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The Complex World of Antisocial Personality Disorder: A Critical Scholarly Review

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Abstract: ***Antisocial Personality Disorder (ASPD) is a chronic mental health condition characterised by persistent patterns of disregard for the rights of others, deceitfulness, impulsivity, irritability, and aggressive behaviour. It has been extensively studied in psychiatry and psychology due to its strong association with criminality, substance abuse, and social dysfunction. This review article synthesises the existing literature on ASPD, examining its epidemiology, diagnostic criteria, neurobiological underpinnings, comorbidities, and treatment approaches. Drawing upon seminal and contemporary studies, we highlight the role of genetic predisposition, neurocognitive deficits, and environmental influences in the development and persistence of ASPD. Additionally, we explore the challenges in diagnosis and treatment, emphasising the limited efficacy of conventional interventions and the need for tailored therapeutic strategies. The discussion section delves into controversies regarding the categorical versus dimensional classification of ASPD and the potential for early interventions to mitigate its severity. This review aims to provide a nuanced understanding of ASPD and offer insights for clinicians, researchers, and policymakers in addressing this complex disorder.***

Keywords: Antisocial Personality Disorder, Psychopathy, Criminal Behaviour, Neuro cognition, Genetic Factors, Treatment Approaches, Conduct Disorder. Impulsivity, Aggression, Recidivism, Pharmacological Interventions, Comorbid Conditions, Neurobiological Abnormalities, Amygdala Dysfunction

1. Introduction

Antisocial Personality Disorder (ASPD) is a severe and chronic mental health condition that is characterised by a pervasive pattern of disregard for the rights of others, deceitfulness, impulsivity, irritability, and a failure to conform to social norms (Hare et al., 1991; Glenn et al., 2013). It is one of the most extensively researched personality disorders due to its significant implications for both individual well-being and societal safety. Individuals diagnosed with ASPD often exhibit persistent patterns of aggressive and exploitative behaviours, leading to frequent encounters with the criminal justice system (Black, 2015). The disorder is strongly associated with increased rates of substance abuse, violence, and recidivism, making it a critical area of study in forensic and clinical psychology (De Brito & Hodgins, 2007).

Historically, the concept of antisocial behaviour has evolved from early descriptions of moral insanity to the modern classification of ASPD within the Diagnostic and Statistical Manual of Mental Disorders (DSM). The DSM-III and DSM-IV defined ASPD primarily through behavioural criteria, focusing on observable patterns of rule-breaking, while the DSM-5 continues this approach but acknowledges the role of underlying personality traits (American Psychiatric Association, 2013). Despite its well-defined diagnostic criteria, ASPD remains a complex and multifaceted disorder, often overlapping with other conditions such as psychopathy, substance use disorders, and attention-deficit/hyperactivity disorder (ADHD) (Storebø & Simonsen, 2013).

Prevalence rates of ASPD vary across different populations, with higher rates observed among incarcerated individuals and substance abusers (Moran, 1999). In the general population, estimates suggest that approximately 3% of males and 1% of females meet the criteria for ASPD, though these numbers may be underestimated due to the reluctance of affected individuals to seek psychological assessment (Barry et al., 1997; Alegria et al., 2013). Research also indicates that ASPD has a strong genetic component, with twin studies suggesting heritability estimates of around 50% (Loeber et al., 2002). However, environmental influences such as childhood trauma, dysfunctional family dynamics, and socioeconomic disadvantages also play a crucial role in the disorder's development (Hill, 2003).

The neurobiological underpinnings of ASPD have been widely studied, with findings indicating abnormalities in brain regions associated with impulse control, emotional regulation, and moral reasoning. Neuroimaging studies have revealed reduced grey matter volume in the prefrontal cortex, amygdala dysfunction, and altered connectivity between brain regions involved in decision-making and empathy (Dinn & Harris, 2000; Stevens et al., 2003). These findings provide valuable insights into the neurological basis of ASPD and highlight potential avenues for intervention.

