# Hyperneuroplasticity as a Transdiagnostic Framework

## Neural Amplification Across Autism, ADHD, Trauma, and Gifted Neurodivergence

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## Abstract

Hyperplasticity, a condition of heightened neural plastic responsiveness, has gained traction as a unifying neurobiological mechanism across neurodivergent and trauma-exposed populations. This paper synthesizes recent findings from transcranial magnetic stimulation (TMS), functional neuroimaging, and neuroinflammatory research to argue that hyperplasticity may underlie both the cognitive strengths and vulnerabilities observed in autism, ADHD, early-life trauma, and gifted neurodivergence. Evidence is examined from electrophysiology, synaptic signaling, and structural connectivity studies, highlighting shared disruptions in excitatory/inhibitory balance, synaptic modulation, and neural circuit architecture. Implications for neurodiversity-affirming support include a shift from enhancement to modulation of plasticity, with emerging strategies offering promising new directions for supporting self-regulation, contextual adaptation, and individualized care in neurodivergent populations.

Building on foundational models such as the Intense World Theory (Markram et al., 2010) and integrating insights from transdiagnostic studies of synaptic regulation (Oberman & Pascual-Leone, 2014; Iuculano et al., 2023), this paper introduces the term *hyperneuroplasticity* as an umbrella construct. This construct reframes traits typically labeled as symptomatic or disordered, such as sensory amplification, cognitive intensity, or emotional reactivity, as context-sensitive expressions of a highly responsive nervous system. Hyperneuroplasticity offers a dimensional, systems-level framework for interpreting neurodevelopmental variation, emphasizing regulation, experiential timing, and environmental fit.

# 1. Introduction

The concept of hyperplasticity, a state of excessively heightened neural plasticity, has gained increasing empirical support, particularly when researching the experience of autistic individuals. Early and recent human studies using transcranial magnetic stimulation (TMS), especially continuous theta-burst stimulation (cTBS), reveal that autistic individuals demonstrate exaggerated synaptic potentiation or suppression effects, indicative of elevated long-term potentiation (LTP) and altered cortical excitability compared to neurotypical controls (Oberman et al., 2012; Desarkar et al., 2025).

Specifically, research employing cTBS over the primary motor cortex (M1) shows that individuals with autism exhibit greater and more persistent suppression or facilitation of motor-evoked potentials (MEPs), a physiological marker of plasticity, than age-matched controls (Oberman et al., 2012; Oberman et al., 2014; Jannati et al., 2020). These results support the notion of hyperplastic cortical responsiveness in this population.

Complementary evidence from electroencephalographic (EEG) and neuroimaging studies suggests increased short-range coherence and reduced long-range connectivity in autism (Barttfeld et al., 2010; Just et al., 2007), consistent with hyperplastic remodeling and local overconnectivity. A recent systematic review by Chen et al. (2024) confirms that autism is marked by atypical plasticity involving altered synaptic modulation, neuroinflammatory processes, and developmental trajectories across the lifespan.

In recognition of its broader relevance across multiple neurodevelopmental and experiential contexts, the emerging term **hyperneuroplasticity** is introduced as an umbrella construct encompassing these heightened and context-sensitive neural plastic responses. Unlike traditional views that isolate such plasticity effects within single diagnoses, hyperneuroplasticity offers a dimensional framework for understanding both enhanced cognitive functioning and increased susceptibility to dysregulation in neurodivergences such as autism, ADHD, cPTSD, and giftedness (Solomon Oldnman & P. Gently, personal communication, 2025).

New intervention models target modulation rather than enhancement of plasticity. Notably, Desarkar et al. (2025) describe targeted repetitive TMS (rTMS) protocols aimed at regulating hyperplastic responses in motor, sensory, and prefrontal regions, with observed improvements in executive and sensory function. These efforts, when supportive rather than corrective, emphasizing regulation rather than normalization, can be neurodiversity-affirming.

# 2. Literature Review

## 2.1 Autism and Hyperplasticity

Autism remains the most extensively investigated neurodevelopmental condition in relation to elevated plasticity. TMS-based protocols reliably demonstrate heightened cortical responsiveness in autistic individuals, evidenced by exaggerated long-term potentiation and abnormal corticospinal excitability (Oberman et al., 2010, 2012, 2016). Subsequent research (Oberman & Pascual-Leone, 2014) indicates this hyperplasticity persists into adulthood and may contribute to cognitive resilience.

Structural MRI studies reveal increased cortical thickness in auditory and temporal regions, including Heschl's gyrus. This finding is linked to sensory hypersensitivity in autism (Hyde et al., 2024; multi-site toddler MRI studies). Elevated cortical gyrification in temporal, parietal, and occipital lobes further suggests that microcircuit over-connectivity underlies atypical information processing.

