Chapter

Perspective Chapter: Neuropsychology of Aggression in Psychopathy and Sociopathy – Insights for the Treatment and Study of Antisocial Personality Disorder

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Abstract

Misunderstanding, stigmatization, and fascination abound in western culture concerning the concepts of psychopathy and sociopathy. These concepts are often used interchangeably—and erroneously—to describe violent or aggressive behavior in humans. These concepts tend also to be hurled as insults at individuals assumed to exhibit a lack empathy, impulsive decision making, and violent behavior. Psychopathy and sociopathy, however, are two concepts that describe different etiologies of the same mental health condition: antisocial personality disorder. This chapter bifurcates between the neurobiological origins of psychopathy and sociopathy, contributing to the destigmatization of a broadly misunderstood mental health condition. This chapter also explores recent findings from functional magnetic resonance imagery studies that analyze neurophysiological activity germane to psychopathy and sociopathy. Using these terms, students, clinicians, and researchers have access to a language that outlines correlations in neural substrate activity between genetic antisocial personality disorder (psychopathy) and epigenetic antisocial personality disorder (sociopathy). These terms might also serve to enhance treatment outcomes, as they implicate discrete neural substrates that have the potential to be treated using psychotherapeutic and psychopharmacological interventions.

Keywords: aggression, ASPD, psychopathy, sociopathy, fMRI, brain

1. Introduction

Acts of aggression are often linked to and stigmatized alongside mental health conditions. Nowhere is this association more readily reflected than in violent portrayals of individuals who live with what is most likely antisocial personality disorder (ASPD). According to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*

(DSM-5), ASPD is a Cluster B personality disorder (relating to unpredictable, erratic thinking and behavior) that implicates a pervasive pattern of physical aggression toward others (fights or assaults), a decreased capacity for remorse for harmful actions perpetrated against others, increased disregard for the safety of self and others, as well as lying or other deceitful behavior, among other symptoms. This constellation of symptoms is often represented in film, television, and literature through the figure of the "serial killer": An individual with homicidal impulses—often portrayed as more beast than human—who perpetrates egregious, patterned acts of murder, often with disturbing motivations, rituals, and other aspects related to the murders [1]. While these serial killers might exhibit symptoms like those outlined in the DSM-5 diagnosis of ASPD, these figures are more often identified by one of two words: a "psychopath" or a "sociopath" [2].

It is unclear how the terms psychopath and sociopath offer any helpful definition for conceptualizing the personalities, behaviors, thoughts, or feelings of these characters, let alone for real people. Definitions for these terms appear to attribute arbitrary characteristics that tend to overlap with one another, offering no clarifying criteria with which to meaningfully bifurcate between terms. Often individuals in the West tend to flippantly wield psychopath and sociopath as derogatory terms to lambast and otherize individuals whom they perceive as lacking empathy, perpetually lying, and or acting aggressively toward others [3]. Not only do media caricatures of "psychopaths" and "sociopaths" paint a grim, limiting picture for real people who live with ASPD, but the casual usage of these terms, with no meaningful distinction between them, obfuscates the real challenges that these individuals face. Indeed, acts of violence, among other things, tend show up more often as challenges for individuals living with ASPD [4], but not all individuals with ASPD experience the exact same series of challenges, for they are complex human beings with similarly complex motivations, much like the rest of humanity. Thus, how might aggression be more accurately reframed in the experience of ASPD? And how might the terms of sociopathy and psychopathy be rescued from the derisive public discourse to serve a helpful clinical purpose in this endeavor?

Accordingly, the present chapter of this book will utilize the framework of clinical neuropsychology—or the study of thoughts, feelings, and behaviors and their relationship to brain area activity [5, 6]—to guide this exploration of aggression, ASPD, psychopathy, and sociopathy. This framework not only provides a concrete scientific medium with which measure and observe the neurophysiological impacts of thoughts, feelings, and actions related to ASPD but also legitimizes the real physiological and behavioral challenges of individuals who live with this condition, destigmatizing the egregious bias against all individuals who live with ASPD as blood-thirsty, unfeeling criminals and killers [7]. Thus, within this framework, data from functional magnetic resonance imagery (fMRI) studies will be used to explore the activity of neural substrates implicated in aggression and ASPD. These data will be used to meaningfully differentiate the terms psychopathy and sociopathy, which have clinical significance with respect to the etiology of ASPD symptoms. Lastly this chapter will establish a jumping off point for mental health researchers and clinicians to better understand the neurophysiology and symptomatological etiology of ASPD, contributing helpfully to the process of destigmatization of a fundamentally misunderstood personality disorder.

