Review

The neurobiology of fear: understanding anxiety in autism spectrum disorder

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Abstract

Although anxiety is not considered a core feature of autism spectrum disorder (ASD), it adds a significant and distinct layer of complexity. While not included in the diagnostic criteria, anxiety disorders, including obsessive-compulsive disorder, are highly prevalent among individuals with ASD. Many exhibit clinically elevated levels of anxiety, which can exacerbate social, cognitive, and emotional challenges. Individuals with heightened anxiety are more prone to sustained negative emotional states, potentially reflecting dysfunction in brain systems involved in emotion regulation. Anxiety is widely understood to have a neurobiological basis, and extensive research has examined how its arousal influences behavioral development, particularly in neurotypical populations. This paper explores the neurodevelopmental underpinnings of anxiety within the context of ASD, with a focus on the amygdala, a structure heavily implicated in both anxiety and autism. We review the role of the prefrontal cortex in modulating amygdala function, and how atypical connectivity between these regions may contribute to the manifestation of anxiety symptoms in individuals with ASD.

Keywords: Autism Spectrum Disorder (ASD), anxiety, amygdala, neural circuits, brain connectivity, functional connectivity, neural mechanisms, emotion regulation, anxiety pathways in autism

1. Introduction

A significant change in contemporary thinking acknowledges that abnormal brain development is crucial to the emergence and manifestation of anxiety symptoms in individuals with autism spectrum disorder (ASD). Decades ago, Leo Kanner (1943), made some of the earliest efforts to understand the origins of autism, suggesting that a lack of emotional warmth from cold and emotionally distant parents can contribute to its development, potentially leading to emotional withdrawal and heightened vulnerability to anxiety. However, Kanner and Eisenberg later revised this perspective, acknowledging that while the emotional environment of the family may influence child development, autism itself appeared to be innate, with genetic and neurobiological underpinnings (Eisenberg & Kanner, 1956). Subsequent research has since firmly refuted the early psychogenic theories, identifying genetic and neurological factors as the primary contributors to autism (Sullivan et al., 2019; Amaral, 2017; Harris & Piven, 2016). Although researchers generally agree on a biological basis for autism, the precise neurodevelopmental pathways that lead to its characteristic brain differences are still under investigation (Geschwind & Levitt, 2007; Markram et al., 2007).

Anxiety is not a rare occurrence in the broader population of individuals with ASD. In fact, it manifests through a variety of behavioral responses and exhibits a wide range of physical expressions (Leachman et al., 2024). It is frequently characterized by heightened sensitivity to social interactions, which can substantially impair daily functioning. Research indicates that up to 40-50% of individuals diagnosed with autism also experience clinically significant anxiety, further complicating their developmental and social experience (Weston et al., 2016; Copeland et al., 2014). Many individuals with ASD experience elevated anxiety stemming from the challenges of navigating social interactions, managing sensory sensitivities, and adapting to unexpected changes in routine. Additionally, the communication difficulties frequently associated with ASD can foster feelings of isolation and misunderstanding, leading to heightened concerns about how they will be perceived or understood by others (Cacha, 2024; Iqbal et al., 2023). Sensory sensitivities may exacerbate anxiety, as overwhelming stimuli can provoke heightened stress responses or episodes of panic. (South & Rodgers, 2017). Furthermore, individuals with ASD often depend on structure and predictability in their daily routines, so unexpected changes or disruptions can be particularly stressful and significantly increase anxiety. Understanding these sensory sensitivities is essential for helping individuals with autism reduce anxiety and navigate their environments more comfortably. The interplay of ASD's unique features often sets the stage for anxiety to thrive unchecked, but with understanding and support, individuals with ASD can learn coping strategies to manage their anxiety effectively.

