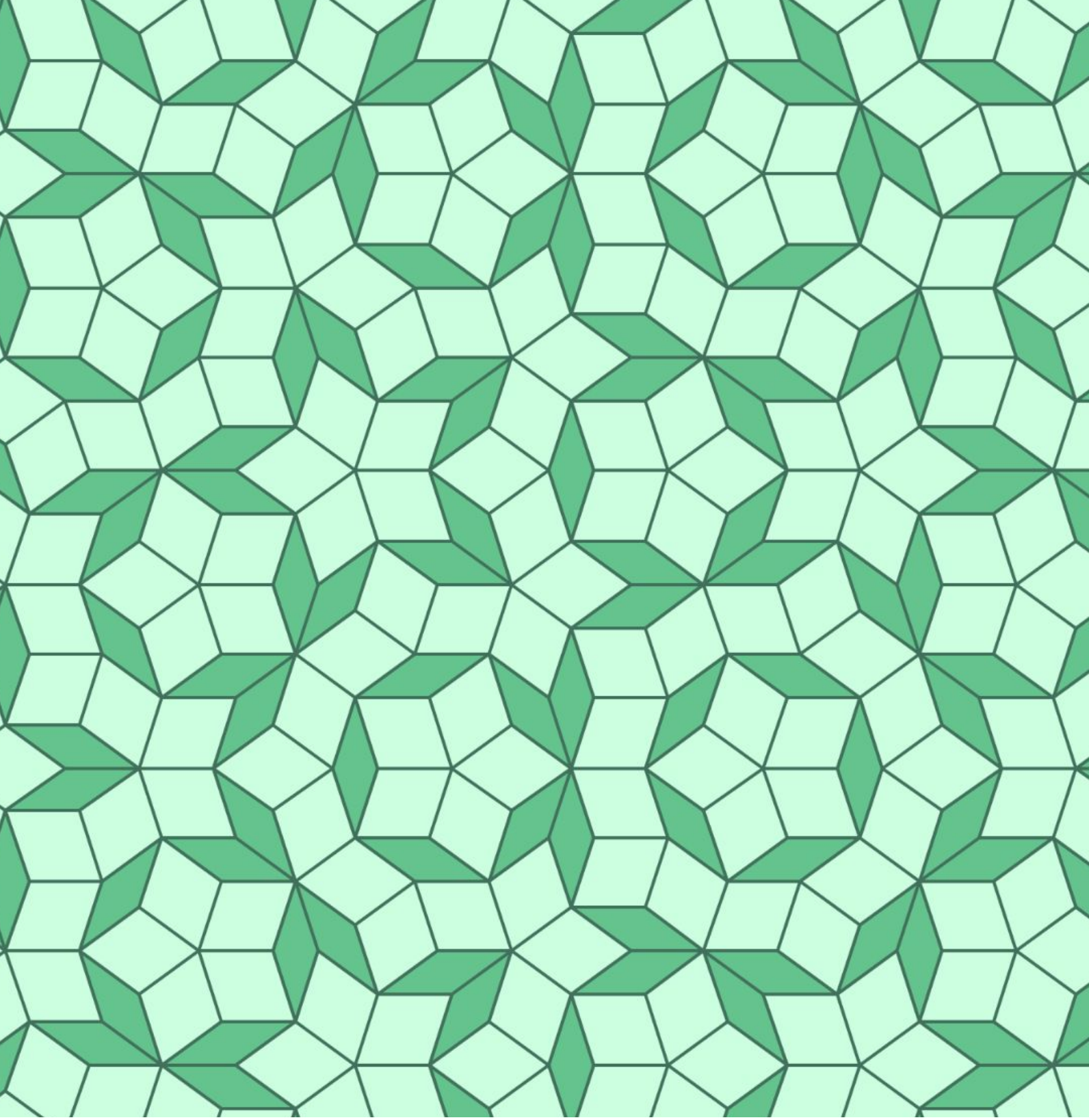

THE PENROSE MAGAZINE

STEM

Issue 6: Neuroscience Mini Issue, 15 Mar 2026

Welcome to the Sixth Issue of Penrose Magazine!

Penrose is a STEM magazine where we hope to establish a community of young people who are passionate about STEM, want to share with their peers and further their knowledge beyond the curriculum. This installment of the magazine consists of the academic articles from the Fluorescent Magazine x Penrose Magazine Neuroscience Collection. We hope to continue fostering an environment where people are encouraged to push themselves to create meaningful work and support each other to grow.

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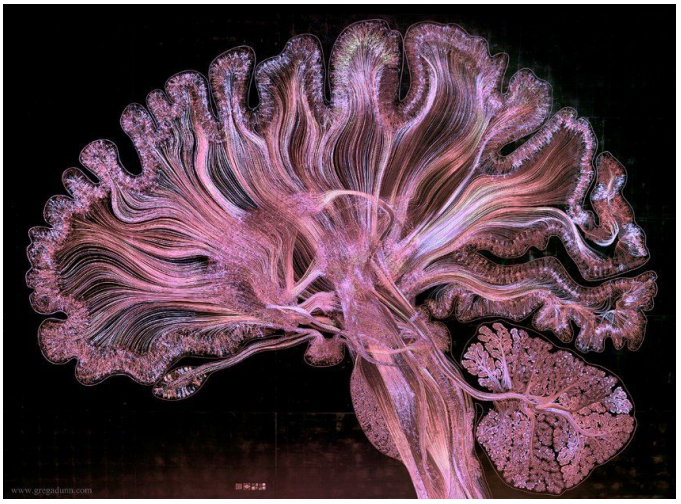


Drivers and Mechanisms of Autism Spectrum Disorder: A Comprehensive Schematic

The Autism Spectrum Disorder (ASD) is a condition in which genetic mutations, heritable variants or prenatal environmental factors influence the pathway of neurons and structure of the brain. Despite ASD being a congenital neurodevelopmental condition, it is often misinterpreted in non-clinical settings, with scientifically discredited hypotheses suggesting a causal link between vaccinations and autism [1]. Individuals often require predictable routines and maintain intense interests; furthermore, they may experience sensory hypersensitivity and face significant challenges with social communication [2].

