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MAGNETIC RESONANCE IMAGING IN THE LEGAL DIGANOSIS OF ANTISOCIAL PERSONALITY DISORDER

by

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Abstract

Antisocial Personality Disorder, APD, is characterized by manipulation and disregard for the emotions of others, and individuals suffering from this disorder often display behaviors that cause them to end up in the criminal justice system. The disorder prevents these individuals from learning from their mistakes, leading them to commit multiple crimes over their lifetime. Persons with APD can be diagnosed by psychiatrists and psychologists using psychological assessment tools meant to measure the attributes related to the disorder. However, the psychological tools available today are not entirely reliable or well validated and the instruments are not always useful in various situations. Neuroscientists have developed three brain theories that aim to identify the underlying mechanisms causing the symptoms of the disorder, which could lead to additional methods for diagnosis of APD in the legal setting. The first theory, called the Septo-Hippocampal theory, suggests that a dysfunction in the hippocampus causes the symptoms of APD. The second theory, the Amygdaloid theory, suggests that APD is actually caused by a dysfunction in the amygdala. The third theory, Connectivity theory, suggests that there is dysfunction in the connections between the hippocampus and amygdala that results in APD. The emergence of these theories opens the possibility of using Magnetic Resonance Imaging (MRI) or functional MRI (fMRI) technology as a diagnostic tool for APD. However, none of these theories can adequately explain the occurrence of all cases of APD, suggesting that the dysfunction could be anywhere within the brain system(s) that include the septal nuclei, hippocampus, amygdala, and/or other limbic structures. MRI and fMRI scans may prove to be useless in diagnosing this disorder.

Magnetic Resonance Imaging in the Legal Diagnosis of Antisocial Personality Disorder

Is Magnetic Resonance Imaging (MRI) and functional Magnetic Resonance Imaging (fMRI) of the brain advanced to the point that the legal community should be comfortable using it in the diagnosis of antisocial personality disorder (APD)? More specifically, are we ready to include evidence from brain MRI and fMRI in the courtroom when APD is a factor? It is the goal of this thesis to reach an answer to this question. To accomplish this task, it is necessary to review current diagnostic tools for APD that are used in the courtroom as well as discuss the advantages and disadvantages of MRI as a diagnostic tool. To accomplish this last task, it is critical to review the research on MRI and fMRI use that is based in three brain theories of APD. This information should be sufficient to formulate at least a preliminary answer to this important question. This review will focus on MRI and fMRI, which look at organ morphology and activity, respectively, and will not discuss other types of brain imaging, like PET and CT scans.

APD is defined as "a personality disorder characterized by a history of continuous and chronic antisocial behavior that is not attributable to severe mental retardation, schizophrenia, or manic episodes" (Stedman, 2004).. Individuals suffering from APD manipulate and antagonize others without any signs of remorse. They lie, cheat, steal, and may also develop drug and alcohol dependence ("Antisocial personality disorder", 2013). The DSM-V recognizes six essential components in the diagnosis of APD (American Psychiatric Association, 2013). First, the individual must show impairments in personality functioning. This can be seen in one of two ways; self-functioning, either in their identity, which is described as self-esteem derived from personal gain, power, or pleasure, or in selfdirection, which is described as the absence of prosocial internal standards associated with

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failure to conform to lawful and culturally normative ethical behavior, or interpersonal functioning. This impairment can be in either in empathy, which can be described as a lack of concern for feelings, needs, or suffering of others, or in intimacy, which is described as an incapacity for mutually intimate relationships or the use of dominance and intimidation to control others. Second, the individual must exhibit certain pathological personality traits. These traits may fall into one of two categories; antagonism or disinhibition. Antagonism traits may include manipulation, deceitfulness, callousness, or hostility. Disinhibition traits may include irresponsibility, impulsivity, or risk taking. Third, the dysfunctions in personality functioning and traits must be stable across time and situations. Fourth, these dysfunctions cannot be better explained by the individual's developmental stage or sociocultural environment. Fifth, these dysfunctions cannot be better explained by substance abuse or another medical condition, like head trauma. Sixth, the individual must be at least 18 years old (American Psychiatric Association, 2013). Personality, and therefore personality disorders, are generally believed to be determined by a combination of genes and environmental factors. It is believed that individuals are born with genes that make them vulnerable to developing APD but the individual would need to experience certain life events for the disorder to actually develop. Individuals diagnosed with APD often experience trauma during early childhood, including verbal, physical, or sexual abuse or the loss of a parent. These individuals are generally diagnosed with a conduct disorder in childhood, indicating that this disorder develops over time ("Antisocial personality disorder", 2013). These individuals will lie in order to exploit others and will engage in unnecessarily risky behaviors. However, they do not feel remorse for their actions and do not learn from the negative consequences. Because these types of traits and behaviors often result in criminal

