Narrative Review: The Intersection of Genetic Predisposition for Autism Spectrum Disorder and Nurture Factors in the Development of Borderline Personality Disorder

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Narrative Review: The Intersection of Genetic Predisposition for Autism Spectrum Disorder and Nurture Factors in the Development of Borderline Personality Disorder

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Abstract

Purpose: This narrative review explores the significant overlaps between Borderline Personality Disorder (BPD) and Autism Spectrum Disorder (ASD), focusing on shared symptoms and genetic underpinnings. The purpose was to investigate neurodivergent traits in BPD similar to autism and examine how genetic predisposition for ASD can increase BPD risk when faced with adverse childhood experiences. Despite different DSM-5 classifications, both disorders share traits like emotional dysregulation, interpersonal difficulties, and cognitive distortions. Genetic markers, including variations in BDNF, COMT, and CNTNAP2 genes, suggest a shared biological vulnerability. The review posits that a family history of ASD may increase BPD likelihood during adolescence, especially when encountering adverse childhood experiences (ACEs). This interplay between genetic predisposition and environmental stressors highlights the need for improved BPD diagnostic criteria and tailored interventions.

Methodology: A comprehensive literature search was conducted using databases such as PubMed, Semantic Scholar, and Google Scholar. Key search terms included "Autism Spectrum Disorder," "Borderline Personality Disorder," "Genetic Overlap," and "Theory of Mind." The review focused on peer-reviewed studies from the last decade, with some foundational papers included for historical context.

Findings: The review uncovered notable overlaps between ASD and BPD in genetics, neurocognition, and symptomatology. Shared genetic factors include variations in the BDNF, COMT, SHANK3, and CNTNAP2 genes. Neurocognitive similarities were evident in impairments like social cognition, emotional regulation, and facial expression processing. Overlapping symptoms such as emotional dysregulation, Theory of Mind deficits, and executive dysfunction emphasize the neurodivergent nature of BPD. Importantly, the review suggests that a family history of ASD, combined with exposure to adverse childhood experiences (ACEs), may increase the risk of developing BPD during adolescence.

Unique Contribution to Theory, Practice and Policy: This review reimagines BPD within a neurodivergent framework, similar to ASD, focusing on its neurobiological foundations. It supports an integrated theoretical model highlighting the neurodivergent traits of BPD which are similar to those of ASD. In clinical practice, the review emphasizes the importance of recognizing overlaps during assessment to improve diagnostic accuracy and inform tailored interventions. It advocates for incorporating neurodiversityaffirming practices into BPD treatment, inspired by approaches used in ASD care. The findings encourage deeper study of the shared genetic and neurodevelopmental aspects of BPD, potentially leading to improved treatment and diagnostic models.

Keywords: Borderline Personality Disorder, Autism Spectrum Disorder, Genetic Predisposition, Neurodivergence, ACEs

JEL Codes of Classification: 110, 112, 114

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INTRODUCTION

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Borderline Personality Disorder (BPD) and Autism Spectrum Disorder (ASD) are both psychological conditions belonging to different categories in the DSM-V. Cognitive complications, social impairments, problems in social cognitive functioning, and emotional dysregulation highly characterize both of the conditions. Recent studies have brought forward rather complicated yet intricate connections between the two disorders, especially when considering the genetic similarities. Individuals with a family history of ASD may be more susceptible to BPD because of genetic similarities (Lichtenstein et al., 2010). This genetic overlap can present itself in the various overlapping symptoms. Furthermore, the inherent and textbook neurodivergent traits further complicate this relationship. Research has demonstrated that behavior and traits of high-functioning ASD often mimic the symptoms of BPD (Darling Rasmussen, 2023). Through extensive research, it is observed that the family history of ASD often posits the genetic vulnerability for developing BPD when faced with Adverse Childhood Experiences (Lichtenstein et al., 2010). Moreover, children with ASD are more vulnerable to developing maladaptive coping mechanisms because of trauma as compared to the neurotypical population when faced with ACEs, this in turn significantly increases the possibility of developing BPD (May et al., 2021).