Deficits in social communication and atypical sensory processing significantly impact adaptive functioning. In high-stimulation environments—such as educational institutions or public commercial spaces—individuals may experience an inability to modulate exogenous stimuli (e.g., intense lighting or auditory input). This failure of sensory gating leads to a state of sensory overload, often manifesting in self-regulatory behaviors or 'shutdowns,' characterized by physical withdrawal or irritability.

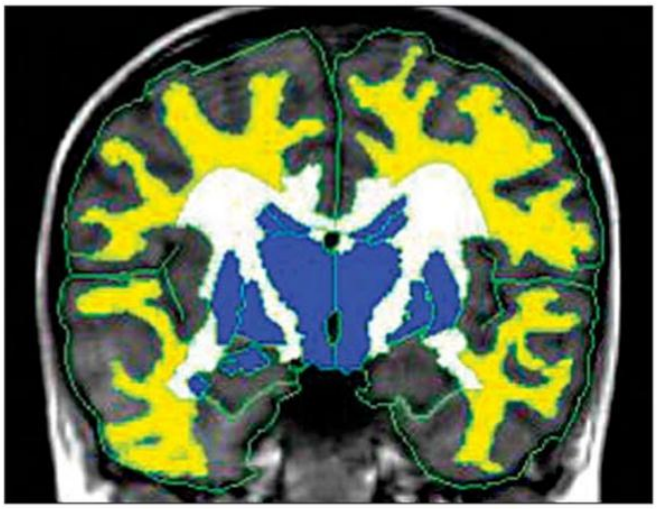
While Autism Spectrum Disorder (ASD) is a lifelong neurodevelopmental condition without a clinical 'cure,' multimodal interventions significantly enhance adaptive functioning and quality of life.

Specialized therapies, including Speech-Language Pathology (SLP), Applied Behavior Analysis (ABA), and Occupational Therapy (OT), address specific communication and behavioral challenges [3]. To manage sensory processing deficits, clinicians often employ sensory integration therapy and sensory diets—customized regimens of proprioceptive and vestibular activities, such as rhythmic swinging or jumping, designed to modulate arousal levels [4]. Furthermore, fine motor skill training is utilized to improve manual dexterity and coordination.

As established, the etiology of Autism Spectrum Disorder (ASD) involves a significant hereditary component, characterized by variants in high-confidence risk genes such as SHANK3, NLGN3, NLGN4, NRXN1, and CNTNAP2. [5] Many of these mutations reside in genes encoding synaptic scaffolding proteins—most notably SHANK3—which are critical for the structural integrity of synapses [6]. Conversely, genes like SCN1A are implicated in neuronal excitability and synaptic plasticity, often exhibiting structural polymorphisms in autistic individuals. Furthermore, the involvement of genes regulating transcription and chromatin remodeling during early corticogenesis underscores the robust genetic foundation of the disorder.

Autism Spectrum Disorder (ASD) exhibits a high heritability coefficient of approximately 0.90 and displays a notable sex-based skew, with a higher prevalence observed in males than in females [7]. The genetic basis of the disorder has been largely elucidated through longitudinal twin studies initially popularized in the early 20th century. These studies demonstrate a significantly higher concordance rate in monozygotic (identical) twins, ranging from 60% to 90%, compared to dizygotic (fraternal) twins. Such a marked discrepancy provides robust evidence for the strong genetic architecture and hereditary nature of the condition.

A prominent study led by psychologist Thomas W. Frazier II utilized data from the Interactive Autism Network (IAN), analyzing a cohort of 568 twin pairs to determine the hereditary patterns of ASD. The findings revealed a monozygotic concordance rate of approximately 76%, highlighting a profound genetic influence. Furthermore, the researchers identified a 34% shared diagnosis rate among same-sex dizygotic twins, which dropped to 18% in opposite-sex pairs [8]. This data further validate the hypothesis that genetic factors are primary drivers of the disorder's phenotypic expression.



Representation of nonuniform growth patterns in autistic brains. The yellow area represents white matter which is in a larger volume in the study of children with autism in comparison to the control group. White area represents bridging and sagittal components (no difference from control). The blue area showed differences but not to a remarkable extent. Image by Martha Herbert, MD, PhD [9].

Current neurobiological perspectives highlight the prevalence of macrocephaly, defined as a head circumference exceeding the 99th percentile, within the ASD population.

This clinical observation prompted extensive investigation into the correlation between cranial overgrowth and the disorder's etiology [10]. Structural Magnetic Resonance Imaging (sMRI) has confirmed a significant increase in total brain volume (TBV) during early childhood; notably, this cerebral enlargement appears to persist throughout childhood but often attenuates by adolescence [11]. Quantitative analyses indicate that this volume increase is driven by expansions in both total cerebral white matter and total cortical gray matter. Utilizing advanced automated parcellation techniques, researchers have documented significant regional variations in gray matter density, further suggesting a complex pattern of early-life brain overgrowth.