activities, it is important for judges and juries to understand that these individuals will continue to reoffend until they are removed from society.

Professionals in the legal system have always been interested in how to diagnose individuals with psychological disorders accurately, and how to use these diagnoses to place the correct amount of culpability on those accused of crimes. The duty of providing accurate diagnoses has traditionally fallen to clinical psychologists who have created assessment tools that rely on a combination of demographics, behavioral history, and the psychologists' own observations to come to decisions about an individual's mental health. However, many neuroscientists have suggested that it may be possible to pinpoint abnormalities in brain structure that cause the psychological symptoms of specific mental disorders (e.g., Castellanos et al., 2002 Sparks et al., 2002, Drevets et al., 2008). If this is true, then it opens the door for these techniques, such as MRI and other brain scanning technology that uncover such abnormalities, to be used as evidence in the courtroom. Currently, scientists are attempting to determine which structures in the brain control certain behaviors and how these might be related to APD (e.g., Anderson et al., 1999; Blair et al., 2001; Degroot & Treit, 2004; Craig et al., 2009; Boccardi et al., 2010) and how MRI, in particular, might be useful in making this connection. If a problem with aspecific structure can be designated as the central abnormality in APD brains and then be reliably imaged in APD brains, this would give mental health professionals and lawyers a more objective and verifiable assessment than what is currently available.

In order to understand APD, neuroscientists have been trying to determine the underlying mechanisms that cause the symptoms of the disorder. There are three prevailing theories in the literature about the neural causes of APD symptoms. The first is the Septo-

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Hippocampal Theory (Blair, Colledge, & Mitchell, 2001), which suggests that a dysfunction in the hippocampus and septum, which controls anxiety reactions and learning, is the main cause of APD symptoms . The second is the Amygdaloid Theory (Sato et al., 2011), which suggests that a dysfunction in the amygdala, which controls fear and anxiety responses, is the main cause of APD symptoms. The third theory is the Connectivity Theory, which contends that both of the Septo-Hippocampal and Amygdaloid theories are partially correct and that both of the systems identified in those theories are involved in the disorder (Craig et al., 2009). Prior to discussing the merits of each of these brain theories, which have been studied using MRI, a discussion of the current legal tools used to diagnose APD for the courts, MRI and fMRI, and the how MRI and/or fMRI might add to a judge or juries understanding of an individual's capacity to be rehabilitated, or not, is relevant.

Current Legal Practice

Today, there are many different psychological instruments for diagnosing APD that are used in legal settings. However, there are only a few that are used regularly in the court system for purposes of informing judges and juries about an individual's mental state . The most common tool is the Psychopathy Checklist – Revised (PCL-R) (Hare et al., 1990). As the test became more popular, it was reworked to apply to populations other than individuals who have already entered the criminal justice system . One such revision, called the Psychopathy Checklist – Screening Version (PCL-SV) (Cooke, et al., 1999), made it possible to evaluate non-criminal individuals. This is important for first time offenders who have no prior criminal history but suffer from the same disorder as individuals who has multiple offenses in their history. It is important to note that scores on PCL-R and PCL-SV are positively correlated with violent behavior and recidivism (Hemphill et al., 2011) and that these instruments are the only ones used consistently in the courtroom. DeMatteo et al.