2. What is aggression?

Before diving into the data and discussion of the neurophysiology of ASPD, it is important that terms are appropriately outlined and defined. First, what is meant by

"aggression?" One might hold certain assumptions about what aggression entails, some of which might be true, but not all of these assumptions are salient with respect to the experience of ASPD. For years researchers have been divided on how to define and conceptualize aggression. Indeed, it is universally accepted and empirically observable that there are multiple types of aggression [8]. Some leaders in the field promote a bivariate framework of aggression, others a multivariate framework [8, 9]; And then there are some who are conceptually agnostic about how to define aggression because presentations are often mixed [10]. Feshbach was among the first to utilize a bivariate model of aggression, outlining "proactive" and "reactive" subtypes [11]. There is also the work of Moyer who devised multivariate categories of different expressions of aggression from his research with animal populations [12]. The categories are as follows: *fear-induced aggression*—an act of aggression toward another animal when one feels cornered and unable to escape; *maternal aggression*—the mother acts aggressively toward a perceived source of threat to the safety of her young; *inter-male aggression*—males of the same species engage in aggressive behavior to compete for resources within a social dominance hierarchy (typically observed among chimpanzees and rodents) [13, 14]; *irritable aggression*—an aggression response to an irritating (whatever that means) stimulus in the environment; *sex-related aggression*—an act of aggression precipitated by a sexual act, typically observed in humans and some animal populations; *predatory aggression*—an aggression act distinct from others in this multivariate model, where an attack response is inspired by viewing an object of prey in line of sight, typically observed in predatory animal populations; and *territorial aggression*—also known as the resident-intruder paradigm in animal studies [15], this aggression response is triggered when one animal (the intruder) enters into the established territory of another animal (the resident). Recent research has expanded upon these complex expressions of aggression in animal subjects [16–18].

With respect to humans, defining aggression is not so simple. Human expressions of aggression often rely on a complex constellation of motivating factors—such as cultural context, experience of race, gender, and sexuality, political persuasion, religious sensibilities, etc.—and mechanisms—such as highly charged affect and strategic action planning. Territorial disputes between nation-states, for example, can involve acts of military-related aggression, but they can also involve complex affects germane to nationalistic beliefs that motivate behavior, and so on [8]. When defining human aggression, there are risks implicated in oversimplifying its phenomenology; namely, common expressions of aggression tend to categorically overlap and do not fall into discrete types, and bivariate models of human aggression do not conform to the complexities of what is known about the decision-making process [19]. Considering these caveats and complexities in the discourse, two definitions emerge that operationalize definitions about human aggression. The first definition comes from a study published in the 90s that stipulates interpersonal human aggression (or aggression between humans) as behaviors perpetrated from an intent to cause harm to someone who does not wish to be harmed [20]. While this definition leaves room for many exogenous factors to be considered, an assumption is made about the motivation of the aggressor, that there is an intent to cause harm. To be sure, causing harm can be extrapolated into a variety of environmental contexts with numerous motivating mechanisms, but there is another general category that summarizes the second half of human aggression: protection. Siegel and Victoroff, for example, propose "defensive rage behavior" as another definitional category through which to conceptualize the intent behind human aggression, where causing harm is of secondary concern to protection in the interaction [8]. Synthesizing these two definitions

of human aggression, therefore, one arrives at the following definition: *behaviors perpetrated from an intent to cause harm to someone who does not wish to be harmed, or from an intent to protect oneself from harm, where the motivation to cause harm is of secondary import*.

Taking this operationalized definition of human aggression, how might it be applied to aggression in the experience of ASPD? While motivations for acts of aggression can vary for individuals living with ASPD, motivations for these same individuals tend to cluster around impulse and premeditation [21]. Within the framework of the operationalized definition proposed in this, motivations for human aggression are accounted for among individuals living with ASPD. These individuals might be motivated to cause harm, whether by impulse or premeditation, and they might be driven to defend themselves, where the secondary harming behaviors could be impulsive or premeditated. It should be inferred, also, that these observations can be refined by complex exogenous factors that influence behavior and motivation.

