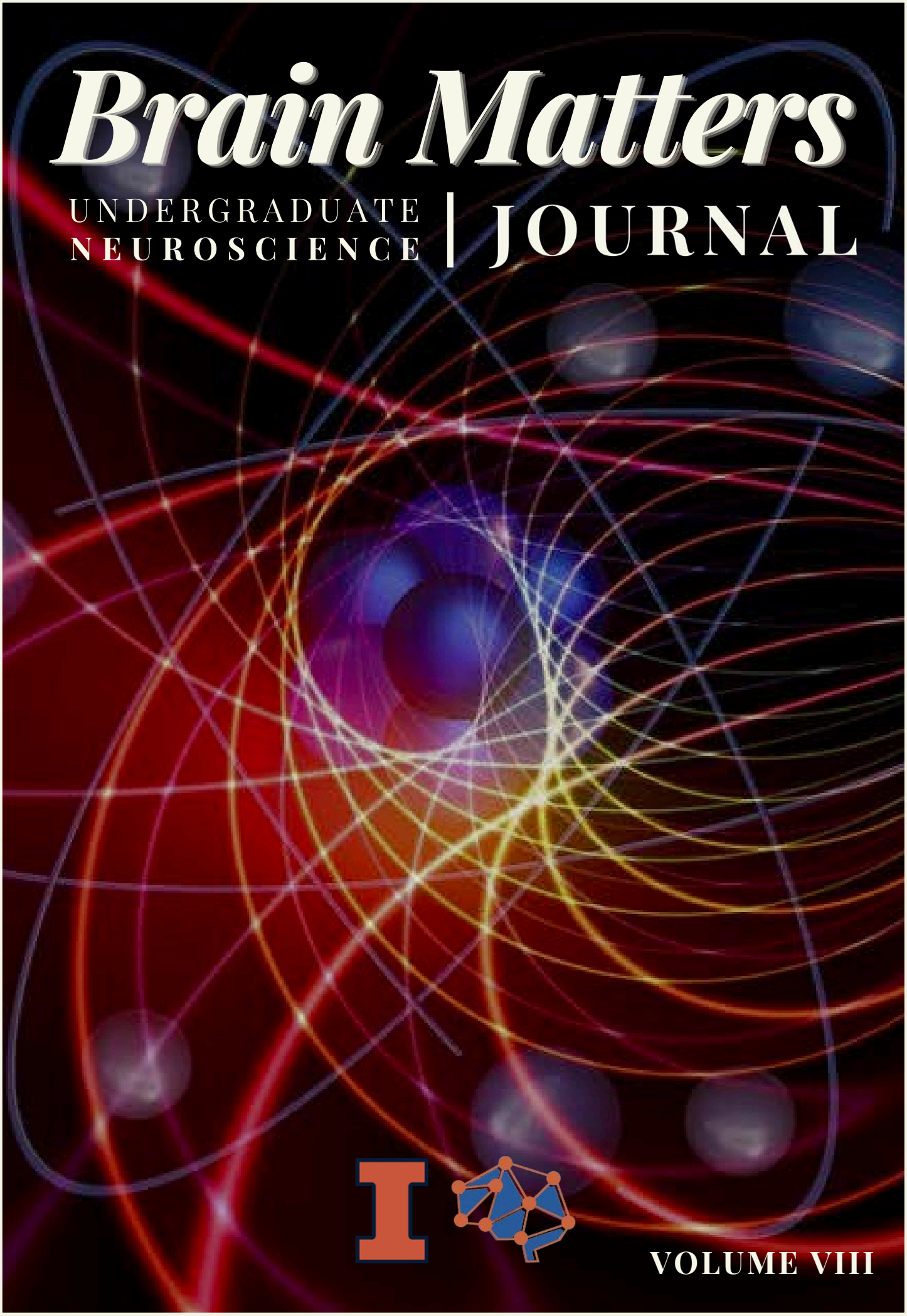
Brain Matters

UNDERGRADUATE NEUROSCIENCE | JOURNAL



VOLUME VIII

Table of Contents

Articles

About Brain Matters

Brain Matters discusses all things neuroscience, psychology, and biology written by UIUC's very own. The journal welcomes all authors no matter their area of study or year, Therefore, authors come from diverse backgrounds, from molecular and cellular biology & psychology, to computer science & engineering. This diversity allows volumes to have a wide range of articles. The journal is mainly written for the college community yet is accessible to anyone as Brain Matters is published in an Open Access format by the University Library at the University of Illinois Urbana-Champaign.

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To the Brain Matters writers, editors, & executive board members, as well as the University of Illinois University Library & Merinda Kaye Hensley for all of your hard work in making this journal possible.

Biological Neural Networks as the Forefront of AI Processing

Written by Edward Lin

Abstract

Graphic processing units (GPUs) are a major component of artificial intelligence (AI) processing power. As AI becomes more sophisticated and more processing power is needed to run these intellectual models, a growing concern of energy demand and extensive AI training becomes an increasing concern. To create more sophisticated machine learning algorithms, scientists in the field of neuromorphic computing studied the brain for its ability to efficiently process and store information. Finding a way to incorporate the brain directly into computing may create novel algorithms to meet the increasing demands of information processing.

Introduction

Graphic processing units (GPUs) are chips within a computer that process data simultaneously, performing parallel computations. Making approximately 36,500 calculations per second, GPUs consist of three major components that use basic arithmetic operations for neural network processing. The GPU's tensor core is the forefront of critical AI operations and AI learning, which uses geometric transformations and large matrix computations for AI neural network optimization. As science further progresses, we obtain more data about the world, which requires more processing power. This exponential growth in data requires increasingly more powerful processing capabilities to interpret this data. However, the current processing power of modern-day computational systems are inefficient and expensive, as they have a high energy and resource demand for running the GPUs and regulating their temperature as shown in Figure 1. Since the late 1980s, scientists and engineers have been studying the structural organization within the brain to help them optimize information processing, giving rise to the field of neuromorphic computing. The processing speed of the brain is similar to that of a supercomputer, and outperforms a supercomputer in terms of energy efficiency, spatial optimization, memory, and storage. Rather than mimicking and studying how brain structures optimize information processing, it may be effective to explore the direct integration of these systems, also known as "reverse neuromorphic computing". Implementing the biological neuronal networks within the brain directly into a computer's processing units can maximize efficiency, leading to greater information processing and more

	Frontier supercomputer (June 2020)	Human brain
Speed	1.102 exaFLOPS	~1 exaFLOPS (estimate)
Power requirements	21 MW	10–20 W
Dimensions	680 m^2 (7,300 sq ft)	1.3–1.4 kg (2.9–3.1 lb)
Cost	$600 million	Not applicable
Cabling	145 km (90 miles)	850,000 km (528,000 miles) of axons and dendrites
Memory	75 TB/s read; 35 TB/s write; 15 billion IOPS flash storage system, along with the 700 PB Orion site-wide Lustre file system	2.5 PB (petabyte)
Storage	58 billion transistors	125 trillion synapses, which can store 4.7 bits of information each

The Hewlett Packard Enterprise Frontier, or OLCF-5, is the world's first exascale supercomputer, hosted at the Oak Ridge Leadership Computing Facility (OLCF) in Tennessee. It is compared here with the human brain. For sources see (6–11).

Figure 1. Comparison Between Supercomputer and Human Brain

sophisticated machine learning algorithms (Kagan et. al, 2023). As the brain receives new information, it reorganizes itself and forms new connections. The incorporation of self-restructuring capabilities to AI opens more dynamic approaches to AI training. In addition, the incorporation of neuroplasticity functions in GPUs may allow neural networks to adjust how they process inputs without rigorous retraining. Such approaches give engineers the freedom to develop more powerful algorithms with deeper, more advanced neural networks. The implementation of neuroplasticity would enable AI models to process complex, continuous flows of data more effectively, allowing the energy-efficient and adaptable data processing of the brain to be manifested in GPUs.

> **The implementation of neuroplasticity would enable AI models to process complex, continuous flows of data more effectively, allowing the energy-efficient and adaptable data processing of the brain to be manifested in GPUs.**

Biological Neural Networks (BNNs)

Biological Neural Networks BNNs, or biological neural networks, are clusters of neurons connected to each other through axons and dendrites (in a sense, they can be described as miniature brains with the most basic complexity). Through these axonal and dendritic connections, BNNs are able to demonstrate characteristics that mirror those of artificial intelligence: computational performance and network plasticity (the ability of neurons to arrange themselves based on the stimulation received). The vast quantity of connections within a BNN enables the biological structure to undergo parallel processing across multiple neuronal signaling pathways and allows stimuli to be distributed vastly. When integrated within computing systems, BNNs have the ability to not only process the information and exhibit a response, but also rearrange themselves according to the stimulus, displaying plasticity and small amounts of memory (Dranias et. al, 2014). Memory consolidation in BNNs can be categorized into two types of memory processes, fading memory and hidden memory. Fading memory relies on the firing activity in response to a stimulus, lasting only for a brief moment, while hidden memory depends on synaptic plasticity to strengthen the connections between neurons. Hidden memory allows the BNN to retain information for prolonged periods. However, the retention of this information is disrupted or restructured when the BNN receives a high-intensity electrical input (stimuli).

Multielectrode Arrays

MEAs, or multielectrode arrays, help researchers integrate neurons within computational devices. Structurally, they are a flat surface with multiple microelectrodes embedded in an array placed underneath a BNN that is in a petri dish. Each electrode independently records extracellular activity. The recorded signals are then digitized and processed by a computer to interpret neural activity. The computer then generates an electrical stimulus that is sent as a response to the neurons to evoke another response, generating a real-time feedback loop.

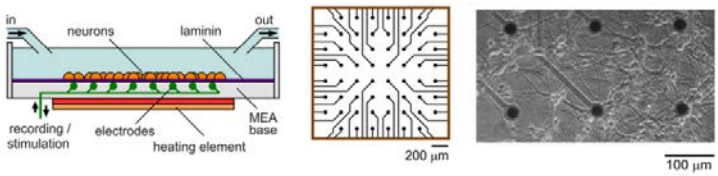

Figure 2. Multielectrode array structure (left to right) schematic, 60 electrode array, cultured neurons

Ping-Pong Experiment

Through an experiment revolving around ping-pong, researchers connected a BNN to an MEA that provided input of the game's environment, allowing the BNN to control a ping-pong paddle in real-time. The neurons received structured feedback through successful (positive reinforcement) and missed hits (negative reinforcement), allowing the BNN to self-correct from an optimal to a more accurate dynamic state. Through this, the BNN could retain task-specific information for brief intervals, displaying a positive correlation between branching ratio (neuroplasticity) and task performance. This enhanced BNN performance for that one specific task. When researchers no longer provided feedback to the BNN, its "hit-to-miss ratio" decreased (Habibollahi and Kagan, 2023). Once connected to a computing system through a multielectrode array, BNNs rearranged themselves until they reached an optimal dynamic state to efficiently process the structured stimuli. Despite reaching an optimal state, BNNs displayed low accuracy but a high level of computing, thus requiring feedback. Feedback was only in regards to one specific task, allowing BNNs to process information efficiently and with high accuracy for one task, making BNNs optimal for computing one task over and over again. However, if required to switch tasks, BNNs needed to rearrange themselves and be given the correct feedback to perform a task swiftly (Habibollahi and Kagan, 2023).

Neuronal Capabilities of Matrix Processing

The ping-pong experiment's success with parallel processing and real-time adaptation points to the potential of larger neural systems. In the brain, neural processing in the cerebral cortex occurs at intervals of a few milliseconds. Despite being slower than a computer, the cortex is able to compensate for this difference through its ability to process vast amounts of information in parallel (Ballard, 1986). Resembling GPUs, this parallel processing in the brain is how the brain receives sensory information and executes motor skills (Sigman and Dehaene, 2008). In addition, hierarchical (information flowing through successive layers of processing) and modular organization (each cortex is divided into specialized regions for particular tasks) of the brain is essential for matrix computations. Present artificial intelligence neural networks follow a similar information flow, but creating a processing system consisting of specialized BNNs can structure information for maximal efficiency and scalability. This hypothetical computational system, much like the brain, will have areas of the computer's processing unit distinguished by function. Each area will have their own specialization of information processing. As information is received, the information will be decomposed and sent to the right processing subregions, taking into advantage a BNN's fading memory. Information processing can then be split into smaller, manageable bits of information that each subregion of BNNs can process, allowing for scalability and efficiency.

Ethics

Despite using only BNNs, which in comparison to a human brain is minuscule in proportion, it is possible to create a sentient life force when such an organization is scaled to an extent. Eventually, if such biotechnological methods are implemented, one will create structures that contain a neuron count that mirrors certain large mammalian organisms. In the ping-pong game, researchers were able to notice a certain degree of self awareness within the BNN, despite being comparatively small to that of a simple organism's (Kagan et. al, 2022). However, if neurons were integrated into technology, will such a system eventually have a form of consciousness? Due to exponential growth of technology, the size of BNN integrated systems will also have to increase to address this demand. But once a certain number of neurons are integrated and interacting, it can eventually have its own life force. It is essential to bring up such topics when discussing the potential future direction advancement of biocomputational devices to prevent the unethical exploitation of sentient beings as inanimate objects. Furthermore, ethical considerations regarding the creation of such conscious systems must be explored, drawing a line as to when a system has developed consciousness.

Conclusion

Although the idea of integrating neurons into the processing system of computers seems efficient and beneficial to the further advancement of AI, it is still fairly new and many caveats have yet to be considered. For example, how would BNNs be integrated at a scale into computers/super computers that will provide enough processing power to match that of the computer? How much energy and resources would it take to maintain the functionality of a BNN once integrated into the computer's processing? Such topics are hard to answer and current research on the topic is in its infancy. In addition, with the global emergence of AI made public in only the past few years. But the benefits of such computational systems are apparent and mitigate the flaws of current information processing systems in computers.The ability of the brain to process information in a hierarchical and modular manner, as well as BNN's plasticity and hidden memory, can allow information processing systems to be optimally efficient. Without the rigid structure of AI neural networks, such a system allows for fluidity of adaptability, while taking on an energy-efficient approach.

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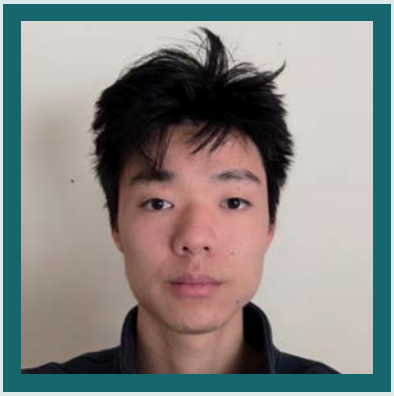

About the Author

Edward is a Sophomore at the University of Illinois majoring in Neural Engineering. Through his studies, he aspires to implement biological mechanisms/systems into computers and explore AI-neural network connections. Some of his interests include playing volleyball, filming, and going on road trips. After graduation, he hopes to attend graduate school.

The Role of Human Leukocyte Antigens in Multiple Sclerosis and Brain Atrophy

Written by Tanisha Mandal

Abstract

Human Leukocyte Antigens (HLA) are significant components of the human immune system responsible for autoimmunity. These genes are located on chromosome 6 and encode for proteins that assist the body in fighting against foreign invaders. However, HLA may have the potential to induce varying degrees of brain atrophy (BA) and multiple sclerosis (MS), both of which are neurodegenerative disorders. Examining the protective and aggravating effects of HLA on these neurological disorders, as well as potential preventive measures that could be implemented through HLA, may prove to have significant effects on the approach to BA and MS.

Introduction

Brain Atrophy (BA) is a progressive neurodegenerative disorder that leads to neuronal loss and connectivity. As a result, brain volume decreases and symptoms such as memory loss, seizures, and aphasia occur. While it is not uncommon to lose neurons as one gets older, BA refers to when an individual has more abnormal brain changes than what is expected for their age. In its most severe forms, BA can become fatal (Harris TC, de Rooij R, Kuhl E., 2019). There are two types of BA, focal and generalized. Focal refers to atrophy in a specific part of the brain, and generalized refers to atrophy spread throughout the brain.

Another chronic neurological disorder is Multiple Sclerosis (MS) which is caused by the autoimmune system, inducing the body to attack the central nervous system (CNS). Common symptoms include muscle weakness, coordination issues, cognitive difficulties such as memory problems, and slowed processing speed. The exact causes of MS remain unclear, but several factors contribute, particularly genetic predisposition. In an individual with MS, the autoimmune system attacks the myelin sheath surrounding the body's neurons, which disrupts communication between neurons (National Institute of Neurological Disorders and Stroke, 2024). The loss of neuronal connections caused by the presence of MS, is also an aforementioned key symptom of BA. Thus, MS can significantly worsen the damage done by atrophy. In addition, MS can also be the primary cause of BA, when the loss of connection is so great that it can be considered abnormal.

Due to the fact that MS is an autoimmune disorder, it can be linked to Human Leukocyte Antigens. Human leukocyte antigens, or HLA for short, are found on chromosome 6 and encode for proteins that present as antigenic peptides on T cells. This means that MS is a T-cell-mediated autoimmune disorder. HLA -DR antigens specifically are membrane heterodimeric glycoproteins, which means that they consist of proteins that are found in cell membranes, and often play a role in communication and signaling (Scholz, E. M, Marcilla, M, Daura, X, et al., 2017). In the case of HLA specifically, they play an important role in the autoimmune response system of the human body. As a result, many autoimmune disorders are caused by defects in the HLA gene. After extensive research, the allele responsible for this development has been narrowed down to Class II HLA-DRB1*1501 (Lorefice L, Fenu G, Sardu C, et al., 2019).

More research has uncovered that HLA can also provide protective effects against the demyelination caused by BA and MS. This is due to another class II allele, specifically DRB1*1302, which works in the CNS and has been shown to prevent the rapid degeneration of neurons associated with BA. Class II HLA has complex effects on the CNS, thus contributing to the development of brain atrophy and multiple sclerosis, with different alleles of the same gene providing vastly different outcomes.

Description of HLA Class II Alleles - Structure and Function

As seen in Figure 1, the primary structural difference between two types of human Leukocyte antigens is that Class 1 HLA molecules have 1 polypeptide chain, combined with a beta-2 microglobulin subunit while class II molecules contain 2 polypeptide chains. This results in the distinct roles that the molecules play in the human body.

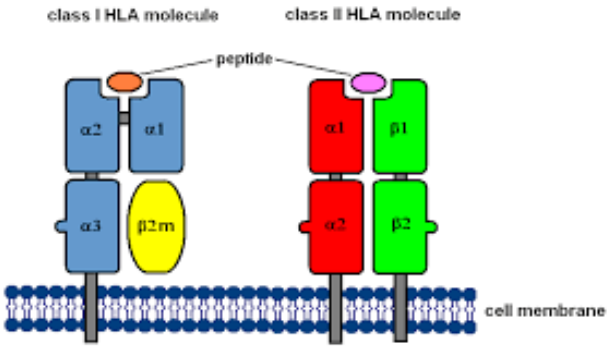

Figure 1: Structure of Class I and Class II HLA Molecules (Grubic, 2017).

Class I molecules are responsible for facilitating cell destruction, whereas class II molecules are responsible for recognizing invaders and producing the appropriate antibodies (James, L. M., & Georgopoulos, A. P., 2019). However, since class II HLA binds to antigen-presenting cells (APC) and is ultimately responsible for determining what the body considers a harmful microorganism, a miscoding in certain alleles can cause the body to attack itself, known as an autoimmune disorder. As previously stated, MS is classified as an autoimmune disorder, meaning that the introduction of MS in the body is almost entirely due to class II HLA molecules. This is why class II HLA molecules are the primary focus concerning BA and MS.

The Protective Effects of DRB1*1302 on Demyelination and Atrophy

Studies have discovered that the frequency of Class II HLA-DRB1*1302 has an inverse association with dementia, which is caused by the loss of synaptic connections, in 14 Western European countries (James, L. M., & Georgopoulos, A. P., 2019). As displayed in Figure 2, this is suspected to be due to the fact that the HLA DRB1*1302 allele helps encode for antibodies that specifically protect against pathogens that cause neurodegeneration. Dementia is linked to BA through the most prominent symptoms of neurodegeneration, loss of memory. Since the frequency of the DRB1*1302 allele has an inverse association with the loss of synaptic connections, it is suspected that the allele protects against pathogens that cause this neurological degeneration, which in turn causes BA. Therefore, it is reasonable to assert that the presence of the DRB1*1302 allele has an inverse effect on the onset of dementia, and thus the onset of BA.

For populations that express a high frequency of DRB1*1302, neurodegeneration can be slowed by around 45.2%. Scientists hypothesize that its protective effect could be due to the removal of antigens that persist in causing gradual brain atrophy. These beneficial effects have also been attributed to DRB1*13:02's binding to cathepsin S, a known harmful substance in brain aging (James, L. M., & Georgopoulos, A. P., 2019). This is best demonstrated in the research done on DRB1*1302's effect on dementia and Gulf War Illness, which are both caused by some levels of neurodegeneration. Thus, it is reasonable to assume that DRB1*1302 demonstrates preventative effects against general atrophy of the brain.

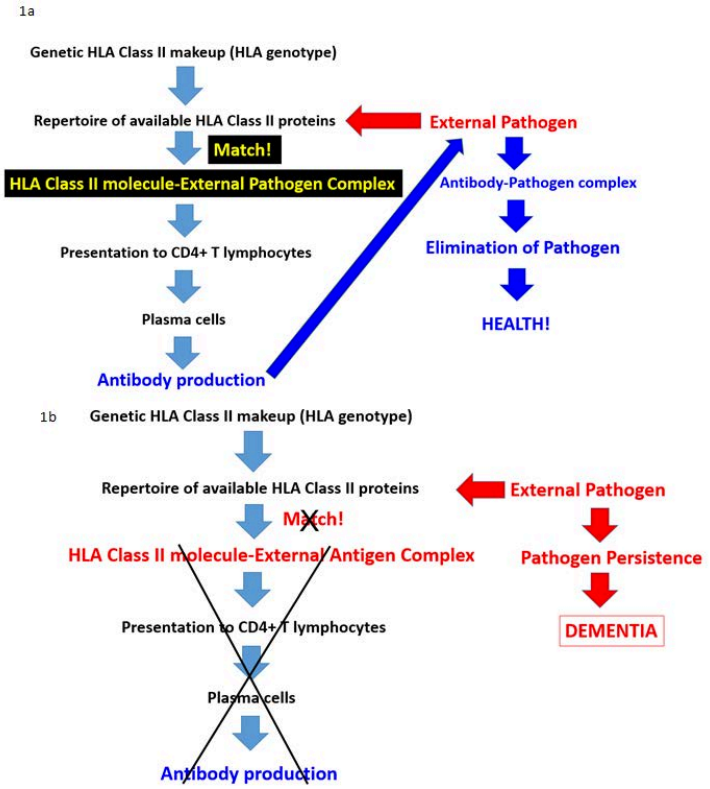

Figure 2: Flowchart Demonstrating Link Between Decreased HLA and Dementia (James, L. M., & Georgopoulos, A. P., 2019)

The Harmful Effects of DRB1*1501 on MS and Brain Atrophy

HLA-DRB1*1501 is an HLA haplotype, a group of closely linked alleles that are frequently inherited together and has been linked to a significantly higher risk of MS, and consequently, also to BA (Scholz, E. M, Marcilla, M, Daura, X, et al., 2017). In fact, DRB1*1501 is the single strongest genetic factor in the development of MS. While T-cells aren't typically considered APC, they do express major histocompatibility complex (MHC is known as HLA in humans) class II antigens, which are precisely what class II HLA control (Pichler, W. J & Wyss-Coray, T, 1994).

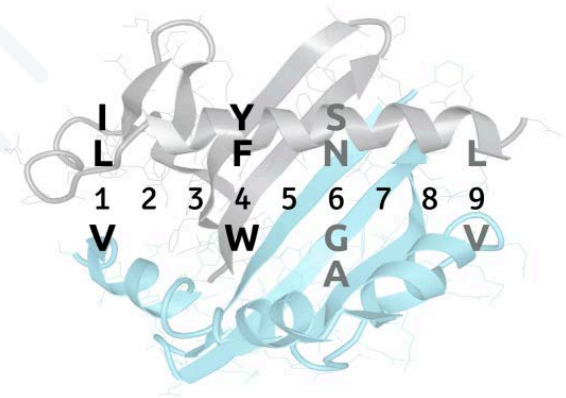

Figure 3: Binding Motif of HLA DRB1*1501 (HLA Protein, 2024).

