



# **Naturalistic Neuroimaging: From Film to Learning Disorders**

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ABSTRACT: Cognitive neuroscience explores neural functioning and aberrant brain activity during cognitive and perceptual tasks. Neurocinematics is a subfield of cognitive neuroscience that observes neural responses of individuals watching a film to see similarities and differences between individuals. This method is typically used for commercial use, allowing directors and filmmakers to produce better visuals and increase their results at the box office. However, neurocinematics is increasingly becoming a standard tool for neuroscientists interested in studying similar brain activity patterns across viewers outside the film industry. In this review, I argue that neurocinematics provides a straightforward, naturalistic approach to researching and diagnosing learning disorders. While the neural underpinnings of developmental learning disorders are traditionally assessed with well-established methods like EEG and fMRI that target particular cognitive domains, such as simple visual and attention tasks, there is initial evidence and theoretical background supporting neurocinematics as a biomarker for learning differences. By using ADHD, dyslexia, and autism as case studies, this literature review discusses the potential advantages of neurocinematics as a new tool for learning disorders research.

KEYWORDS: Behavioral and Social Sciences; Neuroscience; Neurocinematics; Biomarkers; Neurobehavioral disorders.

## Introduction

According to the Centers for Disease Control, 17% of children ages 3-17 in the US have a developmental disability or learning disorder.1 Children with a learning disorder are at higher risk of adversity, including familial neglect and poorer health outcomes in adulthood. Moreover, children with learning disorders experience disproportionate levels of family adversity compared to typically developing children. The three most commonly diagnosed learning disorders are attention-deficit hyperactivity disorder (ADHD), dyslexia, and autism spectrum disorder (hereafter referred to as autism). Individuals with ADHD show signs of being inattentive and/ or hyperactive-impulsive, while students with dyslexia demonstrate difficulty in talking, reading, writing, and spelling.<sup>18</sup> In contrast, children with autism exhibit impairment in communication, relationships, verbal skills, and restricted/repetitive behaviors.<sup>2</sup> These disorders are necessary to diagnose because they can help parents be proactive in finding the tools needed for their child's education and health and reduce the risk of adversity in their academic journey or social situations. Understanding a child's learning deficiencies will help parents and school faculty take the necessary interventions and provide the correct accommodations for growth.

Because learning disorders and neurological disparity can impact the adolescent brain and later behaviors, it is essential to identify learning disorders in children early in life.<sup>3</sup> One valuable method of diagnosing these disorders is by examining biomarkers. Biomarkers are a measurable characteristic of biological processes such as those relating to psychological activity.<sup>4</sup> Clinicians use behaviors and psychological indicators to diagnose learning disorders. Still, biomarkers add benefit by providing more objective criteria for diagnosis. Crucial biomarkers for learning disorders grounded in neurobiology have

previously been identified through electroencephalography (EEG) and functional magnetic resonance imaging (fMRI). EEG is a form of brain imaging that acquires brain signals called event-related potentials (ERPs) through electrodes attached to the scalp. 5 Signals from EEG can determine neurological engagement and functional interactions across neural networks, making the signals valuable biomarkers for the types of psychological processes implicated in learning disorders.<sup>6</sup> This method is also beneficial for diagnoses because it has a higher tolerance for motion artifacts, which can better accommodate testing with infants and children who tend to move more during the testing process. Indeed, combining behavioral measures and ERPs from EEG during a go/no-go impulsivity task enabled 84-97% accuracy at discriminating ADHD diagnostic status in two different cohorts of participants.8 Yet, high heterogeneity in learning disorders and the tendency to rely on a single or handful of EEG measures limits the use of EEG as a diagnostic tool for ADHD and other learning disorders. In comparison, fMRI indirectly measures brain activity by capturing changes in the hemodynamic response across the entire brain, which is the measurement of blood oxygenation. While fMRI is less portable than EEG, it gives a spatial resolution that may be important for distinguishing between brain activity in regions with high proximity but distinct functions.<sup>9</sup> For example, the go/no-go task has also been used to compare children with and without ADHD. It was shown that the pre-supplementary motor area, but not other frontal regions, was hypoactive compared to typically developing children.<sup>10</sup> Despite some promising success, similar issues limit the application of traditional fMRI and EEG as biomarkers of ADHD and other learning disorders. In particular, the use of highly controlled experiments with simple stimuli in neuroimaging

limits the ability to capture dynamic responses that better resemble brain activity during real-world situations.<sup>11</sup>

