

Aetiology and Treatments of Antisocial Personality Disorder

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Abstract: This paper provides a comprehensive overview of Antisocial Personality Disorder (ASPD) by emphasising its characteristics, pathological causes and treatments. ASPD is identified as a type B personality disorder which is characterised by consistently disobeying social norms and lacking remorse. The following sections discuss the contributors to ASPD from both nature (biological) and nurture (environmental) perspectives, highlighting the essential influences of inheritance and adverse childhood experiences. Specifically, this study explores the abnormalities in brain structures involving the frontal lobe and amygdala, as well as the effects of abuse and ignorance in its development. In addition, this paper recalls the treatment methods, with Cognitive Behavioural Therapy (CBT) exhibiting a higher possibility of reducing violent behaviours, while the psychodynamic approach and pharmacological intervention function to mitigate comorbidities of ASPD. Furthermore, ASPD behaviours in real life have been addressed through analysing the case study of Ted Bundy, illustrating the importance of diagnosis and intervention in the early stages. The findings supply useful suggestions for relevant experts and professors and also help society to better understand the complexity of ASPD.

Keywords: Antisocial personality disorder, Literature review, Aetiology, Treatment methods, Intervention.

1. Introduction

Antisocial Personality Disorder (ASPD) is one of the types of personality disorders in cluster B which has been described as 'the wild'. Psychologists and therapists typically define this cluster to be dramatic and erratic [1]. This adult-only disorder is characterised by behaviour concerns and emotional abnormalities which deviate from the social norms and statistical norms. The common phenomenon of persistent abuse of the rights of other individuals along with inadequate remorse for exhibiting irritability and aggressiveness can adversely impact interpersonal connections. It is challenging to diagnose ASPD directly as it often occurs with comorbidities, such as substance abuse and other personality disorders [1]. In the global world, males are the dominant with this disorder. Taking the United States as an example, the prevalence of ASPD in the whole population is in the range between 1 percent and 4 percent, the annual data is estimated to be 0.2 percent to 3 percent according to the Diagnostic and Statistical Manual of Mental Disorders-Fifth Edition (DSM-5) [1]. As ASPD can only be diagnosed over 18, according to DSM-5, if individuals display devastating

behaviours under the age of 15, they have a higher possibility of developing ASPD in future. For instance, juveniles frequently show enmity towards their surroundings and have a great tendency to have issues with lacking control over their negative emotions, or habitually stealing and scuffling [2]. Currently, the research associated with ASPD focuses on its pathological mechanisms, behavioural manifestations, early intervention and treatments. The United States and Europe have dominated relevant studies, concentrating on individual clinical samples and the judicial system. While in Orient countries, researchers are more likely to investigate the impact of cultural diversity. This present study offers a comprehensive analysis of the aetiology and treatment methods of ASPD from multiple perspectives mainly through literature review. It not only provides practical recommendations for parents to help prevent the development of related disorders in their children but also addresses the requirements for modifying social misconceptions, and aims at reducing social stigma and prejudices associated individuals with diagnosed with ASPD. In addition, the latest results of research exhibited in this paper supply an essential reference for clinical therapists to understand the behavioural characteristics of ASPD along with its recent training progress in more depth. Hence, this study has an exigent social value at either the academic or the practical levels.

2. Aetiology of ASPD

Multifactor gathering together promotes the development of ASPD. The ‘nature versus nurture’ model is used to determine the aetiology reasons through using the literature review method in the following section.

