Etiology of Antisocial Personality Disorder: A Review of Brain Function and Childhood Experiences

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Abstract. Antisocial Personality Disorder (ASPD) is a psychological disorder in which an individual engages exclusively or significantly in the deliberate violating of, scamming, as well as violence against other people for their gain. This review aims to provide a comprehensive account of the recent developments regarding biological, neurocognitive, and neuropsychological factors and environmental risks related to ASPD. Neuroimaging studies have been conducted to assess brain function using voxel-based morphometry (VBM) and imaging of functional magnetic resonance (fMRI) to determine structural and functional changes that occur. Previous studies have recognized marked grey matter volume (GMV) disruptions in the prefrontal cortex, temporal lobes, parietal regions, and aberrant functional connectivity profiles — particularly between attention and default mode networks. Of the environmental factor, childhood trauma is crucial for leading to necessary support of ASPD phenotype with emotional or physical abuse during infancy, significantly contributing to risk-prediction capacity—the systematic review methodology involved six critical evaluations of the study on neurobiology and childhood ASPD risk factors. Consistent neuroimaging data on brain structure (grey matter volume reductions) and functional connectivity and the critical role of early life experiences in susceptibility to this disorder are shown throughout. In conclusion, ASPD's etiology is multifactorial, involving a complex interaction of biological and environmental factors. Understanding the neurological underpinnings and developmental experiences leading to ASPD can inform more targeted interventions and treatment strategies for at-risk individuals, highlighting the need for early detection and therapeutic approaches to address brain function deficits and childhood adversities.

Keywords: Antisocial personality disorder, ASPD, brain function, childhood experience.

1. Introduction

Investigating the etiology of ASPD has enormous public health and social implications. Individuals with ASPD diagnoses are highly overrepresented in criminal justice systems, and the disorder is strongly linked with violence, substance abuse, and recidivism. In addition, ASPD is notoriously hard to treat since classic therapeutic approaches have rarely been successful. Consequently, the root causes of ASPD must be identified for more effective strategic intervention and better treatment outcomes. Although research has provided valuable insights, there are still gaps, particularly in understanding the interplay between brain function and environmental influence on shaping antisocial behavior.

Past researchers have persistently pointed out the involvement of structural and functional brain abnormalities in the etiology of ASPD. Neuroimaging studies have demonstrated significant reductions in gray matter volume (GMV) in the prefrontal cortex, temporal lobes, and parietal regions implicated in social information processing, empathy, and cognitive modulation. Studies on functional connectivity have also shown that ASPD patients exhibit abnormal activity patterns of brain networks, especially in the interaction between the Default mode network (DMN) and attention-related brain regions. Thus, it supports the idea that neurobiological underpinnings for ASPD intersect well with disturbances in social cognition and emotional regulation, hence giving rise to the antisocial behaviors in the disorder. However, these neurobiological factors only partly explain ASPD and the disorder is far more complex. Childhood experiences, with particular emphasis on trauma and adversity faced by a child, become essential considerations for the development of ASPD.

There is a wealth of literature that ascertains not only a higher risk of experiencing childhood abuse and neglect but also other adverse events among those with ASPD. Early exposure to violence and dysfunctional family settings seriously disrupts emotional and behavioral development, giving rise to patterns of aggression and rule-breaking behavior that are hallmarks of ASPD. Despite these insights, the specific pathways through which childhood experiences has a compact connection with the development of ASPD and how these factors interface with neurobiological vulnerabilities have received scant attention.

The present review tries to bridge the gaps in the existing literature by synthesizing information on ASPD's neural and environmental causes. This review embarks on the integration of neuroimaging studies on brain structure and functioning with those on childhood trauma and early deviant behaviors to explain the etiology of ASPD. This review methodically draws on significant research on VBM, fMRI, and systematic reviews regarding childhood trauma and aggression. Based on the review, researchers can better understand the possible causes of ASPD and some of the treatment methods that have been attempted in the past.

2. ETIOLOGY OF ASPD

2.1. Brain Function Perspective

Research on the neural correlations of ASPD has advanced significantly, showing the complex interaction between brain function and antisocial behavior. This summary integrates six critical studies to provide an overview of how brain structure and function are implicated in ASPD, focusing on gray matter volume (GMV) reductions, functional connection, and the impact of traumatic brain injuries (TBI).