Research suggests that pathogens involved with demyelination may appear to be similar to the actual myelin itself. As a result, DRB1*1501 could be especially susceptible to being "tricked" by this mimicry and produce agents that begin attacking myelin instead of the pathogens (Sospedra, M., & Martin, R., 2005). In addition, research shows that DRB1*1501 is suspected to interact with specific cytokines that are responsible for signaling immune responses in the brain, causing inflammation and degeneration of cells in the CNS. They essentially secrete proinflammatory cytokines that predispose the body to autoimmune diseases such as MS. When reviewing patients who expressed a high frequency of DRB1*1501, scientists found that "carriage of HLA-DRB1*15 was associated with increases in the development of brain grey and white matter pathology, as reflected by reduced [magnetization transfer ratio] (MTR), a trend toward increased T2 lesion load over 5 years, and greater T2 lesion volumes at each time point over the follow-up" (Tur, C., Ramagopalan, S., Altmann, D. R., et al., 2014). Thus, it was concluded that individuals showed a significant amount of brain atrophy when contrasted with the healthy control group. This shows a strong positive correlation between the presence of the DRB1*1501 allele and abnormal amounts of atrophy in the brain.

Effects of HLA on the Pathways of MS and BA

In nearly every case of MS, the early stages of pathology result from abnormal amounts of inflammation in the brain (Kiss, M. & McAlpine, C.S., 2023). Neuroinflammation is caused by the increased presence of T-cells in response to supposed invaders, which in cases of MS are the body's own cells, and the regulation of these T-cells is linked back to HLA. Individuals carrying the DRB1*1501 allele, have an increased susceptibility to MS and more severe disease progression, which can lead to greater demyelination. This is due to the role of these alleles in promoting an autoimmune response against myelin proteins. Similarly, the onset of BA is caused by abnormal amounts of demyelination in the brain. One of the most common causes of demyelination is inflammation, linking the root cause of BA to the root cause of MS. Thus, BA is similarly influenced by the HLA alleles.

Proposed Preventative Gene Therapy:

Brain Atrophy and Multiple Sclerosis have long been thought to be incurable, with treatment mainly consisting of just improving the quality of life of people afflicted with these diseases. Since different alleles of HLA can produce different effects on BA and MS, a potential goal for gene therapy could be to activate DRB1*1302 more often and suppress the expression of DRB1*1501 in HLA. As a result, preventative effects would be much higher, and the rate of BA and MS in individuals would likely decrease significantly. One form of gene therapy could focus on monoallelic expression, as demonstrated in Figure 4, which is when one allele of a gene is expressed, and another allele of that same gene is silenced.

Techniques such as CRISPR have been used to selectively silence one allele while leaving the other intact, which seems to be exactly what individuals at risk for atrophy need (Hsu, P. D., Lander, E. S., & Zhang, F., 2014). Overall, a preventative cure for atrophy may be just around the corner, providing a means to protect against one of the most complicated and unstoppable neurodegenerative effects.

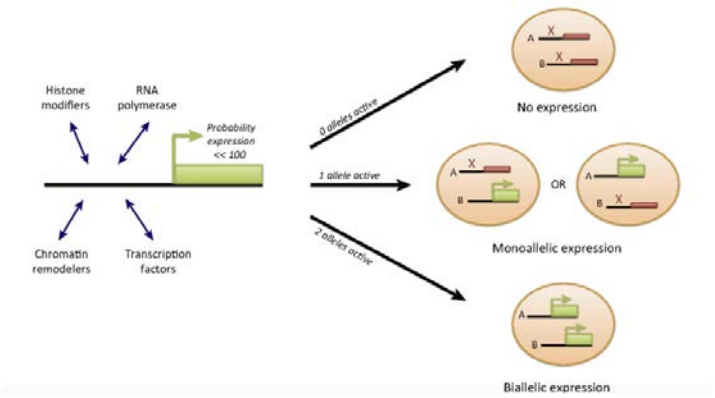

Figure 4: Diagram of Monoallelic Gene Therapy (Eckersley-Maslin, M. A.,& Spector, D. L., 2014)

Conclusion

Although the significance of HLA on the pathways of BA and MS has not been widely explored, the link between the two is most certainly present. Due to both neurodegenerative diseases being directly linked to the loss of synapses onset by autoimmune responses in the brain, and HLA being responsible for the immune system, HLA is a clear factor in the development of BA and MS. The two alleles most closely related to the onset of the diseases, DRB1*1501 and DRB1*1302, are haploids of HLA, so genomic editing and therapy may provide the first preventative cure for BA and MS.

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The Critical Window for Estrogen Replacement Therapy in Menopausal Women: Exploring the Neuroprotective Effects of Estrogen in Reducing Dementia Risk

Written by Sylvia Merz

Abstract

Menopause, characterized by a significant decline in estrogen levels, profoundly impacts brain health, influencing cognitive functions and neurobiological integrity. Estrogen replacement therapy (ERT), especially when administered within a "critical window" near the onset of menopause, has shown promise in mitigating cognitive decline and reducing dementia risk. This paper explores the neurobiological mechanisms underlying estrogen's protective role, including its effects on mitochondrial health, synaptic plasticity, neurogenesis, and amyloid-beta plaque clearance. By synthesizing current literature, we underscore the importance of early ERT initiation and highlight the implications of the critical window hypothesis for optimizing cognitive outcomes in aging women.

Introduction

The onset of menopause is marked by a rapid decline in circulating estrogen levels, which significantly impacts cognitive functioning and neurobiological integrity. Estrogen plays a crucial role in maintaining synaptic density, promoting neurogenesis, and enhancing cellular resilience against age-related stressors, particularly in brain regions critical for memory and executive function (Sherwin, 2012; Brinton, 2013). The reduction in estrogen levels during menopause is associated with accelerated neurodegenerative processes, contributing to an increased risk of dementia in postmenopausal women (Mosconi et al., 2017; Maki & Henderson, 2020).

Emerging evidence supports the notion that ERT can offset cognitive decline; however, the timing of ERT initiation is critical. The "critical window hypothesis" posits that starting ERT during the menopausal transition or shortly thereafter enhances its neuroprotective effects, while delayed initiation may diminish its efficacy or pose risks (Whitmer et al., 2011; Turek & Gąsior, 2023). This paper reviews specific neurobiological mechanisms through which estrogen influences brain health and examines how the timing of ERT initiation aligns with these mechanisms to optimize cognitive outcomes.

Estrogen's Role in Key Brain Regions

Estrogen exerts its neuroprotective effects primarily in the hippocampus and prefrontal cortex, regions essential for memory, learning, and executive function.

The Hippocampus

The hippocampus is critical for neurogenesis and memory formation. Estrogen promotes synaptic plasticity and enhances long-term potentiation (LTP), vital for memory retention and cognitive flexibility (Maki & Henderson, 2020). Research indicates that estrogen supports the maintenance of hippocampal volume by preventing age-related atrophy and promoting synaptic connections (Mosconi et al., 2017; Liu et al., 2019). Moreover, estrogen enhances the expression of synaptic proteins such as synapsin and PSD-95, which are crucial for synaptic stability and plasticity (Zhou et al., 2017). Early initiation of ERT appears to bolster hippocampal integrity, thereby preventing functional decline in this essential region.

The Prefrontal Cortex

The prefrontal cortex is responsible for executive functions, including decision-making, attention, and working memory. ERT can enhance prefrontal cortex function, particularly in tasks requiring cognitive flexibility (Wang et al., 2015). Estrogen increases synaptic density and improves neurotransmission efficiency, facilitating cognitive processes crucial for complex decision-making (Turek & Gąsior, 2023). This enhancement occurs through estrogen's modulation of GABAergic and glutamatergic signaling pathways, which contribute to improved cognitive performance (Daniel & Dohanich, 2015). By sustaining these

neurobiological functions, estrogen mitigates cognitive aging, particularly in domains that decline significantly with age.

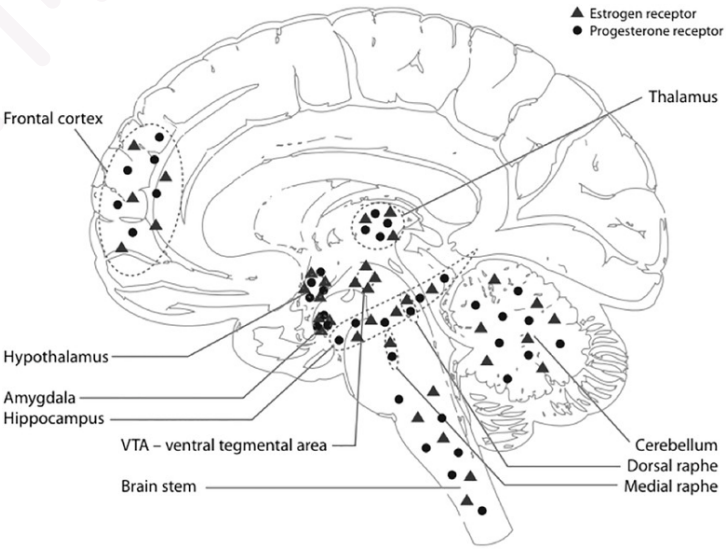

Figure 1. Estrogen and progesterone receptor concentrations throughout the brain, displaying high concentrations in both the hippocampal and prefrontal cortex regions (Boyle et al., 2020)

Neurobiological Mechanisms of Estrogen in the Brain

Estrogen exerts its neuroprotective effects through several cellular and molecular pathways, including mitochondrial function, neurogenesis, synaptic plasticity, and amyloid-beta clearance. These pathways collectively contribute to cognitive resilience and reduce the risk of neurodegenerative diseases.

Mitochondrial Health and Oxidative Stress Reduction
Mitochondria are essential for meeting the high energy demands of brain cells. Estrogen enhances mitochondrial function by promoting ATP production and activating antioxidant systems that neutralize free radicals, thereby reducing oxidative stress (Brinton, 2013; Maki & Henderson, 2020). Estrogen's role in upregulating antioxidant enzymes such as superoxide dismutase (SOD) and glutathione peroxidasehelps protect neurons from oxidative damage (Kumar et al., 2019). This mitochondrial support is especially relevant in the hippocampus and cortex, where energy demands are heightened due to intensive synaptic activity.

Neurogenesis and Brain-Derived Neurotrophic Factor (BDNF) Regulation
Estrogen stimulates neurogenesis in the adult hippocampus, partly by increasing levels of brain-derived neurotrophic factor (BDNF), a protein crucial for neuron survival, growth, and synaptic plasticity (Gibbs et al., 2000). BDNF not only supports memory processes but also enhances cognitive flexibility, which tends to decline during aging.

> **An estrogen-deprived state can lead to reduced BDNF levels, impairing neurogenesis and contributing to cognitive decline (Turek & Gąsior, 2023).**

Furthermore, estrogen facilitates the conversion of neural stem cells into neurons, enhancing the capacity for neurogenesis (Wang et al., 2015).

Synaptic Plasticity and Neurotransmitter Modulation
Synaptic plasticity, the brain's ability to strengthen or weaken connections between neurons, is central to learning and memory. Estrogen enhances synaptic plasticity by modulating glutamatergic and cholinergic neurotransmission, which supports efficient signal transmission across neurons (Daniel & Dohanich, 2015). Estrogen also influences NMDA receptor activity, which plays a key role in synaptic strengthening during memory encoding and retrieval. By supporting these mechanisms, estrogen fosters an environment conducive to cognitive agility and long-term memory storage.

Amyloid-Beta Clearance and Tau Pathology Reduction
The accumulation of amyloid-beta (Aβ) plaques and tau tangles are hallmarks of Alzheimer's disease pathology. Estrogen contributes to the enzymatic breakdown of Aβ plaques and inhibits tau hyperphosphorylation, thus reducing the risk of these toxic accumulations (Mosconi et al., 2017; Maki & Henderson, 2020). Recent findings suggest that estrogen enhances the expression of proteins involved in Aβ clearance, such as apolipoprotein E (ApoE), and promotes the function of microglia, the brain's resident immune cells, in phagocytosing Aβ deposits (Kuhlmann et al., 2020). By initiating ERT within the critical window, these neuroprotective processes are more likely to be effective in staving off Aβ-related pathology and maintaining healthy neural networks.

Timing of Estrogen Replacement Therapy: The Critical Window Hypothesis

The critical window hypothesis proposes that the cognitive benefits of ERT are maximized when initiated during a narrow window around menopause onset. This timing aligns with estrogen's neurobiological effects on brain health, as early intervention supports neurogenesis, mitochondrial

function, and Aβ clearance before age-related declines solidify (Maki & Henderson, 2020; Whitmer et al., 2011). Espeland et al. (2015) reported that women who began ERT during the menopausal transition exhibited better cognitive outcomes compared to those who delayed therapy, underscoring the importance of early intervention.

Conversely, delayed ERT beyond this critical period can have detrimental effects, potentially increasing the risk of cognitive decline. Whitmer et al. (2011) found that late initiation of ERT correlated with an elevated risk of dementia, suggesting that the aging brain, after prolonged estrogen deficiency, may become less responsive to hormonal interventions or even react adversely due to disrupted compensatory mechanisms developed in response to earlier estrogen loss.

Clinical Implications and Future Research Directions

The critical window hypothesis carries substantial clinical implications, advocating for personalized ERT timing based on individual health profiles and menopausal timing. Tailoring ERT protocols could maximize cognitive benefits and minimize risks, particularly for women at high risk of Alzheimer's disease. Emerging studies on selective estrogen receptor modulators (SERMs) indicate potential alternatives to traditional hormone therapy, offering neuroprotective benefits without the associated risks (Shumaker et al., 2003). SERMs, such as bazedoxifene and ospemifene, may selectively target estrogen pathways that promote cognitive health while minimizing the risk of breast cancer, a significant concern with conventional ERT (Huang et al., 2017).

Future research should continue to refine ERT protocols and investigate SERMs and other compounds that modulate estrogenic activity to offer targeted, effective options for cognitive support in postmenopausal women. Additionally, studies examining the interaction between genetic factors, such as APOE ε4 allele status, and the efficacy of ERT may yield valuable insights into optimizing treatment for diverse populations (Vakhitov et al., 2023).

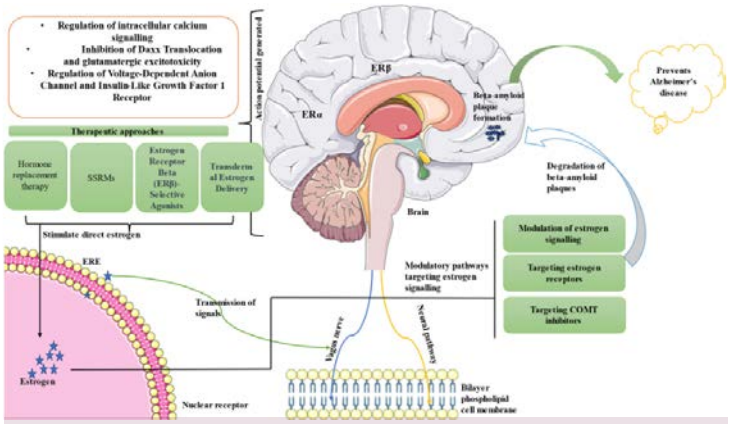

Figure 2. Synthesis of therapeutic strategies targeting estrogen in an effort to degrade accumulated beta amyloid plaque and prevent Alzheimer's Disease (Mishra et al., 2023)

Conclusion

Estrogen's influence on brain health is profound, encompassing mitochondrial support, neurogenesis, synaptic plasticity, and the reduction of neurodegenerative pathology. The timing of ERT initiation, as emphasized by the critical window hypothesis, is crucial for achieving these neuroprotective benefits. Early intervention, aligned with the menopausal transition, coincides with estrogen's mechanisms in preserving cognitive health and lowering dementia risk. Ongoing research should aim to elucidate the complex interplay of estrogen signaling, neuroprotection, and timing to enhance cognitive resilience in aging women.

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About the Author

Sylvia Merz is a junior majoring in Psychology with a concentration in Cognitive Neuroscience and minors in Public Health and Statistics. On campus, she is involved as a research assistant within the Laboratory for the Emotion and Stress Assessment, a course assistant for STAT 212 (Biostatistics), and a member of Girls Next Door (an a cappella group). She also serves as a community representative for the Alzheimer's Association, through which she has integrated her passion for global health and psychology to contribute to aging research. In her free time, Sylvia loves to hike, thrift, and sew!

The Consequential Effects of Sleep Quality on the Academic Performance of University Students

Written by Siwon Park

Abstract

Sleep is a vital biological process essential for proper physiological and cognitive functioning. While its true purpose remains largely theoretical, sleep deprivation has been proven to impair numerous brain functions, particularly in college students who are increasingly susceptible to irregular sleep patterns. Drawing on evolutionary and neurological theories, such as the restorative, synaptic homeostasis, and brain plasticity theories, the article examines the impact of sleep deprivation on cognitive processes, memory retention, attention span, and overall brain connectivity. Through recent studies utilizing tools such as MRI and attention network tests, a consistent decline in brain activity and memory function was observed in sleep-deprived individuals. These effects are especially prevalent in university settings, where academic pressures, lifestyle changes, and increased substance intake contribute to deteriorating sleep quality. The findings highlight that inadequate sleep not only diminishes students' ability to retain and process information but also places them at higher risk for academic failure and long-term health consequences. Ultimately, the article emphasizes that sleep is not only necessary for survival but also fundamental to academic success and cognitive resilience.

Introduction

While sleep is understood as a state of unconsciousness for rest and several dynamic neuroligical processes, the purpose behind its true nature has not been fully discovered. Many correlative theories and evolutionary explanations exist to uncover the true purpose of sleep. Scientifically reasonable theories include inactivity, energy conservation, restoration, synaptic homeostasis, and brain plasticity theory (Division of Sleep Medicine at Harvard Medical School, 2021). The inactivity theory–also described as the evolutionary or adaptive theory–suggests that sleep was an evolutionarily advantageous quality, particularly for survival. The energy conservation theory theorizes another evolutionary explanation for sleep: when sources of food were scarce, conserving energy by sleeping would be an efficient method to maximize the utility of energy. More recent research has led to the other three theories: restorative, synaptic homeostasis, and brain plasticity, all of which refer to the function of the brain and its necessity to reset in terms of necessary chemicals, neural connections, and memory along with developmental aspects. Much like the human body–which cannot sustain exertive physical activity and requires recovery through sustenance, reconnection of muscle fibers, and rest–the brain can function ordinarily through mandatory rest. Regardless of the true physiological objective of sleep, sleep is an essential, life-sustaining activity necessary for function.

Figure 1. Research has indicated multiple phases in one sleep cycle, each with varying characteristics and roles. The main phases include N1, N2, N3, and REM, which describe the physical stages of sleep one experiences throughout their sleep (Suni, 2025).

The detrimental effects of sleep deprivation are evident. Sleep quality is subjective for every person; varying durations of overall sleep, along with duration within each stage of the sleep cycle, can differ. However, a general deviation from quality sleep has demonstrated harmful effects. While the issue of sleep deprivation has been on the

rise for the general American population, the greatest contributors to this statistic are college students.

As the number of sleep-deprived college students rises, the importance of exploring possible causes of sleep deprivation grows. Many students find college to be their first experience away from home, which can be an unfamiliar and uncomfortable environment. Aspects of this new environment, such as dining hall food and roommates, can negatively affect sleep quality. In addition, the potential increase in workload for students can affect the quality and duration of sleep due to factors such as lack of sufficient time and stress. College also presents easier access to alcohol and other substances, which may cause passing out, and although the duration of unconsciousness may be long, the quality of sleep is poor (Gaultney 2010). Furthermore, unregulated intake of caffeine severely affects sleep as the substance indirectly inhibits melatonin synthesis and secretion by preventing the binding of adenosine triphosphate to the active site of the receptor, leading to prolonged time without sleep. With these facets for decreased sleep quality, the potential development of sleep and mental health disorders increases, which can further affect a student's sleep. With the specific analysis of university students and their decreased sleep quality, the correlation between sleep deprivation and academic performance can be explored. This article argues that sleep deprivation significantly impairs the academic performance of university students by reducing cognitive function as well as diminishing memory retention and attention span, ultimately hindering their ability to succeed academically and maintain overall well-being.

Compromise of Cognitive Activity

A study explored the characteristics of sleep deprivation imposed on eight healthy male and female subjects, with normal sleep conditions as the control. Within the study, the brain activity of subjects who received normal sleep and 24 hours of sleep deprivation was monitored. Although "normal sleep" was not specified to an exact time of unconsciousness, "good" or "normal" sleep was accepted as being subjective. Thus, the researchers utilized measurements of fitness, usage of certain substances, presence of sleep disorders, and a Pittsburgh Sleep Quality Index (<5) to filter participants and maintain their norm throughout the study. (L Wang, Y Chen, Y Yao, Y Pan, Y Sun, 2016). With every participant having a Fitbit monitoring their sleep status, subjects were randomly assigned to sessions of an attention network test, followed by an MRI test that measured the amplitude of low-frequency fluctuations (ALFF). The data gathered described higher ALFF areas in the right cuneus and lower ALFF areas in the right lentiform nucleus, right claustrum, left middle frontal gyrus, left dorsolateral prefrontal cortex, and left inferior parietal cortex within the sleep-deprived subjects. The research team associated the lower ALFF in the left dorsolateral prefrontal cortex with a reduction in gray an essential tissue in the brain and spinal cord for many cognitive and motor-control functions, as well as a reduction in regional homogeneity (Wang, Chen, Yao, Pan, Sun, 2016). With the brain scans that demonstrated lower ALFF levels, the cognitive function and the connectivity between the inferior parietal cortex and the medial prefrontal cortex were compromised, thus, detrimentally affecting memory recall, consolidation, and retrieval along with crucial cognitive functions.

The left inferior parietal cortex is involved in memory recall, consolidation, and retrieval, as well as cognitive functions like bodily awareness, responsibility, and moral decision-making (L Wang, Y Chen, Y Yao, Y Pan, Y Sun, 2016). One particular finding demonstrated that "sleep deprivation reduced the left inferior prefrontal cortex deactivation during a visual short-term memory task" (A. Krause, E. Simon, B. Mander, S. Greer, J. Saletin, A. Goldstein-Piekarski, M. Walker, 2017). With crucial cognitive functions within the responsibilities of the left inferior prefrontal cortex and the left inferior parietal cortex, sleep deprivation that contributes to the declining function of both leaves an overall underperforming brain. This research demonstrates the significant effect that sleep deprivation contributes to the rapid degeneration of several crucial areas of the brain and their respective functions.

Within the brain, two primary networks—the default mode network (DMN) and its anticorrelated network (ACN)—exist to establish connections between neural networks to fulfill applicative tasks. Typically, the ACN corresponds to the frontoparietal network (FPN), which shows a negative correlation with DMN activity (L Wang, Y Chen, Y Yao, Y Pan, Y Sun, 2016). "In the sleep-deprived state, there is unstable reciprocal inhibition between task-related FPN activity and DMN activity, and erratic ascending arousal activity influencing thalamic activity" (Krause, Simon, Mander, Greer, Saletin, Goldstein-Piekarski, Walker, 2017). The lower ALFF in these regions describes a weakening of the neurological connections within the brain, ultimately demonstrating detrimental effects on attention, working memory, and overall cognitive function. The consequence of sleep deprivation and the associated reduced function of areas within the brain stimulated compensatory activity from non-orthodox regions such as the lentiform nucleus and claustrum. Although the compensatory activity of these regions of the brain allows for extended cognitive function, the quality of such functions is compromised, with results demonstrating inconsistent cognitive abilities.

Diminishing Memory Retention and Attention Span

Memory is one of the main functions of the brain, and sleep deprivation has significant effects on memory. The tired hippocampus: The molecular impact of sleep deprivation on hippocampal function, published by Curr Opin Neurobiol, states that "Sleep deprivation has the biggest impact on hippocampal memory consolidation in the first few hours

following training when it overlaps with the second wave of cAMP signaling, transcription, and protein synthesis critical for increasing synaptic efficacy and memory storage."