Furthermore, disturbances in the Theory of Mind and conscious awareness, particularly concerning cognitive distortions, play a significant role in understanding the connection between ASD and BPD. These disruptions highlight how genetic traits associated with ASD may contribute to the pathogenesis of BPD, particularly when viewed through the lens of the diathesis-stress model. This model suggests that individuals with a genetic predisposition for ASD may be more vulnerable to developing BPD when exposed to environmental stressors. It is important to note that individuals with BPD score higher on assessments for Autistic traits (Dell'Osso et al., 2023a). Understanding these links is crucial for advancing treatment strategies and fostering a more nuanced recognition of neurodivergent traits in personality disorders (Guilé et al., 2018). Some overlapping symptoms between BPD and ASD include social difficulties, challenges with emotional regulation, sensory sensitivities, impulsivity, and black-and-white thinking. These shared features underline the intricate relationship between the two disorders and emphasize the need for a comprehensive approach to diagnosis and intervention (Gondek, 2021). Lastly, both disorders display dysfunction of the reward pathway. In BPD the individuals often indulge in risky behavior or repetition of maladaptive coping mechanisms (Parr et al., 2022), while in ASD there is reduced sensitivity to social interactions which is linked to atypical dopamine functioning (Blum et al., 2024). Repetitive behavior, impulsive or atypical reward-seeking behavior, and complications in social interactions are some of the overlapping symptoms of BPD and ASD which are associated with a dysfunctional reward system (Drews-Windeck et al., 2023).

This narrative review explores the intricate genetic and environmental interconnections between these disorders, emphasizing the role of nurture factors in shaping outcomes. By examining this intersection, we aim to shed light on the shared and divergent pathways contributing to these conditions, addressing critical research and clinical gaps. This research will address the following research questions: International Journal of Psychology ISSN 2957-6881 (Online)





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- Does a genetic predisposition for Autism Spectrum Disorder (ASD) increase the risk of developing Borderline Personality Disorder (BPD) during adolescence, as explained by the diathesis-stress model?
- Does a genetic predisposition for ASD increase the risk of developing BPD during adolescence?
- How do disturbances in theory of mind and emotional dysregulation manifest in individuals with ASD and BPD, and what are the shared characteristics between the two disorders?
- What role does the reward system dysfunction play in the emotional and mood regulation challenges observed in individuals with ASD and BPD?
- What cognitive distortions are common to both ASD and BPD?

A meta-analysis revealed that genetic factors played a significant role in determining BPD traits in individuals. The review revealed that about 40% to 60% of variations are caused by genetic and environmental influences in the development of BPD (Ansari et al., 2023). The interplay between these factors suggests that individuals who have a family history of ASD are at increased vulnerability to developing BPD due to shared genetic pathways and environmental stressors. Furthermore, A meta-analysis discovered that the pooled prevalence of BPD in ASD was 4% [95% CI 0%-9%] and of ASD in BPD was 3% [95% CI 1%-8%] (May et al., 2021). This further eludes to the fact that the genetic similarity between ASD and BPD can become a significant risk for individuals with family history of ASD to be vulnerable to developing BPD. Likewise, both ASD and BPD are characterized by challenges in social interactions and emotional regulation, which further increases the risk of developing BPD-like symptoms in individuals with ASD (Allman et al., 2024). Some of the symptoms of ASD, make the children more vulnerable to the precursors of BPD (DeShong et al., 2019). Children with ASD already suffer from emotional dysregulation, a dysfunctional reward system, ToM deficits, and social cognitive deficits, these problems make them an easy target for abuse and ACEs.

Theoretical Framework

The theoretical framework for this research integrates genetic studies, neuropsychology, and cognitive theories, including cognitive distortions and theory of mind, to explore the complex relationship between Autism Spectrum Disorder (ASD) and Borderline Personality Disorder (BPD). Genetic studies provide insight into hereditary predispositions, highlighting the potential for shared genetic traits that may increase vulnerability to BPD in individuals with a family history of ASD. Neuropsychology examines the neurological underpinnings of emotional regulation and reward system dysfunction, both critical in understanding mood and behavioral challenges in these disorders. Additionally, it also observes the neurodivergent traits existing in BPD. Cognitive distortions and the Theory of Mind offer a lens to analyze how disruptions in self-awareness and interpersonal understanding contribute to the development of BPD during adolescence. Together, these domains provide a comprehensive framework to investigate the interplay between nature and nurture in this intricate relationship.

Cognitive Distortions

Cognitive distortions refer to biased perspectives that individuals have about themselves, the people around them, and their environment. Moreover, these distorted "perspectives" can often



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lead to negative thought patterns which can further exacerbate emotional dysregulation, a prominent symptom in both ASD and BPD (Sakdalan & Maxwell, 2023).