Clinically significant anxiety is estimated to affect 40-50% of individuals with ASD, emphasizing its prominence as one of the most common co-occurring mental health conditions within this population (Kerns et al., 2020). While the exact reasons are not fully understood, experts suggest it may be linked to challenges with social interactions and heightened sensory sensitivities that can contribute to increase levels of anxiety (Ezell et al., 2020; Spain et al., 2018). There is a neurobiological explanation for this phenomenon which revolves around differences in brain development and functioning (Herrington, et al., 2017). Specifically, the amygdala and prefrontal cortex, which play key roles in processing emotions and managing fear, may not function as efficiently in individuals with autism (Tian et al., 2022). Imagine an intricate network of wires within the brain, each dedicated to a distinct emotional function. In individuals with autism, certain brain circuits that govern emotional processing may be likened to a malfunctioning system, leading to increased levels of anxiety. This disruption in emotional regulation highlights the complexities faced by ASD individuals, who often experience challenges in regulating their emotions and fear responses. These difficulties can be attributed to alterations in brain structure, (Cisler et al., 2010) which impact their ability to process and respond to emotional stimuli effectively.

2. ASD-specific impediments to effective emotion regulation

Research indicates that ASD individuals often face distinct challenges in emotion regulation, largely due to neurodevelopmental differences affecting both cognitive and sensory processing (Jakobson & Rigby, 2021). A central difficulty in this regard is alexithymia, an impaired ability to identify or articulate their own emotions. Individuals with alexithymia often report feeling easily overwhelmed by sensory input, suggesting that such sensitivity may further disrupt the emotional regulation process (Starita & di Pellegrino, 2018). Moreover, heightened sensitivity to sensory stimuli can trigger intense emotional reactions, which may often impede an individual's ability to self-soothe or employ adaptive coping strategies (Syu et al., 2020). Moreover, the social communication deficits inherent in ASD further intensify these issues by limiting access to social support, thereby reducing opportunities for emotional validation and assistance (Leachman et al., 2024; Hassen, 2023; Ibrahim 2019). Consequently, the interplay of these factors cultivates a cycle of increased anxiety and frustration, underscoring the necessity for targeted interventions that promote emotional awareness and regulation skills tailored to the unique needs of individuals on the autism spectrum (Pawluk et al., 2021; Grandin et al., 2011).

Supporting studies demonstrate that emotional regulation is a fundamental component of mental health and overall well-being, it empowers individuals to manage their emotions effectively, navigate challenges with resilience, and foster healthy, balanced relationships (Menefee et al., 2022; Cserép et al., 2022). However, autistic individuals frequently encounter difficulties in effectively managing their emotions (Grandin et al., 2011). Multiple investigations suggest a connection between ASD and atypical amygdala activity, especially in fear and emotional regulation tasks. This connection underscores the importance of the amygdala in understanding the neurobiological underpinnings of these conditions (Gibbard et al., 2018; Gotts et al, 2012). This tiny, almond-shaped region of the brain is responsible for processing visual and auditory information to detect potential threats, triggering the body's fear and stress response (Cacha et al., 2020). This crucial function enables individuals to quickly identify potential threats and respond appropriately, enhancing their ability to stay safe and make effective decisions in high-risk situations (Cacha, 2024; Iqbal, 2023). In individuals with anxiety disorders, the amygdala can become overactive, resulting in heightened fear responses and persistent feelings of worry (Makris, 2022). The concern has become even more difficult with ASD individuals due to the variations in the size and function of their amygdala (Wang & Li, 2023). Previous studies have suggested that individuals with (ASD) may have an enlarged amygdala, which could contribute to difficulties in emotional processing and social understanding (Ecker et al., 2012; Gotts et al., 2012). These findings, however, are inconsistent across age groups, with some studies reporting enlargement primarily in younger children but not in adults. Research has indicated that ASD individuals exhibit variations in the size and functionality of the amygdala compared to neurotypical individuals (Gibbard et al., 2018). However, there is variation in whether amygdala enlargement is found, depending on the imaging methodology, sample characteristics, and developmental stage assessed. Addressing concerns related to individuals with ASD has become increasingly challenging due to the variations in the size and function of their amygdala, which can significantly affect their social interactions and communication abilities (Spagna et al., 2018). Considering that difficulties in social interaction and altered emotional processing are core features of autism, it is unsurprising that significant attention has been directed toward the role of the amygdala in the condition's underlying pathology.

3. The role of the prefrontal cortex, hippocampus, and amygdala in understanding ASD

To understand how anxiety manifests in individuals with autism spectrum disorder, it is essential to examine three key brain regions: the amygdala, hippocampus, and prefrontal cortex (PFC). These brain areas contribute differently: for example, the amygdala to emotion detection, the hippocampus to contextual memory, and the PFC to regulation and decision-making. In individuals with ASD, neural activation and connections among these areas often deviate from typical developmental trajectories, which may contribute to heightened anxiety responses (Amaral et al., 2008).

