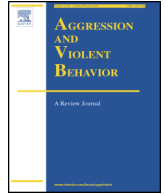


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Aggression and Violent Behavior



Psychopathy versus sociopathy: Why the distinction has become crucial

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ABSTRACT

The terms *psychopath* and *sociopath* are often used interchangeably, but there appears to be some hesitance by researchers in the many disciplines comprising criminology to continue this trend. The problem seems to be that as research has advanced in studies of psychopathy, which is the more common of the two terms, *psychopathy* now commands a much more specific definition, and this is what alienates it from its estranged cousin, *sociopathy*. As language can serve to hinder or confound research, it is crucial that these terms take their proper place in brain science. Here, I present how the two terms are currently used in neuroscience and psychology, and suggest how research in sociopathy should progress.

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1. Introduction

While quibbling over terminology can be a tedious endeavor, it has become necessary to treat the terms *psychopath* and *sociopath* differently. When used in every day conversation the importance in the distinction is not noticeable – the words simply describe a remorseless killer and seem to denote that something could be wrong with the offender's brain. However, in brain science, the difference between these two terms has become crucial for numerous reasons. First, the history of studying psychopathy has now arrived at a point where the word *psychopath* means something very specific. Second, there appears to be a hesitance among scholars to use them interchangeably, preferring to use *sociopathy* if a brain injury or a belief

system resulted in antisocial behavior (there should be no hesitance in the use of terminology). And lastly, in light of the first two reasons, the neurology underlying the psychopath and the sociopath can only be different, a crucial fact to be realized when seeking to understand the etiology, behavioral characteristics, and potential treatments for each.

Here I present a brief history of psychopathy research, followed by the use of sociopathy in brain science, and conclude with some ideas about where the research should go by looking at the life of a recent killer, Anders Breivik.

2. The history and growth of *psychopathy*

Hervey Cleckley was one of the first mental health experts to classify the term *psychopathy* in his book *The Mask of Sanity*, which was first published in 1941. Cleckley listed the numerous psychopathic

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traits with which we have all become familiar, such as superficial charm, lack of remorse, and an impersonal sex life (Ogloff, 2006). However, the next significant breakthrough came from Robert Hare, a Canadian psychologist who devised the Psychopathy Checklist (PCL-R) (Hare, 1991). The PCL-R is used in many studies exploring the behavior and neurology of psychopathic individuals as it is often one of the key criteria for separating psychopathic from the non-psychopathic individuals (Basoglu et al., 2011; Craig et al., 2009; Raine et al., 2004; Vaidyanathan, Hall, Patrick, & Bernat, 2011; Yang, Raine, Colletti, Toga, & Narr, 2010).

In recent years, the PCL-R has met with some controversy. Hare has expressed a preference that the test only be used in an academic setting, rather than in a court room, as there have been cases where mental health experts hired by the defense and by the prosecution come up with suspiciously different results for the defendant.¹

However, the brilliance of the PCL-R should not be undermined. Hare designed the test from a strictly behavioral point of view and managed to list all relevant behaviors of the psychopath and create a scoring system that could tell you if the person was psychopathic or not. Hare has always stressed that in order to identify these behaviors, and to deal with the pathological lying of the psychopath, the test needs to be administered by a trained professional. When the test was first designed, Hare could not have known of the immense screening power the test would have for neuroscientists a decade later.

By using the PCL-R (or the abbreviated PCL-SV) as a screening tool, it was possible to find that psychopaths have reduced gray matter in their frontal lobes (Muller et al., 2008; Raine, Lencz, Birhle, LaCasse, & Colletti, 2000; Yang et al., 2010), increased striatal volume (Glenn, Raine, Yaralian, & Yang, 2010), abnormal asymmetry in the hippocampus (Laakso et al., 2001; Raine et al., 2004), a larger corpus callosum (Raine et al., 2003), a lack of structural integrity in the uncinate fasciculus² (Craig et al., 2009), abnormal activity in the anterior cingulate cortex (ACC) (Kiehl et al., 2001), and deformations within the amygdala (Yang, Raine, Colletti, & Toga, 2009). There are two points to note here; first, the PCL-R works extremely well as the differences in neurology reflect the results of the test (at least in an academic setting). Second, all of these neurological studies have added an immense amount of knowledge to what it means biologically to be a psychopath.

