

The Journal of the IYNA

FEATURED ARTICLES

'Impact of Multitasking on Memory Formation in Teenagers: A Cognitive Neuroscience Perspective'
- May Mourad

'Short-Form Videos Significantly Degrade Long-Term Memory Performance Across a Diverse Age: Implications of Memory Performance in Adolescents and Adults'
- Jolin Cheng

'The Efficacy of Music Therapy for Children and Adolescents with Special Needs'
- Orly Galatin

August 2025
VOL. 9 ISSUE 5

Contents

INTRODUCTION

Letter from the Editors IYNA Editorial Team Page 4

GENERAL NEUROSCIENCE

Addiction's Effects on the Prefrontal Cortex Garv Pattani and Joshua Kim Pages 5-9

This paper explores addiction as a brain disease that alters prefrontal cortex function, disrupts neurotransmitter balance, and follows a three-stage cycle of bingeing, withdrawal, and anticipation. While various therapies aim to restore cognitive control and emotional regulation, addiction's neurological complexity makes treatment and recovery a persistent challenge.

The Efficacy of Music Therapy for Children and Adolescents with Special Needs Orly Galatin Pages 10-16

This paper evaluates the impact of music therapy on children and adolescents with special needs, highlighting improvements in communication, emotional regulation, and social interaction. The author evaluates how research shows that caregiver reports suggest generally positive outcomes, though the effectiveness may vary depending on factors like session structure and delivery method.

The Neurological Foundations of Stress and the Impact of Chronic Stress on the Brain Ashmi Parikh Pages 17-22

The author examines how stress, while evolutionarily designed as a survival mechanism, has become a constant presence in modern human life due to complex thought processes. Unlike animals, humans can create stress through anticipation and reflection, leading to chronic activation of the body's stress systems. The article explores how this chronic stress, regulated by the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic adrenal medullary (SAM) system, can cause hormonal imbalances and structural brain changes like dendrite shrinkage. It also highlights strategies for managing stress and protecting brain health.

Short-Form Videos Significantly Degrade Long-Term Memory Performance Across a Diverse Age: Implications of Memory Performance in Adolescents and Adults Jolin Cheng Pages 23-31

This paper investigates the impact of short-form videos on memory and finds that they significantly degrade long-term memory performance across age groups. Using a within-subjects design, the author identifies research highlighting how exposure to such content may interfere with cognitive function, particularly memory retention.

Impact of Multitasking on Memory Formation in Teenagers: A Cognitive Neuroscience Perspective	May Mourad	Pages 32-35
---	------------	-------------

This paper examines how multitasking, especially common among teenagers in the digital age, negatively affects memory formation from a cognitive neuroscience perspective. Research shows that divided attention impairs brain regions responsible for memory and attention, leading to reduced recall, lower academic performance, and potential long-term cognitive consequences.

Is it Possible to Fully Explain Consciousness?	Alexa Marsh	Pages 36-40
--	-------------	-------------

This article explores whether human consciousness can be fully explained by examining philosophical, neurological, and psychological perspectives. By analyzing key debates, such as monism vs. dualism and the “hard problem” of consciousness, the author contrasts these approaches and proposes a theoretical framework to address the question.

DISEASES AND DISORDERS

The Fight Against ALS: The Role of Artificial Intelligence in Early Diagnosis and Drug Discovery	Angela Li	Pages 41-45
--	-----------	-------------

The author explores how Artificial Intelligence (AI) offers new hope in combating Amyotrophic Lateral Sclerosis (ALS), a fatal neurodegenerative disease that causes progressive muscle paralysis while leaving cognitive function intact. Though there is currently no cure, recent advancements in AI show promise in enabling earlier diagnosis, more accurate detection, and faster drug development to help manage or potentially treat the disease.

Huntington’s Disease: Innovative Computational Diagnosis Methods	Kaushik Tatiraju	Pages 46-49
--	------------------	-------------

This paper explores how emerging artificial intelligence and machine learning technologies are improving the diagnosis of Huntington’s Disease, a genetic neurological disorder often misdiagnosed or undiagnosed. By reviewing recent innovations and computational methods, including tools like Neuralink, the study highlights the growing potential of AI to enhance accuracy and efficiency in HD prognosis and detection.

The Protective Role of REELIN in Alzheimer's Disease: Exploring New Genetic Insights	Arihaan Mallick	Pages 50-53
--	-----------------	----------------

The author highlights recent discoveries linking the REELIN protein to resilience against Alzheimer's disease (AD). While AD is marked by the buildup of amyloid plaques and tau tangles that impair memory and learning, new research reveals that a rare genetic mutation (REELIN-COLBOS) enhances REELIN's neuroprotective functions. This mutation appears to reduce the harmful effects of tau pathology, suggesting that REELIN may play a crucial role in protecting the brain and could offer promising new directions for AD treatment.

Alzheimer's Memory Decline and Daily Activities	Hafsa Ali	Pages 54-59
---	-----------	----------------

This study investigates the relationship between daily activities and memory decline in individuals with Alzheimer's disease using longitudinal data from the Alzheimer's Disease Neuroimaging Initiative. Findings suggest that while time is a significant factor in cognitive decline, difficulties in daily living have a minimal and non-significant impact on memory over time.

CONTRIBUTORS PAGES pages 60-61

· INTRODUCTION ·

Letter From the Editors

Journal Leadership

Dear Readers,

Welcome to the fifth issue of the 9th volume of the IYNA Journal! We greatly appreciate your readership, continued or new. We have worked hard at producing more high-quality articles for everyone to read and encouraging a growing number of high school students from around the world to submit their neuroscience findings, research, and/or interviews to the journal. We've hand-picked a special few to showcase in this month's journal.

We have been receiving many wonderful articles from you guys. It is clear how much the journal is improving as we review each article submission. We would just like to thank everyone who has submitted articles to this issue and prior issues alike. Without your dedication and hardwork, we would not be able to spread the word about the amazing diversity in subject matter that neuroscience, and neuroethics specifically, has to offer. With that being said, here are some previews of the essays published this month:

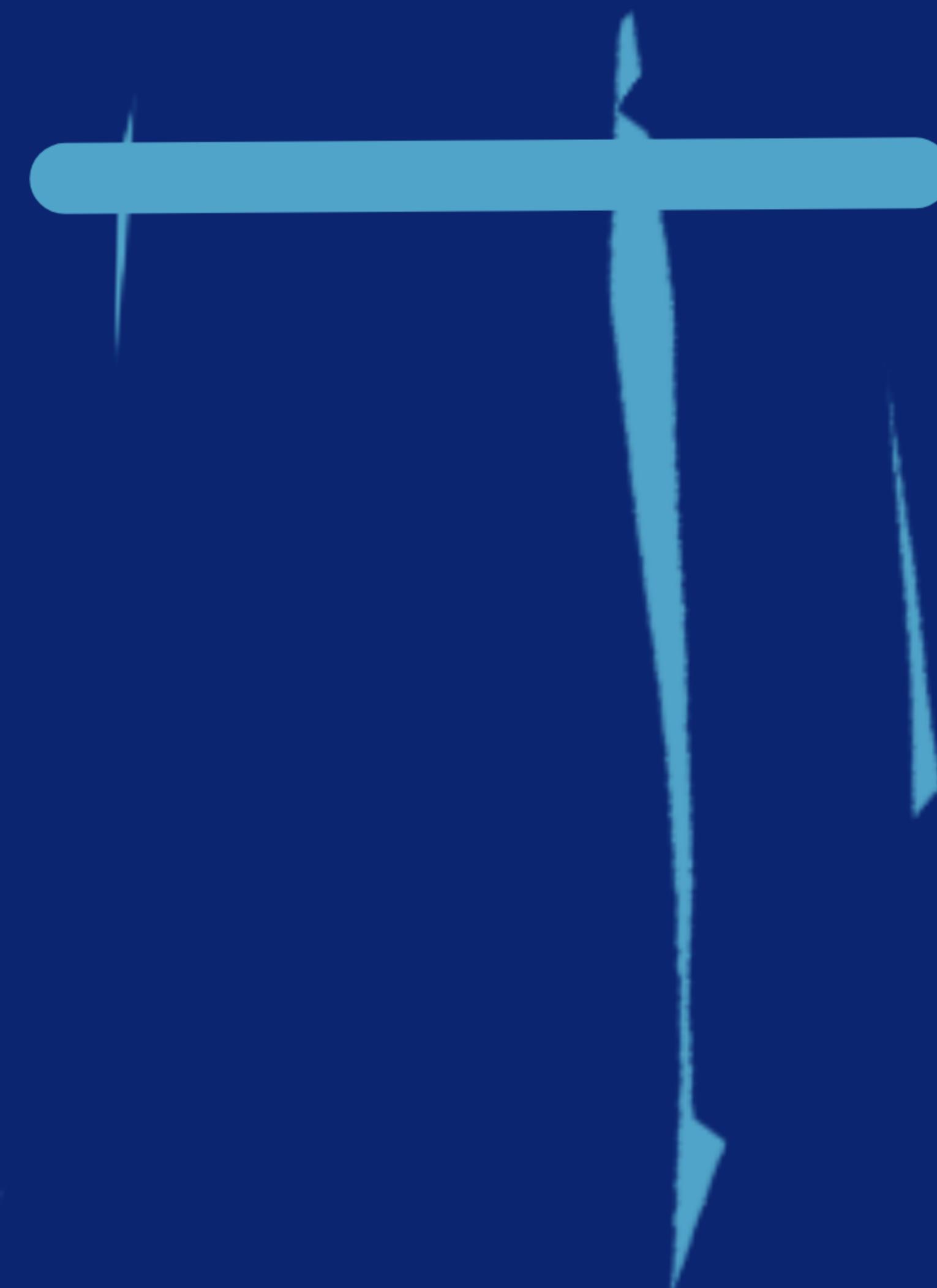
May Mourad explores the impact of multitasking on memory formation in teenagers, Jolin Cheng examines how short-form videos significantly degrade long-term memory performance, and Orly Galatin sheds light on the efficacy of music therapy for children and adolescents with special needs.

We would like to recognize all of our dedicated editors for helping us make this issue the success that it is. You can see all of their names and positions on our Contributors page. If you have any questions, comments, or suggestions for us, please email apan@youthneuro.org. We hope you enjoy reading this issue as much as we enjoyed editing it!

Best Regards,

Annie Pan - IYNA Journal Editor-In-Chief
Ashvin Kumar - Managing Editor
Riyaa Sri Ramanathan - Head of Assembly
Aleksandra Dubno - Head of Outreach
Ananyaa Karthikeyan - Head of Events
Ana Beatriz Araujo - Head of Translation
Shrika Vejandla - Head of Journalists

GENERAL NEUROSCIENCE



Addiction's Effects on the Prefrontal Cortex

Garv Pattani and Joshua Kim

Abstract

Addiction is a brain disease that induces irregular prefrontal cortex function, imbalances in neurotransmitters and hormones, as well as damage to other parts of the body and brain. Addiction generally presents itself in a three-stage cycle: Bingeing, withdrawal, and anticipation. Addicted individuals typically tend to show increased D₂ receptor binding potential, indicative of a much higher tolerance to dopamine. They also show increased mu receptor binding potential. Experiments have also yielded that addicted individuals show increased BOLD responses to drug-related cues. Although addiction can be hard to overcome, there are many treatments and therapies that help patients recover. There are 3 main types of treatments: Cognitive-behavioral therapy, Dialectical-behavioral therapy, and Mindfulness-based practices. These methods have been proven to help the prefrontal cortex regain its ability to control impulses, regulate emotion, and make decisions.

Process of Addiction

Addiction is a prevalent and well-documented condition in which an individual is physically or mentally dependent on a substance or drug. It is well-supported that addiction is a chronic brain disease that can require long periods of recovery treatment. It is characterized by disrupted prefrontal cortex function, as it disrupts dopamine (pleasure hormone and neurotransmitter) flow and increases glutamate activity in the brain. Glutamate is an abundant excitatory neurotransmitter and plays important roles in memory and learning. Substance abuse habits and cravings have been attributed to this increased glutamate activity. Addiction typically involves a cycle of three steps: bingeing, withdrawal, and anticipation. This cycle generally becomes more severe as an individual gets more and more addicted to a certain substance. This impairs many cognitive, behavioral, and response functions of the PFC, and it becomes difficult for the individual to control their substance use.

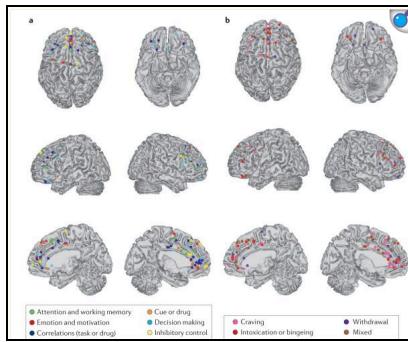


Figure 1. Recent neuroimaging studies of PFC activity in drug-addicted individuals.

The first stage of addiction, the bingeing stage, begins when a person first uses the drug and experiences a high. It affects the basal ganglia, which is responsible for controlling the rewarding effect of the drug, as well as motor control. The drug causes it to produce a large dopamine flood, leading to a high. It can also impact the basal ganglia so that it induces habitual substance taking. It creates strong associations between the surrounding environment and the pleasurable sensations experienced.”

The next stage of this process, withdrawal, happens when an individual comes off drugs and the reward system in the basal ganglia shuts off. This said individual then starts to go through an empty, depressive state. This activates the stress system of the brain, the amygdala, and causes the individuals to feel an urge to return to the binging stage, to bring relief from their current state of depression.

The last stage, anticipation, is activated as an addict starts to crave drugs. An addict starts to desire drugs after a period of abstinence, which can vary in length in proportion to the severity of the addiction. The prefrontal cortex is impacted during this stage, and it activates the brain’s “go system” to cause the individual to seek out drugs in order to return once more to the binging stage.

Effects of Addiction on the Prefrontal Cortex

Addiction can change the functioning of the prefrontal cortex as it gets more severe and can make it even more challenging to break free from it. This can also cause negative effects on the functions associated with the PFC, such as emotions, cognition, behavior, etc.

Dopamine D₂ receptors are found throughout the PFC but are most densely concentrated in subcortical regions such as the dorsal and ventral striatum, as well as the midbrain. According to a journal posted on Nat Rev Neurosci, a Positron Emission Tomography (also called PET, which works by injecting radioactive glucose into a vein and tracking where it is used in the brain with a machine) was carried out and has reported lower dopamine receptor availability in addicted

individuals. High concentrations of dopamine caused by drugs can damage and reduce the sensitivity and number of the brain's dopamine D₂ receptors, which only worsens the addiction. Part of the behavioral impacts caused by addiction can be attributed to this lack of dopamine signaling. To further support this, other PET results showed that the dopamine release in the brain attenuated in correspondence to the administering of a drug, along with the feeling of being high.

Along with dopamine, endogenous opioids also regulate the rewarding feeling of drugs. Endogenous opioids have roles in neurotransmission, and, similar to dopamine, can cause euphoria under certain conditions (although they also have many other, more practical uses like pain moderation). Also like dopamine, it was shown to be released at decreased levels along with drug use. A study (carried out by the National Institute on Drug Abuse) that used carfentanil has yielded that addicted individuals had much higher levels of mu receptor binding potential in their anterior frontal cortex and ACC (Anterior Cingulate Cortex). This lower level of endogenous opioids also results in a plethora of withdrawal symptoms commonly associated with addiction and can be somewhat alleviated with the use of methadone (a substitute drug used to treat addictions). In fact, elevated receptor binding in the ACC and DLPFC (Dorsolateral Prefrontal Cortex) has been suggested to be a better predictor of treatment outcome than base drug use.

Then imbalance of hormones and neurotransmitters caused by addiction can disrupt the function of the PFC, and can also induce iRISA syndrome (impaired response inhibition and salience attribution), which can cause irritability and unusual prioritizing of drugs.

Experiments

The majority of the brain studies related to addiction are taken from the midbrain dopamine areas (substantia nigra and ventral tegmental area) and the basal ganglia structures, which is where they project (ventral striatum and dorsal striatum). These structures are known to be heavily involved in habits and reward systems.

Many fMRI (Functional Magnetic Resonance Imaging, a study that shows the activity in various areas of the brain using a magnet) studies show that stronger BOLD responses (blood-oxygen-level dependent responses, indicative of brain activity) due to drug-related cues are present in the prefrontal cortex of addicted individuals than controls. However, it should be noted that there were some exceptions to this. More specifically, these results were indicated in the left DLPFC, the medial frontal gyrus, and the right subcallosal gyrus in cigarette-addicted individuals, and the bilateral DLPFC and ACC in alcoholics. In addition, in a study held at the University of Heidelberg, Germany, it was found that occasional smokers made more efforts to earn things like money and other desirable objects than cigarettes, as opposed to dependent smokers, who made equal efforts to win both.

Furthermore, many addicted individuals showed stunted striatal dopaminergic function, which is theorized to be the cause behind an addict's unusual prioritization of the drug. According to research done by the National Institute of Alcohol Abuse and Alcoholism (and other

organizations), the OFC (orbitofrontal cortex) also appears to be a crucial modulator of the value reinforcers; disruption in this area may also contribute to the drug reward in addicts. Consistent with this, metabolism in the medial OFC and ventral ACC in addicted individuals increased after the administration of the drugs.

