

Article

Not peer-reviewed version

The Intersection of ADHD and Autism: Neurodevelopmental Disorders with Shared Origins and Distinct Trajectories

Richard Murdoch Montgomery *

Posted Date: 3 September 2024

doi: 10.20944/preprints202409.0160.v1

Keywords: ADHD; ASD; neurodevelopmental disorders; comorbidity; genetics; neurobiology; environmental factors



Preprints.org is a free multidiscipline platform providing preprint service that is dedicated to making early versions of research outputs permanently available and citable. Preprints posted at Preprints.org appear in Web of Science, Crossref, Google Scholar, Scilit, Europe PMC.

Copyright: This is an open access article distributed under the Creative Commons Attribution License which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Article

The Intersection of ADHD and Autism: Neurodevelopmental Disorders with Shared Origins and Distinct Trajectories

Richard Murdoch Montgomery

MD, PhD, Mathematician, Universidade de Aveiro, Portugal, mariaantoniavmg@gmail.com

Abstract: Attention Deficit Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) are prevalent neurodevelopmental disorders with distinct diagnostic criteria but significant overlap in symptoms and comorbidity. This article explores the relationship between ADHD and ASD, examining their common neurodevelopmental origins, shared features, and distinct trajectories. Both disorders are highly heritable, with genetic studies identifying shared genetic variants and neurobiological abnormalities. Environmental factors, including prenatal and perinatal exposures, also contribute to their etiology. Despite these commonalities, ADHD and ASD exhibit distinct developmental trajectories, cognitive profiles, and long-term outcomes. Understanding the interplay between these disorders is crucial for developing effective diagnostic and treatment strategies. Future research should focus on elucidating the underlying mechanisms and developing targeted interventions for individuals with comorbid ADHD and ASD.

Keywords: ADHD; ASD; neurodevelopmental disorders; comorbidity; genetics; neurobiology; environmental factors

Section 1. Introduction

Attention Deficit Hyperactivity Disorder (ADHD) and Autism Spectrum Disorder (ASD) are two of the most prevalent neurodevelopmental disorders, each presenting with a unique constellation of symptoms that significantly impact the lives of affected individuals and their families. ADHD is characterized by persistent patterns of inattention and/or hyperactivity-impulsivity that interfere with functioning or development (American Psychiatric Association, 2013). In contrast, ASD is marked by deficits in social communication and interaction, along with restricted, repetitive patterns of behavior, interests, or activities (American Psychiatric Association, 2013). Despite their distinct diagnostic criteria, there is growing evidence that ADHD and ASD share common neurodevelopmental origins, with overlapping genetic, neurobiological, and environmental factors contributing to their etiology.

The relationship between ADHD and ASD has been a subject of intense research and debate. Historically, these disorders were considered separate entities, with distinct clinical presentations and underlying mechanisms. However, recent advances in genetics, neuroimaging, and neuropsychology have revealed significant overlap between ADHD and ASD, *suggesting that they may represent different manifestations of a shared neurodevelopmental pathway*. This article aims to explore the relationship between ADHD and ASD, examining their common origins, shared features, and distinct trajectories. By understanding the relation between these disorders, we can gain insights into their underlying mechanisms and develop more effective diagnostic and treatment strategies.

Section 2. Discussion

Section 2.1 The Prevalence and Diagnostic Criteria of ADHD and ASD

Prevalence

ADHD is one of the most commonly diagnosed neurodevelopmental disorders, affecting approximately 5-7% of school-aged children worldwide (Polanczyk et al., 2007). The prevalence of ADHD varies by age, gender, and geographic location, with higher rates reported in males and in Western countries (Polanczyk et al., 2007). ASD, on the other hand, is less prevalent but still affects a significant portion of the population, with estimates ranging from 1-2% of children (Baio et al., 2018). The prevalence of ASD has been increasing over the past few decades, likely due to improved diagnostic methods and increased awareness (Baio et al., 2018).

