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Review

NEURODEVELOPMENTAL DISORDERS AND EATING DISORDERS: NEUROBIOLOGICAL CONNECTION

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SUMMARY

In the recent years, the co-occurrence of Neurodevelopmental disorders (NDDs) with Eating disorders (EDs) has gained increasing recognition, through multiple cross-sectional and longitudinal studies that demonstrated a significant overlap between these two conditions. This review aims to explore the neurobiological connections, the comorbidity and the shared pathways that relate these disorders, summarizing current findings.

Key words: *neurodevelopmental disorders - eating disorders – neurobiology – ADHD - autism spectrum disorder*

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INTRODUCTION

Neurodevelopmental disorders (NDDs), such as Attention-Deficit/Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD), are characterized by early-onset deficits in cognitive, behavioral, and social domains. These disorders often continue across the lifespan and are commonly associated with a range of psychiatric comorbidities. Among these, eating disorders (EDs) such as anorexia nervosa (AN), bulimia nervosa (BN), and binge-eating disorder (BED) are increasingly identified as clinically relevant co-occurring conditions (Boltri & Sapuppo 2021; Westwood & Tchanturia 2017). Recent research has highlighted a notable intersection between NDDs and EDs, particularly in shared behavioral phenotypes like impulsivity, emotional instability, and altered reward sensitivity (Kinnaird et al. 2023). Such parallels have prompted investigations into whether both types of disorders may arise from overlapping neurobiological pathways. In particular, dysfunctions in neurotransmitter systems, notably dopamine and serotonin, and abnormalities in brain circuits related to executive control and motivation (e.g., prefrontal cortex, striatum, and mesolimbic regions), have been implicated in both NDDs and EDs (Westwood & Tchanturia 2017).

Intellectual disabilities (ID) are conditions characterized by impairments in mental skills and daily functioning compared to peers (Dinkler et al. 2022). Individuals with ID show diverse challenges in social skills, communication, reasoning, learning, and problem-solving. About 2% of children in the general population have ID, but prevalence varies due to differing criteria, age, and country (Aksović 2023). Rates tend to be higher in youth and middle-income nations. ID must be evident during development, typically in childhood or adolescence, and is more common in males. Severity varies, depending on the level of support needed, and while ID can change over time, it is generally a lifelong condition with notable functional limitations, not always involving intellectual impairment. It has been shown that also these disabilities are correlated to eating disorder.

Understanding the biological connections between these disorders could improve diagnostic accuracy and inform more effective, individualized treatment strategies. Despite increasing interest in this topic, the scientific literature remains dispersed and lacks a cohesive synthesis of the underlying mechanisms linking these conditions.

This review aims to bring together existing findings on the neurobiological associations between neurodevelopmental and eating disorders. The discussion will focus on shared genetic factors, neurochemical alterations, and brain structure and function, as well as on the potential clinical implications of these overlapping features.

OVERVIEW OF NEURODEVELOPMENTAL DISORDERS

NDDs are a group of early-onset conditions characterized by deviations in brain maturation and functioning that affect cognitive, behavioral, and social domains. The most widely studied NDDs are ADHD and ASD, both of which display distinct yet overlapping clinical and neurobiological profiles (Hoogman et al. 2018; Kinnaird et al. 2023).

ADHD: Functional and Neurochemical Signatures

ADHD is commonly associated with alterations in brain morphology and connectivity. Structural neuroimaging studies have consistently identified reduced volumes in the prefrontal cortex, basal ganglia (notably the caudate nucleus and putamen), and cerebellum, particularly in pediatric populations (Hoogman et al. 2018). These anatomical variations are paralleled by disruptions in functional circuits that underlie executive control, attentional regulation, and motor planning, especially in the fronto-striatal and fronto-cerebellar pathways (Fried et al. 2021).

On a neurochemical level, ADHD is primarily linked to dysregulation of the dopaminergic and noradrenergic systems where their hypoactivity contributes to core symptoms such as inattention, impulsivity, and hyperactivity (Arnsten & Rubia 2012). Increased density of dopamine transporters (DAT) in the striatum leads to reduced synaptic dopamine availability, a mechanism targeted by stimulant medications like methylphenidate (Del Campo et al. 2011). In addition, other findings correlate serotonin, glutamate, and GABA in the neurobiology of ADHD, reflecting the disorder's complex and multifactorial nature (Cortese et al. 2021).

