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Review

A neuroscience perspective on antisocial personality disorder (ASPD)

Seyyedeh Sahar Asgari Ghalebin

Department of Psychology, Mohaghegh Ardabili University, Ardabili 56199-1313, Iran; s.asgari@uma.ac.ir

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Abstract: Introduction: Antisocial personality disorder (ASPD) is characterized by a persistent pattern of behavior that disregards and violates the rights of others. This disorder has biological-genetic and environmental roots, with the biological aspects playing a more significant role. Therefore, this article aims to examine the different dimensions of ASPD to adopt treatment and a suitable solution for it, considering its important effects on others and society. Method: Using a predefined search strategy, searches were conducted in databases including Google Scholar, PubMed, Criminal Justice Abstracts, and PsycINFO for published articles related to the research topic. Only studies published in English between 1990 and 2024 and containing information about the neuroscience of ASPD were eligible for inclusion. A total of 37 articles were selected for analysis and synthesis of the results. Results: Findings indicate that the limbic and frontal regions are particularly implicated in ASPD, with notable differences in the upper and lower frontal sulcus compared to typical brain variants. Additionally, individuals with ASPD exhibit larger spectral bands in delta and theta waves during a resting state than healthy subjects. Conclusion: This literature review provides valuable information about the neurobiology of ASPD and can enhance the depth and breadth of our knowledge for a more comprehensive and impactful understanding and treatment of ASPD. However, since there are apparent differences in brain function between ASPD subjects and controls, further research is needed to achieve consensus.

Keywords: antisocial personality disorder (ASPD); neuroscience; brain function; emotion dysregulation

1. Introduction

Personality disorders are mental health conditions characterized by enduring patterns of thought, emotion, and behavior that deviate from cultural expectations and cause significant impairment in various life areas, including social and emotional relationships, work, and education (American Psychiatric Association, 2023). These disorders are categorized into three clusters: A (odd and eccentric), B (dramatic, emotional, or erratic), and C (anxious and fearful) (World Health Organization, 2019). Cluster B includes Antisocial Personality Disorder, Narcissistic Personality Disorder, Borderline Personality Disorder, and Histrionic Personality Disorder. These disorders, particularly ASPD and Borderline Personality Disorder, are extensively studied due to their dramatic and unstable nature, which often results in significant social and medical challenges, including poor adherence to medical and lifestyle recommendations (risk-taking, disregard for others' safety, violence), which can lead to medical problems for the individual as well as members of his social environment (Douzenis et al., 2012). Because patients with ASPD do not seek treatment for their behavioral problems, the National Institutes of Health and Care Excellence specifically emphasizes that patients with ASPD should be the priority of care (Kendall et al. 2009). Psychoeducation is one way to care for ASPD, which is to educate patients about their condition and how

to better manage it. If done sensitively and correctly, it may help to increase the patient's awareness of their behavior problems and how they affect themselves and others (Thylstrup and Hesse, 2016). Currently, there is limited evidence on psychoeducation for ASPD (Zanarini and Frankenburg, 2008).