Cures and Recovery for Addiction

Although addiction can be hard to escape, there are many treatments and therapies to remedy it. These treatments help individuals break free of addiction, and regain control of their lives. There are three main methods that assist patients in achieving lasting recovery: Cognitive-Behavioral Therapy, Dialectical Behavioral Therapy, and Mindfulness-Based Practices.

The first treatment, Cognitive-Behavioral Therapy (CBT), is one of the most effective therapies for recovering from addiction. Numerous research and tests have proven that CBT leads to significant improvement and positive change. This therapy aims to help individuals recognize and change their unhealthy thoughts or behaviors. It focuses on working through a person's current life and finding solutions. CBT helps the prefrontal cortex restore its ability to manage impulses and emotions by focusing on these aspects.

Another productive remedy, Dialectical-Behavioral Therapy (DBT), is a common therapy used to help individuals break free from addiction. This is a treatment program for people with mental health conditions that involve difficulty regulating emotion, like addiction. The treatment process usually involves individual therapy sessions and group training. Through DBT, patients learn to accept themselves and their feelings. This approach is similar to CBT in that it helps patients identify solutions to eliminate their problematic behaviors. These skills are crucial for the prefrontal cortex to regain its function.

This leads to the last method, Mindfulness-Based Practices (MBP). This practice can be performed individually at home, or with a group during a mindfulness session. Mindfulness is a type of meditation, where individuals need to focus on the present moment, without any judgment from others. Spending lots of time working, playing sports, or having negative thoughts can cause people to experience stress, anxiety, and depression. Mindfulness helps them stay calm, and not focus on the past. Some examples of mindfulness practices involve exercises like meditation and yoga. Positive mindfulness has proven to increase self-control, improve emotional regulation, reduce stress, and boost mood for many individuals.

References

[1] The Prefrontal Cortex: A Key Player in Addiction - Haven Detox Little Rock. (10/02/2023). The Haven Detox. <https://arkansasrecovery.com/the-prefrontal-cortex-a-key-player-in-addiction/>. Retrieved: 11/10/2024

[2] Three Stages Of Addiction. Resurgence Behavioral Health. <https://resurgencebehavioralhealth.com/3-stages-of-addiction/>

[6] PET scan: MedlinePlus Medical Encyclopedia. (2015). MedlinePlus. <https://medlineplus.gov/ency/article/003827.htm> Retrieved: 11/11/2024

[7] The Raleigh House (12/31/2020). The Prefrontal Cortex and Addiction. <https://www.theraleighhouse.com/addiction-blog/pref>

Retrieved: 11/12/2024

[3] Goldstein, R. Z., & Volkow, N. D. (2011). Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. *Nature Reviews Neuroscience*, 12(11), 652–669. <https://doi.org/10.1038/nrn3119> Retrieved: 11/10/2024.

[4] Cleveland Clinic. (05/27/2023). Functional MRI – Seeing Brain Activity as it Happens. Cleveland Clinic. <https://my.clevelandclinic.org/health/diagnostics/25034-functional-mri-fmri>. Retrieved: 11/08/2024.

[5] Young, C. B., Sonne, J., & Reddy, V. (07/24/2023). Neuroanatomy, Basal Ganglia. Nih.gov; StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK537141/> Retrieved: 11/10/2024

rontal-cortex-and-addiction. Retrieved: 11/15/2024.

[8] Hillman, Elizabeth M. C. (2014). Coupling Mechanism and Significance of the BOLD Signal: A Status Report. *Annual Review of Neuroscience*, 37(1), 161–181. <https://doi.org/10.1146/annurev-neuro-071013-014111>. Retrieved: 11/15/2024.

[9] Volkow, Nora D. (2005). Activation of Orbital and Medial Prefrontal Cortex by Methylphenidate in Cocaine-Addicted Subjects But Not in Controls: Relevance to Addiction. *Journal of Neuroscience*, 25(15), 3932–3939. <https://doi.org/10.1523/JNEUROSCI.0433-05.2005>. Retrieved: 11/13/2024

[10] de Grecq, M., Supady, A., Thiemann, R., Tempelmann, C., Bogerts, B., Forchner, L., Ploetz, K. v., & Northoff, G. (2009). Decreased neural activity in reward circuitry during personal reference in abstinent alcoholics-A fMRI study. *Human Brain Mapping*, 30(5), 1691–1704. <https://doi.org/10.1002/hbm.20634>(DD/MM/YYYY). Retrieved: 11/14/2024.

The Efficacy of Music Therapy for Children and Adolescents with Special Needs

Orly Galatin

Abstract

This study examines the effects of music therapy on children and adolescents with special needs, with emphasis on communication, social interaction, emotional expression, relaxation, physical coordination, and overall well-being. Data collected from caregivers of youth participating in music therapy sessions indicate predominantly positive effects, particularly in communication skills, emotional regulation, and social engagement. While some participants experienced minimal change, the majority reported increased interest in music, improved coordination, and heightened emotional expression. These findings suggest that music therapy is a valuable intervention for children and adolescents with special needs, though factors such as session structure and modality (e.g., in-person vs. virtual) may influence its effectiveness.

Introduction

Music has long been recognized as a powerful tool for human expression, social connection, and emotional regulation. In recent years, its role in therapeutic interventions, particularly for children and adolescents with special needs, has gained increasing attention [1-4]. Childhood and adolescence are crucial periods for psychological, social, and cognitive development, and research suggests that music can play a significant role in shaping these aspects. Music therapy, which involves structured musical activities to promote well-being, has been used to support youth with special needs by enhancing communication skills, fostering social interactions, and regulating emotions. However, while many studies highlight its benefits, there remains a need for further empirical research to understand its full impact on neurobiological and psychological development.

Neuroscientific research has demonstrated that music engages multiple brain regions, including those responsible for emotion processing, memory, and motor coordination [5]. Music has profound effects on neuroplasticity, particularly in those whose brains are still developing. Engaging with music activates the limbic system, which governs emotions, and stimulates the prefrontal

cortex, which plays a key role in decision-making and self-regulation. These findings suggest that music therapy can be particularly beneficial for children and adolescents with special needs, who often face challenges in emotional regulation and cognitive flexibility.

Moreover, research has shown that rhythm-based interventions can enhance motor coordination and auditory processing, both of which are crucial for individuals with neurodevelopmental disorders [6]. The structured nature of music therapy provides a predictable and engaging environment that can support cognitive and social skills development. The ability of music to synchronize neural activity may explain why youth with special needs respond positively to rhythmic patterns and melodic structures.

The connection between music and youth mental health is well-documented. One influential study explored how music can facilitate emotional expression, strengthen social bonds, and foster creativity [4]. Children and adolescents frequently use music as a means of coping with stress and articulating emotions that may be difficult to express through words. Studies suggest that participation in group musical activities, such as choir or drumming circles, can enhance self-esteem and social belonging. However, excessive exposure to music with negative or distressing themes can also contribute to heightened emotional distress. This underscores the importance of structured, therapeutic interventions that harness music's positive effects while mitigating potential risks.

The present study aims to answer the following questions:

1. How does music therapy impact communication, social interaction, and emotional expression in children and adolescents with special needs?
2. What role does music therapy play in physical coordination, relaxation, and stress reduction?
3. How do caregivers perceive the overall effectiveness of music therapy in their child's development?

Methodology

This study collected data from caregivers whose children participated in music therapy. The sample included children with disabilities such as autism, Down syndrome, and cerebral palsy. A survey based on the Impact Areas Questionnaire (IAQ) [7] containing Likert items and free-response questions was created (see Appendix). Key areas assessed included communication skills, social interaction, emotional expression, relaxation/stress reduction, physical coordination/movement, and overall experience. Caregivers were also asked to describe any behavioral changes observed outside therapy and provide feedback on session effectiveness.

The anonymous survey was distributed to parents at multiple consecutive music therapy sessions. A visual-response-scale questionnaire for the children themselves was considered, but program staff predicted a very low completion rate if using this modality.

Responses to Likert items were tabulated and free-response answers were collected to form a narrative summary. Statistical analyses were performed using Google Sheets.

Results

There were a total of 18 respondents out of a class of 32 individuals, representing a response rate of 56%. Ages of participating children ranged 4-19 years, with varying lengths of participation in music therapy ranging from less than a month to over six months.

Observations of impact in the queried domains are summarized in Figure 1.

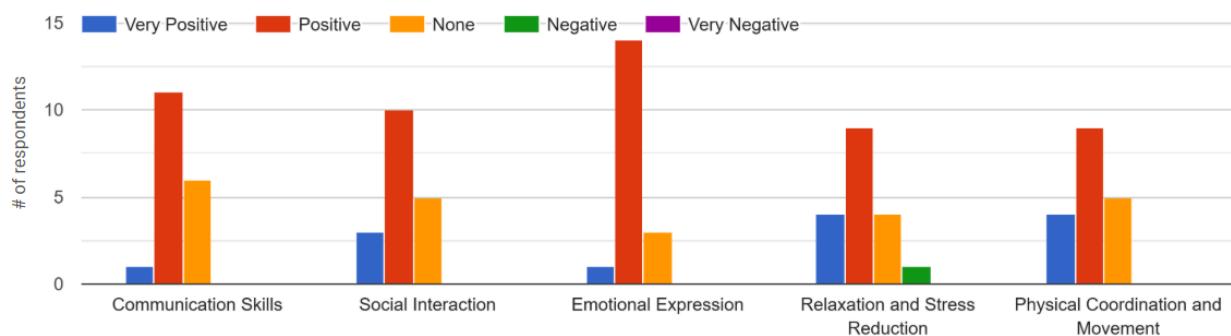


Figure 1. Changes in selected areas since beginning music therapy.

The majority of caregivers (over 70%) reported improvements in communication skills, with some noting that their children became more expressive or engaged in interactive singing. Many participants observed improvements in social interactions, with children becoming more engaged in group activities and responding better to others. One parent highlighted that music therapy provided a structured way to increase social interaction for a child with autism.

Emotional expression showed the strongest positive trend, with children demonstrating increased joy, smiles, and expressiveness. One parent noted that their child had become more emotionally expressive, smiling more and engaging in verbal interactions. In fact, one participant who is non-verbal used his digital tablet to type a song request for the first time.

Most respondents noted a calming effect, though a few saw no significant change. One negative response cited difficulties with virtual therapy sessions, suggesting that the format may have influenced the outcome. Several participants noted improved coordination, particularly with drumming and movement-based activities. One respondent specifically mentioned improved bilateral coordination and midline crossing skills.

Many caregivers provided additional qualitative insights, including:

- Children developing a love for music, dancing, and singing.

-
- Increased ability to self-regulate emotions through music.
 - The importance of structured and flexible sessions to accommodate individual needs.

Overall, caregivers expressed strong satisfaction with music therapy, very positive/positive ratings in 89% of respondents. Free responses cited increased interest in music, dancing, and self-expression. One parent emphasized that their child initially struggled to listen to music but, through therapy, learned to tolerate and engage with it. Strikingly, 94% of caregivers would recommend music therapy to other families of children with special needs. Additionally, 67% reported an increased interest or enjoyment in other musical activities outside therapy (e.g., singing, dancing, or playing an instrument).

Discussion

While past research has extensively explored the neurological and psychological benefits of music, fewer studies have specifically examined its efficacy in structured therapy sessions for children and adolescents with special needs. This study aimed to address that gap by collecting firsthand data from caregivers who have observed their children's responses to music therapy. Our survey assesses changes in communication, social interaction, emotional expression, relaxation, and physical coordination following participation in music therapy.

The findings align with previous research indicating that music therapy enhances communication, emotional regulation, and social engagement in youth with special needs. The high number of positive responses suggests that music therapy provides meaningful benefits, particularly in fostering self-expression and coordination. These results support prior studies indicating that structured musical engagement can be an effective intervention for adolescents with developmental and cognitive challenges. Future research should continue to explore the long-term effects of music therapy and identify specific techniques that maximize its benefits.

Although this study provides valuable insights into the efficacy of music therapy in this population, several limitations must be acknowledged. First, the data relies on caregiver-reported observations, which, while insightful, are inherently subjective and may be influenced by personal biases or expectations. Second, the study does not account for variability in music therapy approaches. Different therapists use distinct techniques, ranging from active participation (e.g., playing instruments, singing) to passive listening, which may yield varying outcomes. Additionally, the duration and frequency of music therapy sessions varied among participants, making it challenging to determine the optimal length of treatment required for significant improvements. Lastly, external factors such as home musical exposure, co-occurring therapies, and individual differences in cognitive and sensory processing were not controlled in this study. Despite these limitations, this study contributes to the growing body of research highlighting the potential of music therapy as a meaningful intervention for young people with special needs.

Conclusion

Music therapy appears to be a valuable tool for youth with special needs, offering benefits in multiple domains of mind-body functioning. While individual experiences vary, the overall caregiver-reported impact is positive, reinforcing the importance of integrating music-based interventions into therapeutic and educational programs. The role of emerging technologies such as more pervasive/on-demand interventions, particularly utilizing artificial intelligence models based on functional MRI response data [8], may be especially helpful for non-verbal individuals, thereby expanding equitable access to treatment in vulnerable populations.

Appendix

Music Therapy Feedback Survey

Thank you for taking the time to complete this anonymous survey. Your feedback is invaluable in helping us better understand how music therapy impacts children with special needs. All responses will be used to evaluate the effectiveness of music therapy and contribute to research in this field.

1. What is your child's age? —

2. How long has your child been participating in music therapy sessions?

- Less than 1 month
- 1-3 months
- 4-6 months
- More than 6 months

3. Has your child experienced any changes in the following areas since starting music therapy?

	Very Positive	Positive	None	Negative	Very Negative
1. Communications Skills	<input type="radio"/>				
2. Social Interaction	<input type="radio"/>				
3. Emotional Expression	<input type="radio"/>				
4. Relaxation and Stress Reduction	<input type="radio"/>				
5. Physical Coordination and Movement	<input type="radio"/>				

4. How would you rate your child's overall experience with music therapy sessions?

- Very Positive
- Positive
- Neutral
- Negative
- Very Negative

5. Have you noticed any additional changes in your child's behavior outside of therapy that you believe are connected to music therapy?

6. Does your child show increased interest or enjoyment in other musical activities outside therapy (e.g., singing, dancing, playing an instrument)?

- Yes
- No

7. Would you recommend music therapy to other families of children with special needs?

- Yes
- No

8. Is there anything else you'd like to share about your child's experience with music therapy or its impact on their development?

Thank you for your answers and feedback!

References

[1] Porter, Sam et al. (27/10/2016). Music therapy for children and adolescents with behavioural and emotional problems: a randomised controlled trial. *Journal of Child Psychology and Psychiatry*, 58(5), 586-594. <https://doi.org/10.1111/jcpp.12656>. Retrieved: 27/04/2025.

[2] Mayer-Benarous, Hanna et al. (08/04/2021). Music therapy for children with autistic spectrum disorder and/or other neurodevelopmental disorders: a systematic review. *Frontiers in Psychiatry*, 12. <https://doi.org/10.3389/fpsyg.2021.643234>. Retrieved: 27/04/2025.

-
- [3] Amirah, Shakira et al. (31/08/2023). Music therapy improves social interaction and verbal communication skill among children with autism spectrum disorder: A systematic review and meta-analysis. *Narra X*, 1(2).
<https://doi.org/10.52225/narrax.vii2.qo>. Retrieved: 27/04/2025.
- [4] Chen, Li. (02/08/2023). Influence of music on the hearing and mental health of adolescents and countermeasures. *Frontiers in Neuroscience*, 17. <https://doi.org/10.3389/fnins.2023.1236638>. Retrieved: 27/04/2025.
- [5] Sharma, Samata and David Silbersweig. (01/06/2018). Setting the stage: Neurobiological effects of music on the brain. <https://remix.berklee.edu/mh-exchange-music-medicine/6>. Retrieved: 27/04/2025.
- [6] Ding, Xiaofen et al. (26/09/2024). The benefit of rhythm-based interventions for individuals with autism spectrum disorder: a systematic review and meta-analysis with random controlled trials. *Frontiers in Psychiatry*, 15. <https://doi.org/10.3389/fpsyg.2024.1436170>. Retrieved: 27/04/2025.
- [7] Tsiris, Giorgos et al. (16/06/2020). The Impact Areas Questionnaire (IAQ): a music therapy service evaluation tool. *Voices: A World Forum for Music Therapy*, 20(3). <https://doi.org/10.15845/voices.v20i2.2816>. Retrieved: 27/04/2025.
- [8] Jiao, Dian. (24/02/2025). Advancing personalized digital therapeutics: integrating music therapy, brainwave entrainment methods, and AI-driven biofeedback. *Frontiers in Digital Health*, 25. <https://doi.org/10.3389/fdgh.2025.1552306>. Retrieved: 27/04/2025.

