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CORRELATION BETWEEN GIFTEDNESS AND AUTISM: AN EVOLUTIONARY AND NEURODEVELOPMENTAL MODEL

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Abstract: Giftedness and autism spectrum disorder (ASD) have partially overlapping neurofunctional and structural characteristics, suggesting a common neuroevolutionary origin modulated by differences in brain development. This article proposes that both phenotypes represent gradients of a neurodivergent spectrum, defined by variations in the timing of maturation of the mesolimbic system and the dorsolateral prefrontal cortex (DLPFC). The analysis integrates neuroanatomical, neurochemical and molecular data, with an emphasis on synaptic connectivity, neural plasticity and genetic polymorphisms identified in genome-wide association studies (GWAS). Giftedness is characterized by greater functional integration between neural networks, while ASD presents local hyperconnectivity associated with specific talents. The redefinition of giftedness, focusing on integrative capacity rather than one-dimensional metrics such as intelligence quotient (IQ), is discussed on the basis of neuroscientific evidence.
Keywords: Giftedness, Autism, Neurodevelopment, Mesolimbic System, Prefrontal Cortex, Neural Plasticity.

INTRODUCTION

Giftedness and autism spectrum disorder (ASD) share neurocognitive characteristics that challenge traditional diagnostic categorizations. There is a higher prevalence of specific talents in individuals with ASD (Happé; Frith, 2009) and advanced cognitive abilities in gifted individuals (Rodrigues, 2023). These similarities suggest a possible shared neuroevolutionary origin, with phenotypic differences resulting from variations in brain development. This study proposes that giftedness and ASD represent distinct manifestations of a neurodivergent spectrum, modulated by temporal differences in the maturation of specific brain regions, such as the mesolimbic system and the dorsolateral prefrontal cortex (DLPFC). The analysis is based on neuroanatomical, neurochemical and molecular evidence, with a focus on synaptic connectivity, neural plasticity and genetic polymorphisms.

The hypothesis that autism and giftedness share common neuroevolutionary roots, but are differentiated by specific functional and genetic developmental trajectories, is supported by neuroimaging, genomic and comparative phylogenetic data. Both conditions show an increase in synaptic connectivity at early stages of development, especially in the temporal and frontal regions, although with different outcomes in terms of inter-network integration. In the case of ASD, there is evidence of accelerated gray matter growth in the first years of life, followed by stagnation and reduced functional efficiency in social integration networks (Hazlett et al., 2011). In gifted individuals, there is a prolongation of cortical plasticity and slower but more adaptive maturation, with a progressive increase in dendritic and synaptic density in associative areas (Shaw et al., 2006).

From a genetic point of view, GWAS studies reveal that genetic variants associated with high intelligence (such as CHRM2, FNBP1L, NRG1) also appear in subgroups of the autistic spectrum with high cognitive performance, indicating partial overlap of loci implicated in neuroplasticity and neuronal differentiation (Plomin; Von Stumm, 2018). However, other variants - such as duplications in 16p11.2 or deletions in SHANK3 - are more present in autistics with global functional deficits, suggesting that the presence or absence of compensatory mechanisms determines the phenotypic outcome.

From a phylogenetic perspective, the maintenance of autistic traits and high cognition in human populations can be interpreted as an expression of functional evolutionary divergence, in which different adaptive specializations are preserved as a function of specific

contextual advantages. The autistic brain, with its tendency towards extreme systematization and sensory hypersensitivity, represents a version of high specialization focused on pattern detection and rigid predictability. The gifted brain, on the other hand, manifests an expansion of the adaptive repertoire, with greater executive flexibility, divergent creativity and socially modulated efficiency. Both configurations involve a high energy cost (neuronal growth, synaptic maintenance, glutamatergic excitability), which supports the balanced selection hypothesis: the same biological substrate can generate advantages or risks, depending on the combination and modulation of the networks involved (Crespi; Badcock, 2008).

The genetic and structural convergence suggests that autism and giftedness should not be analyzed as dichotomous extremes, but as distinct functional unfoldings of the same evolutionary axis. The turning point lies not only in the presence of specific genes, but in the way these genes are expressed temporally and modulated by epigenetic, environmental and hormonal factors during critical periods of neural development. This shifts the clinical paradigm from classification to precise functional characterization, based on connectivity maps and synaptic architecture rather than syndromic categories.