(2014) recorded how often the PCL-R was used in the court system, and they also asked why the tool was used in a particular case, whether the prosecution or defense requested the test, and whether the results of the assessment were challenged by the opposing side. They found that, from 1991, when the test was published, to 2004, the PCL-R had been used in 87 cases, almost all of which were at the state level. Overwhelmingly, the assessment was requested by the prosecution as a way of proving that the defendant was likely to reoffend because he or she could be diagnosed as antisocial. The results of the test were challenged; challenges occurred in 13 of the 87 cases with only two resulting in successful exclusions from trial. Use of the PCL-R as a legal tool is becoming more popular, and it has been used in 348 cases from 2005 to 2011 (DeMatteo et al., 2014). Overall, the use of this test has been extensive and judges are becoming more comfortable with its outcomes.

Overall, the PCL-R appears to be reliable. Hare, the author of the test, and his associates (2000) found a correlation of .90 between two experienced and educated raters rating the same individual using the test suggesting reliable outcomes given qualified raters. The authors also state that the predictive validity of the PCL-R is "unparalleled" (p. 628) and "unprecedented" (p. 628). The researchers found that offenders who had been labeled "psychopaths" with the PCL-R were four times more likely to be violent reoffenders in the first year after being released from prison (Hare et al., 2000). Overall, it seems that this instrument does a reliable job of identifying individuals with APD within the criminal justice system.

Despite the PCL-R's high levels of reliability, there are some problems with the test that have been pointed out over the years. The biggest problem with the PCL-R centers

around the major controversy in the forensic psychology field over whether the results of this test should be reported in two factors (personality and behavior) or in four facets (affective, interpersonal, lifestyle features, and criminal history) (DeMatteo, et al., 2014). Another problem of the PCL-R is that it is very difficult to use in a non-incarcerated sample because it relies on criminal history as an indicator of personality. Due to this limitation, there has been a need to create new tools that may be used with individuals who have not been accused or tried for any previous crimes, including the PCL-SV (Cooke, et al. 1999). Hughes et al. (2013) evaluated 65 male prisoners on both the PCL-SV and the Psychopathic Personality Inventory (PPI) (Benning et al., 2003). The PPI measures psychopathy in two factors, including one for interpersonal interactions, including dominance and anxiety levels and one for social deviance, including antisocial behaviors and substance abuse and is typically used to diagnose individuals in the general population. They found that scores on both of these measures were positively correlated with APD tendencies but that the subscales of the PPI and the facets of the PCL-SV did not correlate at all. This result raises the question, are the PPI and PCL-SV measuring the same thing? If not, there is the possibility that neither instrument is measuring what psychologists think they are measuring, leaving the court system without a valid test for APD.

As the court cannot be completely sure whether the PCL-R and PCL-SV are actually measuring APD tendencies, the need for an additional assessment tool is apparent. If it could be proven that a specific region or a structure of the brain plays a central role is causing the symptoms associated with APD, an MRI could be an effective way to allow neuroscientists to take a look at a brain scan and tell, with at least moderate certainty, whether the individual in question suffered from APD. This would give the courts a test that all involved, including the lawyers, defendant, judge, and jury, would be able to base life-changing decisions on without any doubt as to their choices. Currently, however, scientists have not been able to determine with any degree of certainty what neural abnormalities in the brain, if a specific abnormalities is at fault, cause these symptoms.

MRI Results in Psychopathy

Only results of MRI and fMRI studies are discussed in this thesis as these imaging techniques are of the greatest interest as potential tools for diagnosis of APD in legal settings. MRI studies look at brain scans produced by a MRI machine, which use a magnetic field in order to produce an image or picture of internal organs including the brain. It measures the differences in the response of protons in hydrogen atoms of water molecules to changes in a magentic field in various types of tissue allowing scientists to see the morphology or shape of the organs. For brain tissue, it allows neuroscientists to see the morphology of brain structures. Studies using fMRI use scans generated by the same basic technology as MRI. However, fMRI measures oxygen levels and blood flow through brain structures rather than changes in water molecules, allowing neuroscientists to examine activity level of the structures in the brain, rather than morphology. Either of these technologies could be useful if APD symptoms are caused by a particular dysfunction in the brain.

In order to understand what is causing the symptoms of APD, scientists have looked at activation as well as morphology of different structures in the brain. It is possible that APD can be linked to dysfunction or malformation of a single structure, or it may have to do with a combination of abnormalities that effect communication between structures and thus neural activation. Overall, it is important to understand what role these structures play in behavior so that neuroscientists can look at structures that are related to classic APD symptoms. Three theories of APD have emerged that focus on brain structures implicated in production and modulation of fear and anxiety, which are linked to the most common symptoms of the disorder.

