the fact are forced to sacrifice specificity (i.e. transforming from under or over activation to simply abnormal activation), they can at least coexist, and lend strength to each other. Here we see the researchers picking their battles. They recognize that their potential enemy is also well equipped, and so they call a truce, and compromise for each other.

Finally, a last example of the Kiehl (2001) article being negatively modalized is in an article by Canil and Amin (2002). This article reviews the Kiehl (2001) study, and others like it. Their review negatively modalizes the Kiehl (2001) study by reminding readers that the study is exploratory, not confirmatory, and should therefore be interpreted and applied with caution. Canil and Amin (2002) explain that the Kiehl (2001) study, and others of its kind need to have

their results replicated, and the research extended to be considered more of a confirmed fact: "The fact that the conclusions of any individual study can be softened by alternative interpretations or methodological concerns is a useful reminder that the knowledge-base in these domains is malleable and awaits replication and extension." (p.424). This negative modalization is not undermining or opposing the Kiehl (2001) article per se, rather, it is informing the reader of the exploratory nature of this type of study. However, despite this word of caution, as we have shown, many other articles ignore this and carry the Kiehl (2001) article downstream with positive modalities and use it as confirmatory evidence.

The above examples show that although they are rare, there are negative modalizations of the Kiehl (2001) article. However, these modalizations are not enough to threaten the facticity of the Kiehl (2001) article's assertions. The above examples are evidence that science does not entail pure discovery, but rather a building of facts that make up our reality. The above examples also show that such a building process requires a collectivity, a calculated system of alliances, and a careful use of resources (e.g. enrolling exploratory studies as confirmatory). Detailing the politics of writing does not discredit the process, but rather shows how delicate the process of making neuroscientific facts on psychopathy is. Authors understand who reigns and has influence in the field, and respect their work enough to corroborate with them. This section has also shown how the politics of writing sets conditions where authors (e.g. Deeley et al., 2006) must exercise caution and restraint in presenting their findings in the wake of more credible evidence (i.e. Kiehl et al., 2001). In a way, this dynamic polices the field, as authors must refrain from making sweeping claims without substantial evidence that can contend with more established truths.

CONCLUSION: PSYCHOPATHY AS A NEUROSCIENTIFIC CONSTRUCT AND BOUNDARY OBJECT

This thesis has explored the way neuroscience assertions pertaining to psychopathy become seen as facts. We attempted to answer the question, how is neuroscience being applied to, and redefining psychopathy. In doing so we have presented a brief history of the development of the psychopathic construct into its modern version(s). We have also provided a detailed literature review of the construct, which included a review of psychopathy as a set of personality characteristics exhibited by successful business executives; psychopathy as a correctional tool for risk management purposes; psychopathy as a clinical instrument for diagnosing and treating psychiatric patients; and psychopathy as a scientific construct. This study focused on psychopathy as a scientific construct, and more specifically, how the neurosciences are being applied to the construct. In studying this application, we chose to work with Actor-Network Theory, which sits within the broader paradigm of Science and Technology Studies. This thesis was limited to a single case study of an academic research article on neuroscience and psychopathy, Kiehl et al., (2001). The chosen methodology allowed us to study the inner workings of the Kiehl (2001) laboratory, article, and career of the article as a means of gaining an understanding of how neuroscience is applied to, and redefines, psychopathy.

We have effectively supported our thesis that psychopathy as a built fact is a boundary object as defined by Star and Griesemer (1989). Star (2010) delineates conditions of a boundary object, which include (1) the object resides between social worlds; (2) the object maintains its vague identity across social worlds, but can be tailored and made more specific for local use in a social world; (3) groups cooperate without consensus and tack back-and forth between the many forms of the object. We have shown how psychopathy meets these three conditions. The construct inhabits several intersecting worlds (e.g. criminal justice, psychopharmacology, business, academics, psychology, genetics, and neuroscience) by maintaining plasticity as a characteristic. The common identity of psychopathy is maintained by researchers despite the tailored definitions used for the purpose of their studies (i.e. APD, youth delinquency, violent offenders, aggressive lab animals, etc.). Finally, we showed that through referencing techniques, authors and actors cooperate as they tack back-and forth between forms of the object (i.e. the

Kiehl article references many researchers who discuss the vague identity of psychopathy by tailoring the definition to fit their research). By adapting to the local needs of the Kiehl (2001) article through the translations described in the analysis chapters, the construct takes on new neuroscientific definitions that connect it with heterogeneous social worlds, which all lend it strength. By detailing the ways in which psychopathy is translated in the Kiehl (2001) article, we highlight many processes involved in academic research that create boundary objects. In describing how and why psychopathy is a boundary object, and focusing on how it has been made to adapt to the world of neuroscience, we have elucidated many intricacies of the facticity of psychopathy.

Our Laboratory Analysis chapter has shown how Kiehl's (2001) sophisticated array of black-boxed instruments was able to effectively produce representations of nature that were otherwise inaccessible. They were able to create a reality around psychopathy and affective processing through their ability to create nature, translate it into more accessible forms that represent the original element. Our chapter traced back the representations to their original elements, and displayed to the reader how the temporary redefining of concepts, constructs, and objects through representations (e.g. sample of psychopaths as representation of all psychopaths; PCL-R score cut-offs to represent psychopathy; 4-letter words back to real life affective material; 3D image of the human head/ brain back to raw fMRI data, etc.) was part of the process in producing the Kiehl (2001) facts on psychopathy and neuronal affective processing. We showed how researchers make many subjective judgments in conducting the work that is put into the producing of facts in a laboratory, and how sophisticated the production process is it this initial stage.

We then analyzed the technical writing techniques employed in the Kiehl (2001) article in our Upstream Analysis chapter. We discussed the ways in which the Kiehl (2001) article amassed references both within (i.e. graphs, legends, tables) and outside (i.e. other articles) of the text as a means of building a supporting foundation for their claims. We showed how strategic organization of references where explicit techniques such as the qualification of statements, modifying cited statements, stacking claims, stylisation, stratification of claims, staging and framing, etc. (Latour, 1987) were used to produce the neuroscientific facts around psychopathy

and affective processing. This analysis showed how psychopathy became a flexible construct for the purpose of exploration and fact production, as it was constantly compared to and represented by other entities (e.g. Antisocial personality disorder, brain damaged patients, violent criminals etc.). That is, this chapter showed how psychopathy holds the properties of a boundary object as it adapts to the heterogeneous social worlds to which it connects with. This chapter showed how, just like the laboratory, the Kiehl (2001) article is a sophisticated and calculated product. The Kiehl (2001) article is built like a fortress where allies are chosen and placed carefully. We showed how neuroscience did not simply discover new facts about the psychopathic construct, but rather built new facts that are made to withstand the harshest of terrains.

Finally, we analyzed how the Kiehl (2001) article was taken downstream by proceeding authors, and how their assertions were transformed into facts. This exploration showed us that akin to how the Kiehl (2001) authors positively modalized the articles they cited, proceeding authors positively modalized the Kiehl (2001) article. That is, exploratory research was often cited as confirmatory evidence, and used as premises or support for further assertions. This concretizes psychopathy as a neuroscientific construct, its adapted/ translated form created by the Kiehl (2001) researchers. This analysis chapter also showed how the Kiehl (2001) article, and consequently psychopathy as a neuroscientific construct, was taken downstream and positively modalized by authors in many outside/ neighbouring disciplines. By diversifying the application of the Kiehl (2001) article, psychopathy as a neuroscience construct becomes embedded and fortified by allies that span far beyond the borders of criminal psychopathy. Psychopathy gained strength from both the numbers and diversity of allies. We also showed how the politics of academic writing is a source of strength for the facticity of psychopathy as a neuroscientific construct. Proceeding authors would cautiously report results that were conflicting with the Kiehl (2001) results. That is, they would report in a more inclusive manner, so that they could still connect with the powerful Kiehl (2001) research team. We showed how an author that opposed the Kiehl (2001) assertions was ignored and how this research dynamic allows for challenged assertions to move forward as facts. Lastly, in this downstream analysis, we saw how researchers would zoom in and out of the brain (i.e. discuss specific structures or regions/ systems including many structures) for the purpose of amalgamating and ascertaining evidence on the neural networks responsible for psychopathic affective processing deficits. We saw how

researchers would connect with each other by using the interconnectivity and multiplicity of neural networks as a resource. For psychopathy, this zooming in and out renders the construct more adaptable; a more varied set of researcher articles to connect and further develop the factual status of psychopathy as a neuroscientific construct. We also showed how zooming in and out of the brain gave neuroscience assertions on psychopathy the ability to remain relevant despite changing positions in neuroscience (i.e. the denunciation of the limbic system).

Our study is limited to the analysis of the Kiehl (2001) article and how their assertions became facts. We cannot generalize to the production of all facts that are produced from technical writings on psychopathy and neuroscience. However, we do suggest that similar processes are at work across the field of neuroscience and psychopathy studies. Throughout our exploration we followed many articles both up and downstream from our starting pointand found that most were comparable in their laboratory and referencing techniques. Although this thesis is limited to the conclusions made on the Kiehl (2001) facts, we suggest that this line of inquiry is important for understanding the facticity (the quality or condition of being factual) of psychopathy as a neuroscientific construct. That understanding supports our assertion that psychopathy is a boundary object that is able to adapt/ translate its composition to align itself with multiple worlds. Exploring the world of research articles from this perspective can elucidate much about the field of neurocriminology as a whole. We have provided a deeper understanding of how and why the Kiehl (2001) assertions are factual statements, which goes beyond the face value understanding that "processing of affective stimuli is associated with less limbic activation in criminal psychopaths than in criminal nonpsychopaths… [and that] psychopathic offenders appear to use alternative neural systems to process affective stimuli" (Kiehl et al., 2001, p. 683).