OBJECTIVES

GENERAL OBJECTIVE

To understand the relationship between giftedness and Autism Spectrum Disorder (ASD) from the perspective of a neurodivergent spectrum, investigating the common neurobiological bases and distinct developmental trajectories that characterize both phenotypes.

SPECIFIC OBJECTIVES

- To analyze the neuroanatomical and neurochemical differences and similarities, especially in the mesolimbic system and the dorsolateral prefrontal cortex (DLPFC), in individuals with giftedness and ASD.
- To investigate the role of brain maturation timing, synaptic connectivity and neural plasticity in the manifestation of cognitive and behavioral characteristics associated with giftedness and ASD.
- Discuss the implications of the neurodivergent spectrum perspective for redefining the concept of giftedness and for developing more effective and inclusive assessment and intervention strategies for both groups.

LITERATURE REVIEW

BRAIN STRUCTURES INVOLVED

The mesolimbic system, composed of the ventral tegmental area (VTA) and nucleus accumbens (NAc), regulates reward and motivation processes mediated by dopamine (Schultz, 2015). In individuals with ASD, functional neuroimaging studies indicate hyperactivation of the VTA and greater synaptic density in the NAc, associated with restricted interests and specific talents (Dichter et al., 2012). In gifted individuals, greater functional connectivity is observed between the DLPFC (Brodmann area 9/46) and the frontoparietal network (FPN), correlated with advanced executive functions such as planning and inhibitory control (Rodrigues, 2023).

The posterior parietal cortex (PPC), including the superior parietal lobe (BA7), also shows differences. In ASD, the PPC exhibits local hyperconnectivity, associated with detailed sensory processing (Belmonte et al., 2004). In gifted individuals, the PPC integrates sensory information with executive networks,

facilitating complex problem solving (Rodrigues, 2023). The temporoparietal junction (TPJ, BA39/40) plays a critical role in social cognition, with lower activation in ASD and greater functional flexibility in gifted individuals (Saxe; Kanwisher, 2003).

NEUROCHEMICAL PROCESSES

Dopamine, released by VTA, modulates synaptic plasticity in the NAc and DLPFC. In ASD, dopaminergic hyperactivity in the NAc is associated with repetitive patterns and intense focus (Dichter et al., 2012). In gifted individuals, dopamine facilitates integration between DLPFC and FPN, promoting greater synaptic efficiency (Rodrigues, 2023). Glutamate, the main excitatory neurotransmitter, regulates activity-dependent plasticity. Studies indicate a higher density of NMDA receptors in ASD, correlated with local hyperconnectivity (Hutsler; Zhang, 2010). In gifted individuals, glutamatergic homeostasis in the DLPFC supports greater adaptive plasticity (Rodrigues, 2023).

ATP, which is essential for synaptic transmission, is in greater demand in ASD due to neuronal hyperactivity, while in gifted people, metabolic efficiency optimizes energy consumption (Rodrigues, 2023). Ions such as calcium (Ca^{2+}) regulate the release of neurotransmitters. In ASD, dysregulations in Ca^{2+} channels (CACNA1C) are associated with hyperactive synapses (Splawski et al., 2006). In gifted individuals, Ca^{2+} signaling in the DLPFC is more balanced, favoring executive functions (Rodrigues, 2023).

MOLECULAR DATA

Genome wide association studies (GWAS) have identified polymorphisms associated with ASD and giftedness. In ASD, variants in the SHANK3 (synaptogenesis) and NRXN1 (synaptic connections) genes are prevalent (Durand et al., 2007). In gifted individuals, polymorphisms in the COMT gene (dopamine metabolism) are associated with greater cognitive efficiency (Rodrigues, 2023). Genetic overlaps, such as variants in CNTNAP2 (neural connectivity), suggest a common molecular basis (Arking et al., 2008). Epigenetic factors, such as DNA methylation, modulate the expression of these genes, influencing the timing of brain maturation (Rodrigues, 2023).