ASPD is defined by a long-term pattern of disregard for and violation of the rights of others, with behaviors often including aggression and criminal activity (World Health Organization, 2019). Biological-genetic and environmental factors contribute to the development of ASPD (Schermer et al., 2020). Recent research has concluded that antisocial personality disorder is a neuro-developmental disorder because, according to brain imaging, neurocognition, genetics and epigenetics, neurochemistry, and early effects that have on brain health and development influences indicating that its origins lie in early childhood. Unlike other personality disorders, ASPD's neurological basis is more substantial than environmental influences (Raine, 2018). Research consistently identifies abnormalities in the primary limbic system of individuals with ASPD (Koenigs et al., 2011). The improper development of the limbic system, particularly the amygdala, hippocampus, septal nucleus, and fornix, is implicated in antisocial behaviors (White et al., 2013). This improper development is evident in conditions like cavum septi pellucid (CSP), where the incomplete fusion of the limbic structures during fetal development is noted. CSP is a marker for fetal neural maldevelopment. The septum pellucidum is one component of the septum and consists of a deep, midline, limbic structure made up of two translucent leaves of glia separating the lateral ventricles, forming part of the septohippocampal system (Al-Saedi and Hassan, 2016). During fetal development, approximately in the 12th week of pregnancy, a space between the two layers—the CSP—is created, which begins to close in approximately the 20th week of pregnancy, and this closure occurs shortly after birth (3–6 months postnatally). It ends in ninety percent of cases. Fusion of the CSP is attributed to the rapid development of the hippocampus and other midline structures. Incomplete fusion results in fossa continuity, which, in turn, reflects possible neurodevelopmental anomalies of these midline structures (Raine et al. 2010). According to the report, CSP is weakly related to antisocial personality disorder (Al-Saedi and Hassan, 2016). There are individual differences in the degree of this neurodevelopmental abnormality; whereas some have complete cavity closure, others present with a small degree (>6 mm in the coronal plane) of incomplete closure. The cause of the maldevelopment of midline limbic structures that result in CSP is largely unknown, although it is thought that prenatal alcohol exposure plays an important teratogenic role (Kim et al. 2007). In this review article, we want to examine antisocial personality disorder from different dimensions by reviewing authentic articles so that we can adopt a suitable treatment and solution for it, considering the important effects it can have on others and society.

2. Method

To identify relevant studies, we searched databases including Google Scholar, PubMed, Criminal Justice Abstracts, and PsycINFO. Inclusion and exclusion criteria were defined before the database search to include only studies relevant to the research topic. This search was to extract articles published from 1990 to 2024 using terms such

as “antisocial personality disorder”, “QEEG”, and “MRI” and written in English on antisocial personality disorder. Studies that provide comparative analysis with related disorders, multimodal neuroimaging techniques, address comorbid conditions, focus on neurogenetic and neurodevelopmental aspects, functional connectivity, EEG and QEEG data, longitudinal and developmental studies, focus on emotion processing and empathy, machine learning and predictive modeling, neurofunctional abnormalities across domains, clinical implications and treatment strategies, and transdiagnostic factors have been discussed in the study. Additionally, studies that focused on disorders overlapping with ASPD were excluded. The researcher assessed eligibility for article inclusion in the literature review through screening of titles and abstracts. This search yielded 250 articles. After reviewing titles and abstracts, 37 English abstracts were found suitable for this review, and their full text was subsequently reviewed.

3. Results

Advancements in technology have enabled researchers to explore brain function using various imaging devices, including functional magnetic resonance imaging (fMRI). fMRI is a non-invasive and safe technique that uses magnetic fields and radio waves to investigate a wide range of brain disorders (Kim et al., 2021). fMRI operates by detecting changes in blood flow, as oxygenated hemoglobin is more magnetic than deoxygenated hemoglobin, allowing for the visualization of active brain regions (Hao et al., 2022). This method is employed to diagnose brain disorders and study brain function in response to stimuli or thoughts, including psychiatric conditions like personality disorders (Hinckel et al., 2015). According to a 2020 meta-analysis of 83 studies conducted with fMRI, using a liberal statistical threshold, several key regions were identified in the meta-analysis, mainly during acute threat response, social cognition, and cognitive control tasks (Dugré, 2020). Liu et al. (2014) used fMRI to uncover the neural mechanisms underlying ASPD. Their study showed decreased Amplitude of Low-Frequency Fluctuations (ALFF) in the right orbitofrontal cortex, left temporal pole, right inferior temporal gyrus, and left posterior lobe of the cerebellum. These brain regions are crucial for processing information, emotional states, decision-making, and language comprehension. The right orbitofrontal cortex, in particular, showed a negative correlation with ALFF values and psychopathic deviation scores on the Minnesota Multiphasic Personality Inventory (MMPI). These changes suggest abnormal activity in the frontotemporal network involved in movement control, planning, and complex decision-making in ASPD patients. Jiang et al. (2017a) conducted an fMRI study on 32 individuals with ASPD and 35 controls, examining brain function changes related to antisocial behavior. They found that ASPD patients exhibit longer processing paths for sensory inputs and reduced network efficiency, leading to decreased global brain function integration. Analysis showed reduced modularity and integration of network modules in the frontal areas, affecting the frontoparietal control network. These disruptions may contribute to impaired behavior and cognition in ASPD.