The Neurological Foundations of Stress and the Impact of Chronic Stress on the Brain

Ashmi Parikh

Abstract

In a world of looming deadlines, assessments, and financial troubles, stress has become an inevitable part of human life. Stress has been displayed throughout the animal kingdom as a survival mechanism for life or death situations. However, humans have the incredible ability of complex thought which leads to stressors that don't always involve life or death situations. Instead humans can generate their own stressors from thoughts of the past and future. This leads to imbalances in the body's natural stress response, causing things such as glucocorticoid imbalances and dendrite shrinkage in the brain. This article analyses the root processes of acute and chronic stress in the brain through the hypothalamic–pituitary–adrenal axis and the sympathetic adrenal medullary system. It discusses the long term effects of chronic stress on the brain along with healthy ways to manage stress.

What is Stress

Stress is the body's natural response to challenges or changes [4]. Dr Monteith, an associate professor of clinical neurology at the University of Miami Miller School of Medicine, defines stress as, “a protective mechanism. We all need stress; it signals to us that we may be in an uncomfortable or undesirable situation, and should take steps to remove ourselves from that situation.[4]” Stress is found throughout the animal kingdom and isn't always negative, however it can cause adverse effects in the body when the stress response is activated for a prolonged period of time [8]. Unlike most animals, humans have creativity and imagination which allows for complex thought [8]. This has been a positive characteristic for almost all aspects of human society, however, complex thought gives humans the unique ability to create their own stressors [8]. Humans can generate stressors by ruminating on past events or worrying about future events [8]. These stressors are perceived by the

body as a life-threatening situation, so the stress response is continuously activated leading to chronic stress [8].

Classifications of Stress

There are several different types of stressors based on their duration and nature [1]. The first two categories are acute and chronic stress [1]. Acute stress is the fight or flight response, where the body prepares for a short term challenge [1]. On the other hand, chronic stress is stress that continues for a prolonged period of time, this can lead to long term problems [1]. Another way to classify stressors is based on their nature or quality, this includes eustress and distress [1]. Eustress is stress that is associated with positive experiences, such as making friends or getting married [1]. Distress is stress that is associated with negative experiences, such as financial issues or divorce [1].

The Body's Stress Response

The body's stress response has evolved to help us react to life-threatening situations quickly and effectively [2]. The first step in the body's stress response is detecting the stressor, this is done through the five senses [2]. At the slightest indication that something is wrong, signals are sent to the amygdala to be interpreted [2]. The amygdala is the area of the brain responsible for processing emotions that are crucial to survival, this includes fear, anger, anxiety and more [2]. If the situation is deemed dangerous, distress signals are sent to the hypothalamus, which plays an important role in regulating hormones [2].

Initially, the autonomic nervous system is activated, particularly the sympathetic nervous system [2]. The sympathetic nervous system triggers the body's fight or flight response through the SAM (sympatho-adrenomedullary) system which prompts the adrenal glands to release epinephrine and norepinephrine [2]. This causes immediate changes in the body including faster heart rate, higher blood pressure, rapid breathing, dilated pupils and more [2].

After the first wave of epinephrine, the hypothalamus activates the HPA (Hypothalamus Pituitary Adrenal) Axis [2]. The HPA axis works to prolong the sympathetic nervous system's effects [2]. Through this process, the adrenal glands release cortisol [2]. During chronic stress, there is prolonged activation of the HPA axis which can lead to many negative effects.

The Sympatho-adrenomedullary System and Catecholamines

The Sympatho-adrenomedullary system, also known as the SAM system is the body's acute stress response [ii]. Better known as the fight or flight response, this system helps the body respond to immediate stressors [ii]. It starts off with the activation of the sympathetic nervous system after the amygdala has recognized the presence of a threat [ii]. Then, through autonomic nerves, a distress signal is sent to the adrenal glands [ii]. The adrenal glands are triangular shaped glands situated on top of the kidneys [ii]. The signal is sent specifically to the adrenal medulla, which is the inner part of the adrenal glands [ii]. The adrenal medulla will then release catecholamines such as epinephrine (adrenaline) and norepinephrine (noradrenaline) [ii].

Catecholamines are hormones that can also act as neurotransmitters, this includes dopamine, epinephrine, and norepinephrine [12]. During the SAM system, the adrenal medulla releases both epinephrine and norepinephrine [12]. This causes rapid changes in the body that prepares it for fight or flight [11]. The heart beats faster and stronger to provide ample blood supply to all necessary organs, breathing becomes faster and deeper to ensure the body has enough oxygen, the pupils dilate and focus intensifies [11]. These changes are usually short lived as epinephrine and norepinephrine levels are brought back to normal, which is why the SAM system is used for acute stressors [11].

The Hypothalamic–Pituitary–Adrenal Axis and Glucocorticoids

The Hypothalamic-pituitary-adrenal axis, also known as the HPA axis, works to prolong the SAM system's effects [9]. Starting out with the distress signal reaching the hypothalamus, the SAM system is activated immediately [9]. This distress signal also prompts the activation of the HPA axis (see figure 1) [9]. Starting with the hypothalamus, which releases Corticotropin-releasing hormone (CRH) [9]. The CRH travels to the pituitary gland, which is a small gland located at the base of the brain [9]. As it detects the CRH from the hypothalamus, the pituitary gland releases adrenocorticotrophic hormone (ACTH) [9]. This hormone travels through the bloodstream to the adrenal glands, specifically the adrenal cortex [9]. The adrenal cortex is the outermost layer of the adrenal glands, and ACTH prompts it to release the glucocorticoids [9].

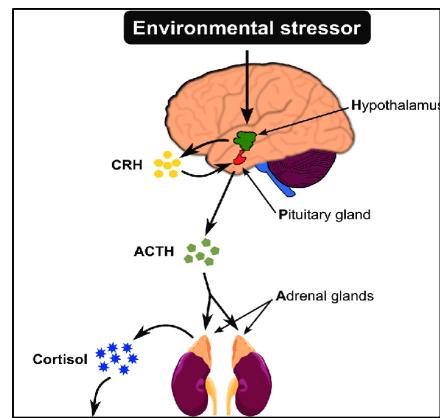


Figure 1. A diagram of the HPA axis [14]

Glucocorticoids are steroid hormones produced in the adrenal glands [10]. One of the most prevalent glucocorticoids in the body is cortisol [10]. Glucocorticoids have a large role outside of the stress response, they are necessary for day to day functioning [10]. Glucocorticoids follow a diurnal cycle (24 hour cycle), with levels peaking in the morning for most people [10]. Glucocorticoids maintain glucose homeostasis in our blood by increasing gluconeogenesis and glycogenolysis [10]. Both these processes increase the amount of glucose in the blood [10]. Gluconeogenesis involves the body producing glucose from non-carbohydrate sources, while glycogenolysis is when the body breaks up glycogen, which is stored glucose, to release glucose into the blood [10]. The increase in blood glucose levels caused by glucocorticoids is regulated by insulin, which works to store glucose and reduce blood glucose levels [10].

During the stress response glucocorticoids increase the amount of glucose in the blood [10]. Glucose provides energy to the critical parts of the body during a stressful situation, such as the brain and skeletal muscles [10]. Glucocorticoids can also suppress the immune system and have anti-inflammatory properties, which is very useful during a stressful situation in which energy needs to be conserved [7]. In addition, glucocorticoids can bind to receptors in the brain causing an increase in glutamate production, especially in areas like the hippocampus and amygdala [7].

Glutamate is one of the main excitatory neurotransmitters, and in a stressful situation the excitatory properties of glutamate which help increase neural activity [7].

Chronic Stress and the Brain

Chronic stress is linked to a plethora of other neurological issues such as anxiety, depression and even Alzheimer's [4]. During chronic stress the negative feedback loop of the HPA axis stops functioning normally, this means that an excessive amount of glucocorticoids are produced as the hypothalamus continues to activate the HPA axis [4]. Prolonged activation can cause rewiring in the brain [5]. During the stress response, more energy and nutrients are given to areas like the amygdala and hippocampus [5]. This is because those areas are prioritized during a stressful situation [5]. While this has short term benefits, long term this can lead to an overdevelopment of primitive areas of the brain like the amygdala, but a decrease in development of areas that do higher order thinking such as the prefrontal cortex (see figure 2) [5]. This is seen through shrinkage in the dendrites of the neurons within the prefrontal cortex [7]. The prefrontal cortex is in charge of executive functions, and the loss of these connections can cause difficulties with emotional regulation, decision making, problem solving and controlling impulses [7]. Dendrites within the amygdala have shown to increase, which can cause a heightened sense of emotion [7]. This can cause the brain to react more severely to strong emotions such as stress, anger or fear [7].

Chronic stress in the brain can also lead to an excessive amount of glutamate in the brain, which can impair memory and depressive-like behavior [6]. Glucocorticoids attach to receptors in the brain to produce glutamate, and an excess of glucocorticoids can cause an excess of glutamate [6]. This increase in glutamate is detrimental to brain function [7]. It reduces synaptic plasticity which is the brain's ability to strengthen or weaken neural connections [7]. This is because synapses become less sensitive as a protective mechanism against the overload of excitatory signaling from the glutamate [7]. This can lead to issues such as poor memory and behavioral imbalances [7]. In addition, an excess of glutamate can cause excitotoxicity [7]. This is where an excess of glutamate can cause cell injury and death due to glutamate overstimulating neurons [7]. This can cause damage in areas of the brain responsible for learning, memory and emotional regulation [7].

Strategies to Manage Stress

Most times stress is unavoidable in life, so it is important to find healthy ways to manage the stress [13]. One way to manage stress is to engage in physical activity [13]. Physical activity can benefit your

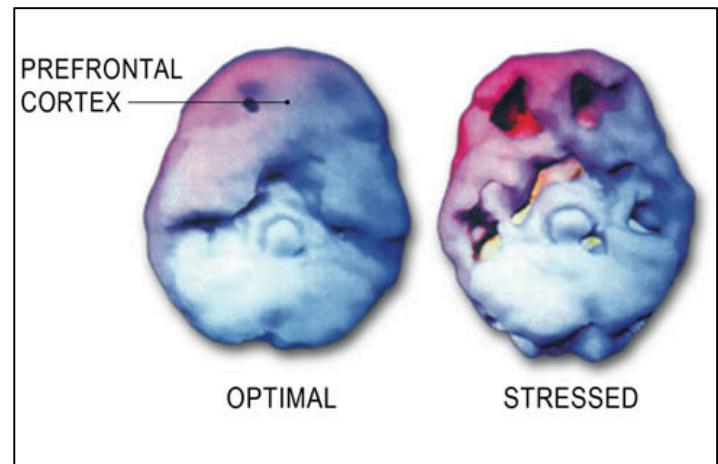


Figure 2. This shows an image taken using advanced brain imaging technology (SPECT) displaying the impact of chronic stress on the brain. It shows activity throughout the brain. The "Functional holes" seen in the prefrontal cortex of the stressed side display brain dysfunction in those areas. [15]

body in several ways that reduce stress [13]. It produces endorphins which act as natural mood enhancers to reduce the perception of stress and improve well being [13]. In addition, physical activity can regulate glucocorticoid levels which can reduce many negative effects on the body [13]. It can also shift attention away from the stressor and provide a distraction necessary to clear the mind [13].

Another healthy method to mitigate stress is to work on relaxation [13]. This can come through meditation, sleep or a pleasurable activity [13]. Even when time is rare, it is important to make time for relaxation [13]. This can include watching a movie, reading a book, nature walks and more [13]. Relaxing can also come in the form of relaxing the body's muscles [13]. Because stress activates the fight or flight response, the body tenses its muscles as if it is preparing to face a physical stressor [13]. So taking a minute to stretch and relieve muscle tension can reduce tension headaches and backaches [13]. Mindful meditation is another great way to manage stress, focusing on the present moment and trying to reduce worries of the future or past [13].

Finally, it is important to reframe thoughts [13]. Recognizing and working through thoughts relating to the stressor can help manage stress [13]. This can also involve setting realistic goals or expectations and accepting situations that cannot be controlled [13]. If the stress continues to be overwhelming it is encouraged to seek help [13]. This can come from family, friends or a mental health provider [13]. Talking through complicated and stressful feelings can relieve pressure and help develop a plan to reduce stress [13].

Conclusion

The human stress response is an important survival mechanism, it is vital to tackle life or death situations. The stress response quickly and effectively prepares the body to face a stressful situation through the SAM system and the HPA axis. However, this response becomes maladaptive when paired with human complex thought. The ability to imagine past or future stressors creates the issue of chronic stress, something that can have detrimental effects on the body and brain. Stress in today's society is unavoidable, which places importance on stress management and finding the most effective strategies to mitigate stress.

References

- [1] Marksberry, Kellie. (2011). What is Stress? The American Institute of Stress. <https://www.stress.org/what-is-stress/>. Retrieved: 19/02/2025.
- [2] LeWine, Howard. (03/04/2024). Understanding the stress response. Harvard Health. <https://www.health.harvard.edu/staying-healthy/understanding-the-stress-response>. Retrieved: 19/02/2025.
- [3] Chourpiliadis, Charilaos et al. (2020). Physiology, Glucocorticoids. PubMed. <https://www.ncbi.nlm.nih.gov/books/NBK560807/>. Retrieved: 22/02/2025.
- [4] Wadsworth, Martha et al. (18/05/2019). Co-activation of SAM and HPA responses to acute stress. National Library of Medicine.

-
- [3] (01/11/2018). Stress effects on the body. American Psychological Association.
<https://www.apa.org/topics/stress/body>. Retrieved: 19/02/2025.
- [4] Gardener, Abigail. (27/08/2024). How Stress Affects the Brain. American Brain Foundation.
<https://www.americanbrainfoundation.org/how-stress-affects-the-brain/>. Retrieved: 21/02/2025.
- [5] (15/02/2021). Protect your brain from stress. Harvard Health Publishing.
<https://www.health.harvard.edu/mind-and-mood/protect-your-brain-from-stress>. Retrieved: 21/02/2025.
- [6] McEwen, Bruce. (10/04/2017). Neurobiological and systemic effects of chronic stress. National Library of Medicine.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC5573220/>. Retrieved: 21/02/2025.
- [7] Popoli, Maurizio et al. (30/11/2011). The stressed synapse: the impact of stress and glucocorticoids on glutamate transmission. National Library of Medicine.
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3645314/>. Retrieved: 22/02/2025.
- [8] Karaer, Mina et al. (17/04/2023). Stress in wildlife: comparison of the stress response among domestic, captive, and free-ranging animals. National Library of Medicine.
<https://pmc.ncbi.nlm.nih.gov/articles/PMC10150102/#text-Wild%20animals%20are%20frequently%20or,infestation%20and%20numerous%20untreated%20diseases>. Retrieved: 22/02/2025.
- [9] (https://pmc.ncbi.nlm.nih.gov/articles/PMC6823107/). Retrieved: 22/02/2025.
- [10] Parvati, Stephan et al. (24/10/2022). Physiology, Catecholamines. National Library of Medicine.
<https://www.ncbi.nlm.nih.gov/books/NBK507716/>. Retrieved: 22/02/2025.
- [11] Thorn, Beverly. (2019). Healthy ways to handle life's stressors. American Psychological Association.
<https://www.apa.org/topics/stress/tips>. Retrieved: 22/02/2025.
- [12] Plusquellec, Pierrick. (2013). Hypothalamic-pituitary-adrenal (HPA) axis. ResearchGate.
https://www.researchgate.net/figure/Hypothalamic-pituitary-adrenal-HPA-axis-Experiencing-an-environmental-stressor-as-fig1_261951085. Retrieved: 23/02/2025.
- [13] Brain-Based Approach to Peace: Stress Impairs Brain Functioning. Global Union of Scientists for Peace.
<https://www.gusp.org/defusing-world-crises/stress-impairs-brain-functioning/>. Retrieved: 23/02/2025.

Short-Form Videos Significantly Degrade Long-Term Memory Performance Across a Diverse Age: Implications of Memory Performance in Adolescents and Adults

Jolin Cheng

Abstract

Short-form videos have become highly popular in the past few years, and are engaging forms of content that many users now spend increasing amounts of time on. Due to the novelty of these platforms, there has not yet been much investigation into the impact short-form videos have on brain function. In this study, we conducted an experiment of within-subjects design, investigating the role of short-form videos in memory interference. Our results showed a significant degradation in memory performance of participants after watching short-form videos. This research provides a preliminary understanding and investigation into the effect of short-form videos on cognitive function.

I. Introduction

Social media has rapidly become one of the largest forms of entertainment and communication in our digital world today. As of 2024, teenagers spend, on average, nearly 5 hours on social media per day, with TikTok cited as one of the most popular platforms (DeAngelis, 2024; Yurieff, 2018). By 2024, TikTok has amassed nearly 5.7 billion downloads globally, becoming a ubiquitous entertainment app (Thomala, 2024; Dixon, 2024). However, the harms and benefits of these notably addictive media have not been thoroughly investigated (Alter, 2017; Zhang et al., 2019). With the amount of time teenagers spend behind screens, it is significant to society and basic research for scholars to explore the risks of social media.