3. What are sociopathy and psychopathy?

With a definition of aggression established and linked to the experience of ASPD, how might sociopathy and psychopathy factor into the discourse? The term psychopath was coined by a cohort of German psychiatrists in the late-1800s—of whom Dr. Julius Koch was a leading voice—to describe the personalities of individuals who appeared to spurn social expectations and desire to cause harm to others [22]. The influence of this term expanded throughout the early 20th century to describe individuals who violated legal and moral expectations placed upon residents of any given society [22]. Generally, speaking the colloquial use of psychopath carried with it an implicit stigma that these individuals are socially undesirable and thus deemed worthy of castigation. Concomitantly, an American psychologist by the name of Dr. George Partridge devised the term sociopathy to describe similar behavior phenomena to those observed by Koch in years past [23]. According to Partridge, sociopaths are individuals who broadly fail to live up to their community's established norms, and these behaviors pose a threat to the safety of other community members [23]. As has been discussed, psychopathy and sociopathy became popularized in media around the world from the mid-20th century onward and have moved into common parlance in the West to describe individuals who lie and who express any form of aggression toward others, especially when it is premeditated [1–3].

The definitions of psychopathy and sociopathy generated by Koch and Partridge, respectively, appear to refer to the same (or at least similar) constellation of behaviors. Unfortunately, the similarity in definitions obfuscates any meaningful difference between terms. And the definitional criteria themselves appear to be highly arbitrary. What exactly constitutes "causing harm to others" and "spurning social norms?" By these definitions, one might categorize a protestor as a psychopathy or sociopath. After all, their acts of protest presumably upset the status quo implicated in their present social norms, and the powers that be might perceive these acts of protest as an intent of the protestor to cause them harm. Does that mean the protestor is *truly* a psychopath or sociopath based on the virtue of protesting? Obviously not! And herein lies the lack of conceptual integrity (and absurdity) laden in Koch and Partridge's

definitions of psychopathy and sociopathy. This way of thinking about aggression and adherence to social norms perpetuates the systems of White Supremacy and anti-Black racism that are woven into the landscape of mental healthcare and social science research, among many other intersectional challenges [24].

Shifting away from these harmful models of aggression and other behaviors, the task is to helpfully redefine the terms of psychopathy and sociopathy in a way that 1) meaningfully differentiates between terms, 2) avoids arbitrary definitions of aggression and other behaviors, and 3) can be useful within clinical and research settings. Based on what is known about the behavioral challenges implicated in experiences of ASPD, aggression and other behaviors associated with psychopathy and sociopathy might find purchase in the context of this personality disorder; not in a derisive, stigmatizing sense but in a manner that is meaningful for clinical diagnosis and recovery. Therefore, to begin unpacking definitions of psychopathy and sociopathy with respect to ASPD, a brief discussion of neurobiological etiology is warranted.

3.1 Neurobiological etiology

For readers who are unfamiliar, neurobiology in humans implicates the study of cells and cellular network function in the nervous system, which informs cognitive, affective, and behavioral processes. For example, studies that addresses patterns of the release of cortisol during episodes of traumatic stress [25] or problems with serotonergic reuptake in cases of depression [26] are both examples of neurobiology. Neurobiology implicates, also, the study of genetics and epigenetics. Genetics involves the study of genes, gene variation, and heritable traits in organisms associated with DNA sequencing, whereas epigenetics implicates the study of heritable phenotype alterations that are not associated with DNA sequencing. In other words, the expression of epigenetic markers in the human body has the potential to change over time, often motivated by experiences in one's social environment; whereas genetic sequencing is heritable and not necessarily impacted by one's social environment [27].

Historically, sociopathy has been identified as an epigenetic phenomenon, developing because of chronic high stress situations that occur over one's lifetime [28]. For example, individuals who survive chronic child abuse have been shown to exhibit an increased expression of altered mRNA methylation markers over time, and these markers are correlated with an increase in antisocial symptomatology [28, 29]. And while there is no "psychopath gene," antisocial behaviors have been observed in individuals with no history of epigenetic stressors and alterations in certain genetic substrates, often figuring within multiple generations of families [30, 31]. In other words, research shows how ASPD has the potential to develop among individuals 1) who experience chronic adverse life situations during crucial developmental periods [32], or 2) whose family exhibits a history of the disorder [33]. Therefore, a meaningful difference between these two terms emerges: psychopathy is genetic, and sociopathy is epigenetic. And if one recalls the earlier discussion about sociopathy and psychopathy describing similar behaviors implicated in ASPD, it might be useful to conceptualize psychopathy and sociopathy as two different manifestations (or, perhaps, subtypes) of ASPD, differentiated, at least in part, by neurobiological etiology. While this framework might help explain the organic origins of sociopathy and psychopathy, is there evidence that these two ASPD subtypes exhibit different neurophysiology across the human lifespan? And if so, how do their neurophysiology impact expressions of aggression?