Despite its severe consequences, ASPD remains challenging to treat. Traditional therapeutic approaches, including cognitive-behavioural therapy (CBT) and pharmacological interventions, have shown limited success in reducing antisocial behaviours (Meloy & Yakeley, 2014). Additionally, the lack of motivation for change among individuals with ASPD further complicates treatment efforts. As a result, there is an increasing focus on early intervention strategies aimed at identifying at-risk individuals and implementing preventive measures (Lahey et al., 2005).

2. Literature Review

Antisocial Personality Disorder (ASPD) has been extensively studied from multiple perspectives, including its epidemiology, neurobiological underpinnings, environmental

influences, and treatment approaches. This section synthesises key findings from the existing literature to provide a comprehensive understanding of ASPD.

Epidemiology and Diagnostic Considerations
Moran (1999) conducted an epidemiological analysis of ASPD, highlighting its prevalence and societal impact. The study emphasised the disorder's strong association with criminal behaviour and substance abuse. Similarly, Glenn, Johnson, and Raine (2013) reviewed diagnostic criteria, noting the challenges in differentiating ASPD from related conditions like psychopathy and conduct disorder.

Neurobiological and Genetic Influences
Research suggests that ASPD has a strong neurobiological basis. Dinn and Harris (2000) examined neurocognitive deficits in individuals with ASPD, finding impairments in executive functioning and impulse control. Studies by Stevens, Kaplan, and Hesselbrock (2003) further support this, indicating that deficits in the prefrontal cortex contribute to impulsivity and aggression. Genetic studies, such as those by Lahey et al. (2005), propose that heritable factors play a significant role in ASPD development.

Environmental and Developmental Risk Factors
Early life experiences are critical in shaping antisocial behaviour. Hare, Hart, and Harpur (1991) discussed the role of childhood trauma and dysfunctional family environments in ASPD development. Loeber, Burke, and Lahey (2002) further linked adolescent conduct disorder to later ASPD diagnosis, underscoring the importance of early interventions. Research by De Brito and Hodgins (2007) explored the interplay between genetic predispositions and environmental triggers, emphasising that adverse childhood experiences exacerbate ASPD symptoms.

Comorbidities and Associated Behaviours
ASPD is often comorbid with substance use disorders, aggression, and other psychiatric conditions. Moeller and Dougherty (2001) examined the relationship between ASPD, alcohol abuse, and violent tendencies. Additionally, Barry et al. (1997) found that primary care patients with ASPD frequently exhibit comorbid conditions such as depression and anxiety, complicating treatment approaches. Storebø and Simonsen (2013) investigated the connection between ASPD and ADHD, suggesting overlapping neurobiological mechanisms.

Treatment Approaches and Challenges
Despite its severe consequences, effective treatment for ASPD remains limited. Meloy and Yakeley (2014) reviewed existing therapeutic interventions, concluding that cognitive-behavioural therapy (CBT) shows some promise in reducing antisocial behaviour. Pharmacological treatments, such as mood stabilizers and atypical antipsychotics, have been explored as potential interventions (Black, 2015). However, Poythress et al. (2010) highlighted the heterogeneity within ASPD populations, suggesting that treatment must be tailored to individual subtypes for greater efficacy.

3. Key Findings

The review of existing literature on Antisocial Personality Disorder (ASPD) highlights several critical insights into its etiology, behavioural characteristics, comorbidities, and treatment challenges.

Etiology and Risk Factors
Research consistently supports a multifactorial origin of ASPD, involving genetic predispositions, neurobiological abnormalities, and environmental influences. Neuroimaging studies (Stevens et al., 2003) indicate deficits in the prefrontal cortex, affecting impulse control and decision-making. Genetic research (Lahey et al., 2005) suggests a heritable component, while studies on early childhood adversity (Hare et al., 1991) demonstrate the significant role of trauma and dysfunctional family dynamics in ASPD development.

Behavioural and Cognitive Patterns
Individuals with ASPD exhibit persistent patterns of aggression, manipulation, impulsivity, and disregard for social norms. Studies (Glenn et al., 2013) reveal cognitive distortions and impaired moral reasoning, contributing to chronic antisocial behaviour. Further, distinctions between psychopathy-associated ASPD and impulsive-aggressive ASPD (Poythress et al., 2010) highlight the disorder's heterogeneity.