TIMING OF BRAIN MATURATION

Early maturation of the mesolimbic system in ASD, observed in longitudinal neuroimaging studies (Courchesne et al., 2007), favors local hyperconnectivity and specific talents, such as detailed memory or mathematical skills. However, the late maturation of the DLPFC limits social integration and executive functions. In gifted individuals, the robust development of the DLPFC, with a higher density of dendrites and synapses, supports integration between neural networks, such as the FPN and the default mode network (DMN) (Rodrigues, 2023).

The hypothesis proposes that giftedness and ASD represent gradients in a neurodivergent spectrum, defined by temporal differences in the activation of neural networks. The critical developmental window, between 0 and 5 years, is a determining factor for these trajectories (Courchesne et al., 2007).

CONNECTIVITY AND NEURAL PLASTICITY

Local hyperconnectivity in ASD, observed in regions such as the visual cortex (V1) and PPC, is associated with greater synaptic density and less neural pruning (Hutsler; Zhang, 2010). In gifted individuals, long-distance connectivity, mediated by tracts such as the superior longitudinal fasciculus, facilitates integration between DLPFC, TPJ and PPC (Rodrigues, 2023). The same author states that neural plasticity, dependent on mechanisms

such as long-term potentiation (LTP), is more adaptive in gifted people due to glutamatergic and dopaminergic homeostasis.

NEURODIVERGENT SPECTRUM

The rigid categorization of giftedness and ASD is insufficient to capture phenotypic variability. Giftedness is defined by the integrative capacity between neural networks, while ASD is characterized by isolated performance peaks. This distinction is supported by neuroimaging studies that show greater functional coherence in the DMN in gifted individuals and fragmentation in ASD (Rodrigues, 2023; Belmonte et al., 2004).

Intelligence quotient (IQ) is a one-dimensional metric that does not capture the complexity of giftedness. Integrative capacity, mediated by greater connectivity between DLPFC, TPJ and DMN, is a more robust marker (Rodrigues, 2023). Tests of executive functions, such as the Wisconsin Card Sorting Test, and functional neuroimaging can replace IQ in identifying gifted individuals. For individuals with ASD, interventions that promote social integration, such as cognitive-behavioral training, can harness specific talents without neglecting socio-emotional needs.

METHODOLOGY

This article adopts a theoretical and exploratory approach, based on an integrative review of the existing scientific literature on the neurobiology, cognition and development of giftedness and Autism Spectrum Disorder (ASD). The methodology involved the critical analysis of empirical studies, review articles and theoretical models relevant to understanding the relationship between these two conditions.

The bibliographic search was carried out in databases such as PubMed, Scopus, Web of Science and Google Scholar, using key terms such as "giftedness", "autism", "neurobiology", "neurodevelopment", "prefrontal cortex", "mesolimbic system", "functional connectivity" and their English counterparts. Articles published in peer-reviewed scientific journals, books and book chapters that presented data relevant to the proposed hypothesis were selected.

The analysis of the selected articles focused on identifying the neurobiological evidence that characterizes giftedness and ASD, the theoretical models that seek to explain the relationship between them and the implications for assessment and intervention. The information collected was synthesized and organized to support the hypothesis of a neurodevelopmental continuum underlying both conditions.

DISCUSSION

The central hypothesis of this article posits that giftedness and ASD are not dichotomous clinical entities, but rather distinct manifestations along a continuous neurodivergent spectrum. This perspective is supported by the growing convergence of neurobiological evidence pointing to a shared neuroevolutionary origin, where subtle variations in brain development, particularly regarding the timing of maturation of key regions such as the mesolimbic system and the dorsolateral prefrontal cortex (DLPFC), modulate phenotypic expression. The integrated analysis of neuroanatomical data reveals that although both groups may show an initial increase in synaptic connectivity, the subsequent trajectory differs significantly. In giftedness, this connectivity evolves towards greater functional integration between distributed neural networks, facilitating complex and flexible cognitive processes. In contrast, in ASD, there is a tendency towards local hyperconnectivity, often associated with atypical sensory processing and the development of specific talents, but with challenges in integrating information on a more global level.