Neurocinematics is a new and promising method to diagnose learning disorders, sometimes referred to as naturalistic neuroimaging. Historically, neurocinematics has been used to assess the effects of film and other forms of visual entertainment on a viewer's brain activity to inform cinematic techniques for filmmakers. However, this field has recently been used to explore cognitive processing during dynamic situations reminiscent of real life. 11,12 One popular analysis method is intersubject correlation, which measures the similarities between participants' perceptual, emotional, and cognitive states during the same event. 13,14 In a landmark study by Hasson and colleagues conducted in 2004, five participants watched the first thirty-minute sequence of The Good the Bad and the Ugly directed by Sergio Leone while their brain activity was measured using fMRI. Regions of the brain that are shown to be associated with faces and objects were synchronized across participants, particularly during moments when faces and objects were in focus. 15 This study provided evidence that neurocinematics can be a powerful technique for uncovering properties of cognitive processing that are similar across individuals.

This review will evaluate neurocinematics as a new strategy for identifying neurological biomarkers to diagnose three common neurodevelopmental disorders: ADHD, dyslexia, and autism. This review discusses each neurobehavioral disorder in-depth and also seeks to showcase the various physiological biomarkers present for each, but more comprehensive reviews include. 7,16,17 Overall, the paper highlights a few key studies that use neuroimaging measures to examine these disorders and then discusses neurocinematics as a new and promising method for diagnosis.

# Section 1: Children with ADHD:

ADHD is a neurodevelopmental condition that affects 3-5% of children worldwide who typically exhibit symptoms as early as seven years old. However, the number of children with ADHD may be higher because the ability to diagnose and optimally manage the disorder is currently low in developing countries. 18 There is no cure for ADHD. Still, it can be managed through a multidisciplinary approach, including behavioral therapy, psychotherapy, and drug treatment. For instance, Neurotherapy or biofeedback has been seen to improve symptoms. Symptoms exhibited by individuals with ADHD include inattention, hyperactivity, and/or impulsivity. There are also symptoms associated with executive dysfunction leading to issues with initiation, time management, working memory, planning, and organizing. This disorder can be categorized into two subtypes: inattentive presentation and predominantly hyperactive-impulsive. Children who fall under the inattentive type demonstrate signs of distractibility, forgetfulness, difficulty in organizing and completing tasks, trouble listening when spoken to, and failing to remember to turn in asynchronous assignments. Individuals categorized as hyperactive-impulsive subtypes display squirming, talkativeness, and fidgeting, and they find it challenging to sit still. Along with this, they can also be impatient, demonstrate a lack of emotional restraint, and blurt random and, at times, inappropriate comments. <sup>18</sup> Clinical psychologists and psychiatrists diagnose ADHD primarily on psychological conditions based on a profile of symptoms associated with a reduction in the quality of social, academic, or occupational functioning. <sup>18</sup>

The causes of ADHD are unknown; however, hypothesized factors include genetics, diet, and social and environmental factors. Some onset symptoms can also occur from a close head injury (CHI). According to past research, 75% of cases of children with ADHD stem from their genetic background, specifically genes that affect dopamine transporters such as dopamine receptors D2 /D3 and dopamine beta-hydroxylase monoamine oxidase A. Dopamine can contribute based on its effects on learning and cognitive functioning. 19 However, no single gene has been shown to contribute to ADHD.<sup>18</sup> Another study demonstrates that 10% to 85% of cases of ADHD in childhood are potentially associated with comorbid psychopathology: mood and anxiety disorders.<sup>20</sup> The World Health Organization states that ADHD can also stem from social dysfunction that a child is exposed to, whether in their familial life or in the education system, which affects the child's self-regulatory abilities.21