Sleep deprivation results in the attenuation of cAMP-PKA-LIMK pathways (Chua, E. C., Fang, E., & Gooley, J. J., 2017), which is the direct cause of the detrimental effects on hippocampal function. Furthermore, this study discovered that sleep deprivation also reduces the translational and transcriptional processes through the inhibition of the mTORC1 pathway. In their study Effects of Total Sleep Deprivation on Divided Attention Performance, Chua, Fang, and Gooley (2017) reported that "divided attention performance was impaired during exposure to total sleep deprivation, as demonstrated by a significant interaction between task load (single, dual, and triple tasks) and time since wake on aGNG response times and errors." The auditory Go/No-Go (aGNG) task, which measures attention and response control, was used to assess participants' performance under varying cognitive loads. Results showed that participants performed worse on the aGNG task when required to divide attention across multiple tasks, compared to when completing the aGNG task alone. This decline in performance was further exacerbated by sleep deprivation, highlighting its significant negative impact on multitasking and overall cognitive functioning.

In addition to impaired memory, distractibility, which is the difficulty in maintaining performance and effort, is increased (Chua, E. C., Fang, E., & Gooley, J. J., 2017). Just like food and water, sleep is a necessity for life, and the homeostatic pressure of sleep and the given task conflict, ultimately resulting in the reduction of attention span and an increase in distractibility.

General Academic Detriments

The possible theories of sleep describe reasons for the necessity of sleep. The Restorative, Synaptic Homeostasis, and Brain Plasticity Theory all propose that the necessity of sleep is correlated with the functional reset of the brain and explains a process of recovery and restoration during sleep. Deviations from quality sleep and resetting of the brain can cause insufficient recovery of the brain. Consequently, the homeostasis of the brain can be offset, and susceptibility to sleep and mental disorders can be escalated.

The poor physiological conditions, along with the deteriorating cognitive functions described previously, are direct results of sleep deprivation, demonstrating the negative consequences of sleep deprivation on academic performance.

Academic performance is critical, as college and university education are crucial steps toward future careers and further education. However, the process of learning is not so simple as going to a lecture and taking notes. The complex pathway to academic success involves a variety of aspects, one of the most important being sleep. With sleep, sufficient information encoding can take place while allowing for brain space to absorb more information. On the contrary, "Students reported insufficient sleep and a discrepancy between weekday and weekend amount of sleep. Students at risk for sleep disorders were overrepresented among students in academic jeopardy (GPA < 2.0)," said Jane F Gaultney, professor of psychology at North Carolina, Charlotte (NIH). Without such a crucial aspect of academics, simply thinking, remembering, and paying attention becomes more difficult. The beneficial aspects of quality sleep, along with the detrimental consequences of sleep deprivation, contribute directly and indirectly to the academic success of an individual, clearly demonstrating the absolute necessity of sleep, not just out of physiological necessity, but for academic success.

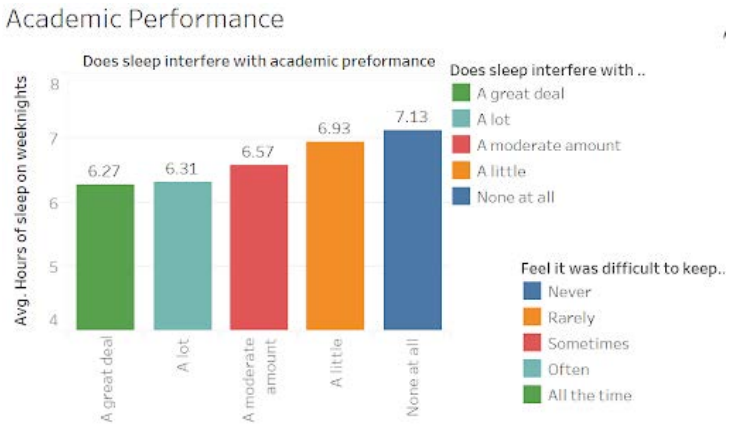

Figure 2. The barplot graph describes a negatively correlated relationship between the average hours of sleep a student gets on weeknights with how much they felt their sleep interfered with their academic performance (McGowan, Coughlin, 2017).

Extensive neurological and behavioral research demonstrates that sleep is not merely a passive state but a fundamental biological process crucial to cognitive functioning and academic performance. With evidence indicating the individual decline of each cognitive function in their effectiveness and efficiency, sleep deprivation has been demonstrated to be a significant detriment. With sleep being an absolute physiological need for humans, sleep deprivation not only harms cognitive function but also the person. With sleep deprivation demonstrating to be an incredible barrier to academic success and damaging to health, the emphasis on quality sleep is truly an imperative message to university students.

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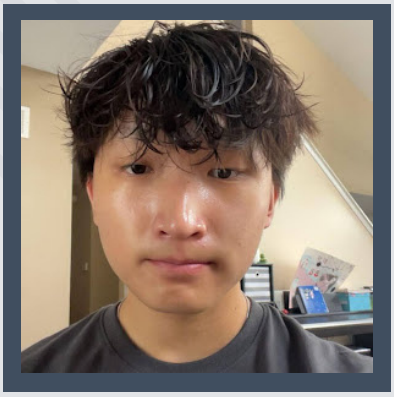

About the Author

Siwon is a pre-medical student majoring in Biochemistry at the University of Illinois Urbana-Champaign, with a strong interest in the intersection of research and clinical medicine. Passionate about understanding the molecular basis of disease and pharmacology. At UIUC, Siwon is engaged in research involving cell culture and cellular differentiation, with a focus on inducing stem cells to become muscle and neuron-like cells. Additionally, Siwon has contributed to research at the Feinberg School of Medicine, studying corneal damage and repair mechanism. Siwon plans to pursue a career in medicine that integrates both clinical practice and biomedical research. Through this dual path, he aims to help bridge laboratory discoveries with therapeutic advances that improve lives.

Understanding the Different Types of Cerebral Palsy and Treatment Options

Written by Leah Rupp

Abstract

Cerebral palsy is caused by damage to the brain during or right after birth. The damage– caused by infections or reduced oxygen supply–may affect certain areas of the brain such as white matter or the motor cortex. These injuries to the brain may lead to abnormal muscle stiffness or strokes, which are major symptoms of cerebral palsy. The three fundamental types of cerebral palsy are spastic, dyskinetic, and mixed. Each have individual classifications of muscle stiffness. Physicians may prescribe anticonvulsants to combat seizures and benzodiazepines to reduce muscle spasms. Recently, new technological advancements have improved the lives of those with cerebral palsy such as Voicett, a talking device.

Introduction

In the United States, between 5,500 and 13,100 children are born with cerebral palsy each year (Cleveland Clinic, 2023). Cerebral palsy is a neurological disease that affects muscle movement. Since there is no test to diagnose someone with cerebral palsy, physicians have to use a combination of Magnetic Resonance Imaging (MRI) and Computed Tomography (CT) scans, as well as neurological tests to determine if an individual has cerebral palsy. Many of the symptoms first appear in early childhood, such as having an abnormal gait, which is when individuals struggle with balance and coordination while walking. These motor impairments are mostly caused by damage to the brain. Further symptoms can arise such as seizure disorders, delayed growth and development, and impaired speech, vision, and hearing.

Causes of This Disease

There are many different causes of cerebral palsy. Birth complications and infections during pregnancy can be risk factors and causes of cerebral palsy. A child born prematurely, specifically before the 37th week of pregnancy, has a higher rate of developing cerebral palsy. Additionally, a baby with a low birthweight, less than 5 pounds and 8 ounces, has a higher rating of developing cerebral palsy (Centers for Disease Control and Prevention, 2025).

Various infections during pregnancy can increase the chance of developing cerebral palsy. For example, recent studies have connected cerebral palsy to chickenpox, rubella, and cytomegalovirus (Cleveland Clinic, 2023). These bacterial infections can infect the placenta causing the baby to be infected. Other infections can lead to a high amount of proteins, such as interleukin cytokines. A recent study conducted by Madison Paton, a researcher at Cerebral Palsy Alliance Research Institute, proved a link between specific cytokines such as IL-6 and IL-10, and cerebral palsy. These proteins are seen in high amounts for those with cerebral palsy. In a similar discovery, Dr. Mark R Schleiss, a pediatrician and researcher at the University of Minnesota, conducted a study finding the correlation between cerebral palsy and cytokines. Dr. Schleiss studied how interleukin cytokines are involved in neuroinflammation. They promote inflammation when the brain is injured through trauma or infections. Inflammation can lead to neuronal damage, causing motor development impairment, as seen in those with cerebral palsy (National Library of Medicine, 2021).

Brain Damage

Many different changes to the brain can cause cerebral palsy such as damage to white matter, cerebral dysgenesis, and intracranial hemorrhage (National Institute of Neurological Disorders and Stroke, 2025). White matter is a tissue in the brain full of a large network of axons, which aid in communicating to the rest of the body. It does this by passing down nerve impulses to neurons. Figure 1 shows six different newborns with acute to severe white matter injury. Patients D and F have low white matter, making them more likely to develop cerebral palsy. White matter can become injured by poor blood flow and nutrients,

causing ineffective communication throughout the body leading to abnormal motor development.

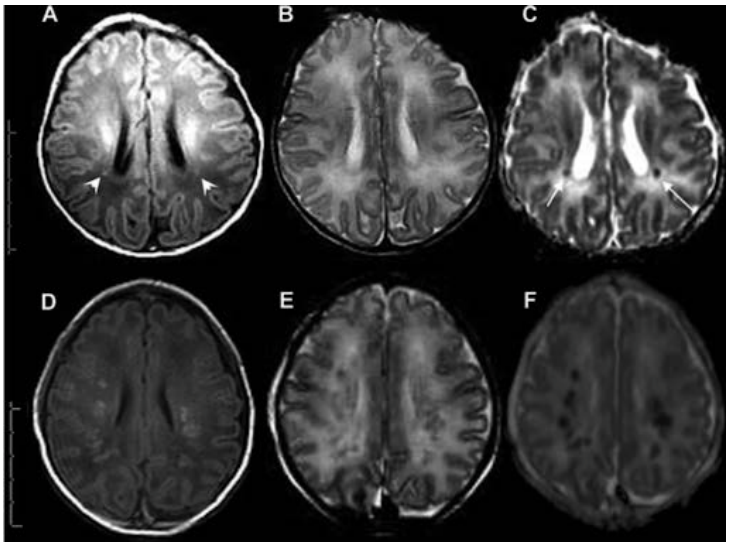

Figure 1. Patients A, B, C have relatively high amounts of white matter. Patients D, E, F have relatively low amounts of white matter, so they have a higher chance to develop cerebral palsy (Nature, 2009).

Cerebral dysgenesis is the abnormal development of the brain. These abnormalities can be caused by trauma, mutations, and infections. Intracranial hemorrhage is bleeding in the brain, commonly caused by a fetal stroke. Babies in the womb can experience a stroke when a blood clot occurs in the placenta. Damage to white matter, cerebral dysgenesis, and intracranial hemorrhage can all lead to abnormal motor development. This happens when areas of the brain that control motor function are affected.

Different Types of Cerebral Palsy

It is important to note that different types of brain damage can lead to various kinds of cerebral palsy. The three fundamental classifications of cerebral palsy are spastic, dyskinetic, and mixed (National Institutes of Health, 2021). Many medical professionals have discovered subclasses to spastic cerebral palsy, including diplegic and quadriplegic.

Spastic cerebal palsy is the most common type, and it is classified by extremely stiff muscles. Muscles can become abnormally stiff when damage is done to the motor cortex in the brain. The motor cortex controls muscles and movement, it can become injured by poor oxygen flow during or after pregnancy (Cleveland Clinic, 2025). One of the main subclasses of spastic cerebral palsy is diplegic. Individuals with diplegic cerebral palsy have stiffness only in the legs, as the arm and neck muscles are unaffected. Intellectual abilities are neurotypical, as well as speech abilities for diplegic cerebral palsy. Quadriplegic cerebral palsy is the second subclass of spastic cerebral palsy and it is the most severe. It is when the legs and arms are stiff, but neck muscles are extremely weak. For those with quadriplegic cerebral palsy speech is most likely impaired. Individuals may need assistance when eating and walking (Cleveland Clinic, 2023).

Dyskinetic cerebral palsy is a second type of cerebral palsy. It is the uncontrollable jerking movements of arms and legs. This is caused by damage to the basal ganglia in the neocortex, which is heavily involved in motor control (National Library of Medicine, 2023). For those with dyskinetic cerebral palsy, balance and motor skills may be abnormal. Facial muscles may also be affected, causing drooling. The last type of cerebral palsy is mixed. Mixed cerebral palsy is rare, and it is is a combination of spastic and dyskinetic.

Treatment Options

There are many great drug options for those diagnosed with cerebral palsy. Anticonvulsants are mainly to combat seizures and benzodiazepines are to reduce muscle spasms. Anticonvulsants work by decreasing the excessive electrical activity in the brain. This is done by altering the electrical activity of neurons. Neurons work by transmitting electrical and chemical signals to each other. When an individual has a seizure, neurons uncontrollably relay these signals. Anticonvulsants can inhibit certain ion channels, including sodium, potassium, chloride ion channels (Cleveland Clinic, 2023). Some of the more common anticonvulsants include topiramate, valproic acid, and phenobarbital.

Benzodiazepines are some of the oldest medical treatments to treat spasticity as seen in those with cerebral palsy. Benzodiazepines notify the brian to release more of a neurotransmitter called gamma-aminobutyric acid (GABA). GABA slows the nervous system down, creating a sedative effect on the body (Cleveland Clinic, 2023). In addition to medications, many new technological advancements have improved the lives of those with cerebral palsy. Voicett is an AI speech device for those with extreme speech impediments. The machine essentially “talks” for the patient with a click of a few buttons. Another great piece of technology is Stasism. Statism is a fun interactive way for children to complete physical therapy. The program utilizes games, sound effects, and colorful graphics.

Conclusion

Cerebral palsy can be caused from pregnancy implications or from brain injuries after birth. Damage to areas of the brain that control motor skills, vision, and speech can cause impairment. Multiple studies have been conducted on the correlation between interleukin cytokines and cerebral palsy (National Library of Medicine, 2021). Dr. Paton and Dr. Schleiss have conducted separate research on the correlation between interleukin cytokines and cerebral palsy. Cytokines are involved in neuroinflammation, where excessive inflammation can lead to neuronal damage, causing motor development impairment. Amount of white

matter is also a factor in developing cerebral palsy. White matter can be damaged through poor blood flow, inhibiting communication to the rest of the body. However, there are many different types of cerebral palsy, caused by different levels of brain damage. Spastic, dyskinetic, and mixed are the main types each effecting different parts of the body. Medical professionals have been utilizing anticonvulsants and benzodiazepines to treat seizures and spacity, respectively. Many more pieces of technology and medications are yet to be invented to better improve the lives of those with cerebral palsy.

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Leah Rupp is a freshman at the University of Illinois in Urbana-Champaign studying Molecular and Cellular Biology within the honors concentration. Leah joined Brain Matters to get the opportunity to learn and write about new neuroscience research. Leah is also a Stress Management Peer with McKinley Health Center and a volunteer with the Food Assistance and Well-being Program. In her free time, Leah enjoys running and playing the piano. Her career aspiration is to become a physician.

Isolation - How it Affects Fear Responses and Anxiety

Written by Ananya Sampathkumar

Abstract

Isolation is a common state for many people to feel. Despite this, isolation can have detrimental effects on people's emotions and brain. In particular, isolation affects the amygdala, specifically when it comes to anxiety and fear responses. Researchers have recently become further interested in the effects of isolation on the brain, especially following the COVID-19 quarantine. Understanding social isolation and its effects on the brain is crucial to learning more about the sociability of human beings, as well as how social anxiety can be caused or affected by interactions with others.

Introduction

Loneliness is commonly referred to as the feeling of social isolation. While this can be a familiar feeling for many people, loneliness is not ingrained into human nature (Finley, Schaefer, 2022). Humans are inherently social creatures: communication is so crucial for people that the usage of isolation as a torture tactic leads to drastic responses such as psychological disintegration or even death (Umberson, Montez, 2011). Like many other animals, complex social behavior and interactions are essential to proper health and survival (Young, 2008). Since socialization is consequential for proper health and development, the lack thereof can lead to several issues. Isolation is associated with higher rates of dementia and cardiovascular disease and an increased risk of mortality (Finley, Schaefer, 2022). When it comes to neurological symptoms, extended isolation can cause issues such as cognitive decline and lack of social understanding (Offord, 2020). These effects can be seen in the amygdala and the processes that it controls.

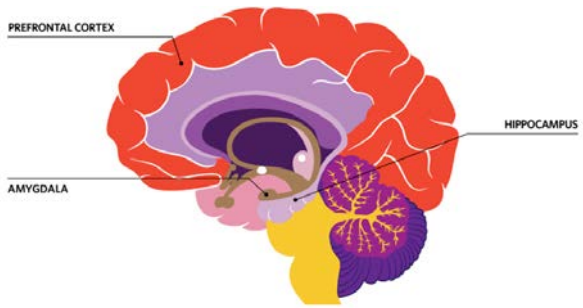

Figure 1. In the brain, the amygdala affects emotions, specifically fear and anger, which are heavily influenced by a lack of social interaction. Our current understanding of the amygdala suggests that increased levels of anxiety and further fear responses are mediated by the amygdala. (IStock.com, JamboJam)

How Social Isolation Changes the Amygdala:

Social isolation can cause the brain to undergo neural plasticity, or the changing and adaptation of the brain. Several circuit changes in the amygdala are associated with the effects of social isolation. To properly study this, arry Harlow performed many well-known experiences with rhesus monkeys in 1944 (Offman, 2020). Harlow separated baby monkeys from their mothers and raised them in clinical settings. It was noticed that these monkeys showed strange behavior such as circling their cage mindlessly and self-mutilating (APS, 2018). Specifically, Harlow demonstrated that baby monkeys born and raised in social isolation were naturally more aggressive and struggled with maintaining social interactions with other monkeys in 1944. These experiments started many conversations regarding how social isolation can affect interactions between different lobes of the brain. One longitudinal study by the Bucharest Early Intervention Project followed 136 adopted children (mean age of 55.56 months) who were exposed to social isolation in institutions such as orphanages as young

children. This study utilized magnetic resonance imaging (MRI) in order to study the brain's structure and neural connections. In children raised in institutions, the connectivity between the PFC and amygdala was immature in comparison to a traditional upbringing: this link is crucial in regulating emotions and influencing fear learning. This could lead to a possible lack of rational decision-making, as if the amygdala is unable to communicate with the prefrontal cortex, then learning aversive stimuli would be strained. Interestingly, children who grew up socially isolated were found to have increased activity between the PFC and hippocampus regions associated with aversive learning through the same study. Although these findings seem to be contradictory, each brain connection is responsible for regulating and expressing specific emotions, reactions, or other important mechanisms; no one connection is responsible for the entirety of one large-scale interaction. The relationship between the PFC and the hippocampus being increased could be causing increased memory storage. By remembering more, the brain is able to make more educated decisions about more things, and this overthinking is often associated with higher levels of anxiety. The majority of other neural pathways affected by the increased social isolation in children who grew up in institutions are between the PFC, amygdala, hippocampus and striatum, all of which are associated with fear, anxiety, and rewards (Xiong, et al. 2023).

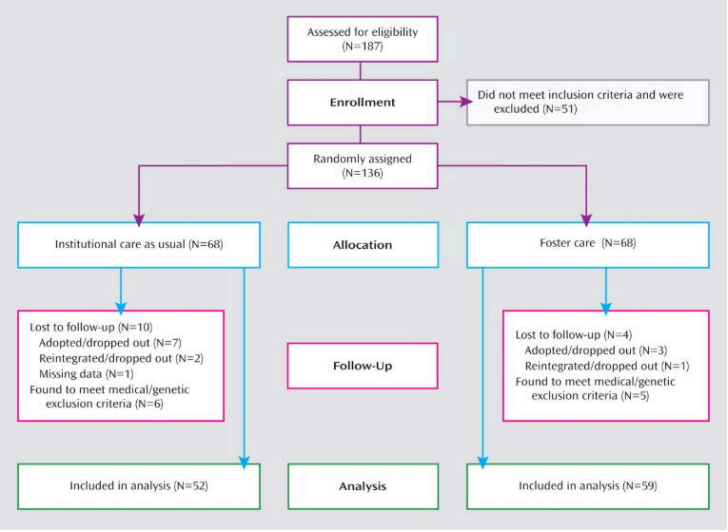

Figure 2. (Zeanah et al., 2009)

Moreover, the size of the brain can be affected by these changes as well. Researchers have found that the size of your amygdala is inversely proportional to the size of your social circle; generally, smaller amygdalas are associated with fewer angry emotions and increased socialization (Offman, 2020). These changes are fascinating and important to consider when it comes to the importance of social interaction in the brain. Finally, chronic stress caused by solitude during childhood has been shown to alter the blood-brain barrier of the amygdala. Inflammation in the brain has been heavily associated with an increase of depression and anxiety, which can all be tied back to the social isolation's effects on the blood barrier of the amygdala (Wu, et al. 2022). Researchers at Peking University Health Science Center utilized male and female mice in a study to test childhood isolation's effects on the brain. Three-week-old mice were either caged alone or with four to six other mice for eight weeks. The results showed that female mice were incredibly affected by this isolation. Interestingly, there were minimal changes observed in male mice in comparison. When the female mice caged in isolation were examined, their brains showed signs of inflammation. Chronic stress can affect the blood barrier of the amygdala, and oftentimes, can lead to inflammation of the brain, and is likely a result of the social isolation that these mice underwent over the eight-week period (Wu, et al. 2022).

> **...each brain connection is responsible for regulating and expressing specific emotions, reactions, or other important mechanisms...**

Long and Short-Term Effects of Social Isolation:

There are many long-term and short-term effects seen by individuals who have experienced social isolation of any kind. One of the major symptoms seen as a result of social isolation is the increase of chronic stress or anxiety. These disorders can be debilitating, and cause many issues and difficulties for many people who struggle with them. Furthermore, social isolation may lead to an increase in the likelihood of developing Alzheimer's. In 2018, researchers at he Tsinghua-Peking Center for Life Sciences studied how mice's ability to recognize one another was affected by social isolation. This was assessed by recording how long the mice spend interacted with one another. The mice were kept in isolation for either one day or seven days, and then given either 150 minutes or 15 minutes to explore a new environment with new mice. Interestingly, when mice were returned to their original enclosures afterward, they were able to interact with and recognize their colony mates again. This effect was also seen when researchers inhibited the Rac1 protein. Rac1 is a small signaling protein that is commonly linked to Alzheimer's and other memory loss issues (Offord, 2020). The inhibition of this protein means that signals are not being expressed as well as necessary for proper function, and specifically can affect the cell cycle and cellular plasticity. This can lead to many detrimental effects

such as memory loss (Tangella, et al. 2020). The expression of this protein during social isolation may contribute to the lack of recognition shown by the isolated mice, and seems to point towards a possible association between Alzheimer's and social isolation (Offord, 2020). These physical changes can cause many differing effects on the behavior of the individual experiencing social isolation such as heightened anxiety and depression. Still, there is still a large possibility that many of these effects are reversible. When it comes to the COVID-19 pandemic, there were many changes witnessed in the amygdala, such as a shrinkage in size. Fortunately, researchers have noted that after the social isolation period of the pandemic passed, the changes in the amygdala have returned to regular size and function, showing the plasticity of the brain (Xiong, et al. 2023).