4. Aggression and the human brain

Before addressing the neurophysiology of the psychopathic and sociopathic subtypes of ASPD, it is worth circling back to the concept of human aggression and exploring how aggression manifests in the human brain. To frame this exploration of aggression in the human brain, it would be worthwhile to circle back to the definition for aggression offered in this chapter: *behaviors perpetrated from an intent to cause harm to someone who does not wish to be harmed, or from an intent to protect oneself from harm, where the motivation to cause harm is of secondary import*. From this definition, two key words emerge: intent and motivation. And with these key words, two driving forces for aggression emerge: causing harm and protecting oneself against harm. First, there are several regions of interest (ROIs) in the human brain associated with intent and motivation. The orbitofrontal cortex (OFC), for example, takes account of salience outcomes with respect to select social situations, or preferential attention to outcomes associated with the motivation task [34]. In other words, if someone is sitting in their living playing sudoku all while a news program is playing on the television, the OFC will activate and help this person direct their full attention to completing this puzzle while discriminating stimuli coming from the television. There is also the dorsolateral prefrontal cortex (dlPFC) which is responsible for cognitively organizing and executing plans for sensorimotor activity related to a motivation task, often co-activating the primary motor cortex (PMC) and the sensorimotor cortex (SMC) [34, 35]. For example, if someone is motivated to get up in the morning and go for a jog, the dlPFC will prepare the person to get out of bed, get dressed in workout clothes and running shoes, and then stretch in preparation for their jog. Additionally, the anterior cingulate cortex (ACC) activates during situations of motivation and intent by synthesizing attentional concerns and sensorimotor activity, appraising the costs and benefits of engaging in a motivation task [34–36]. Using the previous jogging example, the ACC will activate to help this individual assess whether it is worth it to go for a jog. The ACC might prompt this individual to consider how jogging will help them feel good and contribute to a health goal, or perhaps weigh out concerns about not having enough energy to commute to their place of work after they finish their jog. Lastly, there is the ventral tegmental area (VTA): A neuronal cluster located in the midbrain that is responsible for producing dopaminergic and serotonergic responses to motivation tasks, helping to reinforce these behaviors [37]. And while there are many other tangential and smaller ROIs implicated in the complex processes of motivation [34], the ROIs mentioned above present a general constellation of important regions correlated with motivation in humans.

These ROIs play crucial roles in the motivation or intent behind aggressive behavior. To illustrate these roles, consider the following story. Robert is going for a hike in the Rocky Mountains of Western Colorado. Halfway through his hike he stops to take a break and eat his lunch. Suddenly, Robert hears rustling in the bushes approximately 20 feet from his location. Then, a mountain lion emerges and begins growling at Robert, preparing to attack. While mountain lion attacks are incredibly rare, readers will observe in this example how Robert's brain springs into action to react to the threat posed by this mountain lion. At once, Robert's OFC helps him to focus his attention on the approaching mountain lion and ignore ostensibly irrelevant stimuli and environmental details around him. Simultaneously, Robert's dlPFC recruits is motor cortices to prepare him to stand up, shout at the mountain lion, and throw sticks and stones at it. Robert's ACC synthesizes this information and,

assessing the crucial motivation to preserve his life, decides to throw sticks and stones at the mountain lion in hopes of discouraging it from attacking further. Thankfully, Robert's strategy was a success. The mountain lion initially perceived Robert as a prey animal because he was sitting down eating his lunch. Once he stood up and acted aggressively, the mountain lion perceived Robert as a threat, not a prey animal, and ran away. Among the waves of relief that washed over Robert in the aftermath of this event, his VTA activated and flooded him with dopamine, reinforcing for Robert that acting aggressively and looking intimidating toward a mountain lion might be a useful skill, should he find himself in a similar situation in the future. Readers can assume that Robert's broader motivations for acting aggressively implicated a desire to protect himself against harm, as this mountain lion presented a considerable risk to his physical safety. All these ROIs, as well as countless others, worked together in the span of milliseconds to help Robert survive.