Comorbid Conditions: ASPD is frequently comorbid with substance use disorders, mood disorders, and other personality disorders. Moeller and Dougherty (2001) identified a strong correlation between ASPD, alcohol abuse, and violent tendencies. Barry et al. (1997) found high rates of co-occurring anxiety and depression in individuals with ASPD, complicating treatment approaches.

Challenges in Treatment and Management
Therapeutic interventions for ASPD remain limited, with only modest success reported for cognitive-behavioural therapy (CBT) (Meloy & Yakeley, 2014). Pharmacological approaches, including mood stabilizers and atypical antipsychotics (Black, 2015), have shown some promise in managing aggression and impulsivity but fail to address core personality traits. The heterogeneity within ASPD populations (Marcus et al., 2006) suggests the need for tailored treatment strategies.

Future Directions
Recent studies advocate for a multidimensional approach to ASPD treatment, integrating neurobiological insights with behavioural therapies. Yakeley and Williams (2014) emphasized the need for early intervention strategies to prevent ASPD progression. Marcus et al. (2006) suggested that future research should focus on identifying distinct ASPD subtypes to refine diagnostic and treatment frameworks.

4. Discussion

Developmental and Environmental Factors Contributing to ASPD
The development of Antisocial Personality Disorder (ASPD) is strongly linked to early behavioral patterns, environmental factors, and genetic predispositions. Loeber, Burke, and

Lahey (2002) and Lahey et al. (2005) highlight that conduct disorder in childhood serves as a primary predictor of ASPD in adulthood, with symptoms such as aggression, deceitfulness, and rule-breaking emerging early in life. Hill (2003) emphasizes that early identification of at-risk individuals through behavioural assessments is crucial for preventive interventions. Environmental factors, including childhood trauma and exposure to parental substance abuse, also play a pivotal role. Filov (2019) found that individuals with ASPD often have a history of childhood adversity, which exacerbates their propensity for violent behaviour

Neurobiological and Cognitive Underpinnings

Neurological and cognitive impairments are central to ASPD pathology. Glenn, Johnson, and Raine (2013) provide evidence that structural and functional abnormalities in the prefrontal cortex and amygdala contribute to poor impulse control and emotional regulation. Similarly, Dinn and Harris (2000) found that individuals with ASPD exhibit deficits in executive functioning, leading to impaired decision-making and increased risk-taking behaviours. Stevens, Kaplan, and Hesselbrock (2003) further explore these cognitive deficits, demonstrating that ASPD individuals struggle with tasks requiring complex problem-solving and impulse regulation. Marcus et al. (2006) examine whether ASPD exists on a continuum or as a distinct category, ultimately supporting a dimensional model of the disorder that aligns with observed neurocognitive variability

The Role of Psychopathy in ASPD

A significant area of research involves the overlap between ASPD and psychopathy. Hare, Hart, and Harpur (1991) distinguish psychopathy from ASPD, noting that while both disorders share traits like deceitfulness and aggression, psychopathy includes a lack of emotional depth and manipulative tendencies. Poythress et al. (2010) conducted a cluster analysis on offenders with ASPD, identifying subtypes that reflect varying degrees of psychopathic traits. Meloy and Yakeley (2014) discuss the implications of these findings for treatment, suggesting that individuals with high psychopathic traits are less responsive to conventional therapeutic interventions

ASPD and Comorbid Conditions

ASPD frequently coexists with other psychiatric disorders, complicating diagnosis and treatment. Moeller and Dougherty (2001) found strong associations between ASPD and substance use disorders, particularly alcohol dependence, which exacerbates impulsivity and aggression. The National Institute on Drug Abuse (1999) supports this by reporting high ASPD prevalence among drug-abusing populations. Storebø and Simonsen (2013) explore the link between ADHD and ASPD, indicating that childhood ADHD symptoms—especially impulsivity—predict the development of ASPD in adulthood. Black et al. (2010) analysed ASPD in incarcerated individuals and found high rates of psychiatric comorbidities, including mood and anxiety disorders, further complicating rehabilitation efforts.