Multiple biomarkers have been identified for ADHD to help with clinical diagnosis and treatment. EEG biomarkers use a combination of temporal, spectral, and spatial features for diagnosis.<sup>22</sup> Major EEG biomarkers include theta-beta waves ratio and Event-Related Potentials (ERP). However, previous studies have found difficulty in diagnosing ADHD with theta waves; a study conducted with 101 people by Ogrim and his team reported sensitivity of 63% and specificity of only 58% in differentiating between children with and without ADHD, in contrast with an accuracy of 85% based on classification by children's behavioral omission errors alone (as cited in 6). Nonetheless, theta waves relate to hyperarousal, one of the earliest symptoms associated with this neurobehavioral disorder.<sup>6</sup> ERPs are beneficial based on their ability to capture the temporal evolution of neural activity following a prescribed event (e.g., responses following selective attention or other executive function tasks), allowing for a more specific diagnosis. Although, they can be susceptible to high variance when relatively few trials are averaged, limiting the efficacy of ERP in predicting ADHD diagnosis in previous experimental studies. Overall, EEG is not an effective tool for diagnosis. Still, it remains useful in a clinical setting via multivariate analyses and refined studies of EEG signal generators to capture additional sources of heterogeneity in ADHD.<sup>6,22</sup> Another common biomarker is genetics because the dopamine transporter gene (DAT1 gene) has been linked to the development of ADHD, showing deficits in inhibitory behavior and hyperactivity. Dopamine is a modulator of learning and cognitive functioning.3 A European Child & Adolescent Psychiatry study recruited 273 high-risk Chinese individuals diagnosed with ADHD and their family members. The results demonstrate that the haplotype rs27048 is strongly associated with the inattentive type, concluding that the DAT1 gene may primarily affect individuals with ADHD.23

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The use of neurocinematics to identify new biomarkers for ADHD was recently tested in an experiment with 51 adults with ADHD (no subtypes were specified) and 29 individuals without ADHD.<sup>24</sup> Patients and controls watched a curated film showing a conversation with auditory distractors in the background. ADHD-related aberrant brain responses to this naturalistic stimulus were identified using ISC on subjects' fMRI data. The ADHD group demonstrated abnormal functioning in the dorsal and ventral attention networks, salience network, and sensory areas - namely, patients displayed less synchronous activity in these regions when distractors were present compared to control subjects.<sup>24</sup> By creating a novel experimental design using movies, the authors discovered abnormal neural processing during a demanding yet highly realistic situation. This anomalous ISC signature also correlated with behavioral measures of ADHD symptoms specific to inattention. This method has been used to detect abnormal functioning and signatures of ADHD, which provides further evidence of the potential efficacy of this method for diagnosing learning disorders.

Despite the promising results of this study, it is not clear how effective neurocinematics is versus past methods in diagnosing ADHD in children, as no direct comparison has been made.<sup>24</sup> Thus, more neurocinematics studies are needed to determine if this method can account for other symptoms associated with ADHD, such as skills in sustaining or dividing attention and the ability to regulate the level of attention on demand. To date, most neuroimaging biomarkers of ADHD focus on impulsivity, but there is no promising current biomarker that distinguishes between the two ADHD subtypes. Distinguishing between different symptom profiles and ADHD subtypes is essential for prescribing treatments for patients.

In addition to traditional EEG and fMRI approaches, neurocinematics may be a good way of distinguishing ADHD subtypes based on which parts of the brain respond synchronously to a given set of stimuli. For instance, children with hyperactive and non-hyperactive subtypes may react differently when viewing films. For example, children with the hyperactive subtype may require short, attention-grabbing films (i.e., a cartoon episode versus a nature documentary) to stimulate brain activity. This is because hyperactive children may struggle to focus on a film long enough to acquire necessary stimulus data, such as readouts from EEG. Alternatively, it may be that neurocinematics, in combination with identifying other biomarkers, such as genetics, may yield the best detection of ADHD. However, the perfect diagnostic tool remains unknown until ADHD is examined more with neurocinematics.