If one discusses aggressive behavior, they cannot ignore the fight or flight response. This behavioral response helps individuals prepare to fend off potential threats in the environment, including by means of acts of aggression. A crucial ROI system implicated in the fight or flight response is the limbic system. The limbic system is a subcortical midbrain network that helps individuals process emotional reactions to various environmental stimuli [38]. Particularly, the limbic system specializes in threat detection and prepares the body accordingly to respond to threat [39]. Two principal ROIs implicated in the limbic system's threat detection and response system are the amygdala and the hippocampus. About the size of a green pea, the amygdala is situated at the front of the limbic system and acts as the primary interface for emotional processing in the human body [40, 41]. The amygdala helps individuals process whether something in the environment is threat as well as complex networks of emotions related to motivation for action [41, 42]. The amygdala helps individuals process if they desire to act aggressively to protect themselves from potential harm (i.e., threat detection), cause harm (i.e., anger, disgust, jealousy, etc.), or some combination thereof [43]. With respect to the motivation to cause harm associated with the amygdala, consider an example of a person named Stacy who sees a fly buzzing around her living room. When Stacy sees this fly, her amygdala might not perceive it as a threat to safety but, instead, as a gross pest. Stacy's amygdala registers feelings of disgust and frustration toward the fly, and these feelings motivate Stacy to act aggressively toward the fly by swatting at it.

Working together with the amygdala in aggressive behavior is the hippocampus. Located behind the amygdala, the hippocampus serves as the primary center for short-term memory operationalization and long-term memory encoding in the human brain [44]. The hippocampus activates when retrieving memories that are associated with feelings processed by the amygdala in real time [45]. To situate these roles of the hippocampus in the context of aggression, consider once more the example of Stacy and the fly. When experiencing frustration and disgust at witnessing this fly buzzing around her living room, Stacy's hippocampus recalls a long-term memory from Stacy's childhood where a fly kept buzzing around her head as she laid on her bed in her bedroom. Stacy remembered that this previous experience with a fly really bothered her, recollecting similar feelings of disgust and frustration. This childhood memory helped prompt Stacy to swat at the fly buzzing around her living room. Thus, the hippocampus serves a crucial role toward accessing encoded memories associated with feelings expressed in the present moment, and these memories help reinforce behaviors associated with these feelings [46].

One might also include the insula as an important ROI implicated in aggressive behavior. Like the amygdala, the insula is a key emotion processing center in the human brain [47]. However, the insula also specializes in registering and processing both emotional and physical pain [47], as both types of pain are undifferentiated by the insula [48]. It has been studied for decades among human and animal subjects that the experience of pain can inspire acts of aggression for a variety of reasons, including protecting against further injury and pain, meting vengeance for a perceived slight, and so on [49]. The insula moderates sensations of pain in the human brain, which can lead to acts of aggression if other ROIs determine that the experience of pain is sufficient to warrant an aggressive response.

Lastly, there are the thalamus and brainstem which play key roles in moderating aggression and the human brain. The thalamus is an egg-shaped structure in the middle of the midbrain region which moderates afferent motor and sensory stimuli from the body to the brain [50]. On the converse, the brainstem is a small stalk-like structure connecting the brain the spinal column and is responsible for channeling efferent stimuli to moderate processes of the nervous system [51]. In effect, the thalamus and brainstem create "loop" of sorts to moderate input from various stimuli, helping the brain and body communicate and function accordingly [52]. This "loop" becomes particularly relevant when the fight or flight response is activated. When someone perceives an external threat in their environment and decides to act upon it by engaging in acts of aggression, the brainstem communicates to other areas of the central nervous system (CNS) that it is "time to fight." These areas of the CNS include the heart, lungs, major muscle and tendon groups, sweat glands, digestive tract, pancreas, and so on [53]. For example, the brainstem informs the heart pump more blood to the extremities; the lungs dilate to increase airflow and oxygenation; muscles and tendons tense up, reinforced from blood sent by the heart; the pancreas sends adrenaline throughout the body to energy the person before fighting; and much more. Accordingly, the thalamus and brainstem are crucial ROIS with respect to helping facilitate acts of aggression.

These ROIs mentioned above are observed to activate typically within most human beings during acts of aggression. The question remains, however, if these same ROIs activate in the same capacity among individuals who live with genetic ASPD (psychopathy) or epigenetic ASPD (sociopathy). Additionally, are there observable differences in neurophysiological activity between these ASPD subtypes? Thus, the following subsections of this chapter will compare extant findings for ROI activity among adults living with psychopathy and sociopathy, exploring crucial neurophysiological differences between each ASPD subtype. Children and adolescents will not be considered in this chapter's collection of findings to account for crucial neuroanatomical changes that occur during development. Findings from all studies in both subsections were collected using functional magnetic resonance imagery (fMRI).