Functionally, autism is marked by relatively stronger short-range coherence and weaker long-range connectivity, particularly across fronto-occipital and interhemispheric networks. These connectivity patterns align with hyperplastic microcircuit models and support intense, localized neural responsivity (Barttfeld et al., 2010; Just et al., 2007). Multimodal imaging combining EEG, fMRI, and DTI further reveals that altered structure-function relationships in autism are associated with atypical neural dynamics and flexible adaptation strategies.

Animal and developmental models of autism additionally identify disrupted critical-period plasticity linked to sleep disturbances and synaptic pruning irregularities, suggesting developmental timing of plasticity may remain prolonged in autism and contribute to heightened sensitivity (Medina, 2022).

Together, this converging neuroimaging and neurophysiological evidence solidifies autism as a prototypical example of hyperneuroplasticity, where intensified excitatory-inhibitory imbalance, disrupted synaptic modulation, and regionally heightened responsivity coalesce to create both pronounced strengths and regulatory vulnerabilities. This framework invites a developmental and systems-level interpretation of autistic neurobiology, emphasizing adaptation over pathology.

## 2.2 ADHD and Executive Differences

ADHD research suggests atypical or unstable plasticity, particularly in frontostriatal and prefrontal networks. Neuroimaging studies indicate cortical thinning and delayed maturation in medial and superior prefrontal regions essential for executive control and inhibition (Shaw et al., 2006; Hoogman et al., 2019). Reduced subcortical volumes, including in the amygdala and hippocampus, have also been reported, reflecting altered emotional regulation and memory systems.

Diffusion tensor imaging (DTI) reveals diminished white matter integrity in key tracts such as the corpus callosum and superior longitudinal fasciculus, suggesting compromised connectivity across executive networks (Hoogman et al., 2019). Functional MRI meta-analyses highlight reduced activation in fronto-cingulo-striatal circuits during attention and working memory tasks (Rubia et al., 2018), while resting-state studies report increased variability in posterior cingulate, medial prefrontal, and salience networks, findings indicative of heightened neural responsivity to internal and external cues (Misra & Gandhi, 2023).

Together, these findings support the view that ADHD is not simply a deficit in attention or inhibition, reflecting rather divergent regulation of neural plasticity. From a neurodiversity-affirming lens, this reinforces the importance of supporting cognitive modulation strategies that respect the individual's heightened responsivity, rather than enforcing normative pacing or attentional structures.

## 2.3 Trauma and Sensory Modulation

Chronic or early-life trauma can lead to hyper-reactivity in sensory and limbic networks through stress-mediated plasticity shifts. Neuroimaging studies have shown trauma-specific volumetric changes, such as reduced hippocampal and prefrontal cortex volumes, and altered connectivity between limbic and regulatory structures (Sullivan & Opendak, 2020).

Resting-state and task-based fMRI analyses reveal disrupted amygdala-prefrontal coupling, while prolonged activation of the hypothalamic-pituitary-adrenal (HPA) axis is associated with sustained elevation of pro-inflammatory markers, such as interleukin-6 and TNF-α (Estes & McAllister, 2015). These immune shifts interfere with normative synaptic pruning, dendritic arborization, and neurogenesis, particularly during sensitive developmental windows (Teicher et al., 2016).

Distinct trauma subtypes appear to yield divergent neurostructural and connectivity signatures. For example, childhood sexual abuse is associated with increased amygdala volume and hyperreactivity, while emotional neglect has been linked to reduced connectivity in

reward-processing circuits, including the nucleus accumbens and medial prefrontal pathways (Teicher et al., 2016; Hanson et al., 2021)

Such findings illustrate how early environmental threat shapes a hyperneuroplastic nervous system to prioritize safety, often at the cost of flexible regulation. Rather than viewing these adaptations as pathological, a neurodiversity-affirming interpretation recognizes them as contextually intelligible responses to adversity. The challenge lies in creating co-regulating environments that foster safety and choice, allowing these neural systems to recalibrate without shame or coercion.

## 2.4 Giftedness and Heightened Responsivity

Gifted individuals often exhibit rapid learning, sensitivity to complexity, and deep pattern recognition, suggesting elevated plastic responsiveness. Neuroimaging studies have documented that individuals with high intelligence show increased cortical thickness and greater gray matter volume in prefrontal and parietal cortices, along with elevated white matter coherence in regions associated with executive functioning and abstract reasoning (Narr et al., 2007; Choi et al., 2008; Jung & Haier, 2007; Shaw et al., 2006). DTI research further reveals efficient connectivity across the corpus callosum and fronto-parietal networks (Navas-Sánchez et al., 2016), aligning with the Parieto-Frontal Integration Theory (P-FIT) of intelligence. PET and fMRI studies have additionally demonstrated that individuals with higher IQ exhibit more efficient neural recruitment and metabolic activity during complex cognitive tasks (Haier et al., 2004).