A Case Study - Covid's Effects on the Brain

In late 2019, the world witnessed the most recent pandemic caused by SARS-CoV-2. This infectious disease spread throughout the world, forcing everyone into isolation in order to protect themselves and their loved ones from getting sick. As a result, the COVID pandemic stands as a great case study for the effects of social isolation on people for a long period of time. For many teenagers, this social isolation caused many mental health issues and neural changes. Adolescence is a major time for brain development, and the social isolation forced onto teenagers all over the world led to notable changes in their brain. A study performed on 16-year-olds reported that teenage brains had substantial physical changes from before and after the pandemic (Corrigan, et al. 2024). Following the pandemic, teenagers had lower average brain cortical thickness and larger bilateral hippocampal and amygdala volumes. These changes seem to indicate that the brains of these teenagers are maturing at a faster rate than they were prior to the pandemic. This can lead to issues such as early cognitive decline. This trend can be seen in children as well. Interestingly, this trend is more prominent in females than males. In order to track these changes, researchers utilized was 1.4 years, meaning that post pandemic male's brains were 1.4 years older than they should have been. The difference between female brains prior to and after the pandemic is more drastic. The average difference in ages between pre-pandemic female 16-year-old brains and post pandemic 16-year-old brains is about 4.2 years. This means that female brains have aged at a much faster rate than male brains during the pandemic (Corrigan, et al. 2024). While researchers are unsure as to exactly why male and female brains have aged, aging in the brain is often associated with increased rates of anxiety and depression. The faster your brain ages, the more likely anxiety and depression are to be risk factors for you (Han, et al. 2021).

Conclusion

Social isolation is the state of having very little social interaction or contact with other people. Human society is built upon complex interactions with one another due to our social nature, and a lack of social interaction can lead to many issues. Whether those issues are neurological such as increased anxiety or more physical such as increased likelihood to develop cardiovascular disease, social isolation can be detrimental to the health of people. This is seen specifically in the amygdala, where we see increased levels of anxiety and fear response as a result of the neural circuits and structure being adapted by prolonged isolation. These long term and short term effects can be extremely difficult to adapt to, and this is clearly shown in experiments discussing the COVID-19 pandemic. Now that the peak of the pandemic was about three years ago, researchers have finally begun to start understanding the mental health effects of the quarantine on differing groups. We have finally started getting results about the neural implications of the pandemic, but there is still a lot of room to learn and understand more about the effects of social isolation on the brain and the lives of the people around us.

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The Detrimental Effects of Aberrant Regulation on the Hedgehog Signaling Pathway

Written by Emily Aldrich

What is Sonic Hedgehog?

The Hedgehog (Hh) signaling pathway is a conserved neural pathway that plays an important role in the embryonic development of both invertebrates and vertebrates. This pathway was originally discovered in the species Drosophila melanogaster, the common fruit fly, and is found among a variety of species. The signal transmission from cell membranes are regulated by the Hh signaling pathway and dictate embryonic development. There are three main Hedgehog ligand proteins that regulate the transcription of target genes for this pathway. The three types of Hedgehog in the mammalian body are Sonic Hedgehog (Shh), important in the specification of cells in the nervous system, Desert Hedgehog (Dhh) which is seen in the hormone-producing gonad glands involved in reproduction, and Indian Hedgehog (Ihh) which plays a role in skeletal development (Carballo et al., 2018). All of the components in the Hh pathway are found in the primary cilium, which is an immobile organelle that juts out from the side of a cell and can sense the surrounding environment (Gigante & Caspary, 2020). When the pathway is regulated, typical development can occur. However, the dysregulation of the Hh signaling pathway may lead to a variety of diseases and disorders, including tumorigenesis of medulloblastoma, the most common malignant brain tumor.

> “...the dysregulation of the Hh signaling pathway may lead to a variety of diseases and disorders, including tumorigenesis of medulloblastoma, the most common malignant brain tumor.”

The Signal Transduction Pathway of Sonic Hedgehog

The Shh pathway is most commonly activated by canonical signaling, in which there are ligand-dependent interactions or receptor-induced signalings. Without the glycoprotein Shh, this signaling pathway does not occur. Smoothened (Smo) is a GPCR-like transmembrane protein that is usually inhibited by another transmembrane protein called Patched (Ptch 1) when the glycoprotein Shh is absent. Gli transcription factors are present in the cilia in a complex with Kif7, an IFT-kinesin that moves necessary materials toward the cilium during cellular signaling. Additionally, the

repressor factor Sufu promotes the truncation of Gli proteins, in which Gli proteins are shortened and turned into the GliR repressor form. This inhibits the transcription of Shh target genes (Traiffort et al., 2012). During Shh canonical signaling, the glycoprotein Shh binds to and inactivates Ptch 1, which, in turn, activates Smo. When this protein is activated, it accumulates at the primary cilium. This accumulation relieves the inhibition that Sufu exerts on Gli proteins, which allows them to turn into the GliA activated form, and move into the nucleus to activate the transcription of Shh target genes. Each target gene has a specific function. For example, Gli1 and Ptch1 are involved in pathway feedback, Cyclin-D1 and Myc promote cell proliferation, and CCND2 and CCNE1 regulate the cell cycle. In addition, bcl2 regulates apoptosis, AGN1/2 are involved in angiogenesis, SNAIL is involved with epithelial-to-mesenchymal transitions, and NANOG and SOX2 regulate the self-renewal of stem cells.

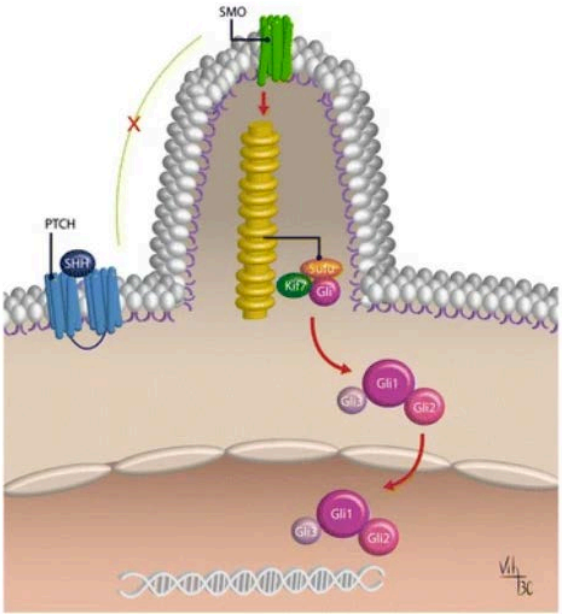

Figure 1. Canonical activation of the sonic hedgehog (Shh) signaling pathway occurring at the primary cilium (Adapted from Robbins et al., 2012).

Dysregulation of Hh Signaling Pathway

When the Hh signaling pathway is not carefully controlled, the effects on the development of cells and tissues can be very harmful. The aberrant activation of the Hh signaling pathway is caused either by mutations in pathway-related genes or by the excessive expression of Hh signaling molecules. This uncontrolled activation is what leads to tumorigenesis. The Shh pathway plays a particularly important role in regulating neural development in the cerebellum, a part of the brain linked to motor learning and coordination. Aberrant activation of this pathway is linked to pathway-activating mutations in Ptc (a protein found in Drosophila, the common fruit fly, that is similar to Ptch in humans), Sufu, or Smo, which all have key roles in the regulation of the Hh signaling pathway. It has been seen in mice medulloblastoma brain tumor stem cells that there is markedly higher Gli1 expression than in the normal stem cells. These cells do not undergo apoptosis (programmed cell death). Instead, they continue to proliferate when they are not supposed to. This suggests that there is a lack of protective mechanisms in place for these malignant stem cells, whereas non-malignant stem cells are able to control excessive proliferation in response to signals that promote mitosis. Interestingly, only 25% of medulloblastomas displaying abnormally high Hh signaling pathway activation have been found with mutations in Ptc, Sufu, or Smo (Traiffort et al., 2012). This means that there are other genetic pathways associated with Hh signaling that play a role in the development of cancer cells. Tumor suppressors are genes that regulate cell growth in order to prevent the development of cancer. Without them, cells will not perform apoptosis and instead continuously divide uncontrollably. Tumor suppressors such as Ren(KCTD11), Numb, and p53 have suppressive effects on Gli-dependent activation of Hh target genes. The activity of these tumor suppressors may decrease and lead to unregulated Glli protein activation, contributing to cancer development.

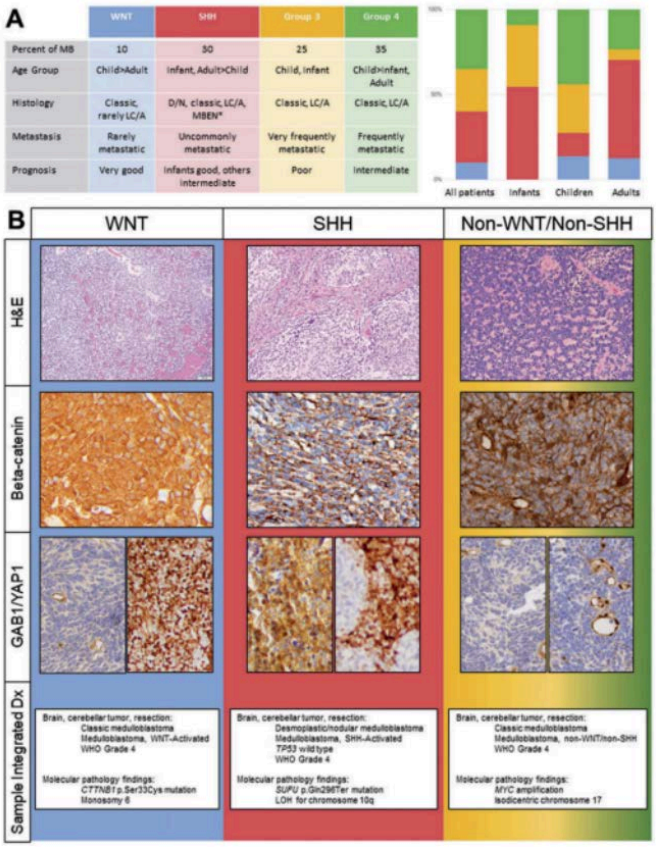

Figure 2. Medulloblastoma subgroups and the histological characteristics (Cotter & Hawkins, 2021).

Research for Improvement and Treatments

The findings of how the Hh signaling pathway works and its involvement in tumorigenesis have opened up the possibilities of developing methods of molecular targeting

and tumor prevention associated with the pathway. Several studies support the hypothesis that malignant tumors are initiated and maintained by cancer stem cells (Tan et al., 2006; Xie et al., 2022). Specific neuronal cancer stem cells can be found in a niche, where neurons and glial cells are generated from stem cells or progenitor cells. The niche provides signals that regulate whether the stem cells should differentiate, remain dormant, or actively divide. Shh is very important for determining cell fate and patterning during embryo development. It was discovered that the level of Shh signaling pathway activation in adulthood played an important role in regulating the balance between dormant and activated neuron stem cells (Carballo et al., 2018). Currently, the standard treatment for most brain tumors is the removal of the majority of the tumor, followed by chemotherapy and radiotherapy. Researchers are currently trying to determine alternative treatments involving the inhibition of the Shh pathway activation in cancer stem cells. There is great interest in targeted Hh signaling pathway inhibition (HPI) as a type of treatment for aggressive cancer cells when radiotherapy and surgery are not effective (Skoda et al., 2018). There have been multiple HPI molecules identified that act at different levels of the Hh pathway. One group is Hh ligand inhibitors, HPIs that inhibit the binding of the Hh protein to Ptch receptors, keeping Smo inhibited and therefore the rest of the pathway blocked from activating target genes. This includes Cyclopamine, Vismodegib, and Sonidegib. Another group is Smo antagonists which bind to a specific site on the Smo receptor that prevents the downstream activation of the Hh signaling cascade. However, clinical studies have shown that the use of Smo inhibitors can induce development of mutations that lead to treatment resistance. Moreover, Shh medulloblastomas are highly mutated tumors, and it is not uncommon for these tumors to develop a resistance to Smo inhibition, as they present alterations in downstream Shh pathway genes such as Sufu and Gli2. This turned researchers to Gli-based inhibitors, which is an alternative group of Shh antagonists that act directly in Gli to block transcription factors. This includes NVPLDE-225 and BMS-833923, which are currently being tested in brain tumors (Carballo et al., 2018). While most of the HPIs that have entered clinical trials mainly target Smo, the resistance to these inhibitors have lead to the discovery of new HPIs that may be essential to bypass these resistance mechanisms and control the tumorigenesis of medulloblastoma.

A Gateway Into the Future

The Hh signaling pathway plays a critical role in healthy embryonic development, putting into action a multitude of target genes that are needed for the initial stages of development. There are many steps in the transduction pathway leading to expression of target genes. Mutations that form can cause this highly regulated pathway to either activate uncontrollably or become inhibited at the wrong times. When this occurs, continuous and inappropriate cell division can lead to the rapid growth of tumors in the body. With the knowledge that scientists have today about the Hh signaling pathway, there is great potential for certain treatments and therapies that can inhibit the aberrant regulation of the Hh pathway, either from ligand-dependent or ligand-independent signaling inhibition. Further research into which pathway mechanisms are most likely to elicit a strong response to inhibition can provide a greater understanding of the Hh pathway, and can be used to create better, more effective, and safer anti-cancer therapies.

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About the Author

Emily Aldrich is a Freshman majoring in Neuroscience with minors in Linguistics and Psychology on the pre-med track. Emily joined Brain Matters to gain a deeper understanding of the brain through exploring current research topics in neuroscience. In her free time, she enjoys listening to music, reading, and spending time with friends.

The Influence of Maternal Stress on a Child's Development in the Womb and the Long-term Effects on the Child's Neurodevelopment and Mental Health

Written by Alexa DiVito

Introduction

Stress during pregnancy is common amongst both new and experienced mothers. (Dunkel Schetter and Tanner, 2012) Many can attest to the fact that a stressed mother can impact her children at any age, whether it be requiring them to do extra chores or causing anxiety. However, what many people may not realize is the immense impact stress during pregnancy has on children. Maternal stress does not only affect the mother but the fetus as well, especially during their development and later life. Research shows that when pregnant women experience various stressors (such as grief, daily challenges, or natural disasters), it can lead to significant changes in their children's neurodevelopment. These changes can include a higher likelihood of autism, emotional disorders, and diminished cognitive abilities.

The Fetal Programming Hypothesis

'The Fetal Programming Hypothesis' states that during critical and sensitive periods of development, a disturbance in environmental factors has an organizational effect on biological systems (Seckl and Holmes, 2007). These systems include the central nervous system (the brain and spinal cord), autonomic nervous system (regulates involuntary physiological process such as heart rate and respiration), neuroendocrine (comprised of the hypothalamic-pituitary-adrenal axis), cardiovascular, and immune systems. (Bale, 2015). Disturbances can also negatively affect intrinsic plasticity, which is the nervous system's ability to change its activity in response to stimuli. This can be done by reorganizing its structure, fruition, or connections, to react and adapt to environmental influences. This hypothesis states that fetal conditions can have major impacts later in life. These impacts can be presented in many ways, depending on which part of the brain is affected.

How the Fetal Brain is Physically Affected

Certain parts of a fetus's brain can be affected by maternal stress, hindering proper fetal brain development. For example,in animal models, acute periods of prenatal or postnatal stress have profound effects on HPA function and behavior in adult offspring. This means that maternal stress during prenatal development can affect how the fetus's brain and associated parts of the body will respond to stress later in life. These effects can be different based on sex. Chronic maternal stress increases locomotor activity, which can cause higher sensitivity to change or trouble focusing in adult male offspring; on the other hand, it decreases sensorimotor gating (regulation of sensory information) in adult female offspring. (Emack and Matthew, 2011). Naturalistic studies with humans find similar effects on motor behavior (Huizink et al. 2004). Studies of prenatal maternal anxiety (O'Connor et al. 2002), prenatal exposure to stressful life events (Stott 1973), and even prenatal exposure to dexamethasone (a synthetic glucocorticoid that is an anti-inflammatory and immunosuppressive steroid) (Trautman et al. 1995) are associated with children who are more withdrawn, anxious and depressed.

How the Brain is Cognitively Affected

Various research shows that prenatal exposure to maternal stress increases the risk of behavioral and mental health problems later in life (Van den Bergh et al. 2020). A classic study by Hutten and Niskanen (1978) examined rates of mental illness in samples of children from Finland whose fathers had died either during pregnancy or within their first year of life. Significantly greater rates of schizophrenia and other mental illness were found in the prenatal stress exposure group. Van Os and Selton (1998) also found a significant increase in rates of schizophrenia in Holland as a function of the German invasion during World War II. These studies show specific effects of the timing of the stressor during pregnancy, with the most noxious effects being associated with exposure during the second trimester. Because of this, it has been hypothesized that the timing of the disruption in fetal neural development, rather than the type of disruption, may be even more critical in determining the risk for negative outcomes and which developmental processes are likely to be affected (Mednick et al. 1998). These findings alter the impact of prenatal stress timing on development, a concept further explored by researchers in Project Ice Storm, which examined the effects of a large-scale natural disaster on pregnant women and their children.

Project Ice Storm

Through January 5th and 9th of 1998, a series of freezing rain storms hit Southern Quebec, Canada. There was widespread flooding and ice accumulation causing around 1.3 million power outages,which led to water filtration plants being shut down, leaving many people to seek new shelter. While the meteorological event itself occurred over a period of four days, the recovery process lasted even longer, with long-term impacts lingering for months afterwards (Senesac, 2019). Shortly after, Project Ice Storm was initiated. The objective of this project was to determine the nature and duration of the effects of an independent stressor during pregnancy on the unborn child by using a prospective design with a large sample of families. By conducting repeated assessments of affected women and their children over several years, the impact of objective stress exposure and subjective stress reaction on perinatal outcomes, maternal postpartum depression, and the behavioral, physical, and cognitive development of the children was found. The test group, STORM32, split mothers into three groups depending on the stress levels demonstrated (low, moderate, and high stress), and contrasted the low stress group with the combined moderate-high stress group. They found that moderate to high objective prenatal maternal stress is associated with poorer intellectual and language functioning at the age of two when scientists examined interactions between trimester of exposure and severity of ice storm stress. This is demonstrated by Graph 1.1.

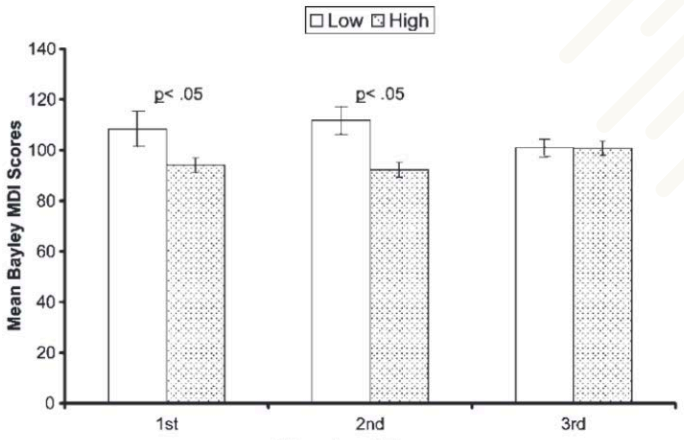

Figure 1. Toddlers' mean (+/- standard error) Bayley mental developmental index (MDI) scores at 2 years of age, as a function of objective prenatal maternal stress levels (low, high) and trimester of exposure (1st, 2nd, or 3rd).

These findings were reinforced by the results of their analyses of the children's play behaviors (Laplante et al.2004). For intellectual abilities, children whose mothers were exposed to the ice storm during their 1st or 2nd trimester of pregnancy and who experienced moderate or high objective stress had significantly lower Bayley MDI scores (a neural development assessment). This is demonstrated by Graph 1.2.

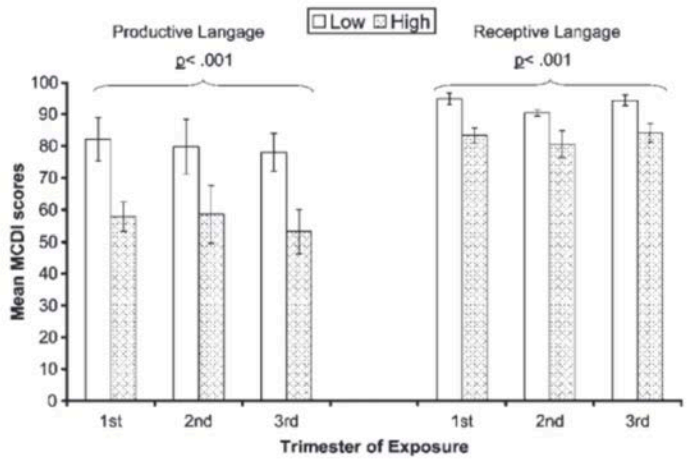

Figure 2. Toddlers' mean (+/- standard error) MacArthur communicative development index (MCDI) scores for productive and receptive language abilities at 2 years of age, as a function objective prenatal maternal stress levels (low, high) and trimester of exposure (1st, 2nd, or 3rd).

The findings from Project Ice Storm strongly suggest that major stressful events, independent of maternal personality factors, can have a negative impact on cognitive and language development of the unborn child.

Conclusion

Maternal stress during pregnancy has far-reaching effects that go beyond the immediate challenges of pregnancy. The evidence shows that stress during this crucial period can leave lasting marks on a child's neural development and mental health, with impacts that may only surface after birth. Whether it's everyday stress or more severe circumstances, the timing and intensity of stress exposure during pregnancy can influence the child's risk for cognitive and emotional difficulties. Supporting mothers during pregnancy isn't just about the mother's well-being at the moment– it's also about safeguarding the long-term health and development of their children. Addressing maternal stress will help ensure healthier outcomes for future generations.

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About the Author

Alexa DiVito is a freshman at the University of Illinois. She is currently an undeclared major on the Pre-Nursing track and plans to declare as a Psychology major next year. Alexa became part of Brain Matters to develop her knowledge of the brain and share her new knowledge with others. Apart from writing for Brain Matters, Alexa is involved in Greek life, RSO's, and is working on getting her CNA license.

Can We Train Our Brains to Break Social Media Addiction?

Written by Meha Goswami

Introduction

Social media has revolutionized communication, allowing people to stay connected with family and friends, and build relationships across the globe. While these benefits enhance social and psychological well-being, excessive use of social media can have detrimental effects on mental and physical health (Cheng et al., 2022). Many individuals turn to social media as an escape from reality, using it to avoid problems rather than address them. Cheng et al., (2022) found that when social media consumption becomes compulsive and interferes with daily life, it can develop into social media addiction—a behavioral addiction characterized by excessive use that disrupts crucial aspects of well-being, such as physical health, productivity, and interpersonal relationships.

Similar to substance use disorders, social media addiction is driven by neurobiological mechanisms that reinforce compulsive behavior. At the core of this addiction lies the brain's dopamine reward system, which is exploited by social media platforms through unpredictable rewards such as likes, comments, and shares that trigger dopamine release and reinforce habitual use (Cheng et al., 2022). Understanding how social media manipulates these neural pathways is essential for developing effective strategies to reduce dependence and restore healthy digital habits.

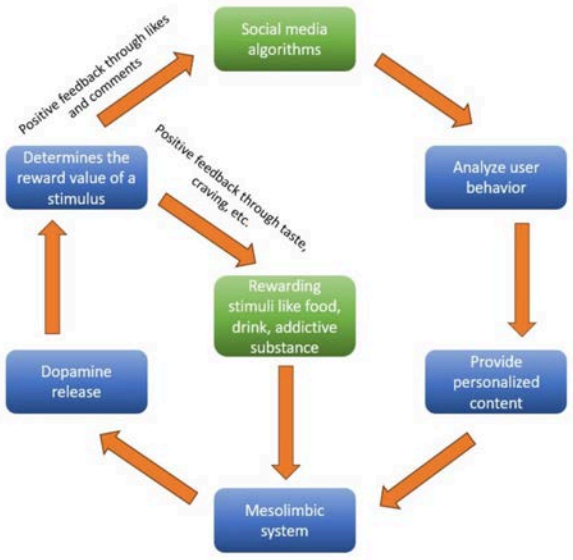
The Neuroscience of Social Media Addiction

To fully grasp the addictive nature of social media, it is important to explore the underlying neural mechanisms that sustain compulsive digital behaviors. This section delves into the neuroscience behind social media addiction, examining the brain's reward pathways, structural differences, and cognitive impacts.