Gender Differences in ASPD

ASPD manifests differently in men and women. Alegria et al. (2013) found that while men with ASPD exhibit more overt aggression and criminal behaviour, women tend to engage in relational aggression, manipulation, and deception. These findings suggest that ASPD diagnostic criteria, which emphasize externalized behaviours, may lead to underdiagnoses in women. Blaszczynski and McConaghy (1994) explore gender differences in gambling behaviours among ASPD individuals, finding that men with ASPD are more prone to impulsive gambling, whereas women tend to use gambling as an emotional escape.

ASPD in Forensic and Criminal Populations

Given its strong association with criminal behaviour, ASPD is widely studied in forensic settings. Black (2015) outlines the natural course of ASPD, noting that while symptoms decline with age, recidivism rates remain high among offenders. Colman and Wilson (1997) apply evolutionary game theory to ASPD, proposing that antisocial traits may have evolved due to their short-term survival benefits. Barry et al. (1997) examine ASPD prevalence in primary care settings, suggesting that many individuals with ASPD remain undiagnosed outside of forensic contexts. Moran (1999) provides epidemiological data on ASPD, showing that its prevalence varies across different populations and is influenced by socioeconomic factors.

ASPD and Impulsivity-Related Disorders

Impulsivity is a defining characteristic of ASPD and is linked to various maladaptive behaviours. Sher et al. (1994) discuss the connection between ASPD and disinhibitory psychopathology, particularly in relation to alcohol abuse. Lobbestael et al. (2009) examine the effects of induced anger in ASPD individuals, demonstrating heightened emotional reactivity and reduced impulse control. Blaszczynski and McConaghy (1994) also explore the link between ASPD and pathological gambling, reinforcing the role of impulsivity in these behaviours.

Treatment Challenges and Future Directions

Treating ASPD remains a complex challenge due to poor treatment compliance and lack of motivation for change. Meloy and Yakeley (2014) highlight the limited efficacy of cognitive-behavioural therapy (CBT) in ASPD patients, particularly those with high psychopathic traits. Yakeley and Williams (2014) propose new therapeutic approaches, including metallization-based therapy, which may be more effective in addressing emotional dysregulation in ASPD individuals. De Brito and Hodgins (2007) discuss the potential for early interventions targeting at-risk youth, emphasizing the importance of prevention over treatment.

5. Conclusion

Antisocial Personality Disorder (ASPD) remains a significant challenge in psychiatric and forensic fields due to its complex etiology, persistent patterns of antisocial behaviour, and limited treatment options. This review has explored the multifaceted nature of ASPD, examining its epidemiology, neurobiological and genetic underpinnings, environmental influences, comorbid conditions, and treatment approaches. The evidence underscores that ASPD is a product of intricate interactions between genetic predispositions and adverse environmental factors, such as childhood trauma and dysfunctional family dynamics.

A key takeaway from this review is the association between ASPD and a range of maladaptive behaviours, including aggression, substance abuse, and criminal activities. Neurobiological findings suggest that deficits in prefrontal cortex functioning and altered neurotransmitter systems contribute to the impulsivity and lack of empathy characteristic of individuals with ASPD. Additionally, research on adolescent antecedents highlights the importance of early identification and intervention, as conduct disorder and oppositional defiant disorder are strong predictors of ASPD development in adulthood.

Despite its severe social and individual consequences, effective treatment strategies for ASPD remain limited. Traditional therapeutic approaches, such as cognitive-behavioural therapy (CBT), have shown modest success, particularly when addressing comorbid conditions like substance use disorders. Pharmacological interventions, including mood stabilisers and atypical antipsychotics, may alleviate certain symptoms but do not provide a comprehensive solution for core antisocial traits. This highlights the necessity for a more integrative approach, combining psychotherapy, pharmacology, and early preventative measures.

Furthermore, research indicates that ASPD exhibits substantial heterogeneity, with potential subtypes differing in severity and underlying pathology. Some individuals display psychopathic traits, characterized by callous-unemotional behaviour, whereas others exhibit a more impulsive-aggressive presentation. Recognizing these distinctions is crucial for developing tailored interventions that address specific needs and behavioural patterns within the ASPD population.