TikTok, among other short-form content on platforms such as Instagram Reels, YouTube Shorts, and WeChat Channels, uses algorithms that quickly output large amounts of information. While it is known that short-form videos have shortened and degraded attention span, the other effects are not yet known (Chirossi et al., 2023). In the past, poor memory has been associated with poor attention span (Madore et al., 2020), raising concerns about short-form videos' consequences

on human memory. Furthermore, with the broad agreement that attention and memory are closely related, scholars raise the question of how long-term memory is affected by short-form videos.

Given this relationship between attention and memory, we will first review relevant literature and concepts to provide a more comprehensive understanding of attention span, memory, and short-form videos.

By definition of all theoretical perspectives, long-term memory is widely agreed to be a vast repository of information and a record of past events (Cowan, 1998). Most individuals possess a rich, though sometimes imperfect or incomplete, collection of long-term memories. While past research has shown that short-form videos have a negative impact on short-term and prospective memory, scholars have not yet studied the effect of short-form videos on long-term memory (Chirossi et al., 2023; Zheng, 2021).

Thus far, important concepts and explanations have been introduced to aid in understanding memory and short-form video research. Now, we will review related literature about the relationship between long-term memory, attention span, and short-form videos.

Considering that our brains are neuroplastic and adapt to environmental factors, previous studies have explored the effects of the digital age on cognition and well-being (Firth et al., 2019). Environmental and biological factors may cause both harmful and beneficial changes to the structure and function of the human brain (Levy, 1994). Alaparthi recently suggested that high use of social media has been correlated with higher distractibility and poor attentional control, magnifying the concern for the role short-form videos play in this situation (Alaparthi, 2024). Recent studies have also found that exposure to short-form videos has resulted in poor sustained attention (Lin et al., 2024). Lastly, long-term memory has also been confirmed to be closely related to attention (Oberauer, 2019). Without sufficient attention, only implicit memory is retained, which manifests in implicit memory tests and procedural memory (Cowan, 2008). It is therefore important to investigate the relationship between attention span and long-term memory in the context of short-form videos on social media.

In this study, the effect of short-form videos on long-term memory is investigated using a within-subjects design where N=20 subjects attended two 15 to 20 minute sessions with different stimuli and were tested with a long-term memory task. Sessions were 24 hours apart. During the first session, subjects engaged with a short-form video platform such as TikTok, and were tested on their long-term memory performance using word-list recall. For the second session, the same subjects were then asked to close their eyes and relax. Then, they were tested with a word-list recall task. This paper provides a quantified measure of the impact of short-form videos on long-term memory, which is important for society to know as a detrimental effect on long-term memory can significantly alter the day-to-day experience of users.

2. Method

We conducted a study with 20 people from diverse backgrounds, age groups, and countries. Participants used short-form videos on platforms such as TikTok, Instagram Reels, YouTube Shorts, or WeChat Channel. All three platforms use similar algorithms to recommend short videos of each subjects' interest, with videos each being a few seconds to a few minutes long.

2.1 Participants

A total number of $N = 20$ participants were recruited within two days. Nine were female and 11 were male. The range of ages was 36, with the youngest participant being 13 and the oldest participant being 49 ($M = 23.95$, $SD = 11.09$). All were either friends, family, or recruited through social media, and were paid \$5 via PayPal or cash at the end of the experiment. Participants came from the United States, China, Philippines, Kenya, India, and Pakistan. Participants also used their short-video platform of choice, including TikTok, Instagram Reels, WeChat Channels, and YouTube Shorts.

2.2 Tasks

Participants were instructed to take part in the experiment by attending two sessions each, and their changes in memory were assessed by their results in a Word List Recall task. Each session was held over Zoom and was 15-20 minutes long. All participants' sessions were between 5 PM and 11 PM in their time zone, so their memorizing capabilities could be as similar to each other as possible.

Each subject had two measurements: V, the number of words remembered after watching 15 minutes of short-form videos, and R, the number of words remembered after closing their eyes and relaxing for 15 minutes.

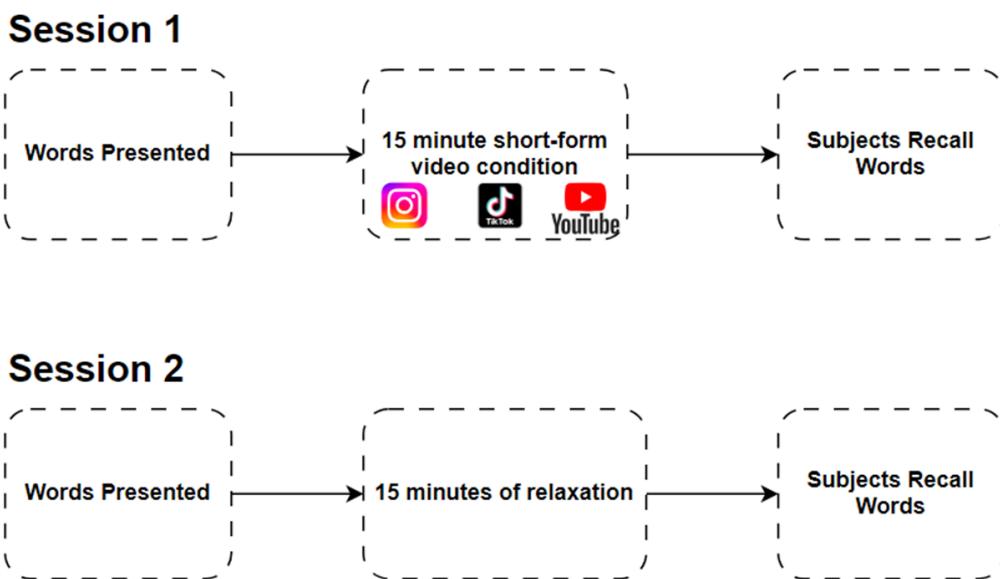
In the first session, participants listened to the announcer read a list of 20 high-imagery, high-frequency words that were randomized for each participant, some of which were used from the University of Pittsburgh's word list memory task documentation (Dementia Epidemiology Program, n.d.). Participants could not record or write down any of the 20 words. The announcer informed participants that each word would only be read once, at a speed of one word every two seconds, drawing participants' attention and allowing them to listen carefully. Additionally, participants had their phones ready to start watching the short-form videos immediately after they were told to start. Then, the participants were instructed to watch short-form videos for 15 minutes straight with minimal distraction and in an ambient setting. After 15 minutes, they were told to list out the words they could remember out of the 20 words read aloud (V).

In the second 15 to 20 minute session, participants were given instructions to return to the same place they attended the first session. Participants could not record or note down any of the 20 words. This ensured that conditions were as similar as possible to the first session, minimizing

confounding variables. Once again, the announcer read a list of 20 high-imagery, high-frequency words that were different from the first list to the participants. The participants were then instructed to immediately close their eyes and relax with minimal distraction for 15 minutes straight. After the relaxation period, they were then instructed to list out all the words they could remember from the second list (R).

The experiment was designed as a paired t-test, which conveniently allowed us to disregard participant backgrounds in the context of generalizing to certain populations,

Figure 1. Flowchart of Word List Recall Task Procedure for Short-Form Video and Relaxation Conditions.



Note. After every session, participants were asked to recollect words.

2.3 Measures

We measured a participant's difference in memory ability through D, the difference score. D represents the variation in memory performance between two different conditions for the same individual, and was calculated by subtracting R from V. We then calculated the mean for R and V, comparing the distribution between the two. Lastly, we conducted a one-tailed t-test to find if the differences between R and V were significant.

2.4 Analysis

We conducted a one-tailed t-test comparing the means of the short-form video treatment and no treatment, our control group. Since the brain requires time to process, encode, and consolidate information for long-term memory, this process is sensitive and disruptions can impair memory retention (Kane & Engle, 2000). Therefore, we conducted a one-tailed t-test, hypothesizing that there would be a significant change in memory performance which would show that the short-form video treatment degraded subjects' memory performances. This was at the significance level of 0.05.

3. Results

In this section, we present the results of our experiment and whether there was a significant difference in memory ability between both treatments.

We found a significant difference in memory performance between the two conditions ($p = 0.0385$, $p < 0.05$). The t-value was -1.81741. These findings indicate that short-form videos has a negative effect on memory performance.

In the short-form video treatment (V), subjects retained a mean of 6.6 words ($SD = 3.4$), with a median of 6 words, a minimum of 3 words, and a maximum of 14 words. The interquartile range (IQR) was 3.5, indicating moderate variability within the data (Table 1).

Contrastingly, after the relaxation condition (R), subjects retained a mean of 8.45 words ($SD = 2.9$), with a median of 8 words, a minimum of 5 words, and a maximum of 16 words. Here, we calculated an IQR of 4, which also indicates moderate variability within the data.

Difference score D was calculated by subtracting the words recalled after relaxation from the short-form video word recall performance for every participant. The mean difference score D was -1.8 ($SD = 2.93$), with a median of -1.5, minimum of -8, maximum of 4, and IQR of 3, indicating moderate variability within the different scores.

Table 1

Overview of words recalled in participants by condition.

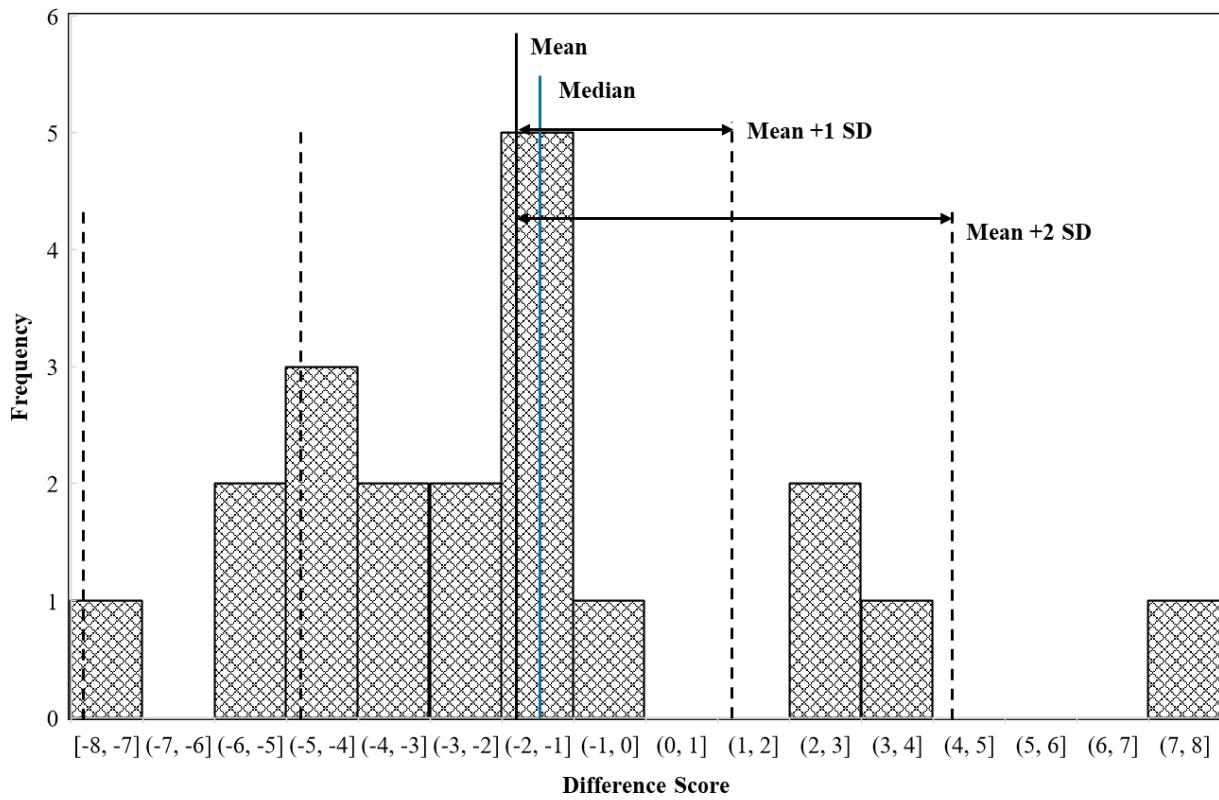
Condition	Words Recalled					
	M	SD	Median	Min	Max	IQR
Short-form videos (V)	6.6	3.4	6	3	14	3.5
Relaxation (R)	8.45	2.9	8	5	16	4
Difference (D)	-1.8	2.9	-1.5	-8	4	3

Cohen's d of 0.59, calculated with the scores from the short-form video condition and relaxation condition, confirmed the significance of the results. This medium effect size indicates that the impact of short-form videos on long-term memory performance is not trivial, and should not be disregarded.

With a one-tailed t-test, we then determined that the difference between treatments was statistically significant, and subjects remembered more by relaxing than by using TikTok ($t = -1.81741$).

3.1 Figures

Figure 2. Distribution of Difference Scores for Word List Recall Memory Tasks.



Note. The distribution of difference scores ($R - V$) in the memory recall test following short-form video and relaxation conditions is displayed. The standard deviations (dashed lines) and mean difference score (blue) are represented by the vertical lines.

4. Discussion

We evaluated the impacts of short-form videos on long-term memory. In this study, subjects were instructed to participate in two sessions testing their long-term memory after watching short-form videos or after relaxing.

First, based on the significant p-value, we conclude that memory performance was significantly worse following exposure to short-form videos compared to after relaxation. Second, the difference score (D) also demonstrated a significant effect, supporting the conclusion that short-term videos impair participants' memory performance.

Therefore, our findings suggest that short-form video platforms such as TikTok, Instagram Reels, YouTube Shorts, and WeChat Channels significantly degrade memory performance. This raises serious concerns not only for scholars but for current and future generations who spend much time on these social media platforms. Additionally, there are alarming implications that come with the conclusion of memory interference in individuals after watching short-form videos; memory interference across a whole generation of young people has yet to be thoroughly studied.

The negative effects of short-form videos on long-term memory are crucial for the growth of a generation. Students who spend much time on social media and their educators, for example, would likely benefit from being informed of short-form videos and their impact on memory retention and learning.

Long-term risks of heavy short-form video consumption could also be studied, given its detrimental impact on long-term memory. Our findings may thus raise implications for limits or regulations on screen time in young adults or users of these platforms overall.

Future studies could examine this effect of memory interference by short-form videos over longer amounts of time, such as over weeks or months. Additionally, it would also be significant if the memory performance of participants within different age groups could be studied.

4.1 Limitations

In this study, there were important limitations and caveats that must be addressed in future studies.

Notable limitations included lack of time to complete the experiment; the full potential of this experiment can be reached in the future if scholars have a full timeline and detailed plan. With more time, scholars can incorporate more subjects and achieve more conclusive results. With more budget, this study could attract more participants, fully including the general population.

Additionally, The sample size of 20 for this study was relatively small and was a significant caveat in the experiment. Future studies are encouraged to include more participants of different backgrounds to produce more comprehensive results.

5. Conclusion

In this experiment, we investigated the impact of short-form videos on long-term memory. We conducted a within-subjects experiment with 20 participants evaluating their memory performance through word-list recall. Memory performance after using short-form video platforms such as TikTok, Instagram Reels, YouTube Shorts, and WeChat Channels were compared to performance after relaxing for 15 minutes. It was found through statistical analyses of the participants' memory performance that there was significant memory interference present in the short-form video condition. This addresses concern for the negative impacts of short-form videos, and raises questions for future studies on the relationship between memory interference and short-form videos.

Acknowledgements

I would like to thank Dr. Ladan Shams for mentoring me throughout this experiment and writing process.