This thesis provided a contribution to the knowledge of psychopathy as a neuroscience construct using STS/ ANT. As we mentioned at the beginning of this thesis, Pickersgill (2009) also used STS to research psychopathy and neuroscience, which aligned well with our own theoretical positioning. However, Pickersgill (2009) used a different data set (i.e. researchers comments on scientific assertions), and his research object was different than ours; while Pickersgill (2009) was interested in discourses on neuroscience colonization in the mental health

domain, we were interested in the material semiotic and how facts come to be created using material elements (i.e. nature created through scientific texts). This research was indeed inspired by works such as Pickersgill (2009, 2010), but while his research focused on qualifying the neuroscience facts and discussing their implications, we unfolded the facts and discussed the conditions of fact-making, or, facticity.

This understanding of the facticity of psychopathy as a neuroscience construct is essential considering that the neurosciences are indeed penetrating the realm of the social and promulgating a discourse that unites brain, mind and behaviour (Ortega & Vidal, 2011; Pickersgill, 2009; Rose & Abi-Rached, 2013). With regards to criminology, it has been proposed that we are seeing a paradigm shift toward 'neurocriminology' as a new discipline (Walsh & Beaver, 2009). Although our study does not comment on how this may affect the way professionals apply the construct, others have. For example, neurological causation begets a likely trajectory of pre-emptive 'screening and intervening' (e.g. preventative detention) on risky brains in adults and delinquent youth (Eastman & Campbell, 2006; Rose, 2010; Salekin, Rosenbaum, Lee, & Lester, 2009; Sevecke, 2011; Slough, & McMahon, 2008); increased psychotropic and genetic interventions (Leifer, 2000; Rose, 2000; Rose, 2010); and ultimately, a CJS that reduces violent criminality to individual neurobiological factors, and fails to endorse policy that targets broader systemic societal factors.

Considering the possible implications of such a paradigm shift, we assert that this thesis is a necessary exploration. It is essential that we fully understand the facticity of assertions made under this new paradigm, so that they can be put to appropriate application. We propose that further research of this kind is performed so as to broaden our understanding, and tighten our grasp on the facticity of psychopathy as a neuroscientific construct, and more generally, neurocriminology. In our assertion that psychopathy is a boarder object, we recognize the implications this has for diagnosing/ classifying and treating offenders who are designated as psychopaths. This turn toward neuroscience embraces an increasingly flexible construct, and possibly more flexible targets for intervention. For example, in the Kiehl (2001) article we saw the inclusiveness of psychopathy expand to those with lower cut-off PCL-R scores. Would using these sub-psychopaths as samples for research participants eventually warrant the treatment of

this population in criminal intervention practices? As neurosciences render psychopathy as a construct more adaptable, it is plausible that our diagnostic and intervention practices for criminal justice purposes will follow suit. Some researchers have already began to discuss the ethics of biological interventions on psychopathic prisoners (Choy, Berryessa, & Raine, 2016). They discuss Deep Brain Stimulation (DPS) therapy and explain that there are differences in subjective experiences of suffering between primary and secondary psychopaths that must be considered when determining what is ethical. Choy, Berryessa and Raine (2016) also consider how, like psychopaths, those with ADHD or schizophrenia often have callous unemotional traits, and can be treated by DBS. This is an example of how the psychopathy construct adapts to fit with neighbouring constructs and corresponding treatment options. As we have shown how psychopathy is a boarder object that adapts for neuroscience practices, we can see evidence of how it adapts to fit with available biological treatment options (i.e. associate with schizophrenia and ADHD interventions through its common ground- callous unemotional traits). This has implications for neurocriminology and biological intervention as a whole, beyond just the psychopathy construct. As neuroscience researchers target individual traits that intersect multiple disorders/ constructs (e.g. callousness), we could see intervention practices (e.g. DBS) target individual traits and be utilized for treating multiple disorders that share the trait. Additionally, as constructs that are being redefined by neuroscience become more fluid, flexible, and adaptable, we could see diagnostic and intervention practices that are more inclusive of who can be labelled. For instance, as psychopathy reduces its cut-off score more can be labelled as psychopathic, or as ADHD children and schizophrenic patients display callous and unemotional traits they are labelled with psychopathic tendencies. As a construct becomes more flexible through neuroscience research, so too does its diagnosis and application in practice.

REFERENCES

Adolphs R, Tranel D, Damasio AR (1998): The human amygdala in social judgment. *Nature*, 393, 470–474.

Adolphs, R., Tranel, D., Hamann, S., Young, A. W., Calder, A. J., Phelps, E. A., & Damasio, A. R. (1999). Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia*, *37*(10), 1111-1117.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorder: DSM-5*. ManMag.

Ammons RB, Ammons CH (1962): The Quick Test (Qt): Provisional Manual. *Psychol Rep,* 11, 111–161.

Anderson, S. W., Bechara, A., Damasio, H., Tranel, D., & Damasio, A. R. (1999). Impairment of social and moral behavior related to early damage in human prefrontal cortex. *Nature neuroscience*, *2*(11), 1032-1037.

Andrews, D.A. (2000). Principles of effective correctional programs. In L.L. Motiuk & R.C. Serin (Eds.), *2000 Compendium on Effective*

Andrews, D. A., & Bonta, J. (2010). *The psychology of criminal conduct*. Elsevier.

Andrews, D., & Bonta, J. (2016). LSI-R Level of Service Inventory-Revised.

Arrigo, B. A., & Shipley, S. (2001). The confusion over psychopathy (I): Historical considerations. *International Journal of Offender Therapy and Comparative Criminology*, *45*(3), 325-344.

Aspinwall, L. G., Brown, T. R., & Tabery, J. (2012). The double-edged sword: Does biomechanism increase or decrease judges' sentencing of psychopaths? *Science*, *337*(6096), 846-849.

Babiak, P. (1995). When psychopaths go to work: A case study of an industrial psychopath. *Applied Psychology*, *44*(2), 171-188.

Babiak, P. (1996). Psychopathic manipulation in organizations: Pawns, patrons, and patsies. *Issues in criminological and legal psychology*, *24*, 12-17.

Babiak, P. (2000). Psychopathic manipulation at work. *The clinical and forensic assessment of psychopathy: A practitioner's guide*, 287-311.

Babiak, P., & Hare, R. D. (2006). *Snakes in suits: When psychopaths go to work*. Regan Books/Harper Collins Publishers.

Babiak, P., & Hare, R. D. (2012). The B-Scan 360 Manual. *Toronto, ON: Multi-Health Systems*.

Babiak, P., Neumann, C. S., & Hare, R. D. (2010). Corporate psychopathy: Talking the walk. *Behavioral sciences & the law*, *28*(2), 174-193.

Barbaree, H. E., Seto, M. C., Langton, C. M., & Peacock, E. J. (2001). Evaluating the predictive accuracy of six risk assessment instruments for adult sex offenders. *Criminal Justice and Behavior*, *28*(4), 490-521.

Barnow, S., & Freyberger, H. J. (2010). The family environment in early life and aggressive behavior in adolescents and young adults. In *Neurobiology of Aggression*, 213-229.

Bartels, D. M., & Pizarro, D. A. (2011). The mismeasure of morals: Antisocial personality traits predict utilitarian responses to moral dilemmas. *Cognition*, *121*(1), 154-161.

Bechara A, Damasio AR, Damasio H, Anderson SW (1994): Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, 50, 7–15.

Bechara A, Damasio H, Damasio AR, Lee GP (1999): Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *J Neurosci*, 19, 5473–5481.

Beck, A., Kline, S., & Greenfield, L. (1988). Survey of youth in custody: special report. *Washington, DC, Department of justice, Bureau of justice Statistics*.

Beitchman, J. H., Zai, C. C., Muir, K., Berall, L., Nowrouzi, B., Choi, E., & Kennedy, J. L. (2012). Childhood aggression, callous-unemotional traits and oxytocin genes. *European child & adolescent psychiatry*, *21*(3), 125-132.

Berthoz, S., Grezes, J., Armony, J. L., Passingham, R. E., & Dolan, R. J. (2006). Affective response to one's own moral violations. *Neuroimage*, *31*(2), 945-950.

Birbaumer, N., Veit, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., & Flor, H. (2005). Deficient fear conditioning in psychopathy: a functional magnetic resonance imaging study. *Archives of general psychiatry*, *62*(7), 799-805.

Blackburn, R. (2006). Other theoretical models of psychopathy. *Handbook of psychopathy*, 35-57.

Blair, R. J. R. (2001). Neurocognitive models of aggression, the antisocial personality disorders, and psychopathy. *Journal of Neurology, Neurosurgery & Psychiatry*, *71*(6), 727-731.

Blair, R. J. R. (2003). Facial expressions, their communicatory functions and neuro-cognitive substrates. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *358*(1431), 561-572.

Blair, R. J. R. (2003). Neurobiological basis of psychopathy. *British Journal of Psychiatry, 182*, 5-7.

Blair, R. J. (2006). Subcortical brain systems in psychopathy. *Handbook of psychopathy*, 296-312.

Blair, R. J. R. (2008). The amygdala and ventromedial prefrontal cortex: functional contributions and dysfunction in psychopathy. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *363*(1503), 2557-2565.

Blair, R. J. R. (2013). Empathic responsiveness in amygdala and anterior cingulate cortex in youths with psychopathic traits. *Journal of Child Psychology and Psychiatry*, *54*(8), 900-910.

Blair, R. J. R., Budhani, S., Colledge, E., & Scott, S. (2005). Deafness to fear in boys with psychopathic tendencies. *Journal of Child Psychology and Psychiatry*, *46*(3), 327-336.

Blair, R. J. R., Colledge, E., & Mitchell, D. G. V. (2001). Somatic markers and response reversal: is there orbitofrontal cortex dysfunction in boys with psychopathic tendencies? *Journal of abnormal child psychology*, *29*(6), 499-511.

Blair, R. J. R., Mitchell, D. G. V., Richell, R. A., Kelly, S., Leonard, A., Newman, C., & Scott, S. K. (2002). Turning a deaf ear to fear: Impaired recognition of vocal affect in psychopathic individuals. *Journal of Abnormal Psychology*, *111*(4), 682-686.