4.1 Neural substrates correlated with psychopathy

Data for ROIs implicated in aggression for individuals living with psychopathy are represented in **Table 1** and organized by areas that are activated and deactivated during aggression. It is to be understood also that "activation" and "deactivation" to not implicate typical neurophysiological phenomena associated with aggression but disordered activation and deactivation of these ROIs to account for discrepancies in how individuals with ASPD respond to aggression stimuli in the environment versus

individuals without ASPD. The same principle applies also for data from the next subsection on sociopathy and aggression.

First, **Table 1** shows that individuals living with psychopathy often tend to exhibit disordered activation of the OFC [54, 55, 58, 59, 61, 62]. As has been discussed, the OFC typically activates when processing motivation to complete and repeat a motivation task [34]. In cases of psychopathy, the OFC appears to activate in a typical fashion, but the activation responses are to atypical reward tasks, often tasks that contrast from values in the individual's cultural context [63]. Thus, the neurological circuitry of OFC itself is not inhibited nor impaired but is disordered by the response impulses it receives from other ROIs. Thus, if other areas of the brain inform someone to crash a car or steal someone's belongings, the OFC will process these behavioral stimuli without moderation that typically occurs in OFC function.

Principally, the OFC is informed by the medial prefrontal cortex (mPFC), which figures often as an area of disordered activation [54–57, 60, 61]. The mPFC is responsible for action planning, impulse control, social/moral behavior, relationship building, problem solving, and other functions, similarly to the dlPFC described earlier [64]. For individuals with psychopathy, however, the mPFC appears to play a key role in how they process relationships, impulse control, and social/moral behavior, which then informs the habituation of behavior through the OFC [65]. There appears to be a marked volumetric difference in mPFC size among individuals living with psychopathy versus those who do not ($p < .004$ Bonferroni correction) [66]. It has also been observed among those living with psychopathy that the mPFC appears to exhibit less functional connectivity between other social/moral behavior and emotion processing centers in the brain [67]. Thus, when translated to contexts of aggression for those living with psychopathy, the mPFC activates consistently but exhibits challenges in sending crucial impulses to trigger empathetic emotional responses, moderate behavioral impulses, and cognitively reflect on motivations for actions. Using the previous example of a person stealing someone's belongings, an individual with psychopathy might have considerable challenges processing how the other person might feel if they stole their belongings, moderating the impulse to commit theft in the first place, and ultimately reflect on the motivation for stealing belongings in the first place. Thus, individuals who live with psychopathy, though they might engage in

Sources	Brain area activation	Brain area deactivation
Anderson and Kiehl [54]	OFC, mPFC, and ACC	Amygdala
Blair [55]	OFC, mPFC, and ACC	Amygdala
Glen and Raine [56]	mPFC and ACC	Amygdala
Harenski and Kiehl [57]	mPFC	Amygdala
Lam et al. [58]	OFC	
Nummenmaa et al. [59]	OFC, ACC, PMC	Insula and amygdala
Patrick [60]	ACC and mPFC	Amygdala
Perez [61]	OFC and mPFC	
Schiffer et al. [62]	ACC and OFC	Insula and amygdala

Note: OFC = orbitofrontal cortex; mPFC = medial prefrontal cortex; ACC = anterior cingulate cortex; and PMC = primary motor cortex.

Table 1.
Brain area activity during aggression in psychopathy.

behaviors perceived as egregious or even heinous in certain cultural contexts, are not sub-human monsters, volitionally devoid of moral character. Rather, they are human beings living with the impacts of a genetically disordered mPFC that does not connect properly to crucial ROIs implicated in various prosocial behaviors.

Lastly, **Table 1** shows a marked deactivation of the amygdala among individuals who live with psychopathy [54–57, 59, 60, 62]. Ordinarily, the amygdala activates in response to threat stimuli in the environment, signaling that the individual should either prepare to fight or run away [40, 41]. For individuals living with psychopathy, however, the amygdala often deactivates when encountering threat stimuli in the environment. This phenomenon is explained in part by decreased functional connectivity of the amygdala with other ROIs, indicating a [decreased capacity for] "contextual fear conditioning, and insensitivity to cues predicting capture" [54]. Deactivation of the amygdala during acts of aggression among individuals living with psychopathy correlates to a lack of stimulation of the thalamus and brainstem, which do not prompt the CNS to activate crucial survival regions in the human body when engaging in acts of aggression [68]. However, on the occasions when the amygdala does activate, it usually occurs during very high stress situations, which often lead to a decreased capacity for amygdalar affective downregulation [54–56]. Thus, individuals who live with psychopathy appear not to respond to aggression stimuli in the environment out of fear or a need to survive, as their amygdalae typically deactivate. However, the motivation to engage in acts of aggression appears to come from their social and moral reasoning, which often does not implicate prosocial reasoning and behavior. Thus, individuals who live with psychopathy appear to engage in acts of aggression because they are human beings who exhibit genetically altered neurophysiological activity in their amygdalae, activity which is often reflected in antisocial behaviors.