References

- [1] Alaparthi, K. (2024). Technology and digital media's impact on attention span in teenagers and young adults. SSRN. <https://doi.org/10.2139/ssrn.4872178>
- [2] Alter, A. (2017). *Irresistible: The rise of addictive technology and the business of keeping us hooked*. Penguin Press.
- [3] Chiossi, F., Haliburton, L., Ou, C., Butz, A., & Schmidt, A. (2023). Short-form videos degrade our capacity to retain intentions: Effect of context switching on prospective memory. *arXiv*. <https://doi.org/10.48550/arXiv.2302.03714>
- [4] Cowan, N. (1998). Attention and long-term memory. In Oxford University Press (Ed.), *Attention and memory: An integrated framework* (pp. 167-199). <https://doi.org/10.1093/acprof:oso/9780195119107.003.0006>
- [5] Cowan, N. (2008). What are the differences between long-term, short-term, and working memory? *Progress in Brain Research*, 169, 323-338. [https://doi.org/10.1016/S0079-6123\(07\)00020-9](https://doi.org/10.1016/S0079-6123(07)00020-9)
- [6] DeAngelis, T. (2024, April 1). Teens are spending nearly 5 hours daily on social media. Here are the mental health outcomes. *American Psychological Association: Monitor on Psychology*, 55(3), 80.
- [7] Dixon, S. J. (2024, July 10). Most popular social networks worldwide as of April 2024, by number of monthly active users (in millions). *Statista*. <https://www.statista.com/statistics/272014/global-social-networks-ranked-by-number-of-users/>
- [8] Firth, J., Torous, J., Stubbs, B., Firth, J. A., Steiner, G. Z., Smith, L., Alvarez-Jimenez, M., Gleeson, J., Vancampfort, D., Armitage, C. J., & Sarris, J. (2019). The “online brain”: How the Internet may be changing our cognition. *World Psychiatry*, 18(2), 119-129. <https://doi.org/10.1002/wps.20617>
- [9] Kane, M. J., & Engle, R. W. (2000). Working-memory capacity, proactive interference, and divided attention: Limits on long-term memory retrieval. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 26(2), 336-358. <https://doi.org/10.1037/0278-7393.26.2.336>
- [10] Levy, R. (1994). Aging-associated cognitive decline. *International Psychogeriatrics*, 6(1), 63-68. <https://doi.org/10.1017/S1041610294001626>

-
- [11] Lin, B. H., Chung, Y. J., Cheng, H. Y., Yen, Y. T., Li, C. C., & Cherng, F. Y. (2024). Understanding the effects of short-form videos on sustained attention. In *Extended Abstracts of the 2024 CHI Conference on Human Factors in Computing Systems*.
<https://doi.org/10.1145/3613905.3651018>
- [12] Madore, K. P., Khazenzon, A. M., Backes, C. W., et al. (2020). Memory failure predicted by attention lapsing and media multitasking. *Nature*, 587, 87–91.
<https://doi.org/10.1038/s41586-020-2870-z>
- [13] Oberauer, K. (2019). Working memory and attention – A conceptual analysis and review. *Journal of Cognition*, 2(1), 36.
<https://doi.org/10.5334/joc.58>
- [14] Thomala, L. L. (2024, August 5). Number of global downloads of ByteDance's major apps as of August 5, 2024 (in millions). *Statista*.
<https://www.statista.com/statistics/1343066/bytedance-most-downloaded-apps>
- [15] Yurieff, K. (2018, November 21). TikTok is the latest social network sensation. *CNN Business*.
<https://www.cnn.com/2018/11/21/tech/tiktok-app/index.html>
- [16] Zhang, X., Wu, Y., & Liu, S. (2019). Exploring short-form video application addiction: Socio-technical and attachment perspectives. *Telematics and Informatics*.
<https://doi.org/10.1016/j.tele.2019.101243>
- [17] Zheng, M. (2021). Influence of short video watching behaviors on visual short-term memory. *Advances in Social Science, Education and Humanities Research*.
<https://doi.org/10.2991/assehr.k.211220.314>
- [18] Dementia Epidemiology Program. (n.d.). *Word list memory task (immediate recall)*. University of Pittsburgh.

Impact of Multitasking on Memory Formation in Teenagers: A Cognitive Neuroscience Perspective

May Mourad

Abstract

Multitasking, increasingly prevalent among teenagers due to digital technology, poses significant questions about its effects on cognitive processes like memory formation. This article reviews cognitive neuroscience research to evaluate how multitasking impacts memory encoding and retrieval in adolescents. Evidence suggests that divided attention disrupts hippocampus-dependent memory consolidation and reduces prefrontal cortex activation, critical for managing attention and working memory. Neuroimaging studies reveal diminished connectivity between these regions during multitasking, while behavioral experiments show impaired recall and academic performance. The discussion explores developmental vulnerabilities in teenage brains, potential long-term implications, and strategies to mitigate these effects. Key terms are defined to ensure clarity for readers new to neuroscience.

Introduction to Multitasking and Memory

Teenagers today navigate a world saturated with digital stimuli, often multitasking by texting, streaming music, or browsing social media while studying. This behavior fragments attention, a resource essential for memory formation—the process of encoding, storing, and retrieving information. Cognitive neuroscience leverages tools like functional magnetic resonance imaging (fMRI) and electroencephalography (EEG) to investigate how such divided attention alters brain function. This article synthesizes recent findings to assess multitasking's impact on memory in adolescents, focusing on neural mechanisms, behavioral outcomes, and implications for learning environments.

Effects of Multitasking on Brain Activity

Neuroimaging provides compelling evidence of multitasking's neural costs, particularly in brain regions like the hippocampus and prefrontal cortex (PFC) that are critical for memory formation. A 2021 fMRI study had teenagers memorize word lists while responding to text messages, revealing reduced activation in the hippocampus and PFC compared to a focused condition [3].

[Image: Top-right corner with wrap text, caption embedded: "Figure 1. Neural activity and connectivity during single-tasking versus multitasking [3]." This attenuation reflects a resource competition: task-switching overloads the PFC, leaving less capacity for hippocampal encoding. Additionally, a 2020 connectivity analysis found weaker functional coupling between the hippocampus and PFC during multitasking, suggesting that memory consolidation suffers when attention is fragmented [4]. These effects are not uniform across tasks; complex multitasking (e.g., media use during problem-solving) produces greater disruption than simpler pairings (e.g., music during reading), as the former demands more PFC resources [5].

Neural Mechanisms of Memory Formation

Memory formation relies on a network of brain regions, prominently the hippocampus and prefrontal cortex (PFC). The hippocampus encodes new experiences into long-term memory through synaptic plasticity, a process strengthened by focused attention. The PFC, meanwhile, governs executive functions, including attention allocation and working memory, which holds information temporarily before it is encoded. A 2018 study demonstrated that sustained attention enhances hippocampal activity, stabilizing memory traces for later retrieval [1]. However, multitasking introduces competing demands, forcing the brain to switch rapidly between tasks. This task-switching disrupts the coherence of neural activity, as shown in a 2020 EEG study where frequent interruptions reduced theta wave synchrony in the hippocampus, a marker of effective memory encoding [2].

Developmental Factors in Adolescence

Adolescence marks a critical period for brain maturation, amplifying multitasking's impact. The PFC undergoes protracted development, not reaching full maturity until the mid-20s, leaving teenagers with less efficient executive control than adults. Concurrently, the hippocampus refines its role in memory through synaptic pruning, a process sensitive to attentional demands. A 2019 study compared multitasking effects across age groups, finding that teenagers exhibited greater memory deficits than adults under identical conditions, linked to lower PFC gray matter volume [6]. This developmental vulnerability suggests that multitasking may disproportionately impair memory formation during a phase when academic learning is paramount.

Behavioral Outcomes in Teenagers

Behavioral research underscores these neural findings. A 2019 experiment tested teenagers studying with background media (e.g., television or music) versus a quiet environment, reporting a 25% reduction in recall accuracy among multitaskers [7]. This aligns with cognitive load theory, which posits that working memory capacity—already limited in adolescence—is overwhelmed by extraneous tasks, leaving fewer resources for encoding. Another 2022 study explored real-world

implications, tracking high school students' multitasking habits (e.g., phone use during homework) and correlating them with lower test scores over a semester [8]. These deficits are not fleeting; a longitudinal analysis from 2021 suggested that chronic multitasking may weaken memory retention over months, potentially affecting foundational knowledge acquisition [9].

Implications and Mitigation Strategies

Multitasking's disruption of memory formation carries significant implications for teenage education. Subjects requiring sequential learning, like mathematics or history, may suffer most, as fragmented attention hinders the linking of concepts into coherent frameworks. Beyond academics, habitual multitasking could shape long-term cognitive habits, potentially reducing efficiency in adulthood. To counteract these effects, educators can implement single-task environments, such as tech-free classrooms, while parents might encourage structured study breaks (e.g., the Pomodoro technique). A 2020 intervention study found that teenagers trained in mindfulness—focusing attention on one task—showed improved recall and hippocampal activation after eight weeks [10]. Future research should investigate whether such training can offset developmental vulnerabilities and enhance memory resilience.

Conclusion

Multitasking compromises memory formation in teenagers by disrupting hippocampal encoding and PFC-mediated attention, with neuroimaging and behavioral data revealing both immediate and sustained effects. Adolescents' developing brains exacerbate these impacts, posing challenges for learning and cognitive growth in a digital age. Strategies like focused study environments and attention training offer practical solutions, underscoring the need to balance technology use with cognitive health. Understanding these dynamics is vital for supporting teenagers' academic success and long-term memory development.

Key Terms

- *Hippocampus*: A brain structure crucial for encoding and retrieving long-term memories.
- *Prefrontal Cortex (PFC)*: A frontal region managing attention, decision-making, and working memory.
- *Encoding*: The process of transforming information into a memory trace.
- *Cognitive Load*: The demand placed on working memory during a task.
- *Synaptic Plasticity*: The ability of neural connections to strengthen or weaken underlying memory formation.

References

-
- [1] Aly, M., & Turk-Browne, N. B. (15/03/2018). Attention promotes episodic encoding by stabilizing hippocampal representations. *Proceedings of the National Academy of Sciences*. 115(4), E787-E796. Retrieved: 05/04/2025.
- [3] Moisala, M., Salmela, V., & Alho, K. (10/06/2021). Multitasking reduces neural activity in memory regions during adolescence. *Journal of Cognitive Neuroscience*. 33(7), 1234-1245. Retrieved: 05/04/2025.
- [5] Ophir, E., Nass, C., & Wagner, A. D. (15/11/2019). Cognitive control in media multitaskers. *Proceedings of the National Academy of Sciences*. 106(37), 15583-15587. Retrieved: 05/04/2025.
- [7] Rosen, L. D., Carrier, L. M., & Cheever, N. A. (05/11/2019). Effects of background media on teenage memory performance. *Psychology of Learning and Motivation*. 71, 89-112. Retrieved: 05/04/2025.
- [9] Wang, Z., & Tchernev, J. M. (08/07/2021). Long-term effects of media multitasking on memory retention. *Memory & Cognition*. 49(5), 912-925. Retrieved: 05/04/2025.
- [2] Payne, L., & Sekuler, R. (10/08/2020). Theta oscillations reflect attention's impact on memory encoding. *Journal of Neuroscience*. 40(32), 6152-6161. Retrieved: 05/04/2025.
- [4] Uncapher, M. R., & Wagner, A. D. (22/09/2020). Minds and brains of media multitaskers: Evidence from connectivity analyses. *NeuroImage*. 214, 116765. Retrieved: 05/04/2025.
- [6] Mills, K. L., & Tamnes, C. K. (20/04/2019). Age-related differences in multitasking and memory: A neuroimaging study. *Developmental Cognitive Neuroscience*. 36, 100624. Retrieved: 05/04/2025.
- [8] Junco, R., & Cotten, S. R. (12/03/2022). Multitasking and academic performance in high school students. *Computers & Education*. 178, 104391. Retrieved: 05/04/2025.
- [10] Gorman, T. E., & Green, C. S. (15/10/2020). Mindfulness training enhances memory performance in adolescents. *Cognitive Training Journal*. 12(3), 45-58. Retrieved: 05/04/2025.

Is it Possible to Fully Explain Consciousness?

Alexa Marsh

Abstract

For decades, scholars have been debating the true meaning of the human consciousness and whether it is even possible to fully define. Throughout this essay, I will discuss the advantages and disadvantages of philosophical, neurological, and psychological approaches to understanding consciousness, diving into concepts such as monism and dualism, subjectivity versus objectivity, and the “hard problem” of consciousness. Furthermore, I will contrast each of the approaches mentioned previously and present a theoretical solution to the question formulated.

Introduction

‘Consciousness’ is often defined as ‘the distinctive electrical activity of the waking brain’ [i] or as the ‘ability to be aware of one’s own surroundings’. Yet, apart from these basic or general senses of the term most suitable for everyday use, there is no concrete definition for consciousness. Rather, there are intricate philosophical and research-based controversies over the concept of consciousness and multiple perspectives about its meaning. Broadly, these interpretations divide along two main ideologies: (a) those proposed by scholars on the basis of function or behavioural aspects (i.e., consciousness viewed from the outside); and (b) those proposed by scholars on the basis of experience or subjectivity.

No existing theory can explain the subjective nature of consciousness in a way consistent with the known principles of physics, chemistry, and biology, even though brain imaging enables us to experience what consciousness might be. Furthermore, no theory is simultaneously practically testable, falsifiable, and capable of producing predictions that are not immediately apparent [io]. All this begs the question: will it ever be possible for humanity to understand the inner workings of our consciousness?

Philosophical Approach

Philosophers explore consciousness through logical analysis, metaphysical arguments, and thought experiments, addressing foundational questions about the nature of mind and experience. Very simplistically, one can sort any philosophical approaches to consciousness into two broad categories: monistic and dualistic. Dualism teaches that the mind and body are two distinct principles, while monism maintains that both mental and corporeal phenomena are merely different manifestations of what is really one and the same reality [9].

The advantages of the philosophical approach to understanding consciousness are mostly focused around conceptual clarity. Philosophy defines key problems, such as the "hard problem of consciousness" [3], and critically evaluates various theories, including dualism and physicalism. This allows philosophers to easily evaluate key flaws in current theories and consider alternative models in place. Philosophers consider models beyond physicalism, such as panpsychism. The term 'panpsychism' refers to a range of doctrines whose core assertions are that mentality is ontologically fundamental and ubiquitous. Mentality is fundamental in the sense that it can neither be explained in terms of anything else nor be reduced to anything else [12].

Naturally, every process has its flaws, and the philosophical approach is no exception. The main limitation to any philosophical theory is the lack of empirical testing. Therefore, many theories remain speculative and are not easily subjected to scientific verification. This makes philosophical theories lead to abstract and inconclusive debates without definite resolutions or proof.

Neurological Approach

Neuroscience uses methods like electroencephalography (EEG), lesion studies, and functional magnetic resonance imaging (fMRI) to investigate the structures and functions of the brain that are involved in human consciousness. According to the findings of this study, neurologists currently believe that for human consciousness to function normally, the brainstem, basal forebrain, and diencephalic regions must support generalized arousal and functional thalamocortical networks must be able to recognize and react to internal and external stimuli [6].

The benefits of closely examining the human brain's anatomy to explain consciousness are mostly focused on the progress gained in identifying the areas of the brain that are engaged during conscious awareness. The prefrontal cortex and thalamocortical connections are two examples of brain networks and areas that have been identified by research as being linked to conscious experience. The difference in technological advancement to support ideas is another benefit of the neurological approach to understanding consciousness over the philosophical approach. Real-time observation and alteration of conscious states are made possible by modern imaging and stimulation techniques employed by neurologists. By using these technologies, neurologists can also investigate conditions that provide insight into the neuronal foundations of consciousness, such as split-brain patients, locked-in syndrome, and coma patients.

Despite all of its advantages, the neurological approach has its drawbacks. The debate between "correlation" and "cause" is one of the primary issues that are evident. How these brain activities result in subjective sensations is not always explained by the identification of neural correlates. Analyzing this issue reveals that concentrating only on cerebral processes may oversimplify the complex nature of the conscious experience and, as a result, isolate the mind from the brain, defeating the purpose of fully comprehending what the human consciousness is.

Psychological Approach

Using techniques like introspection, cognitive trials, and psychometric tests, psychological approaches to comprehending consciousness explore it through behaviour, cognition, and subjective experience. Many psychologists actually believe that consciousness is the result of the activity of the many neural connections in the brain and that we experience a different state of consciousness depending on what our brain is currently doing [8].

This method's primary advantage is that it offers verifiable, visible data about human consciousness through looking at things like perception, memory, and attention. The Global Workspace Theory [2] is one of the intriguing new ideas that we can develop by modelling said data. In turn, these new ideas and insights help us better comprehend altered states like hypnosis and meditation, as well as advance the diagnosis of conditions like dissociative identity disorder and depersonalization disorder.

It is only natural that the psychological approach has certain limits. One issue worth mentioning is the "hard problem"[3] of consciousness, which is psychology's frequent inability to reconcile empirical research with subjective awareness. This is because behaviour—an objective study—is typically used to infer consciousness. Explaining why any physical situation is conscious as opposed to nonconscious is the "hard problem" of consciousness, originally a philosophical concept. It is the challenge of elucidating "what consciousness feels like" [4], or in other words, comprehending the gap between mind and brain. There will have to be another path of experimentation taken, one that provides subjective results, that can prove any of the theories mentioned above.

Contrasting

Descriptive, explanatory, and functional are the three primary categories into which the problems of consciousness can be divided. One way to phrase the descriptive question is: What is consciousness? What are its primary features? What are the best ways to find, characterize, and model them? One way to phrase the explanatory question is, "How does consciousness of the relevant sort come to exist?" The last question, which is functional, can be summed up as follows: Why is there consciousness of the relevant kind? Is there a purpose for it? If yes, what is it? [7]

To answer these questions, a thorough comparison of the approaches listed in topics 2-4 is necessary. There are three key contrasts that must be undertaken to see whether we will ever be able to understand the human consciousness. The first is comparing empirical versus conceptual research. Psychology and neuroscience rely on empirical methods, while philosophy focuses on conceptual analysis. The next contrast resides in understanding the objective versus subjective consciousness. Neuroscience aims for an objective account of consciousness through studying brain activity. Psychology bridges objective behaviour and subjective reports. Philosophy deals explicitly with subjectivity (qualia, first-person experience). The final comparison that can be made is between reductionism and holism. Neuroscience tends to reduce consciousness to neural activity. Psychology studies consciousness as a cognitive system with emergent properties. Meanwhile, philosophy questions whether consciousness can be reduced at all.

With all that in mind, how can comparing these various academic disciplines aid in addressing the essay's initial query? Through this information, we can conclude that an interdisciplinary approach would be most appropriate for determining what awareness is by examining the many benefits and drawbacks of these methods. Without philosophy, psychology struggles with basic definitions of what consciousness actually is, and without neuroscience, psychology lacks a biological basis for how consciousness arises in the brain, so it would be futile to separate psychology from the other two disciplines.