Blumer, D., Benson, D.F., 1975. Personality changes with frontal lobe lesions. In: Benson, D.F.,

Blumer, D. (Eds.), Psychiatric Aspects of Neurological Disease. Grune and Stratton, New York, 151 – 170.

Bobes, M. A., Ostrosky, F., Diaz, K., Romero, C., Borja, K., Santos, Y., & Valdés-Sosa, M. (2013). Linkage of functional and structural anomalies in the left amygdala of reactive aggressive men. *Social cognitive and affective neuroscience*, *8*(8), 928-936.

Boccardi, M., Ganzola, R., Rossi, R., Sabattoli, F., Laakso, M. P., Repo-Tiihonen, E., & Frisoni, G. B. (2010). Abnormal hippocampal shape in offenders with psychopathy. *Human brain mapping*, *31*(3), 438-447.

Bonta, J., & Motiuk, L. (2015). High-Risk Violent Offenders in Canada. Retrieved from http://www.csc-scc.gc.ca/research/r50e-eng.shtml

Brotman, L. M., Gouley, K. K., Huang, K. Y., Kamboukos, D., Fratto, C., & Pine, D. S. (2007). Effects of a psychosocial family-based preventive intervention on cortisol response to a social challenge in preschoolers at high risk for antisocial behavior. *Archives of General Psychiatry*, *64*(10), 1172-1179.

Buchheim, A., Roth, G., Schiepek, G., Pogarell, O., & Karch, S. (2013). Neurobiology of borderline personality disorder (BPD) and antisocial personality disorder (APD). *Swiss Archives Neural Psychiatry*, *164*(4), 115-22

Budhani, S., & Blair, R. J. R. (2005). Response reversal and children with psychopathic tendencies: success is a function of salience of contingency change. *Journal of Child Psychology and Psychiatry*, *46*(9), 972-981.

Callon, M. (1986). Some elements of a sociology of translation: domestication of the scallops and the fishermen of St Brieuc Bay. *J. Law, Power, action and belief: a new sociology of knowledge? 1*, 196-223.

Campbell, C., & Eastman, N. (2012). The neurobiology of violence: Science and law. *I Know What You're Thinking: Brain imaging and mental privacy*, 139.

Canli, T., & Amin, Z. (2002). Neuroimaging of emotion and personality: Scientific evidence and ethical considerations. *Brain and cognition*, *50*(3), 414-431.

Carré, J. M., Hyde, L. W., Neumann, C. S., Viding, E., & Hariri, A. R. (2013). The neural signatures of distinct psychopathic traits. *Social neuroscience*, *8*(2), 122-135.

Choy, O., Berryessa, C. M., & Raine, A. (2016). The Ethics of Biological Interventions on Psychopathic Prisoners. *AJOB Neuroscience*, *7*(3), 154-156.

Ciaramelli, E., Muccioli, M., Ladavas, E., & di Pellegrino, G. (2007). Selective deficit in personal moral judgment following damage to ventromedial prefrontal cortex. *Social cognitive and affective neuroscience*, *2*(2), 84-92.

Cipriani, G., Borin, G., Vedovello, M., Di Fiorino, A., & Nuti, A. (2013). Sociopathic behavior and dementia. *Acta Neurologica Belgica*, *113*(2), 111-115.

Cleckley, H. (1941, 1955, 1976, 1982). The mask of sanity, *St. Louis: Mosby*.

Clemens, B., Voß, B., Pawliczek, C., Mingoia, G., Weyer, D., Repple, J. Habel, U. (2015). Effect of MAOA Genotype on Resting-State Networks in Healthy Participants. *Cerebral Cortex*, *25*(7), 1771-1781.

Coccaro, E. F., Sripada, C. S., Yanowitch, R. N., & Phan, K. L. (2011). Corticolimbic function in impulsive aggressive behavior. *Biological psychiatry*, *69*(12), 1153-1159.

Cooke, D. J., Michie, C., Hart, S. D., & Clark, D. A. (2004). Reconstructing psychopathy: Clarifying the significance of antisocial and socially deviant behavior in the diagnosis of psychopathic personality disorder. *Journal of personality disorders*, *18*(4), 337.

Cooke, D. J., Michie, C., & Hart, S. D. (2006). Facets of clinical psychopathy. *Handbook of psychopathy*, 91-106.

Cooke, D. J., Michie, C., & Skeem, J. (2007). Understanding the structure of the Psychopathy Checklist–Revised An exploration of methodological confusion. *The British Journal of Psychiatry*, *190*(49), 39-50.

Cooke, D. J., Forth, A. E., & Hare, R. D. (Eds.). (2012). *Psychopathy: Theory, research and implications for society* (Vol. 88). Springer Science & Business Media.

Cope, L. M., Ermer, E., Nyalakanti, P. K., Calhoun, V. D., & Kiehl, K. A. (2014). Paralimbic gray matter reductions in incarcerated adolescent females with psychopathic traits. *Journal of abnormal child psychology*, *42*(4), 659-668.

Cornell, D. G., Warren, J., Hawk, G., Stafford, E., Oram, G., & Pine, D. (1996). Psychopathy in instrumental and reactive violent offenders. *Journal of consulting and clinical psychology*, *64*(4), 783.

Correctional Services Canada. (2015). FORUM On Corrections research.

Craig, M. C., Catani, M., Deeley, Q., Latham, R., Daly, E., Kanaan, R., & Murphy, D. G. (2009). Altered connections on the road to psychopathy. *Molecular psychiatry*, *14*(10), 946-953.

Dadds, M. R., & Rhodes, T. (2008). Aggression in young children with concurrent callous unemotional traits: can the neurosciences inform progress and innovation in treatment approaches? *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *363*(1503), 2567-2576.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation--a possible prelude to violence. *Science*, *289*(5479), 591-594.

Damasio, H., Grabowski, T., Frank, R., Galaburda, A.M., Damasio, A.R., 1994. The return of Phineas Gage: clues about the brain from the skull of a famous patient. Science 264 (5162), 1102 – 1105.

Davis, M., Walker, D. L., & Lee, Y. (1997). Amygdala and bed nucleus of the stria terminalis: differential roles in fear and anxiety measured with the acoustic startle reflex. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, *352*(1362), 1675-1687.

Decety, J., & Ickes, W. (2011). *The social neuroscience of empathy*. MIT Press.

Del-Ben, C. M. (2005). Neurobiology of anti-social personality disorder. *Archives of Clinical Psychiatry (São Paulo)*, *32*(1), 27-36.

Deeley, Q., Daly, E., Surguladze, S., Tunstall, N., Mezey, G., Beer, D. & Clarke, A. (2006). Facial emotion processing in criminal psychopathy. *The British Journal of Psychiatry*, *189*(6), 533-539.

Dellacherie, D., Hasboun, D., Baulac, M., Belin, P., & Samson, S. (2011). Impaired recognition of fear in voices and reduced anxiety after unilateral temporal lobe resection. *Neuropsychologia*, *49*(4), 618-629.

Dias, R., Robbins, T. W., & Roberts, A. C. (1996). Dissociation in prefrontal cortex of affective and attentional shifts. *Nature*, *380*(6569), 69-72.

Dikman ZV, Allen JJB (2000): Error monitoring during reward and avoidance learning in high- and low-socialized individuals. *Psychophysiology,* 37, 43–54.

Dolan, B., & Coid, J. (1993). *Psychopathic and antisocial personality disorders: Treatment and research issues*. Gaskell/Royal College of Psychiatrists.

Dolan, M., & Doyle, M. (2000). Violence risk prediction. *The British Journal of Psychiatry*, *177*(4), 303-311.

Dougherty, D. D., Rauch, S. L., Deckersbach, T., Marci, C., Loh, R., Shin, L. M. & Fava, M. (2004). Ventromedial prefrontal cortex and amygdala dysfunction during an anger induction positron emission tomography study in patients with major depressive disorder with anger attacks. *Archives of General Psychiatry*, *61*(8), 795-804.

Douglas, K. S., Ogloff, J. R., Nicholls, T. L., & Grant, I. (1999). Assessing risk for violence among psychiatric patients: the HCR-20 violence risk assessment scheme and the Psychopathy Checklist: Screening Version. *Journal of consulting and clinical psychology*, *67*(6), 917.

Douglas, K. S., Vincent, G. M., & Edens, J. F. (2006). Risk for criminal recidivism: The role of psychopathy.

Eastman, N., & Campbell, C. (2006). Neuroscience and legal determination of criminal responsibility. *Nature reviews neuroscience*, *7*(4), 311-318.

Edens, J. F. (2006). Unresolved controversies concerning psychopathy: Implications for clinical and forensic decision making. *Professional Psychology: Research and Practice*, *37*(1), 59.

Edens, J. F., Petrila, J., & Buffington-Vollum, J. K. (2001). Psychopathy and the death penalty: can the Psychopathy Checklist-Revised identify offenders who represent "a continuing threat to society"? *The Journal of Psychiatry & Law*, *29*(4), 433-481.

Edens, J. F., Skeem, J. L., Cruise, K. R., & Cauffman, E. (2001). Assessment of "juvenile psychopathy" and its association with violence: a critical review. *Behavioral Sciences & the Law*, *19*(1), 53-80.

Edwards, B. G., Calhoun, V. D., & Kiehl, K. A. (2012). Joint ICA of ERP and fMRI during error-monitoring. *Neuroimage*, *59*(2), 1896-1903.

Ellard, J. (1988). The history and present status of moral insanity. *Australian and New Zealand Journal of Psychiatry*, *22*, 383-389.

Elliott, F. A. (1978). Neurological aspects of antisocial behavior. *The psychopath: A comprehensive study of antisocial disorders and behaviors*, 146-189.

Ermer, E., Cope, L. M., Nyalakanti, P. K., Calhoun, V. D., & Kiehl, K. A. (2012). Aberrant paralimbic gray matter in criminal psychopathy. *Journal of abnormal psychology*, *121*(3), 649.