Septo-Hippocampal Theory

The septo-hippocampal system refers to a group of structures that are connected in a way that allows interaction between the septum and hippocampus. The septum is located in the frontal lobe, while the hippocampus located in the temporal lobe (Kiernan, 2009). The hippocampal formation includes the dentate gyrus and the formation connections with the entorhinal cortex, the subicular area, and the posterior cingulate cortex (Gray & McNaughton, 2003; Degroot & Treit, 2004; Kiernan, 2009).

Structures of the septo-hippocampal system are thought to play a central and general role in learning and memory (Gazzaniga, Ivry, & Mangun, 2009; Kiernan, 2009), and the hippocampus and surrounding structures may also play a specific role in spatial learning and memory (O'Keefe & Dostrovsky, 1971; O'Keefe & Nadel, 1979). Further, Treit and Menard (1999, 2000) have shown that the septum plays a large role in initiating anxiety and fear reactions, and Treit and Menard (1997) found that the hippocampus is important in learning to avoid painful or stressful stimuli. Therefore, these structures also seem to play a role in fear, stress and anxiety.

The Septo-Hippocampal Theory of APD (Sato et al., 2011) states that abnormalities in structures within the septo-hippocampal system are causing the symptoms of the disorder. The septum, which plays a critical role in identifying anxiety inducing stimuli, may function abnormally, causing a person not to feel anxiety over appropriately stress-inducing stimuli. The hippocampus, through its role in learning and memory, plays a role in learned avoidance behavior (Black et al., 1977). When these two structures do not communicate correctly, a person is more likely to take risks and engage in antisocial behavior because either he or she doesn'tfeel stress or anxiety or does not appropriately relate that stress to the cue and so does not learn to avoid situations producing stress or anxiety. The theory contends that APD symptoms are caused by this lack of anxiety over lying, illegal activities, and other types of activities commonly engaged in by afflicted individuals.

As evidenced by typical risk factors of the disorder, as well as the necessary conduct disorder in childhood needed for diagnosis, it is clear that there is a developmental factor to APD. As such, adolescent brains may show indicators of the disorder before diagnosis. White et al. (2013), in another MRI study, compared the size of juvenile participant's cavum septum pellucidum (CSP), which fuses shortly after birth and affects the development of the amygdalae, hippocampi, septal nuclei, which are involved in reward and reinforcement (Olds & Milner, 1954, Baxter & Murray, 2002, Schmelzeis & Mittleman, 1996), and the fornix (Standring, 2008), which is involved in communication between the hippocampus and hypothalamus, and that participant's diagnosis with a disruptive behavior disorder and the severity of the symptoms (Raine et al., 2010). What they found was that all adolescents who were diagnosed with a disorder had a larger than average CSP but not every participant who had a large CSP also had a diagnosis. Also, the size of the CSP did not predict severity of symptoms. This study aimed to determine how developmental abnormalities may relate to APD. The results of this study show that the CSP is related to APD but fail to explain exactly how. Regardless of the specifics, this study further cements the notion that there is a developmental component to the disorder. Overall, this study demonstrates at the size of the CSP, which directly affects the development of the septum and hippocampus, is correlated

with disorders that produce symptoms similar to APD. This further cements the idea that these two structures may be responsible for creating the disorder.

Sato et al. (2011) conducted an MRI study in which participants, half of whom met criteria for APD, underwent an MRI scan. Upon looking at the scans from individuals suffering from the disorder and those who did not, the researchers found that there were structural differences in the superior temporal sulcus, superior temporal gyrus, and the left occipital cortex and posterior cingulate gyrus. The superior temporal sulcus is involved in determining where others are directing their emotion; this can be accomplished by an individual through recognizing head position and gaze direction of others (Gazzaniga, Ivry, &Mangun, 2009), The superior temporal gyrus is involved in processing spoken and written language (Gazzaniga, Ivry, & Mangun, 2009). The occipital cortex and cingulate gyrus are involved in human awareness, such as the ability to perceive stimuli through all senses, psychological understanding, life-purpose, self-actualization, and attention control (Gazzaniga, Ivry, & Mangun, 2009). The second part of the Sato et al. (2011) study involved having interpreters blind to subject condition look at the brain scans from the participants. The interpreters would then decide whether to diagnose them as dysfunctional (i.e., with APD) or healthy. The researchers found that the interpreters were accurate on about 80% of the categorizations. This suggests that the brain differences in the septo-hippocampal area are distinct in individuals with APD and that these differences are uniform enough across the disorder for them to be recognizable to a trained eye. This suggests that MRI could be used to accurately identify individuals suffering from APD.