Foundational work by Greenough et al. (1987), based on environmental enrichment in animal models, demonstrated that experience can directly shape neural architecture through dendritic growth and synaptogenesis. These principles later became central to the field of experience-dependent plasticity. From a developmental perspective, Gagné (2004) similarly proposed that enriched environments act as catalysts in transforming potential into realized talent. Together, these perspectives support the idea that heightened responsiveness in gifted individuals may emerge from the dynamic interaction between biological sensitivity and enriched input.

Comparative studies between gifted and autistic populations, such as Riccioni et al. (2021), reveal shared electrophysiological patterns and circuit-level dynamics, including increased local coherence and altered excitation-inhibition ratios. This builds on earlier conceptual work like the Intense World Theory (Markram et al., 2007), which proposes hyper-reactivity and local hyperplasticity in neural microcircuits—traits that may appear in both gifted and autistic individuals in differing contexts. Iuculano et al. (2023) also report that gifted and autistic

children may utilize variant learning pathways, involving either enhanced representational plasticity or adaptive neural stability.

Further evidence highlights co-occurring biological sensitivities, including atypical neuroimmune responses and disrupted synaptic pruning, reinforcing the plausibility of a shared plasticity-based substrate across gifted and neurodivergent profiles (Estes & McAllister, 2015; Hanson et al., 2013). Oberman and Pascual-Leone (2014) further suggest that elevated cortical plasticity, observed in autistic individuals, may provide cognitive protection later in life, supporting the broader developmental value of modulated hyperplasticity across populations.

## 2.5 Converging Mechanisms: Excitation/Inhibition, Inflammation, and Connectivity

Across neurodivergent and trauma-related conditions, several common neurobiological mechanisms emerge that offer insight into the underlying basis for hyperplasticity. One of the most well-documented involves the disruption in excitatory and inhibitory neurotransmission. Typically, a balance between glutamatergic excitation and GABAergic inhibition ensures regulated synaptic responsiveness. In autistic individuals, for example, evidence points to excessive excitation via NMDA receptor overactivation, coupled with insufficient inhibitory GABAergic tone (Rubenstein & Merzenich, 2003; Nelson & Valakh, 2015). This excitatory/inhibitory (E/I) imbalance creates a lower threshold for synaptic plasticity, resulting in circuits that are more readily modified in response to input. Similar patterns have been observed in ADHD and trauma, where prefrontal cortical networks may be either hyperexcitable or insufficiently inhibited, leading to either erratic attention or chronic hypervigilance (Sagvolden et al., 2005; Biederman et al., 2005).

A second converging mechanism centers on the role of neuroinflammation. Cytokine signaling, microglial activation, and other immune processes have been shown to interfere with synaptic pruning, dendritic arborization, and neurogenesis, particularly during sensitive developmental windows (Estes & McAllister, 2015). In trauma-exposed individuals, prolonged activation of the hypothalamic-pituitary-adrenal (HPA) axis can result in chronic elevation of inflammatory markers, which in turn disrupt normal plastic adaptation and maintenance (Heim et al., 2008). In autism, elevated levels of interleukin-6 and tumor necrosis factor-alpha have been associated with altered plasticity and connectivity, suggesting that immune signaling may be a significant modulator of hyperplastic tendencies (Ashwood et al., 2011; Li et al., 2009).

A third domain involves patterns of neural connectivity. Hyperplasticity is often accompanied by increased local connectivity, likely a result of overactive short-range synaptogenesis, and reduced long-range coherence, which impairs integrative processing across distributed

networks. In autism, this is evidenced by stronger short-range synchronization in sensory and associative cortices and diminished connectivity between frontal and posterior regions (Barttfeld et al., 2010; Just et al., 2007; Courchesne et al., 2011; Zikopoulos & Barbas, 2010). These architectural patterns suggest that while local specialization may be enhanced, the capacity for top-down modulation and global coordination is weakened. This profile aligns closely with the behavioral presentation of enhanced detail focus alongside difficulty with context integration or executive oversight.

# 3. Implications

The synthesis of findings across autism, ADHD, trauma-exposed populations, and giftedness supports the view that hyperplasticity represents a cross-cutting neurobiological feature, one that amplifies both adaptability and sensitivity to context. In contrast to models that position plasticity as an unqualified benefit, this framework emphasizes its dialectical nature: heightened plasticity can lead to exceptional cognitive development or to heightened vulnerability, depending on regulatory mechanisms and environmental input.