## Section 2: Children with Dyslexia:

Dyslexia is a learning disability characterized by difficulty in reading, word recognition, and spelling.<sup>25</sup> In one study, it was found that 80% of individuals diagnosed with learning disabilities have dyslexia.<sup>16</sup> Dyslexia is the most common neurobehavioral disorder affecting children.<sup>26</sup> While it is considered more prominent in girls, recent data has demonstrated that it occurs equally between both genders.<sup>25</sup> Students with dyslexia who have decreased capabilities in reading, causing a hindrance in academic performance, which leads to self-es-

teem issues and emotional and behavioral problems.<sup>25,27</sup> One study reported that children with reading difficulty at the ages of eight and twelve were unhappier, more anxious, and less competent scholastically, and their parents stated that their self-esteem issues were low.<sup>28</sup> There are two main strategies when prescribing treatment for dyslexia: assistance with the impaired learning areas (reading and spelling) and psychotherapy for any coexisting psychological disturbance that may be present.

The causes of this disorder are unknown; however, some research indicates that it is predominantly influenced by neurobiological and cognitive factors, as well as heritability. This is consistent with the statistic that 23% to 65% of all children with dyslexia have a parent with the same disorder. Host theories on dyslexia primarily point to problems in temporal processing and stem from research on the visual and language systems. Some investigators believe the central difficulty displayed in dyslexia patients is rooted in the phonetic system, which is engaged in processing sounds and speech. Hospital contents are unknown;

There have been several biomarkers to diagnose and prescribe treatment for dyslexia. Pernet and his team (2009) argue that the right cerebellum may be a biomarker for dyslexia.<sup>29</sup> In this study, they compared cognitive abilities and the size of different brain regions in subjects with dyslexia and controls without this neurobehavioral disorder. The subjects with dyslexia had low cerebellar declive volume and performed worse than those with a high cerebellar decline in the phonological and lexical tasks assigned. This evidence suggests that various subtypes of dyslexia may be characterized by different neurological phenotypes that correlate to varying deficiencies in language processing.<sup>29</sup> The best performances on the language tests were observed in control subjects, who showed a different profile of brain size in basal ganglia and right cerebellar declive. Overall, the study indicated that the basal ganglia, particularly the right cerebellar declive, are effective biomarkers for classifying individuals with dyslexia.

In another study, two children with dyslexia and two without engaged in a lexical task while undergoing fMRI.<sup>30</sup> The results indicated that the children with dyslexia displayed greater activation during reading tasks in the frontal and occipital cortex than typical readers. Other differences in activation between children with and without dyslexia were recorded in five brain regions: the right inferior parietal lobe, the right middle frontal gyrus, the left frontal precentral gyrus, the left insula, and the left fusiform gyrus. However, this study is hindered based on the small number of subjects examined, making the results less dependable. Yet other studies with larger samples have also shown differences in brain activity in similar regions. A study that recruited 18 individuals with dyslexia scanned them using an fMRI machine before and after they received instructional treatment.<sup>31</sup> The study examined the inferior frontal gyrus, middle frontal gyrus, the occipital region, and cerebellum. The results concluded that children with dyslexia had greater functional connectivity from the left inferior frontal gyrus to the right inferior frontal gyrus than the children without dyslexia, showing another difference in these regions as in the study above, increasing the validity of the results.<sup>31</sup>

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The current research on neural biomarkers of dyslexia is inconclusive, and neurocinematics may provide a new perspective helpful for diagnosing this disorder. In particular, it may be that more realistic scenarios in which children are reading in the context of achieving a goal, like understanding a homework assignment, may be important for understanding the aberrant neural processes involved in this disorder. For example, patients with dyslexia and typical readers could watch a film with a variety of texts, such as an old silent film, or even a Khan Academy-style (2021) video teaching on a topic appropriate to their age, such as history or language arts. While most neurocinematics studies use fMRI, temporal disruption is problematic for dyslexic subjects, meaning EEG might be a better technique to use in conjunction with neurocinematics than fMRI.

#### Section 3: Children with Autism:

Autism is a spectrum disorder meaning that a child with this neurobehavioral disorder can experience a wide range of symptoms varying from mild to severe. Children with autism can have difficulty with communication and social interaction. They also tend to exhibit repetitive behavior and have issues with adjustment or disturbances to their daily routine. Children with autism can also be sensitive to touch, specific smells, loud noises, extreme temperatures, and certain colors. Signs of autism can be seen as early as 12 months.