Eysenck, H. J., Arnold, W., & Meili, R. (1972). *Encyclopedia of psychology*. Seabury Press.

Fahim, C., He, Y., Yoon, U., Chen, J., Evans, A., & Pérusse, D. (2011). Neuroanatomy of childhood disruptive behavior disorders. *Aggressive Behavior*, *37*(4), 326-337.

Fanselow, M. S. (2000). Contextual fear, gestalt memories, and the hippocampus. *Behavioural brain research*, *110*(1), 73-81.

Farmer, E. (2011). The age of criminal responsibility: developmental science and human rights perspectives. *Journal of children's services*, *6*(2), 86-95.

Farrington, D. P. (2006). Family background and psychopathy. *Handbook of psychopathy*, 229-250.

Feeley, M. M., & Simon, J. (1992). The New Penology: Notes on the Emerging Strategy of Corrections and its Implications. *Criminology*, *30*(4), 449-474.

Fellows, L. K., & Farah, M. J. (2005). Different underlying impairments in decision-making following ventromedial and dorsolateral frontal lobe damage in humans. *Cerebral cortex*, *15*(1), 58-63.

Finger, E. C., Marsh, A. A., Mitchell, D. G., Reid, M. E., Sims, C., Budhani, S., & Pine, D. S. (2008). Abnormal ventromedial prefrontal cortex function in children with psychopathic traits during reversal learning. *Archives of general psychiatry*, *65*(5), 586-594.

Fisher, P. A., Stoolmiller, M., Gunnar, M. R., & Burraston, B. O. (2007). Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. *Psychoneuroendocrinology*, *32*(8), 892-905.

Forth, A. E. (2000). *Assessing psychopathy with the PCL-R*. Sinclair Seminars, San Diego, CA.

Fowles, D. C. (1993). Electrodermal activity and antisocial behavior: Empirical findings and theoretical issues. *Progress in electrodermal research*, 223-237.

Fowles, D. C., & Dindo, L. (2006). A dual-deficit model of psychopathy. *Handbook of psychopathy*, 14-34.

Fowles, D. C., & Missel, K. A. (1994). Electrodermal hyporeactivity, motivation, and psychopathy: theoretical issues. *Progress in experimental personality & psychopathology research*, 263.

Frances, A. (1994). *Diagnostic and statistical manual of mental disorders: DSM-IV*. American Psychiatric Association.

Friston KJ, Ashburner J, Frith CD, Poline J-B, Heather JD, Frackowiak RSJ (1995a): Spatial registration and normalization of images. *Hum Brain Mapp*, 2, 165–189.

Friston KJ, Holmes AP, Poline J-P, Grasby PJ, Williams SC, Frackowiak RS, and Turner R (1995b): Analysis of fMRI timeseries revisited. *Neuroimage*, 2, 45–53.

Friston KJ, Holmes AP, Worsley KJ, Poline J-P, Frith CD, Frackowiak RSJ (1995c): Statistical parametric maps in functional imaging: a general linear approach. *Hum Brain Mapp,* 2, 189–210.

Gacono, C. B. (Ed.). (2015). *The clinical and forensic assessment of psychopathy: a practitioner's guide*. Routledge.

Gendreau, P., Goggin, C., & Smith, P. (2002). Is the PCL-R really the "unparalleled" measure of offender risk? A lesson in knowledge cumulation. *Criminal Justice and Behavior*, *29*(4), 397-426

Glenn, A. L., & Raine, A. (2014). *Psychopathy: An introduction to biological findings and their implications*. NYU Press.

Glenn, A. L., Raine, A., & Schug, R. A. (2009). The neural correlates of moral decision-making in psychopathy.

Glenn, A. L., Raine, A., Schug, R. A., Young, L., & Hauser, M. (2009). Increased DLPFC activity during moral decision-making in psychopathy. *Molecular Psychiatry*, *14*(10), 909.

Glenn, A. L., Raine, A., Yaralian, P. S., & Yang, Y. (2010). Increased volume of the striatum in psychopathic individuals. *Biological psychiatry*, *67*(1), 52-58.

Glenn, A. L., Yang, Y., Raine, A., & Colletti, P. (2010). No volumetric differences in the anterior cingulate of psychopathic individuals. *Psychiatry Research: Neuroimaging*, *183*(2), 140-143.

Glenn, A. L., & Yang, Y. (2012). The potential role of the striatum in antisocial behavior and psychopathy. *Biological psychiatry*, *72*(10), 817-822.

Glover, A. J., Nicholson, D. E., Hemmati, T., Bernfeld, G. A., & Quinsey, V. L. (2002). A comparison of predictors of general and violent recidivism among high-risk federal offenders. *Criminal justice and behavior*, *29*(3), 235-249.

Golub, M. S., Hogrefe, C. E., & Germann, S. L. (2007). Iron deprivation during fetal development changes the behavior of juvenile rhesus monkeys. *The Journal of nutrition*, *137*(4), 979-984.

Golub, M. S., Hogrefe, C. E., Widaman, K. F., & Capitanio, J. P. (2009). Iron deficiency anemia and affective response in rhesus monkey infants. *Developmental psychobiology*, *51*(1), 47-59.

Gopal, A., Clark, E., Allgair, A., D'Amato, C., Furman, M., Gansler, D. A., & Fulwiler, C. (2013). Dorsal/ventral parcellation of the amygdala: relevance to impulsivity and aggression. *Psychiatry Research: Neuroimaging*, *211*(1), 24-30.

Gordon, H. L., Baird, A. A., & End, A. (2004). Functional differences among those high and low on a trait measure of psychopathy. *Biological psychiatry*, *56*(7), 516-521.

Graustella, A. J., & MacLeod, C. (2012). A critical review of the influence of oxytocin nasal spray on social cognition in humans: evidence and future directions. *Hormones and behavior*, *61*(3), 410-418.

Gray, J. A. (1970). The psychophysiological basis of introversion-extraversion. *Behaviour research and therapy*, *8*(3), 249-266.

Greene, J. D., Sommerville, R. B., Nystrom, L. E., Darley, J. M., & Cohen, J. D. (2001). An fMRI investigation of emotional engagement in moral judgment. *Science*, *293*(5537), 2105-2108.

Gustafson, S. B., & Ritzer, D. R. (1995). The dark side of normal: a psychopathy-linked pattern called aberrant self-promotion. *European Journal of Personality*, *9*(3), 147-183.

Hall, J. R., & Benning, S. D. (2006). The "successful" psychopath. *Handbook of psychopathy*, 459-478.

Halligan, S. L., Herbert, J., Goodyer, I. M., & Murray, L. (2004). Exposure to postnatal depression predicts elevated cortisol in adolescent offspring. *Biological psychiatry*, *55*(4), 376-381.

Harenski, C. L., & Hamann, S. (2006). Neural correlates of regulating negative emotions related to moral violations. *Neuroimage*, *30*(1), 313-324.

Harenski, C. L., Harenski, K. A., Shane, M. S., & Kiehl, K. A. (2010). Aberrant neural processing of moral violations in criminal psychopaths. *Journal of abnormal psychology*, *119*(4), 863.

Hare RD (1965): Psychopathy, fear arousal and anticipated pain. *Psychol Rep*, 16, 499–502.

Hare RD (1968): Psychopathy, autonomic functioning, and the orienting response. *J Abn Psychol*, 73, 1–24.

Hare RD (1982): Psychopathy and physiological activity during anticipation of an aversive stimulus in a distraction paradigm. *Psychophysiology*, 19, 266–271.

Hare, R. D. (1984). Performance of psychopaths on cognitive tasks related to frontal lobe function. *Journal of Abnormal Psychology*, *93*(2), 133.

Hare, R. D. (1991). *The Hare psychopathy checklist-revised: Manual*. Multi-Health Systems, Incorporated.

Hare, R. D. (1995). *Without conscience: The disturbing world of the psychopaths among us*. Guilford Press.

Hare, R. D. (1996). Psychopathy a clinical construct whose time has come. *Criminal justice and behavior*, *23*(1), 25-54.

Hare, R. D. (1998). Psychopathy, affect and behavior. In *Psychopathy: Theory, research and implications for society*, 105-137

Hare, R. D. (2000). Assessing psychopathy with the PCL-R. Sinclair Seminars, San Diego, CA.

Hare, R. D. (2004). Hare PCL-R: Technical Manual. *Toronto: MHS*.

Hare, R. D., Cooke, D. J., & Hart, S. D. (1999). Psychopathy and sadistic personality disorder.

Hare RD, Frazelle J, Cox DN (1978): Psychopathy and physiological responses to threat of an aversive stimulus. *Psychophysiology*, 15, 165–172.

Hare, R. D., & Hart, S. D. (1993). Psychopathy, mental disorder, and crime.

Hare, R. D., & Hervé, H. (1999). Hare P-Scan: Research version. *Toronto, ON: Multi-Health Systems*.

Hare, R. D., & Jutai, J. W. (1988). Psychopathy and cerebral asymmetry in semantic processing. *Personality and Individual Differences*, *9*(2), 329-337.

Hare, R. D., & Neumann, C. S. (2006). The PCL-R assessment of psychopathy. *Handbook of psychopathy*, 58-88.

Hare, R. D., & Neumann, C. S. (2008). Psychopathy as a clinical and empirical construct. *Annu. Rev. Clin. Psychol.*, *4*, 217-246.

Hare RD, Quinn MJ (1971): Psychopathy and autonomic conditioning. *J Abn Psychol*, 77, 223–235.

Harris, G. T., & Rice, M. E. (2006). Treatment of psychopathy. *Handbook of psychopathy*, 555-572.

Harris, G. T., Rice, M. E., & Quinsey, V. L. (1993). Violent recidivism of mentally disordered offenders: The development of a statistical prediction instrument. *Criminal justice and behavior*, *20*(4), 315-335.

Harris, G. T., Skilling, T. A., & Rice, M. E. (2001). The construct of psychopathy. *Crime and Justice*, *28*, 197-264.