ASD: Brain Connectivity and Excitation/Inhibition Imbalance

In contrast, ASD involves atypical development of neural connectivity, particularly in circuits associated with social cognition, language, and sensory integration. Early brain overgrowth, followed by disrupted pruning and connectivity, has been reported in several longitudinal Magnetic Resonance Imaging (MRI) studies (Courchesne et al. 2011). These findings include variations in the frontal and temporal lobes, amygdala, cerebellum, and corpus callosum (Ecker et al. 2015).

At the synaptic level, ASD is characterized by dysfunction in genes involved in neuronal communication and synapse formation such as SHANK3, neurexin, and neuroligin, which contribute to altered synaptic plasticity (Zoghbi & Bear 2012). Moreover, an imbalance between excitatory (glutamatergic) and inhibitory (GABAergic) signaling is a core hypothesis in ASD pathophysiology, potentially underlying sensory hypersensitivity, social difficulties, and repetitive behaviors (Rubenstein & Merzenich, 2003). Serotonergic dysregulation is another well-documented feature of ASD. In fact, elevated blood serotonin levels (hyperserotonemia), along with altered serotonin transporter and receptor function, have been found in both postmortem and neuroimaging studies (Müller et al. 2016). Dopaminergic dysfunction, particularly in mesocorticolimbic pathways, may contribute to motivational and reward-processing differences observed in autistic individuals (Pavăl 2017).

Genetic Architecture of NDDs

Both ADHD and ASD show high heritability and complex genetic underpinnings. ADHD is associated with common variants in genes regulating dopamine and norepinephrine transmission such as DAT1 and DRD4 (Thapar et al. 2013). In ASD, genome-wide association studies have identified hundreds of risk genes, many of which are involved in synaptic scaffolding, transcriptional regulation, and neurodevelopmental signaling pathways (Sanders et al. 2015). On the other hand, de novo mutations and copy number variants further support the heterogeneous and polygenic nature of autism (Satterstrom et al. 2020).

OVERVIEW OF EATING DISORDERS: NEUROBIOLOGICAL FOUNDATIONS

EDs, including AN, BN, and BED, are complex mental health conditions involving persistent disturbances in eating behavior and a distorted perception of body image. In recent years, growing attention has been given to their neurobiological underpinnings, particularly the roles of serotonergic and dopaminergic systems, as well as the hypothalamic–pituitary–adrenal (HPA) axis.

Serotonin: Mood, Appetite, and Control

Serotonin (5-HT) plays a crucial role in regulating satiety, mood, and impulse control. In individuals with AN, research has shown altered serotonergic activity, especially in the hypothalamus and prefrontal cortex, which may contribute to appetite suppression and heightened anxiety around eating (Kaye et al. 2009). These individuals often present with reduced levels of 5-HIAA, a serotonin metabolite, in cerebrospinal fluid, particularly during the acute phase of the illness, with partial normalization during weight restoration (Frank et al. 2013).

In bulimia nervosa, serotonin dysfunction has been linked to impulsive eating behaviors and difficulties with inhibition. However, findings are less consistent than in AN, possibly due to the episodic nature of BN and the influence of binge/purge cycles on neurochemistry (Brewerton et al. 1995; Jimerson et al. 1990).

Dopamine: Reward Processing and Motivation

Dopamine (DA) is deeply involved in reward evaluation, reinforcement learning, and motivational drive. In AN, studies have reported decreased dopaminergic activity and lower levels of homovanillic acid (HVA), a dopamine metabolite, suggesting blunted sensitivity to reward and possibly a paradoxical anxiety response to food stimuli (Kaye et al. 2013). Functional imaging has highlighted abnormal activation of striatal regions, especially the nucleus accumbens and caudate, during food anticipation and consumption (Frank et al. 2013).

In contrast, individuals with BN and BED often exhibit heightened dopamine responses to highly palatable food cues. This hypersensitivity may promote binge eating behaviors, especially in the context of impaired inhibitory control or emotional dysregulation (Wang et al. 2004; Kuikka et al. 2001).

HPA Axis: Chronic Stress and Endocrine Dysregulation

The HPA axis orchestrates the body's response to stress, and its dysregulation is frequently observed in individuals with EDs. Elevated cortisol levels, both at baseline and in response to stress, have been reported in AN and BN, indicating a hyperactive stress response system (Monteleone et al. 2016). Interestingly, while baseline cortisol is often high, acute reactivity to stressors can be blunted, suggesting a potential desensitization of the HPA axis due to chronic activation (Lawson et al. 2011).

This hormonal imbalance can affect multiple brain regions, including the hippocampus and amygdala, and has been associated with persistent anxiety, disrupted hunger cues, and reduced reward sensitivity (Culbert et al. 2016).