In summary, Autism Spectrum Disorder is characterized by lifelong atypicalities in neural processing, marked by distinct patterns of regional hyper- and hypo-connectivity across the cerebral cortex. Enhancing public and clinical awareness of these neurobiological challenges is essential for mitigating social stigma and fostering a more inclusive environment. Despite significant advancements, the field of neurodivergence remains an under-researched frontier with vast opportunities for meaningful scientific contribution. Expanding our understanding of the genetic and neuroanatomical architecture of ASD not only advances the global body of knowledge but provides vital support for individuals and families navigating the complexities of the spectrum.

By Ahlam Parambath Sunil

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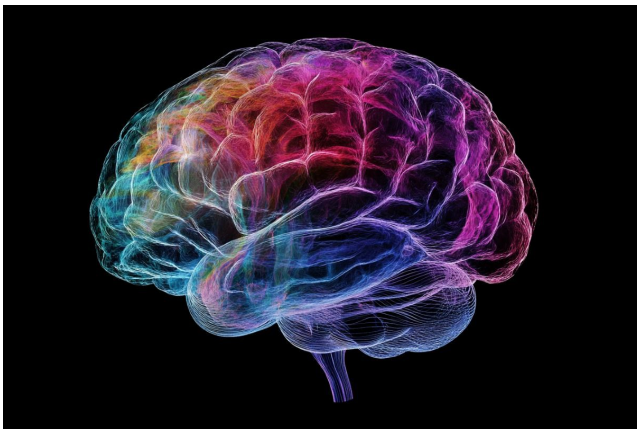

How Brains of Individuals with Autism Spectrum Disorder Differ from Neurotypical Individuals: Structure, Function and Development.

Autism spectrum disorder (ASD) is a neurodevelopmental condition that is characterised by persistent difficulties in social communication, impacted sensory processing, and restrictive, repetitive movements. Due to its roots in the brain, ASD exhibits in children as young as 6 months old. Children with ASD may show signs of social impairment, such as failure to make eye contact or not responding to their name being called. They may also fail to hit their key milestones, for instance talking at the same time as their peers. There are many reasons that contribute to these traits, including brain structure, development and other factors, namely genetics and neurotransmission.

Brain development in neurotypical individuals follows a predictable track. Babies' brains develop and increase in size rapidly, and as they grow, a process called synaptic pruning occurs [1]. However, in autistic individuals, the developmental path can differ slightly. Firstly, their brains tend to increase in volume at a faster rate, leading to overgrowth in areas like the amygdala. The amygdala is responsible for processing emotions, for example interpreting facial expressions or feeling fear. Multiple studies have found that rapid growth of the amygdala occurs between 6 and 12 months of age, and the severity of the overgrowth directly relates to the severity of ASD traits presented [2]. It has been suggested that the overgrowth of the amygdala is caused by difficulty processing sensory information, stressing the amygdala and leading to faster growth than the rest of the brain. Age-specific size differences can also be seen; while certain structures can be oversized in early development, more severe decreases in structural volumes are also observed, leading to slightly smaller brain size by 12-16 years of age [3]. However, these are not uniform or universal to all people with ASD; therefore, finding a correlation is difficult.

Additionally, autistic brains often neglect the pruning process. Synaptic pruning is when unnecessary connections between neural cells and neuronal axons and dendrites in the brain are broken down. They are degraded and engulfed via phagocytosis by immune-like cells called microglia. Phagocytosis is the process in which specialised cells engulf and digest unneeded structures in the body, this can happen for pathogens, protozoa or unneeded cells. This is particularly important during the first few years of life to maintain the integrity of neural pathways, as the connections are usually overproduced in the early stages of development [4].

It has been found that individuals with autism may experience trouble with the different “markers” working in the brain to flag or spare neurons from phagocytosis. There is a complement system in the brain that works with microglia to mark certain neurons for destruction. Complement proteins like C1q or C3 label neurons with an “eat me” signal for microglia [5]. However, in people with ASD, the system may be dysregulated. For example, C1q or C3 proteins could be unexpressed or malfunctioning, which means microglia may not receive the correct signals for the right neurons.

Lastly, microglial cells may be less responsive in some autistic individuals, as studies have shown that sometimes microglia fail to respond adequately to “eat me” signals from neurons, which can cause insufficient pruning. Leaving unused connections in the brain directly links to social impairments, as the brain has become too much of a busy environment to prioritise and process

information efficiently. Insufficient pruning can also lead to the characteristic sensory sensitivities observed in autistic individuals, as the excessive neural links can cause overwhelming communication between parts of the brain, leading to stressful experiences regarding external stimuli.

Furthermore, the prefrontal cortex, which controls behaviour, is also affected by insufficient pruning. The prefrontal cortex relies on clear, precise signals; when it receives too many, the brain struggles to filter out unimportant information, making it harder to coordinate responses, resulting in characteristic struggles with social interaction, narrowed interests and difficulties adapting to change.

There may also be a neurotransmitter factor in expressed traits of autism. For example, an imbalance of excitatory and inhibitory transmitters can cause a deficit in cognitive processing and social behaviour. In some autistic individuals, excessive levels of glutamate (excitatory) and decreased levels of GABA (inhibitory) have been found [6]. This can result in neuronal hyperexcitability, contributing to sensory sensitivities and anxiety. Moreover, some research indicates that the mGluR5 receptor, a type of glutamate receptor, may be overactive in individuals with ASD [7].