2.1. Nature Perspectives

Recent experiments have started to verify that genes could potentially influence the development of antisocial behaviours. According to a Canadian study, researchers aimed to determine whether the relationship between the monoamine oxidase A (MAOA) gene and aggression remains constant over the entire distribution of exposure to violence. The experiment finally involved 327 boys from French Canadian kindergartens in Québec and all of them had experienced abuse by their parents, either sexual or physical [3]. The original sample consisted of two subgroups: 2000 randomly selected students (49.95% girls) with normal behaviours, and 1017 children (about 41.7% girls) with the highest level of behavioural issues, assessed through using a Social Behavioural Questionnaire [3,4]. Researchers then chose common single nucleotide polymorphisms (SNPs) upwards the transcriptional site five kilobases to investigate the differences between participants’ genotypes. After comparing the results with the referential scale, 327 valid data remained (standard critical value: -2.33, $p=0.001$). At the ages of 15 and 21, participants will be assessed again with the Diagnostic Interview Schedule for Children (DISC) and DIS under semi-structured interviews based on DSM-III-R [5]. The findings indicated that suffering physical abuse was linked to participant’s mental and physical assaults. The eventual results illustrated a non-linear correlation between the MAOA gene and violent behaviours, implying that genetic moderation only occurs when individuals encounter extremely high levels of aggression. All five MAOA SNPs were associated with antisocial symptoms, with two of them (rs5906893 and rs979605) contributing the most. Individuals with low-frequency alleles exhibited higher rates of violence in their relationships and had more apparent antisocial traits in comparison with other individuals who contained high-frequency ones (see Fig.1) [3]. One concern of this study is after assessing from several different scales, the remaining valid sample consisted solely of males, therefore the results could lead to gender bias as the gender ratio was entirely imbalanced. However, the ultimate sample could verify that males are more likely to obtain ASPD than females.

ASPD can be considered a neurodevelopmental disorder as it meets traits such as having brain anomalies and neurocognitive deficits [1]. The amount of evidence could support a correlation between brain structures and ASPD as previous studies suggest that the frontal lobe and limbic system are strongly associated with the features of the disorder. Researchers used the MRI technique to scan the dimensions of the limbic system in the early stages of people with ASPD and compare their shapes with typically developing (TD) persons. Cavum septum pellucidum (CSP) is composed of the lateral ventricles separated by two leaves of glia. The normal brains of TD persons showed a more open space than the ASPD patients (see Fig.2) [6]. Investigators found that either adults or juveniles detected with CSP all obtained higher marks on ASPD-relevant assessments. Hence, their findings indicated that individuals with limbic developmental abnormalities are more likely to be diagnosed as ASPD patients [6]. Moreover, psychologists found that the amygdala showed a reduction in its volume and function in ASPD patients [7]. The researchers scanned the situations of the cerebrum cortex and they found that the frontal and temporal lobes showed impairment as well. The results showed that the prefrontal cortex (PFC) could function in contributing to ASPD as TD individuals who encountered accidents in the PFC will have the probability of experiencing antisocial behaviours [7]. The phenomenon of the increasing volume of brain tissue also occurred in people with ASPD. For example, the volume of the striatum amplified by 9.6% [8]. The main function of it is to provide rewards for relevant signals during cognitive processes. It has been verified that the increasing concentration of dopamine secreted in the striatum has a relationship with psychopathic behaviours. People with ASPD exhibited more interest in intrinsic motivations and did not care about the punishment for their threatening behaviours [9].

Raine used meta-analysis to further demonstrate the dysfunction of the brain structures had a strong inevitable association with antisocial behaviours. That is, poor behaviours led by prefrontal deficits have influenced the forming of an antisocial personality, with the entire effect $d=0.44$ [6]. Matthys and his colleagues guessed that the neurocognition of the amygdala might have deficits because its function is to deal with emotional information [10]. Therefore, by comparing several traits such as fear and annoyance, they discovered that people with ASPD exhibited very insensitivity to fear, which suggested the reason why patients always committed illegal actions. In addition, as mentioned earlier, the striatum functions to deal with cognition. The lower sensation of rewarding actions in the patients resulted in a lack of attention to social norms. Hence, people with ASPD showed deficits to conform normal social behaviours.