4.2 Neural substrates correlated with sociopathy

Data for ROIs implicated in aggression for individuals living with sociopathy are represented in **Table 2** and organized by areas that are activated and deactivated during aggression. In contrast to psychopathy, which is a genetic form of ASPD, sociopathy is an epigenetic form of ASPD, developed primarily by environmental factors that the individual encounters. Thus, at birth, individuals with sociopathy do not tend to exhibit any noticeable neurophysiological differences [78]. Once they encounter major or persistent stressors, like chronic child abuse or community violence, then ROIs begin exhibit disordered activation which could lead to ASPD [79]. Across all data in **Table 2**, individuals living with sociopathy tend to exhibit similar activation and functional connectivity issues with the OFC and mPFC to those living with psychopathy [69–77]. Like in psychopathy, individual living with sociopathy tend to process behavioral stimuli in their OFC in a typical manner, but these inputs are coming from disordered social and moral reasoning and decreased capacity for impulse exhibited by the mPFC [69, 70, 80]. Thus, when considering these specific cognitive functions and behaviors related to aggression, individuals living with sociopathy and psychopathy are virtually indistinguishable.

The primary difference in presentations of sociopathy and aggression, however, come with amygdala activity. According to data in **Table 2**, individuals living with sociopathy appear to exhibit consistent amygdala activation when engaging in acts of aggression [70–77]. In contrast to psychopathy, individuals living with sociopathy appear to exhibit more functional connectivity between the amygdala, thalamus,

Sources	Brain area activation	Brain area deactivation
Blair and Cipolotti [69]	Amygdala, OFC	
Bower and Price [70]	mPFC, OFC, amygdala	
Cipriani et al. [71]	mPFC, OFC, amygdala	
Damasio et al. [72]	mPFC, amygdala	
de Oliveira-Souza et al. [73]	mPFC, amygdala	
Gregory et al. [74]	mPFC, amygdala, insula	
Mendez et al. [75]	OFC, mPFC, amygdala	
Mendez et al. [76]	OFC, mPFC, amygdala	
Tang et al. [77]	ACC, mPFC, OFC, amygdala	

Note: OFC = orbitofrontal cortex; mPFC = medial prefrontal cortex; and ACC = anterior cingulate cortex.

Table 2.
Brain area activity during aggression in sociopathy.

brainstem, and thus the CNS [81]. Individuals with sociopathy appear to respond in more typical fashion to fear and threat stimuli in the environment [82]. To be sure, when compared with individuals living with psychopathy, individuals living with sociopathy exhibited a consistently higher emotional response rate to threat and fear stimuli ($p < .001$) [83]. Thus, a key difference between sociopathy and psychopathy appears to be the nature in which the amygdala activates. However, in the context of sociopathy, amygdala activation correlates only with fear and threat stimuli but not with moral reasoning dependent on cultural values, which is indicated by functional connectivity issues in the mPFC, like those exhibited by individuals living with psychopathy. Accordingly, these data can lead readers to infer that individuals who live with sociopathy appear to engage in acts of aggression due to differential and disordered activation of key ROIs implicated in aggressive behavior.

5. Implications for mental healthcare clinicians and researchers

Findings from this chapter present several implications for mental healthcare clinicians and researchers. First, this chapter categorizes psychopathy and sociopathy as two subtypes of ASPD, one forming genetically (psychopathy) and the other forming epigenetically (sociopathy). This categorization aims to destigmatize the experience of ASPD during clinical work as individuals with ASPD are often castigated as "heartless" or "inhuman" [7]. To be sure, categorizing psychopathy and sociopathy in this way does not make any less egregious the actions that these individuals might have committed, nor render them any less culpable in a court of law for crimes committed. Rather, this categorization helps to engender compassion for these individuals as human beings who live with a challenging and often misunderstood personality disorder.

Additionally, this genetic and epigenetic differentiation could be used as helpful criteria for determining which type of treatment to use in a therapeutic context. Referring to data from **Tables 1** and **2**, individuals living with psychopathy appear to exhibit amygdala deactivation during acts of aggression, whereas individuals living with sociopathy tend to exhibit consistent activation and functional connectivity.