Dopamine Reward System

Social media platforms exploit the brain's reward system, specially the mesolimbic pathway, by triggering dopamine release in response to engagement on accounts. Dopamine-driven reinforcement contributes to problematic behaviors including excessive social media use (De et al., 2025). Variable reward systems activate the brain's mesolimbic dopamine system, the same pathway involved in drug addiction. This system gauges the reward value of these social experiences and reinforces behavior that would lead to more pleasurable outcomes; this creates a cycle of reinforcement where users continue checking social media in hopes of receiving pleasurable stimuli. Moreover, the anticipation of these rewards can be more impactful than the rewards themselves. Over time, this pattern of expectation strengthens neural circuits associated with craving and compulsive behaviors (De et al., 2025). Highly active neural circuits reinforce the habitual use of social media and makes it even tougher to resist overuse.

fMRI studies reveal increased activity in the ventral striatum, a key reward-processing center, when individuals engage with social media, further reinforcing addictive tendencies (De et al., 2025).

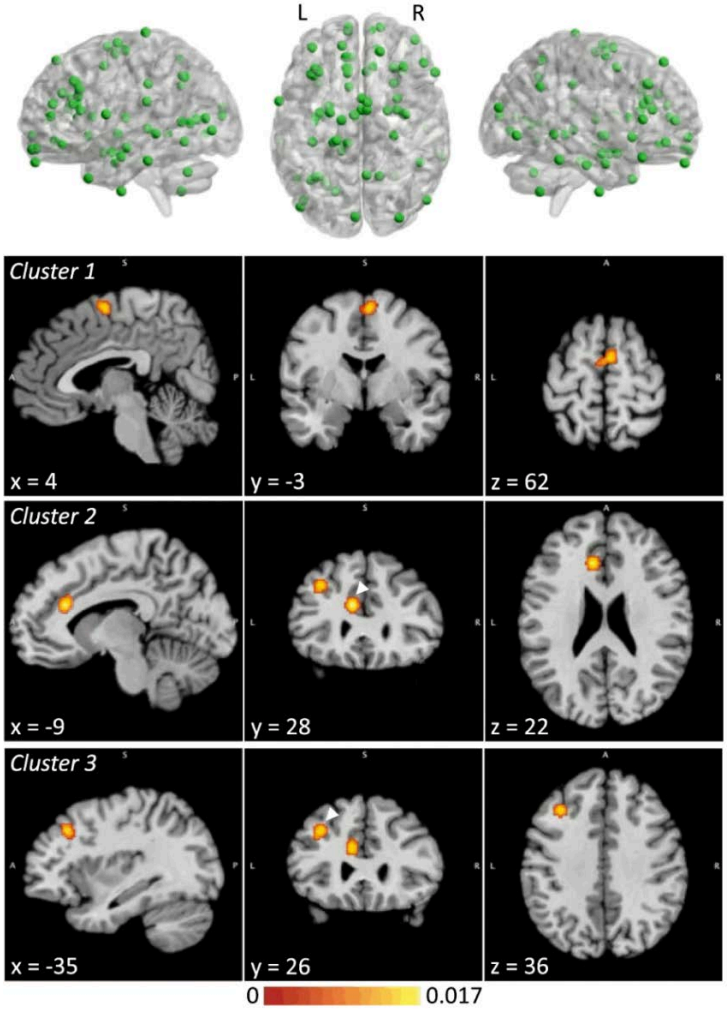


Impulse Control and Gray Matter Differences in the Brain

Gray matter is a crucial component of the brain that contains most of the neuronal cell bodies and plays a vital role in processing information, decision-making, and impulse control. Solly et al., (2022) found that chronic social media use may alter brain structures responsible for decision-making and self-control. For example, excessive social media engagement is linked to reduced gray matter volume in areas such as the left anterior cingulate cortex (ACC), supplementary motor area (SMA), and left dorsolateral prefrontal cortex (DLPFC). These regions play a crucial role in inhibitory control and impulse regulation. These alterations contribute to reduced inhibitory control, making it more difficult for individuals to regulate their social media consumption (Solly et al., 2022). Additionally, Solly et al. (2022) highlights that compulsive digital engagement leads to neuroplastic changes that reinforce habitual behaviors, further impairing self-regulation. Overall, chronic social media use can significantly impact brain structures involved in self-control, potentially creating a cycle of compulsive behavior and diminished ability to regulate usage.

Cognitive Overload and Impact on Attention

The constant multitasking required by social media—switching between apps, notifications, and conversations—overloads cognitive resources and weakens the brain's ability to sustain attention on a single task (Ophir et al., 2009). Over time, multitasking leads to decreased working memory capacity, making it harder to focus and engage deeply in offline activities. Neuroscientific research suggests that excessive digital multitasking affects the prefrontal cortex (i.e., the brain region responsible for executive function), attention control, and working memory (Ophir et al., 2009). Prolonged social media use has been associated with reduced efficiency in attentional networks, leading to difficulties in filtering distractions and maintaining sustained focus on cognitively demanding tasks. Together, these effects illustrate how habitual digital multitasking can compromise core cognitive functions, ultimately diminishing our ability to concentrate and perform effectively in everyday life.



Strategies to Break Social Media Addiction

Given the profound neurological and cognitive impact of excessive social media use, it is vital to explore evidence-based strategies for intervention. The following sections outline psychological and neurobiological approaches that have shown promise in reducing social media dependence and restoring healthy digital habits.

Cognitive Behavioral Therapy (CBT)

CBT is a well-established method for treating behavioral addictions, including social media dependence. CBT focuses on developing alternative coping mechanisms, delaying gratification, and utilizing self-monitoring tools to track and reduce usage (Dong et al., 2025). By identifying triggers and restructuring thought patterns, CBT helps individuals regain control over their digital habits and has been shown to be effective in addressing problematic internet use and social media addiction.

Individuals undergoing CBT interventions showed significant reductions in compulsive digital engagement and improvements in impulse control (Dong et al., 2025). These findings suggest that targeted behavioral strategies can help retrain the brain's response to social media stimuli and reduce dependence over time. CBT promotes healthier coping strategies, self-monitoring, and delayed gratification to manage digital habits—tools that directly counter the instant-reward mechanisms of social media platforms. By encouraging individuals to recognize and interrupt automatic thought patterns and behaviors associated with compulsive use, CBT helps break the cycle of addiction. Self-monitoring increases awareness of usage patterns, while learning to delay gratification reduces reliance on the immediate dopamine-driven rewards of likes, notifications, or scrolling. Together, these techniques support lasting behavioral change and greater psychological resilience in the face of digital temptations.

Neurofeedback Training

Neurofeedback training is a promising therapeutic approach for addressing social media addiction. By providing real-time feedback on brain activity, neurofeedback enables individuals to learn how to regulate neural responses to addictive stimuli, such as social media engagement (Marzbani et al., 2016). Neurofeedback training can enhance self-regulation and reduce compulsive behaviors, which are key factors in addiction. Marzbani et al. (2016) found that neurofeedback training significantly improved impulse control and reduced cravings in individuals with addictive behaviors, suggesting its potential applicability in treating social media addiction. Neurofeedback could help users break the cycle of compulsive social media checking by promoting healthier brain responses and greater control over digital impulses.

Cognitive Overload and Impact on Attention

A digital detox involves consciously limiting social media use to prevent excessive dopamine release (Anandpara et al., 2024). Specific strategies—such as turning off notifications, using digital wellbeing apps to monitor and limit screen use, and implementing tech-free hours—have been shown to improve sleep, reduce anxiety, and enhance emotional regulation (Hoeppner, 2024). Research highlights the effectiveness of digital detox strategies in improving mental well-being and reducing screen dependency. For example, limiting screen time through controlled interventions, such as setting time limits and engaging in offline activities, led to better emotional regulation and reduced reliance on digital devices (Anandpara et al., 2024). These findings suggest that digital detox strategies can help individuals break free from compulsive social media use, ultimately promoting healthier behavioral patterns and improving overall mental health.

Conclusion

Social media addiction alters key neural pathways related to reward, impulse control, and attention, making it difficult for individuals to break free from compulsive use. Central to this addiction is the brain's dopamine system, which is triggered by unpredictable rewards from social media engagement, reinforcing habitual use. However, emerging neuroscience-based strategies—such as cognitive behavioral therapy, neurofeedback training, and digital detox—offer promising solutions for retraining the brain and fostering healthier digital habits. By implementing these strategies, individuals can regain control over their online behaviors, reduce dependence on social media, and promote long-term well-being.

Despite these advancements, current research on social media addiction has notable limitations. Many studies rely heavily on self-reported data, which can be biased or inaccurate, and often lack longitudinal designs that are necessary to determine long-term neural and behavioral changes. Additionally, much of the existing research has focused on adolescents and young adults, leaving gaps in understanding how social media affects other age groups. The neurobiological mechanisms underlying digital addiction are still not fully understood, and interventions often lack standardization, making it difficult to assess their efficacy across diverse populations. Future research should aim to address these shortcomings by incorporating more objective neuroimaging data, expanding demographic diversity, and developing standardized protocols for intervention to better support sustainable behavioral change.

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About the Author

Meha Goswami is a sophomore majoring in Psychology, with an interest in double majoring in Molecular and Cellular Biology, and is on the pre-med track. Outside of Brain Matters, she is involved with Phi Chi, Delta Kappa Delta, and Illini Sheltering Hands Society, and she works as a research assistant in the Vision Lab. In her free time, Meha enjoys painting, listening to music, and spending time with her friends!

Curcumin and Glioblastoma: How Turmeric Can Be a Dietary Supplement Against Brain Cancer

Written by Kathryn Kennedy

Introduction

Imagine a brain tumor so aggressive that it can resist both chemotherapy and surgery, causing an alarming survival expectancy of only 12 to 18 months after diagnosis. This intimidating and complex disease is glioblastoma (GBM), the most common and deadliest brain tumor in adults (Yalamarty, et al., 2023). Researchers are urgently exploring new methods to prevent this serious disease. One particular intriguing dietary supplement researchers have studied as a means to reduce GBM tumor cell development is increased curcumin intake. Understanding how curcumin affects GBM cell mechanisms and how to incorporate turmeric into a diet are key components to taking advantage of this fascinating scientific breakthrough.

A Promising Supplement

Curcumin is a compound found within turmeric, a relatively accessible root vegetable native to India and Southeast Asia. Curcumin has been known to possess antioxidant, anti-inflammatory, neuroprotective, and antiproliferative properties, leading researchers to explore curcumin's potential anticancer mechanisms.

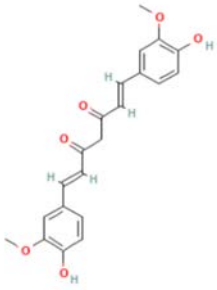

Figure 1. 2D Structure of Curcumin. National Center for Biotechnology Information.

In a recent meta-analysis of studies involving curcumin's specific role in preventing and mitigating the danger of GBM cells, Dr. Ângelo Luís et al. identified that a compelling attribute of curcumin is that it can target signaling pathways properties and can modulate pathways involved in GBM cell growth. Notably, curcumin impacts GBM cell proliferation, cell death, and tumor cell mobility, among other functions (Luís, et al., 2024). To come to this conclusion, Dr. Luís et al. analyzed the efficacy of curcumin on the tumor volume in animal subjects before and after curcumin consumption across 24 studies. Another study conducted by researchers Zexia Wang et al. at the Hubei

University of Science and Technology found that after comparing GBM tumor volume in mice before and after curcumin consumption, curcumin was effective at reducing the volume of the tumors. These results suggest that curcumin can be a useful supplement in preventing cancer cell growth, specifically for GBM (Wang et al., 2020). To better understand how curcumin has these effects, it is important to explore its molecular mechanisms.

Effect on Specific GBM Pathways

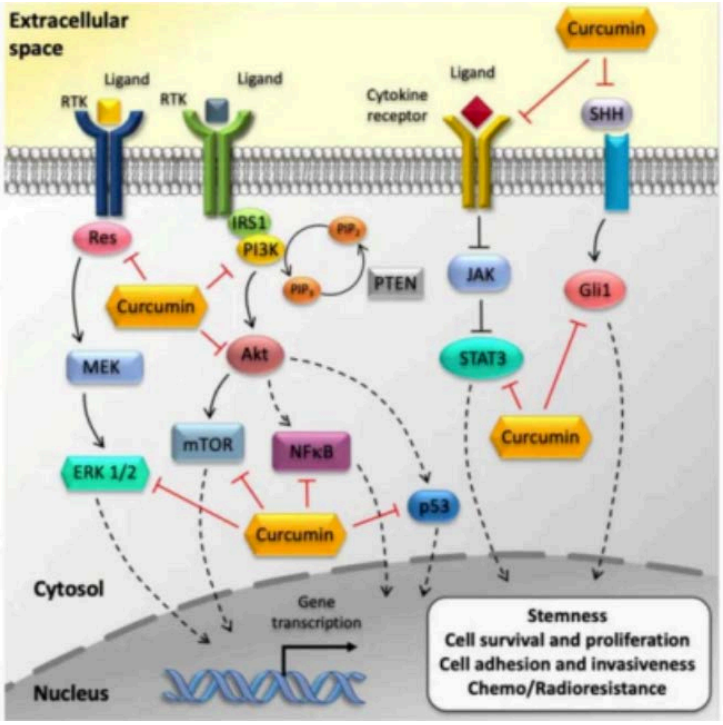

Figure 2. Effects of curcumin on GBM cancer stem cells (GSCs). Molecules.

Curcumin has been found to have pleiotropic effects, meaning it can modulate a number of signaling pathways, specifically major GBM pathways. It hinders cell growth and proliferation by inhibiting tumor-promoting pathways such as nuclear factor κB (NF-kB) and phosphoinositide 3-kinases/Akt/mammalian target of rapamycin (PI3K/Akt/mTOR). The NF-kB pathway also plays a role in enhancing proinflammatory genes, so curcumin's ability to inhibit this pathway contributes to its anti-inflammatory properties. As an antioxidant, curcumin protects cells from damage caused by free radicals, a major source of oxidative stress, which is known to activate the PI3K pathway. By reducing oxidative stress, curcumin is able to inhibit the Pl3K pathway, and therefore suppress tumor cell proliferation. The compound has also been shown to influence pathways involving cell cycle arrest, chemo-sensitizing effects, and cell migration and invasion (Ryskalin et al., 2020), though further research is needed to determine the specific quantity of curcumin necessary to maximize these effects.

Phytochemical Properties

Curcumin is also known to be a type of phytochemical, which are "plant-based bioactive compounds produced by plants for their protection... [that] can be derived from various sources such as whole grains, fruits, vegetables, nuts, and herbs" (Kumar, et al., 2023). As a phytochemical, curcumin has been widely studied for its potential properties associated with other phytochemicals, such as its benefits to counteract stress, mitochondrial damage, synaptic dysfunction, and neuro-inflammation. Phytochemicals also play a role in protecting against major diseases such as diabetes, obesity, cancer, cardiovascular diseases, and lung and prostate cancers. The vast health benefits of phytochemicals, and therefore curcumin, are fascinating, and stress the importance and reward of knowing how to effectively increase curcumin intake.

Figure 3. Ground turmeric. Marco Verch.

Incorporating Curcumin in a Diet

Knowing how to effectively incorporate curcumin into a diet is crucial for maximizing its vast health benefits. Mary-Eve Brown, an oncology clinical dietitian at Johns Hopkins Medicine, provides recommendations on how to safely increase curcumin intake through turmeric. She notes that consuming too much curcumin can be risky, so it is wise to avoid turmeric supplements and instead boost curcumin intake by including turmeric into meals. She recommends adding turmeric to stews, chilis, chicken soup, and making tea with turmeric root. She also encourages frequently finding and cooking healthy recipes online that contain turmeric as an ingredient (Brown, 2024).

GBM currently remains a dangerous and complex disease, and its resistance to current treatment stresses the urgent need for further GBM research and new prevention methods. The intriguing and relatively recent finding of curcumin's anticancer properties may be a promising step forward as a supplement to aid the prognosis of GBM. However, it is crucial to note that curcumin is not a

treatment for GBM – it cannot replace chemotherapy and surgery. Instead, it may complement these treatments as a dietary supplement. Further research in how this compound can target GBM signaling pathways can determine how to maximize its effect in humans. Though curcumin's full anticancer potential is unknown, taking advantage of its known properties can support health benefits.

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About the Author

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Understanding the Genetics of ADHD

Written by Meredith Kremitzki

Abstract

Attention Deficit Hyperactivity Disorder (ADHD) is one of the most common childhood disorders. Recently, it has been a source of debate and criticism in the media due to the increase in diagnoses. Despite the controversies surrounding overdiagnosis, treatment, and the disorder itself, one clear thing is that there is a genetic component to ADHD. In understanding ADHD as a whole, the discussion must start with the history and symptoms of the disorder, then focus on heritability, searching for a causal gene, and finally, analyzing possible genes of interest.

Introduction

Attention Deficit Hyperactivity Disorder (ADHD) has been described for centuries and is now the most common childhood disorder, affecting around 10% of the US population. ADHD has three core symptoms, which include inattention, hyperactivity, and impulsiveness that generally disrupt functioning (Mahone, 2017). The impairment must also be present in multiple environments (Holland, 2019). This disorder was initially named hyperactive/hyperkinetic syndrome in the 1980s, and the use of stimulants as treatment led people to believe that the root cause was some sort of brain damage. Then, as research into this disorder continued, the name evolved to Attention Deficit Disorder (ADD). With the publication of the DSM-III, it was finally renamed ADHD, which included the inattentive, hyperactive/impulsive, and combined subtypes with the DSM-IV. The DSM-V diagnostic guidelines include the age of onset being 12 years of age, with symptoms of inattention and/or impulsivity/hyperactivity being present. It also added an addendum where those over 17 could be diagnosed if they had five symptoms of inattention and/or impulsivity/hyperactivity (Mahone, 2017). Like many mental disorders, the definition of ADHD has changed over time, adjusting for new knowledge and research.

Heritability of ADHD

Research involving twin studies has found that the heritability is between 70-80%, and if a person has a first-degree relative with ADHD, they will have 5-105-10 times the risk of also developing ADHD (Mahone, 2017). In their paper, Faraone and Larsson review the data on twin, family, and adoption studies, as well as discuss genome-wide association studies, when discussing the heritability of ADHD (Faraone, 2019). One of the studies Faraone and Larsson reviewed encompassed a study of 894 people with ADHD who had siblings between the ages of 5-17. This study found that their rate of ADHD was 9 times higher than in those who did not have siblings with ADHD (Faraone, 2019). Additionally, adoption studies suggest that genetic factors could be more impactful than environmental factors. Then, when working with twin studies, researchers take advantage of the differences between monozygotic and dizygotic twins (Faraone, 2019). Monozygotic twins are commonly called identical twins, and they share 100% of their genetic makeup. On the other hand, dizygotic twins only share 50% of their DNA and are no more related to each other than a non-twin sibling would be. Overall, studies with both types of twins are very important in determining the heritability of a gene/disorder. Specifically, monozygotic twins help to compare the effects of the environment on the development of a disorder. In these twin studies, they estimated that the mean heritability of ADHD was 74% (Faraone, 2019). Faraone conducted a meta-analysis that included a Swedish study composed of 16,366 twins that found a strong connection between the extreme and subthreshold criteria of ADHD. The findings of Faraone 2019

are significant to the discussion surrounding heritability due to the amount of data they compiled into their meta-analysis.

Faraone and team compiled data around the heritability of ADHD from many different studies in Figure 1 of their paper. These studies had publication years from 1971 to 2017. These comparisons are significant as they show that the heritability of ADHD has been in scientific discussion for the past 50 years. Additionally, this comparison shows that all mentioned studies, with one exception, had a heritability of 0.6 or greater, indicating the heritability of ADHD to be greater than 50% at a minimum. This measure means that the differences seen are due to genetic factors rather than other factors. Additionally, this comparison strengthens the argument that ADHD has a strong genetic component.

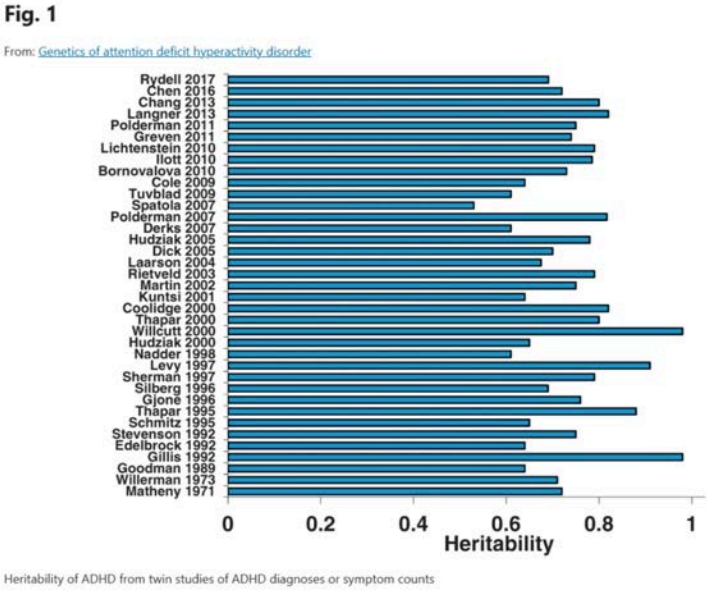

Figure 1. Bar chart showing the heritability of ADHD across multiple studies.

While the heritability of ADHD is well supported, it is also worth noting that a reporter effect has been seen in self-reporting. In this case, the incidence of self-rating of ADHD symptoms with different teachers of a twin pair showed lower heritability, around 30-40%, than with the same teachers of a twin pair showed a heritability around 70-80%. Of course, this difference could be because of different raters for each twin, which could have introduced effects where the rater experiences different ADHD symptoms (Faraone, 2019).

The Search for the ADHD Gene

After the positive results of the heritability of ADHD, the search for a gene began. This started with genetic linkage studies, which found linkage on chromosome 16. Then, using linkage across multigenerational populations, evidence was found indicating chromosomes 4, 5, 8, 11, and 17 (Faraone, 2019). Then, a candidate gene association study (CGAS) was done to try and find a specific gene. CGAS are studies that use knowledge about biology or biological function to target specific genes they hypothesize might be of interest. After doing a CGAS, there were 6 genes found: serotonin transporter 5HTT, dopamine transporter DAT1, dopamine receptor DRD4, dopamine receptor DRD5, serotonin 1B HTR1B, and a synaptic vesicle regulating protein SNAP25 (Faraone, 2019). What can be seen from these studies is that it is not known what exact gene causes ADHD, nor where this gene is located. However, the CGAS did give researchers some possible genes to start with for in their ADHD research.

Genome-wide association studies (GWAS) are similar to CGAS except that they look across the entire genome and see if there are genetic variations that are found in those with ADHD versus those without ADHD. A meta-analysis with 2455 controls, 896 people with ADHD, and 2064 trios of two parents and an ADHD child found no significant genes. Overall, GWAS shows that a significant portion of the heritability of ADHD was due to the influence of multiple genes that all have a small effect. A specific single-nucleotide polymorphism was found to have a heritability of 22%, making up a third of the heritability found in twin studies. This multi-gene theory was confirmed by using a polygenic risk score that predicted ADHD. In even more support for the multi-gene theory, it discusses how certain polymorphisms are located in places in the genome that are important for brain function (Faraone, 2019). The difficulty of a disorder like ADHD is that while multiple genes are likely to be involved, genetic studies are still unable to determine which genes are involved and to what extent.

Promising ADHD Genes

Despite the lack of specific data on the genetic cause of ADHD, there has been a lot of research investigating certain genes and pathways that could be involved. Bidwell et al. discuss three specific genes of interest: the dopamine receptor gene (DRD4), the dopamine transporter gene (DAT1), and the serotonin transporter (5HTT).

Bidwell et al. first start with the dopamine receptor gene, DRD4. The DRD4 gene is located on chromosome 11 and is interesting because DRD4 receptors are expressed in regions associated with attention and inhibition. Many studies have specifically looked into a 48-base-pair variable repeat polymorphism in a specific exon that codes for a loop around the receptor. Generally, the 4-repeat polymorphism is most common in the population, but the 7-repeat allele has been associated with ADHD. In response to this change, research has been focused on testing whether this difference in repeats has made a difference in the efficacy of drugs, but there have not been consistent

results in this kind of testing (Bidwell, 2011). Another reason that dopamine is of interest to those studying ADHD is because of how many brain functions dopamine signaling is involved in.