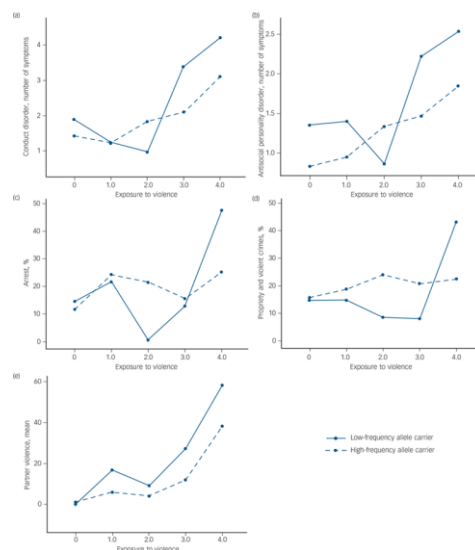


Figure 1: The comparison of the aggression level between low-frequency allele carriers and high-frequency allele carriers [3].

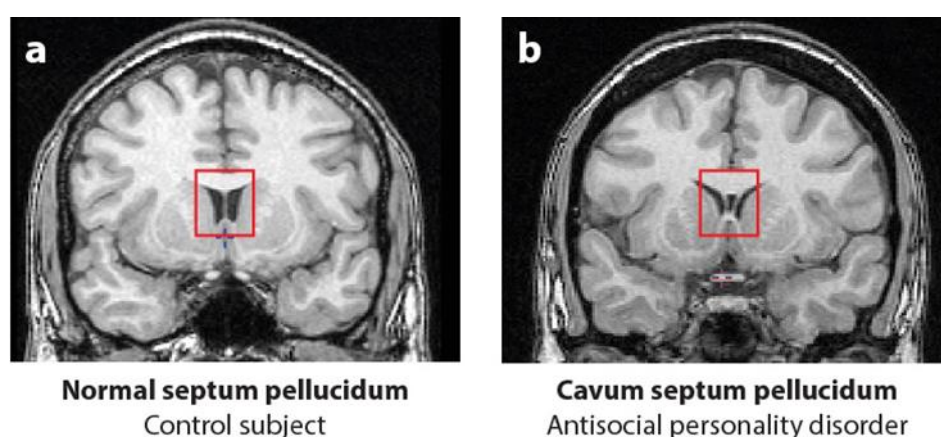


Figure 2: The comparison of the volume of cavum septum pellucidum between normal individuals and ASPD patients [6].

2.2. Nurture Perspectives

Research on the association between adverse childhood experiences (ACE) and ASPD suggests that abuse and neglect are risk factors for ASPD. The first factor is the effect of child maltreatment on the incidence of ASPD. A study conducted in 2008 examined the relationship between child sexual abuse (CSA) child corporal punishment or abuse (CPA) and mental disorders (e.g. ASPD) in early adulthood. The study proposed that children who had been sexually abused were 2 to 4 times more likely to develop ASPD between the ages of 18 to 25 than children who had not been sexually abused [11]. Similarly, the rate of ASPD among those who have been physically abused is 2 to 7 times higher than among those who have not been abused. Therefore, the researchers argued that physical abuse was a predictor of ASPD symptoms, and they suspected that physical abuse caused children's feelings of hostility and distrust toward adult authority figures, which laid the foundation for ASPD [11]. In addition, since ASPD is often associated with substance dependence (SD), subsequent research has expanded the category of ACE from child abuse to drug abuse (such as alcohol and cocaine), witnessing or experiencing violent and serious crimes, and CSA or CPA. As a result, those three types of ACE increased the odds of developing ASPD by 1.47, demonstrating a relationship with the prevalence of ASPD [12]. When it comes to neglect, to examine whether it has the same effect as abuse on ASPD prevalence, the investigators used data from the Collaborative Longitudinal Study of Personality Disorders, including abuse (emotional, verbal, physical, and caregiver) and neglect (physical and emotional) [13]. The findings showed that verbal abuse and caregiver sexual abuse were associated with increased odds of children developing ASPD. One explanation for caregiver sexual abuse is that caregivers who exhibit symptoms of ASPD (or antisocial behaviours) are more likely to develop ASPD in children [14]. In addition, concerning neglect, researchers also using data from 'Delinquent Development' study from the University of Cambridge, identified that among several risk factors for ASPD in children and adolescents, paternal disinterest in their children or parental disinterest in their children's education increased the risk of ASPD [15].