In the case of ASD, cognitive distortions often manifest as rigid thinking patterns, misinterpretations of social cues, and inability to change routines (Kulu & Ozsoy, 2020). This sort of "Black and White" rigidity, (their chosen routine is good and inability to adjust to a different routine) contributes to emotional dysregulation, especially when faced when changes or unexpected challenges (Kulu & Ozsoy, 2020). As for BPD, individuals also face "Black and White" thinking, catastrophizing, or personalizing situations. They often experience extreme emotions and react intensely when things do not go according to what they consider "comfortable or right" (Sakdalan & Maxwell, 2023). Additionally, the cognitive distortions in BPD can lead to emotional dysregulation, feelings of abandonment, and impulsive decisions.

Theory of Mind

Theory of Mind is known as the cognitive ability to understand that others have their own thoughts, beliefs, desires, and intentions. ToM deficits are common and prominent in both ASD and BPD.

In ASD, individuals experience significant challenges in ToM, especially in inferring others' emotions, and intentions. This can further cause social cognition problems and communication problems (Milosavljevic et al., 2016). Additionally, it impacts their interpersonal relationships which leads to feelings of isolation and frustration. On the other hand, individuals with BPD struggle with cognitive and affective components of ToM (Németh et al., 2018). Even if they can understand mental states of others, they often misinterpret them. Similarly, it leads to interpersonal relationships conflicts which further contributes to feelings of isolation, frustration, and even rejection (Németh et al., 2018).

Genetic Predisposition and Neurodivergence

Research suggests that common genetic markers could explain the similarity of symptoms in both disorders. For instance, variants in the **BDNF** (**Brain-Derived Neurotrophic Factor**) gene have been linked to both ASD (Salinas et al., 2020) and BPD (Khanzada et al., 2017). BDNF is responsible for mood regulation and cognitive functioning, contributing to traits observed in both disorders. In individuals with ASD, lower serum levels of BDNF are associated with emotional dysregulation and impairments in cognitive functioning, leading to challenges in social interactions and communication (Nangdev et al., 2024). Similarly in BPD, variants of the BDNF gene cause mood dysregulation and impulsivity, which is often considered a problem of cognitive functioning, and not understanding the risk of situations (Nangdev et al., 2024).

Another significant genetic marker is **CNTNAP2** (**Contactin Associated Protein-like 2**), which has also been implicated in both ASD and BPD. CNTNAP2 is essential for prosocial behavior, impulse control, and repetitive behaviors (Memis et al., 2022). In ASD, individuals face difficulties in prosocial behavior and understanding social cues this problem is linked to CNTNAP2 gene (Memis et al., 2022). In BPD, individuals often face problems with prosocial behavior and forming meaningful relationships, along with impulsive behavior which is often linked to disruptions in synaptic signaling due to CNTNAP2 mutations (Nangdev et al., 2024). This shared genetic basis highlights how variations in these genes may contribute to overlapping symptoms across both disorders.



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Another well-researched genetic marker common to ASD and BPD is **COMT** (**Catechol-O-Methyltransferase**). COMT is responsible for the metabolism of catecholamines, including dopamine, norepinephrine, and epinephrine. This breakdown of catecholamines helps regulate dopamine levels in the brain (Esmaiel et al., 2020). As indicated by research, mutations in the COMT gene can lead to the development of several ASD traits, especially the ones associated with the dysfunction of the reward pathway. This is often exhibited in the form of repetitive behavior and problems with communication (Gadow et al., 2009). Furthermore, mutations in the COMT gene can lead to difficulties in forming productive and healthy social interactions, as well as increased emotional reactivity and impulsive behavior (Esmaiel et al., 2020). Additionally, the mutations can cause emotional reactivity and increase impulsive behavior as well (Mitrović et al., 2024). In the case of BPD, mutations and variations in COMT gene causes similar problems, heightened emotional reactivity, impulsivity, aggressive behaviour, and problems with maintaining interpersonal social relationships (Qayyum et al., 2015). These findings underscore a notable genetic similarity between ASD and BPD, highlighting the involvement of COMT in the characteristic symptoms of both disorders.

Furthermore, another genetic marker, although not studied extensively, common to ASD and BPD is **SHANK3**. SHANK3 encodes a protein that forms and maintains synapses to ensure smooth communication between neurons (Erickson, 2016). In individuals with ASD, mutations in SHANK3 are linked to several core symptoms, including social communication deficits, repetitive behaviors, and challenges in sensory processing (Brown et al., 2018). Research indicates that these mutations disrupt synaptic function, which may contribute to the characteristic difficulties in social interactions and behavioral regulation observed in ASD (Brown et al., 2018). Similarly, alterations in the SHANK3 gene have been identified in individuals with BPD. These genetic variations are associated with impulsivity, emotional dysregulation, and communication difficulties. The impact of SHANK3 mutations on synaptic signaling may exacerbate the emotional instability and interpersonal challenges commonly experienced by those with BPD (Wan et al., 2022).