Hart, S. D. (1998). The role of psychopathy in assessing risk for violence: Conceptual and methodological issues. *Legal and criminological psychology*, *3*(1), 121-137.

Hart, S. D., & Hare, R. D. (1997). Psychopathy: Assessment and association with criminal conduct.

Hemphill, J. (1992). Psychopathy and recidivism following release from a therapeutic community treatment program. *Unpublished ms. Saskatoon: Department of Psychology, University of Saskatchewan*, 60.

Hemphill, J. F., Hare, R. D., & Wong, S. (1998). Psychopathy and recidivism: A review. *Legal and criminological Psychology*, *3*(1), 139-170.

Hemphill, J. F., & Hart, S. D. (2003). Forensic and clinical issues in the assessment of psychopathy. *Handbook of psychology*.

Hemphill, J. F., & Hare, R. D. (2004). Some misconceptions about the Hare PCL-R and risk assessment a reply to Gendreau, Goggin, and Smith. *Criminal Justice and Behavior*, *31*(2), 203-243.

Hibbeln, J. R. (2001). Homicide mortality rates and seafood consumption: A cross-national analysis. *World Review of Nutrition and Dietetics*, *88*, 41-46.

Hicks, B. M., Markon, K. E., Patrick, C. J., Krueger, R. F., & Newman, J. P. (2004). Identifying psychopathy subtypes on the basis of personality structure. *Psychological assessment*, *16*(3), 276.

Hollingshead AdB, Redlich FC (1958): *Social Class and Mental Illness: A Community Study*. New York: Wiley.

Holt, S. E., & Strack, S. (1999). Sadism and psychopathy in violent and sexually violent offenders. *Journal of the American Academy of Psychiatry and the Law Online*, *27*(1), 23-32.

Hoptman, M. J., & Antonius, D. (2011). Neuroimaging correlates of aggression in schizophrenia: an update. *Current opinion in psychiatry*, *24*(2), 100.

Horley, J. (2014). The emergence and development of psychopathy. *History of the Human Sciences*, *27*(5), 91-110.

Hornak, J., Bramham, J., Rolls, E.T., Morris, R.G., O'Doherty, J., Bullock, P.R., Polkey, C.E., 2003. Changes in emotion after circumscribed surgical lesions of the orbitofrontal and cingulate cortices. Brain, 126, 1691 – 1712.

Hyde, L. W., Byrd, A. L., Votruba-Drzal, E., Hariri, A. R., & Manuck, S. B. (2014). Amygdala reactivity and negative emotionality: Divergent correlates of antisocial personality and psychopathy traits in a community sample. *Journal of abnormal psychology*, *123*(1), 214.

Intrator J, Hare R, Stritzke P, et al (1997): A brain imaging (single photon emission computerized tomography) study of semantic and affective processing in psychopaths. *Biol Psychiatry*, 42, 96–103.

Irwin W, Davidson RJ, Lowe MJ, Mock BJ, Sorenson JA, Turski PA (1996): Human amygdala activation detected with echoplanar functional magnetic resonance imaging. *Neuroreport*, 7, 1765–1769.

Jalava, J. V. (2007). *Science of conscience: Metaphysics, morality, and rhetoric in psychopath research* (Doctoral dissertation, Dept. of Psychology-Simon Fraser University).

Jones, A. P., Laurens, K. R., Herba, C. M., Barker, G. J., & Viding, E. (2009). Amygdala hypoactivity to fearful faces in boys with conduct problems and callous-unemotional traits. *American Journal of Psychiatry*, *166*(1), 95-102.

John Howard Society of Alberta. (2000). Offender Risk Assessment.

Juárez, M., Kiehl, K. A., & Calhoun, V. D. (2013). Intrinsic limbic and paralimbic networks are associated with criminal psychopathy. *Human brain mapping*, *34*(8), 1921-1930.

Kalbe, E., Brand, M., Thiel, A., Kessler, J., & Markowitsch, H. J. (2008). Neuropsychological and neural correlates of autobiographical deficits in a mother who killed her children. *Neurocase*, *14*(1), 15-28.

Kiehl, K. A. (2006). A cognitive neuroscience perspective on psychopathy: Evidence for paralimbic system dysfunction. *Psychiatry Research, 142*(2-3), 107-128.

Kiehl, K. A., & Hoffman, M. B. (2011). The criminal psychopath: history, neuroscience, treatment, and economics. *Jurimetrics, 51*, 355.

Kiehl, K. A., Hare, R. D., McDonald, J. J., & Brink, J. (1999). Semantic and affective processing in psychopaths: An event-related potential (ERP) study. *Psychophysiology, 36*(6), 765 774.

Kiehl, K. A., Smith, A. M., Hare, R. D., Mendrek, A., Forster, B. B., Brink, J., & Liddle, P. F. (2001). Limbic abnormalities in affective processing by criminal psychopaths as revealed by functional magnetic resonance imaging. Biological Psychiatry, 50(9), 677-684.

Kiehl, K. A., Smith, A. M., Mendrek, A., Forster, B. B., Hare, R. D., & Liddle, P. F. (2004). Temporal lobe abnormalities in semantic processing by criminal psychopaths as revealed by functional magnetic resonance imaging. *Psychiatry Research: Neuroimaging, 130*(1), 27-42.

Kinscherff, R. (2010). Proposition: a personality disorder may nullify responsibility for a criminal act.

Krischer, M. K., & Sevecke, K. (2008). Early traumatization and psychopathy in female and male juvenile offenders. *International journal of law and psychiatry, 31*(3), 253-262.

Kitajka, K., Sinclair, A. J., Weisinger, R. S., Weisinger, H. S., Mathai, M., Jayasooriya, A. P., & Puskás, L. G. (2004). Effects of dietary omega-3 polyunsaturated fatty acids on brain gene expression. *Proceedings of the National Academy of Sciences of the United States of America, 101*(30), 10931-10936.

Knoch, D., Treyer, V., Regard, M., Müri, R. M., Buck, A., & Weber, B. (2006). Lateralized frequency-dependent effects of prefrontal rTMS on regional cerebral blood flow. *Neuroimage, 31*(2), 641-648.

Koenigs, M., Young, L., Adolphs, R., Tranel, D., Cushman, F., Hauser, M., & Damasio, A. (2007). Damage to the prefrontal cortex increases utilitarian moral judgements. *Nature, 446*(7138), 908-911.

Kohlboeck, G., Glaser, C., Tiesler, C., Demmelmair, H., Standl, M., Romanos, M. & LISAplus Study Group. (2011). Effect of fatty acid status in cord blood serum on children's behavioral difficulties at 10 y of age: results from the LISAplus Study. *The American journal of clinical nutrition, 94*(6), 1592-1599.

Kropp, P. R., Hart, S. D., Webster, C. D., & Eaves, D. (1999). Spousal assault risk assessment guide (SARA) Multi-Health Systems. *Inc., Toronto*.

Laakso, M. P., Vaurio, O., Koivisto, E., Savolainen, L., Eronen, M., Aronen, H. J., & Tiihonen, J. (2001). Psychopathy and the posterior hippocampus. *Behavioural brain research*, *118*(2), 187-193.

LaBar KS, LeDoux JE, Spencer DD, Phelps EA (1995): Impaired fear conditioning following unilateral temporal lobectomy in humans. *J Neurosci,* 15, 6846–55.

Lang, P. J., Davis, M., & Öhman, A. (2000). Fear and anxiety: animal models and human cognitive psychophysiology. *Journal of affective disorders*, *61*(3), 137-159.

Latour, B. (1987). *Science in action: How to follow scientists and engineers through society*. Harvard university press.

Latour, B. (1999). Circulating reference: Sampling the soil in the Amazon forest.

Latour, B. (2005). *Reassembling the social: An introduction to actor-network-theory*. Oxford university press.

Latour, B. and Woolgar, S. (1979) Laboratory Life, the Social Construction of Scientific Facts. London: Sage.

LeDoux, J. E. (1995). In search of an emotional system in the brain: leaping from fear to emotion and consciousness.

LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual review of neuroscience*, *23*(1), 155 184.

LeDoux, J. (2003). The emotional brain, fear, and the amygdala. *Cellular and molecular neurobiology*, *23*(4), 727-738.

Leifer, R. (2001). A critique of medical coercive psychiatry, and an invitation to dialogue. *Ethical Human Sciences and Services*, *3*(3), 161-173

Levenson, M. R., Kiehl, K. A., & Fitzpatrick, C. M. (1995). Assessing psychopathic attributes in a noninstitutionalized population. *Journal of personality and social psychology*, *68*(1), 151.

Levenston, GK, Patrick CJ, Bradley MM, Lang PJ (2000) The psychopath as observer: Emotion and attention in picture processing. *J Abn Psychol,* 109, 373–385.

Li, R. L., Kosten, T. R., & Sinha, R. (2006). Antisocial personality and stress-induced brain activation in cocaine-dependent patients. *Neuroreport*, *17*(3), 243-247.

Lilienfeld, S. O., & Andrews, B. P. (1996). Development and preliminary validation of a self-report measure of psychopathic personality traits in noncriminal population. *Journal of personality assessment*, *66*(3), 488-524.

Lim, M. M., & Young, L. J. (2006). Neuropeptidergic regulation of affiliative behavior and social bonding in animals. *Hormones and behavior*, *50*(4), 506-517.

Liu, J. (2004). Childhood externalizing behavior: theory and implications. *Journal of child and adolescent psychiatric nursing*, *17*(3), 93-103.

Liu, J. (2011). Early health risk factors for violence: Conceptualization, evidence, and implications. *Aggression and violent behavior*, *16*(1), 63-73.

Loza, W., & Dhaliwal, G. K. (1997). Psychometric Evaluation of the Risk Appraisal Guide (RAG) A Tool for Assessing Violent Recidivism. *Journal of Interpersonal Violence*, *12*(6), 779-793.

Loza, W., & Simourd, D. J. (1994). Psychometric evaluation of the Level of Supervision Inventory (LSI) among male Canadian federal offenders. *Criminal Justice and Behavior*, *21*(4), 468-480.