A number of the neurological differences mentioned above are associated with interruptions to their respective developmental pathways in early life. Raine et al. (2003) point out that the corpus callosum starts out larger in childhood but “trims back” as the brain develops; and Raine et al. (2004) state that the kind of asymmetry noticed in the hippocampi in psychopaths reflects the asymmetry seen in the fetus – an asymmetry which diminishes throughout the development of the child. So, is something happening to children in their early lives that could end up orchestrating the construction of the psychopath brain? Farrington (2005) explains that antisocial behavior in children appears to be correlated with physical abuse, parental conflict, and antisocial parents (among a number of other factors), and it would not surprise anyone if this extremely stressful home life stunted the child’s brain from developing normally.

There have also been many studies on the autonomic functioning of psychopaths, some of which preceded the advent of the PCL-R. Hare (1968) summarizes many of the studies before 1968, and concludes through his own investigation that autonomic functioning in the psychopath does appear to be slightly different from the non-psychopath while at rest, as demonstrated by a lower level of skin conductance and lower heart rate variability. Later studies have also found reduced

physiological changes when psychopaths are presented with fear imagery (Levenston, Patrick, Bradley, & Lang, 2000; Patrick, Cuthbert, & Lang, 1994). These physiological differences in the psychopath have prompted many to wonder if these response deficits are why psychopaths appear affectively blind and do not have an in depth understanding of emotions. Gao, Raine, and Schug (2012) suggest that psychopaths are genuinely unable to appreciate their own body sensations; therefore, the pathological lying dimension to psychopathic behavior does not extend to understanding their own emotional experiences.

Hopefully, I have shown that the term *psychopath* means a great deal to those in brain science. And it is precisely because of this rich history in psychopath research that having another term can only serve to confound our understanding of this dangerous condition.

3. Finding a place for *sociopathy*

Hare and Babieck (2006) acknowledge a clear difference between *psychopathy* and *sociopathy*. Psychopathy, which is the condition Hare successfully captured with the PCL-R, means the individual will have no empathy or sense of morality among a number of other traits (Hare, 1991). Sociopathy, on the other hand, is indicative of having a sense of morality and a well-developed conscience, but the sense of right and wrong is not that of the parent culture. As this difference is reflected in the brain, the distinction again shows its use.

4. Morality and beliefs

To help give sociopathy its own neurological correlate, a study of the empathy circuit (Baron-Cohen, 2011) is useful. The empathy circuit, proposed by Baron-Cohen (2011), consists of 10 regions, all with an arguable role in empathy; as empathy represents our capacity to understand another’s emotional disposition, it allows neuroscience to stake a claim in discussions of morality. Not surprisingly, some of the regions mentioned are areas that failed to develop in the psychopath; these regions include certain areas of the frontal cortex, the ACC, and the amygdala. Consequently, here we see that psychopaths, who have no sense of morality, have compromised areas in the empathy circuit. If sociopaths do have a sense of morality, then it would be crucial to know how their brain compares to psychopaths.

The fact that sociopaths do have a sense of morality and a sense of right and wrong reflects that they have beliefs about the social world. There are some regions in the empathy circuit (Baron-Cohen, 2011) that are affected by our beliefs and their impact on the rest of the brain is not necessarily hindered by injury or dementia. The ACC is known for its involvement in pain recognition in ourselves (Lane et al., 1998), and activity in this region has been correlated with whether we think punitive actions against others are just or unjust (Singer et al., 2006). Singer et al. (2006), using functional magnetic resonance imaging (fMRI), found that the activity in the ACC decreases when people believe that the punishment another person receives is considered fair; this was particularly true of the male participants. While neurodevelopmental differences between the male and female ACC could facilitate this difference in judgment, deciding on whether a punishment is just or fair is a reflection of beliefs. If beliefs can cause a variance of activation in any part of the empathy circuit, we can see how it might be possible for differences in belief to cause subtle neurological differences that promote different moral behavior.