Interaction Between Systems

The serotonergic, dopaminergic, and neuroendocrine systems do not act independently. For instance, serotonin receptors, especially 5-HT_2C, modulate the release of corticotropin-releasing hormone (CRH), linking serotonergic tone to HPA axis activation (Tao et al. 2007). Additionally, neuropeptides such as leptin, ghrelin, and peptide YY that play a crucial role in regulating appetite and energy balance, interact with dopamine-mediated circuits, influencing feeding motivation and homeostatic regulation (Monteleone et al. 2018).

These intertwined systems contribute to the maintenance of maladaptive behaviors in EDs by reinforcing food restriction or binge-purge cycles, and by altering the perception of hunger, stress, and reward.

COMORBIDITY BETWEEN NEURODEVELOPMENTAL DISORDERS AND EATING DISORDERS: EPIDEMIOLOGICAL INSIGHTS

ADHD and Eating Disorders

Meta-analytic evidence encompassing over 4,000 individuals with ADHD and nearly 30,000 controls has shown that individuals with ADHD have approximately a fourfold increased likelihood of developing any eating disorder compared to those without ADHD (pooled odds ratio [OR] = 3.82; 95% confidence interval [CI]: 2.34–6.24) (Cortese et al. 2016). This elevated risk extends to specific ED diagnoses, including AN (OR = 4.28), BN (OR = 5.71), and BED (OR = 4.13) (Cortese et al. 2016). Notably, studies employing clinical diagnostic interviews reported higher comorbidity rates than those relying on self-reported ADHD symptoms.

Conversely, the prevalence of ADHD among individuals diagnosed with an eating disorder is also higher than in the general population, with pooled OR estimates around 2.57 (95% CI: 1.30–5.11) (Yilmaz et al. 2017). This association is particularly pronounced in BED, where ADHD prevalence can reach up to 20%, compared to lower rates in restrictive AN (3–16%) and BN (9–35%) (Instanes et al. 2020). National survey data further corroborate these findings, indicating that ADHD symptoms often precede the onset of eating disorders, particularly those characterized by bingeing and purging behaviors (Ulfvebrand et al. 2015). Longitudinal studies reinforce the temporal link, suggesting that early ADHD traits may predispose individuals to developing disordered eating patterns later in life.

Additionally, individuals diagnosed with avoidant/restrictive food intake disorder (ARFID) also face a greater likelihood of having ADHD. For instance, research has indicated that up to 39% of children and teenagers undergoing treatment for ARFID also meet the diagnostic criteria for ADHD, compared to approximately 10% of the general population (Dinkler et al. 2022; APA 2022). Similar to other eating disorders, scientists believe that the higher occurrence of ARFID and co-existing ADHD may be linked to certain common traits shared by both conditions. These traits generally include being easily distracted, difficulties with impulsiveness, heightened activity levels, and struggling to remain seated to complete a meal, which might clarify why people with ADHD frequently exhibit the limited food consumption features associated with ARFID (Westwood & Tchanturia 2017).

ASD and Eating Disorders

There is increasing evidence that autism spectrum traits are disproportionately represented among individuals with anorexia nervosa. Systematic reviews and meta-analyses reveal that approximately 29% of people with AN score above clinical thresholds for ASD traits, a prevalence notably higher than that found in the general population (Westwood et al. 2017). Long-term follow-up studies of adolescents with AN show that up to 27% meet diagnostic criteria for ASD even 18 years after initial assessment (Huke et al. 2013). Additionally, the estimated prevalence of formal ASD diagnoses in AN populations is around 22.9% (Huke et al. 2013). These comorbidities may reflect overlapping neuropsychological characteristics, including cognitive rigidity, difficulties with set-shifting, and social communication challenges, which can contribute to both the development and persistence of eating disorder symptoms (Bentz et al. 2017).

Researchers have also discovered that individuals with autism appear to be at an increased risk for other types of eating disorders, including an eating disorder where individuals eat non-food items called pica, and ARFID, both of which are significantly more prevalent within this group (Kennedy et al.2022; Fields et al. 2021). For instance, studies have shown that as many as 30% of autistic children exhibit symptoms of pica, and 21% of autistic people have been diagnosed with ARFID (Baraskewich al. 2021; Koomar et al. 2021). The most common eating and feeding challenges observed in autistic individuals, also frequently seen in those diagnosed with ARFID or pica, include limited food preferences, heightened sensitivity to food textures, consuming only a specific brand of food, pocketing food without swallowing, restrictive mealtime routines, and ingesting non-food substances (pica) (Mayes et al. 2019). Although the connection between autism and ARFID has not been completely clarified, research has identified a genetic association (zswim6 gene) linking the two conditions (Koomar et al. 2021; Kozak et al. 2023).