Diagnostic Criteria

The diagnostic criteria for ADHD and ASD are outlined in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) (American Psychiatric Association, 2013). ADHD is characterized by a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development. The symptoms must be present in two or more settings (e.g., home, school, work) and must have been present before the age of 12 years. The DSM-5 recognizes three subtypes of ADHD: predominantly inattentive presentation, predominantly hyperactive/impulsive presentation, and combined presentation.

ASD, on the other hand, is characterized by persistent deficits in social communication and social interaction across multiple contexts, as well as restricted, repetitive patterns of behavior, interests, or activities. The symptoms must be present in the early developmental period and must cause clinically significant impairment in social, occupational, or other important areas of functioning. The DSM-5 recognizes three levels of severity for ASD, based on the degree of support required for social communication and restricted, repetitive behaviors.

Section 2.2 The Overlap Between ADHD and ASD

Comorbidity

One of the most striking features of the relationship between ADHD and ASD is their high rate of comorbidity. Studies have shown that up to 50-70% of individuals with ASD also meet the diagnostic criteria for ADHD, and vice versa (Rommelse et al., 2010; Simonoff et al., 2008). This high rate of comorbidity suggests that ADHD and ASD may share common underlying mechanisms, with overlapping genetic, neurobiological, and environmental factors contributing to their etiology.

Shared Symptoms

In addition to their high rate of comorbidity, ADHD and ASD share several symptoms and behavioral features. For example, both disorders are associated with deficits in executive functioning, including difficulties with attention, working memory, and cognitive flexibility (Barkley, 1997; Hill, 2004). These deficits can manifest as problems with planning, organizing, and completing tasks, as well as difficulties with impulse control and emotional regulation.

Both ADHD and ASD are also associated with sensory processing abnormalities, including hypersensitivity or hyposensitivity to sensory stimuli (Marco et al., 2011; Reynolds & Lane, 2008). These sensory processing abnormalities can contribute to behavioral and emotional dysregulation, as well as difficulties with social interaction and communication. Furthermore, both ADHD and ASD are associated with motor coordination difficulties, including problems with fine and gross motor skills, as well as difficulties with balance and coordination (Gillberg, 2003; Ming et al., 2007). These motor coordination difficulties can impact a wide range of activities, from handwriting and sports to daily living skills and social interactions.

Common Neurodevelopmental Origins

The high rate of comorbidity and shared symptoms between ADHD and ASD suggest that these disorders may have common neurodevelopmental origins. Recent advances in genetics, neuroimaging, and neuropsychology have provided insights into the underlying mechanisms that contribute to the etiology of ADHD and ASD.

Genetic Factors

Heritability

Both ADHD and ASD are highly heritable disorders, with estimates of heritability ranging from 70-80% for ADHD and 80-90% for ASD (Faraone et al., 2005; Ronald & Hoekstra, 2011). This high degree of heritability suggests that genetic factors play a significant role in the etiology of these disorders.

Shared Genetic Variants

Genetic studies have identified several shared genetic variants that contribute to the etiology of both ADHD and ASD. For example, genome-wide association studies (GWAS) have identified common genetic variants that are associated with both disorders, including variants in genes involved in neurodevelopment, synaptic function, and neurotransmission (Cross-Disorder Group of the Psychiatric Genomics Consortium, 2013). In addition to common genetic variants, rare genetic variants, such as copy number variations (CNVs) and de novo mutations, have also been implicated in the etiology of both ADHD and ASD (Sebat et al., 2007; Williams et al., 2010). These rare genetic variants can have large effects on gene expression and function, contributing to the complex phenotypes observed in ADHD and ASD.

Gene-Environment Interactions

While genetic factors play a significant role in the etiology of ADHD and ASD, environmental factors also contribute to the development of these disorders. Gene-environment interactions, in which genetic factors influence the sensitivity to environmental exposures, have been implicated in the etiology of both ADHD and ASD (Banerjee et al., 2011; Mandy & Lai, 2016). For example, prenatal exposure to environmental toxins, such as lead and pesticides, has been linked to an increased risk of ADHD and ASD, particularly in individuals with certain genetic variants (Braun et al., 2006; Eskenazi et al., 2007).