The amygdala, a central structure within the limbic system, plays a critical role in emotional processing, particularly in the detection and response to fear and threat-related stimuli. In individuals with ASD, the amygdala frequently exhibits heightened activity in response to social and environmental cues, contributing to increased emotional reactivity and heightened fear responses (Baron et al., 2020). This hyperactivity strengthens the emotional tagging of memories, causing neutral or ambiguous situations to be perceived as threatening, thus contributing to chronic anxiety (Kleinhans et al., 2020).

The hippocampus plays a central role in the formation and contextualization of memories, especially those related to fear or stress. It helps the brain distinguish between safe and dangerous environments by organizing memories in spatial and temporal contexts. However, in conditions involving chronic stress or trauma, such as Post-Traumatic Stress Disorder (PTSD), the hippocampus often shows structural abnormalities, including reduced volume, which can lead to fragmented or disorganized memory recall (Bremner et al., 1995). Similar abnormalities—such as altered hippocampal volume and disrupted connectivity have been observed in individuals with ASD (Schumann et al., 2009). These impairments can interfere with the ability to appropriately contextualize experiences, making it more difficult to discriminate between actual threats and benign situations, thereby exacerbating anxiety symptoms.

The prefrontal cortex (PFC) is crucial for executive functioning, including decision-making, attention regulation, and emotional control. It acts as a regulatory hub that modulates limbic system activity, particularly by down regulating the amygdala during perceived threats (Etkin et al., 2004). In individuals exposed to trauma or chronic stress, the PFC is often underactive, diminishing its ability to regulate emotional responses effectively (Shin et al., 2001). In ASD, hypoactivation or atypical connectivity within the PFC may contribute to difficulties in emotion regulation, poor impulse control, and heightened stress sensitivity (Di Martino et al., 2009). This reduced top-down control from the PFC to the amygdala further amplifies fear responses and may contribute to the persistence of anxiety symptoms in ASD populations.

Together, dysfunctions in these three brain regions form a neurobiological framework for understanding the elevated prevalence and intensity of anxiety in individuals with ASD. The interplay between heightened emotional salience (amygdala), impaired contextual processing (hippocampus), and reduced regulatory control (PFC) underpins the challenges these individuals face in navigating a world often perceived as unpredictable or threatening (Herrington et al., 2017a).

Difficulties in these three key areas of the brain help explain why individuals with ASD often experience higher levels of anxiety. The amygdala intensifies emotional responses, making feelings more overwhelming (Andrews et al., 2022). The hippocampus struggles to interpret the context of situations, which can lead to confusion or misreading social cues. Meanwhile, the prefrontal