Electroencephalography (EEG) is a technique used to record the brain’s electrical activity through electrodes placed on the scalp, capturing the electrical signals

produced by the brain's nerve cells. Quantitative electroencephalography (QEEG) enhances this by numerically analyzing brain waves and utilizing statistical algorithms to detect changes in brain wave frequency, amplitude, and shape (Kesebir and Yosmaoğlu, 2018). QEEG offers real-time insights into brain activity and a better understanding of brain function under various conditions (Koberda et al., 2013). Using machine learning analysis, Baumgartl et al. (2020) conducted an EEG study comparing 42 patients with ASPD to 42 healthy controls. The results revealed that ASPD patients had larger spectral bands in delta and theta waves during the resting state than healthy subjects. Delta waves are typically associated with deep sleep and decreased consciousness, while theta waves increase during sleep and dreaming. The presence of these bands during wakefulness indicates brain dysfunction and may predict aggressive behaviors. Additionally, ASPD patients showed a decrease in alpha-band activity. Delta and theta indexes were the primary predictors differentiating ASPD patients from healthy individuals. A study of 84 violent criminals, including 50 with ASPD, QEEG, and low-resolution electromagnetic tomography (LORETA) analyses, was used to inspect the theta band of the EEG spectrum. The ASPD group exhibited excessive theta-delta and reduced alpha-band activity in the right frontotemporal and left temporal-parietal regions. These findings suggest that QEEG and source localization techniques can uncover differences in electrical brain activity among offenders with ASPD (Calzada-Reyes et al., 2012). EEG studies indicate that ASPD subjects show slow and paroxysmal abnormalities in the temporal and frontal regions. The frontal lobes, which regulate impulse control, and the temporal lobes, involved in sensory and emotional processing, exhibit dysfunction in ASPD subjects, potentially leading to impulsive and inappropriate behaviors (Ramos et al., 2018). As factors 1 (emotional and interpersonal) and factor 2 (antisocial and behavioral) traits increase, people with antisocial personality disorder have worsening psychological traits (Basoglu et al., 2011). Further research by Tillem et al. (2016) showed that in male inmates with ASPD, higher theta coherence was associated with worse psychological traits when exposed to unpleasant images. Psychopathy was linked to reduced gamma coherence in tasks involving emotional sharing (Decety et al., 2015a). P50 gating, a reduction in P50 amplitude due to repetition, acts as a preattentive filter, impacting cognitive processes and task performance. Studies indicate that ASPD is associated with impaired P50 gating and delayed responses, suggesting abnormal information filtering. Research by Lijffijt et al. (2009) found a continuum of weaker P50 gating from childhood-onset antisocial behavior to ASPD, mainly linked to conduct disorder symptoms. Individuals with ASPD generally show abnormal brain wave patterns, notably a reduced P300 amplitude. The P300 is an event-related potential (ERP) associated with attention and cognitive processing. Reduced P300 amplitude reflects deficits in these areas in individuals with ASPD. The results of a study by Calzada-Reyes et al. (2012) showed that ASPD was present in violent offenders. QEEG analysis showed a pattern of excessive theta-delta activity and a decreased alpha band on the right frontal-temporal and left temporoparietal regions in the ASPD group. Low-resolution electromagnetic tomography (LORETA) showed increased theta activity in the ASPD group compared to the non-ASPD group within the left temporal and parietal regions. Kesebir and Yosmaolu (2018) discuss how QEEG has diagnostic and prognostic value, can be a

biomarker and endophenotype in affective disorders, and can contribute to personalized treatment design.

Medical comorbidities in ASPD can significantly influence brain structure and function. In this regard, Douzenis et al. (2012) concluded that cluster B personality disorders are associated with Axis I psychiatric disorders such as addiction that have serious and life-threatening physical comorbidities. Lifestyle and health behaviors related to cluster B personality disorders lead to medical problems and enhance preexisting physical problems.