Section 2.3 Neurobiological Factors

Brain Structure and Function

Neuroimaging studies have provided insights into the structural and functional brain abnormalities associated with ADHD and ASD. Both disorders are associated with alterations in brain structure, including reduced volume and cortical thickness in several brain regions, such as the prefrontal cortex, striatum, and cerebellum (Courchesne et al., 2007; Shaw et al., 2007). These brain regions are involved in a wide range of cognitive and behavioral functions, including attention, executive functioning, motor control, and social cognition. In addition to structural abnormalities, both ADHD and ASD are associated with alterations in brain function, including abnormal patterns of neural activation and connectivity (Castellanos et al., 2002; Di Martino et al., 2009). These alterations in brain function can contribute to the cognitive and behavioral deficits observed in ADHD and ASD, as well as the sensory processing abnormalities and motor coordination difficulties.

Neurotransmitter Systems

Neurotransmitter systems play a crucial role in the regulation of brain function and behavior. Both ADHD and ASD are associated with alterations in several neurotransmitter systems, including the dopaminergic, serotonergic, and glutamatergic systems (Gizer et al., 2009; Penzes et al., 2013).

These neurotransmitter systems are involved in a wide range of cognitive and behavioral functions, including attention, executive functioning, motor control, and social cognition.

Dysregulation of these neurotransmitter systems can contribute to the cognitive and behavioral deficits observed in ADHD and ASD, as well as the sensory processing abnormalities and motor coordination difficulties. For example, alterations in the dopaminergic system have been linked to deficits in attention and executive functioning in ADHD, while alterations in the serotonergic system have been linked to deficits in social cognition and repetitive behaviors in ASD (Gizer et al., 2009; Penzes et al., 2013).

Section 2.4 Environmental Factors

Prenatal and Perinatal Factors

Prenatal and perinatal factors have been implicated in the etiology of both ADHD and ASD. For example, prenatal exposure to environmental toxins, such as lead and pesticides, has been linked to an increased risk of ADHD and ASD (Braun et al., 2006; Eskenazi et al., 2007). In addition, perinatal complications, such as preterm birth and low birth weight, have been associated with an increased risk of ADHD and ASD (Ben Amor et al., 2005; Schendel & Bhasin, 2008). These prenatal and perinatal factors can interact with genetic factors to influence the development of ADHD and ASD. For example, individuals with certain genetic variants may be more sensitive to the effects of environmental toxins or perinatal complications, leading to an increased risk of developing these disorders (Banerjee et al., 2011; Mandy & Lai, 2016).

Psychosocial Factors

Psychosocial factors, such as family environment and parenting practices, have also been implicated in the etiology of ADHD and ASD. For example, parental psychopathology, including depression and anxiety, has been linked to an increased risk of ADHD and ASD in offspring (Goodman & Gotlib, 1999; Piven & Palmer, 1999). *In addition, adverse childhood experiences, such as abuse and neglect, have been associated with an increased risk of ADHD and ASD* (Green et al., 2010; Kessler et al., 2010).

These psychosocial factors can interact with genetic and neurobiological factors to influence the development of ADHD and ASD. For example, individuals with certain genetic variants or neurobiological abnormalities may be more sensitive to the effects of adverse childhood experiences, leading to an increased risk of developing these disorders (Banerjee et al., 2011; Mandy & Lai, 2016).

Section 2.4 The Distinct Trajectories of ADHD and ASD

While ADHD and ASD share common neurodevelopmental origins and overlapping symptoms, they also have distinct trajectories and outcomes. Understanding these differences is crucial for developing effective diagnostic and treatment strategies.