Additionally, Muller et al. found that there were elevated levels of serotonin in the blood of 25% of children with ASD, acting as an early biomarker for autism [8]. However, both low and high levels of serotonin have been associated with autism, suggesting a complex relationship between the two. Low levels of serotonin can cause increased repetitive behaviours and anxiety, while high levels cause social difficulties and communication deficits [9]. Each of the two effects are highly prominent autistic traits, confirming the tie between autism and both excitatory and inhibitory neurotransmitters. Finally, this is a brief exploration of the comorbidity of autism and other syndromes. Comorbidity is the term used to describe when two or more disorders often appear together in patients.

There are many neurological aspects of autism that remain unexplored. However, we can use the comorbidity of autism with numerous other conditions to make assumptions about how the autistic brain works. For example, 8% of people with epilepsy are also diagnosed with ASD [10], meaning the neurological factors that cause epilepsy may line up with autism. Epilepsy is characterised by unregulated electrical discharges in the brain. Genetic studies have identified various genes that are common in both autism and epilepsy, and the majority of these are responsible for neuronal synaptic structure and function [11].

This suggests that not only is there a problem with synaptic pruning in the autistic brain but also a difference in the structure of the synapses themselves. The comorbidity of autism and other disorders has been a point of interest for decades, and it has been found that autism usually presents with other conditions like anxiety, depression and ADHD. These disorders are usually the products of irregular neurotransmission in the brain, or genetic differences that code for specific brain structures. This once again confirms the theory that genetic and neurological factors play a big role in the development of autism, and it is not purely environmental as once believed.

Overall, it has been shown that autism is a neurological disorder that is caused by differences in brain function and development, rather than a solely psychological disorder that can be cured. Autism is caused by structural differences in the brain, such as increased amygdala volume and variations in growth speed.

By Anabell Bujwid

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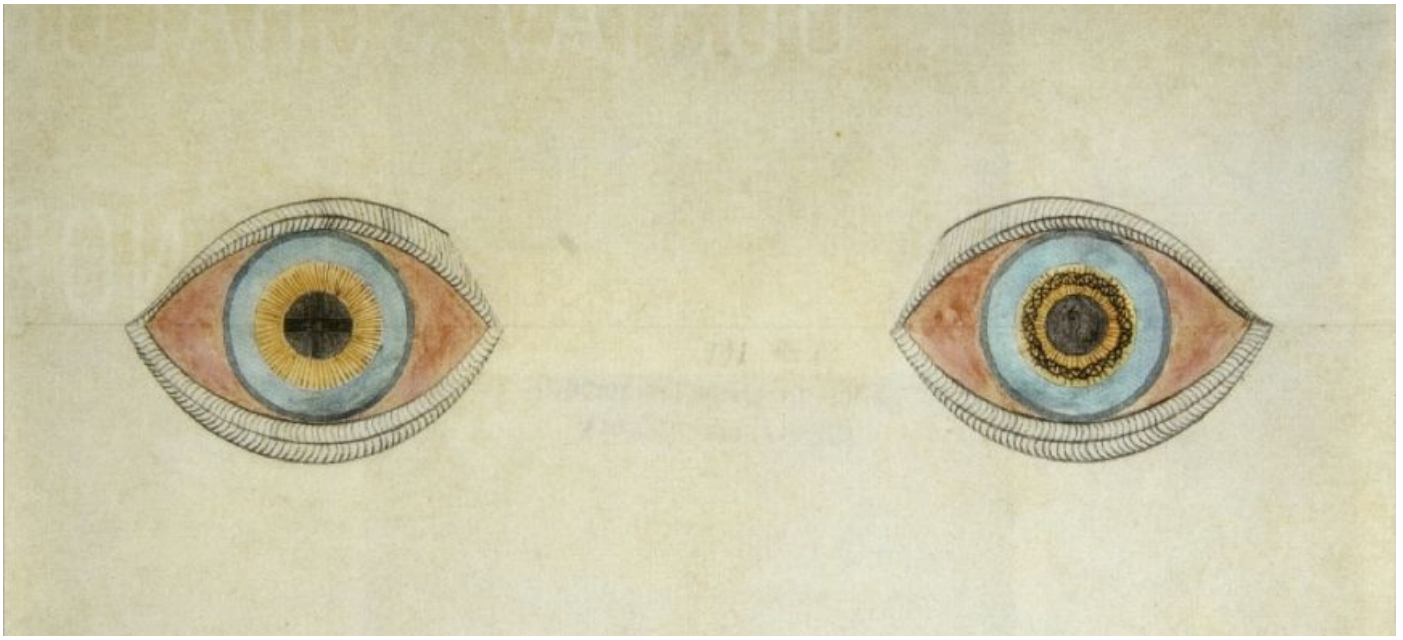

Brain Activity and the Role of Neurotransmitters during Hallucinations

Hallucinations are a complex occurrence where one senses something that does not actually happen. These can happen in many psychiatric and medical disorders, and can be debilitating and distressing for those affected.

Hallucinations are estimated to be one of the top 10 most costly mental health problems. A study in Western Australia from July 2013 to July 2015 looks into this. 0.09% of all A&E visits are caused by hallucinations, which accounts for 1.8% of all mental health presentations. Of this, one-third are psychotic disorders, and 17% of cases do not show any clear medical or psychiatric cause. Furthermore, 65.8% of patients presented two or more times during the two-year period [1]. Patient demographics show that they have a higher likelihood of being male, unemployed, an urban resident and unmarried. This could illustrate links between hallucinations and lifestyle. For example, loneliness could be a factor, as unemployed people have no contact with people in the workplace, and unmarried people are likely to have smaller amounts of contact in the home. Limitations of this study include the fact that it is purely based on data from emergency settings, which is not ideal since diagnosis lack a comprehensive psychiatric assessment [1].