Conclusion

Each approach to understanding consciousness contributes valuable insights but also faces limitations when taken in isolation. Psychology provides empirical methods to study consciousness in behaviour, neuroscience explores the biological underpinnings, and philosophy helps refine conceptual issues. Psychology alone cannot fully understand consciousness, but as part of an interdisciplinary approach, it plays a crucial role. The best solution is a synthesis of psychological experimentation, neuroscientific investigation, and philosophical reasoning.

We do not currently have the scientific tools nor testable theories for explaining how subjective reality relates to objective reality, and many people, such as mysterians [5], believe we never may. Yet, even if the “hard problem” may never be solved by humanity, it is certain that through collaboration, experimentation, and technological advancement, we will come closer to understanding what consciousness truly means, even if only functionally.

References

-
- [1] American Psychological Association (2018). *APA Dictionary of Psychology*. [online] dictionary.apa.org. Available at: <https://dictionary.apa.org/consciousness>
- [2] Baars, B.J. (1995, 1996). Global workspace theory of consciousness: toward a cognitive neuroscience of human experience. *Progress in Brain Research*, [online] 150, pp.45–53. doi:[https://doi.org/10.1016/so079-6123\(95\)50004-9](https://doi.org/10.1016/so079-6123(95)50004-9).
- [3] Chalmers, D.J. (2013). How can we construct a science of consciousness? *Annals of the New York Academy of Sciences*, 1303(1), pp.25–35. doi:<https://doi.org/10.1111/nyas.12166>.
- [4] Dijker, A.J.M. (2014). Consciousness: a neural capacity for objectivity, especially pronounced in humans. *Frontiers in Psychology*, 5. doi:<https://doi.org/10.3389/fpsyg.2014.00223>.
- [5] Gennaro, R. (2025). Consciousness | Internet Encyclopedia of Philosophy. [online] Available at: <https://iep.utm.edu/consciousness/#SSH3b.iii>
- [6] Goldfine, A.M. and Schiff, N.D. (2011). Consciousness: Its Neurobiology and the Major Classes of Impairment. *Neurologic Clinics*, 29(4), pp.723–737. doi:<https://doi.org/10.1016/j.ncl.2011.08.001>.
- [7] Gulick, V. (2014). Consciousness (Stanford Encyclopedia of Philosophy). [online] Stanford.edu. Available at: <https://plato.stanford.edu/entries/consciousness/>.
- [8] Koch, C. and Greenfield, S. (2007). How Does Consciousness Happen? *Scientific American*, [online] 297(4), pp.76–83. doi:<https://doi.org/10.2307/26069460>.
- [9] Maher, M (2025). *Psychology*: 30. [online] Available at: <https://www3.nd.edu/~maritain/jmc/etext/psycho30.htm>
- [10] Pepperell, R. (2024). *Why science can't explain consciousness scientifically*. [online] Available at: <https://medium.com/what-matter-feels/why-science-can-t-explain-consciousness-scientifically-acd5e79bc2>
- [11] Psychology Today. (2023). *An Overview of the Leading Theories of Consciousness*. [online] Available at: <https://www.psychologytoday.com/ca/blog/finding-purpose/202308/an-overview-of-the-leading-theories-of-consciousness>.
- [12] Seager, W (1999). Panpsychism 1. What is Panpsychism?. Available at: https://www.utsc.utoronto.ca/~seager/pan_seager.pdf.
- [13] Weisberg, J. (2024). *Hard Problem of Consciousness* | Internet Encyclopedia of Philosophy. [online] Internet Encyclopedia of Philosophy. Available at: <https://iep.utm.edu/hard-problem-of-consciousness/>

DISEASES & DISORDERS



The Fight Against ALS: The Role of Artificial Intelligence in Early Diagnosis and Drug Discovery

Angela Li

Abstract

Imagine waking up one morning with a cramp in your hand. At first, you dismiss it—it's just fatigue or a minor cramp. It can't be too serious, right? But then, days turn into weeks, and the weakness begins to spread throughout your body. Daily tasks like buttoning a shirt or holding a pen become progressively more difficult. Eventually, breathing becomes a struggle as the paralysis spreads to the lungs. This is the devastating reality of Amyotrophic Lateral Sclerosis (ALS), a fatal neurodegenerative disease. ALS targets alpha motor neurons (neurons that control muscles), while leaving the rest of the brain intact. Thus, most patients are fully conscious of their disease as their muscles wither away. Currently, there is no cure for ALS; however, recent advancements in Artificial Intelligence (AI) have shed hope on this topic. AI has the potential to detect ALS earlier in patients, provide accurate diagnoses, and even accelerate drug discovery.

What Is ALS?

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease afflicting up to 5000 patients each year in the United States. The disease can affect people at any age but most commonly between the ages of 55 and 75 [1]. What starts off as minor cramps or weakness spreads to the entire body, leading to total paralysis. Within two to five years, a patient will lose their ability to breathe as the paralysis spreads to respiratory muscles. Paralysis begins with alpha motor neurons—neurons found in the brain and spinal cord. Each alpha motor neuron controls a set of muscle cells, called muscle spindles [2]. As these neurons die, muscles will stop receiving signals, causing them to waste away in a process called muscular atrophy.

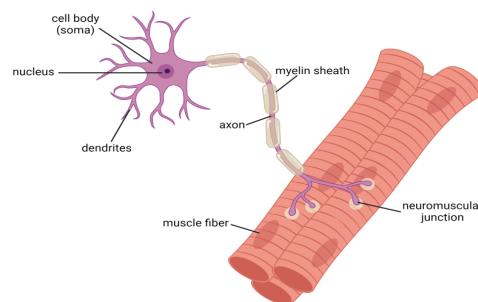


Figure #1. Image of an alpha motor neuron controlling a muscle [2]

Causes

About 10% of ALS cases are caused by genetic mutations, known as “familial ALS”. The genes SOD1 and C9orf72 have been linked to ALS [3]. The gene SOD1 produces an enzyme, superoxide dismutase 1, which breaks down free radicals, a toxic byproduct of cellular metabolism [4]. When SOD1 is mutated, the enzyme will not break down free radicals as effectively. Free radicals accumulate, and the buildup of these molecules is believed to kill motor neurons. The C9orf72 gene has a similar theory: A gene mutation in C9orf72 causes the production of a harmful protein known as a dipeptide repeat protein. The defective proteins build up in the cell body, killing the neurons [5]. These genes are only associated with ALS [6]. The definitive cause of ALS is still unknown, and further research must be done.

The other 90% of ALS cases are sporadic, meaning they randomly occur with no genetic linkage. Some studies suggest that environmental factors may play a role. For example, veterans are 2.5 times more likely to develop ALS, possibly because of exposure to toxic chemicals [7]. Additionally, viral infections, intense physical exertion, and smoking have also been investigated as potential risk factors. Men are more likely to develop ALS than women, suggesting that gender may also contribute to disease risk. However, the reasons remain unclear.

Treatment:

While there is no way to stop or reverse ALS, some drugs can dampen the symptoms and improve one’s quality of life. The FDA has approved the following drugs for ALS patients: Riluzole, Edavarone, and Tofersen (Qalsody). Riluzole is an oral medication that reduces the levels of glutamate, a neurotransmitter that carries messages through the brain. Researchers believe that too much glutamate is toxic because it over stimulates the cell — a condition known as excitotoxicity. Thus, reducing glutamate may slow down the death of alpha motor neurons. Clinical trials have shown that Riluzole may lengthen a patient’s life by a few months. Edaravone is another option available to ALS patients. Instead of neurotransmitters, this drug targets free radicals by neutralizing them—turning these harmful, unstable molecules into more stable, safer ones. Edaravone may slow functional decline in some patients. Finally, Tofersen (Qalsody) is an injection delivered into a patient’s spinal cord. This drug is given to ALS patients with an SOD1 mutation. Tofersen binds to the mRNA—a messenger that carries instructions for protein production—in the SOD1 gene, preventing the cell from making more faulty SOD1 protein. The benefits of this drug are still unclear, and more research is needed [1].

The Challenges of ALS in Diagnosis and Treatment:

ALS is notoriously difficult to treat for a variety of reasons. There are a multitude of genetic mutations that are linked to this disease. One treatment may address *one* gene related to ALS, but what about all the *other* genes and the complex role each gene plays? Once a treatment is found, it must cross the blood-brain barrier that protects the brain and spinal cord. The challenge of this barrier further limits treatment options.

ALS is especially hard to diagnose since it requires a diagnosis of exclusion. Doctors must rule out other diseases—like multiple sclerosis, spinal muscular atrophy, or other neurodegenerative diseases—which share overlapping symptoms with ALS. There is no single test that can diagnose ALS. Traditional diagnosis is a long process that involves examining the patient's medical record; MRIs, which take pictures of the brain; an electromyography (EMG), a test that measures muscle activity; and a nerve conduction velocity test (NCV) [8]. By the time they receive their diagnosis, it would be too late: significant neuron loss would have already occurred, and the patient may be beyond the window for intervention.

The Impact of AI

Despite these challenges, artificial intelligence (AI) remains a promising field of research. AI has the potential to transform the way we treat and diagnose diseases. In a Mayo Clinic study, an AI-assisted screening tool was found to be more accurate than a mammogram when diagnosing left ventricular dysfunction, a heart disease. The AI detected patients at risk of the disease 93% of the time compared to a mammogram's 85% [9].

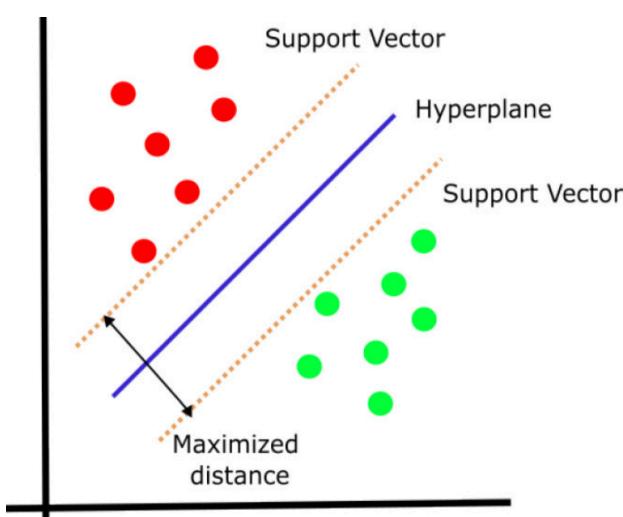


Figure #2 Support Vector Machine graph [10]

AI could likely diagnose neurodegenerative diseases too. Through a process called machine learning, AI can be trained to provide earlier and more accurate diagnoses. Unlike humans, AIs can analyze millions of patient cases at once. Using this enormous data set, the AI uses an algorithm to predict patterns. One such algorithm is called a Support Vector Machine (SVM), which aims to maximize the margin between data sets [10]. Once the millions of patient records are scanned into the AI, it is classified into two groups, “No ALS” and “Has ALS”, and plotted on a graph.

During machine learning, the Support Vector Machine draws a boundary line, called a hyperplane, that maximizes the distance between the hyperplane and the closest data points from both groups (as seen in Figure 2). Through constant adjustments and refinements, the AI becomes more accurate at distinguishing ALS from non ALS patients. After this process, the AI would be ready to analyze new patient data, determining which side—ALS or non ALS—of the hydroplane the patient falls.

AI could also be used to accelerate drug discovery for ALS. Utilizing AI's capacity to analyze massive data sets, AI could potentially transform drug testing. Using its machine learning models, AI could scan vast chemical libraries. On average, developing a new drug takes 12 years and billions of dollars [ii], but AI could significantly cut this time.

Conclusion

Amyotrophic Lateral Sclerosis remains one of the most devastating diseases today. Its sporadic nature, genetic complexity, and difficulty to diagnose leaves treatment challenging. Although more research is needed, AI represents a powerful force in the fight against ALS, bringing us closer to a cure.

Reference

- [1] Marin, B., Boumèdiène, F., Logroscino, G., et al. (13/05/2016). Variation in worldwide incidence of amyotrophic lateral sclerosis: a meta-analysis. International Journal of Epidemiology, 46(1), 57-74. <https://academic.oup.com/ije/article/46/1/57/2617177>. Retrieved: 07/03/2025.
- [2] Society for Neuroscience. Brain Facts Book. (2018). https://www.brainfacts.org/-/media/Brainfacts2/Brain_Facts-Book/Brain-Facts-Book-2018-high-res.pdf. Retrieved: 07/03/2025.
- [3] Amyotrophic Lateral Sclerosis Association. (2023). ALS Genes and Mutations. <https://www.als.org/research/als-research-topics/genetics>. Retrieved: 07/03/2025.
- [4] Amyotrophic Lateral Sclerosis Association. (25/04/2023). Tofersen.. <https://www.als.org/navigating-als/living-with-als/fda-approved-drugs/tofersen#:~:text=Mutations%20in%20he%20SOD1%20gene%20are%20thought%20to%20cause%20the,in%20ALS%20development%20and%20progression>. Retrieved: 07/03/2025.
- [5] National Center for Biotechnology Information.
- [7] Beghi, E., Logroscino, G., Chio, A., et al. (09/09/2020). Military service and related risk factors for amyotrophic lateral sclerosis. Acta Neurologica Scandinavica, 143(1), 39-50. <https://onlinelibrary.wiley.com/doi/10.1111/ane.13345>. Retrieved: 07/03/2025.
- [8] MedlinePlus. (10/04/2024). Electromyography (EMG) and Nerve Conduction Studies. <https://medlineplus.gov/lab-tests/electromyography-emg-and-nerve-conduction-studies/>. Retrieved: 07/03/2025.
- [9] Jiang, F., Jiang, Y., Zhi, H., et al. (19/12/2017). Artificial intelligence in healthcare: past, present and future. Stroke and Vascular Neurology, 2(4), 230-243. <https://svn.bmjjournals.org/content/2/4/230>. Retrieved: 07/03/2025.
- [10] Geeks forgeeks (27/01/2025). Support Vector Machine (SVM) Algorithm. <https://www.geeksforgeeks.org/support-vector-machine-algorithm/>. Retrieved: 07/03/2025.
- [ii] Congressional Budget Office. (01/04/2021). Research and

(08/01/2015). C9orf72 Frontotemporal Dementia and/or Amyotrophic Lateral Sclerosis.
<https://www.ncbi.nlm.nih.gov/books/NBK268647/>.
Retrieved: 07/03/2025.

[6] Kwiatkowski, T. J., Jr., Bosco, D. A., Leclerc, A. L., et al. (27/02/2009). Mutations in the FUS/TLS Gene on Chromosome 16 Cause Familial Amyotrophic Lateral Sclerosis. *Science*, 323(5918), 1208-1211.
<https://www.science.org/doi/10.1126/science.1166066>.
Retrieved: 07/03/2025.

Development in the Pharmaceutical Industry.
<https://www.cbo.gov/publication/57126>. Retrieved: 07/03/2025.

[12] University of Utah. (2024). Motor Neuron Anatomy. *Utah Education Network*.
<https://uen.pressbooks.pub/app/uploads/sites/382/2024/03/U12-012-Motor-Neuron-anatomy-1024x717.png>. Retrieved: 07/03/2025.

Huntington's Disease: Innovative Computational Diagnosis Methods

Kaushik Tatiraju

Abstract

Huntington's Disease (HD) is a genetic neurological disorder, meaning that it affects the nervous system of the complex human body. It is characterized as one of the diseases with the most upcoming treatments. It has major potential in the future due to recent advancements in artificial intelligence and machine learning. Such inventions have the potential in helping with improving diagnosis rates, partially because of the amount of misdiagnosed individuals, where symptoms are not properly characterized and associated with this disease, or undiagnosed, where the symptoms of the disease go unnoticed until later in the development of this disease. This article looks at various scientific studies and upcoming computational diagnostic methods utilizing artificial intelligence and machine learning to help increase the accuracy and efficiency of diagnosis for HD. Specifically, this article will focus on HD prognosis and development, given the addition of upcoming artificial intelligence tools and resources. Artificial intelligence plays a large extent in the improvement of diagnosis rates, as machines can be created and trained to recognize the symptoms of Huntington's Disease, and accurately detect it in upcoming patients. With the rise of artificial intelligence comes new innovations, as demonstrated by Neuralink, a company which created a chip that can be inserted into a human brain and control functions of the brain that are conventionally controlled by the nervous system of the body. Recent innovations, like Neuralink, make the promise of novel innovations utilizing AI to find accurate diagnosis methods for HD even greater.

Current Symptoms and Diagnosis

As Huntington's Disease is a neurological disorder, it affects the body's ability to transfer messages throughout the body, or, in other words, there is a miscommunication between different parts of the body. HD impacts the nervous system, specifically the autonomic nervous system, which controls all the involuntary functions of the body, such as breathing [1]. The nervous system is split into two parts that have various other subsets within them: the central nervous system, consisting of the autonomic system, which can be further reduced into the sympathetic and parasympathetic nervous system; and the peripheral nervous system [2][3]. The main unit of the nervous system is a neuron or nerve cell, consisting of many different structures, as shown in Figure 1 below [3]. Huntington's Disease impacts the locomotive actions of the human body, which can cause issues with movement of impacted and affected individuals [4]. Obviously, since HD is neurological, this disorder is also characterized by several mental and neurological symptoms.