Differences in the amygdala have also been correlated with a difference in worldviews. Kanai, Feilden, Firth, and Rees (2011) found that the right amygdala in conservatives was larger than in liberals. While interpreting this result remains a challenge, it is nevertheless a neurological difference that reflects a worldview and a different moral outlook. Clearly, further studies need to examine if the worldview resulted in this neurological difference, or if the neurological difference occurred independently of any worldview, but favored acquisition of the worldview – or if they at some point began to

¹ Dr. Hare expresses his concern over the use of the PCL-R in the legal system on NPR <http://www.npr.org/2011/05/27/136723357/creator-of-psychopathy-test-worries-about-its-use>

² The uncinate fasciculus is the white matter tract that allows communication between the frontal cortex and the limbic structures, particularly the amygdala.

perpetuate one another. Whatever the result, however, an enlarged right amygdala is indicative of a conservative morality, and demonstrates that beliefs could either result in a change to this part of the empathy circuit, or certain kinds of beliefs will be perpetuated as a result of this change to this part of the empathy circuit.

In order to develop a sense of morality in the first instance, it seems crucial that certain areas in the prefrontal cortex are at least partly functional. For example, Amodio and Frith (2006) explain the importance of the anterior region of the rostral medial frontal cortex (arMFC); it is needed for mentalizing (thinking about the intentions, desires, and beliefs of others), self-knowledge (distinguishing yourself from others), and person knowledge (being aware of the perception and judgments of others). These three important methods of social reflection seem pivotal in establishing a sense of morality because they are how we understand the behaviors of others and determine whether or not we like this behavior, which we can measure against how we choose to behave based upon self-reflection, our awareness of others, and our own beliefs. Sociopathy, unlike psychopathy, therefore, requires a higher degree of neuronal integrity in the medial prefrontal cortex.

To summarize, there are neurological correlates for how beliefs could promote specific kinds of attitudinal or behavioral moral outcomes. These correlates provide a basis for studying how beliefs create our moral identity by affecting our empathy circuit. However, a large component of sociopathy involves antisocial behavior, and I am unaware of any neurological study that ties beliefs to antisocial behavior.

5. Injury and dementia

Numerous physicians and neuroscientists refer to sociopathy in terms of *acquired sociopathy* (Barrash, Tranel, & Anderson, 2000; Blair & Cipolotti, 2000; Mendez, Chen, Shapira, & Miller, 2005), which is usually when a brain injury or dementia in the frontal lobe(s) results in antisocial behavior. It must be noted here that this definition of *sociopathy* is vastly different from the one offered by Hare and Babieck (2006). Perhaps the most famous story of acquired sociopathy is the case of Phineas Gage; Gage, who worked on the railroad in the nineteenth century, received a metal spike through his frontal lobe as the result of an explosion (Mendez, Shapira, & Saul, 2011). A closer analysis of the accident has led some to believe that the rod pierced through Gage's ventromedial prefrontal cortex (VMPFC) (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994). Gage apparently went from being a responsible worker to irresponsible, profane, and indifferent to the social conventions of his time (Sanfey, Hastie, Colvin, & Grafman, 2003). It is worth asking if Gage still had a sense of morality, but it is this kind of personality change caused by damage to the frontal lobe that defines *acquired sociopathy*.

The first person to use the term *acquired sociopathy* was Antonio Damasio after his lengthy examinations of the patient, E. V. R., who had lesions to the orbitofrontal cortex and subsequently changed from being a happily married professional to being divorced numerous times with a failed business (Blair & Cipolotti, 2000). The resultant personality changes that led patients to exhibit a lack of concern for others and a lack of remorse led Damasio to suggest that psychopathy could be the developmental counterpart to this acquired sociopathy (Blair & Cipolotti, 2000). This seems a little overly ambitious, as an abnormal frontal lobe is only one piece of the psychopathy puzzle — as we have seen, differences can also arise in the amygdala, hippocampus, corpus callosum, striatum, and the uncinate fasciculus, although there is little doubt that damage to the frontal lobe can easily result in antisocial behavior (Brower & Price, 2001; Kandel & Freed, 1989).