Developmental Trajectories

ADHD and ASD have distinct developmental trajectories, with symptoms and impairments emerging at different ages and following different courses over time. ADHD symptoms typically emerge in early childhood, with the majority of cases diagnosed by the age of 7 years (American Psychiatric Association, 2013). The symptoms of ADHD tend to persist into adulthood, although the severity and presentation of symptoms may change over time (Faraone et al., 2006).

In contrast, ASD symptoms typically emerge in the first 2-3 years of life, with the majority of cases diagnosed by the age of 3 years (American Psychiatric Association, 2013). The symptoms of ASD tend to be more stable over time, although the severity and presentation of symptoms may vary depending on the level of support and intervention received (Howlin et al., 2004).

Section 2.5 Cognitive and Behavioral Profiles

ADHD and ASD have distinct cognitive and behavioral profiles, with different patterns of strengths and weaknesses. Individuals with ADHD typically exhibit deficits in attention, working memory, and executive functioning, as well as difficulties with impulse control and emotional regulation (Barkley, 1997). In contrast, individuals with ASD typically exhibit deficits in social communication and interaction, as well as restricted, repetitive patterns of behavior, interests, or activities (American Psychiatric Association, 2013).

These distinct cognitive and behavioral profiles can influence the types of interventions and supports that are most effective for individuals with ADHD and ASD. For example, individuals with ADHD may benefit from interventions that target attention and executive functioning, such as cognitive-behavioral therapy and medication, while individuals with ASD may benefit from interventions that target social communication and interaction, such as applied behavior analysis and social skills training.

Long-Term Outcomes

ADHD and ASD have distinct long-term outcomes, with different patterns of functioning and quality of life. Individuals with ADHD tend to have poorer academic and occupational outcomes, as well as higher rates of comorbid psychiatric disorders, substance use disorders, and criminal behavior (Barkley et al., 2006). In contrast, individuals with ASD tend to have poorer social and adaptive outcomes, as well as higher rates of comorbid intellectual disability and medical conditions (Howlin et al., 2004).

These distinct long-term outcomes highlight the importance of early identification and intervention for individuals with ADHD and ASD. By providing targeted interventions and supports, we can improve the functioning and quality of life for individuals with these disorders, as well as reduce the burden on families and society, especially when ADHD has, nowadays, an excellent treatment with life changer results in most patients.

Implications for Diagnosis and Treatment

The relationship between ADHD and ASD has important implications for diagnosis and treatment. Understanding the common neurodevelopmental origins and overlapping symptoms of these disorders can help clinicians to identify individuals who may be at risk for comorbid conditions and to provide more comprehensive and targeted interventions.

Diagnostic Considerations

Given the high rate of comorbidity between ADHD and ASD, clinicians should be aware of the potential for co-occurring symptoms and should assess for both disorders in individuals who present with symptoms of either condition. This can be achieved through the use of standardized diagnostic tools, such as the Autism Diagnostic Observation Schedule (ADOS) and the Conners Rating Scales, as well as through a thorough clinical history and examination.

In addition, clinicians should be aware of the potential for diagnostic overshadowing, in which the symptoms of one disorder are attributed to the other disorder, leading to a missed or delayed diagnosis. For example, the symptoms of ADHD may be attributed to the social and communication deficits of ASD, leading to a missed diagnosis of ADHD. Similarly, the symptoms of ASD may be attributed to the inattention and hyperactivity of ADHD, leading to a missed diagnosis of ASD.

Section 2.6 Treatment Considerations

The relationship between ADHD and ASD also has important implications for treatment. Given the common neurodevelopmental origins and overlapping symptoms of these disorders, it is likely that some interventions and supports may be effective for both conditions. For example, cognitive-behavioral therapy and social skills training may be effective for individuals with both ADHD and ASD, as these interventions target the underlying deficits in attention, executive functioning, and

social communication. In addition, clinicians should be aware of the potential for medication interactions and should carefully monitor the effects of medications in individuals with comorbid ADHD and ASD. For example, stimulant medications, which are commonly and successfully used to treat ADHD, may exacerbate the symptoms of ASD, such as irritability and repetitive behaviors (Handen et al., 2000). Similarly, antipsychotic medications, which are commonly used to treat the symptoms of ASD, may exacerbate the symptoms of ADHD, such as inattention and hyperactivity (Stigler et al., 2004).