Several studies focus on the connection between brain morphology and psychopathic traits. Based on the study of a sample of male violent offenders, all of whom had been convicted of severe crimes, conducted by Hofhansel et al. and voxel-based morphometry (VBM), people with higher scores in the Psychopathy Checklist-Revised (PCL-R), particularly on the antisocial behavior, they had reduced GMV in the prefrontal cortex, temporal regions, and parietal lobes. Specifically, these offenders exhibited GMV reductions in the right superior frontal gyrus, right middle and superior temporal areas, and the left inferior parietal lobe [1]. This finding conforms to a report from Gregory et al., which shows significant GMV reduction in the anterior rostral prefrontal cortex (Brodmann area 10) and temporal poles (Brodmann area 20/38) among violent offenders with antisocial personality disorder and psychopathy (ASPD+P) [2]. These regions, where deficits may contribute to antisocial behavior, are critical for social information processing.

Research from Tang et al. employed fMRI to explore the neurobiological underpinnings of antisocial personality disorder (ASPD) by identifying abnormal functional connectivity patterns in the brains of affected individuals. To enhance the accuracy of their analysis, they designed a machine learning classifier designed to learn from data and make predictions. This classifier could distinguish ASPD subjects from healthy controls with an accuracy of 86.57%, achieving a sensitivity of 77.14% (correctly identifying ASPD individuals) and a specificity of 96.88% (correctly identifying non-ASPD controls). The study found that ASPD subjects' most significant neural alteration was uncoupling the default mode network (DMN) and the attention network [3]. Significantly, here are some areas identified as regions with high discriminative power as the precuneus, superior parietal gyrus, and cerebellum, indicating that these regions may be critical to the functional deficits observed in ASPD. This approach is the same as previous findings by Schiffer et al., who observed structural brain abnormalities in violent offenders, such as increased GMV in the mesolimbic reward system, including the amygdala and nucleus accumbens and decreased GMV in the insula, linking these regions to dysfunctional emotional and reward processing in ASPD [4].

Maresca et al. systematically reviewed the relationship between TBI and aggressive behavior. The review included 27 studies from an initial 738 articles, highlighting that individuals with a history of TBI, particularly those injured at a young age, exhibited higher levels of aggression and antisocial

behavior [5]. Neuroimaging studies within this review pointed to damage in the frontal lobe, insula, and limbic system as crucial contributors to these behavioral outcomes. This review complements findings from Hofhansel et al. and Gregory et al., highlighting brain regions' role in exacerbating antisocial behaviors through similar neural pathways.

Miles et al. investigated irritability, anger, and aggression (IAA) trajectories in veterans with TBI, identifying four distinct subgroups based on IAA scores: no IAA at admission or discharge (25.72%), resolved IAA (17.63%), delayed onset IAA (8.96%), and persistent IAA (47.69%) [6]. The study found that the only consistent predictor of IAA across these subgroups was the more significant post-traumatic stress disorder (PTSD) symptoms. Notably, the research highlighted that specific brain regions, particularly those took part in the regulation of emotion and cognitive control—such as the frontal and temporal lobes—were often implicated in the persistence of aggressive behaviors post-TBI. This finding underscores the critical position of PTSD in the development of these behaviors. It suggests that targeted interventions must consider TBI's psychological and neurobiological aspects. The study's conclusions conform to the neurobiological findings of Schiffer et al. and Tang et al., giving more highlight on the importance of brain changes in dealing method of ASPD.

The convergence of these studies illustrates a multifaceted etiology of ASPD rooted in structural and functional brain abnormalities. GMV reductions in the prefrontal cortex, temporal regions, and parietal lobes are recurrent findings, suggesting these areas' crucial roles in cognitive control, empathy, and social behavior. For example, Hofhansel et al. reported significant GMV reductions in the superior frontal gyrus, middle and superior temporal regions, and inferior parietal lobe [1], while Gregory et al. found reductions in the anterior rostral prefrontal cortex and temporal poles in violent offenders with ASPD+P [2].

Additionally, disruptions in functional connectivity, particularly between the attention network and the default mode network, highlight the dynamic interplay between different brain regions contributing to antisocial behavior. Tang et al. demonstrated that these connectivity disruptions were primarily between the default mode network and the attention network, with the superior parietal gyrus and precuneus showing the highest discriminative power [3].

The impact of TBIs further complicates the picture, as brain injuries can exacerbate existing vulnerabilities in brain structure and function, leading to increased aggression and antisocial behaviors. Maresca et al. highlighted that TBIs, especially those occurring at a young age, are associated with a higher possibility of aggression and antisocial behavior due to damage in special brain areas.