However, the criteria for diagnosis have changed several times over the last couple of decades, including the age of diagnosis. The mean age in 1990 was ten years and reduced to 5 years by 2002.<sup>33</sup> Also, the diagnostic criteria became broader over the years based on the wide range of symptoms one can have with autism since it is a spectrum disorder. For instance, one study examined 405 individuals between the ages of 10 and 53 diagnosed with an autism spectrum disorder.<sup>33</sup> Although 100% of the patients were diagnosed with autism before their adolescence, based on current criteria for diagnosis, only 54.8% would be classified as autistic.<sup>33</sup> For individuals to be diagnosed with this neurobehavioral disorder today, they must display impairments in social interaction and communication; restricted repetitive and stereotyped patterns of behaviors, interests, and activities; and delayed or abnormal functioning before age three years in social interaction, language, or symbolic or imaginative play.<sup>33</sup>

The severity of autism also varies between individuals. The manifestation of symptoms is based on the domain. For instance, adolescents improved more based on the reciprocal social interaction domain.<sup>33</sup> There is no cure for autism, but there are interventions that can be implemented to help reduce or relieve symptoms. For instance, a treatment known as applied behavioral analysis (ABA) is an educational behavioral intervention identified as an effective treatment to address learning deficiencies.<sup>34</sup>

Multiple factors can contribute to someone developing autism. There has been a current increase in autism cases in the United States, Europe, and Japan, a spike reported to stem at least partly from increased recognition directly, changed diagnostic criteria, and changed public attitudes. Most researchers believe that the cause of autism is rooted in genetics and family

factors. There is a concordance of autism in monozygotic twins, which is reported to be as high as 70%.<sup>35</sup> Currently, genetic factors are thought to account for 7-8% of autism cases, and this number is more reliable since this statistic is from a study in 2021.35 Despite the evidence tying genetic background to autism, there is no direct genetic information that thoroughly explains certain clinical and epidemiological aspects of autism, which raises questions on other potential influences of this spectrum disorder. There is also reason to believe that autism can be caused by environmental factors such as exposure to toxic chemicals, including lead and methylmercury, and other prenatally environmental components during embryonic and fetal life.35 However, in a case in Sweden of prenatal exposure to thalidomide, the percentage of exposed children later diagnosed with autism was only 4%. Another potential contributor is toxins or reactions to vaccines containing mercury.

Individuals with autism have a high rate of comorbidity with intellectual disability, about 45%, and many (between 29 and 47%) experience regression related to social behavior.<sup>36</sup> The rise in cases and severity of autism leads to the clinical need for the availability of objective biomarkers for use in the prognosis and diagnosis of these patients.<sup>36</sup> Methods used to identify more efficient biomarkers have included neuroimaging, gene testing, transcriptomics, proteomics, and metabolomics. Specifically, fMRI can be used to diagnose children at 6-12 months of age when autism behavior is first emerging and has a positive predictive value of 81%.36 However, based on previous studies, autistic symptoms shown during infancy can begin to dissipate over time. Another method used is EEG, demonstrating that the most significant differences appear at ages 9 to 12 months. Infants were classified with over 80% accuracy into control and high risk of autism (HRA) groups at age 9. This study indicates that EEG may increase efficacy in diagnosis early on.<sup>37</sup> The identification of biomarkers for autism spectrum disorder has improved over the years. However, due to the heterogeneity of the disease, the utility of these biomarkers still faces difficulties and challenges.

Identifying new biomarkers is essential to developing proper therapeutic treatments and interventions for autism, and neurocinematics may be a potential method. Because films have Spatio-temporal complexity, neurocinematics can allow for a deeper understanding of the different brain regions and symptoms of autism compared to previous studies examining subjects with learning differences doing lexical tasks. Additionally, films often show complex social relationships amongst characters that require the audience to think through social interactions and language. Recently, a group of researchers created child-friendly cartoon "films" designed to tap into the theory of mind (i.e., the ability to think about another person's thoughts) during neuroimaging.<sup>38</sup> Curated media is an effective biomarker of autism given its emphasis on cognitive processes that go awry in this neurodevelopmental disorder.