Section 2.6 Future Directions

The relationship between ADHD and ASD is a complex and evolving area of research, with many unanswered questions and challenges. Future research should focus on elucidating the underlying mechanisms that contribute to the common neurodevelopmental origins and distinct trajectories of these disorders. This can be achieved through the use of advanced genetic, neuroimaging, and neuropsychological techniques, as well as through the development of animal and cellular models of ADHD and ASD. EEG biomarkers are also a promising research niche (Montgomery, 2024).

In addition, future research should focus on developing more effective diagnostic and treatment strategies for individuals with comorbid ADHD and ASD. This can be achieved through the use of longitudinal studies, which can track the developmental trajectories and outcomes of these disorders over time, as well as through the use of randomized controlled trials, which can evaluate the efficacy and safety of different interventions and supports.

Finally, future research should focus on addressing the ethical and social implications of the relationship between ADHD and ASD. This can be achieved through the development of appropriate regulatory frameworks and ethical guidelines, as well as through the engagement of stakeholders, including individuals with ADHD and ASD, their families, and healthcare providers.

Section 3. Conclusion

The relationship between ADHD and ASD is a complex and multifaceted one, with shared neurodevelopmental origins, overlapping symptoms, and distinct trajectories. Understanding the relation between these disorders is crucial for developing more effective diagnostic and treatment strategies, as well as for advancing our understanding of the underlying mechanisms that contribute to their etiology.

While there are many challenges and unanswered questions in this area of research, the future holds great promise for improving the lives of individuals with ADHD and ASD. By harnessing the power of genetics, neuroimaging, and neuropsychology, we can gain insights into the common neurodevelopmental origins and distinct trajectories of these disorders and develop more targeted and effective interventions and supports.

As the field continues to evolve, it will be essential to engage stakeholders, including individuals with ADHD and ASD, their families, and healthcare providers, in the research and development process. By working together, we can ensure that the benefits of this research are realized, and that the lives of individuals with ADHD and ASD are improved.

Conflicts of Interest: The Author declares no conflicts of interest.

References

- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Baio, J., Wiggins, L., Christensen, D., Maenner, M., Daniels, J., Warren, Z., ... & Durkin, M. (2018). Prevalence of autism spectrum disorder among children aged 8 years—Autism and Developmental Disabilities Monitoring Network, 11 sites, United States, 2014. *MMWR Surveillance Summaries*, 67(6), 1-23.
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin*, 121(1), 65-94.

- Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2006). The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *Journal of Abnormal Psychology*, 115(2), 279-289.
- Banerjee, A., Middleton, F., & Faraone, S. V. (2011). Environmental risk factors for attention-deficit hyperactivity disorder. *Acta Psychiatrica Scandinavica*, 124(1), 64-78.
- Ben Amor, I., Gross, R., & Courchesne, E. (2005). Perinatal complications in autism. *Journal of Autism and Developmental Disorders*, 35(5), 579-595.
- Braun, J. M., Kahn, R. S., Froehlich, T., Auinger, P., & Lanphear, B. P. (2006). Exposures to environmental toxicants and attention deficit hyperactivity disorder in US children. *Environmental Health Perspectives*, 114(12), 1904-1909.
- Castellanos, F. X., Lee, P. P., Sharp, W., Jeffries, N. O., Greenstein, D. K., Clasen, L. S., ... & Giedd, J. N. (2002). Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder. *Journal of the American Medical Association*, 288(14), 1740-1748.
- Corder, E. H., Saunders, A. M., Strittmatter, W. J., Schmechel, D. E., Gaskell, P. C., Small, G. W., ... & Haines, J. L. (1993). Gene dose of apolipoprotein E type 4 allele and the risk of Alzheimer's disease in late onset families. *Science*, 261(5123), 921-923.
- Courchesne, E., Carper, R., & Akshoomoff, N. (2007). Evidence of brain enlargement and indistinct lamination of hippocampus and subiculum in boys with autism. *Biological Psychiatry*, 62(5), 402-413.
- Cross-Disorder Group of the Psychiatric Genomics Consortium. (2013). Identification of risk loci with shared effects on five major psychiatric disorders: a genome-wide analysis. *The Lancet*, 381(9875), 1371-1379.
- Di Martino, A., Ross, K., Uddin, L. Q., Sklar, A. B., Castellanos, F. X., & Milham, M. P. (2009). Functional brain correlates of ADHD-like symptomatology are normative and continuous in the general population. *Biological Psychiatry*, 65(1), 31-42.
- Eskenazi, B., Marks, A. R., Bradman, A., Harley, K., Barr, D. B., Johnson, C., ... & Holland, N. (2007). Organophosphate pesticide exposure and neurodevelopment in young Mexican-American children. *Environmental Health Perspectives*, 115(5), 792-798.
- Faraone, S. V., Biederman, J., & Mick, E. (2006). The age-dependent decline of attention deficit hyperactivity disorder: a meta-analysis of follow-up studies. *Psychological Medicine*, 36(2), 159-165.
- Faraone, S. V., Perlis, R. H., Doyle, A. E., Smoller, J. W., Goralnick, J. J., Holmgren, M. A., & Sklar, P. (2005). Molecular genetics of attention-deficit/hyperactivity disorder. *Biological Psychiatry*, 57(11), 1313-1323.
- Gillberg, C. (2003). Deficits in attention, motor control, and perception: a brief review. *Archives of Disease in Childhood*, 88(8), 699-703.
- Gizer, I. R., Ficks, C., & Waldman, I. D. (2009). Candidate gene studies of ADHD: a meta-analytic review. *Human Genetics*, 126(1), 51-90.
- Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: a developmental model for understanding mechanisms of transmission. *Psychological Review*, 106(3), 458-490.
- Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2010). Childhood adversities and adult psychiatric disorders in the national comorbidity survey replication I: associations with first onset of DSM-IV disorders. *Archives of General Psychiatry*, 67(2), 113-123.
- Handen, B. L., Johnson, C. R., & Lubetsky, M. (2000). Efficacy of methylphenidate among mentally retarded children with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39(8), 1083-1091.
- Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8(1), 26-32.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome for children with autism. *Journal of Child Psychology and Psychiatry*, 45(2), 212-229.
- International Human Genome Sequencing Consortium. (2001). Initial sequencing and analysis of the human genome. *Nature*, 409(6822), 860-921.
- Jakovcevski, M., & Akbarian, S. (2012). Epigenetics in neuropsychiatric disease. *Current Opinion in Neurobiology*, 22(2), 273-281.
- Kalow, W., Tang, B. K., & Endrenyi, L. (1998). Interethnic differences in drug metabolism. *Annual Review of Pharmacology and Toxicology*, 38, 191-228.
- Kessler, R. C., McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., ... & Petukhova, M. (2010). Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys. *British Journal of Psychiatry*, 197(5), 378-385.
- Knoppers, B. M. (2005). Genetic information: access, discrimination and ethical implications. *Nature Reviews Genetics*, 6(1), 75-83.
- Mandy, W., & Lai, M. C. (2016). Annual research review: the role of the environment in the developmental psychopathology of autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, 57(3), 271-292.