Another gene of interest is the dopamine transporter gene, which is located on chromosome 5. This gene is of specific interest because it is heavily expressed in the striatum, a region of the brain associated with attention, working memory, reward, and decision-making, where its main function is to reuptake dopamine. Additionally, this gene is interesting because dopamine transporters are the primary site that stimulants used to treat ADHD target. The most popular polymorphism is a variable repeat that is found in the untranslated region of the DAT1 gene. Untranslated regions are part of the genome that are not translated into protein, so these untranslated regions do not affect the protein of the dopamine transporter. However, the significance of this sequence is that it is believed to affect the expression of the dopamine transporter (Bidwell, 2011). Overall, this gene is significant because it can affect the dopamine levels in the brain regions associated with attention, memory, etc, which could cause some of the symptoms commonly seen in ADHD.

Another gene that Bidwell et al. focus on is the serotonin transporter gene (5HTT). Unlike the dopamine-associated genes, the role of serotonin has been less studied. Where this 5HTT has been promising is in animal studies that show serotonin having a key role in regulating things like attention. Additionally, when this transporter has been disrupted, there has been an increase in hyperactivity in mice. This is interesting, as one of the hallmarks of ADHD is hyperactivity. Similar to the DAT1 gene, the polymorphism of interest is associated with changes in transcription and activity of the transporter. Specifically, this polymorphism is a 44-base pair deletion in the promoter region of the gene. The function of promoter regions is to regulate the transcription of specific genes. With this specific change, researchers see less transcription and a reduced amount of the transporter itself (Bidwell, 2011).

Given the data on the three genes, Bidwell et al. performed a family-based association test (FBAT) on the polymorphism surrounding the DRD4, 5HTT, and dopamine transporter genes. They then performed testing to determine how often that variance was associated with either overall ADHD or a certain symptom, like inattention or hyperactivity/impulsivity. This data can be seen in Table 4 from their paper. What they found was that the DRD4 gene was statistically significant for all types of ADHD compared to. Additionally, they found statistically significant results with the dopamine transporter gene when comparing against total ADHD and the inattentive phenotype. However, this result was not seen in the hyperactive/inattentive phenotype (Bidwell, 2011). These results from Bidwell et al. are significant as they could give further insight into a specific polymorphism in a particular gene that is associated with ADHD. It also provides a guide for further studies to repeat this analysis and to compare with different polymorphisms.

Table 4 Results of FBAT statistics for each candidate polymorphism and the total ADHD and dimensional symptom phenotypes

From: A Family Based Association Study of DRD4, DAT1, and 5HTT and Continuous Traits of Attention-Deficit Hyperactivity Disorder

Polymorphism	Informative families	FBAT-GEE					
		Total ADHD		Inattentive		Hyperactive/impulsive	
		p-value	Variance explained	*p*-value	Variance explained	*p*-value	Variance explained
DRD4 exon III VNTR							
4-repeat	160	.007 *	.01	.007 *	.01	.003 *	.01
7-repeat	122	.05 ~	.01	.21	.01	.08	.01
DAT1 3′ UTR VNTR							
9-repeat	139	.03 ~	.00	.009 ~	.01	.57	.00
10-repeat	141	.02 *	.01	.005 *	.02	.47	.00
5HTTLPR							
Short	110	.23	.00	–	–	–	–
Long	110	.23	.00	–	–	–	–

"+" Indicates allele is positively associated with phenotype, "−" indicates allele is negatively associated with phenotype
Bold indicates statistical significance at a level of $p < .05$

Figure 2. Table with FBAT results for each polymorphism and related ADHD phenotype with significance value.

Despite any controversies surrounding ADHD, the research has shown that this is a multifaceted disorder. Unlike many other disorders or illnesses, no one gene can explain the cause of ADHD. However, whatever factors influence this disorder have a strong genetic component that can be observed in heritability studies. The many different hypotheses of the root cause of ADHD include problems with dopamine and serotonin. Further research could test combinations of genes and ADHD. These studies could investigate whether a change in activity in both the dopamine and serotonin genes is correlated with increased ADHD symptoms. This could help understand the likely polygenic aspect of ADHD.

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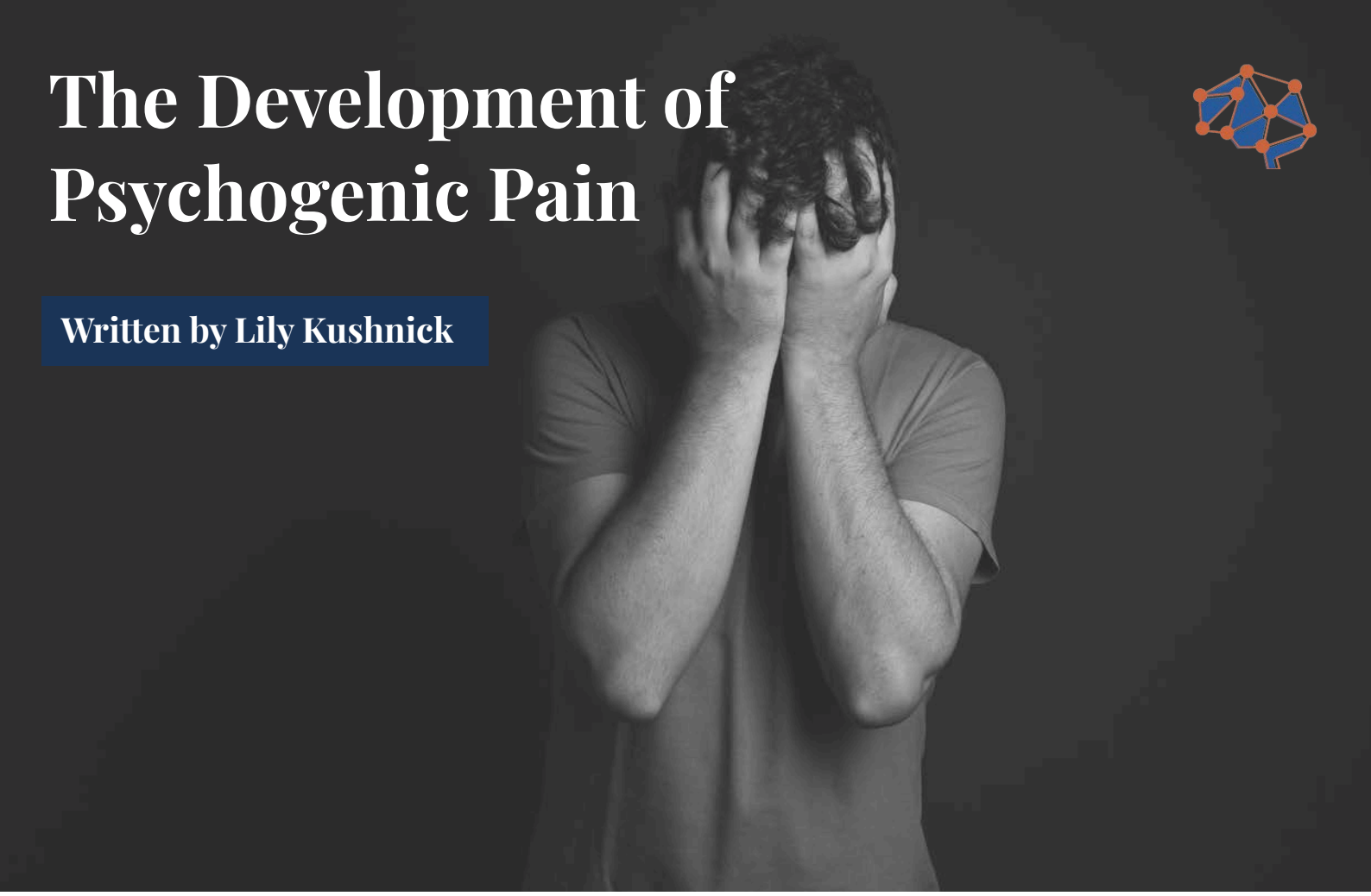
About the Author

Meredith Kremitzki is a junior at the University of Illinois, majoring in psychology with a concentration in cognitive neuroscience and a minor in integrative biology. She became involved with Brain Matters to learn more about the different topics in neuroscience. Along with writing for Brain Matters, Meredith is a laboratory teaching assistant for the chemistry department. She hopes to become a doctor and continue learning about the brain and body.

The Development of Psychogenic Pain



Written by Lily Kushnick

Introduction

Pain is a complex experience, but it can be better understood when divided into three broad categories: nociceptive pain, caused by tissue damage, neuropathic pain, resulting from nerve damage, and the newly recognized psychogenic pain (also known as nociplastic pain). Nociplastic pain refers to physical pain caused or increased by psychological, emotional, or social factors rather than physical or neurological damage. Nociceptors (i.e. pain receptors) release neurotransmitters to the thalamus and other parts of the brain through the nervous system. Pain manifests as a physical and subjective experience. Despite the lack of a clear physical cause, and though some dismiss it as entirely psychological, psychogenic pain remains a genuine condition as it is both physical and psychological. While there is no clear underlying physical cause, and although some people may discount it being all in a person's head, psychogenic pain is still real.

The Development of Pain

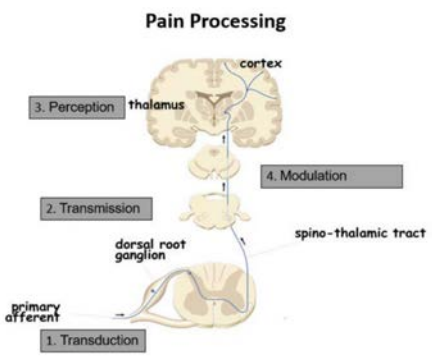


Figure 1. Pathway of pain signaling and processing (NIH, 2022).

There are four major processes in the development of pain. It starts with transduction, which refers to the activation of pain receptors in response to stimuli—either mechanical (i.e. pressure), heat, or chemical. Next, transmission involves the nociceptive message being sent from the peripheral nervous system (PNS) to the central nervous system (CNS). These nociceptive messages are encoded in the patterns and frequency of impulses from the nociceptor. Along this pathway, modulation occurs, altering the pain signals as they travel. Modulation is one of the reasons people experience pain in different severities, even with similar stimuli (Kirkpatrick et al., 2015). For example, the activation of nociceptors may not always lead to a sensation of stronger pain due to modulation. Finally, perception occurs and is the cumulative subjective experience resulting from an array of sensory signals. This step includes the attention, expectation, and interpretation of the pain messages and cannot be objectively measured unlike the other neural processes.

For instance, when you stub your toe, nociceptors are activated in response to a mechanical stimulus. The message would travel through the anterolateral system (a sensory pathway that carries information about stimuli such as temperature and touch) in the spinal cord and then to the brain (specifically, the thalamus then to various areas of the

cerebral cortex) as an electrical impulse, which is interpreted and experienced as pain (Institute of Medicine (US) Committee on Pain, Disability, and Chronic Illness Behavior et al., 1987)

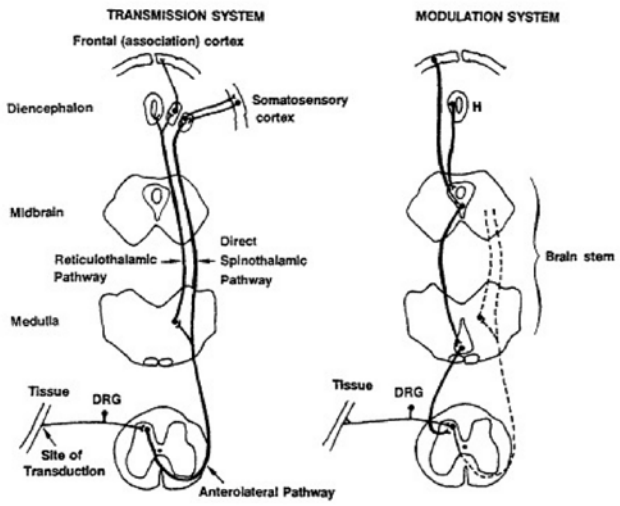


Figure 2. The major neural structures relevant to pain and pathway/development of pain from nociceptive transduction of stimulus from tissue. In psychogenic (nociplastic) pain, there is no physical stimulus (Institute of Medicine (US) Committee on Pain, Disability, and Chronic Illness Behavior et al., 1987).

The Detection of Pain

Nociception—the process by which the nervous system detects painful stimuli—is crucial to understanding psychogenic pain and the mechanisms behind pain in general. Nociceptive pain refers to the activation of nociceptors in response to actual or threatened tissue damage. On the other hand, nociplastic (psychogenic) pain arises from the activation of nociceptors without the presence or clear threat of physical damage (Milner & Doherty, 2015). In this case, the excitation of nociceptors is mediated by retrograde activation by messages from the sympathetic nervous system (SNS). This means that the postsynaptic neuron, the cell receiving a signal, sends information back to the presynaptic neuron, the cell sending a signal. Nociceptors detect harmful stimuli and signal the CNS which causes the sensation of pain. So, retrograde activation in this context would involve the injured areas sending signals back to the nociceptors (Tao & Poo, 2001). Another way nociceptors could be activated is by reflex muscle tension. Prolonged muscle tension is often accompanied by increased sensitization of nociceptor terminals in muscles (Isagulyan & Kashcheev, 2022). Muscle tension is a reflexive response to stress and can significantly decrease the mechanical threshold for nociceptor activation in muscles (Chen et al., 2011). Essentially, elevated and extended periods of stress heighten muscle tension which generally lowers pain tolerance or increases sensitivity to stimuli.

Factors Effecting the Development of Psychogenic Pain

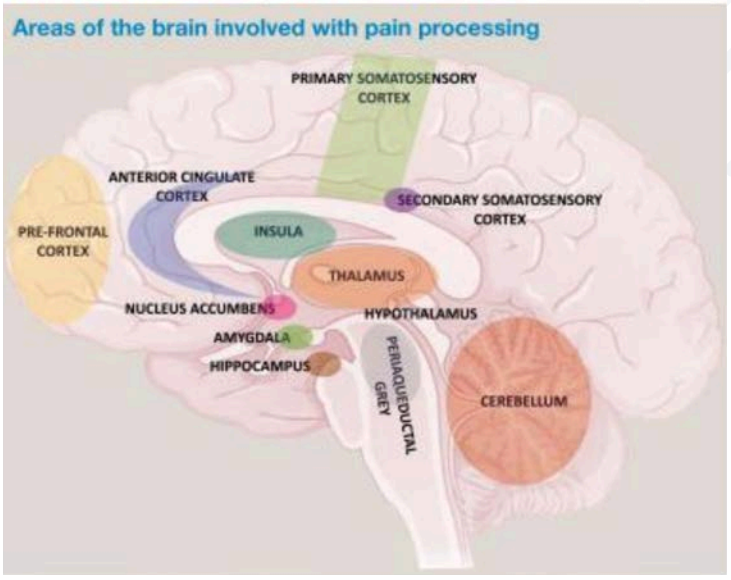


Figure 3. Areas of the brain involved with pain processing (Gore, 2022).

There are a few proposed mechanisms for the development of psychogenic pain. Pathophysiologically, one mechanism pointed to is hyperresponsiveness to pain stimuli. Nociceptors may become more sensitive to stimuli and lower the threshold for activation, causing even seemingly nonpainful stimuli to produce pain (Bułdyś et al., 2023). The extent of nociceptor activation determines the input the CNS receives which determines the severity of pain experienced. Nociceptors sensitize, meaning their excitability can increase. As a result, there is a reduction of the threshold and an increase in magnitude of response to a stimulus (Gold & Gebhart, 2010).

Another potential influence could be hyperactivity and connectivity between regions of the brain responsible for perceiving pain, such as the medial prefrontal cortex (mPFC), anterior cingulate cortex (ACC), thalamus, and somatosensory cortex, which would also cause an amplified response to pain signals. Decreased activity and connectivity in the regions of the brain such as the rostral ventromedial medulla (RVM) and periaqueductal gray (PAG) can impact the development of psychogenic pain as well (Bułdyś et al., 2023). The PAG activates a pain inhibitory system and influences pain modulation by its connections with the RVM, which can both facilitate and inhibit nociceptive inputs (Ossipov et al., 2014).

Additionally, psychogenic pain may be caused by the nervous system's recognition of pain that has already healed, supporting the idea that confused signaling may be a part of the cause (Moini et al., 2023). Pain plasticity—the adaptive processes of the nervous system in response to pain stimulus—may lead to changes in nociceptors, causing them to activate atypically. Because of this plasticity

plasticity, signaling is amplified and a "pain memory" is formed (Price & Inyang, 2015). It is most likely a combination of these factors as many regions of the brain (ACC, mPFC, thalamus, somatosensory cortex, RVM, and PAG) work together to create the experience of pain.

Other important factors are cognitive and psychological. Psychogenic pain is commonly associated with psychosocial and emotional conflicts, as the brain can interpret mental distress as physical pain (Moini et al., 2023). Chronic stress can trigger or exacerbate pain because it can contribute to the sensitization of nociceptors, causing the brain to become hypersensitive to pain signaling (Hannibal & Bishop, 2014). In addition, problems with emotional regulation may lead to somatization—the expression of emotional/psychological conflicts as physical (somatic) symptoms (Lumley & Schubiner, 2019). Generally, positive emotions inhibit pain while negative emotions facilitate it. Issues with regulating negative emotions can heighten the amplification of pain signaling (Toledo et al., 2024).

The idea of "catastrophizing" can also be associated with the development of psychogenic pain. People who tend to catastrophize (i.e. exaggerating negative mentality) are more likely to experience more intense pain and have more difficulty managing it. This is linked to activation of PFC, ACC, and amygdala, which is involved in perception of pain and emotional regulation (Sullivan et al., 2000).

Conclusion

Psychogenic pain most commonly manifests as headaches, stomach aches, and back pain, and is overall commonly associated with mental disorders like depression and anxiety (Galli, 2023). However, psychogenic pain is very complex and still not fully understood, so there is not a standard diagnosis or treatment of the condition yet. Due to its novelty, misunderstanding, and the general dismissive attitude towards it up until only recently, many patients suffering from psychogenic pain have not received the attention they deserve. With many technological advancements and development of new research, new techniques for diagnosis, treatment and prevention of psychogenic pain are not far out of reach. In the meantime, a greater emphasis on recognizing and accepting this condition and approaching it with compassion and an open mind is crucial to ensuring patients receive the appropriate care.

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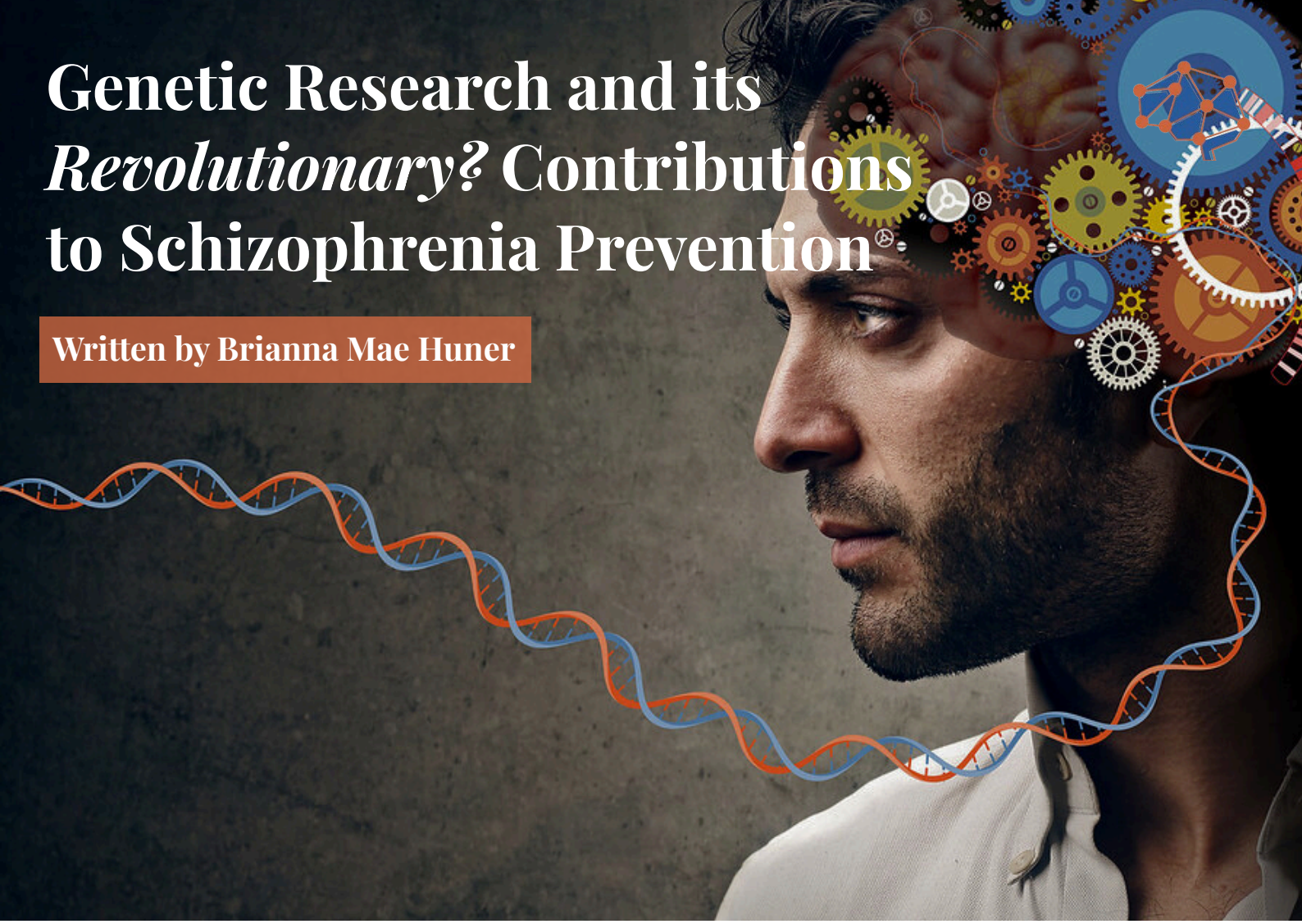
Genetic Research and its *Revolutionary?* Contributions to Schizophrenia Prevention



Written by Brianna Mae Huner

Introduction

For over a century, physicians, neurologists, psychiatrists, biologists, and eventually geneticists have been working hard to answer the question: what causes schizophrenia and its related disorders? In earlier days of research, much of the focus was placed on the treatment of such conditions, and scientists have made great advancements in this area. For example, in the early 19th century, it was typical to see patients with schizophrenia exhibit catatonia, which is a symptom characterized by a decrease in reactivity to environmental stimuli. These patients were stuck within these states for days or weeks. Now, catatonia is treated with benzodiazepines, and patients can return to non-catatonic functioning over a much shorter period of time. Sienart et al., 2014). This pharmacological discovery has improved the lives of schizophrenia patients dramatically and has shifted the typical symptomology observed in this disorder. Yet, the question remains: what causes this disorder, and can we use that knowledge to prevent its development in the first place? Investigations into the etiology (or causes) of schizophrenia have proven to be less fruitful than their treatment-based counterparts. The knowledge that schizophrenia is at least partially genetic motivated a surge of investigations into the human genome. This research began with studies using molecular genetics, a method proven to be incredibly effective in discoveries into etiology of other genetic diseases. With the completion of the Human Genome Project (HGP), genome-wide investigations came to the forefront. It was initially projected that the data collected from the HGP would revolutionize the screening, diagnosis, and treatment of mental illnesses. However, 20 years after this prediction was made, the etiology of these conditions remains unclear, as the nature of these illnesses has been found to be difficult to discern through genetic studies.