ID AND EATING DISORDERS

It has been demonstrated that ID correlate with eating disorders. A study found that up to 80% of children with ID experience feeding or eating issues, such as trouble chewing, sucking, or swallowing, food texture or temperature preferences, extreme pickiness, disruptive behaviors during meals, and reflux or regurgitation (Gal et al. 2011). Those with more severe needs may have greater feeding difficulties and are at higher risk of aspiration, which can lead to serious health problems like poor growth, nutritional deficiencies, or the need for feeding tubes, all impacting quality of life (Dumont et al. 2023).

Pica is more common among people with ID, affecting up to 26% of children, especially in institutional settings (Kennedy et al. 2022). Additionally, up to 38% of children and teens with ARFID also have ID (Westwood & Tchanturia 2017). Higher rates of rumination disorder are also seen, with studies showing about 6-10% of individuals with ID engage in repetitive regurgitation behaviors, which can have a calming or stimulating effect, similar to other repetitive behaviors like head-banging (APA 2022; Wilder & Lipschultz 2016).

SHARED NEUROBIOLOGICAL PATHWAYS IN NEURODEVELOPMENTAL AND EATING DISORDERS

There is growing recognition that NDDs and EDs exhibit overlapping neurobiological substrates, which may help explain their frequent coexistence. These shared pathways encompass disruptions in neurotransmitter systems, brain circuits involved in reward and cognitive regulation, as well as dysregulation of the HPA axis.

Altered Dopaminergic Signaling

Abnormal dopamine function is a hallmark in both NDDs and EDs. Dopamine is essential for processes related to motivation, reward, and executive control. In ADHD, evidence points to reduced dopamine transporter availability and impaired dopamine transmission, which are linked to symptoms like inattention and impulsivity (Volkow et al. 2009). Likewise, in EDs, especially in binge eating disorder and bulimia nervosa, altered dopaminergic activity has been observed in response to food cues, reflecting disrupted reward processing (Wang et al. 2011).

Such atypical dopamine signaling patterns may hinder reward learning and cognitive flexibility, fostering maladaptive eating habits and challenges with self-regulation (Kaye et al. 2013; Instanes et al. 2020).

Serotonergic System Disruptions

Dysfunctions in serotonergic pathways have been found in both EDs and NDDs. For instance, decreased serotonergic activity correlates with heightened impulsivity and anxiety often reported in ADHD (Biederman et al. 2008), whereas abnormal serotonin receptor availability and transporter function have been consistently documented in anorexia nervosa and bulimia nervosa (Frank et al. 2013). These serotonergic abnormalities likely contribute to core clinical features such as restrictive or binge-purge eating patterns, as well as to deficits in executive function and emotional regulation observed across these disorders (Hale et al. 2017).

Executive Control and Cognitive Flexibility Networks

Neuroimaging research has highlighted impairments in frontostriatal circuits, key for executive functions like inhibition, mental flexibility, and working memory, in both NDDs and EDs (Halperin & Schulz 2006; Tchanturia et al. 2012). Difficulties in cognitive flexibility and impulse control may underlie the rigid, perseverative behaviors characteristic of ASD and anorexia nervosa, as well as the impulsive symptoms seen in ADHD and binge-type EDs (Roberts et al. 2016).

Furthermore, disrupted functional connectivity between the prefrontal cortex and subcortical regions such as the striatum and amygdala influences emotional regulation and reward processing in these disorders (Monkul et al. 2007; Kaye et al. 2013).

Dysregulation of the HPA Axis

Altered stress response systems, particularly involving the HPA axis, have been implicated in both neurodevelopmental and eating disorders. Increased cortisol secretion and changes in glucocorticoid receptor sensitivity have been reported in ADHD and anorexia

nervosa populations, suggesting a shared neuroendocrine imbalance (Kotwicki et al. 2020; Monteleone et al. 2016).

Such HPA axis dysfunction can intensify anxiety, disrupt appetite control, and negatively influence neurodevelopment, thereby perpetuating symptoms in both clinical conditions (Herman et al. 2016).