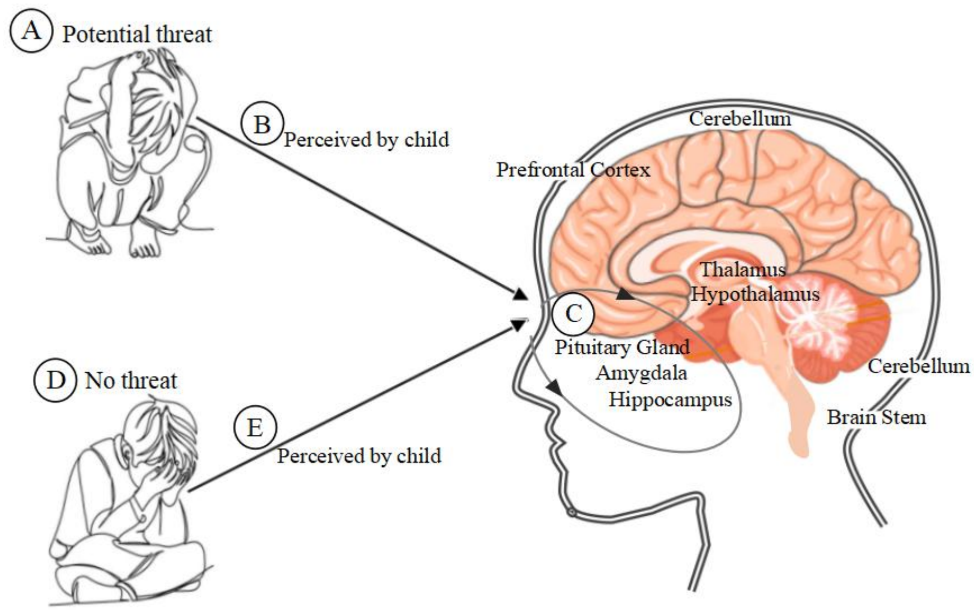

Figure 1: When a potential threat[A] is perceived by a child[B] with autism, the prefrontal cortex engages with the limbic system[C], which governs emotional responses. This interaction activates brain regions responsible for processing emotions, including the amygdala, hippocampus, and hypothalamus. Positioned below the thalamus and just above the pituitary gland, the hypothalamus plays a central role in managing the body's reaction to stress. In children diagnosed with ASD, repeated exposure to traumatic or stressful experiences can heighten their sensitivity to potential threats. As a result, even in the absence of actual danger[D], neutral or unclear stimuli may be misinterpreted as threatening[E]. This misinterpretation can set off the body's stress response by triggering the hypothalamic-pituitary-adrenal (HPA) axis. Over time, this pattern of heightened arousal may lead to persistent fear responses and could result in both structural and functional changes in the limbic system.

cortex (PFC) has trouble regulating reactions, making it harder to manage strong emotions (Sun et al., 2023). Together, these challenges make it difficult to navigate a world that often feels unpredictable or frightening.

Neuroimaging studies indicate that children with autism spectrum disorder (ASD) often exhibit an enlarged amygdala, particularly during early childhood, as well as atypical connectivity between the amygdala and prefrontal cortex (PFC), a pattern that may underlie difficulties in emotional regulation and stress management (Sofologi et al., 2020). These children also tend to display heightened physiological arousal, such as elevated heart rate or increased sweating in response to stress, reactions closely associated with amygdala activity (Kushki et al., 2013).

In addition to these limbic system differences, cerebellum and brainstem. Such alterations may disrupt neural connectivity and contribute to deficits in motor coordination, sensory processing, and autonomic regulation domains frequently impacted in ASD (Leisman et al., 2023). Furthermore, abnormalities in the brainstem may interfere with sleep regulation, arousal modulation, and communication between neural systems, adding to the complex neurobiological profile of autism.

Moreover, exposure to early life stress such as familial conflict or illness—has been associated with amygdala structural differences in children with ASD; positive parenting may help moderate these effects (Kuenzel et al., 2025). In general populations, chronic stress and trauma are known to reconfigure neural stress circuits, particularly impacting the amygdala and hippocampus. While explicit longitudinal data in ASD populations is still emerging, preliminary findings suggest that similar neuroplastic changes may occur, potentially compounding emotional dysregulation and anxiety symptoms over time.

4. Altered amygdala function in ASD: implication to HPA Axis dysregulation

Functional MRI studies have consistently demonstrated that individuals with autism spectrum disorder (ASD) often experience elevated levels of anxiety, which correspond with increased activation in the amygdala (Yang et al., 2024; Herrington et al., 2017a). Interestingly, this contrasts with findings that link core ASD symptoms, particularly social and emotional impairments with reduced amygdala activation (Herrington et al., 2017b). These patterns suggest that the amygdala serves a dual role: not only does it reflect an individual's internal emotional states, such as anxiety, but it also mirrors the cognitive demands they face in social situations.

Supporting this view, Tottenham & Gabard-Durnam (2017) reported that while anxiety symptoms in youth with ASD are associated with heightened amygdala activity, the defining traits of ASD are linked to reduced activation in this region. This imbalance in neural processing may contribute to difficulties in emotional regulation and social engagement, central challenges in ASD (Tottenham et al., 2014). Consistent with previous interpretations, Herrington et al. (2016) proposed that the amygdala functions as a "hybrid signal," encoding both emotional reactivity and social-cognitive effort.