Most people have experienced some form of hallucination. These can vary significantly, from fairly day-to-day, minor examples like tinnitus, to much more significant experiences, such as voices promoting self-harm or suicide [2]. Typical stimuli are able to create sensations in the brain and cause these phenomena. Specialised receptor cells, for example, photoreceptor cells for light and chemoreceptors for odour (smell), send an electrical impulse to the brain through nerves. These receptor cells are specialised nerve endings and are normally receptive to a particular class of stimulus energy within a specific range of intensity [2]. Because of these restrictions, each particular receptor has adequate and inadequate stimuli.

Regarding the previous example of photoreceptor cells, visible light would be an adequate stimulus, but some inadequate stimuli are also able to activate the receptor, should they be intense enough. One may ‘see’ pressure and a phosphene (a bright spot) by applying moderate pressure to a closed eye [2]. This is a hallucination since the bright spot does not occur in reality; however, it does not cause any abnormal brain activity since it is caused by the sensory receptors rather than the brain. Hence, it is less of a brain-generated hallucination and more of a sensory illusion.

A common cause of hallucinations is psychosis. Psychosis is a condition where a person loses some or all contact with reality.

These typically involve hallucinations, delusions, and disordered thinking and speaking. Psychosis itself has many causes. For example, it can be caused by various mental health disorders, such as schizophrenia and bipolar disorder, and also one-off experiences such as drug misuse or severe stress and anxiety. It is thought that psychosis is caused by increased activity of neurotransmitters in the brain, especially dopamine [2], [3].

During many auditory hallucinations, the brain's auditory cortex and superior temporal gyrus show heightened activity without external stimuli from the ears. These are the regions in the brain that typically process sounds. This activity could be driven by internal cognitive processes, but also imbalances and increased activity of neurotransmitters. These include dopamine and serotonin [2].

Dopamine has been shown to be a cause of the abnormal firing of neurons, which could cause hallucinations, particularly in conditions like schizophrenia [2]. Dopamine synthesis and discharge can be caused by many drugs, such as recreational drugs like cocaine, alcohol and methamphetamine. These also cause changes in neuronal activities [4]. Evidence for dopamine's effect on hallucinations is that drugs that are known to reduce the effect of dopamine on the brain usually reduce the symptoms of psychosis [3].

Visual hallucinations involve activity in the visual cortex similar to that in the auditory cortex, as discussed above. This area becomes active without any external visual input during a hallucination, generating images that can appear to be real. These, too, are affected by psychedelic and hallucinogenic drugs, in particular serotonin-based drugs [2].

Serotonin is a neurotransmitter that carries messages between nerve cells in the central nervous system, inside the brain, and the peripheral nervous system, around the body. This neurotransmitter is thought to affect learning, memory and happiness, as well as regulating body temperature, sleep and hunger. Lack of serotonin can cause depression, anxiety, mania and other health conditions [5].

With psychedelic and hallucinogenic drugs, the brain's serotonin (5-HT) receptors are activated in ways that alter typical sensory processing. These create complex visual phenomena [2]. This is the most common method by which hallucinogenic drugs work [6]. Regarding the 5-HT receptors, in particular the 5-HT2A receptor, there are two major pathways which are utilised to create the hallucination.

Classic hallucinogenic drugs such as Lysergic acid Diethylamide, more commonly known as LSD, stimulate the 5-HT2A receptor, a particular serotonin receptor subtype. It is proven that the 5-HT2A receptor is critical to the function of hallucinogenic drugs since blocking it with a different drug, ketanserin stops the hallucination. The affinity of a drug for the 5-HT2A receptor has a positive correlation to its potency, which is its ability to create hallucinatory experiences; for example, LSD has a high affinity and high potency. Hallucinogenic drugs are complementary to the 5-HT2A receptors, so they mimic the action of serotonin and bind to the receptors, which 'tricks' them into believing that serotonin has bound to it [6].

The other pathway is serotonin syndrome. When the central nervous system experiences excessive serotonin activity, the 5-HT2A receptor is overstimulated. This dysregulation can cause auditory and visual hallucinations, as well as delusions and other symptoms [7].

To conclude, hallucinations are complex phenomena that encompass a wide range of experiences. These can be caused by a range of causes, such as mental disorders like bipolar disorder and schizophrenia, but also hallucinogenic and psychedelic drugs, such as LSD and methamphetamine. Neurotransmitters and receptor cells, especially the neurotransmitters serotonin and dopamine, handle communications between various parts of the nervous system, which are key to understanding

hallucinations. This is a rapidly evolving field, with researchers continually pushing the boundaries of innovation and research to help patients suffering from hallucinations with new treatments and strategies to mitigate the effects.

By Anuron (Niyor) Das

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Learning behavior analysis using digital tools

Acknowledgement

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1. Introduction

Despite significant advances in learning analytics and intelligent educational systems, several important research gaps remain insufficiently addressed [1], [2]. This study aims to contribute to the field by targeting the following gaps: limited integration of cognitive interpretation in learning analytics, the use of outcome-based evaluation models, and the lack of behavior-centric personalization mechanisms.

1.1. Limited Cognitive Integration in Learning Analytics

Many existing learning analytics systems focus on performance data without providing a deeper understanding of the cognitive interpretations of learner behavior [3], [4].

Temporal and interaction data are usually overlooked or used only in a statistical manner. This work addresses this gap by linking measurable user behavior to well-established cognitive concepts.