## 3.1 Implications of the Hyperneuroplasticity Umbrella Framework

Understanding hyperneuroplasticity as an umbrella construct across neurodivergent and trauma-related conditions carries several key implications:

First, it invites a diagnostic reframing. Rather than isolating autism, ADHD, trauma responses, and giftedness as distinct clinical categories, this perspective encourages dimensional understanding based on shared neurobiological tendencies. Heightened plastic responsiveness can be seen as a foundational substrate expressed differently across developmental pathways, depending on timing, context, and regulation.

Second, it shifts intervention design toward shared principles of modulation, regulation, and environmental attunement. If dysregulated plasticity contributes to both strengths and struggles across conditions, then unified approaches to supporting plasticity, such as sensory integration, emotional co-regulation, and timing-sensitive learning supports, become more viable than diagnosis-specific protocols.

Third, it expands research methodology. Neuroscience can move beyond siloed studies of "disorder-specific" biomarkers to focus on cross-cutting patterns in connectivity, synaptic signaling, immune activation, and structural variability. Research emphasis can shift toward investigating how brains adapt under different contextual loads, rather than pathologizing variation.

Fourth, it reorients clinical and educational practice. Professionals are encouraged to shape environments that respect individual neurobiological thresholds and amplify self-guided growth. This includes predictable, attuned, and sensory-stabilizing contexts that reduce overwhelm while promoting learning, agency, and safety.

Fifth, it reduces stigma. When hyperplasticity is framed as a neurodevelopmental variant rather than a pathology, its expression in emotional intensity, sensory reactivity, divergent thinking, or heightened vigilance can be interpreted with compassion, respect, and developmental insight.

## 3.2 Therapeutic and Environmental Considerations

These implications call for a shift in how we conceptualize intervention. Rather than viewing heightened plasticity as a dysfunction to be normalized, a neurodiversity-affirming framework recognizes it as a core difference that interacts dynamically with environment and context. Interventions should aim to support individuals in navigating these with agency and self-awareness, rather than conforming and reducing plasticity itself.

Targeted modulation of neural responses, particularly through environmental co-regulation, interpersonal attunement, and adaptive sensory supports, can be effective when aligned with individual goals and lived experience. Desarkar et al. (2025) demonstrate that modulation of plasticity can support sensory and executive regulation in autistic adults. Iuculano et al. (2023) show that autistic children may rely on stable rather than shifting neural activation patterns, calling for interventions that reflect these unique strategies. Oberman and Pascual-Leone (2014) report lifelong preservation of cortical plasticity in autism, while Markram et al. (2010) suggest that intense neural responsivity amplifies both memory encoding and environmental sensitivity.

These findings underscore the need for collaborative, individualized approaches. Neuroimaging studies by Narr et al. (2007) and Choi et al. (2008) document that individuals with high intelligence exhibit increased cortical thickness, enhanced gray matter volume, and elevated white matter coherence, supporting the view that hyperplastic profiles can be developmentally beneficial, rather than viewed as deficient.

Within psychosocial and educational contexts, hyperplastic brains are more influenced by their environments. In enriched, structured, and attuned conditions, plasticity may foster learning, creativity, and emotional insight. In contrast, chaotic or misattuned environments may intensify emotional reactivity and encode maladaptive patterns. Support strategies should move beyond accommodation to the intentional shaping of environments that affirm identity, promote agency, and reduce systemic stressors.

Finally, naming hyperneuroplasticity as an umbrella construct helps reconceptualize symptomatology as a shared expression of neurobiological variation. This invites dimensional, developmentally sensitive, and person-centered models that emphasize contextual fit and reject deficit-based assumptions. Spriggs et al. (2024) further suggest that hyperplastic states can be induced or modulated pharmacologically, reinforcing the biological malleability and context sensitivity of plasticity itself.

## 4. Toward a Synthesis

Conceptualizing hyperplasticity as a transdiagnostic mechanism invites a reconfiguration of neurodevelopmental models. Instead of treating autism, ADHD, trauma responses, and giftedness as distinct entities, each with unique etiologies and symptomologies, we might view them as different expressions of a common neurobiological substrate organized along dimensions of regulation, developmental timing, and experiential context.

This reconceptualization aligns with the emerging construct of **hyperneuroplasticity**, which integrates the shared mechanisms of neural amplification across diagnostic boundaries. Rather than situating each condition within siloed etiological narratives, hyperneuroplasticity emphasizes plastic responsivity as a dynamic trait influenced by genetic predispositions, environmental input, and systemic regulation. It introduces a framework for understanding how vulnerability relates to capacity and how dysregulation may give rise to exceptional adaptation.

In this view, differences arise not from the presence or absence of plasticity, but from how that plasticity is expressed, constrained, or amplified in the context of biology, history, and environment. This shift opens up new avenues for research and care that are dimensional, personalized, and grounded in developmental neuroscience and lived experience.

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