Additional neuroimaging studies have revealed that the amygdala in individuals with ASD is not only more active but often structurally larger than in neurotypical peers (Yang et al., 2024). These differences may underlie the atypical emotional experiences and regulation strategies observed in autism (Andrews et al., 2022). Several studies have also linked these structural and functional variations to heightened stress reactivity and anxiety (Gao et al., 2022). This hyper-reactivity is believed to disrupt the hypothalamic-pituitary-adrenal (HPA) axis, the body's central stress response system, leading to abnormal cortisol levels and an increased risk for anxiety-related comorbidities (Pagliaccio et al., 2015; Weston, 2019).

The amygdala plays a central role in initiating the stress response through the hypothalamic-pituitary-adrenal (HPA) axis. Acting as the brain's alarm system, it rapidly detects emotionally significant stimuli such as potential threats and triggers the body's stress response (Hinds et al., 2022). Once activated, the HPA axis sets off a hormonal cascade: the hypothalamus releases corticotropin-releasing hormone (CRH), which stimulates the pituitary gland to secrete adrenocorticotropic hormone (ACTH). The ACTH subsequently stimulates the adrenal glands to secrete cortisol, a glucocorticoid hormone essential for orchestrating the body's stress response by mobilizing available energy, increasing vigilance, and facilitating adaptive reactions to perceived threats (Barry et al., 2017).

The amygdala initiates the stress response by engaging the HPA axis, which begins with the release of corticotropin-releasing hormone (CRH) from the hypothalamus. This triggers the pituitary gland to secrete adrenocorticotropic hormone (ACTH), which in turn stimulates the adrenal glands to release cortisol, a key hormone in managing the body's response to stress (Barry et al., 2017; Hinds et al., 2022). While the amygdala drives this immediate fear or anxiety-based reaction, the prefrontal cortex serves a counterbalancing role by evaluating the threat and regulating emotional output. In ASD, disruptions in this regulatory loop often result in abnormal cortisol patterns, such as elevated baseline levels, which may further impair emotional regulation (Cacha et al., 2020).

Abnormalities in amygdala function have been increasingly identified as a critical factor in ASD-related emotional and social processing (Fishman et al., 2018; Ogundele, 2018). Atypical activation patterns in this region are associated with deficits in recognizing emotional cues and managing interpersonal interactions. These effects may stem from altered development or overactivation of the amygdala, which could explain the heightened anxiety and stress sensitivity observed in this population (Kredlow, 2022; Hassen et al., 2023; Cacha, 2024). Emerging evidence also highlights dysfunction in the HPA axis among children and adolescents with ASD, suggesting a neuroendocrine basis for the anxiety symptoms frequently seen in this group (Makris et al., 2022).

Amygdala dysfunction, often observed in both ASD and anxiety disorders, has been linked to impairments in emotional regulation and social communication

(Frye, 2022; Gibbard et al., 2018; Sun et al., 2023). Neuroimaging studies have further shown disrupted connectivity between the amygdala and regions involved in social cognition, such as the prefrontal cortex and superior temporal sulcus (Dichter, 2012; Ecker et al., 2012). These findings suggest that the severity of social difficulties in ASD may be closely tied to the extent of amygdala irregularities, underlining the need for biologically-informed interventions.

Although the amygdala is a primary center for processing emotional and threat-related information, regions within the prefrontal cortex also play a pivotal role in regulating fear and anxiety responses. In individuals with ASD, atypical connectivity between the prefrontal cortex and amygdala may contribute to altered responses to fear-inducing stimuli (Shin & Liberzon, 2010; Herrington et al., 2016; Dichter et al., 2012). These findings emphasize the importance of considering the dynamic interplay between cortical and subcortical structures in understanding emotional regulation and stress responses in ASD.

5. Prefrontal cortex pathways for threat perception and response

Fear-processing circuits within the prefrontal cortex (PFC) constitute a neural network involved in the detection, interpretation, and modulation of responses to potentially threatening stimuli (Etkin et al., 2011). Neuroimaging studies have consistently reported atypical activation and connectivity patterns in the prefrontal cortex (PFC) of individuals with ASD, indicating both functional and structural alterations that may emerge early in development and persist across the lifespan (Uddin et al., 2013; Ecker et al., 2012).