1.2. The Use of Outcome-Based Evaluation Models

Traditional educational analytics frequently emphasize outcomes rather than the learning process [5], [6], thus limiting the system's ability to understand how learners engage, adapt, and persist over time. This work addresses this gap by shifting the focus to the process of learning and behavior modeling.

1.3. The Lack of Behavior-Centric Personalization Mechanisms

Most traditional educational analytics rely on static user profiles or attributes, resulting in limited personalization that does not account for real-time learner behavior [3], [5]. This mechanism has reduced adaptability and responsiveness. The proposed system to prioritize dynamic data over static data fills this gap by continuously updating behavioral indicators, thus allowing adaptive interventions to reflect the learner's current cognitive state as manifested through interaction behavior.

1.4. The Objective of This Paper

This paper explores using digital tools to analyze learning behavior and how AI-driven adaptation shapes the learning process. It proposes a scalable system architecture for adaptive learning analytics, contributing an applied framework that integrates digital platforms with AI-based personalization.

2. System Overview: ThinkR

2.1. Platform Architecture

ThinkR is implemented as a modular, multi-layer web application composed of a frontend layer, backend services, a data storage component, and AI-based analytics modules. This architecture supports scalability, modularity, and efficient processing of learner interaction data.

The frontend layer handles user interaction and data visualization and is implemented using HTML5, CSS3, and JavaScript to ensure a responsive and accessible interface. User actions, including task completion and assessment interactions, are captured at this level and transmitted to the backend for further processing.

The backend is implemented using Firebase and is responsible for authentication, data validation, and session management. Firebase Authentication ensures secure user identity management, while the backend mediates communication between the frontend and the data storage layer.

All platform data is stored in Firebase Firestore, a cloud-based NoSQL database organized into structured collections linked to unique user identifiers. The system collects three main categories of data: temporal, interaction, and performance data. Temporal data (e.g., time-on-task, session duration, and breaks) reflects attention and cognitive effort. Interaction data captures exploratory behavior through metrics such as sessions initiated and content generated. Performance data, including test scores, provides insight into learning consolidation and cognitive load.

2.2. AI and analytics modules

ThinkR integrates AI-based components to support content generation and learning adaptation. The platform utilizes the OpenAI API to generate learning materials, including lessons, study plans, and assessments, based on predefined pedagogical and structural constraints.

3. Learning Behavior Modeling

3.1. Behavioral Features

Each behavioral feature considered in this study can be computed using the data from users during engagement with the platform.

Behavioral feature	Computation	Cognitive Interpretation
Engagement	Active time / session time	Attentional stability
Consistency	Activity variance over time	Habit formation
Responsiveness	Behavior changes after feedback	Cognitive flexibility
Persistence	Retries per task	Motivation and effort

Table 1. Behavioral features

The objective of this approach is not to measure neural activity or infer underlying brain states, but to analyze and observe the behavioral manifestation of cognition during learning activities. The focus of this study is the measurable user behavior; the proposed feature set enables cognitive interpretation [7], [8].

3.2. AI-Based Analysis

AI infers learning states from observable behavior, analogous to cognitive neuroscience [7], [8]. Rule-based inference extracts behavioral features into high-level learning states, enabling transparent reasoning. Trend detection across sessions identifies changes in learner behavior over time, allowing the system to track learning dynamics without direct neural measurements.

4. Adaptive Learning Mechanism

4.1. Personalization Logic

The personalization logic behind the content generation across the tool integrates static and dynamic user data stored in the platform database. Static

data includes user-defined attributes such as learning style and scheduling preferences, while dynamic data is derived from ongoing interactions and behavioral indicators. By prioritizing dynamic behavioral data, the system adapts learning content to the learner's current cognitive and motivational state, reflecting a neuroscience-informed distinction between stable traits and fluctuating cognitive states [9].

4.2. Impact on Learning Behavior

The impact of the adaptive learning mechanism is evaluated through observable changes in learner behavior following personalization interventions. Rather than focusing on learning outcomes alone, the analysis emphasizes behavioral indicators that reflect how learners interact with the system over time.

5. Experimental Setup

The experimental study involved higher-education students from diverse academic disciplines and levels, who voluntarily used the ThinkR platform over one week as part of their regular study routines. No predefined tasks or exclusion criteria were imposed beyond active platform use, allowing for natural interaction across varied learning contexts. Participants used ThinkR to generate study materials, create assessments, complete learning sessions, and monitor progress, while all interactions were logged automatically without disrupting the learning process.

6. Results and Analysis

The analysis was based on behavioral metrics derived from interaction, temporal, and performance data collected over the one-week study period. Key behavioral features (engagement, consistency, responsiveness, and persistence) were computed using rule-based methods and analyzed longitudinally to identify trends across user sessions rather than isolated events. Overall, engagement increased over time, reflected by higher active time and reduced idle periods, indicating improved attentional stability and sustained cognitive effort without externally imposed study schedules. Consistency measures showed reduced variance in activity patterns for most users, suggesting the formation of more regular study routines supported by continuous behavioral feedback. Users also demonstrated responsiveness to adaptive feedback, adjusting session length, pacing, and task selection, which indicates increased cognitive flexibility and active strategy refinement. Persistence metrics revealed sustained engagement and increased task retries following lower performance outcomes, pointing to improved motivational regulation independent of immediate performance gains. Collectively, these results show that behavior-based learning analytics capture meaningful changes in the learning process that are not reflected by outcome metrics alone, supporting the effectiveness of behavior-centric, AI-adaptive learning systems in promoting deeper understanding and long-term engagement.