Lykken, D. T. (1957). A study of anxiety in the sociopathic personality. *The Journal of Abnormal and Social Psychology*, *55*(1), 6.

Lykken, D. T. (1995). *The antisocial personalities*. Psychology Press.

Lykken, D. T. (2006). Psychopathic personality: The scope of the problem. *Handbook of psychopathy*, 3-13.

Lösel, F. (1998). Treatment and management of psychopaths. In *Psychopathy: Theory, research and implications for society*, pp. 303-354.

Iacono, W. G. (2006). Toward an integrated perspective on the etiology of psychopathy. *Handbook of psychopathy*, 375-385.

Mahmut, M. K., Homewood, J., & Stevenson, R. J. (2008). The characteristics of non-criminals with high psychopathy traits: Are they similar to criminal psychopaths? *Journal of Research in Personality*, *42*(3), 679-692.

Manning, N. (2000). Psychiatric diagnosis under conditions of uncertainty: personality disorder, science and professional legitimacy. *Sociology of health & illness*, *22*(5), 621-639.

Marinkovic, K., Oscar-Berman, M., Urban, T., O'Reilly, C. E., Howard, J. A., Sawyer, K., & Harris, G. J. (2009). Alcoholism and dampened temporal limbic activation to emotional faces. *Alcoholism: Clinical and Experimental Research*, *33*(11), 1880-1892.

Marsh, A. A., Finger, E. C., Mitchell, D. G., Reid, M. E., Sims, C., Kosson, D. S., & Blair, R. J. R. (2008). Reduced amygdala response to fearful expressions in children and adolescents with callous-unemotional traits and disruptive behavior disorders. *American Journal of Psychiatry*.

Matthewman, S. (2011). *Technology and social theory*. Palgrave Macmillan.

Maughs, S. (1941). A concept of psychopathy and psychopathic personality: its evolution and historical development. *Journal of Criminal Psychopathology*.

May, J. S., & Beaver, K. M. (2014). The neuropsychological contributors to psychopathic personality traits in adolescence. *International journal of offender therapy and comparative criminology*, *58*(3), 265-285.

McCord, W., & McCord, J. (1964). The psychopath: An essay on the criminal mind.

McDonald, R., Dodson, M. C., Rosenfield, D., & Jouriles, E. N. (2011). Effects of a parenting intervention on features of psychopathy in children. *Journal of abnormal child psychology*, *39*(7), 1013-1023.

McNaughton, N., & Gray, J. A. (2000). Anxiolytic action on the behavioural inhibition system implies multiple types of arousal contribute to anxiety. *Journal of affective disorders*, *61*(3), 161-176.

Meloy, J. R. (2000). Violence risk and threat assessment. *San Diego, CA: Specialized Training Services*.

Mendez, M. F., Anderson, E., & Shapira, J. S. (2005). An investigation of moral judgement in frontotemporal dementia. *Cognitive and behavioral neurology*, *18*(4), 193-197.

Mier, D., Lis, S., Zygrodnik, K., Sauer, C., Ulferts, J., Gallhofer, B., & Kirsch, P. (2014). Evidence for altered amygdala activation in schizophrenia in an adaptive emotion recognition task. *Psychiatry Research: Neuroimaging*, *221*(3), 195-203.

Miller, A. K., Rufino, K. A., Boccaccini, M. T., Jackson, R. L., & Murrie, D. C. (2011). On individual differences in person perception: Raters' personality traits relate to their psychopathy checklist-revised scoring tendencies. *Assessment*, *18*(2), 253-260.

Millon, T. (1981). *Disorders of personality: DSM-III, axis II*. John Wiley & Sons.

Millon, T., Simonsen, E., & Birket-Smith, M. (1998). Historical conceptions of psychopathy in the United States and Europe. *Psychopathy: Antisocial, criminal, and violent behavior*, 3-31. New York: Guilford.

Millon, T., Simonsen, E., Birket-Smith, M., & Davis, R. D. (Eds.). (2003).*Psychopathy: Antisocial, criminal, and violent behavior*. Guilford Press.

Minzenberg, M. J., & Siever, L. J. (2006). Neurochemistry and pharmacology of psychopathy and related disorders. *Handbook of psychopathy*, 251-277

Mitchell, D., Colledge, E., Leonard, A., Blair, R.J.R., 2002. Risky decisions and response reversal: is there evidence of orbitofrontal cortex dysfunction in psychopathic individuals? Neuropsychologia, 2013 – 2022.

Mokros, A., Osterheider, M., Hucker, S. J., & Nitschke, J. (2011). Psychopathy and sexual sadism. *Law and human behavior*, *35*(3), 188.

Moll, J., de Oliveira-Souza, R., Bramati, I. E., & Grafman, J. (2002). Functional networks in emotional moral and nonmoral social judgments. *Neuroimage*, *16*(3), 696-703.

Moltó, J., Poy, R., & Torrubia, R. (2000). Standardization of the Hare Psychopathy Checklist Revised in a Spanish prison sample. *Journal of Personality Disorders*, *14*(1), 84.

Monahan, J., Brodsky, S. L., & Shan, S. A. (1981). *Predicting violent behavior: An assessment of clinical techniques*, 95-128. Beverly Hills, CA: Sage Publications.

Monahan, J., Steadman, H. J., Silver, E., Appelbaum, P. S., Robbins, P. C., Mulvey, E. P., & Banks, S. (2001). Rethinking risk assessment. *The MacArthur study of mental disorder and violence*.

Müller, J. L., Gänßbauer, S., Sommer, M., Döhnel, K., Weber, T., Schmidt-Wilcke, T., & Hajak, G. (2008). Gray matter changes in right superior temporal gyrus in criminal psychopaths. Evidence from voxel-based morphometry. *Psychiatry Research: Neuroimaging*, *163*(3), 213-222.

Müller, J. L., Sommer, M., Wagner, V., Lange, K., Taschler, H., Röder, C. H., Hajak, G. (2003). Abnormalities in emotion processing within cortical and subcortical regions in criminal psychopaths: Evidence from a functional magnetic resonance imaging study using pictures with emotional content. Biological Psychiatry, 54(2), 152-162.

Nelson HE, O'Connell A., (1978): Dementia: the estimation of premorbid intelligence levels using the New Adult Reading Test. *Cortex*, 14, 234–244.

Neumann, C. S., Hare, R. D., & Newman, J. P. (2007). The super-ordinate nature of the psychopathy checklist-revised. *Journal of personality disorders*, *21*(2), 102.

Neumann, C. S., Vitacco, M. J., Hare, R. D., & Wupperman, P. (2005). Reconstruing the "reconstruction" of psychopathy: A comment on Cooke, Michie, Hart, and Clark. *Journal of personality disorders*, *19*(6), 624-640.

Newman, J. P., & Kosson, D. S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of abnormal psychology*, *95*(3), 252.

Newman, J. P., Patterson, C. M., & Kosson, D. S. (1987). Response perseveration in psychopaths. *Journal of abnormal psychology*, *96*(2), 145.

Newman, J. P., Widom, C. S., & Nathan, S. (1985). Passive avoidance in syndromes of disinhibition: Psychopathy and extraversion. *Journal of personality and social psychology*, *48*(5), 1316.

Oliveira-Souza, R., Hare, R. D., Bramati, I. E., Garrido, G. J., Ignácio, F. A., Tovar-Moll, F., & Moll, J. (2008). Psychopathy as a disorder of the moral brain: fronto-temporo-limbic grey matter reductions demonstrated by voxel-based morphometry. *Neuroimage*, *40*(3), 1202-1213.

O'Neal, C. R., Brotman, L. M., Huang, K. Y., Gouley, K. K., Kamboukos, D., Calzada, E. J., & Pine, D. S. (2010). Understanding relations among early family environment, cortisol response, and child aggression via a prevention experiment. *Child development*, *81*(1), 290-305.

Ortega, F. G., & Vidal, F. (Eds.). (2011). *Neurocultures: glimpses into an expanding universe*. P. Lang.

Osumi, T., Nakao, T., Kasuya, Y., Shinoda, J., Yamada, J., & Ohira, H. (2012). Amygdala dysfunction attenuates frustration-induced aggression in psychopathic individuals in a non-criminal population. *Journal of affective disorders*, *142*(1), 331-338.

Patrick, C. J. (Ed.). (2006). *Handbook of psychopathy*. Guilford Press.

Patrick, C. J. (2007). Getting to the Heart of Psychopathy.

Patrick CJ, Bradley MM, Lang PJ (1993): Emotion in the criminal psychopath: startle reflex modulation. *J Abn Psychol*, 102, 82–92.

Patrick CJ, Cuthbert BN, Lang PJ (1994): Emotion in the criminal psychopath: fear image processing. *J Abn Psychol*, 103, 523–534.

Patrick, C. J., & Lang, A. R. (1999). Psychopathic traits and intoxicated states: Affective concomitants and conceptual links.

Patrick, C. J., Venables, N. C., & Skeem, J. (2012). Psychopathy and brain function: Empirical findings and legal implications. *Psychopathy and law for practitioners. New York, NY: Wiley*.

Patrick, C. J., & Zempolich, K. A. (1999). Emotion and aggression in the psychopathic personality. *Aggression and violent behavior*, *3*(4), 303-338.

Peng, H., Zheng, H., Li, L., Liu, J., Zhang, Y., Shan, B. & Zhou, J. (2012). High-frequency rTMS treatment increases white matter FA in the left middle frontal gyrus in young patients with treatment-resistant depression. *Journal of affective disorders*, *136*(3), 249 257.

Phillips, M. L., Drevets, W. C., Rauch, S. L., & Lane, R. (2003). Neurobiology of emotion perception I: the neural basis of normal emotion perception. *Biological psychiatry*, *54*(5), 504-514.

Pickering, A. D., & Gray, J. A. (1999). The neuroscience of personality. *Handbook of personality: Theory and research*, *2*, 277-299.