Therapeutically speaking, individuals as having the psychopathy subtype might benefit from interventions that help build fluency with emotional fluency and moral reasoning. For example, clinicians might utilize interventions such as schema therapy (ST) or psychodynamic psychotherapy (PD). ST is a well-studied cognitive therapy that has been indicated for successful long-term use with individuals living with personality disorders [84]. ST helps individuals identify and restructure cognitive schemas associated with unhelpful beliefs, thoughts, emotions, and actions [84]. Additionally, PD is a therapy derived from psychoanalysis that is insight-oriented and invites clients to explore emotional distress through developing skills in self-exploration and self-reflection [85, 86] PD in its short-term iteration has also been indicated for successful treatment for individuals living with ASPD, as it encourages individuals to explore and challenge motivations for tasks associated with aggressive behavior [86, 87]. Either of these interventions might provide some beneficial results for individuals living with psychopathy who seek treatment. For individuals living with sociopathy, an alternative approach to treatment could involve utilizing mentalization-based therapy (MBT). MBT is long-term, manualize, composite psychotherapy that utilizes psychodynamic, cognitive, and ecological aspects to aid individuals with the process of metacognition [88]. MBT has been indicated for use with individuals live with personality disorders and struggle with challenges germane to affect regulation [89]. MBT has the potential to aid individuals living with the sociopathy subtype to reconceptualize their motivations for aggressive behavior, learn effective affect regulation skills, and address cognitive challenges with respect to moral reasoning and affect identification [89]. With this model and these findings, individuals living with psychopathy and sociopathy might receive more suitable treatment that addresses ROIs implicated in their individual mental health conditions.

With respect to researchers, findings from this chapter prompt further investigation into key differences between sociopathy and psychopathy. Namely, further study is warranted concerning differences in amygdala activation and deactivation between sociopathy and psychopathy [90]. Further evidence in this domain could lead to increased understanding in the specific amygdalar mechanisms that are implicated in each mental health condition. Additionally, further study is needed on the role of mPFC in presentations of psychopathy and sociopathy to determine why exactly these functional connectivity deficits correlate with challenges in emotional fluency and social/moral reasoning. Lastly, further study on ACC activity in psychopathy and sociopathy would help increase understanding of the synthesis between cognitive processes and moral reasoning for these individuals. Indeed, further ACC research in this domain could help unravel questions relation to social/moral reasoning challenges associated with frontal lobe regions in the brains of individuals living with sociopathy and psychopathy. With all things considered, this chapter offers helpful considerations for both mental healthcare clinicians and researchers to re-envision ASPD as well as psychopathy and sociopathy, opening tantalizing channels for innovative therapeutic care and groundbreaking research.

6. Conclusion

This chapter explored differences between psychopathy and sociopathy using a neuropsychological perspective, considering differences in neurophysiology and neurobiology with respect to psychological and behavioral phenomena. This chapter offered a definition of aggression in human beings: *behaviors perpetrated from an*

intent to cause harm to someone who does not wish to be harmed, or from an intent to protect oneself from harm, where the motivation to cause harm is of secondary import. It has been shown how individuals who live with ASPD often exhibit aggression utilizing both general aspects of this definition. Additionally, this chapter has discussed how ASPD—and aggressive behaviors exhibited therewith—have the potential to develop both genetically and epigenetically, the phenomenology of which is defined by psychopathy and sociopathy, respectively. This chapter included a discussion about how aggression manifests neurophysiologically and identified key ROIs implicated in aggressive behavior. Data was then collated and presented to distinguish neurophysiological activity between individuals living with psychopathy and sociopathy. This chapter identified that there are similar patterns of disordered activation of the mPFC and OFC individuals living with psychopathy and sociopathy. However, a key difference between both ASPD subtypes was indicated by differences in amygdalar activity; namely, individuals living with psychopathy tended to exhibit amygdala deactivation during acts of aggression, while individuals living with sociopathy tended to exhibit consistent amygdala activation and functional connectivity. These neurophysiological differences explained behavioral differences as well. Individuals living with psychopathy tended to engage in acts of aggression because of motivation from their social/moral reasoning, whereas individuals living with sociopathy tended to engage in acts of aggression in response to typical threat and fear cues in the environment. Findings from this chapter hold several key implications for clinical work and research, including distinguishing modes of treatment for individuals living with these different ASPD subtypes and exploring further how and why each ROI impacted by these conditions contributes to their symptomatology. Overall, however, this chapter presents a fresh perspective on ASPD, psychopathy, and sociopathy, humanizing the individuals living with these conditions, and encouraging innovation in mental health treatment and research for the future.

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