7. Ethical Considerations

Access to stored data is strictly controlled. Users are permitted to access only their own personal data, ensuring privacy and data isolation at the individual level. The only exception to this restriction occurs within the global leaderboard feature, where limited information, specifically the user's experience points (XP) and chosen username, is visible to other users. To preserve privacy, no real names or personally identifiable information are displayed. This design ensures transparency and competitiveness while maintaining user anonymity and data protection.

8. Conclusions

This study demonstrates that behavior-centric digital tools are practical and scalable, and can effectively analyze the learning process and support adaptation in educational systems, contributing to improved engagement and long-term learning performance.

By Buhai Iulia Georgiana

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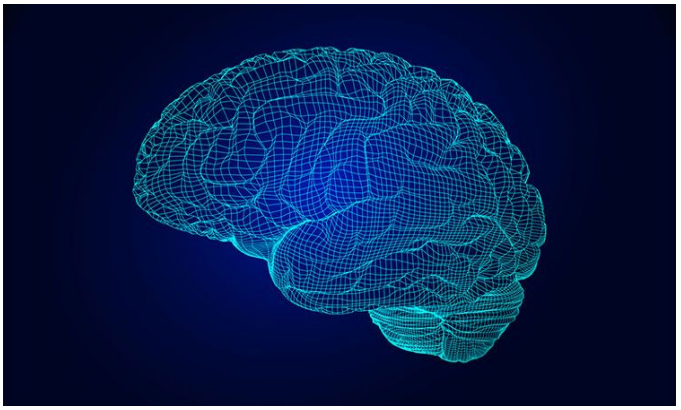

How Artificial Intelligence Makes Neural Activity Interpretable

Currently, artificial intelligence has reached a point of advancement that it not only analyzes external data, but it can also interpret the neural responses and predict subsequent brain activity. By learning the patterns of neural networks, AI can map out what people see, say, react to, and break down complex situations. Through the intersection of AI and neuroscience, the next step for medical rehabilitation is scientific research and human-centered applications. However, these developments bring up ethical concerns about whether to alter or understand the human brain.

AI Models that Decode the Brain

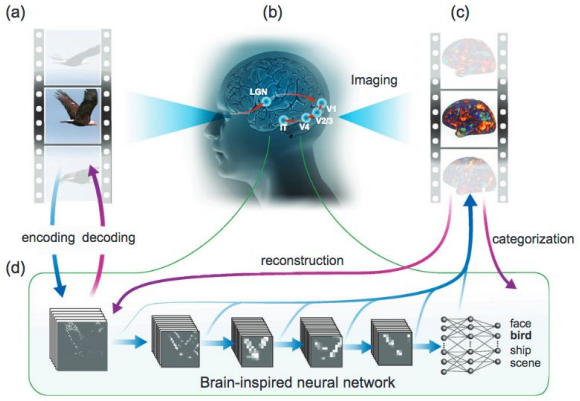

Fig. 1. Conceptual pipeline showing how neural signals are decoded using intelligence models.

The concept behind decoding the brain is simple: take signals from the brain, like electrical pulses, neuronal firing patterns, or fluctuations in blood oxygenation, and then transform the pulses into text images or movement commands. Although these actions require different data applications and machine learning, they all rely on the relationships between brain activity and perception.

Deep Learning for fMRI Reconstructs Images.

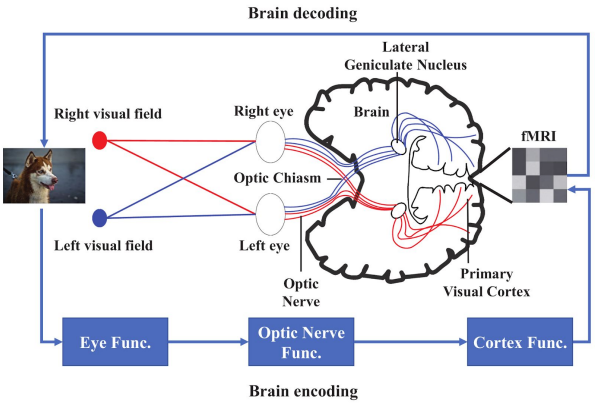

Fig. 2. Overview of neural encoding and decoding, which shows how brain activity is transformed into interpretable representations.

fMRI data is highly dimensional and complex. The immense volume of data points renders manual interpretation impractical. In the past researchers would need to show a human thousands of images in an MRI to learn how the brain recognizes images.

In 2022, a team from Osaka University attempted to incorporate the “Middleman” strategy [1]. Given that AI has already trained on millions of images to recognize concepts such as a dog or a clock, it is capable of representing the images in a mathematical sense. The researchers then aligned the brain signals to the mathematical concepts. Consequently, the AI not only reconstructed approximate shapes but also captured semantic content, meaning that the AI could categorize this information, which could assist with future implications like speech decoding and language model prediction.

Speech Decoding for Paralyzed Patients

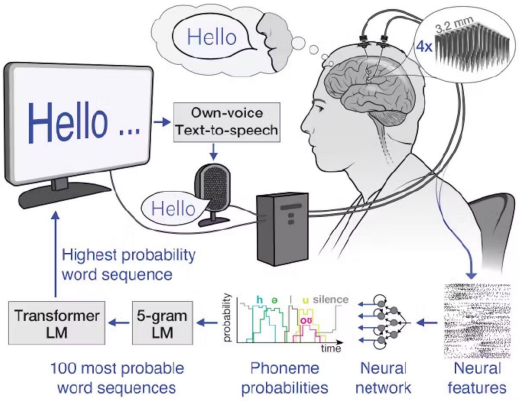

Fig. 3. Illustration of neural speech decoding technology used to restore communication in paralyzed patients.