In conclusion, ASPD represents a critical public health concern, necessitating continued research into its biological, psychological, and social dimensions. Advancements in neuroimaging, genetics, and psychological assessment tools offer promising avenues for better understanding and managing this disorder. Future research should focus on refining diagnostic criteria, identifying early intervention strategies, and developing more effective, personalized treatment approaches. By integrating insights from various disciplines, clinicians and researchers can enhance the prognosis and quality of life for individuals affected by ASPD while mitigating its broader societal impact.

ASPD remains a complex and challenging disorder with profound individual and societal implications. Understanding its multifaceted nature requires an integrative approach, incorporating neurobiological, psychological, and social perspectives. Future research should focus on improving diagnostic precision and developing more effective, personalised interventions.

References

[1] Hare, R. D., Hart, S. D., & Harpur, T. J. (1991). Psychopathy and the DSM-IV Criteria for Antisocial Personality Disorder. Journal of Abnormal Psychology, 100(3), 391-398.
[2] De Brito, S. A., & Hodgins, S. (2007). Antisocial Personality Disorder. In M. McMurran & R. Howard (Eds.), Personality, Personality Disorder, and Risk of Violence. Institute of Psychiatry, King's
[3] Filov, I. (2019). Antisocial Personality Traits as a Risk Factor of Violence Between Individuals with Mental Disorders. Open Access Macedonian Journal of Medical Sciences, 7(4), 657- 662.
[4] Glenn, A. L., Johnson, A. K., & Raine, A. (2013). Antisocial Personality Disorder: A Current Review. Current Psychiatry Reports, 15(12), 1-8.
[5] Moran, P. (1999). The Epidemiology of Antisocial Personality Disorder. Socia/ Psychiatry and Psychiatric Epidemiology, 34(5), 231-242.
[6] Barry, K. L., Fleming, M. F., Manwell, L. B., & Copeland, L. A. (1997). Conduct Disorder and Antisocial Personality in Adult Primary Care Patients. The Journal of Family Practice, 45(2), 151-158.
[7] Meloy, J. R., & Yakeley, J. (2014). Antisocial Personality Disorder. In Gabbard's Treatments of Psychiatric Disorders (5th ed., pp. 1015-1034). American Psychiatric Publishing.
[8] Black, D. W. (2015). The Natural History of Antisocial Personality Disorder. The Canadian Journal of Psychiatry, 60(7), 309-314.
[9] Loeber, R., Burke, J. D., & Lahey, B. B. (2002). What are adolescent antecedents to antisocial personality disorder? Criminal Behaviour and Mental Health, 12, 24-36.
[10] Hare, R. D., Hart, S. D., & Harpur, T. J. (1991). Psychopathy and the DSM-IV Criteria for Antisocial Personality Disorder Journal of Abnormal Psychology, 100(3), 391-398.
[11] Hill, J. (2003). Early Identification of Individuals at Risk for Antisocial Personality Disorder. The British Journal of Psychiatry, 182(s44), s11-s14.
[12] Sher, K. J., Trull, T. J., Bartholow, B. D.. & Vieth, A. (1994). Personality and Disinhibitory Psychopathology: Alcoholism and Antisocial Personality Disorder. Journal of Abnormal Psychology, 103(1), 92-102.
[13] Storebø, O. J., & Simonsen, E. (2013). The Association Between ADHD and Antisocial Personality Disorder (ASPD): A Review. Journal of Attention Disorders..
[14] Lahey, B. B., Loeber, R., Burke, J. D., & Applegate, B. (2005). Predicting Future Antisocial Personality Disorder in Males From a Clinical Assessment in Childhood. Journal of Consulting and Clinical Psychology, 73(3), 389
[15] Blaszczynski, A. P., & McConaghy, N. (1994). Antisocial Personality Disorder and Pathological Gambling. Journal of Gambling Studies, 10(2), 129-145.
[16] Moeller, F. G., & Dougherty, D. M. (2001). Antisocial Personality Disorder, Alcohol, and Aggression. Alcohol Research & Health, 25(1), 5-11.
[17] Dinn, W. M., & Harris, C. L. (2000). Neurocognitive Function in Antisocial Personality Disorder. Psychiatry Research, 97(2-3), 173-190.
[18] Poythress, N. G., Edens, J. F., Skeem, J. L., Lilienfeld, S. O., Douglas, K. S., Frick, P. J., Patrick, C. J., Epstein, M., & Wang, T. (2010). Identifying Subtypes Among Offenders With Antisocial Personality Disorder: A