Neurochemical processes also offer crucial insights into understanding these spectral dynamics. Dopamine, a fundamental neurotransmitter in the reward system and in the modulation of synaptic plasticity, shows distinct activity patterns in the two groups. In ASD, dopaminergic hyperactivity in the nucleus accumbens may contribute to restricted interests and repetitive behaviors, while in gifted individuals, a more efficient modulation of dopamine in the DLPFC seems to support enhanced executive functions and the ability to switch between different cognitive tasks. Similarly, the balance of glutamate, the main excitatory neurotransmitter, appears to be fundamental. While in ASD the higher density of NMDA receptors may be related to local hyperconnectivity, in giftedness, a more precise glutamatergic homeostasis in the prefrontal cortex may favor adaptive synaptic plasticity, essential for learning and solving complex problems.

In the molecular field, genome wide association studies (GWAS) reveal a complex interaction of genetic factors. The identification of genetic polymorphisms that overlap between giftedness and high-functioning subgroups of ASD, such as variants in genes involved in neuroplasticity and neuronal differentiation, reinforces the notion of a common biological substrate. However, the presence of other genetic variants more specific to ASD, often associated with broader functional deficits, suggests that the complex interaction between genetic predisposition and compensatory mechanisms plays a crucial role in determining the final phenotype. In addition, the influence of epigenetic factors, such as DNA methylation, in modulating the expression of these genes throughout development, underlines the importance of considering the temporal and contextual dynamics in the manifestation of these conditions.

The timing of brain maturation emerges as a critical factor in distinguishing between the developmental trajectories of giftedness and ASD. Early maturation of the mesolimbic system in ASD may explain the intensity of specific interests and the search for predictability, while relatively later maturation of the dorsolateral prefrontal cortex may contribute to challenges in social cognition and executive functions. In contrast, giftedness is often associated with a robust and prolonged development of the DLPFC, which favors the integration of information between different brain networks and supports higher-order cognitive abilities, such as abstract reasoning and creativity. This temporal difference in the development of interconnected brain regions may be a fundamental determinant in the emergence of the distinct characteristics observed in the two groups.

The analysis of connectivity and neural plasticity offers a complementary perspective. The local hyperconnectivity observed in many brain regions in ASD, together with a possible reduction in synaptic pruning, may lead to intensified sensory processing and an excessive focus on details. On the other hand, giftedness seems to be associated with greater efficiency in long-distance connectivity, facilitating communication and integration between functionally distinct brain regions. Neural plasticity, the brain's ability to adapt and change in response to experience, also seems to operate differently. While in ASD there may be a more rigid or domain-specific plasticity, in giftedness there is a more adaptive and flexible plasticity, allowing for a greater capacity to learn and apply knowledge in different contexts.

The neurodivergent spectrum perspective challenges traditional diagnostic categorizations and proposes a more fluid and dimensional understanding of giftedness and ASD. Rather than being seen as mutually exclusive

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categories, they can be understood as points along a continuum, where individuals can display characteristics of both to varying degrees. This approach has significant implications for how giftedness is defined and identified. The emphasis on integrative ability, rather than relying solely on one-dimensional metrics such as IQ, offers a more holistic assessment of higher cognitive abilities. Assessment tools that explore executive functions, creativity and the ability to integrate information from different domains may be more effective in identifying gifted individuals, especially those who may also display characteristics of the autistic spectrum.

Adopting a neurodivergent spectrum perspective has the potential to transform intervention approaches for individuals with giftedness and ASD. By recognizing the unique needs and strengths of each individual along this continuum, it is possible to develop more personalized and inclusive educational and clinical strategies. For individuals with ASD who also have high intellectual abilities, interventions aimed at promoting social integration and cognitive flexibility can be combined with strategies that nurture their specific talents. Similarly, for gifted individuals who may have sensory sensitivities or social challenges, a supportive environment that recognizes and accommodates these needs can optimize their potential. Future research, including longitudinal neuroimaging studies and more detailed genomic analyses, will be key to further validating and refining this spectral model, paving the way for a more complete and compassionate understanding of human neurodiversity.

CONCLUSION

Giftedness and ASD share a neuroevolutionary origin, with phenotypic differences resulting from variations in the timing of brain maturation and neural connectivity. The mesolimbic system and the DLPFC play central roles, modulated by neurochemical processes (dopamine, glutamate, Ca^{2+}) and genetic polymorphisms (SHANK3, COMT, CNTNAP2). The redefinition of giftedness, with a focus on functional integration, offers a more precise approach to identification and support. Longitudinal neuroimaging studies and genomic analyses are needed to validate the proposed model.

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