Diathesis-Stress Model and ACEs

The diathesis-stress model proposed for BPD posits that having a family history of ASD can put the individual at risk of developing BPD in adolescence if the individual faces Adverse Childhood Experiences (ACEs). The genetic similarities between the two disorders lay a strong biological foundation for the model (Dudas et al., 2017). Moreover, individuals who already exhibit some traits of ASD, because of family history and genetics, are often more vulnerable to stressors which further increases their chance of developing BPD (Carpita et al., 2023).

The focus is more on BPD as compared to other disorders such as Major Depressive Disorder, Generalized Anxiety Disorder, or PTSD, because BPD shares a lot of neurodivergent traits with ASD and is a result of significant childhood trauma. In contrast, while MDD, GAD, and PTSD are major mental health concerns, they do not exhibit the same degree of overlap with neurodivergent traits as seen in BPD (Rinaldi et al., 2021). This distinction highlights the unique relationship between childhood trauma and the development of BPD, particularly in individuals with a predisposition for ASD. Another critical aspect of the relationship between ASD and BPD is the impairment in Theory of Mind (ToM), which refers to the ability to understand and attribute mental states; such as beliefs, intentions, and emotions, to oneself and others (Rinaldi et al., 2021). Deficits in ToM are a hallmark symptom of ASD and have been



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observed in individuals with BPD as well (Rinaldi et al., 2021). These impairments can lead to significant challenges in social interactions and emotional regulation, further complicating the clinical picture for those affected by both conditions.

Furthermore, the diathesis-stress model highlights how genetic vulnerabilities and environmental stressors influence outcomes. For individuals with a family history of ASD, specific genetic variations, such as BDNF, SHANK3, CNTNAP2, and COMT, may create a biological foundation that heightens sensitivity to stress, and the negative impacts of abuse and ACEs (Woo et al., 2023). These genetic factors coupled with the environmental challenges, increase the likelihood of emotional cognitive, and behavioral problems often associated with BPD. Additionally, when considering the neurobiological or genetic factors, it is important to note how the genetic predispositions linked with ASD can lead to changes in brain areas like the amygdala and the prefrontal cortex, which are essential for regulating emotions and navigating social interactions (Dimitrovski et al., 2024). Individuals with these vulnerabilities often show heightened sensitivity to stress (Woo et al., 2023). When faced with ACEs, like childhood neglect and physical, emotional, and sexual abuse, their heightened stress can amplify physiological and psychological reactions, increasing the risk of developing BPD.

Furthermore, these stressful experiences can induce epigenetic changes, altering certain genes' functions and further impacting emotional regulation, cognition, and social behavior (Woo et al., 2023). Likewise, early life stressors occurring during critical periods of brain development can interfere with neuroplasticity, impacting how the brain matures and adapts. For individuals with ASD genetic traits, this disruption can hinder emotional regulation, mentalization, and interpersonal relations, all of which are central to both ASD and BPD (Harry et al., 2024).

Disturbance in Theory of Mind

The theory of mind is the ability of individuals to attribute mental states. This includes beliefs, intentions, desires, and even emotions, not just to oneself but to others as well. This is a crucial cognitive skill that allows individuals to understand social interactions and to interact in a socially desirable way (Rakoczy, 2022). Significant impairments in ToM are noticed in both ASD and BPD. Individuals suffering from ASD have severe deficits in ToM due to social cognitive deficits. Studies have shown that children or individuals who are diagnosed with ASD have lower performance on the tasks of ToM when compared to neurotypical peers (Cheney et al., 2023). Moreover, impairments in both cognitive and affective aspects of ToM are exhibited in ASD (Bar et al., 2021). On the other hand, individuals with BPD show significant impairment in ToM, particularly in understanding the perspectives of other individuals when they are in emotionally charged situations (Carminati et al., 2024). Moreover, individuals with BPD experience significant problems in reflective thinking, which is crucial for understanding social situations and analyzing the consequences of an action (Aldao et al., 2010). They also experience difficulty in understanding their options and the emotions of those around them (Németh et al., 2018). This often leads to heightened emotional sensitivity and emotional overreactions (Németh et al., 2018). Likewise, several studies have observed similarities between ASD and BPD based on ToM related testing. For instance, a study related to "Reading the Mind in the Eyes" test showed that individuals with ASD and BPD recognized fewer Faux Pas motifs compared to healthy individuals (Bar et al., 2021). Furthermore, impairments in ToM also correlate with decreased empathy in ASD and BPD (Bar et al., 2021).