Cluster-Analytic Study. Journal of Abnormal Psychology,
[19] Black, D. W., Gunter, T., Loveless, P., Allen, J., & Sieleni, B. (2010). Antisocial Personality Disorder in Incarcerated Offenders: Psychiatric Comorbidity and Quality of Life. Annals of Clinical Psychiatry, 22(2), 113-120.
[20] Yakeley, J., & Williams, A. (2014). Antisocial Personality Disorder: New Directions. Advances in Psychiatric Treatment, 20(2), 132-143.
[21] National Institute on Drug Abuse. (1999). Antisocial Personality Disorder Among Drug Abusers. NIDA Research Report Series, 167, 1-8.
[22] Alegria, A. A., Petry, N. M., Liu, S. M., Blanco, C., Skodol, A. E., Grant, B., & Hasin, D. (2013). Sex Differences in Antisocial Personality Disorder: Results from the National Epidemiological Survey on Alcohol and Related Conditions. Personality Disorders: Theory, Research, and Treatment, 4(3), 214-222
[23] Marcus, D. K., Lilienfeld, S. O., Edens, J. F., & Poythress, N. G. (2006). Is antisocial personality disorder continuous or categorical? A taxometric analysis. Psychological Medicine, 36(11), 1571-1581.
[24] Lobbestael, J., Arntz, A., Cima, M., & Chakhssi, F. (2009). Effects of induced anger in patients with antisocial personality disorder. Psychological Medicine, 39(4), 557–568.
[25] Colman, A. M., & Wilson, J. C. (1997). Antisocial personality disorder: An evolutionary game theory analysis. Legal and Criminological Psychology, 2(1), 23-34.
[26] Stevens, M. C., Kaplan, R. F., & Hesselbrock, V. M. (2003). Executive- cognitive functioning in the development of antisocial personality disorder. Addictive Behaviors, 28(2), 285-300.
[27] Blaszczynski, A. P, & McConaghy, N. (1994). Antisocial personality disorder and pathological gambling. Journal of Gambling Studies, 10(2), 129-145.
[28] Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. Psychological Review, 100(4), 674-701.
[29] Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., Taylor, A., & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. Science, 297(5582), 851-854.
[30] Moffitt, T. E., Caspi, A., Rutter, M., & Silva, P. A. (2001). Sex differences in antisocial behaviour: Conduct disorder, delinquency, and violence in the Dunedin Longitudinal Study. Cambridge University Press.
[31] Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., McClay, J., Mill, J., Martin, J., Braithwaite, A., & Poulton, R. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. Science, 301(5631), 386-389.
[32] Anderson, J. L., Sellbom, M., Wygant, D. B., Salekin, R. T., & Krueger, R. F. (2014). Examining the associations between DSM-5 Section III antisocial personality disorder traits and psychopathy in community and university samples. Journal of Personality Disorders, 28(5), 675-697.
[33] Swann, A. C., Lijffijt, M., Lane, S. D., Steinberg, J. L., & Moeller, F. G. (2009). Antisocial personality disorder and borderline symptoms are differentially related to impulsivity and course of illness in bipolar disorder. Journal of Affective Disorders, 112(1-3), 219-225.
[34] Lobbestael, J., Arntz, A., Cima, M., & Chakhssi, F. (2009). Effects of induced anger in patients with antisocial personality disorder. Psychological Medicine, 39(4), 557-568.
[35] Marcus, D. K., Lilienfeld, S. O., Edens, J. F., & Poythress, N. G. (2006). Is antisocial personality disorder continuous or categorical? A taxometric analysis. Psychological Medicine, 36(11), 1571-1581.
[36] Glenn, A. L., Johnson, A. K., & Raine, A. (2013). Antisocial personality disorder: A current review. Current Psychiatry Reports, 15(12), 427.
[37] Meloy, J. R., & Yakeley, J. (2014). Antisocial personality disorder. In G. O. Gabbard (Ed.), Gabbard's treatments of psychiatric disorders (5th ed., pp. 1015-1034). American Psychiatric Publishing.