Schizophrenia Spectrum Disorder (also known as schizophrenia) is characterized by symptoms of three types: positive, negative, and disorganized symptoms, with a poor prognosis and heavy cost toward the individual, their family, and society. Positive symptoms are referred to as such because their symptoms add characteristics that are not already present in normal functioning, such as hallucinations or delusions. Negative symptoms are the opposite, “taking away” from normative functioning, such as avolition (lack of motivation) and diminished emotional expression. Disorganized symptoms refer to a lack of order and form in terms of thinking or activity, such as disorganized speech and motor behavior (American Psychological Association, 2022). Schizophrenia exists on a spectrum of disorders and is believed to be at least partially caused by genetics. The risk of developing schizophrenia is linked to the degree of relation to a family member with schizophrenia. As seen in Figure 1, a first degree relative to a patient with schizophrenia would have a 3.0% hospitalization rate for psychosis, and a second degree relative would have a 2.2% hospitalization rate, both

compared to the general population hospitalization rate of 0.9% (Karlsson, 1971). This disorder can be debilitating, often requiring full-time hospitalization and lifetime treatment. Many patients never return to normal functioning, even between episodes. There is also a higher risk of suicide and early death associated with this disorder (Jobe & Harrow, 2005). The severity of such a disease, combined with its proven genetic component, has driven large-scale research into the etiology of schizophrenia at the order of the genes, producing a myriad of results.

Relationship	Population born 1881–1910		Population born 1911–1940	
	N	Hosp rate	N	Hosp. rate
First degree relatives	*1547*	*5.7*	*492*	*3.0*
Fathers	159	2.5	- -	- -
Mothers	165	7.9		
Brothers	571	5.3	198	3.5
Sisters	511	6.1	179	2.8
Sons	73	5.5	57	1.8
Daughters	68	8.8	58	3.4
Second degree relatives	*726*	*3.5*	*511*	*2.2*
Uncles	242	2.1	-	- -
Aunts	207	4.8		
Nephews	139	1.4	269	1.9
Nieces	138	5.8	242	2.5
Third degree relatives	*879*	*2.2*	*1206*	*0.6*
Male 1st cousins	441	1.4	608	0.5
Female 1st cousins	438	3.0	598	0.7
General population	*6700*	*1.4*	*14447*	*0.9*
Males	3456	1.1	7330	0.8
Females	3244	1.8	7117	0.9

Table 1. Rates of hospitalization with functional psychosis in relatives of psychotic index cases in Iceland.

Research Method #1: Molecular Genetics

Biologists have been using the varying tools at their disposal over time to investigate the etiology of genetic diseases, with one of these methods being molecular genetics. This research revealed key insights into the fields of biology, neurology, and genetics, though it was not as comparatively impactful in psychiatry. Molecular genetics, a field of research that studies genes on a molecular level, focuses on variations in DNA. Studies using this framework have shed light on the genetic mutations behind a number of neurological disorders, such as Huntington's disease (HD), Fragile X Syndrome (FXS), and Amyotrophic Lateral Sclerosis (ALS) (Cowan et al., 2002). These discoveries allowed for improved screening and early identification of these conditions. However, studies using molecular genetics to find the causes of mental illnesses gave mixed results of small effect sizes. This difference can be explained through the nature of these conditions, particularly within the inheritance patterns of the disorder whose causes were found.

Many Americans are made aware of Mendelian patterns of inheritance, though they may not know it by that name, while taking high school biology, through lessons about yellow and green peas and Punnett squares. Classic Mendelian patterns of inheritance involve patterns of segregation such as dominant, recessive, and X-linked (Chial, 2008). One can follow along using genetic information to make reasonable predictions about the phenotype, such as hair or eye color, of an organism by simply examining their pedigree. The diseases with causes found through molecular genetics followed Mendelian patterns of inheritance, such as Huntington's disease, whose pattern of inheritance can be observed in Figure 2. By comparing the genes of people who both do and do not have a familial disorder, target genes can be identified. Additionally, disorders such as HD and ALS are caused by a single defective gene that can be isolated and identified relatively easily. On the contrary, mental illnesses do not follow Mendelian patterns of inheritance. They are likely polygenic (caused by multiple genes) and multifactorial (caused by genetic and environmental factors, as well as interactions between genes and environment). By using these methods of searching and comparing target areas of the genome, there were genes identified as possibly being involved in mental illnesses, but the results weren't as clear-cut as finding one gene that causes a certain disorder.

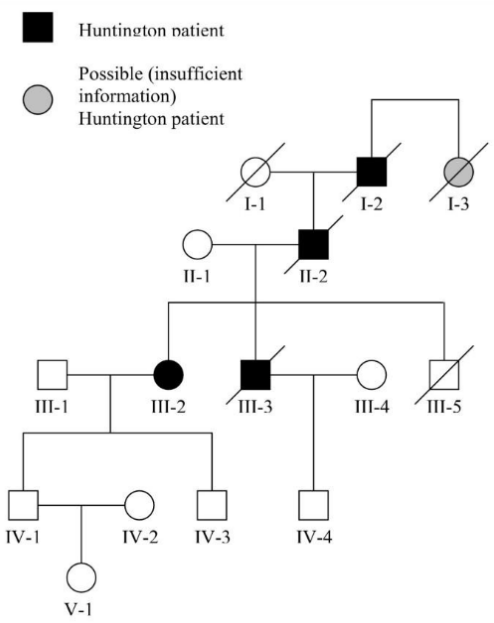


Figure 1. Correa & Guimaraes (2006). Pedigree showcasing the inheritance of Huntington's Disease (HD).

While investigating the human genome to find associations between certain genes and specific psychiatric disorders, a number of loci were identified. Linkage analysis was often used in these studies prior to the completion of the Human Genome Project, which uses genetic markers to find areas on the genome that are close to genes thought to cause certain conditions (Pulst, 1999). Some well-known findings from analyses looking into schizophrenia identified chromosomal regions associated with the serotonin 5HT2A receptor gene (13q14.1-32), as well as chromosomal regions related to synapse-related genes (22q11-12) (Cowan et al., 2002). These findings garnered interest, considering the importance of synapses in neuronal function and serotonin's role in mood regulation and homeostatic roles (Mohammad-Zadeh & Bryant, 2008). With the advent of new

techniques and technology, it was believed that we would be able to find clearer answers in these areas and their connection to schizophrenia.

> **One can follow along using genetic information to make reasonable predictions about the phenotype, such as hair or eye color, of an organism by simply examining their pedigree.**

Research Method #2: Data Analysis from The Human Genome Project

The Human Genome Project (HGP) was a moon-shot project, with the goal of sequencing the entire human genome, and its completion created a new wave of data and discoveries into the field of genetic biology. Launching in 1990 and concluding in 2003, the HGP cost $3 billion (Gannett, 2023). The ability to map the entire human genome created a vast bank of data that could be utilized when researching, for example, cancer and its treatment (Rood & Regev, 2021). It was predicted that the discoveries resulting from the HGP data would allow for gene-based treatments for critical disease such as diabetes and hypertension, as well as change the game for the treatment of mental illnesses. Some scientists went so far as to say that the impact the HGP would have on the field of medicine would be comparable to the discovery of antibiotics (Torrey, 2024). Genome wide association studies (GWAS) were conducted using the new data, and nearly 300 single nucleotide polymorphism (SNP) genetic loci were linked to an increased risk of the development of schizophrenia. Such findings seemed to signal that major breakthroughs were on the way.

Unfortunately, much of the evidence uncovered through GWAS did not hold up under scrutiny. Through comparisons between studies, about half of the genomic loci associated with schizophrenia was discovered to also be associated with other disorders believed to be partially genetic, such as bipolar disorder and autism (Torrey, 2024). Therefore, an argument can be made that these loci are associated with psychiatric disorders as a whole, rather than schizophrenia specifically. Additionally, many of the SNPs identified were found to have a very small effect size, and while they could be linked to schizophrenia, they could not be linked to a cause of schizophrenia. This bears resemblance to how a reduction in size of the frontal lobe is associated with depression, but it is not known if this reduction causes depression, or is caused by depression (Joseph et al., 2025). Despite these discouraging findings, there may be one with promise.

A change in the region of the genome known as the Major Histocompatibility Complex presents strong evidence of an association with schizophrenia, potentially revealing a link between the disorder and the immune system. The Major Histocompatibility Complex (MHC), located on chromosome 6, is associated with the regulation of immune functions, as well as many infectious diseases and autoimmune disorders (Abualros et al., 2021). A GWAS revealed an association between a small alteration in this area and the development of schizophrenia (Caseras et al., 2024). While this finding may initially seem confusing, it does connect some previous findings and hypotheses about schizophrenia. The presence of some infectious diseases during pregnancy, such as influenza, though new evidence reveals that more research is needed in this area (Fung et al., 2022), and Toxoplasma gondii (Yang et al., 2024), are known to slightly increase the risk of the child developing schizophrenia. The MHC's association to autoimmune disorders could also shed light onto the hypothesis that differences in the functions of microglia (the "brain's immune system") could explain some of the symptoms of schizophrenia, as it is hypothesized that essential synapses could be erroneously tagged for consumption by microglia as a part of the brain's synaptic pruning process used to clear unused and unneeded synapses (Li et al., 2023). This incorrect deletion of synapses could explain the cognitive and disorganized issues often associated with schizophrenia, as vital neuronal pathways could be disrupted. Unfortunately, this finding only had a polygenic risk score, which gives an estimate of an individual's genetic risk for developing a specific trait, to explain less than 10% of the variation in liability for the disease (Andreassen et al., 2023). While these findings are promising, they are far from the revolution that was predicted.

Future Directions

Despite the lack of definitive findings through genetic studies, there may still be something to be gleaned from this data. The results of the HGP still have hopes of finding significant results, as researchers hope to use newly introduced machine learning functions to aid in their search. They are also making efforts to factor in potential epigenetic changes caused by environmental factors and gene x environment interactions, or perhaps the solution lies in something that hasn't been considered yet.

It is possible that the flaw in this system is the question itself. It is widely accepted that there is no single or primary cause of many mental disorders, and it is possible that these disorders are the result of multiple different pathways. To explain this, we may look toward an example in the medical world: obesity, and its association with Leptin and Leptin genes. Leptin is a hormone that signals satiety, telling the body when it should stop eating. Leptin receptor deficiency is a rare genetic disorder which causes a mutation in the Leptin gene that results in an inability for the body to produce Leptin. Without a satiety signal, the body's ability to regulate eating is compromised, and the subject becomes

obese. However, when this subject is treated with Leptin supplements, their food intake regulation ability is restored, and they return to a normal weight. In this case, this mutation causes obesity. But not all cases can be explained this way. Leptin treatment only works on obese patients who have this mutation, and if they do not, the treatment produces little effect (Milan et al., 2021). Obesity has multiple causes, and treatment must be derived based on the specific cause. Could a similar approach be applied to schizophrenia, where different treatments could be used for different causes? Could we pinpoint these different causes?

One of the most important traits needed in research is curiosity. While many may be discouraged by the lack of major breakthroughs in the genetic research of schizophrenia and other psychiatric disorders, others may see this as eye-opening, and an invitation to think nontraditionally about these findings. While it was originally believed that genetic research such as molecular genetics, linkage analysis, genome wide association studies, and more would be the key to understanding the etiology of schizophrenia, this was found to be inaccurate, but that doesn't mean that all hope is lost. All that is needed is a new question to be asked.

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Pathophysiology of Postpartum Depression: Etiology and Interplay of Structural and Functional Brain Changes

Written by Sylvia Merz

Abstract

Postpartum depression (PPD) affects a significant portion of new mothers, leading to severe disruptions in maternal mental health, such as persistent feelings of sadness, anxiety and emotional numbness. These symptoms not only hinder the mother's well-being but also interfere with critical maternal-infant bonding and early caregiving, which can have lasting developmental consequences for the child. Despite the well-documented emotional and cognitive consequences of PPD, the neurobiological mechanisms underlying this condition remain are still not fully understood. Structural and functional brain alterations in areas such as the prefrontal cortex (PFC), hippocampus, and amygdala have been implicated in the development of PPD. Neuroimaging studies offer promising insights into the brain changes associated with this mood disorder. Understanding these modifications could pave the way for earlier identification and more targeted interventions to improve maternal mental health outcomes.

Introduction

Postpartum depression (PPD) is a major mood disorder that affects approximately 10-15% of new mothers, manifesting as mood disturbances, cognitive impairments, and difficulty bonding with the infant (Epperson et al., 2014). These mood disturbances, which can include persistent sadness, irritability, and anxiety, often last for months and, in some cases, may continue for years (Leight et al., 2020). The consequences of PPD extend beyond the individual, impacting child development and family dynamics, and it has long-lasting effects that can persist well beyond the postpartum period. Studies have shown that untreated PPD is associated with negative outcomes in child development, including delays in emotional and cognitive development (Stewart et al., 2018). Furthermore, the effects of PPD can persist well beyond the postpartum period, with women reporting increased risks of future depressive episodes and impaired functioning in social and occupational domains (Elliott et al., 2021). Despite its prevalence, the underlying neurobiological mechanisms that drive PPD remain poorly understood, creating challenges in early diagnosis and treatment. Recent advancements in neuroimaging have provided insights into the brain changes associated with PPD. Much like major depressive disorder (MDD), PPD involves changes in the structure and function of brain regions implicated in emotional regulation, memory, and stress processing (Gingnell et al., 2018). PPD has been associated with alterations in prefrontal cortex (PFC) connectivity and amygdala hyperactivity, which are both implicated in mood regulation and stress response (Stewart et al., 2019). Understanding these brain changes in PPD may offer clues to how this disorder develops and, more importantly, provide opportunities for earlier identification and more personalized interventions.

Structural Brain Changes in PPD

Research has shown that PPD is associated with significant structural brain changes, particularly in regions critical for emotional regulation and memory processing. One of the most consistent findings is a reduction in gray matter volume, with studies reporting up to a 9% reduction in PFC volume and an 11% reduction in hippocampal volume in women with PPD compared to healthy controls (Epperson et al., 2014). The PFC plays a crucial role in executive functions such as decision-making, emotional regulation, and social behavior, and its atrophy may contribute to the impaired emotional regulation seen in PPD (Weber et al., 2012). By contrast, the hippocampus is involved in memory processes and stress regulation, and loss of volume in this area may be associated with an impaired ability to cope with the stresses of motherhood, increasing vulnerability to depression (Epperson et al., 2014).

In addition to gray matter changes, white matter abnormalities have been observed in PPD. Studies have identified a 15-20% reduction in white matter integrity in emotion-regulation pathways between the PFC and other brain regions, which may exacerbate difficulties in regulating emotional responses to stress (Han et al., 2014). These structural changes represent significant brain damage, as they involve the loss of neurons and the connections between them, leading to a decrease in brain volume—a condition known as focal brain atrophy (Cleveland Clinic, 2022). Such damage underscores the severity of PPD's impact on the brain's physical structure. The disruption of the brain's network integrity in PPD makes it challenging to restore normal function once these alterations occur. The long-term consequences of such disruptions include increased vulnerability to recurrent depressive episodes and the potential for chronic mood disturbances (Lisofsky et al., 2018). This heightened risk emphasizes the importance of early detection and intervention in PPD to prevent enduring neurological and psychological impairments.

Functional Brain Alterations in PPD

Functional brain modifications in PPD have been characterized by disruptions in brain networks involved in self-referential processing and emotional regulation. One such network is the Default Mode Network (DMN), which is active during self-referential thoughts and mind-wandering, and disruptions here may contribute to the ruminative thought patterns often seen in PPD (Gingnell et al., 2018). Notably, rumination is also a core feature of obsessive-compulsive disorder (OCD), raising the possibility of shared cognitive vulnerabilities between the two conditions. Emerging research suggests that perinatal OCD often co-occurs with PPD, with overlapping symptomatology, including intrusive thoughts and compulsive worry (Russell et al., 2013). This potential comorbidity highlights the need for further investigation into the common neural mechanisms that may drive maladaptive thought patterns in both disorders.

Another area of concern is the amygdala-PFC circuitry. The amygdala, which is responsible for processing emotional responses, shows hyperactivity in PPD, particularly in response to emotionally salient stimuli (Weber et al., 2012). This heightened amygdala response, coupled with hypoactivity in the PFC (which is responsible for regulating emotional responses), impairs the ability to modulate emotions and results in exaggerated feelings of fear and anxiety. This dysregulation in neural circuitry may underlie the emotional volatility and heightened stress sensitivity observed in individuals with PPD, contributing to difficulties in both emotional self-regulation and maternal-infant bonding.

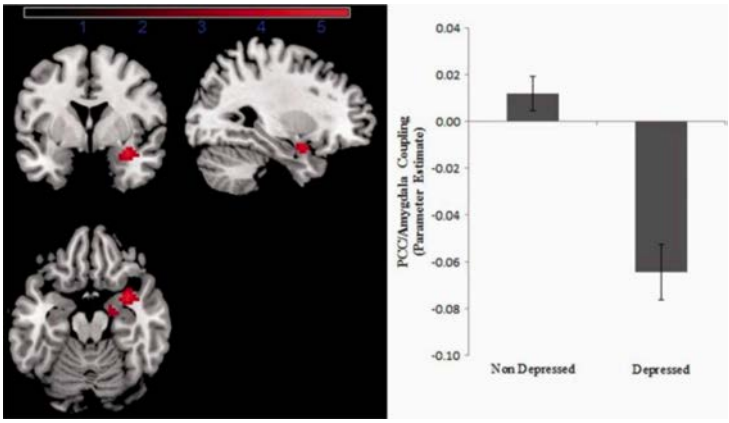


Figure 1: Research by Chase et al. (2013) displayed weaker connectivity between the PCC and right amygdala in depressed vs. healthy moms (peak: 33, 5, −20; P = 0.043, FWE). This area overlaps with the basolateral and superficial amygdala. Bar graph shows average connectivity levels (± standard error).

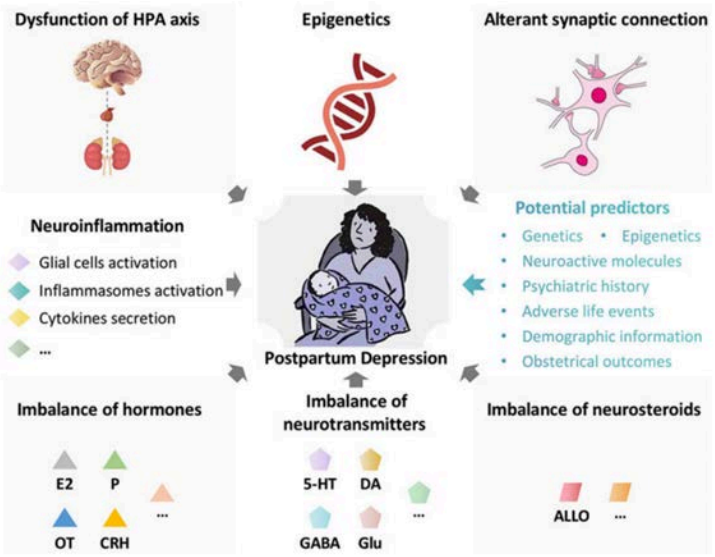
These functional brain alterations align with the broader symptomatology of PPD, which extends beyond mood disturbances. While PPD is classified as a major depressive disorder with peripartum onset, its clinical presentation frequently includes heightened anxiety, excessive worry, and intrusive fears—symptoms traditionally associated with anxiety disorders (American Psychiatric Association, 2013). The observed disruptions in the DMN and amygdala-PFC circuitry may underlie not only depressive symptoms but also the excessive threat sensitivity and cognitive rigidity characteristic of PPD. This neural dysregulation highlights the importance of considering PPD as a multidimensional disorder that encompasses both affective and anxiety-related components.

Interaction Between Structural and Functional Abnormalities

The interaction between structural and functional abnormalities in PPD is complex and multifactorial. One significant contributor is the dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, a critical component of the body's stress response system. Under normal conditions, the HPA axis helps regulate the release of cortisol, a hormone that prepares the body to respond to stress. However, chronic stress, as often observed in depression, can disrupt this system, leading to prolonged elevations or irregularities in cortisol levels. This dysregulation, often observed in depression, can result in lasting changes to brain structures such as the hippocampus and amygdala, regions that are central to emotional regulation and memory processes (Pampallona et al., 2017). Notably, cortisol dysregulation may not only affect the individual but also be passed down generationally, increasing the risk for subsequent generations to experience similar disruptions in stress regulation and mood disorders (Lupien et al., 2009). Although research specifically targeting PPD is limited, it is well established that stress-related changes in brain structures are a hallmark of mood disorders, suggesting that PPD is likely influenced by similar mechanisms.

Epigenetic Influences on PPD Vulnerability

Epigenetic mechanisms may also play a role in shaping an individual's vulnerability to PPD. Gene-environment interactions, such as DNA methylation and histone modification, have been implicated in the development of mood disorders, including major depression. In their study, (Gingnell et al., 2018) examined how maternal stress during pregnancy affects the epigenetic regulation of genes involved in mood and stress responses. They found that prenatal stress can lead to changes in DNA methylation patterns, which in turn affect the expression of genes related to the hypothalamic-pituitary-adrenal (HPA) axis and its stress response. This evidence suggests that prenatal stress may not only increase the risk of developing major depression but could also heighten the susceptibility to PPD. While direct research on epigenetics in PPD is limited, studies on depression suggest that maternal stress during pregnancy and the postpartum period could lead to epigenetic changes that increase the risk of developing PPD, particularly through alterations in the HPA axis. These changes in stress regulation are thought to predispose individuals to emotional dysregulation and mood disturbances (Pampallona et al., 2017). Understanding these mechanisms could provide novel insights into how environmental factors, such as stress and hormonal fluctuations, interact with genetic predispositions to influence the development of PPD.



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Clinical Implications and Future Research

The integration of neuroimaging into clinical practice holds great promise for improving early identification of PPD. Structural and functional brain biomarkers, such as modifications in the PFC, amygdala, and DMN could provide critical information for diagnosing PPD before symptoms fully manifest. Personalized interventions that target these neural biomarkers could enhance the efficacy of treatments, potentially improving outcomes for both mothers and their children. There is a clear need for longitudinal research that combines neuroimaging, hormonal, and genetic data to deepen our understanding of PPD. This approach will help clarify how the brain's structural and functional changes interact with hormonal fluctuations and genetic vulnerabilities over time, paving the way for more effective prevention and intervention strategies.

Conclusion

Postpartum depression is a complex disorder with significant implications for both maternal and infant health. Structural and functional brain alterations contribute to the symptomatology of PPD, highlighting the importance of early intervention in mitigating long-term effects. Future research focused on neuroimaging, hormonal influences, and genetic biomarkers is crucial for developing more effective diagnostic tools and personalized treatment options for PPD. Early identification and intervention will both improve maternal mental health as well as foster better developmental outcomes for children.

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About the Author

Sylvia Merz is a junior majoring in Psychology with a concentration in Cognitive Neuroscience and minors in Public Health and Statistics. On campus, she is involved as a research assistant within the Laboratory for the Emotion and Stress Assessment, a course assistant for STAT 212 (Biostatistics), and a member of Girls Next Door (an a cappella group). She also serves as a community representative for the Alzheimer's Association, through which she has integrated her passion for global health and psychology to contribute to aging research. In her free time, Sylvia loves to hike, thrift, and sew!

The Ethics of Brain-Computer Interfaces (BCIs)

Written by Ruchi Prakash

Fifteen years after a stroke left Cathy Hutchinson paralyzed, she discovered a surprising path to independence. Using nothing but her thoughts, she can now control a robotic arm to feed herself and perform everyday tasks, something that was once thought impossible (Image 1). This life-changing feat is made possible by brain-computer interfaces (BCIs), an upcoming technology that establishes a direct link between the brain and an external device (Orenstein, 2012).

Image 1. Cathy controls a robotic arm with her thoughts, despite being paralyzed.

BCIs capture and translate brain signals into computer commands, which are then interpreted by external devices such as robotic arms, wheelchairs, and speech neuroprosthetics. Speech neuroprosthetics, a specialized type of BCI, translate brain activity into text or synthesized speech. This technology enables users to bypass the peripheral nervous system and muscles entirely, restoring their ability to communicate and interact with their environment.

Beyond simply replacing lost motor function, BCIs can also serve as rehabilitation tools. Feedback from BCI systems can help rewire or strengthen brain circuits, promoting the restoration of native motor functions over time (Daly & Wolpaw, 2008). Although originally limited to restoring motor functions, the scope of BCIs is expanding into exciting new frontiers. Today, BCIs can be used for everything from controlling smart devices to cognitive enhancement and interacting with virtual worlds. Leading companies in the field like Neuralink and Synchron are developing BCIs that could allow people to interact with technology in ways that were previously only seen in science fiction.