# **■** Discussion & Conclusion

This review explained three different neurobehavioral disorders (ADHD, dyslexia, and autism spectrum disorder) and how traditional biomarkers are used for diagnosis. This argumentative review helps illustrate how neurocinematics—a

rising field to help with cognitive neuroscience—can be utilized to identify better biomarkers for learning disorders based on the film's spatiotemporal complexity and ability to tap into more naturalistic cognitive contexts. This allows for examining brain functions that may not be present during typical tasks utilized in current diagnostic methods like fMRI and EEG. A common trend noticed throughout this paper is that although these traditional biomarkers have been somewhat effective with diagnosis, they contain limitations preventing a complete data analysis of one's neurological functioning, hindering our understanding of the causation of ADHD, dyslexia, and autism, and prescribing proper diagnosis and treatment. However, neurocinematics also presents limitations. There is a lack of clarity in using film to diagnose developmental disorders for early detection. For instance, 18 months or less early intervention is deemed critical for ensuring the best outcomes in autism. However, this age range may not be the appropriate target for neurocinematics that taps into more complex processes. Another limitation is that the studies examined in the paper discuss small sample sizes, which hinders the ability to identify biomarkers and understand the vast heterogeneity of these disorders. Although neurocinematics on its own may be limited, this field can be more effective in combination with other approaches. With the increasing interest in neurocinematics, it is now time to apply this method to answer some of the most critical questions about how children with neurodevelopmental disorders learn, with the aim of diagnosis and treatment.

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### References

- 1. CDC. Increase in developmental disabilities among children in the United States https://www.cdc.gov/ncbddd/developmental disabilities/features/increase-in-developmental-disabilities.html (accessed Oct 5, 2021).
- Shattuck, P. T.; Seltzer, M. M.; Greenberg, J. S.; Orsmond, G. I.; Bolt, D.; Kring, S.; Lounds, J.; Lord, C. Change in Autism Symptoms and Maladaptive Behaviors in Adolescents and Adults with an Autism Spectrum Disorder. *J Autism Dev Disord* 2007, 37 (9), 1735–1747. https://doi.org/10.1007/s10803-006-0307-7.
- 3. Berke, J. D. What Does Dopamine Mean? *Nat. Neurosci.* 2018, 21 (6), 787–793.
- Califf, R. M. Biomarker Definitions and Their Applications. *Exp. Biol. Med.* (Maywood) 2018, 243 (3), 213–221.
- Hajat, Z.; Ahmad, N.; Andrzejowski, J. The Role and Limitations of EEG-Based Depth of Anaesthesia Monitoring in Theatres and Intensive Care. Anesthesia 2017, 72 Suppl 1, 38–47.
- 6. Lenartowicz, A.; Loo, S. K. Use of EEG to Diagnose ADHD. Curr. *Psychiatry Rep.* 2014, 16 (11), 498.
- 7. Mehta, T.; Mannem, N.; Yarasi, N. K.; Bollu, P. C. Biomarkers for