Pickersgill, M. D. (2010). Psyche, soma, and science studies: New directions in the sociology of mental health and illness. *Journal of Mental Health*.

Porter, S., & Woodworth, M. (2006). Psychopathy and aggression. *Handbook of psychopathy*, 481-494.

Poser, A. B. (2009). *Techniques for BOLD and blood volume weighted fMRI*.

Poythress, N. G., & Skeem, J. L. (2006). Disaggregating psychopathy.*Handbook of psychopathy*, 172-192.

Prichard, J. C. (1837/1973). *A treatise on insanity and other disorders affecting the mind*. New York: Arno.

Public Safety Canada. (2015). Corrections research in Canada: Taking stock.

Quinsey, V. L., Harris, G. T., Rice, M. E., & Cormier, C. A. (1998). Violent offenders: Appraising and managing risk. *Washington DC*.

Raine, A., Ishikawa, S. S., Arce, E., Lencz, T., Knuth, K. H., Bihrle, S., & Colletti, P. (2004). Hippocampal structural asymmetry in unsuccessful psychopaths. *Biological psychiatry*, *55*(2), 185-191.

Raine A, Lencz T, Bihrle S, LaCasse L, Colletti P (2000): Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. *Arch Gen Psychiatry* 57, 119–27

Raine, A., Lencz, T., Taylor, K., Hellige, J. B., Bihrle, S., Lacasse, L., & Colletti, P. (2003). Corpus callosum abnormalities in psychopathic antisocial individuals. *Archives of General Psychiatry*, *60*(11), 1134-1142.

Raine, A., Meloy, J. R., Bihrle, S., Stoddard, J., LaCasse, L., & Buchsbaum, M. S. (1998). Reduced prefrontal and increased subcortical brain functioning assessed using positron

emission tomography in predatory and affective murderers. *Behavioral sciences & the law*, *16*(3), 319-332.

Raine, A., & Sanmartín, J. (2001). *Violence and psychopathy*. Springer Science & Business Media.

Raine, A., & Yang, Y. (2006). The neuroanatomical bases of psychopathy. *Handbook of psychopathy*, 278-295.

Reise, S. P., & Oliver, C. J. (1994). Development of a California Q-set indicator of primary psychopathy. *Journal of Personality Assessment*, *62*(1), 130-144. Rice, M. E., Harris, G. T., & Cormier, C. A. (1992). An evaluation of a maximum security therapeutic community for psychopaths and other mentally disordered offenders. *Law and human behavior*, *16*(4), 399.

Rilling, J. K., Glenn, A. L., Jairam, M. R., Pagnoni, G., Goldsmith, D. R., Elfenbein, H. A., & Lilienfeld, S. O. (2007). Neural correlates of social cooperation and non-cooperation as a function of psychopathy. *Biological psychiatry*, *61*(11), 1260-1271.

Rogers, R. D. (2006). The Functional Architecture of the Frontal Lobes: Implications for Research with Psychopathic Offenders. *Handbook of psychopathy*, 313-333.

Rolls, E. T., Hornak, J., Wade, D., & McGrath, J. (1994). Emotion-related learning in patients with social and emotional changes associated with frontal lobe damage. *Journal of Neurology, Neurosurgery & Psychiatry*, *57*(12), 1518-1524.

Ronson, J. (2011). *The psychopath test: A journey through the madness industry*. Penguin.

Rose, N. (2000). The biology of culpability: Pathological identity and crime control in a biological culture. *Theoretical criminology*, *4*(1), 5-34.

Rose, N. (2010). 'Screen and intervene': governing risky brains. *History of the Human Sciences*, *23*(1), 79-105.

Rose, N. and Abi-Rached J. M. (2013). Neuro. The New Brain Sciences and the Management of the Mind. Princeton, Princeton University Press.

Rosen, G. M., Deinard, A. S., Schwartz, S., Smith, C., Stephenson, B., & Grabenstein, B. (1985). Iron deficiency among incarcerated juvenile delinquents. *Journal of Adolescent Health Care*, *6*(6), 419-423.

Rubia, K. (2011). "Cool" inferior frontostriatal dysfunction in attention-deficit/hyperactivity disorder versus "hot" ventromedial orbitofrontal-limbic dysfunction in conduct disorder: a review. *Biological psychiatry*, *69*(12), 69-87.

Rush, B. (1812). *Medical inquiries and observations upon the diseases of the mind*. Philadelphia:

Kimber & Richardson.

Salekin, R. T. (2002). Psychopathy and therapeutic pessimism: Clinical lore or clinical reality? *Clinical psychology review*, *22*(1), 79-112.

Salekin, R. T., Rosenbaum, J., Lee, Z., & Lester, W. S. (2009). Child and adolescent psychopathy: Like a painting by Monet. *Youth Violence and Juvenile Justice*, *7*(3), 239 255.

Salekin, R. T., Rogers, R., & Sewell, K. W. (1996). A review and meta-analysis of the Psychopathy Checklist and Psychopathy Checklist-Revised: Predictive validity of dangerousness. *Clinical Psychology: Science and Practice*, *3*(3), 203-215.

Salekin, R. T., Worley, C., & Grimes, R. D. (2010). Treatment of psychopathy: A review and brief introduction to the mental model approach for psychopathy. *Behavioral sciences & the law*, *28*(2), 235-266.

Sarkar, S., Craig, M. C., Catani, M., Dell'Acqua, F., Fahy, T., Deeley, Q., & Murphy, D. G. (2013). Frontotemporal white-matter microstructural abnormalities in adolescents with conduct disorder: a diffusion tensor imaging study. *Psychological Medicine*, *43*(2), 401 411.

Sassi, R. B., Brambilla, P., Hatch, J. P., Nicoletti, M. A., Mallinger, A. G., Frank, E., ... & Soares, J. C. (2004). Reduced left anterior cingulate volumes in untreated bipolar patients. *Biological psychiatry*, *56*(7), 467-475.

Scarpa, A., & Raine, A. (2000). Violence associated with anger and impulsivity. *The neuropsychology of emotion*, 320-339.

Scerbo, A., Raine, A., O'Brien, M., Chan, C. J., Rhee, C., & Smiley, N. (1990). Reward dominance and passive avoidance learning in adolescent psychopaths. *Journal of Abnormal Child Psychology*, *18*(4), 451-463.

Schachter, S., & Latane, B. (1964). Crime, cognition, and the autonomic nervous system. In *Nebraska symposium on motivation*. University of Nebraska Press.

Schmauk, F. J. (1970). Punishment, arousal, and avoidance learning in sociopaths. *Journal of abnormal psychology*, *76*(3), 325.

Seagrave, D., & Grisso, T. (2002). Adolescent development and the measurement of juvenile psychopathy. *Law and human behavior*, *26*(2), 219.

Serin, R. C. (1996). Violent recidivism in criminal psychopaths. *Law and Human Behavior*, *20*(2), 207.

Serin, R. C., Mailloux, D. L., & Hucker, S. J. (2000). *The utility of clinical and actuarial risk assessments for offenders in pre-release psychiatric decision-making*. Correctional Service Canada, Research Branch, Corporate Development.

Serin, R. C., Peters, R. D., & Barbaree, H. E. (1990). Predictors of psychopathy and release outcome in a criminal population. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, *2*(4), 419.

Seto, M. C., & Barbaree, H. E. (1999). Psychopathy, treatment behavior, and sex offender recidivism. *Journal of interpersonal violence*, *14*(12), 1235-1248.

Seto, M. C., & Quinsey, V. L. (2006). Toward the Future. *Handbook of psychopathy*, 229-250.

Sevecke, K., et al. (2011). Personality disorders in adolescence: Controversy and perspectives. Kindheit Und Entwicklung 20(4), 256-264.

Sharpe K, O'Carroll R (1991): Estimating premorbid intellectual level in dementia using the National Adult Reading Test: a Canadian study. *Br J Clin Psychol,* 30, 381–384.

Siddle, D. A. T., & Trasler, G. B. (1981). The psychophysiology of psychopathic behavior. *Foundations of psychosomatics*, 283-303.

Siever, L. J. (2008). Neurobiology of aggression and violence. *American Journal of Psychiatry*, *165*(4), 429-442.

Silver, E., Mulvey, E. P., & Monahan, J. (1999). Assessing violence risk among discharged psychiatric patients: toward an ecological approach. *Law and human behavior*, *23*(2), 237.

Simourd, D. J., & Malcolm, P. B. (1998). Reliability and validity of the Level of Service Inventory-Revised among federally incarcerated sex offenders. *Journal of Interpersonal Violence*, *13*(2), 261-274.

Sismondo, S. (2010). *An introduction to science and technology studies* (Vol. 1). Chichester: Wiley-Blackwell.

Skeem, J. L., Monahan, J., & Mulvey, E. P. (2002). Psychopathy, treatment involvement, and subsequent violence among civil psychiatric patients. *Law and human behavior*, *26*(6), 577.

Skeem, J. L., Poythress, N., Edens, J. F., Lilienfeld, S. O., & Cale, E. M. (2003). Psychopathic personality or personalities? Exploring potential variants of psychopathy and their implications for risk assessment. *Aggression and Violent Behavior*, *8*(5), 513-546.

Slack, J. D., & Wise, J. M. (2005). Culture and technology. *A Primer, New*.

Slough, N. M., & McMahon, R. J. (2008). Preventing serious conduct problems in school-age youth: The Fast Track program. *Cognitive and behavioral practice*, *15*(1), 3-17.

Smith, R. J. (1978). *Personality and psychopathology: A series of monographs, texts, and treatises*. New York: Academic Press.

Smith, D., Smith, R., & Misquitta, D. (2016). Neuroimaging and violence. *Psychiatric Clinics*, *39*(4), 579-597.

Snyder, H. N., & Sickmund, M. (1995). *Juvenile offenders and victims: A focus on violence* (p. 14). US Department of Justice, Office of Justice Programs, Office of Juvenile Justice and Delinquency Prevention.