Shared Characteristics

In both disorders, ASD and BPD there are overlaps in symptoms, which include difficulties in social recognition, increased emotional reactivity, cognitive rigidity, decrease in cognitive empathy, and an increase in impulsive behavior.

Social Recognition and Interactions

Social difficulties are common to both ASD and BPD, although the manifestation is often different. Individuals with ASD often find it difficult to form meaningful connections because of their inability to connect with neurotypicals in a way that seems suitable to the general neurotypical population (Sasson et al., 2017). They often have difficulty expressing their emotions and can get overwhelmed easily (Sasson et al., 2017). As for individuals with BPD, they often get overwhelmed easily and face difficulty reading emotions.

In the case of ASD, it is common to have difficulties with reading emotions and often not understanding sarcasm or the tone of voice which leads to communication problems and frequent conflicts as well (Sasson et al., 2017). Individuals with BPD, find it difficult to read emotions especially when they are overwhelmed and often mistake that someone means to hurt them and or cause them damage (Lazarus et al., 2014). Likewise, individuals with ASD frequently have the same misconception, which stems from their inability to understand social cues and read social situations (Allman et al., 2024). While the similarities are present it is important to acknowledge that the mechanisms behind the symptoms differ significantly. The communication challenges present in ASD stem from cognitive functioning impairments which makes it difficult for individuals with ASD to communicate with neurotypicals (Allman et al., 2024). While in the case of BPD, the social difficulties or communication problems stem from complications resulting from childhood trauma and a fear of abandonment (Allman et al., 2024). Moreover, if the cause behind the social recognition and interaction problems is not studied properly it can lead to misdiagnosis of BPD for ASD, especially for women with ASD (Pires et al., 2023). That being said, it is important to consider the genetic similarities between ASD and BPD especially when it comes to deficits in social cognitive and communication problems. For instance, mutations in COMT gene is common to both of the disorders and are linked to problems with maintaining social connections and emotional regulation, both of these complications impact social interactions (Qayyum et al., 2015). Furthermore, emotional dysregulation can make it difficult to maintain social relationships which further complicates social interactions (Allman et al., 2024). When it comes to neural processing, both of the disorders exhibit atypical neural processing (May et al., 2021). Additionally, individuals with BPD exhibit severe alteration in regions of the brain responsible for emotional processing, similar issues have been observed in the case of ASD too (Mier et al., 2013).

Cognitive Rigidity

Cognitive Rigidity is another common symptom between the two disorders. It is expressed in different forms in ASD and BPD but often the root cause is the "Black and White Thinking" cognitive distortion, dysfunction of the reward system, or inflexible thinking patterns. In the case of ASD, individuals often find it difficult to shift from one routine to another, it even leads to distress and tantrums in children. This can be associated with Black and White Thinking, that a regular and familiar routine is considered good but a different routine is bad. In the case of ASD, individuals are focused or fixated on certain behaviors or certain routines that they do



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not want to be changed (Petrolini et al., 2023). Moreover, they often engage in repetitive behaviors associated with a dysfunctional reward system. Additionally, smaller grey area volume in the posterior parietal lobule is linked to cognitive rigidity (Watanabe et al., 2019). This further suggests cognitive perception impairment which causes this problem. Moreover, social cognition deficits contribute to cognitive rigidity in ASD (Allman et al., 2024). Similarly, in the case of BPD, individuals face difficulties updating their beliefs and schemas leading to continuously or repetitively engaging in certain behaviors (Herzog et al., 2022). This behavior is often negative or self-sabotaging as well. Furthermore, individuals with BPD even face difficulty changing their beliefs about themselves (Herzog et al., 2022). However, it is important to note that this usually stems from severe childhood trauma as the trauma impacts their cognitive perception (Herzog et al., 2022). Additionally, individuals with BPD experience "Black and White Thinking" as well when it comes to their beliefs; they might stick to a belief for a very long time as it seems the right option to them and any other belief apart from that seems wrong to them (Kube & Rozenkrantz, 2021). This pattern of thinking further leads to cognitive rigidity and difficulty in changing behavior and schemas (Kube & Rozenkrantz, 2021). In summary, cognitive rigidity represents a significant area of overlap between ASD and BPD, highlighting the complex interplay between neurodevelopmental factors and environmental influences in shaping cognitive patterns. Understanding these similarities is vital for designing targeted interventions and improving outcomes for individuals with either or both conditions. Notably, cognitive rigidity, often seen as a defining characteristic of neurodivergence, is also markedly present in BPD, underscoring its broader relevance beyond strictly neurodevelopmental contexts.