Schiffer et al. found that violent offenders had more significant GM volumes in the mesolimbic reward system and less GM volume in the insula, highlighting the distinct neural profiles associated with persistent violent behavior and substance use disorders. The consistent finding across studies that specific brain regions, such as the nucleus accumbens and some other areas, are involved in both psychopathy and ASPD emphasizes that a single treatment method is not enough to solve the causes of this disorder.

These integrated findings suggest that neurological factors are partial determinants of ASPD, with significant contributions from brain structure and function abnormalities. The consistent involvement of specific brain regions across studies highlights the importance of targeted interventions that address these neural deficits to manage and treat ASPD effectively. For example, addressing PTSD symptoms in veterans with TBI, as suggested by Miles et al., could be a critical component of comprehensive treatment strategies for ASPD.

2.2. Childhood Experience Perspective

Many early studies showed that childhood development is significant in the etiology of ASPD. Several studies focus on childhood trauma and adverse experiences, which are foundational for the derivation of ASPD. For example, Schorr et al. conducted a study of Childhood trauma (CT) and parental bonding (PB). They found that of 346 hospitalized cocaine users, physical and emotional hurts were the factors connecting with ASPD most closely. It showed that unfortunate childhood

experiences, especially from trusted people, can significantly influence the development of ASPD [7]. They used machine-learning approaches to demonstrate their results. The study revealed complex interactions between different childhood traumas and parental bonding, indicating that early experiences of abuse will profoundly impact personality development and force children to antisocial behavior. This result was the same as the study from Moreira et al., who researched the connection between psychopathic issues and adverse childhood experiences (ACEs). According to PRISMA procedures, they obtained much data from multiple databases to support their research. 12 of the 77 documents were identified as eligible for inclusion and chosen for further analysis; besides databases, there were also 7 selected manual searches, contributing to a total of 19 studies. This research guided the conclusion that more negative samples experienced a higher degree of hurt in early life than less negative samples. The results were also powerful evidence for academic arguments about psychopathic subtypes [8].

Beyond trauma, early behavioral patterns, particularly aggression, are also critical indicators of later ASPD. Schaeffer et al. collected teachers' evaluations of 297 urban, primarily boys from grades 1-7 to identify different trajectories of aggressive behavior in boys from elementary through middle school [9]. Four distinct trajectories of aggressive behavior were identified: Chronic high aggression - Boys' aggression was high from the beginning and remained high throughout elementary school. Moderate aggression - Boys consistently displayed moderate levels of aggression. Increasing aggression - Boys' aggression ranged from low to high but increased over time. Low aggression - Boys consistently displayed low levels of aggression. The study found that boys who followed chronic high or increasing aggression trajectories were at a significantly higher risk for developing conduct disorder (CD), juvenile delinquency, and, ultimately, ASPD. It supports the notion that persistent and escalating aggression during formative school years is a developmental pathway to antisocial behavior.

Furthermore, the study emphasizes the important role of intervention and identification in children displaying aggressive tendencies to prevent the progression to more severe antisocial behaviors. Similarly, DeLisi et al. research focused on the environmental factors in the formation of ASPD, with a focus on adverse childhood experiences and child psychology. Childhood negative experiences, including various forms of poverty and abuse, are essential factors in the sample experience of antisocial behavior [10]. It underscored the significant impact of early abuse, especially those involving aggression and rule-breaking, as direct pathways to ASPD.

Parental influence and environmental factors in early childhood further promote the enhancing of ASPD. Glenn et al. believed that the classification of ASPD in the Diagnostic and Statistical Manual of Mental Disorders (DSM 5) was not detailed enough, so they studied the differences between the subtypes of ASPD [11]. At the same time, they discussed research on the etiology of ASPD and found that some genetic and environmental factors can lead to the development and persistence of antisocial behavior. In addition, Glenn et al. also conducted brain imaging research and preliminary research on ways to cure ASPD symptoms. The study by Schorr et al. also emphasized the importance of parent-child relationship factors in the development of ASPD. The data showed that physical and emotional hurts were usually associated with higher ASPD values; that is, poor parental care in childhood would greatly promote the formation of ASPD. From this, it can be concluded that early childhood care and the quality of relationships between parent and child are vital determinants of the formation of ASPD symptoms, further highlighting the importance of better early childhood education and a more stable family parent-child environment in reducing the risk of ASPD.