Mendez et al. (2011) examined acquired sociopathy in dementia patients. The study involved 33 dementia patients, but the criteria for sociopathy included a range of behavior that included physical assault, theft, indecent exposure, and stalking with homicidal behavior.

The trouble with this study is that the behaviors are not unique to any kind of neurology, after all, the patients had a range of different dementias. Examples cited in the study include a man with Huntington's Disease who stalked his ex-wife and threatened to kill her (he had mild cortical atrophy and hypometabolism bilaterally in the caudate and putamen), a man diagnosed with Alzheimer's Disease who threatened individuals he believed were trying to steal his belongings, and a man with subacute dementia who could not stop from making inappropriate comments or inappropriately touching others. In these instances, *acquired sociopathy* appears to be an umbrella term for antisocial behavior resulting from a range of dementias. If this is to remain a definition for *sociopathy* it will be as useless to mental health experts as the term *insane*.

Another point to make about Mendez et al. (2011) is that many who develop sociopathic behavior from dementia are likely to be over the age of 40; Alzheimer's Disease is typically diagnosed around age 65, although sometimes the individual can be as young as 30,³ and Huntington's Disease, despite rare occurrences of it appearing in childhood or adolescence, typically appears in those in their 30s and over.⁴ The usual onset of dementia does not fit in well with the age-crime curve (a curve that shows the likelihood of people of different ages committing a crime), which peaks in the late teens (Loeber & Stallings, 2011). This means that sociopathy is extremely unlikely to be prevalent in young offenders, unlike psychopathy which can be diagnosed in the form of extreme antisocial personality disorder at age 18. This diminishes the utility of using *sociopathy* in criminology, especially since many with dementia do not develop sociopathic behavior.

6. Conclusion

I have presented three reasons why *psychopathy* cannot be treated the same way as *sociopathy*. First, psychopathy research has been so rich and deep that the term has taken on a tremendous amount of meaning, partly because psychopathy is a developmental disorder that is associated with specific kinds of behavior. Second, if we use the definition that sociopaths have a sense of morality, we have to allow for brain differences that are not present in the psychopath. And last, *acquired sociopathy*, which is very different from psychopathy, seems to be satisfied by the mere presence of antisocial behavior brought about by lesions, from both trauma and surgery, or dementia.

Providing *sociopathy* with its own distinct and useful meaning, I think, has to come from the second reason, which could provide a neurological correlate to the definition supplied by Hare and Babieck (2006). Discovering how beliefs affect the brain and our moral judgment is crucial research; however, how ideas can cause physiological changes in the brain is certainly not as obvious as finding damage or abnormalities. If we can expand on the studies that already exist, we can see how the beliefs of a subculture provide a morality and a worldview that could permit the individual to indulge in heinous acts such as mass killing.

I would like to contend that this new approach for understanding sociopathy would allow us to understand individuals such as Anders Breivik, the Norwegian gunman who on the 22nd July, 2011, murdered 77 people; 8 from a car bomb in Oslo, and 69 who he shot at a summer camp on the island of Utoeya.⁵ Breivik had written a 1801 page manifesto and was very steeped in his own version of national socialism.⁶ He saw his place in the world as a crusader and a martyr for his cause, and consequently his acts were necessary.

³ Alzheimer's Association: http://www.alz.org/downloads/facts_figures_2012.pdf

⁴ National Institute for Health, Genetics Home Reference: <http://ghr.nlm.nih.gov/condition/huntington-disease>

⁵ BBC Article: Anders Breivik pleads not guilty at murder trial. <http://www.bbc.co.uk/news/world-europe-17724535>

⁶ Guardian, Anders Behring Breivik attacks inspired by Serbian nationalists, Helen Pidd. <http://www.guardian.co.uk/world/2012/apr/18/anders-behring-breivik-serb-nationalists>

When he opened fire at the many children in the camp was there limited activity in his ACC? And, if so, how long had his ACC promoted this outlook? Breivik had a sense of right and wrong that clearly differed from the morality of the parent culture and he killed remorselessly.

The key to understanding sociopathy has to be in the power that ideas exert on the brain.

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