Furthermore, Black and White thinking contributes to distress by impairing the flexible problem solving ability of the individual and escalating the interpersonal conflicts. A study showed that black-and-white thinking was correlated to increased social challenges (Suzuki & Hirai, 2023). Additionally, another study showed that in BPD patients there was intensified emotional dysregulation and interpersonal difficulties due to dichotomous thinking (McClure et al., 2016). The study highlighted that there was a need of cognitive flexibility that can improve problem-solving skills and reduce the interpersonal conflicts in both disorders.

Cognitive Empathy

Impairments in cognitive empathy are common to both ASD and BPD. This problem makes it difficult for them to assess another individual's cognitive or mental state, which further leads to difficulty in social and interpersonal relationships. In case of ASD, this problem is often associated with deficits and complications in ToM (Schnitzler & Fuchs, 2024). Furthermore, these deficits also lead to challenges in social understanding and perspective-taking (Kimmig et al., 2024). Additionally, it often makes communication difficult for individuals with ASD as they cannot interpret social cues easily (Kimmig et al., 2024). Similarly, in the case of BPD individuals also face complications in cognitive empathy. They find it difficult to assess the emotional state of those around them (Preston et al., 2020). This problem often leads to hurdles in maintaining interpersonal relationships (Pedrosa et al., 2015). Research suggests that this difficulty in understanding others' mental states often results in volatile social interactions, as individuals with BPD may misperceive others' intentions or react disproportionately to perceived slights (Salgado et al., 2020). In short, both disorders experience deficits in cognitive empathy, further complicating social relationships and communication.



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The link between cognitive empathy deficits and emotional dysregulation often creates a cycle. When individuals struggle to understand others' mental states, they may experience increased anxiety and frustration, which further compromises their emotional regulation abilities (Hatata et al., 2024). Additionally, this emotional dysregulation then impairs their capacity for cognitive empathy, considering heightened emotional states may cloud judgment and perspective-taking abilities (Hatata et al., 2024).

In the case of BPD, this pattern is undeniable, considering the combination of cognitive empathy deficits and emotional dysregulation often leads to hypersensitivity to perceived rejection or abandonment (Pyszkowska et al., 2023). This further leads to intense emotional reactions, emotional meltdowns, and impulsivity, which can strain relationships. Similarly, in ASD, the interplay between cognitive empathy deficits and emotional dysregulation results in meltdowns or shutdowns in response to social overstimulation or confusion (Hervás, 2024).

Executive Functioning

Impaired executive functioning is another symptom common to both ASD and BPD. In ASD, executive dysfunction often occurs in planning and organizing daily activities and tasks (Eleni A. Demetriou et al., 2018). Moreover, they typically experience trouble with changing routines, often preferring consistency in various aspects of life (Margari et al., 2016). Additionally, they frequently experience deficits in sustained attention and working memory (Eleni A. Demetriou et al., 2019). Furthermore, response inhibition challenges are observed among individuals with ASD too (Eleni A. Demetriou et al., 2019). However, all of these symptoms are not just unique to ASD, they are also prevalent in individuals with BPD. Research indicates that executive functioning deficits in BPD are divergent in nature and usually they are observed in the patient's parents too (Hila Z. Gvirts et al., 2012). Likewise, individuals with BPD face impairments in working memory especially under high cognitive load (Hagenhoff et al., 2013). As observed in ASD, organization and planning abilities are also negatively impacted in BPD (H.Z. Gvirts et al., 2015). Notably, adolescents with BPD who exhibit eternalizing behaviors tend to have inhibitory response deficits too (Kalpakci et al., 2018). In conclusion, executive functioning deficits are present in both of the disorders and several studies suggest that the nature of executive dysfunction in BPD is neurodivergent. While the specific manifestations may differ, the underlying cognitive challenges such as planning, working memory, attention, and response inhibition are remarkably similar.