Neuroinflammation and Immune System Involvement

Recent studies propose that neuroinflammatory processes may contribute to the pathogenesis of both NDDs and EDs. Elevated levels of pro-inflammatory cytokines have been identified in individuals with ASD, ADHD, and anorexia nervosa, indicating immune dysregulation as a potential shared factor affecting symptom severity and progression (Müller et al. 2015; Solmi et al. 2021). Such immune alterations might impact synaptic function, neurotransmitter systems, and brain development, providing a plausible biological link between these disorders (Modabbernia et al. 2017). In conclusion, converging evidence points to common neurobiological alterations, including in dopamine and serotonin pathways, executive function networks, HPA axis regulation, and neuroimmune activity, that likely contribute to the overlapping clinical features and high rates of comorbidity observed between NDDs and EDs. This shared neurobiology highlights the importance of comprehensive assessment and integrated treatment approaches.

CLINICAL IMPLICATIONS

Differential Diagnosis

The frequent overlap between NDDs and various EDs complicates the diagnostic process. Shared traits like inflexible thinking, rigid routines, or unusual sensory sensitivities may blur the boundaries between these disorders, leading to delayed recognition or diagnostic confusion (Westwood & Tchanturia 2017). For example, selective eating or ritualized food behaviors in ASD might be mistaken for restrictive eating patterns seen in AN. A detailed clinical evaluation that includes neurodevelopmental history, behavioral observation, and neuropsychological screening tools is essential to accurately differentiate primary diagnoses from overlapping presentations (Kinnaird et al. 2019). Early identification of comorbidities is fundamental for treatment planning and prognosis.

Impact on Treatment and Therapeutic Management

When EDs and NDDs co-occur, treatment often becomes more complex and less responsive to conventional protocols. Standard therapies such as cognitive-behavioral therapy (CBT), may need to be adapted for individuals with executive functioning impairments, social cognition difficulties, or heightened sensory reactivity (Brede et al. 2020). For instance, patients with ASD may struggle with metaphorical language or abstract reasoning, requiring more structured and concrete interventions. Similarly, impulsivity and emotional dysregulation in ADHD can reduce compliance and consistency in therapy, demanding more flexible and reinforcement-based approaches (Koomar et al. 2021).

Pharmacological strategies may also need to be personalized, especially when treating comorbid symptoms like anxiety, mood disturbances, or attentional difficulties. This highlights the importance of interdisciplinary collaboration and continuous monitoring throughout the course of care.

Personalization of Interventions

Given the significant variability in clinical expression and neurobiological underpinnings, individualized treatment is essential for patients with concurrent EDs and NDDs. Tailoring interventions to specific cognitive styles, emotional needs, and behavioral profiles can significantly enhance therapeutic engagement and outcomes (Tchanturia et al. 2021). For example, integrating sensory-based techniques, visual supports, and emotion regulation training may help address challenges in patients with ASD, while structured routines and reward systems can support individuals with ADHD.

Additionally, involving families and caregivers in psychoeducation and behavior management increases treatment adherence and fosters a supportive recovery environment (Kinnaird et al. 2019). As our understanding of neurobiological mechanisms evolves, novel strategies such as digital therapeutics or neuromodulation may offer further personalization opportunities in this high-risk group.

DISCUSSION

The co-occurrence of NDDs and EDs represents an area of growing clinical relevance and scientific inquiry. A growing body of research, including epidemiological surveys, neurobiological findings, and clinical observations, has demonstrated that these disorders frequently coexist and may be linked through shared biological and cognitive pathways. These include disruptions in dopaminergic and serotonergic transmission, impairments in executive functioning, altered stress regulation via the HPA axis, and signs of neuroinflammatory involvement.

Such overlapping features underline the importance of heightened diagnostic vigilance, as symptom similarities can obscure accurate identification and delay the start of appropriate care. Differentiating between primary and comorbid presentations is essential for

tailoring interventions that reflect the individual's full clinical profile. Treatment outcomes can be significantly improved when approaches are customized to address the cognitive style, emotional regulation capacities, and sensory needs of each individual. Involving families in psychoeducational and behavioral strategies can further enhance engagement and reduce the likelihood of relapse. Moreover, advances in our understanding of shared neurobiological mechanisms may pave the way for innovative therapies, including neuromodulatory interventions and biologically-informed models of care.

CONCLUSIONS

Supporting individuals affected by both NDDs and EDs requires a multidisciplinary, flexible, and patient-centered approach. Continued collaboration between clinicians, researchers, and caregivers will be key to developing more effective and comprehensive interventions. Future research should focus on longitudinal designs and neurobiological biomarkers to better understand the shared etiology of NDDs and EDs.

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Contribution of individual authors:

Giada Juli & Rebecca Juli: conceptualization, data curation, formal analysis, investigation, methodology, project administration, visualization, validation, writing ± original draft, writing ± review & editing, supervision.

Alfredo Juli & Luigi Juli: conceptualization, visualization, literature searches and analyses, review & editing.

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