The methods of development towards ASPD are influenced by environmental factors and neurobiological factors influenced by early experiences. Glenn et al. reviewed brain imaging studies in previous data. They found some findings in the field of neurobiology: Studies have shown that childhood abuse and long-term neglect in parent-child relationships are more likely to cause changes in brain structure, such as a reduction in the volume of the prefrontal cortex, which is critical for emotion regulation and impulse control. These neurobiological changes are believed to be the cause of emotional and behavioral disorders in ASPD patients, and research on this physiological lesion

will also help further efforts to treat ASPD. This conclusion is like the developmental model proposed by Schaeffer et al., which suggests that early aggressive behavior, influenced by environmental and biological factors, significantly predisposes to antisocial behavior later in life. The interaction between these factors suggests that multiple genetic and environmental factors must be considered simultaneously to fully understand the formation of ASPD and adequately manage this dangerous condition.

These multi-direction findings highlighted the critical role of early intervention and precise preventive measures in reducing the likelihood of ASPD development. Identifying children at risk—characterized by elevated aggression, symptoms of conduct disorder, or experiences of emotional or physical trauma—at an early stage can prove the steps on the way for personalized interventions aimed at derailing the trajectory toward antisocial behaviors. Schaeffer et al. proposed that varying forms of aggression necessitated distinct intervention methods; children exhibiting progressive aggression may find programs emphasizing self-regulation and social competencies beneficial, and those with persistent high aggression levels, require more interventions, such as multi-function therapy or medication. By targeting the unique developmental pathways that lead to ASPD through timely interventions and holistic treatment plans, educators and researchers can significantly enhance the prospects for at-risk youth and reduce the occurrence of extreme antisocial behaviors in their adult lives.

To sum up, the comprehensive research corpus underscored the crucial role of childhood development in the origins of ASPD. The formative years, particularly those hurt by trauma and adverse childhood experiences, lay the foundational motive power for the path to ASPD. The hurt from emotional and physical abuse during these years, especially by trusted guardians, profoundly impacts the emergence of ASPD. Additionally, a pattern of enduring aggressive behavior in childhood serves as a significant omen of ASPD. Boys who display consistent or enhancing aggression are riskier for having conduct disorders, which may finally lead to ASPD. The research also highlights the significance of parental influence and environmental environment, with childhood care not enough and unstable family settings emerging as essential factors in the manifestation of ASPD symptoms. These comprehensive viewpoints highlighted the critical need for timely intervention and the development of tailored preventive strategies designed to meet at-risk children's distinct needs. By tackling the myriad pathways that lead to ASPD, we are devoted to enhancing the attention of at-risk youth, potentially avoiding the progression of antisocial behaviors into adulthood.

3. Conclusion

The present review emphasized the multifactorial, complexly interacting etiology of ASPD. Major neurobiological and environmental contingencies were identified about the disorder. Structural abnormalities within neuroanatomical structures that form the substratum of social cognition and emotional functions have been reported in neuroimaging studies. A decrease in GMV in the temporal lobes, prefrontal cortex, and parietal regions are usual findings in antisocial individuals' brains. Beyond this, functional disruptions between critical neural networks, especially between the attention-related regions of the default mode network, form the underpinning neural mechanisms of antisocial behavior. Traumatic brain injuries further complicate these vulnerabilities and heighten aggressive, and antisocial tendencies. Simultaneously, among the environmental factors in ASPD development, adverse childhood experiences of emotional and physical abuse constitute, together with early manifestations of aggression, the most important ones. These neurobiological and experiential factors underline ASPD etiology's complex nature.

The implications of the study are reflected in two areas. First, an integrative approach is needed to understand ASPD-neurobiological assessments, combined with comprehensive evaluations of early environmental experiences. It would be crucial in furthering preventive strategies and therapeutic interventions. For that, the identification of children at risk would be of prime importance because of early aggression or trauma for timely intervention. Second, the strong connection between TBI, PTSD,

and ASPD necessitates a multi-direction treatment method that deals with both the psychological and neurobiological dimensions of the disorder.

Future research should aim to improve intervention strategies by integrating neurobiological insights, especially those gained from brain imaging and studies on functional connectivity. Discovering biomarkers linked to ASPD could help in creating personalized treatment plans. Additionally, conducting long-term studies that track the progression from childhood trauma to ASPD while also observing changes in brain function over time would offer valuable insights for both prevention and treatment. Addressing these gaps in the literature will enhance one's understanding of ASPD and contribute to developing more effective, evidence-based therapeutic interventions.

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