With the immense potential of this technology comes important ethical questions: Who owns and controls the data from our minds? Could cognitive enhancement through BCIs deepen social divides? How do we protect our privacy and autonomy over which thoughts are converted into digital signals? As BCIs become more integrated into daily life, addressing these ethical concerns will be essential in shaping the future of human agency.

Identity and Agency

BCIs raise important concerns regarding identity and agency. Many of these technologies are considered invasive because they require electrodes to be implanted directly onto or into brain tissue to record and stimulate neural activity with high precision. Unlike electroencephalography (EEG), which uses electrodes placed on the scalp to passively measure general brain activity, invasive BCIs bypass the skull to achieve greater accuracy and control. This procedure, however, carries several risks, including infection, tissue damage, and gradual electrode degradation (Burwell et al., 2017).

While many BCIs used in clinical and research settings today are non-invasive and rely on EEG, they offer lower resolution compared to invasive systems that require surgical implantation. These medical risks are compounded by ethical dilemmas, particularly when BCIs are used by patients with motor disabilities or neurodegenerative disorders.

One major issue is the difficulty in ensuring ongoing informed consent, especially in patients with cognitive impairment. Conditions like Alzheimer's and Parkinson's can impair decision-making, making it difficult for patients to understand and assess the risks involved. Similarly, individuals with conditions like amyotrophic lateral sclerosis (ALS), which affect speech and communication, may struggle to provide clear and consistent consent (Klein & Ojemann, 2016). On the other hand, using this technology may empower individuals and provide a sense of autonomy not previously possible as seen in the case of Cathy Hutchinson.

Mood disorders, such as depression, further complicate the consent process. Research has shown that depression can impair decision-making, potentially influencing a patient's willingness to continue participation in BCI studies or treatment (Dunn et al., 2011). These factors highlight the importance of continuously assessing a patient's capacity to consent to ensure their autonomy is respected.

Alongside these concerns, BCIs raise questions about control. A common fear is that these devices are capable of "mind reading" and can extract any information from the user's brain. As such, many worry that this technology could alter an individual's sense of self and free will. It is crucial to understand that BCIs do not operate autonomously but instead work together with the user to initiate actions (Shih et al., 2012). This joint action ensures that the user's agency and intentionality are not compromised.

Privacy and Data Security

As BCIs become more widespread, concerns over privacy and data security are growing. BCIs generate highly sensitive neural data that could reveal a person's thoughts, intentions, and emotions. Without proper safeguards, this information could be hacked, misused, or even sold without consent.

Martinovic first introduced the term "brain spyware" to describe the security risks involved with collecting EEG data through BCIs. Using a low-cost gaming headset, Martinovic and his team created an application capable of secretly collecting brain data while showing the user different images. For example, to infer a bank PIN, the system would flash digits on the screen while monitoring brain signals. When a familiar number appeared, the user's brain would produce a P300 brain wave, revealing recognition without any conscious input (Martinovic et al., 2012). Future experiments concluded that it took less than 13.3 milliseconds of presenting specific visual stimuli to extract this sensitive information (Takabi et al., 2016). This research highlights the ease with which BCIs can be misused. With the growing commercialization of BCIs and their integration into games and mobile apps (Image 2.), the threat of data breaches is increasing.

Image 2. New generation of BCIs are being used to improve the gaming experience.

Currently, there is no unified framework for regulating the ownership of neural data, creating uncertainty about whether the neural data belongs to the user, the company, or healthcare provider? As BCIs continue to collect personal information, experts argue for stronger collaboration between manufacturers and governments to address these privacy issues. Xia et al. (2023) recommend enhancing encryption, adding noise to the data, and separating relevant from irrelevant data to better protect user privacy. While these solutions are a step in the right direction, much more work remains to be done.

Equity and Cognitive Enhancement

Given the novelty of this technology, BCIs are currently expensive and not widely available. This limited affordability of BCIs can enlarge social inequities as only privileged hospitals or institutions with access to such technology would be able to offer these treatments to patients. Moreover, some BCIs aim to enhance cognitive and physical abilities in healthy individuals, a concept known as

neuroenhancement. While some fear that advancing this technology could deepen social divides and disrupt human nature, others view it as a potential way to integrate man and machine and enhance human capabilities. Through this, they believe humankind can move closer to perfection and improve moral judgement, emotional perception, and reasoning (Khan & Aziz, 2019). Regardless of the rationale, this debate emphasizes the need for better regulations to ensure equitable access to this life-changing technology.

While brain-computer interfaces offer remarkable potential to transform healthcare, enhance cognitive abilities, and the quality of life, they also raise significant ethical and privacy concerns. As the technology continues to evolve, ethical guidelines and safeguards must be established to protect individual autonomy, safety, and access. By addressing these challenges thoughtfully, we can harness the full potential of BCIs while minimizing risks and inequalities.

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About the Author

Ruchi is a junior at the University of Illinois at Urbana-Champaign majoring in Neuroscience and Psychology. She joined Brain Matters to explore her passion for the brain and stay connected to cutting-edge research in the field. In addition to writing for Brain Matters, Ruchi serves as the Vice President of NeuroTech@UIUC, a project-based organization focused on the intersection of neuroscience and technology. She looks forward to pursuing graduate studies and expanding her experience in research and innovation.

Brain Matters Board

President

Michelle Bishka

Michelle Bishka is a senior majoring in Specialized Chemistry and minoring in Computer Science. Outside of Brain Matters, she is an undergraduate researcher in the Silverman Lab and a member of American Chemical Society. She later hopes to pursue graduate studies in chemistry.

Chief Editor

Andrew Hamilton

Andrew Hamilton is a junior with a major in Neuroscience and minors in Spanish and Chemistry. One thing he enjoys about editing is that he gets to read so many interesting articles about science-related discoveries every day! Outside of the club, he pursues research regarding optimization with on-tissue chemical derivatization.

Vice President

Praise Kim

Praise Kim is the Vice President of Brain Matters and an undergraduate researcher pursuing a BSLAS in Brain and Cognitive Science. Currently, as a research assistant in the Gratton Lab, she studies the Fronto-Parietal Network in cognitive control tasks across different mental states. In the past, she has also presented work on the infant parasympathetic response and maternal depression with the Interdisciplinary Lab for Social Development. She is broadly interested in cognition in the brain and throughout development, also presenting work on social cognitive development at Stanford University. Outside of research, she lifts weights, reads fantasy novels, and spends time with her church. Her future goals are to continue researching the brain—whether as a post-bacc, doctoral student, post-doc, or professor.

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Krisha Agarwal is a junior in MCB Honors with a minor in Informatics. She is the Editor-in-chief of Brain Matters and a member of American Medical Women's Association. She is also an undergraduate researcher at the KV Prasanth Lab in Cell and Developmental Biology. In the future, she hopes to attend graduate school and work in the biotechnology industry. In her free time, Krisha enjoys crocheting, reading, sketching, and spending time with friends.

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Macy Hoeveler is a sophomore in the Brain & Cognitive Science program at UIUC. She is pursuing a double minor in Integrative Biology and Music. Aside from being the Editor-in-Chief of Brain Matters, she is a writing consultant with the Writer's Workshop. In addition, she is a Beckman Fellow with the Auditory Cognitive Neuroscience Lab and a lab assistant at the Dolezal Bee Research Lab. In her free time, Macy is a violinist in the Philharmonia Orchestra and enjoys reading, listening to music, and collecting bugs. She hopes to continue pursuing biology in graduate school, studying behavioral genetics and neurobiology.

Treasurer

Vraj Patel

Vraj Patel is a sophomore majoring in Neuroscience with minors in Chemistry and Psychology. Vraj joined Brain Matters to learn about more niche topics in neuroscience and research in the field. In addition to being treasurer for Brain Matters, Vraj is an undergraduate researcher in the Sweeney Lab, which studies neuroscience in the context of feeding and related behaviors. He is also a volunteer for Avicenna Community Health Center, a course assistant for STAT 200, and a peer mentor for first-year students in the neuroscience major. Vraj hopes to explore more in the field of neuroscience from a medical perspective in the future!

Social Media Chair

Vani Sharma

Vani Sharma is pursuing a Bachelor of Science in Molecular and Cellular Biology (MCB) with an honors concentration, alongside a minor in Public Health and a Neuroscience certificate. As a writer for Brain Matters, she investigates the intricate interplay between the brain and diverse phenomena, including the neural foundations of gratitude, the influence of music on cognitive processes, and the complexities of neuroanatomy and neurological disorders. Through her work, she blends rigorous scientific research with engaging narratives to illuminate the brain's extraordinary intricacies while promoting scientific literacy and making complex concepts accessible to a broader audience.

Social Media Chair

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Erin Ford is a junior majoring in Chemical Engineering with a concentration in Biomolecular Engineering. In her free time, she enjoys playing tennis and painting. She hopes to help others increase their knowledge about neuroscience through her writing in Brain Matters.

Social Chair

Isabelle Afshari

Isabelle is a sophomore at the University of Illinois majoring in Neuroscience. Isabelle became involved in Brain Matters to learn more about writing scientific articles and innovations in neuroscience. In addition to writing for Brain Matters, Isabelle is involved in McKinley Health Stress Management Peers, LAS Leaders, and Women's Glee Club. In the future, Isabelle hopes to attend medical school and continue reading and writing about new scientific innovations!

Social Chair

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Kaitlyn Tuvilleja is a junior in Bioengineering with a Statistics minor. She is an undergraduate research assistant for Bhargava Lab and I^2 Lab. Besides Brain Matters, Kaitlyn is involved with SWE, WIE, and BMES. In her spare time, she enjoys baking and running with her friends.

Design Head

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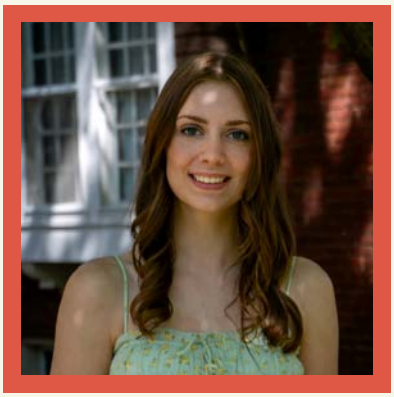
Sarah is a junior studying Psychology and Information Sciences with a minor in Art & Design. Some of her academic interests include cognition, human-computer interaction, and treating psychiatric disorders. She enjoys drawing, finding new music, and crocheting as well! Outside of Brain Matters, Sarah is also involved in Design Innovation Illinois and Psi Eta Mu, a professional information sciences fraternity. She hopes to continue furthering her understanding of neuroscience and exploring topics she's passionate about through the journal.

Design Board



Ruth Anderson

Ruth Anderson is a rising Sophomore at the University of Illinois majoring in neuroscience and minoring in psychology. She joined Brain Matters to get involved with the neuroscience community on campus and learn more about the field. Ruth currently is hoping to pursue a career in research. She is passionate about womens health and child development. Outside of school Ruth enjoys hanging out with her friends, crocheting, and reading.

Esther Nam

Esther Nam is a junior on the pre-medical track majoring in Psychology with a minor in Public Health. She is interested in exploring the cognitive and neurological impacts of bilingualism, and is currently a research assistant in the Educational Psychology Psycholinguistics Lab with a focus on cognitive psych. In her free time, she loves to draw, play games, and spend time with friends. After undergrad, Esther hopes to attend medical school to become a physician.

Jeslyn Chen

I'm a rising sophomore majoring in psychology and minoring in chemistry. I joined Brain Matters to combine my interests of neuroscience, psychology, and journalism. Outside of this magazine, I plan to become a student EMT at UIUC and enjoy drawing, going to concerts, and thrifting.

Jessica George

Hi! My name is Jessica George and I'm a junior majoring in Molecular and Cellular Biology and Brain and Cognitive Science. Outside of school, I volunteer at a nursing home in the activities department, where I work closely with residents who have dementia. In my free time I love dancing, listening to music, and trying new restaurants!

Lisa Patel

Lisa Patel is a rising junior and an Integrative Biology major on the pre-medical track with minors in Chemistry and Nutrition at UIUC. Passionate about medicine and community outreach, she co-founded and serves as President of the Illini Sheltering Hands Society, where she teaches basic life-support skills and organizes volunteering initiatives. As Public Relations Coordinator for REACT, Lisa coordinates hands-on chemistry demonstrations at local elementary and middle schools. She's volunteered over 300 hours at UI Health Hospital while assisting across Emergency, Diagnostics, Radiology, University Health Services, and Surgical departments. She also directs community health initiatives as Director of Medicine for UIUC's MEDLIFE chapter. Her end goal is to become a physician and she is dedicated to expanding her knowledge to better serve her community.

Sania Shah

Sania Shah is a sophomore majoring in Brain and Cognitive Science with a minor in Data Science. She works as an undergraduate research assistant in the Cognitive Decision-Making Lab and dances competitively with the Illini Raas team. Outside of Brain Matters, Sania enjoys playing badminton with friends and curling up with a good book and an iced coffee.

Editors

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Thiya is a sophomore at UIUC majoring in MCB with a minor in Psychology, hoping to one day become a Physician Assistant. In her free time, she likes to run, crochet, and play the piano. She is currently involved in research at the Liang Lab for Behavioral Neuroscience. Through her involvement with Brain Matters, she hopes to broaden her knowledge and gain deeper insights into the fields of neuroscience and psychology.

Kathryn Kennedy

Kathryn Kennedy is a freshman studying Biology with minors in Health Technology and Spanish. She joined Brain Matters to learn more about neuroscience, psychology, and improve her writing and editing skills. Outside of the journal, she is involved in Global Medical Training and Education and Training 4 Health. She also dances with PSA Barkada, sings with the St. John's church choir, and plays guitar in her free time. Her career goal is to be a pediatrician.

Nicholas Opiola

Nicholas Opiola is a recent ’24 MCB alumni. He is a lifelong learner and has always loved studying across all academic disciplines, especially neuroscience! Nicholas joined Brain Matters to immerse himself in all the latest exciting work being performed in the field of neuroscience and to utilize his writing skills towards helping others produce their best work. In his free time, Nicholas loves to watch fútbol, dance, sing karaoke, spend time with family and close friends, play video games, and spend time amongst nature. In the future, Nicholas hopes to devote his career towards making a lasting, positive change in as many lives as possible.

Megan Lu

Megan Lu is a Junior majoring in Brain & Cognitive Science with a minor in Health Administration and Business. She is involved in various RSOs on campus, including FHCE (Future Healthcare Executives) and Alpha Epsilon Delta (a pre-health fraternity). She is also currently involved in research with the Illinois Alternative Protein Project. In her free time, Megan spends most of her time at the gym working out, cooking new recipes, or listening to true crime podcasts. She hopes to deepen her understanding and appreciation of the brain through writing with Brain Matters and will graduate this year.

Yuliia Kohut

Yuliia Kohut is a Freshman in Bioengineering on a pre-medical track and a student from Ukraine. Apart from Brain Matters, on campus she is a Global Health executive member in the American Medical Student Association, and she is also a student volunteer at Carle Hospital. Yuliia is an undergraduate researcher in Dr. Best-Popescu lab at Beckman Institute, working on developing imaging tools for cellular neuroscience research. In her free time Yuliia enjoys cross-stitching, cooking Ukrainian food, and reading sci-fi novels. She joined the editing and writing team of Brain Matters to share her fascination with neuroscience with UIUC!

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Kaitlyn Tuvilleja is a junior in Bioengineering with a Statistics minor. She is an undergraduate research assistant for Bhargava Lab and I^2 Lab. Besides Brain Matters, Kaitlyn is involved with SWE, WIE, and BMES. In her spare time, she enjoys baking and running with her friends.

Gus Dorman

Gus Dorman is a freshman majoring in Neuroscience with a minor in Computer Science. He joined Brain Matters as an editor to learn more about the field while also getting a feel for what research articles are like. If he's not studying, he's probably longboarding around campus, playing a video game, or watching shows.

Praise Kim

Praise Kim is the Vice President of Brain Matters and an undergraduate researcher pursuing a BSLAS in Brain and Cognitive Science. Currently, as a research assistant in the Gratton Lab, she studies the Fronto-Parietal Network in cognitive control tasks across different mental states. In the past, she has also presented work on the infant parasympathetic response and maternal depression with the Interdisciplinary Lab for Social Development. She is broadly interested in cognition in the brain and throughout development, also presenting work on social cognitive development at Stanford University. Outside of research, she lifts weights, reads fantasy novels, and spends time with her church. Her future goals are to continue researching the brain—whether as a post-bacc, doctoral student, post-doc, or professor.

Jessica Chen

Jessica Chen is a sophomore studying clinical-community psychology. With Brain Matters, she has been excited to integrate her interests in neuroscience, linguistics, and psychology. She has appreciated groundbreaking applications of neuroscience in skill acquisition, discrimination, addiction, and more. Currently a research assistant with the Health Equity and Action Lab and the Social Cognition Lab, Jessica examines parenting and child health outcomes across cultural contexts, and neural network dissection of trends in biases. Aside from academics, Jessica is most likely baking a sweet treat or lounging at a matcha cafe.

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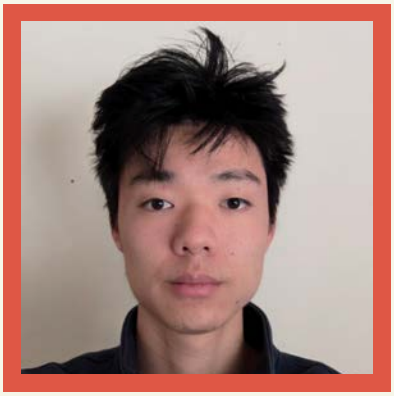
Natalia is a Freshman at the University of Illinois majoring in Neuroscience. Natalia became involved in Brain Matters to further her passion for the brain and to become familiar with modern topics of neuroscience. In addition to Brain Matters, Natalia is involved in the American Medical Women's Association at the University of Illinois. Natalia hopes to continue her studies in the medical field specifically with neurology to continue learning about the brain!

Brain Matters Writers



Edward Lin

Edward is a Sophomore at the University of Illinois majoring in Neural Engineering. Through his studies, he aspires to implement biological mechanisms/systems into computers and explore AI-neural network connections. Some of his interests include playing volleyball, filming, and going on road trips. After graduation, he hopes to attend graduate school.

Tanisha Mandal

Tanisha Mandal is a freshman at the University of Illinois, studying Neural Engineering with a minor in Computer Science. Her interests in neuroscience include computational neuroscience, specifically its applications in treating neurodegenerative diseases, and internal causes of severe brain lesions, such as brain cancer. She also enjoys going skiing, playing cards, and listening to music. Tanisha was interested in being a writer for Brain Matters to have the opportunity to practice writing her own research papers in the future and explore new neuroscience topics in depth. Outside of Brain Matters, Tanisha is involved in research programs such as NeuroTech's Cortex Codex and UR2PhD, and fun RSOs such as The Cooking Collective and UIUC's Book Club!

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Sylvia Merz is a junior majoring in Psychology with a concentration in Cognitive Neuroscience and minors in Public Health and Statistics. On campus, she is involved as a research assistant within the Laboratory for the Emotion and Stress Assessment, a course assistant for STAT 212 (Biostatistics), and a member of Girls Next Door (an a cappella group). She also serves as a community representative for the Alzheimer's Association, through which she has integrated her passion for global health and psychology to contribute to aging research. In her free time, Sylvia loves to hike, thrift, and sew!

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Siwon is a pre-medical student majoring in Biochemistry at the University of Illinois Urbana-Champaign, with a strong interest in the intersection of research and clinical medicine. Passionate about understanding the molecular basis of disease and pharmacology. At UIUC, Siwon is engaged in research involving cell culture and cellular differentiation, with a focus on inducing stem cells to become muscle and neuron-like cells. Additionally, Siwon has contributed to research at the Feinberg School of Medicine, studying corneal damage and repair mechanism. Siwon plans to pursue a career in medicine that integrates both clinical practice and biomedical research. Through this dual path, he aims to help bridge laboratory discoveries with therapeutic advances that improve lives.

Leah Rupp

Leah Rupp is a freshman at the University of Illinois in Urbana-Champaign studying Molecular and Cellular Biology within the honors concentration. Leah joined Brain Matters to get the opportunity to learn and write about new neuroscience research. Leah is also a Stress Management Peer with McKinley Health Center and a volunteer with the Food Assistance and Well-being Program. In her free time, Leah enjoys running and playing the piano. Her career aspiration is to become a physician.

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Ananya Sampathkumar is a sophomore, majoring in Neuroscience with minors in Chemistry and Public Health. Outside of Brain Matters, Ananya is an assistant editor-in-chief for Double Helix Digest, a member of Starcourse, a volunteer at Carle Hospital, and works at the Office of Undergraduate Admissions as a tour guide and student ambassador. In her free time, Ananya likes to read books, make jewelry, watch movies, and hang out with her friends.

Emily Aldrich

Emily Aldrich is a Freshman majoring in Neuroscience with minors in Linguistics and Psychology on the pre-med track. Emily joined Brain Matters to gain a deeper understanding of the brain through exploring current research topics in neuroscience. In her free time, she enjoys listening to music, reading, and spending time with friends.

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Alexa DiVito is a freshman at the University of Illinois. She is currently an undeclared major on the Pre-Nursing track and plans to declare as a Psychology major next year. Alexa became part of Brain Matters to develop her knowledge of the brain and share her new knowledge with others. Apart from writing for Brain Matters, Alexa is involved in Greek life, RSO's, and is working on getting her CNA license.

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Meha Goswami is a sophomore majoring in Psychology, with an interest in double majoring in Molecular and Cellular Biology, and is on the pre-med track. Outside of Brain Matters, she is involved with Phi Chi, Delta Kappa Delta, and Illini Sheltering Hands Society, and she works as a research assistant in the Vision Lab. In her free time, Meha enjoys painting, listening to music, and spending time with her friends!

Kathryn Kennedy

Kathryn Kennedy is a freshman studying Biology with minors in Health Technology and Spanish. She joined Brain Matters to learn more about neuroscience, psychology, and improve her writing and editing skills. Outside of the journal, she is involved in Global Medical Training and Education and Training 4 Health. She also dances with PSA Barkada, sings with the St. John's church choir, and plays guitar in her free time. Her career goal is to be a pediatrician.

Meredith Kremitzki

Meredith Kremitzki is a junior at the University of Illinois, majoring in psychology with a concentration in cognitive neuroscience and a minor in integrative biology. She became involved with Brain Matters to learn more about the different topics in neuroscience. Along with writing for Brain Matters, Meredith is a laboratory teaching assistant for the chemistry department. She hopes to become a doctor and continue learning about the brain and body.

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Lily Kushnick is a freshman at the University of Illinois majoring in neuroscience. Lily became involved in Brain Matters to learn more about the process of writing scientific articles and about current neuroscience research and innovations. In addition to writing for Brain Matters, she is a member of Healthcare Book Club and volunteers at Carle Hospital.

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Brianna Mae is a Junior at the University of Illinois majoring in Clinical/Community Psychology. She became involved in Brain Matters to gain more experience researching and writing about the current research in Neuroscience. When she is not writing for Brain Matters, she is also involved in Dr. Kwapil's Project on Life Experiences Lab, and is the Treasurer for the Psychology Research and Community Club (PRACC). Brianna Mae is hoping to pursue a PhD in Clinical Neuropsychology and conduct research about the neurological basis behind different clinical disorders.

Ruchi Prakash

Ruchi is a junior at the University of Illinois at Urbana-Champaign majoring in Neuroscience and Psychology. She joined Brain Matters to explore her passion for the brain and stay connected to cutting-edge research in the field. In addition to writing for Brain Matters, Ruchi serves as the Vice President of NeuroTech@UIUC, a project-based organization focused on the intersection of neuroscience and technology. She looks forward to pursuing graduate studies and expanding her experience in research and innovation.

Want To Get Involved?

Brain Matters is a Registered Student Organization (RSO) on campus that welcomes all years and majors. Please email brainmattersuiuc@gmail.com with inquiries about getting involved with the journal or RSO.