The PFC plays a crucial role in fear processing, particularly in the acquisition of fear responses and in formulating strategies to regulate or keep fear reactions in check (Sun et al., 2023; Kredlow et al., 2022). It is apparent that amygdala and prefrontal cortex are two crucial brain structures responsible for processing emotions. Their connection is vital for regulating emotions effectively. Individuals diagnosed with ASD frequently demonstrate a diminished connection between frontal and posterior brain regions in comparison to neurotypical individuals (Just et al., 2012). This weakened connection can result in challenges in effectively managing fear and anxiety, which can exacerbate symptoms of the disorder (Kredlow et al., 2022). This weakened connection can also be a contributing factor to the social impairments often observed in individuals with ASD (Cho et al., 2013; Just et al., 2012).

The amygdala and prefrontal cortex play crucial and complimentary roles in the processing and regulating emotions. The amygdala is primarily responsible for the rapid detection of emotionally salient stimuli, particularly those related to fear and threat, and it initiates immediate emotional and physiological responses (Gibbard et al., 2018; LeDoux, 2000). In contrast, the prefrontal cortex handles higher cognitive functions including decision-making, impulse control, and emotion regulation (Jung et al., 2014). The PFC modulates these responses by exerting top-down control, enabling individuals to regulate emotional reactions, assess contextual factors, and engage in adaptive decision-making (Ochsner et al., 2012). The functional connection between these two structures is critical for maintaining emotional balance; the prefrontal cortex helps modulate the activity of the amygdala, allowing individuals to respond to emotional stimuli in a controlled and socially appropriate manner (Sai Sun et al., 2023; Berboth & Morawetz, 2021). Together, these structures form a dynamic neural network that balances emotional reactivity with cognitive regulation (Ibrahim et al., 2019; Etkin et al., 2011).

However, in individuals with ASD, this regulatory pathway is often disrupted. Studies have shown atypical connectivity between the prefrontal cortex and amygdala in ASD, which may contribute to heightened emotional reactivity, difficulty in interpreting social cues, and impaired emotion regulation (Uddin et al., 2013; Tottenham & Gabard-Durnam, 2017). Furthermore, disruptions in the connectivity between the amygdala and other brain regions, such as the prefrontal cortex and insula, have been implicated in anxiety symptoms in autism (Gibbard, 2018). These alterations may lead to difficulties in regulating emotions and responding appropriately to stressors, contributing to heightened levels of anxiety (Di Martino et al., 2009). This dysregulation can manifest as challenges in managing anxiety, frustration, or social engagement, often leading to behavioral responses that appear disproportionate or inappropriate in social context These neural differences may emerge early in development and persist across the lifespan, contributing to the core social-affective difficulties characteristic of ASD (Mosconi et al., 2009).

Recent work indicates that individuals with ASD often exhibit early enlargement of the amygdala, followed by normalization or reduction in volume, particularly within the basolateral amygdala subregions are changes associated with social impairments (Nordahl et al, 2015)