Star, S. L., & Griesemer, J. R. (1989). Institutional ecology, translations' and boundary objects: Amateurs and professionals in Berkeley's Museum of Vertebrate Zoology, 1907-39. *Social studies of science*, *19*(3), 387-420.

Sterzer, P., & Stadler, C. (2009). Neuroimaging of aggressive and violent behaviour in children and adolescents. *Frontiers in behavioral neuroscience*, *3*.

Strüber, N., Strüber, D., & Roth, G. (2014). Impact of early adversity on glucocorticoid regulation and later mental disorders. *Neuroscience & Biobehavioral Reviews*, *38*, 17-37.

Suedfeld, P., & Landon, P. B. (1978). Approaches to treatment. *Psychopathic behavior: Approaches to research*, 347-376.

Tancredi, L. R. (2009). Imaging and Genetics: Future Applications in the Emergency Room. *Primary Psychiatry*, *16*(9).

Taren, A. A., Creswell, J. D., & Gianaros, P. J. (2013). Dispositional mindfulness co-varies with smaller amygdala and caudate volumes in community adults. *PLoS One*, *8*(5), e64574.

Taylor, J., Loney, B. R., Bobadilla, L., Iacono, W. G., & McGue, M. (2003). Genetic and environmental influences on psychopathy trait dimensions in a community sample of male twins. *Journal of abnormal child psychology*, *31*(6), 633-645.

Tassy, S., Oullier, O., Duclos, Y., Coulon, O., Mancini, J., Deruelle, C. & Wicker, B. (2011). Disrupting the right prefrontal cortex alters moral judgement. *Social cognitive and affective neuroscience*.

Tikàsz, A., Potvin, S., Lungu, O., Joyal, C. C., Hodgins, S., Mendrek, A., & Dumais, A. (2016). Anterior cingulate hyperactivations during negative emotion processing among men with schizophrenia and a history of violent behavior. *Neuropsychiatric disease and treatment*, *12*, 1397.

Toch, H. (1998). Psychopathy or antisocial personality in forensic settings. *Psychopathy:*

Antisocial, criminal, and violent behavior, 144-158.

Toglia, M. P., & Battig, W. F. (1978). *Handbook of semantic word norms*. Lawrence Erlbaum.

Tranel D, Damasio H (1994): Neuroanatomical correlates of electrodermal skin conductance responses. *Psychophysiology*, 31, 427–438.

Ulmer, S., & Jansen, O. (2013). *fMRI*. Springer.

Venturini, T. (2010). Diving in magma: How to explore controversies with actor-network theory. *Public understanding of science*, *19*(3), 258-273.

Viding, E., & Jones, A. P. (2008). Cognition to genes via the brain in the study of conduct disorder. *The Quarterly Journal of Experimental Psychology*, *61*(1), 171-181.

Völlm, B. A., Taylor, A. N. W., Richardson, P., Corcoran, R., Stirling, J., McKie, S., Elliott, R. (2006). Neuronal correlates of theory of mind and empathy: A functional magnetic resonance imaging study in a nonverbal task. *NeuroImage*, *29*(1), 90-98.

Waldman, I. D., & Rhee, S. H. (2006). Genetic and environmental influences on psychopathy and antisocial behavior. *Handbook of psychopathy*, 205-228.

Walsh, A. & K.V. Beaver, eds. (2009). *Biosocial Criminology. New Directions in Theory and Research*. New York: Routledge.

Walters, G. D. (2004). The trouble with psychopathy as a general theory of crime. *International Journal of Offender Therapy and Comparative Criminology*, *48*(2), 133-148.

Webster, C. D. (1997). HCR-20: Assessing risk for violence.

Webster, C. D., Douglas, K. S., Eaves, D., & Hart, S. D. (1997). Assessing risk of violence to others. *Impulsivity: Theory, assessment, and treatment*, 251-277.

Webster, C. D., & Hucker, S. J. (2007).*Violence risk: assessment and management*. Chichester, West Sussex, England: Wiley.

Werlinder, H. (1978). Psychopathy: A History of the Concepts: Analysis of the Origin and Development of a Family of Concepts in Psychopathology.

Wetzell, R. F. (2000). *Inventing the criminal: A history of German criminology, 1880-1945*. University of North Carolina Press.

Wheeler, S., Book, A., & Costello, K. (2009). Psychopathic traits and perceptions of victim vulnerability. *Criminal Justice and Behavior*, *36*(6), 635-648.

Williamson, S., Hare, R. D., & Wong, S. (1987). Violence: Criminal psychopaths and their

victims. *Canadian Journal of Behavioural Science/Revue canadienne des sciences du comportement*, *19*(4), 454.

Williamson S, Harpur TJ, Hare RD (1991): Abnormal processing of affective words by psychopaths. *Psychophysiology,* 28, 260–273.

Widiger, T. A. (2006). Psychopathy and DSM-IV psychopathology. *Handbook of psychopathy*, 156-171.

Wilson, J. Q., & Herrnstein, R. J. (1998). *Crime human nature: The definitive study of the causes of crime*. Simon and Schuster.

Wolpe, P. R., Foster, K. R., & Langleben, D. D. (2005). Emerging neurotechnologies for lie detection: promises and perils. *The American Journal of Bioethics*, *5*(2), 39-49.

Wong, S., & Hare, R. D. (2001). Program guidelines for the institutional treatment of violent psychopathic offenders. *Toronto: Multi-Health Systems*.

Yang, Y., Raine, A., Narr, K. L., Colletti, P., & Toga, A. W. (2009). Localization of deformations within the amygdala in individuals with psychopathy. *Archives of general psychiatry*, *66*(9), 986-994.

Yang, Y., Raine, A., Colletti, P., Toga, A. W., & Narr, K. L. (2011). Abnormal structural correlates of response perseveration in individuals with psychopathy. *The Journal of neuropsychiatry and clinical neurosciences*.

Young, M., Justice, J., Gacono, C. B., & Kivisto, A. J. (2000). The Incarcerated Psychopath in Psychiatric Treatment. *The Clinical and Forensic Assessment of Psychopathy: A Practitioner's Guide*, 313-350.

Zahn, T. P., Schooler, C., & Murphy, D. L. (1986). Autonomic correlates of sensation seeking and monoamine oxidase activity: Using confirmatory factor analysis on psychophysiological data. *Psychophysiology*, *23*(5), 521-531.

APPENDIX

Table 1: Symptoms of Psychopathy as Delineated by Hervey Cleckley

1. Superficial charm and good intelligence	9. Pathologic egocentricity and incapacity for love
2. Absence of delusions and other signs of irrational thinking	10. General poverty in major affective reactions
3. Absence of nervousness or psychoneurotic manifestations	11. Specific loss of insight
4. Unreliability	12. Unresponsiveness in general personal relations
5. Untruthfulness and insincerity	13. Fantastic and uninviting behaviour with drink and sometimes without
6. Lack of remorse and shame	14. Suicide threats rarely carried out
7. Inadequately motivated antisocial behaviour	15. Sex life trivial, impersonal, and poorly integrated
8. Poor Judgement and failure to learn from experience	16. Failure to follow any life plan

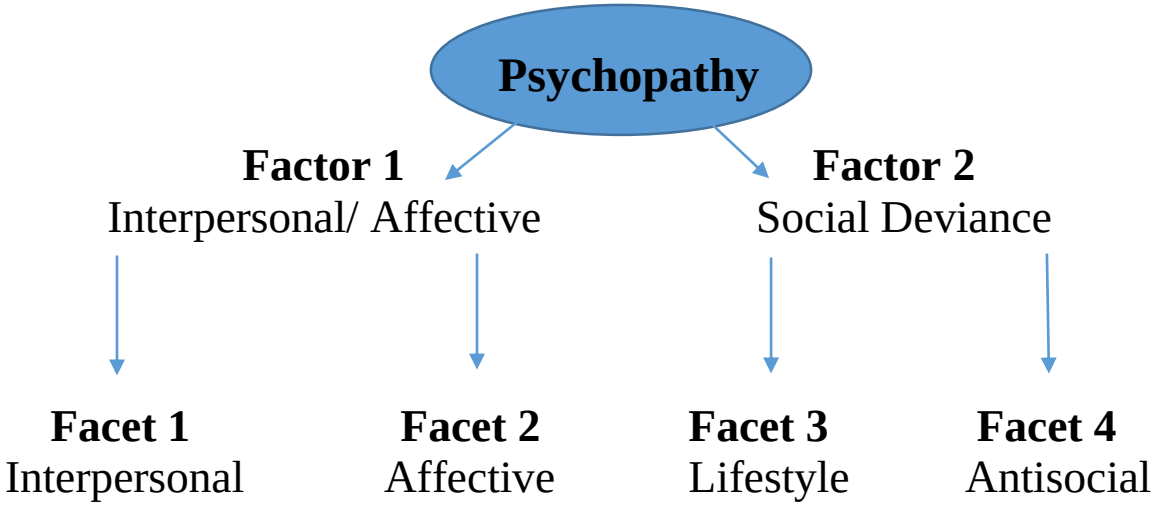
(Cleckley, 1976, pp 337-364)

Table 2: The Psychopathy Checklist- Revised

1. Glib and superficial charm	11. Sexual promiscuity
2. Grandiose (exaggeratedly high) estimation of self	12. Early behaviour problems
3. Need for stimulation	13. Lack of realistic long-term goals
4. Pathological lying	14. Impulsivity
5. Cunning and manipulativeness	15. Irresponsibility
6. Lack of remorse of guild	16. Failure to accept responsibility for own actions
7. Shallow affect (superficial emotional responsiveness)	17. Many short-term marital relationships
8. Callousness and lack of empathy	18. Juvenile delinquency
9. Parasitic lifestyle	19. revocation of conditional release
10. Poor behavioural controls	20. Criminal versatility

(Hare, 2004, pp. 35-46)

Table 3: The Two-Factor, Four-Facet, Structure of Psychopathy



(Hare, 2004, p. 7)