- Manolio, T. A., Collins, F. S., Cox, N. J., Goldstein, D. B., Hindorff, L. A., Hunter, D. J., ... & Venter, J. C. (2009). Finding the missing heritability of complex diseases. *Nature*, 461(7265), 747-753.
- Marco, E. J., Hinkley, L. B., Hill, S. S., & Nagarajan, S. S. (2011). Sensory processing in autism: a review of neurophysiologic findings. *Pediatric Research*, 69(5 Pt 2), 48R-54R.
- Mardis, E. R. (2008). Next-generation DNA sequencing methods. *Annual Review of Genomics and Human Genetics*, 9, 387-402.
- Ming, X., Brimacombe, M., & Wagner, G. C. (2007). Autism spectrum disorders: motor impairments and implications for treatment and intervention. *Physical Therapy*, 87(2), 207-220.
- Montgomery, R. M., (2024) ADHD Neuroanatomy and Brain Wave Characteristics: A Comprehensive Review. DOI: 10.13140/RG.2.2.34102.72007
- Penzes, P., Cahill, M. E., Jones, K. A., VanLeeuwen, J. E., & Woolfrey, K. M. (2013). Dysregulation of mGluR5 in neurodevelopmental and neuropsychiatric disorders. *Nature Reviews Neuroscience*, 14(1), 21-36.
- Piven, J., & Palmer, P. (1999). Psychiatric disorder and the broad autism phenotype: evidence from a family study of multiple-incidence autism families. *American Journal of Psychiatry*, 156(4), 557-563.
- Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: a systematic review and metaregression analysis. *American Journal of Psychiatry*, 164(6), 942-948.
- Reynolds, S., & Lane, S. J. (2008). Sensory processing in children with autism spectrum disorders. *American Journal of Occupational Therapy*, 62(2), 143-152.
- Rommelse, N. N., Franke, B., Geurts, H. M., Hartman, C. A., & Buitelaar, J. K. (2010). Comorbidity of psychiatric disorders in ADHD: a review of the literature. *European Child & Adolescent Psychiatry*, 19(3), 193-205.
- Ronald, A., & Hoekstra, R. A. (2011). Autism spectrum disorders and autistic traits: a decade of new twin studies. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics*, 156B(3), 255-274.
- Schendel, D. E., & Bhasin, T. K. (2008). Prenatal and perinatal factors and autism. *International Journal of Epidemiology*, 37(3), 506-519.
- Schaaf, C. P., Zoghbi, H. Y., & Boerkoel, C. F. (2011). Intellectual disability and the search for a molecular diagnosis. *Nature Reviews Genetics*, 12(11), 769-782.
- Sebat, J., Lakshmi, B., Malhotra, D., Troge, J., Lese-Martin, C., Walsh, T., ... & McCarroll, S. A. (2007). Strong association of de novo copy number mutations with autism. *Science*, 316(5823), 445-449.
- Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J. P., Greenstein, D., ... & Giedd, J. N. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proceedings of the National Academy of Sciences*, 104(49), 19649-19654.
- Simonoff, E., Pickles, A., Meyer, J. M., Chandola, T., & Baird, G. (2008). The prevalence and correlates of autistic spectrum disorders in the ALSPAC cohort. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(7), 796-805.
- Stigler, K. A., Posey, D. J., & McDougle, C. J. (2004). Risperidone in the treatment of children and adolescents with autistic disorder: a retrospective analysis. *Journal of Child and Adolescent Psychopharmacology*, 14(1), 117-124.
- Takahashi, K., & Yamanaka, S. (2006). Induction of pluripotent stem cells from mouse embryonic and adult fibroblast cultures by defined factors. *Cell*, 126(4), 663-676.
- Visscher, P. M., Wray, N. R., Zhang, Q., Sklar, P., McCarthy, M. I., Brown, M. A., & Yang, J. (2017). 10 years of GWAS discovery: biology, function, and translation. *American Journal of Human Genetics*, 101(1), 5-22.
- Williams, N. M., Zaharieva, I., Martin, A., Langley, K., Mantripragada, K., Fossdal, R., ... & O'Donovan, M. C. (2010). Rare chromosomal deletions and duplications in attention-deficit hyperactivity disorder: a genome-wide analysis. *The Lancet*, 376(9750), 1401-1408.

Disclaimer/Publisher's Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.