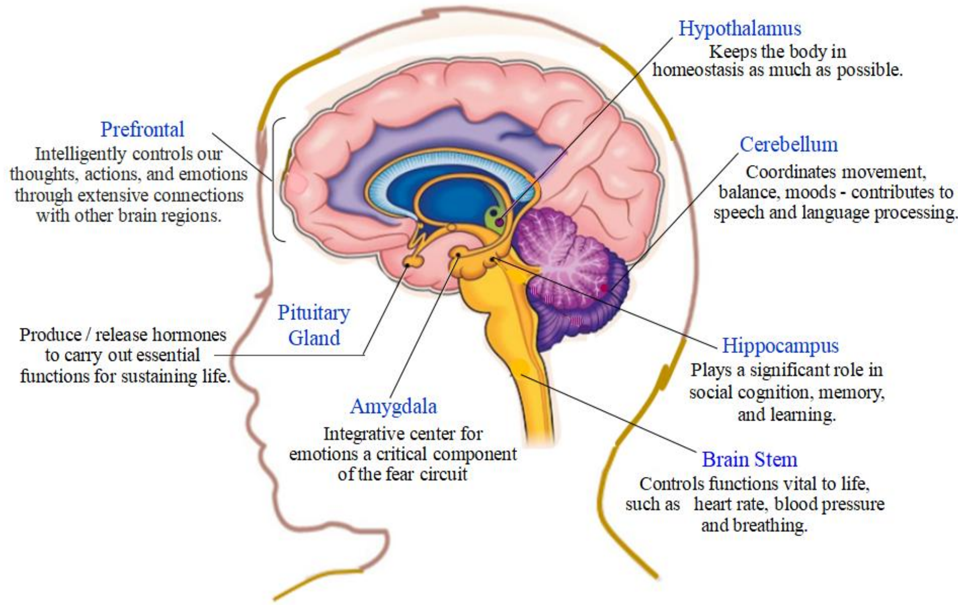

Figure 2: Highlights the key brain structures commonly associated with autism spectrum disorder, including the amygdala, prefrontal cortex, hypothalamus, pituitary gland, cerebellum, and brainstem. Gaining insights into how autism impacts the brain requires familiarity and understanding of the functions of these regions, which play critical roles in emotion regulation, social cognition, sensory processing, and motor coordination. Collectively, these structures are integral to the neural systems believed to mediate the core characteristics of ASD, such as challenges in social communication, the presence of repetitive behaviors, and atypical responses to sensory stimuli.

2015) commonly observed in ASD. Functional neuroimaging studies reveal decreased connectivity between the amygdala and medial prefrontal cortex in young children with ASD, which correlates with symptom severity (Shen et al., 2016). Additionally, dynamic increases in orbitofrontal cortex–amygdala functional connectivity have been linked to deficits in executive functioning and elevated repetitive and restricted behaviors (Chen et al., 2024). These neural differences may underlie challenges in emotion recognition, social interpretation, and relationship formation. By deepening our understanding of amygdala-related circuitry in ASD, researchers aim to develop more effective interventions to enhance social functioning and quality of life for affected individuals.

For people with autism spectrum disorder, this brain circuit may not work typically, making it harder to recognize and respond appropriately to social signals (Barry et al., 2017). Research has shown that individuals with ASD often have differences in the structure and function of both the amygdala and prefrontal cortex (Andrews et al., 2022). These differences may contribute to challenges in recognizing emotions, interpreting social situations, and forming relationships with others. By better understanding how this circuit operates in individuals with ASD, researchers hope to develop more effective interventions and therapies to improve social functioning and quality of life for those affected by the disorder.

6. Moving beyond the brain: autism through their subjective experience

To move beyond traditional understanding that often reduce fear-induced anxiety in ASD to mere amygdala hyperactivity or genetic predispositions (Herrington et al., 2016), it is crucial to shift our focus toward the lived experiences of autistic individuals. Fear in autism should not be narrowly conceptualized as a purely neurological reaction or inherited vulnerability (Leachman et al., 2024). Instead, it must be recognized as a profoundly embodied, cognitive, and existential event, one that unfolds dynamically in real time, intimately shaped by how each person feels, interprets, and ascribes meaning to their experiences (Wang & Li, 2023). Acknowledging this complexity is essential for a more humane and complete understanding.

Understanding the complex nature of fear and anxiety within the autistic community requires a broadened perspective that moves beyond the limitations of the conventional biological model. While biological explanations are essential, they often risk reducing fear to a mere physiological reaction, overlooking the complex, subjective and deeply personal ways in which it is truly experienced (Cummins et al., 2020). For autistic individuals, fear is not simply an automatic response to threat, it is profoundly shaped by sensory sensitivities, subjective awareness, social context, and personal history (Spain et al., 2018). It impacts not only physiological arousal but also cognitive processing, emotional regulation, and one's core sense of identity (Cserép et al., 2022). A similar multidimensional approach is necessary when considering the etiology of ADHD. Although genetic and neurobiological factors are significant, psychosocial influences, such as family dynamics, parenting styles, and environmental stressors also play a critical role in the development and expression of symptoms (Syu et al., 2020). Children exposed to chaotic or highly stressful environments may exhibit more severe ADHD-related behaviors, highlighting the importance of context in understanding neurodevelopmental conditions. (Wang & Li, 2023)

Every autistic person's experience of fear is deeply personal and multifaceted human experience, often shaped by a complex interplay of internal sensations, cognitive interpretations, and existential meanings that cannot be fully captured by brain scans or genetic markers alone (Ibrahim et al., 2019). By embracing this complexity, it encourages us to consider emotional, social, and subjective experiences, especially those that are harder to quantify, like fear, anxiety, or sensory sensitivity, one that honors the full humanity of autistic individuals and recognizes the diverse ways fear shapes their everyday lives (Pagliaccio et al., 2015).