The traditional and most widely used method of diagnosis for this disease is genetic testing. This is conducted by checking the DNA of an individual for numerous CAG (Cytosine, Adenine, Guanine) amino acid sequences on a DNA strand, as this strand appears on the gene that is known to cause Huntington's Disease itself: huntingtin. Yet, like all disorders, Huntington's disease when diagnosed may have some complications, and according to the article "Huntington's disease: diagnosis and management," these complications are tending to become more widespread among patients in recent years [4]. These complications lead to the necessity of more practical and accurate diagnosis methods, where artificial intelligence can play a big role.

Promising Diagnosis Methods

Especially with the severity of Huntington's Disease, it is essential and would benefit the lives of many affected individuals if more efficient diagnosis methods were found. One such diagnosis method that is currently being tested is recognizing and utilizing biological indicators that are commonly associated with HD. These markers, being biological indicators, are essential as it is probably for these indicators to appear in more or all HD victims. Therefore, this would allow for future patients to be tested for those indicators, whether it be through MRI scans or other methods, and, thus, make a conclusion on the identity of the disease. The biological indicators that are currently being researched show up in scans of the brain, and it has demonstrated novel information to scientists, such as the fact that the brain begins to lose function even before Huntington's disease is diagnosed and characteristics of HD start to appear; it continues to deteriorate well into the main phase of HD. These indicators hopefully allow for more data to be collected and studies to be conducted in order to advance the research being done for diagnosis methods and treatments for Huntington's disease, such as through different treatment methods for different individuals based on their unique circumstances with the disease [5].

Yet, while biological indicators definitely have potential, it is likely that Artificial Intelligence and Machine Learning (AI/ML) will have more capabilities in the medical field, especially in obtaining these novel diagnostic methods and treatment options. AI/ML allows for a computer / machine to be trained, usually by mass amounts of data, to the point where it can utilize the data to teach itself, and help it find patterns. If it is possible for a computer to obtain the patient data of individuals who have HD, it could be programmed so that it could possibly find new patterns that humans have not yet discovered, and would detect the disease more accurately so that earlier treatment can be given. According to the article "Exploring Huntington's Disease Diagnosis via Artificial Intelligence Models: A Comprehensive Review", in the future, AI might be able to go through specific data, such as scans and hereditary tests, and make accurate diagnoses using this information [6].

New Findings

As novel inventions and concepts start to arise in the world, modifications continue to be made in the field of medicine. The importance of Artificial Intelligence has been shown in past inventions, but the most recent and upcoming inventions tend to utilize AI and technology more than ever.

One such invention is talked about in the article, “A biological classification of Huntington’s disease: the Integrated Staging System”. In this article, a new software, the Huntington’s Disease Integrated Staging System has been created, using technology and AI, to obtain information about patient factors, especially related to their past medical history. In essence, the software assigns patients a specific stage, from zero to four, based on their level of development of the presence of Huntingtin in the patient. This new system is a key example of Artificial Intelligence and technology being used, as the system itself is able to diagnose and detect an individual’s specific circumstances in regards to HD [7].

In addition, another article, titled “Neuroinflammatory Proteins in Huntington’s Disease: Insights into Mechanisms, Diagnosis, and Therapeutic Implications”, states how various amino acids were detected in patients and affected organisms in experiments on various creatures and parts of organs of patients. It has also been researched and found that specific body systems were found to be activated because of Huntington’s Disease, such as peripheral aspects of the lymphatic system of the human body [8].

Lastly, recent research has been done on eyesight effects caused by Huntington’s. One recent study, talked about in the article, “Huntington’s disease and neurovascular structure of retina”, had the idea of assessing structures of the eye, and checking if they were indicative of Huntington’s disease. In this study, participants were asked and checked about the severity of their disease, and various different tests were performed to check for the depth of a fiber in the retina structure of the eye. The study found that individuals who had Huntington’s disease had a decreased depth of a specific fiber in the eye, which they concluded might be helpful in identifying victims of HD in upcoming years. This study opens the gate for future, more in-depth, studies to be conducted on possible indicators of the sensory system for Huntington’s disease [9].

All of these new findings show the capabilities of new technology, such as detection programs and tests for HD, and how it is important to continue to utilize diagnosis methods in Huntington’s disease, and other neurological disorders as well [7] [9].

Conclusion

As the population of the Earth grows, more and more individuals are impacted and affected by various neurological diseases, including Huntington’s disease. Many of these individuals are not able to get the treatment that is essential for their survival, which is a separate issue. However, many of the individuals who have access to treatment don’t get it, due to the fact that they don’t even know of the disease inside of them. It would benefit everyone if consistent and accurate diagnosis methods were created, that would allow everyone to know and get the information they need. Therefore, it is essential

that individuals take control of the upcoming tools and resources that are available to us, like Artificial Intelligence, and use it to create a better world, with healthier individuals. It is important to use AI, and further the research and inventions in the field of neuroscience, especially to help improve the lives of neurologically affected individuals by conducting research on Huntington's disease, as well as other neurological disorders.

References

- [1] Kobal, Jan et al. (2014). Cognitive and autonomic dysfunction in presymptomatic and early Huntington's disease. *Journal of neurology*. <https://doi.org/10.1007/s00415-014-7319-6>. Retrieved: 12/15/2024
- [2] Wehrwein, Erica et al. (06/13/2016). Overview of the Anatomy, Physiology, and Pharmacology of the Autonomic Nervous System. *Comprehensive Physiology*. <https://doi.org/10.1002/cphy.c150037>. Retrieved: 12/15/2024
- [3] What are the parts of the nervous system?. National Institute of Child Health and Human Development. <https://www.nichd.nih.gov/health/topics/neuro/conditioninfo/part>. Retrieved: 12/15/2024
- [4] Stoker, Thomas et al. Huntington's disease: diagnosis and management. *Practical Neurology*. <https://doi.org/10.1136/practneurol-2021-003074>. Retrieved: 12/15/2024
- [5] Ross, Christopher et al. (2014). Huntington disease: natural history, biomarkers and prospects for therapeutics. *Nature reviews. Neurology*. <https://doi.org/10.1038/nrneurol.2014.24>. Retrieved: 12/15/2024
- [6] Ganesh, Sowmyalakshmi et al. (12/03/2023). Exploring Huntington's Disease Diagnosis via Artificial Intelligence Models: A Comprehensive Review. *Diagnostics*. <https://doi.org/10.3390/diagnostics13233592>. Retrieved: 12/15/2024
- [7] Tabrizi, Sarah et al. (2022). A biological classification of Huntington's disease: the Integrated Staging System. *The Lancet, Neurology*. [https://doi.org/10.1016/S1474-4422\(22\)00120-X](https://doi.org/10.1016/S1474-4422(22)00120-X). Retrieved: 12/18/2024
- [8] Li, Xinhui et al. (11/02/2024). Neuroinflammatory Proteins in Huntington's Disease: Insights into Mechanisms, Diagnosis, and Therapeutic Implications. *International journal of molecular sciences*. <https://doi.org/10.3390/ijms252111787>. Retrieved: 12/18/2024
- [9] Amini, Elahe et al. (2022). Huntington's disease and neurovascular structure of retina. *Neurological sciences: official journal of the Italian Neurological Society and the Italian Society of Clinical Neurophysiology*. <https://doi.org/10.1007/s10072-022-06232-3>. Retrieved: 12/18/2024

The Protective Role of REELIN in Alzheimer's Disease: Exploring New Genetic Insights

Arihaan Mallick

Abstract

Alzheimer's disease (AD) is a neurodegenerative disorder characterized by the progressive accumulation of amyloid plaques and tau tangles in the brain. These pathological markers contribute to the decline in cognitive functions, particularly memory and learning. While numerous studies have focused on understanding the mechanisms behind these protein aggregates, recent findings suggest that genetic factors may influence an individual's vulnerability or resilience to AD. One such factor is the REELIN protein, which plays a critical role in neuronal development, synaptic function, and brain plasticity. Mutations in REELIN have been implicated in several neurodevelopmental disorders, including autism and schizophrenia. However, a recent breakthrough has identified a rare mutation in the REELIN gene, termed REELIN-COLBOS, that appears to offer significant protection against Alzheimer's disease. This mutation increases the function of the REELIN protein, enhancing its neuroprotective effects and mitigating the effects of tau pathology in the brain.

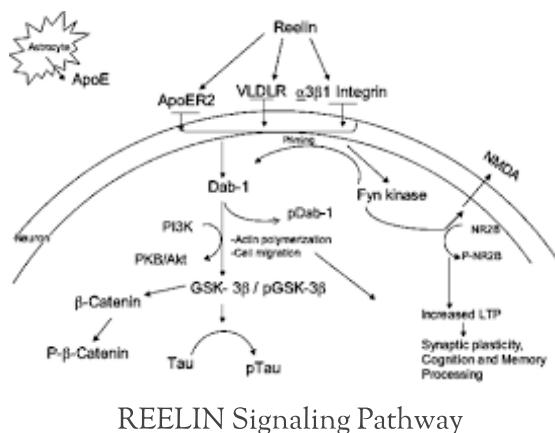
Molecular Mechanisms of REELIN in Alzheimer's Pathology

One of the most significant ways in which REELIN is thought to affect AD pathology is through its regulation of tau phosphorylation. Tau, a microtubule-associated protein, stabilizes microtubules in neurons. However, in Alzheimer's disease, tau becomes hyperphosphorylated and aggregates into neurofibrillary tangles. These tangles are one of the hallmark features of AD and contribute to neuronal dysfunction and death.

REELIN influences tau phosphorylation through its interaction with specific receptors on neuronal cell membranes, such as ApoER2 and VLDL receptors. These receptors are involved in the REELIN signaling pathway, which activates a downstream cascade that modulates the activity of kinases responsible for tau phosphorylation. In particular, the binding of REELIN to these receptors inhibits the activation of GSK-3 β (glycogen synthase kinase 3-beta), a kinase that is known to phosphorylate tau at several critical sites. By preventing GSK-3 β activation, REELIN reduces tau phosphorylation, thereby preventing the formation of tau tangles [i].

However, when REELIN signaling is disrupted, either through mutations in the REELIN gene or loss of receptor function, tau becomes more susceptible to hyperphosphorylation. Studies

have shown that in AD patients, the expression of REELIN is often decreased in areas of the brain affected by tau pathology, such as the entorhinal cortex and hippocampus. This loss of REELIN function accelerates tau aggregation and the subsequent neurodegenerative processes in AD.



REELIN and Amyloid Beta (A β) Plaques

Although tau pathology is central to AD progression, the accumulation of amyloid-beta (A β) plaques also plays a critical role in the disease. A β plaques are composed of small peptides derived from the amyloid precursor protein (APP), which aggregate into plaques that disrupt neuronal function and trigger inflammatory responses. The relationship between REELIN and A β pathology is less direct than its role in tau phosphorylation, but recent studies suggest that REELIN may influence A β deposition indirectly through its effects on neuronal signaling and synaptic function.

Research indicates that REELIN interacts with A β by modulating the trafficking and processing of APP. In particular, REELIN may influence the clearance of A β from the extracellular space by regulating the activity of certain receptors that mediate the uptake and degradation of A β . In mouse models of AD, enhanced REELIN signaling has been shown to reduce A β plaque deposition, suggesting that boosting REELIN activity might help mitigate one of the defining characteristics of AD pathology [2].

Moreover, the REELIN-COLBOS mutation, a variant that increases REELIN activity, has been linked to a reduced burden of both tau tangles and A β plaques in certain individuals, further supporting the hypothesis that REELIN may exert protective effects against amyloid-related neurodegeneration. This mutation has been associated with cognitive resilience in individuals who carry the genetic predisposition for early-onset AD, despite high amyloid levels in the brain.

Case Studies and Experimental Data

One of the most compelling cases comes from a Colombian man who carried the PSEN1-E280A mutation, which is strongly linked to early-onset familial Alzheimer's disease. Despite this genetic predisposition, the individual remained cognitively healthy until his late 60s, well beyond the typical onset age for carriers of the mutation.

Detailed neuroimaging and biomarker analysis revealed that while his brain exhibited a high burden of amyloid plaques, the tau pathology, particularly in the entorhinal cortex, was significantly reduced. Genetic analysis identified a previously unknown mutation in the REELIN gene, termed REELIN-COLBOS, which enhances the protein's function. This mutation was shown to mitigate tau pathology in key brain regions responsible for memory and cognition [3].

Interestingly, the REELIN-COLBOS case has parallels with a previously studied case of a woman carrying the APOE₃-Christchurch mutation, another rare genetic variant associated with cognitive resilience in AD. Both cases highlight the importance of specific genetic mutations in modulating the progression of Alzheimer's pathology. While the APOE₃-Christchurch variant primarily affects amyloid-related pathways, the REELIN-COLBOS mutation appears to act predominantly through tau-related mechanisms.

Mice with enhanced REELIN expression or carrying the REELIN-COLBOS mutation exhibited significantly lower levels of tau tangles in the entorhinal cortex compared to control mice. Moreover, these mice showed preserved cognitive function in behavioral tests, such as maze navigation and memory recall tasks, even when exposed to high levels of amyloid plaques [3].

Implications for Therapeutics

One of the most direct therapeutic approaches involves enhancing REELIN activity to mimic the effects of the REELIN-COLBOS mutation. Gene therapies that upregulate REELIN expression in the brain could offer a way to bolster the protein's protective effects. Alternatively, small molecules or biologics could be designed to activate REELIN's downstream signaling pathways, thereby promoting its neuroprotective effects without directly increasing protein levels.

Given the multifactorial nature of Alzheimer's disease, therapies targeting REELIN could be combined with other approaches to maximize their efficacy. Current drugs that reduce amyloid plaque accumulation, such as monoclonal antibodies targeting amyloid-beta, could be paired with treatments enhancing REELIN activity to provide synergistic benefits [4].

Conclusion

The discovery of the REELIN-COLBOS mutation and its ability to protect against Alzheimer's pathology marks a significant breakthrough in our understanding of the genetic factors that contribute to resilience against neurodegeneration. Enhancing REELIN function offers hope

for new preventative and therapeutic strategies, particularly for individuals with a genetic predisposition to the disease.

References

- [1] Lopera, Francisco et al. (16/05/2023). REELIN-COLBOS variant in autosomal dominant Alzheimer's. *Nature Medicine*. <https://doi.org/10.1038/s41591-023-02318-3>. Retrieved: 09/12/2024.
- [2] Arboleda-Velasquez, Joseph F et al. (2023). Resilience to Alzheimer's Disease in a Reelin-COLBOS Heterozygous Man. *Nature Medicine*. <https://doi.org/10.1038/s41591-023-02318-3>. Retrieved: 09/12/2024.
- [3] Tsai, Li-Huei et al. (2023). Neural Vulnerabilities and Resilience in Alzheimer's Disease Explored. *MIT News*. <https://news.mit.edu/2023/neural-vulnerabilities-resilience-alzheimers-0516>. Retrieved: 09/12/2024.
- [4] Sepulveda-Falla, Diego et al. (2023). Genetic variants of Reelin and their implications in Alzheimer's Disease. *ALZFORUM*. <https://www.alzforum.org/news/research-news/reelin-variant-protects-against-alzheimers>. Retrieved: 09/12/2024.

Alzheimer's Memory Decline and Daily Activities

Hafsa Ali

Abstract

Alzheimer's is a progressive disease that destroys memory and other important mental functions. It is the most common cause of dementia — a gradual decline in memory, thinking, behavior and social skills, affecting a person's ability to function[1]. The purpose of the study is to explore the link between daily activities in people with Alzheimer's disease, and cognitive decline. Data were analyzed from Alzheimer's disease

Neuroimaging Initiative, in participants who had biomarker confirmed AD. Longitudinal regression modeling was used to examine the relationship between memory decline and the lifestyle of an individual with Alzheimer's. I found that overall difficulties in daily living may not strongly predict cognitive outcomes, changes in specific activities may become more or less significant over time. In this study, time is a much stronger factor in memory decline than difficulties in daily living activities. FAQ variables have a very small, non-significant effect on cognitive function at the start of the study and over time. The general trend is a slight decline in cognitive function over time, but the effect is small. The interactions between FAQ variables and time are also very small and not statistically significant, indicating that the relationship between daily living difficulties and cognitive decline does not change as much as the disease.

Introduction

The purpose of this study is to find if people who work, travel, engage their mind with puzzles and games have a higher/lower rate of memory decline compared to those who don't. I also want to see if years of education have any correlation with Alzheimer's disease and the speed at which memory declines for individuals having different lifestyles. It is essential to understand this relationship to have early targeted interventions to help delay the rate of memory decline.

So far we know that Alzheimer's disease is a brain disorder that slowly destroys memory and thinking skills, and eventually, the ability to carry out the simplest tasks[2] and a person is likely to need help with washing, brushing their teeth and taking care of their hair, hands and fingernails[3]. My study explores the effect that different daily living activities have on the rate of memory decline

Therefore the purpose of this scientific paper is to explore the link between memory

decline and the lifestyle of an individual with Alzheimer's.