Reward System Dysfunction

Both ASD and BPD experience significant complications in the reward pathway and it is all further complicated by the involvement of the COMT gene. Individuals with ASD tend to have lower activity in the brain's reward system, specifically in an area known as nucleus accumbens, when they expect or receive awards (Kohls et al., 2013). This is specially obvious when it comes to monetary rewards and it suggests that the reward system and the concept attached to monetary value may work differently in individuals with ASD (Baumeister et al., 2023). Furthermore, for individuals with ASD the neurotransmitter pathways, especially the ones involving dopamine, do not always function properly (Walker, 2008). This imbalance can disrupt the brain's reward system impacting social and cognitive motivation (Blum et al., 2024). Additionally, this impacts social behavior, emotional regulation, and interpersonal relationships (Blum et al., 2024). For individuals with BPD, there are impairments in the reward system too. They often engage in risky behavior and keep on indulging in repetitive



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maladaptive behavior because of this implication (Vega et al., 2013). Likewise, individuals with BPD often experience challenges with how their brain processes rewards and it often impacts their behavior. This leads to risk taking and impulsive behavior because they cannot differentiate between loss and success or wins (Stewart et al., 2019). It also leads to trouble handling financial decisions (Andreou et al., 2015).

Involvement of the COMT gene has been linked to reward system dysfunction in both disorders. The COMT Val15Met gene variants regulate dopamine levels in the brain, particularly in the prefrontal cortex, which impacts thinking and decision making abilities (Mitrović et al., 2024). Hence, thinking patterns are impaired in both ASD and BPD, with repetitive behavior and inability to change routines in ASD and repetitively indulging in maladaptive behaviors in BPD. Individuals with the Met allele of this gene often show greater challenges with social interactions, which is relevant for both ASD and BPD (Millenet et al., 2018). Variations in the COMT gene can affect how dopamine is regulated, which may influence behavior and traits in people with ASD (Esmaiel et al., 2020). These changes could play a role in how rewards are processed and how social interactions are experienced. In BPD, changes in the COMT gene like altered DNA methylation can disrupt dopamine metabolism, which could also impact reward processing and related behaviors (Thomas et al., 2019). The shared traits and genetic factors between ASD and BPD show just how closely these too conditions are connected. Challenges in the brain's reward system, influenced by the COMT gene, may help explain some of their overlapping symptoms, such as repetitive behaviors, unusual patterns of seeking rewards, and difficulties with social interactions.

Furthermore, the dysregulation of the reward system in BPD is associated with impulsive and risky behavior and emotional instability due to problems related to catecholamines. Additionally, they often experience heightened sensitivity to both positive and negative stimuli, leading to rapid mood swings and impulsive actions (Shadara et al., 2021). The reward system, mediated primarily by dopamine, is indicated in BPD. Moreover, abnormalities in the dopaminergic pathways, especially in the frontal-striatal circuits, lead to impulsivity and emotional dysregulation (Franczak et al., 2024). As for ASD, the reward system dysfunction leads to an obsession with certain stimuli, interacting with those stimuli end up becoming the source of "reward" (Saha et al., 2023). Moreover, dysfunction in the GABAergic system has been implicated in ASD and also interacts with the reward system. This combination further contributes to an imbalance in excitatory/inhibitory neurotransmission, which affects social behavior and reward processing (Zhao et al., 2022).

Overlapping Cognitive Distortions

ASD and BPD share several cognitive distortions, particularly in how individuals perceive and respond to the world around them (Allman et al., 2024). One common distortion is "Black and White Thinking". In ASD, this often appears as a rigid adherence to routines, where one specific way of doing things is seen as entirely correct while alternatives are perceived as completely wrong (Amaral et al., 2012). Similarly, individuals with BPD often exhibit extreme thinking patterns, rapidly shifting between idealizing and devaluing others or viewing situations as entirely correct or entirely wrong (Genziana Lay, 2019). Another common cognitive distortion is "Cognitive Rigidity" or the inability to adapt one's thinking to new information or changing circumstances. In ASD, this often manifests as concrete and literal thinking, difficulties in adapting to new demands, and struggles with considering new



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perspectives (Lage et al., 2024). For those with BPD, cognitive rigidity can present as impaired decision making in emotionally charged situations (Bozzatello et al., 2023). The problems related to changing patterns and behaviors, and considering new perspectives are also present in BPD, as they find it difficult to change their behaviors and their update their beliefs (Bozzatello et al., 2023).

Social cognition challenges further highlight the overlap in cognitive distortions between ASD and BPD (Carminati et al., 2024). Individuals with ASD often struggle with interpreting nonverbal cues, understanding abstract social concepts, and recognizing or responding appropriately to others' emotions and intentions (Levy et al., 2022). Similarly, those with BPD may misinterpret other's intentions often assuming negative motives, and experience difficulty in maintaining stable perceptions of others across different situations (Preißler et al., 2010). In short, these cognitive distortions make it difficult to maintain social relationships, have a stable sense of self, and even disturb executive functioning in both of the disorders.