Boccardi et al. (2010) looked directly at the relationship between shape of the hippocampus and APD using MRI scans. Previous MRI studies (Birbaumer et al., 2005;

Kiehl et al., 2001) had shown that APD symptom severity was negatively correlated with hippocampal volume, but no researchers had looked at the actual morphology of the structure. Boccardi and colleagues (2010) looked at brain scans from individuals who scored high, medium, or low on the PCL-R. Overall, they found no specific areas within the hippocampus with less volume in individuals with APD than in persons without APD. However, the researchers did notice that there was a significant depression along the longitudinal hippocampal axis in psychopathic participants and that the depth of this depression was positively correlated with higher scores on the PCL-R. They also found some evidence that the lateral borders of the hippocampus may be enlarged in those scoring high in APD. Boccardi and her colleagues hypothesize that the high APD scores may relate to structural differences. Specifically, they suggest that because the hippocampus is involved in fear conditioning, which involves avoidance behavior, and because the longitudinal depression may change the way hippocampus functions and thus, the way APD individuals react to acquisition and retrieval in this type of conditioning, the resulting reduced connection of anxiety to appropriate situations may lead to risky behaviors exhibited by these individuals (Boccardi et al., 2010).

Overall, the Septo-Hipoocampal Theory contends that reduced anxiety may be responsible for the observable symptoms of APD. Reduced anxiety may be because the septum does not recognize danger in a functionally normal way, because the hippocampus does not respond to the anxiety in a functionally appropriate way, or because these two structures simply do not communicate with each other effectively. This theory also suggests that, because the disorder is dependent on development (Blair, Colledge, & Mitchel, 2001), APD will look very much the same across individuals. Because specific accidents or injuries particular to an individual are not likely to cause APD, it is possible to pinpoint one or more specific structures that are abnormal in shape or activity across all individuals. This last point would find support from studies finding that it is indeed possible, with fairly high accuracy, to have interpreters assign individuals to the APD or healthy (i.e. not APD) groups just by looking at the shape of a structures in MRI scans.

Amygdaloid Theory

The limbic system is composed of several structures from the midbrain, diencephalon, and forebrain that encircle the brain stem. The structures include, but are not limited to, the cingulate gyrus, hypothalamus, anterior thalamic nuclei, hippocampus, and amygdala (Gazzaniga, Ivry, & Mangun, 2009). Communicating with the rest of the limbic system the amygdala is thought to be a key structure in the system and sits anterior to the hippocampus in the temporal lobe and inferior to the corpus striatum (Kiernan, 2009).

The functions of the amygdalae are tied closely to the basic functions of the limbic system, which include emotion, learning and memory (Gazzanigaet al., 2009). In particular, the amygdalae are thought to control emotion, emotional behavior, learned emotion, and emotional memory (Kiernan, 2009; Phelps & Anderson, 1997), and the structures are thought to be involved in general arousal and to control fear (Phillips & LeDoux, 1992). The amygdalae have a role in metabolic level and restlessness, grooming and dietary choice in animals, and sexual arousal and behavior (Goddard, 1964). It plays important roles in reward and punishment as well as learned avoidance behavior of anxiety-inducing stimuli through communication with the hippocampus (Phillips & LeDoux, 1992). Most importantly, the amygdalae control fear and anger responses (Goddard, 1964).

The proponents of the Amygdaloid Theory of APD contend that the dysfunction leading to antisocial behavior are due to problems within the amygdala and in its communication with other limbic system structures. Abnormalities in this brain structure, whether developmental or related to external injury, result in impaired ability to process fearful expressions and a deficiency in aversive conditioning (Flor et al., 2002). Individuals suffering from ADP have decreased amygdala volume and experience less amygdala activation when completing tasks that include an emotional component (Gordon et al., 2004). However, there have also been studies (Tranel, 1994; Blair, 2001) that looked at brain lesions and whether lesions in this part of the brain could cause what Tranel (2002) called "acquired sociopathy" (p.343) and have shown that individuals with amygdala lesions can exhibit symptoms similar to those typically found in cases of APD. Overall, abnormalities in the amygdala can prevent individuals from feeling the fear that is typically associated with behaviors exhibited by individuals with APD.