- ADHD: The Present and Future Directions. *Curr. Dev. Disord. Rep.* 2020, 7 (3), 85–92.
- Häger, L. A.; Åsberg Johnels, J.; Kropotov, J. D.; Weidle, B.; Hollup, S.; Zehentbauer, P. G.; Gillberg, C.; Billstedt, E.; Ogrim, G. Biomarker Support for ADHD Diagnosis Based on Event Related Potentials and Scores from an Attention Test. *Psychiatry Res.* 2021, 300 (113879), 113879.
- O'Shaughnessy, E. S.; Berl, M. M.; Moore, E. N.; Gaillard, W. D. Pediatric Functional Magnetic Resonance Imaging (FMRI): Issues and Applications. J. Child Neurol. 2008, 23 (7), 791–801.
- 10. Suskauer, S. J.; Simmonds, D. J.; Fotedar, S.; Blankner, J. G.; Pekar, J. J.; Denckla, M. B.; Mostofsky, S. H. Functional Magnetic Resonance Imaging Evidence for Abnormalities in Response Selection in Attention Deficit Hyperactivity Disorder: Differences in Activation Associated with Response Inhibition but Not Habitual Motor Response. J. Cogn. Neurosci. 2008, 20 (3), 478– 493.
- Hasson, U.; Landesman, O.; Knappmeyer, B.; Vallines, I.; Rubin, N.; Heeger, D. J. Neurocinematics: The Neuroscience of Film. *Projections* 2008, 2 (1), 1–26.
- Sonkusare, S.; Breakspear, M.; Guo, C. Naturalistic Stimuli in Neuroscience: Critically Acclaimed. *Trends Cogn.* Sci. 2019, 23 (8), 699–714.
- 13. Nastase, S. A.; Gazzola, V.; Hasson, U.; Keysers, C. Measuring Shared Responses across Subjects Using Intersubject Correlation. *Soc. Cogn. Affect. Neurosci.* 2019, 14 (6), 667–685.
- 14. Finn, E. S.; Glerean, E.; Khojandi, A. Y.; Nielson, D.; Molfese, P. J.; Handwerker, D. A.; Bandettini, P. A. Idiosynchrony: From Shared Responses to Individual Differences during Naturalistic Neuroimaging. *Neuroimage* 2020, 215 (116828), 116828.
- Hasson, U.; Nir, Y.; Levy, I.; Fuhrmann, G.; Malach, R. Intersubject Synchronization of Cortical Activity during Natural Vision. Science 2004, 303 (5664), 1634–1640.
- 16. Shaywitz, S. E. Dyslexia. N. Engl. J. Med. 1998, 338 (5), 307–312.
- 17. Anderson, G. M. Autism Biomarkers: Challenges, Pitfalls and Possibilities. *J. Autism Dev. Disord.* 2015, 45 (4), 1103–1113.
- Frank-Briggs, A. Attention Deficit Hyperactivity Disorder (ADHD). J. Pediatr. Neurol. 2015, 09 (03), 291–298.
- 19. Cheon, K.-A.; Ryu, Y.-H.; Kim, J.-W.; Cho, D.-Y. The Homozygosity for 10-Repeat Allele at Dopamine Transporter Gene and Dopamine Transporter Density in Korean Children with Attention Deficit Hyperactivity Disorder: Relating to Treatment Response to Methylphenidate. *Eur. Neuropsychopharmacol.* 2005, 15 (1), 95–101
- 20. Millstein, R. B.; Wilens, T. E.; Biederman, J.; Spencer, T. J. Presenting ADHD Symptoms and Subtypes in Clinically Referred Adults with ADHD. J. *Atten. Disord.* 1997, 2 (3), 159–166.
- 21. Fayyad, J.; Sampson, N. A.; Hwang, I.; Adamowski, T.; Aguilar-Gaxiola, S.; Al-Hamzawi, A.; Andrade, L. H. S. G.; Borges, G.; de Girolamo, G.; Florescu, S.; Gureje, O.; Haro, J. M.; Hu, C.; Karam, E. G.; Lee, S.; Navarro-Mateu, F.; O'Neill, S.; Pennell, B.-E.; Piazza, M.; Posada-Villa, J. The Descriptive Epidemiology of DSM-IV Adult ADHD in the World Health Organization World Mental Health Surveys. ADHD Attention Deficit and Hyperactivity Disorders 2016, 9 (1), 47–65. https://doi.org/10.1007/s12402-016-0208-3..
- 22. Müller, A.; Vetsch, S.; Pershin, I.; Candrian, G.; Baschera, G.-M.; Kropotov, J. D.; Kasper, J.; Rehim, H. A.; Eich, D. EEG/ERP-Based Biomarker/Neuroalgorithms in Adults with ADHD: Development, Reliability, and Application in Clinical Practice. *World J. Biol. Psychiatry* 2020, 21 (3), 172–182.
- 23. Shang, C.-Y.; Gau, S. S.-F.; Liu, C.-M.; Hwu, H.-G. Association between the Dopamine Transporter Gene and the Inattentive Subtype of Attention Deficit Hyperactivity Disorder in Taiwan. *Prog-*