Discussion

The link between ASD and BPD can be established with the help of several research studies, especially when it comes to genetics and overlapping neurocognitive and behavioral symptoms. The traits between the two disorders overlap significantly. Especially regarding emotional regulation the individuals suffering from these two specific disorders struggle with not only emotional impulsivity, and intense emotional responses but also face difficulties while interacting with other people (Dell'Osso et al., 2023). Similarly in neurocognitive profiles between ASD and BPD, it can be observed that early trauma plays a mediating role (Carminati et al., 2024). Both of the disorders shared features such as rigidity, black-and-white thinking, and severe emotion dysregulation (Carminati et al., 2024). This overlap causes challenges not only in the diagnosis of the individuals but also increases the risk of individuals with an autism spectrum disorder to be at greater risk to develop BPD particularly when exposed to environmental stresses (Allman et al., 2024). There is a significant need for detailed research related to BPD, especially when it comes to pathophysiology and treatment models, but understanding that a certain population is at risk, individuals with family history of ASD, can significantly help with treatment and prevention models. Furthermore, it is important to consider the element of neurodivergence in BPD. Cognitive rigidity, severe executive dysfunction, social cognition deficits, reward system dysfunction, and Theory of Mind deficits all align with neurodivergent profiles.

The connection between genetic vulnerabilities for ASD and environmental stressors in the development of BPD is a multi-layered and evolving topic. Genetic factors, particularly those that influence dopamine regulation, seems to play an important role in both conditions. Disruptions in dopamine signaling and variation in the COMT gene have been linked to ASD and BPD, suggesting a shred biological basis. Moreover, genes such as BDNF, CNTNAP2, and SHANK3 have been associated with both disorders. These genes impact functions like social cognition, communication, mood regulation, and even the connection between the neurons. Furthermore, the executive dysfunction present in both of the disorders is similar to a great degree. These shared characteristics hint at a deeper connection between the two connections, rooted in genetics, and environment.



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CONCLUSION AND RECOMMENDATIONS

Conclusion

The relationship between ASD and BPD is complex, especially when considering the impact of adverse childhood experiences ACEs. Several studies indicate that genetic vulnerabilities to ASD may increase the likelihood of being diagnosed with BPD especially in the crucial phase of adolescence, particularly in the individuals who have experienced ACEs. Both disorders, BPD and ASD, share significant genetic components. Studies show that the heritability of BPD is quite high, which means there is a 46% chance for the child to have BPD if the parent has been diagnosed with it (Skoglund et al., 2021). Similar is the case for ASD, research has indicated that autism has a genetic basis, with numerous studies identifying specific genetic markers of the disorder (Genovese & Butler, 2023). There is an obvious overlap in genetic prepositions and vulnerability among individuals with ASD and BPD (Khanzada et al., 2017). A research study has identified 23 genes common to both ASD and BPD (Khanzada et al., 2017). This is highly specific if the individuals have suffered from environmental stressors such as ACEs.

Research has also discussed how ACEs contribute to neurocognitive impairment that mediates the association between trauma and borderline personality disorder. Changes in the brain regions caused by the trauma such as the amygdala, hippocampus, and prefrontal cortex were identified as the key agents that link to BPD symptoms like impulsivity and emotional risk regulation, these symptoms significantly overlap with ASD (Estric et al., 2022). It is crucial to address the neurodevelopment traits in BPD, to better understand the disorder and identify the vulnerable populations.

Recommendations

The shared features of ASD and BPD present diagnostic challenges due to overlapping traits in social cognition, emotional regulation, and sensory processing, emphasizing the need for careful diagnostic evaluation. Insights into these connectives have practical implications for treatment. Early interventions targeting individuals with a family history of ASD may mitigate the effects of ACEs and reduce BPD risks. Moreover, understanding the genetic similarities and acknowledging the neurodivergent traits in BPD can improve treatment models. Instead of focusing mostly on Dialectical Behavior Therapy for BPD there can be modified versions of Behavioral Therapies used for BPD. Furthermore, this narrative review can be used as the foundation for understanding the neurocognitive deficits in BPD.

Future research should focus on several key areas to deepen our understanding of the relationship between ASD and BPD. Longitudinal studies tracking individuals with a family history of ASD through adolescence are needed to clarify developmental trajectories and identify early indicators of BPD symptoms. Investigating specific genetic markers could help predict vulnerability to both conditions while exploring protective factors may uncover ways to mitigate the impact of ACEs in genetically predisposed individuals. Additionally, developing targeted interventions tailored to the overlapping traits of ASD and BPD can address the unique needs of this population, improving outcomes and informing clinical practices.

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