An fMRI study by Contreras-Rodriguez et al. (2014) looked at an emotional facematching task for both healthy controls and criminal individuals diagnosed with psychopathy. Participants underwent a psychological assessment in which a trained psychiatrist rated each individual on the PCL-R. Other information, including criminal history and substance abuse, was collected as well. Individuals were presented with a target face and were made to match that face's emotion to one of two probe faces that were presented. The authors found that healthy controls and individuals with APD performed equally on this task. However, MRI data of the participants showed that healthy controls had activation in their amygdala during the task while individuals with APD did not (Contreras-Rodriguez et al., 2014). It is clear that, while the APD individuals may recognize expressions when they are expressed on another human face, a frontal lobe function, they do not feel the emotion themselves. Studies like that from Contreras-Rodriguez et al. (2014), Flor et al. (2002), and Gordonet al. (2004) clearly implicate limbic system structures, and particularly the amygdala, in APD.

Another fMRI study conducted by Rilling et al. (2007) aimed to determine just how amygdala dysfunction could translate not only into the deficits in emotional processing but also into effects on social interactions. The researchers had participants engage in a Prisoner's Dilemma game while in an fMRI scanner. Participants were told to pick either "Cooperate" or "Defect" and a confederate outside the scanner was given the same choice. When the participant and the confederate both chose to cooperate, each made two dollars. If one cooperated while the other defected, the defecter would get three dollars while the cooperater got nothing. If they both defected, each would make one dollar. This game models what the researchers call reciprocal altruism. Interestingly, APD scores did not correlate with a participant's decision to either cooperate or defect. The researchers found that individuals with APD were more likely to defect for the first few rounds, until the confederate was fairly confident that they would continue to defect (called the "tit for tat" strategy) and suddenly cooperate, leaving the confederate with no money (Rilling et al., 2007). This may be because, with their deficits in aversive conditioning, psychopaths do not learn to avoid these situations (e.g., Flor et al., 2002). The fMRI scans from Rilling et al. (2007) supported this claim, showing decreased amygdala activation for those with APD in this kind of situation. Overall, it appears that the amygdala may be involved in how an individual feels after manipulating another person and possibly in their willingness to do so again. As individuals with APD do not experience fear in the same way as healthy

individuals, as evidenced by Marsh & Cardinale (2012) they do not respond well to aversive conditioning. They do not learn to fear an aversive consequence, because they do not relate the consequence with their actions, and so they do not avoid the actions that bring about the consequence (e.g., Flor et al. 2002). These individuals do not feel badly after manipulating another person and so they are less likely to avoid doing it in the future. This lack of fear also extends to a lack of a fear of being manipulated by others (Marsh & Cardinale, 2012) and so they are more willing to put themselves in situations where they may be manipulated.

Ermer et al. (2012)considered the entire limbic system in a holistic way, in an MRI study, as important to understanding APD, as the limbic and paralimbic systems include many structures that are involved in impulsivity, aggression, empathy, and emotional selfregulation, all of which are implicated in APD (American Psychiatric Association, 2004). Ermer and associates looked at white matter volume within the structures of the limbic systems in APD offenders and compared them to healthy controls. They found that those persons who had been diagnosed with APD had less white matter volume in limbic and paralimbic areas. Taking a broader look at control and offender brains, the researchers found that there were no areas in the entire brain where psychopaths had greater volume of white matter than healthy controls (Ermer et al., 2012). These results show that the limbic system can be implicated in psychopathy in a holistic way rather than only in a specific dysfunctional structure-by-dysfunctional structure manner. This introduces an interesting question. It is unclear, based on current research, if the symptoms of APD are a product of pathology in a specific structure, problems throughout the entire limbic system, or in the connecting tissue between the structures. Taken together, research on the amygdala (e.g., Gordon et al., 2004) and the limbic system (e.g., Ermer et al., 2012), both suggest that

individuals with APD simply have smaller amygdalae than healthy individuals and this may cause abnormal function in the limbic system which leads to APD symptoms.