Non-invasive neuroimaging is helpful in monitoring and early diagnosing neural disorders such as ASPD to prevent their progress to a severe level. Hence, Kim et al. (2021) suggested incorporating a broader range of non-invasive brain imaging technologies, such as near-infrared optical bioimaging, alongside fMRI and QEEG to gain a more comprehensive understanding of ASPD. On the other hand, neurogenetic research is needed to complete neuroimaging studies. Studies by Dwyer et al. (2023) and Liang et al. (2024) emphasize investigating genetic markers alongside developmental pathways that can help develop a more robust model of ASPD as a neurodevelopmental disorder.

Understanding the connectivity patterns between various brain regions can elucidate how these disruptions translate into antisocial behavior. Results of a study by Jiang et al. (2017a) suggest that ASPD is associated with both reduced brain integration and segregation in the topological organization of functional brain networks, particularly in the frontoparietal control network. These disruptions may contribute to disturbances in behavior and cognition in patients with ASPD.

Conducting longitudinal studies to track the development of ASPD from childhood through adulthood could help identify critical periods for intervention and the role of early-life factors. In this regard, Atherton et al. (2020) examined the relationships between effortful control and ADHD, ODD, and CD symptoms over time using multimethod data from a longitudinal study. Results showed that parental ASPD symptoms were associated with increased CD but had no significant effect on effortful control, ADHD, or ODD.

To investigate how emotions are processed in ASPD individuals, Hyde et al. (2014) examined the differential relationship between dimensions of psychopathy and ASPD with negative emotions and amygdala reactivity to emotional faces and concluded that ASPD and psychopathy differ despite overlapping in several features, especially negative emotions and amygdala reactivity to social signals of threat. So, higher psychopathy scores were associated with lower negative emotions and lower amygdala reactivity, while higher ASPD scores were associated with more negative emotions and higher amygdala reactivity.

Decety et al. (2015b), Jiang et al. (2017b), Sundram et al. (2012) and Waller et al. (2017) reported that regions including the splenium of the corpus callosum, left posterior corona radiate/posterior thalamic radiate, right superior longitudinal fasciculus, cingulum, corticospinal tract, and inferior frontal-occipital fasciculus can be potential biomarkers, which would be of great interest in further understanding the pathomechanism of ASPD. As well, Liu et al. (2014) suggest that APD patients may be associated with abnormal activities in the frontotemporal network. Results of a study by Tully et al. (2023) showed that in antisocial individuals, there was

significantly increased variability for total gray matter and overall decreases in mean volume for total whole brain, total grey matter, and amygdala, compared with healthy controls.

Baumgartl et al. (2020) recommend a novel machine-learning approach for the detection of ASPD based on electroencephalography recordings. This approach can identify potential biomarkers for early diagnosis and intervention.

Some studies have proposed a broader assessment of neurofunctional abnormalities across various cognitive and affective domains, such as impulse control and reward processing, to provide a holistic view of ASPD. In this regard, Dugré et al. (2020), in a meta-analysis of whole-brain fMRI studies on antisocial individuals based on distinct neurocognitive domains, concluded that the most prominent functional brain deficits arise during acute threat response, social cognitions, and cognitive control neurocognitive domains. Results of a study by Stuppy-Sullivan and Baskin-Sommers (2019) aimed at identifying and specifying APD-related dysfunction in cognitive and reward factors showed that APD was associated with worse executive functioning during conscious high rewards as well as worse inhibition during high rewards when working memory demands were high. There was no APD-related performance difference during probabilistic decision-making.

There remains a lack of evidence-based pharmacological treatments for individuals with ASPD. Δ9-Tetrahydrocannabinol, the primary psychoactive constituent of cannabis, is an exogenous cannabinoid that stimulates the endocannabinoid system (ECS). Preliminary results of a study by Ho and Kolla (2022) suggest that alterations of the ECS may be present in ASPD. The use of drug treatments for patients with personality disorders remains controversial. Sadly, Stoffers-Winterling et al. (2021) and Khalifa et al. (2020) have come to the same conclusion as deBattista and Glick (1995). 29 years ago, pharmacotherapy could not be regarded as a cure for personality disorders. However, the result of a study by Mulder (1996) showed that anti-psychotics, mood stabilizers, and antidepressants may sometimes be helpful to patients with ASPD. Sesso and Masi (2023) reported that second-generation antipsychotics, lithium, anti-epileptic drugs, and stimulants are first-line medications, according to different target symptoms. Black (2017) reported that patients with ASPD should not routinely be treated with psychotropic medication. Medications may help treat comorbid psychiatric disorders such as major depression or aggressive symptoms common in these patients. Cognitive-behavior therapy may be helpful for those with milder syndromes.