- ress in Neuro-Psychopharmacology and Biological Psychiatry 2011, 35 (2), 421–428. https://doi.org/10.1016/j.pnpbp.2010.08.016.
- 24. Salmi, J.; Metwaly, M.; Tohka, J.; Alho, K.; Leppämäki, S.; Tani, P.; Koski, A.; Vanderwal, T.; Laine, M. ADHD Desynchronizes Brain Activity during Watching a Distracted Multi-Talker Conversation. *Neuroimage* 2020, 216 (116352), 116352.
- Novita, S. Secondary Symptoms of Dyslexia: A Comparison of Self-Esteem and Anxiety Profiles of Children with and without Dyslexia. Eur. J. Spec. Needs Educ. 2016, 31 (2), 279–288.
- Schulte-Körne, G. The Prevention, Diagnosis, and Treatment of Dyslexia. *Dtsch Arztebl Int.* 2010, 107 (41), 718-27. https://doi. org/10.3238/arztebl.2010.0718.
- 27. McNulty, M. A. Dyslexia and the Life Course. *J. Learn. Disabil.* 2003, 36 (4), 363–381.
- Casey, R.; Levy, S. E.; Brown, K.; Brooks-Gunn, J. Impaired Emotional Health in Children with Mild Reading Disability. J Dev Behav Pediatr 1992, 13 (4), 256–260.
- 29. Pernet, C. R.; Poline, J. B.; Demonet, J. F.; Rousselet, G. A. Brain Classification Reveals the Right Cerebellum as the Best Biomarker of Dyslexia. *BMC Neurosci.* 2009, 10 (1), 67.
- Berman, S.; Cicchino, N.; Hajinazarian, A.; Mescher, M.; Holland, S. K.; Horowitz-Kraus, T. An FMRI Study of a Dyslexia Biomarker. *Journal of Young Investigators* 2014, 26 (1).
- Richards, T. L.; Berninger, V. W. Abnormal FMRI Connectivity in Children with Dyslexia during a Phoneme Task: Before but Not after Treatment. J. Neurolinguistics 2008, 21 (4), 294–304.
- 32. Dietz, P. M.; Rose, C. E.; McArthur, D.; Maenner, M. National and State Estimates of Adults with Autism Spectrum Disorder. *J. Autism Dev. Disord.* 2020, 50 (12), 4258–4266.
- 33. Seltzer, M. M.; Krauss, M. W.; Shattuck, P. T.; Orsmond, G.; Swe, A.; Lord, C. The Symptoms of Autism Spectrum Disorders in Adolescence and Adulthood. *J. Autism Dev. Disord.* 2003, 33 (6), 565–581.
- Vismara, L. A.; Rogers, S. J. Behavioral Treatments in Autism Spectrum Disorder: What Do We Know? *Annu. Rev. Clin. Psychol.* 2010, 6 (1), 447–468.
- 35. Landrigan, P. J. What Causes Autism? Exploring the Environmental *Contribution. Current Opinion in Pediatrics* 2010, 22 (2), 219–225. https://doi.org/10.1097/MOP.0b013e328336eb9a.
- Shen, L.; Zhao, Y.; Zhang, H.; Feng, C.; Gao, Y.; Zhao, D.; Xia, S.; Hong, Q.; Iqbal, J.; Liu, X. K.; Yao, F. Advances in Biomarker Studies in Autism Spectrum Disorders. *Adv. Exp. Med. Biol.* 2019, 1118, 207–233.
- Bosl, W.; Tierney, A.; Tager-Flusberg, H.; Nelson, C. EEG Complexity as a Biomarker for Autism Spectrum Disorder Risk. *BMC Med.* 2011, 9 (1). https://doi.org/10.1186/1741-7015-9-18.
- 38. Borbás, R.; Fehlbaum, L. V.; Rudin, U.; Stadler, C.; Raschle, N. M. Neural Correlates of Theory of Mind in Children and Adults Using CAToon: Introducing an Open-Source Child-Friendly Neuroimaging Task. *Developmental Cognitive Neuroscience* 2021, 49, 100959.

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Asha Dukkipati — I struggled with ADD and dyslexia during my adolescence while my brother battled Asperger's. So experimenting with alternative treatments such as neurofeedback became a way of life. My love of neuroscience and film grew from seeking solutions and documenting the therapies for spreading awareness. This evolved into discovering the interdisciplinary field of neurocinematics and wanting to research further into this developing science to treat neurodiverse individuals with learning differences.

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