Overall, the Amygdaloid Theory contends that abnormal function in the limbic system, and specifically the amygdala, prevents individuals with APD from experiencing fear. Among other things, this implies that these individuals do not learn from their mistakes, do not fear negative consequences, and consequently, are more likely to engage in risky behavior. However, this theory seems more focused on the function of a particular structure, the amygdala, that is not dependent on the morphology of that structure. This would make MRI scans useful only to determine that there is no physical injury to the brain that might better explain the symptoms. While it is possible that the fMRI may be used to determine level of amygdala activation under certain circumstances, courts would then need to make sure that individuals were being stimulated properly during the fMRI scans. This would make fMRI scans susceptible to much more criticism than a simple MRI scan designed to look at amygdala structural shape and size. However, it is clear that the amygdala plays an important role in APD and cannot be disregarded as a potential root cause of the disorder.

Connectivity Theory

Connectivity Theory examines a pathway that utilizes both the hippocampus and amygdala. It is clear that emotional memories are remembered better than non-emotional ones, whether that be through encoding, storage, or retrieval mechanisms or processes (Cahill et al., 1995; Dolcos et al., 2004). Some studies have recognized the importance of the amygdala and the hippocampus in this phenomenon, as these structures link emotion and memory in a direct pathway, which may allow for more efficient encoding (Maratos et al., 2001; Smith et al, 2004). These structures also work together to control fear responses and fear conditioning. Aversive information, such as a loud noise, enters the brain through the lower auditory system and is transmitted to both the auditory forebrain and the amygdala (Bordi & LeDoux, 1992). If auditory signals occur in conjunction with an aversive stimulus, the amygdala can create a learned fear response. This combined information is then distributed to the hippocampus where contextual information is recieved, and it is thought that this combined neural processing of some benign event and aversive stimulation in connection with contextual input is what provides for aversive conditioning (Phillips & LeDoux, 1992).

The Connectivity Theory of APD states that the problem leading to psychopathy actually lies in dysfunctional communication between the amygdala and hippocampus (Craig et al., 2009). Information does not relay from the amygdala to the hippocampus easily, or at all, and so fear conditioning does not occur (e.g., Phelps & Anderson, 1997; Phillips & LeDoux, 1992). If fear conditioning does not occur, individuals with APD do not learn from their mistakes and consequently do not attempt to avoid unpleasant situations. This may cause a lot of the behavioral symptoms of APD. However, there are other ways that the communication between amygdala and hippocampus may create problems. Craig et al. (2009) suggested, in an fMRI study, that disorders such as Kluver-Bucey syndrome, which is a disconnectivity syndrome that is characterized by aggressive behavior, loss of normal anger and fear responses, and decreased inhibition, and Aggressive Borderline Personality Disorder may show similar patterns of disconnectivity. These researchers studied fMRI scans of eighteen non-incarcerated individuals, nine with high PCL-R scores and nine healthy controls. Looking at activity between the amygdala and hippocampus, Craig and colleagues (2009) found that those individuals with high PCL-R scores had significantly less activity.

However, as this pattern of behavior is not specific to APD (American Psychological Association, 2004), a lot more research would need to be done for this theory to be useful in a clinical or legal setting.

Advantages and Disadvantages of MRI in APD Diagnosis

There are many possible advantages to using the MRI or fMRI as a diagnostic tool in the courtroom. First, MRI scans are very useful for detecting small differences in morphology of brain structure ("What are the risks and benefits of MRI?", 2015). If neuroscientists are able to pinpoint a central cause of APD (e.g., Sato et al., 2011, Rilling et al., 2007, Craig et al., 2009), MRI would make the diagnosis of the disorder much more objective. This means that a diagnosis will hold more weight in a criminal prosecution, ensuring that dangerous individuals are dealt with in a way that is beneficial to society. Secondly, as there is no radiation involved in an MRI scan ("What are the risks and benefits of MRI?", 2015), there are virtually no health risks. This is an advantage over other types of brain scans, such as a CT scan ("U.S. Food and Drug Administration", 2015).

