How do anomalies in Sensory Processing Integration drive Transdiagnostic Phenotypes in Neurodevelopmental and Psychiatric Disorders?

Anomalous sensory integration appears to be a key transdiagnostic driver of neurodevelopmental and psychiatric phenotypes.

Across autism, ADHD, and a wide range of psychiatric disorders, atypical sensory processing and multisensory integration are common, strongly linked to core symptoms, and often cut across traditional diagnostic boundaries.

Transdiagnostic Sensory Phenotypes

- A meta-analysis of 33 studies (N=2008) found elevated **low registration, sensory sensitivity, and sensory avoiding**, with reduced sensory seeking, across diverse psychiatric disorders, supporting sensory difficulties as a **non-specific transdiagnostic phenotype**. (Van Den Boogert et al., 2022)- In autism and ADHD, five data-driven **sensory phenotypes** were nearly identical across diagnoses and predicted similar patterns of clinical traits, indicating shared underlying mechanisms. (Scheerer et al., 2021)- Resting-state fMRI shows that multisensory network connectivity clusters children by **severity of sensory issues, not diagnosis**, again implying transdiagnostic neurobiology. (Choi et al., 2025)## **Links to Emotion, Attention, and Social Phenotypes**
- In preschool NDDs, atypical sensory processing (~40% prevalence) correlates with **behavioral problems** and may drive abnormal arousal, distress, and emotional dysregulation that worsen communication and social skills. (Gigliotti et al., 2024)- In a mixed neurodevelopmental cohort, distinct sensory subtypes (over-responsive, under-responsive, seeking) map onto **anxiety, ADHD symptoms, and emotion dysregulation**, suggesting common regulatory mechanisms. (Brandes-Aitken et al., 2024)- Meta-analysis in ADHD shows robust elevations in all four sensory quadrants (sensitivity, avoiding, seeking, low registration), arguing sensory atypicalities are central to that phenotype as well. (Jurek et al., 2025)### Examples across conditions

Condition / spectrum	Key sensory anomaly	Linked phenotype	Papers
Autism (children)	Hyper/hypo-reactivity, proprioceptive/vestibular issues	Worsened core social/communication symptoms	(Gigliotti et al., 2024)
Autism & ADHD	Shared sensory phenotypes	Similar patterns of ASD/ADHD traits	(Scheerer et al., 2021)
Psychiatric disorders broadly	Low registration, sensitivity, avoiding	Transdiagnostic sensory difficulty profile	(Van Den Boogert et al., 2022)

FIGURE 1 Sensory anomalies and associated transdiagnostic traits

Mechanistic Pathways: Circuits and Multisensory Integration

- Reviews highlight altered **multisensory integration (MSI)** and delayed MSI development in autism and schizophrenia, often with intact unisensory processing but impaired integration as complexity increases, impacting verbal and non-verbal social communication. (Cascio et al., 2019; Beker et al., 2017)- Genetic and environmental disruptions of sensory circuit development, especially thalamocortical and multisensory cortical circuits, are proposed as common roots of sensory dysfunction across the neuropsychiatric spectrum. (Hornix et al., 2019)- In ASD, proposed mechanisms include **E–I imbalance, thalamocortical and long-range connectivity abnormalities, and temporal/multisensory integration deficits**, which together alter gain control, timing, and reliance on raw sensory input, with downstream effects on attention and emotion. (Consoli et al., 2025)- Experimental work in Sensory Processing Disorder (SPD) and ASD shows reduced behavioral benefit from audiovisual redundancy and altered early and later MSI ERP components, consistent with disrupted integration or later attentional processing. (Molholm et al., 2020; Passarello et al., 2022)## **Developmental Cascades and Intervention Implications**
- Multiple reviews argue that early sensory dysregulation in ASD can **cascade into social deficits** over development, via altered arousal, attention, and learning from social cues. (Thye et al., 2017; Baum et al., 2015)- Sensory processing difficulties in NDDs and SPDs are framed as **risk factors for broader emotional, behavioral, and social problems**, often persisting into adulthood and influencing treatment response. (Gigliotti et al., 2024; Passarello et al., 2022)- Neuroplasticity-based models in ASD propose that maladaptive plasticity in sensory circuits (including altered glutamate/GABA balance and connectivity) underlies sensory integration deficits, and that **targeted sensory and neuromodulation interventions** may reshape these circuits. (Suprunowicz et al., 2025)## **Conclusion**

Across neurodevelopmental and psychiatric disorders, anomalous sensory processing and multisensory integration form shared, transdiagnostic phenotypes. These anomalies appear to shape emotion regulation, attention, and social communication through developmental cascades built on disrupted sensory circuits and MSI networks. Current work increasingly views diagnoses like autism, ADHD, and broader psychiatric conditions as partly different expressions of common sensory–neurobiological liabilities, highlighting sensory circuits as promising targets for assessment and intervention.

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