Social stigma experienced by children in early childhood has a negative impact on their mental health and might contribute to ASPD. Out-of-home care (OOHC) is defined as a service provided to children who cannot be cared for by their family of origin due to birth Out of wedlock, family breakdown, death of a parent, or abuse. An and his colleagues demonstrated that there is an association between social stigma and the prevalence of ASPD. The study examined the effects of stigma on the development of children living in OOHC environments, focusing on self-esteem and antisocial behaviour [16]. The study used stratified sampling to collect data from five OOHC institutions (341 children aged 11 or 12 years) between 2011 and 2015 and analyzed the findings

through Potential Growth Modeling (LGM). Researchers found that individuals show different levels of stigma at the age of 11 or 12, and the level of stigma may decrease with time (average intercepted value: 2.146; $p < 0.001$, average slope: -0.038; $p < 0.001$) [16]. Subsequently, they compared the development of OOHC children with that of the general population and found that OOHC children had slower physical development and poorer mental health (including, being more likely to feel depressed, socially excluded and devalued), speculating that it may even lead to negative developmental outcomes [16,17]. Secondly, the stigma initially received at age 11 or 12 had a statistically significant negative impact on their self-esteem four years later. In addition, over five years between ages 11-12 and 15-16, the results of the stigma slope suggest that stigma has a negative impact on self-esteem and is an influential factor in antisocial behaviour [16]. The results suggest that social stigma is a contributing factor to ASPD.

3. Treatments of ASPD

Each psychological disorder has its most appropriate treatment to try to cure the symptoms of the patients and the abundance of them could be mitigated by the same methodologies.

3.1. Psychological Perspective

Pieces of evidence could verify that cognitive behavioural therapy (CBT) could lighten the condition of ASPD patients' substance abuse such as drug addiction [18]. However, inadequate information cannot promise that using CBT can assist patients to perform better in other aspects. Therefore, Davidson and his colleagues aimed to see whether utilizing CBT could reduce violent behaviours as well as increase health extent in people with ASPD [19]. The sample consisted of 52 males between the ages of 18 to 65 years old either from London or Glasgow and had been diagnosed with ASPD based on DSM-IV [19]. None of the participants had any extra Psychological counselling in the six months before engaging in the experiment. After having assessments, they were divided into two groups: CBT (25 males) and treatment as usual (TAU) (27 males). CBT emphasized the behaviours that inhibited communication and functional adaptations in addition to thoughts about oneself and other people. It also encouraged participants to bravely express their issues with self-cognitions. To help the participants achieve the optimum effects, they were all asked to attend 15 one-hour counselling lessons over six months or double lessons in one year. As for the participants in TAU, they received normal treatment in the communities they lived. To assess the acceptance of therapy and the influences on the desire to have attacking behaviours, participants underwent a semi-structured interview along with the marking scale. Consequently, participants in both CBT and TAU showed reductions in aggressive behaviours, but those who received CBT could control their drug-abusing symptoms better than TAU ones. Their interviews also indicated that the participants who gained CBT generally exhibited higher satisfaction compared with the ones who belonged to TAU (4.0 versus 3.2). However, TAU participants were more willing to have deeper contact with therapists [19].

The development and alteration of interpersonal communication in the counselling process is the core of psychodynamic theory. Establishing mutually long-lasting relationships along with fostering satisfying self-recognition are the two essential goals of the development of personalities. They are likely to be the obstacles in treating processes [20]. This approach could be used to mitigate the symptoms of other comorbidities of ASPD such as anxiety disorders or borderline personality disorder and has been verified to be effective. However, ASPD cannot be cured by psychodynamic treatment separately and the therapists are expecting to see progress in future [21].