Studies by Bijsterbosch et al. (2020) and Kebets et al. (2021) explore transdiagnostic factors that overlap between ASPD and other psychiatric disorders; their results support emotion dysregulation as a transdiagnostic dimension with neurobiological underpinnings that transcend diagnostic boundaries and add evidence to neural variability being a relevant proxy of neural efficiency.

4. Discussion

Research indicates that antisocial personality disorder (ASPD) has a clinical trajectory potentially influenced by environmental factors, such as adverse childhood experiences (ACE) (Wong, 2023). The disorder often manifests initially as abnormal

and aggressive behavior. Over time, individuals may display defiance toward rules and authority figures, progressing to symptoms of Oppositional Defiant Disorder (ODD). During adolescence, these behaviors may escalate into Conduct Disorder (CD) and, if left unaddressed, can evolve into ASPD in adulthood, characterized by intensified and transformed symptoms (Atherton et al., 2020). Many individuals with ASPD also exhibit symptoms of Attention Deficit Hyperactivity Disorder (ADHD) during childhood (Noordermeer et al., 2016). It is important to note that this developmental course is not uniform; some individuals may not exhibit apparent symptoms during adolescence or childhood (Burke et al., 2010). Because ASPD has its roots in childhood and develops in a relatively constant growth period during childhood and adolescence until adulthood, it shows a neurodevelopmental disorder (Raine, 2018). Analysis of the selected studies revealed that the limbic system and frontal brain regions are significantly involved in ASPD. Variations in the upper and lower frontal sulcuses were observed when compared to neurotypical individuals. Furthermore, individuals with ASPD showed a larger spectral band in delta and theta waves in a resting state, suggesting altered brain function. These findings underscore the need for further research to better understand the neurological underpinnings of ASPD and to develop more effective treatment protocols. In conclusion, while there are evident differences in brain function between individuals with ASPD and healthy controls, the lack of consensus among studies indicates that further research is necessary to fully elucidate these differences.

5. Conclusion

Antisocial Personality Disorder (ASPD) is a psychological disorder commonly associated with legal issues. Individuals with ASPD tend to deceive and exploit others without remorse and may display aggressive behaviors. This disorder is characterized by notable changes in brain structure and function, resulting in low cognitive flexibility and a weak understanding of emotions, causing indifference to the pain and suffering of others. Due to these structural and functional brain differences observable since childhood, many researchers have classified ASPD as a neurodevelopmental disorder.

The results of studies have revealed that various brain networks are involved in ASPD, each contributing to behavior formation. Key differences from normal variants include impairments in emotional understanding and specific brain regions, such as:

- The right orbitofrontal cortex is crucial for processing information, reflecting emotional states, and making decisions.
- The left temporal pole is essential for interpreting information, language, and understanding meanings.
- The right inferior temporal gyrus and the posterior lobe of the left cerebellum.

Moreover, individuals with ASPD exhibit larger spectral bands in delta and theta waves during the resting state than healthy subjects. Excessive theta-delta activity reduced alpha band activity in the right frontal-temporal and left temporal-parietal regions, and increased theta activity has been observed in ASPD subjects. These irregularities in brain function can explain the abnormal behaviors in this group and

suggest potential interventions to reduce the disorder's impact on individuals and society.

6. Limitations

- Access to all sources was challenging, leading to the use of some second-hand sources.
- Only English-language articles were reviewed due to language limitations.

7. Recommendations

For a more comprehensive understanding of ASPD, the following are recommended:

- Conduct organized neurogenetic research alongside neuroscience studies.
- Examine criteria and descriptive definitions from brain examinations, considering ASPD subjects as part of a spectrum similar to other neurodevelopmental disorders, to refine psychiatric diagnoses and improve treatment strategies.

Conflict of interest: The author declares no conflict of interest.

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