However, the use of MRI scans also has disadvantages. Many scientists believe that the use of the MRI could lead to more accurate diagnoses ("What are the risks and benefits of MRI?", 2015). While some experts might find this to be an advantage, it may also be a disadvantage. Many psychologists believe that when someone is labeled as part of a specific group, they tend to act more like the members of that group (e.g. Link et al., 1989, Bernburg et al., 2006). This is seen often in juvenile delinquents, but can also be seen in adults; once an individual is told that he or she is a "criminal" he or she will continue to engage in criminal activities (Paternoster & Iovanni, 1989). Some psychologists believe that by labeling someone with APD and using an objective measure, which sends the message that there is no room for error in the diagnosis, individuals who might have been rehabilitated will continue with the behaviors that led them to the courtroom. This becomes a problem when someone who does not actually suffer from APD is misdiagnosed using this method. A second disadvantage is that MRI machines are enclosed and require a person to lay still for an extended period of time. It may be difficult to get accused individuals to be cooperative. Additionally, not everyone can get an MRI scan. People with metal devices in their bodies, such as cochlear implants, pacemakers, intrauterine devices, or even dental fillings. On top of this, MRI scans are expensive (Glover, 2014)and may create too much of a burden on federal or state governments if they were expected to pay for the scans.

Overall, the advantages of using an MRI outweigh the potential disadvantages. MRI would make diagnosing individuals with APD, a dangerous disorder, more reliable and it would also decrease the rate of error in diagnosis if APD symptoms have an identifiable neurological cause,.. This wouldassure that defendants who suffer from APD are not put into regular prisons, where they are a danger to other inmates as well as prison personnel. However, unless neuroscientists are able to determine exactly what causes ADP, MRI will not be very useful in diagnosis.

Discussion

The three brain theories of APD that are current in research, reports and discussions within the neuroscientific and legal, as well as other, communities might present opportunities for a more objective assessment of the disorder. The first theory, the Septo-Hippocampal theory (e.g., Sato et al., 2011), suggests that it may be possible to diagnose APD based on the shape and size of the hippocampus. The second theory, the Amygdaloid theory (e.g., Contreras-Rodriguez et al., 2014), suggests that diagnosing APD based on the

size and shape of the amygdala may be a better approach. Finally, the third theory, Connectivity theory (e.g., Craig et al., 2009), suggests that, instead of the actual shape of a structure, APD might be related to activity between two structures. An MRI or fMRI could be used to look systematically at anatomy or levels of activity of brain structures implicated in APD. This examination could then lead to a more objective decision for diagnosis. However, it is unclear whether is it caused by dysfunction in a specific structure, an entire brain system, or in the connections between structures. A review of the research suggests that it is a combination of the three. It is also impossible to make the statement that all cases of APD, which are diagnosed based on observable behaviors, are all caused by the same neurological dysfunction. As such, psychological tests are better tests for APD than MRI scans.

It is important for the legal system to have accurate assessments of APD and other mental disorders because the criminal justice system is largely focused on rehabilitation, for those who can be rehabilitated. If an individual cannot be rehabilitated, the criminal justice system must be used as a way to isolate those individuals from society. Having information regarding what APD is and definitive proof of whether the accused actually suffers from the disorder would allow judges and juries to understand a person's ability to learn from their mistakes and the probability that the person will reoffend. This would mean that someone whom, by the current understanding of the disorder, is likely to commit more crimes if released from prison would be given a sentence that separates them from society for as long as possible.

Over all, the question is whether the MRI is a viable candidate to accompany the PCL-R in diagnosis decisions in the courtroom. While it may be possible to reach a

preliminary diagnosis of APD with the use of MRIs, it is nearly impossible to tell what exactly is causing the symptoms. It is true that there are some studies that suggest a fairly accurate ability of the MRI to diagnosis this disorder, but it is still unclear what underlying mechanism is causing the symptoms. Unless a consensus can be reached and studies done to show this ability, an MRI is nearly useless in the diagnosis of this disorder.. It is likely that the this question will never be answered as the underlying mechanisms are probably too complex to be seen with MRI technology. The best option for this line of inquiry would be to wait until new brain scanning technologies are developed that allow for a better understanding of that complex system that is the human brain.

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