3.2. Pharmacological Perspective

There is no specific drug approved by the Food and Drug Administration (FDA) in the United States to mitigate the symptoms of ASPD at present. However, some of the existing medicines such as antidepressants, antipsychotics and other mood stabilisers could be used to help individuals with a lower extent of antisocial behaviours to manage their violence and control their moods such as rage and depression. The function of antidepressants is to regulate the level of serotonin in human brains to obtain more stable emotions and the type widely used is selective serotonin reuptake inhibitors (SSRIs) [22]. The drugs are useful as ASPD often co-occurs with depression. In addition, antipsychotics could also play a role in regulating the behaviours of people with ASPD as the function of this type of drug is to reduce the level of aggression.

People with ASPD are always been labelled as psychopaths, and they occasionally are stigmatized as being malevolent [23]. Smith and his colleagues found that individuals commonly thought ASPD patients were dangerous based on the data collected from their survey [24]. Even in the justice administration, people with ASPD were still labelled to be aggressive and impossible to treat [23]. Therefore, to inquire into whether the misconception still appears in society at present, a questionnaire has been generated to investigate the awareness and attitudes of Chinese people toward ASPD.

4. A real-life example of people in ASPD

Research on a sample of serial killers has found a very high incidence of antisocial personality disorder and narcissistic traits in their personality profile [25,26,27]. The classic case is Ted Bundy, a serial killer who was active from 1973 to 1978.

He was arrested by Highway Patrol officers for a traffic violation in Utah in 1975, while he was a third-year law student at the University of Utah and a suspect in several murders of women [28].

Bundy, who was in prison on suspicion of murdering "Caryn Eileen Campbell", jumped out of a second-storey window in the library behind the courthouse on 7 June 1977 and was recaptured by police after breaking his leg and running into the mountains to hide for six days. Ted's second escape occurred on 30 December 1977, when he managed to drill a hole in the ceiling of the prison through an intense diet and escaped [28].

Ted Bundy had "gamed" the justice system, and studies have shown that he would satisfy his need for thrills by stealing before committing murder.

Research suggests that he would satisfy his need for excitement before murdering by stealing, which he seemed to regard as a relatively relaxing form of recreation [29].

Ted has admitted that he is aware that killing is wrong and will, therefore, rationalise his actions [29].

On the other hand, it seems that Ted would engage in some very typical legitimate leisure activities during the cooling-off period after the killings [30], which may also help him to resolve the conflict that regulates him internally after realising that he is a serial killer when he considers himself a normal human being [29].

From his range of experiences, it is clear that he has a very typical antisocial personality disorder. He lacks empathy and enjoys murdering his victims after raping and torturing them. He has never been remorseful after committing a crime, taking pleasure in toying with law enforcement officers and in a series of criminal acts from beginning to end, even stating that "at this stage, the hunt was all mental, a game".

5. Conclusion

In summary, this paper comprehensively analyses the aetiology of Antisocial Personality Disorder (ASPD) and its treatments through the literature analysis method, revealing the complexity and

multiple causes of this mental disorder. The findings suggest that the formation of ASPD is influenced by a combination of both natural and environmental factors. Genetic abnormalities, especially variations in the MAOA gene, have a significant non-linear relationship with violent behaviour, while developmental abnormalities in brain structures, especially functional deficits in the prefrontal cortex and amygdala, are closely related to the core symptoms of ASPD. In addition, adverse experiences such as abuse and neglect during childhood provide the basis for the development of ASPD, and these environmental factors often increase the risk of ASPD through their negative impact on an individual's mental health.

In addition to this, our study has some social value. First, research on the aetiology of ASPD provides practical advice to parents and educators to help them prevent the development of psychological problems in children at an early age. Second, society's misconceptions and prejudices about people with ASPD often exacerbate their plight, and by revealing the pathological mechanisms behind ASPD, this paper helps to reduce the social stigma attached to these patients. Finally, clinical psychologists can use the evidence provided by this study to better understand the behavioural characteristics of patients with ASPD and apply methods such as cognitive behavioural therapy (CBT) in their practice to help alleviate violent behaviour and other symptoms in patients.

Looking forward, the limitation of this paper is that, cultural differences, which are significant in shaping an individual's behaviour, were not discussed. Future research should focus on longitudinal studies to observe the development of ASPD over time, to provide more effective treatment and care for patients with ASPD through multi-level research and practice application.

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