Materials and Methods

The Alzheimer's Diseases Neuroimaging Initiative is a longitudinal, multi-center, observational study whose goal is to validate biomarkers for Alzheimer's disease (AD) clinical trials[4]. For this study, ADNI data sets were used that comprise clinical information about each subject including recruitment, demographics, physical examinations, and cognitive assessment data. The measures used included Neuropsychological Battery, activities of daily living measures, Neuropsychological Functional Activities Questionnaire, Logical Memory - Immediate Recall and Logical Memory - Delayed Recall.

Data was used from Alzheimer's disease neuroimaging initiative which included activities of daily living measures, Neuropsychological Functional Activities , neuropsychological battery and Adnimerge data. MOCA and MMSE were used as the memory variables in this study.These measures have previously been described in the ADNI protocol (cite ADNI protocol publication)).

ADNI data was filtered by removing anyone without clinically or pathologically diagnosed Alzheimer's Disease and individuals without neuropsych data were also removed. R was used to code and analyze the data over time.

The Regression model was used for Combined Memory Score and All FAQ Variables.

Heat maps were also used. The heatmap shows the correlation coefficients between each FAQ variable and the combined MMSE/MoCA and memory scores.Red squares indicate a positive correlation, meaning that as the FAQ score increases, so does the cognitive decline measure.Blue squares indicate a negative correlation, meaning that as the FAQ score increases, the cognitive decline measure decreases. White squares indicate no correlation. This heatmap helps identify which FAQ variables are most strongly related to cognitive decline, guiding further analysis or intervention strategies.

Results

Figure 1 was used to show changes over time and how it is related to difficulties in daily living activities (as measured by FAQTOTAL).The average combined memory score starts at approximately 3.9 when FAQTOTAL and Visit Number are both at their starting points (FAQTOTAL = 0, Visit Number = 1). FAQTOTAL (-0.001): The negative estimate suggests that as FAQTOTAL (indicating higher difficulty in daily activities) increases, the combined memory score slightly decreases. However, this relationship is very weak and not statistically significant (as shown by the t-value of -0.555). The negative visit number of -0.622 estimate indicates that as the visit number increases, which represents the progression of time, the combined memory

score decreases significantly. This suggests that memory tends to worsen over time.

Figure 2 analyzed how the combined cognitive severity score (from MMSE and MoCA) changes over time and how it relates to FAQTOTAL.

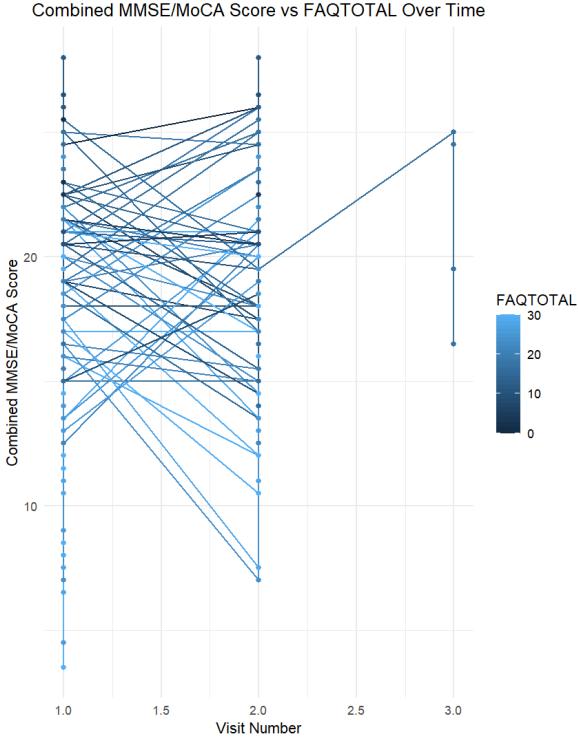


Figure 2. combined MMSE/MoCA Score Model

cognition.

Figure 3 shows the correlation coefficients between each FAQ variable and the combined MMSE/MoCA and memory scores and whether these relationships change as time progresses. For the longitudinal mixed effects model, the intercept (19.71) represents the average combined MMSE/MoCA score when all FAQ variables and Visit Number are at their baseline (typically when FAQ variables are at their mean, and Visit Number is 1). Each FAQ variable represents a different aspect of daily living. For instance, FAQFINAN relates to financial management, FAQFORM to filling out forms, etc. The coefficients for these variables indicate how much they influence the combined cognitive

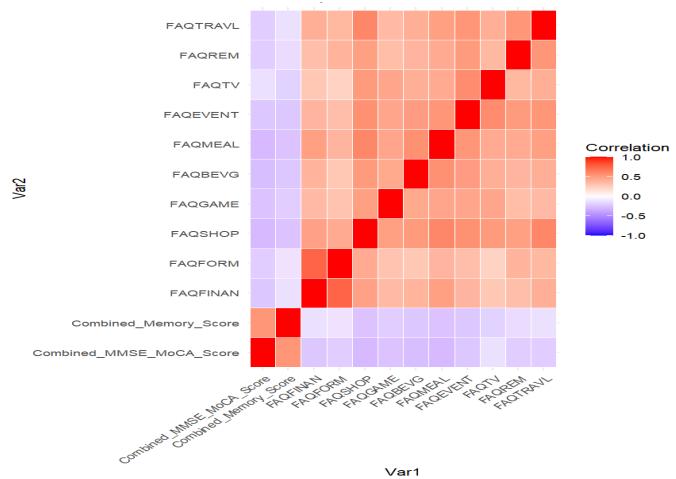


Figure 3. Heatmap of correlations between FAQ variables

score at the participants' baseline visit (Visit Number = 1). Negative coefficients like FAQFINAN = -0.057 suggest that higher difficulty in this area of daily living is associated with a slight decrease in cognitive function. However, most of these effects are very small and not statistically significant. Visit Number (-0.126) is a coefficient that represents the general trend of cognitive function over time. Our model suggests that the impact of financial difficulties on cognitive decline might slightly increase over time, but this change is very small and not significant (correlation coefficient = 0.018). PTID (Intercept) variance (10.278) represents how much individual cognitive scores vary around the average score. The standard deviation of 3.206 indicates the standard deviation of these individual differences. The residual variance (4.943) represents the variation in cognitive scores that is not explained by the model.

Discussion

The purpose of this study was to explore the link between memory decline and the lifestyle of an individual with Alzheimer's disease. We analyzed data from the Alzheimer's Disease Neuroimaging Initiative (ADNI) database.

First, I examined the relationship between memory and global cognition over time. Memory, as measured by the RAVLT and WMS-IV Logical Memory, as well as global cognitive function, as measured by combined MMSE/MoCA scores, generally declined over time. This decline was consistent across multiple memory and global cognition measures, consistent with the progressive nature of Alzheimer's disease.

Second, regression models showed that FAQTOTAL (a summary measure of daily living activities) had a weak and non-significant relationship with both memory and global cognitive function. This suggests that overall difficulties in daily activities were not strongly predictive of cognitive decline in this sample. However, when looking at individual FAQ subscales (such as financial management, meal preparation, etc.), some specific aspects of daily living were significantly related to memory and cognitive function, both at participants' initial visit and over time. For example, financial management was positively associated with memory performance initially but became less impactful over time. This suggests that in early disease stages, memory may have a significant impact on individuals' ability to manage their finances. As the disease progresses and family members or friends take over financial management, less decline may be evident due to decreasing independence in activities of daily living.

Longitudinal models suggested that the relationship between certain daily activities and cognitive decline changes as the disease progresses. For example, the ability to travel became more important for memory performance over time, while the impact of other activities, like financial management, decreased.

The correlation analysis and resulting heatmap helped visualize which FAQ variables

were most strongly related to memory and cognitive decline. Some daily activities had positive correlations with cognitive decline like the ability to travel, shop and attend events, indicating that difficulties in these areas were associated with greater cognitive decline, while others had negative or no significant correlations like FAQTV and FAQGAME. Understanding this relationship is essential for intervention design, as some activities of daily living may be more important to target in early intervention. This may be especially important when designing interventions to provide caregiver support, as certain activities of daily living may require additional time and effort from the person with AD and their family carers. It is important to understand these results in the context of the sample from which the data were taken. This sample may not be fully representative of the general American public. This sample was primarily White (93.2%), which may not represent the actual prevalence of Alzheimer's disease in the general population. Black participants in Alzheimer's disease research studies were 35% less likely to be diagnosed with Alzheimer's or related dementias than white participants[5]. Previous studies have shown that for the overall U.S. population, Black Americans are roughly 1.5 to 2 times as likely as White Americans to develop Alzheimer's or related dementias[5].

The age distribution and education levels of the participants were also documented, which are essential in understanding cognitive decline. The average age of this sample was 74.7 years old ($SD=8$ years), which is consistent with the Alzheimer's Association's most recently reported prevalence report, which states, "An estimated 6.9 million Americans age 65 and older are living with Alzheimer's in 2024. Seventy-three percent are 75 or older"[6].

The average number of years of education in this sample was 15.8 years ($SD=2.49$ years), which is higher than the national average of 13.7 years.

In sum, while this sample likely reflects the typical age range of individuals living with Alzheimer's disease, this sample consists of primarily White, highly educated individuals, which may make these results less generalizable to a broader audience.

There are several limitations to this study. First, I only used verbal memory measures, not nonverbal/visual. MoCA and MMSE are both screening measures, not in depth indicator of cognitive function

Future research should consider nonverbal and visual memory measures as well.

Conclusion

This study highlights the importance of examining specific daily activities when understanding cognitive decline in Alzheimer's disease. While overall difficulties in daily living may not strongly predict cognitive outcomes, changes in specific activities may become more or less significant over time. This suggests that other factors beyond time and daily living difficulties may play a role in cognitive changes in individuals with Alzheimer's disease. This finding

underscores the need for targeted interventions that focus on specific areas of daily functioning to help manage cognitive decline in individuals with Alzheimer's disease.

References

- [1] (10/07/2024). Alzheimer's disease. Mayo Clinic. <https://www.mayoclinic.org/diseases-conditions/alzheimers-disease/symptoms-causes/syc-20350447>. Retrieved: 15/09/2024.
- [2] (05/04/2023). Alzheimer's Disease Fact Sheet. National Institute on Aging. <https://www.nia.nih.gov/health/alzheimers-and-dementia/alzheimers-disease-fact-sheet>. Retrieved 15/09/2024.
- [3] (06/2023). Activities of daily living. Alzheimer's Research UK. <https://www.alzheimersresearchuk.org/dementia-information/how-dementia-affects-everyday-life/activities-of-daily-living/>. Retrieved 15/09/2024.
- [4] About the Study. Alzheimer's Disease Neuroimaging Initiative. <https://adni.loni.usc.edu/>. Retrieved 15/09/2024.
- [5] (16/12/2021). Data shows racial disparities in Alzheimer's disease diagnosis between Black and white research study participants. National institute on aging. <https://www.nia.nih.gov/news/data-shows-racial-disparities-alzheimers-disease-diagnosis-between-black-and-white-research>. Retrieved: 10/6/2024.
- [6] (2024). Alzheimer's Disease Facts and Figures. Alzheimer's Association. <https://www.alz.org/media/Documents/alzheimers-facts-and-figures.pdf>. Retrieved: 12/07/2024.

Contributors Pages

IYNA EDITING TEAM:

Editor-In-Chief: Annie Pan

Managing Editor: Ashvin Kumar

Head of Outreach: Aleksandra Dubno

Head of Assembly: Riyaa Sri Ramanathan

Head of Events: Ananya Karthikeyan

Artist-in-Residence: Kavya Chintakayala

Senior Editors: Divyash Shah, Sai Snigdha Kodali, Vaishnavi Kode, Eesha Oza, Ishani Ghosh, Irene Zhang

Junior Editors: Youlan Li, Mary Zhang, Erin Yoo, Ashley Jing, Gayeong Kim, Xinxin Zhu, Faheem Alam, Abigail Molero, Vibha Yadav Ganji

Head of Journalists: Shrika Vejandla

Journalists: Ambalika Basak, Ellen Seo, Ashra Roshy, Katherine Carpio, Alex Kim

Head of Translation: Ana Beatriz Araujo

Translators: Sebastian Castro, María Fernanda Montiel Quiñones, Brianna Silva, Sofia Fothergill, Purva Sareen, Ipsita Adarsh, Sarthak Kamalkishor Dhole, Namish Balakerthy Punyakoti, GK Tejhaswini, Shokan Zhumadillayev, Sasha Bahdanava, Ayazhan Karimova, Samira Ageyeva

CONTRIBUTING AUTHORS:

Featured Writers: May Mourad, Jolin Cheng, Orly Galatin

Writers: Garv Pattani, Joshua Kim, Orly Galatin, Ashmi Parikh, Jolin Cheng, May Mourad, Alexa Marsh, Angela Li, Kaushik Tatiraju, Arihaan Mallick, Hafsa Ali

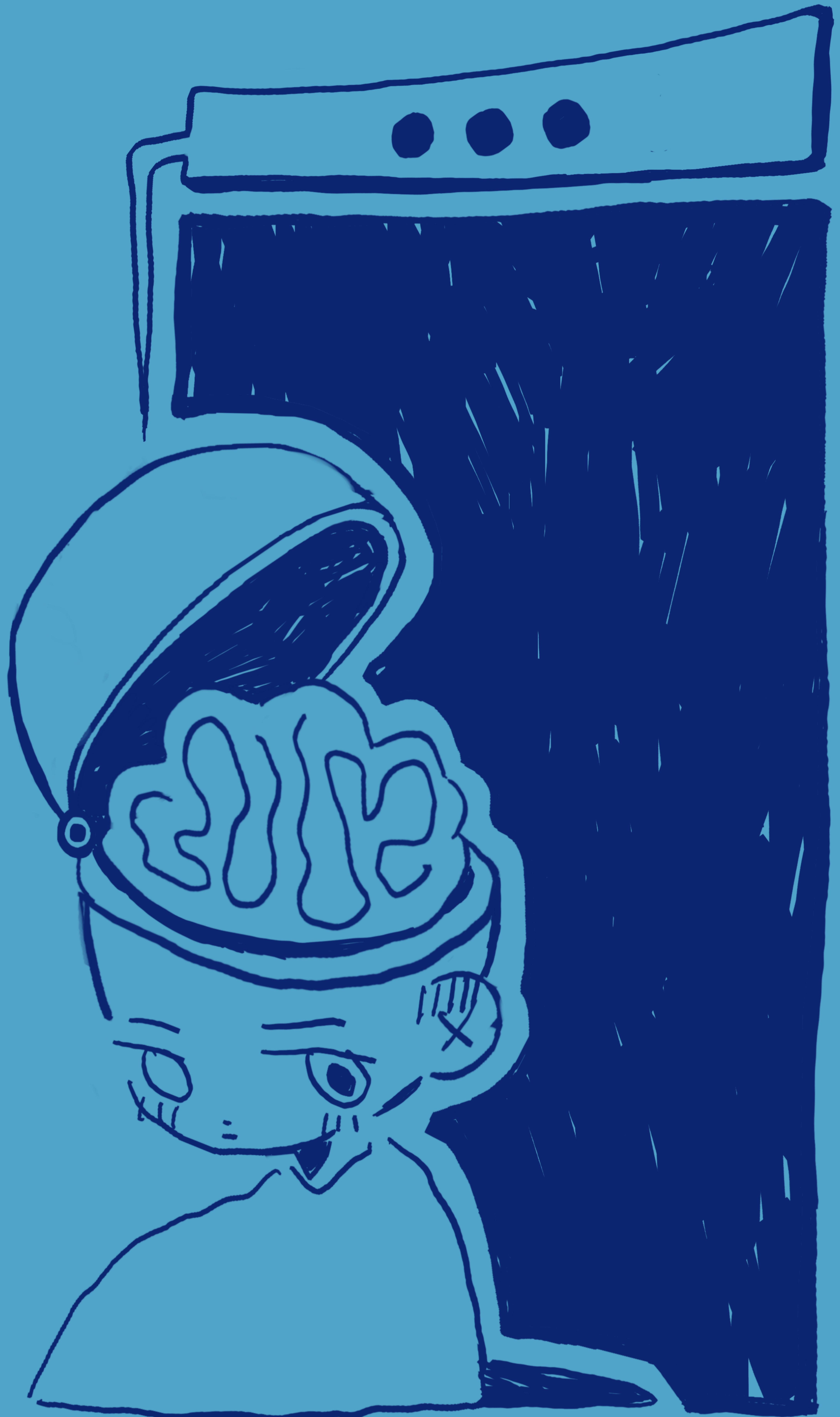
IYNA BOARD OF DIRECTORS:

Chief Executive Officers: Irene Zhang and Eesha Oza

Board Chair: Jacob Umans

ADVISORY BOARD:

Advisory Board Members: Dr. Norbert Myslinski, Dr. Olajide Williams, Dr. Jafri Abdullah,
Dr. Mark Hallett, Elaine Snell



© 2025 The International Youth Neuroscience Association