Fear, whether from sensory overload, social misunderstanding, or past trauma, is a real and influential part of life for many autistic people (Jung et al., 2014). A deeper understanding of autism must recognize how fear affects behavior, communication, and well-being, rather than dismissing it as irrational or problematic behavior. This call for a shift from a purely scientific or clinical view of autism to one that respects and explores the emotional and lived experiences of autistic individuals (Copeland et al., 2014). It emphasizes empathy, depth, and a broader understanding, particularly around how fear impacts their daily life. Seeing fear as something that affects the body, mind, and sense of existence pushes us to move beyond simple explanations that often fail to capture the complexity of autistic experiences. This multidimensional understanding helps create approaches that truly connect with and validate the lived experiences of individuals on the autism spectrum.

Fear in autistic individuals often manifests through heightened physiological responses, such as elevated heart rate or muscle tension, triggered by sensory overload or social interactions. Cognitively, these individuals may interpret social cues differently, leading to misinterpretations that intensify anxiety. Existentially, fear may stem from a deep sense of alienation or challenges in forming identity within a neurotypical world that often marginalizes neurodiversity (Spain et al., 2018).

Addressing these complexities requires a holistic, empathetic approach to therapeutic practices. Trauma-informed care, for instance, can help practitioners understand the historical context of fear in autistic individuals, particularly in environments that have been unaccommodating or hostile (Brake et al., 2023). Additionally, mindfulness-based interventions can equip autistic individuals with tools to manage fear, fostering resilience and self-advocacy.

7. Conclusion

In conclusion, a growing body of evidence supports the view that anxiety in autism spectrum disorder (ASD) is rooted in neurodevelopmental differences, especially involving the amygdala and prefrontal cortex. The amygdala, often hyperactive or enlarged in ASD, contributes to heightened emotional reactivity and stress sensitivity. When its function is poorly regulated by the prefrontal cortex, the result is diminished top-down control over fear and emotional responses. This imbalance is further compounded by common features of ASD such as alexithymia, sensory hypersensitivity, and social communication difficulties, that impede the development of effective coping strategies.

The amygdala, often found to be enlarged or hyperactive in ASD, plays a key role in the heightened anxiety commonly observed in this population. Its dysregulation can lead to exaggerated stress responses and difficulties in social-emotional processing. Compounding this, abnormal connectivity with the prefrontal cortex compromises top-down regulation of fear and emotional reactivity. This neurobiological dysfunction is further intensified by factors such as alexithymia, sensory sensitivities, and social communication challenges, which together limit the development of adaptive coping strategies.

Understanding the interplay between the amygdala, prefrontal cortex, and the hypothalamic-pituitary-adrenal (HPA) axis offers critical insight into the biological mechanisms underlying anxiety in ASD. Disruptions in this interconnected system contribute to persistent physiological arousal, difficulty modulating emotional responses, and increased vulnerability to environmental stressors. These findings underscore the need for targeted interventions that support emotion regulation, stress management, and sensory integration, tailored to the unique neurodevelopmental profiles of autistic individuals.

Yet, anxiety in autism is not reducible to neural circuitry or genetic predisposition alone. It is a *lived, embodied experience*, deeply personal and shaped by the moment-to-moment realities of perception, interaction, and selfhood. A truly comprehensive understanding of fear in autism must account for its cognitive, emotional, and existential dimensions. This requires a shift toward empathetic, respectful methodologies that engage with the full complexity of autistic experience, rather than reducing it to pathology.

In this light, scientific inquiry must not only investigate the biological underpinnings of anxiety but also attend to how fear is felt, interpreted, and internalized, and how it informs behavior, identity, and well-being. This integrative perspective does not reject biology, but rather situates it within a broader, human context, where neurobiology, perception, *and* lived experience converge.

Conflict of interest

The authors declare no commercial or financial relationships that could create a conflict of interest.

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