Brain-computer interfaces (BCIs) are devices that convert brain signals into words. In 2023, Stanford's neural speech prosthesis placed tiny sensors in the motor speech area of a woman's brain, which controls how the mouth and tongue move [2]. Despite the paralysis, her brain continued to transmit neural-related commands, managed by an AI neural network controlling the electric signals. It learnt the neural patterns corresponding to her attempts at speech. As a result, a recurrent neural network decoded these patterns into text at 62 words per minute (nearly conversational speed). This demonstrates that language is controlled not only by vocal musculature, but also by a neural code that AI can now interpret.

Language Models Predicting the Brain Signals

One of the direct applications of large language models (LLMs) has been to use them for extracting semantic meaning from fMRI. In 2023, scientists at the University of Texas at Austin conducted a study examining the relationship between brain activity and GPT-like models [3]. When subjects listened to stories, the models did not reproduce the exact text but captured their overall semantic content. They were able to capture the overall meaning of stories, showing that AI can translate brain activity into language at the conceptual level.

What AI teaches us about the Brain

AI helps scientists in elucidating brain function by analyzing problem-solving patterns. A discovery in computational neuroscience is that conventional neural networks (CNNs) are designed to classify images; they develop internal layers that are similar to the structure of the human visual system [4]. In the brain, early visual areas detect features like edges and textures, while later areas recognize the object as a whole.

Furthermore, AI facilitates the study of higher-order cognitive functions such as memory, attention, and language.

Previously, researchers relied on labor-intensive, manually constructed models, but new AI models offer more convenient methods. For example, Attention Mechanisms in AI are similar to how humans hyperfocus on certain details. When neural network's predictions to real neural data are compared, scientists can better determine which computational principles the brain actually utilizes. Researchers argue that AI is helping to "reverse engineer" the mind more rapidly than previous methods.

Real Applications with human-computer interactions

The newly emerging ability to decode and model brain activity already has far-reaching practical implications in various domains. With the advent of thought-controlled interfaces, AI can translate images and movements into cursor control, the ability to move a computer pointer using neural signals. Experimental systems are also exploring emotional recognition, intent decoding, and cognitive argumentation. This changes how humans interact with technology, and it increases the potential for personalized adaptive digital experiences.

Medical

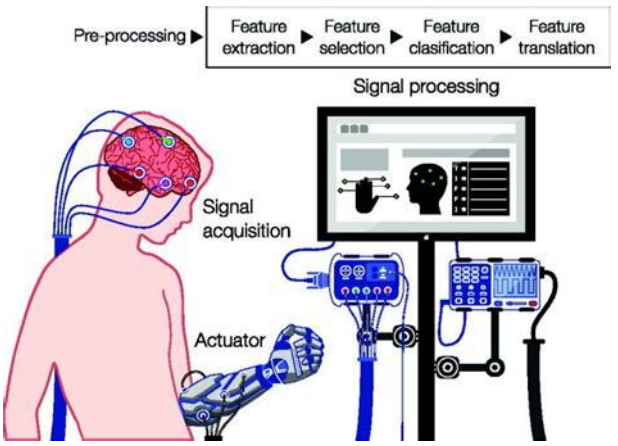

Fig. 4. Architecture of EEG-based BCI systems including signal acquisition, feature extraction, and classification.

AI-powered BCIs help individuals with ALS, spinal cord injuries, or stroke interact with their environment despite severe motor impairments. These systems track signals from the brain and turn them into actions like typing or moving a wheelchair. AI can also analyze brain signals to predict

seizures before they happen, giving them valuable time to prepare. It can also predict degenerative diseases like Alzheimer's and Parkinson's in brain scans much earlier than human clinics can, improving patient outcomes [5].

Research

AI excels at analyzing massive datasets of neural recordings, image scans, and cell type atlases. Through these analyses, AI enables the discovery of previously unidentified neuron types, understanding how large groups of neurons work together, and testing what might happen in the brain under different situations without doing real experiments. ML allows researchers to pose and address complex brain-related questions that were previously intractable.

Risks and Ethical Challenges

Currently, the integration of artificial intelligence into neuroscience raises significant ethical concerns that must be addressed alongside the growing technical developments. The concerns are primarily centered around privacy, physical safety, and fairness in access and application. As demonstrated by recent advances in neural image reconstruction, speech decoding, and semantic interpretation of brain signals, these ethical risks are no longer theoretical but are increasingly practical.

Privacy and Autonomy

Brain data is the most sensitive data humans can produce. Neural activity can disclose individual preferences, intentions, and private mental content. This has led to legal scholars to propose establishing cognitive liberty and mental privacy as fundamental rights [6]. The transmission of brain data introduces medical and security risks. As the brain data is vulnerable, if streams are intercepted or hacked, invasive BCIs could require surgery, carrying risks of infection, scar tissue, or device failure.

Although current models cannot decode complex thoughts without user cooperation, this limitation is likely temporary as technology continues to advance. Misuse of brain-decoding technology could coerce individuals, manipulate behavior, or covertly monitor mental activity, which would be a serious violation of privacy. Furthermore, even non-invasive systems could pose psychological or cognitive burdens, raising ethical questions about patient safety and long-term monitoring. Even though BCIs enabled breakthroughs such as neural speech prostheses for paralyzed patients, researchers and ethicists have weighed the benefits against the long-term health risks, device dependence, and challenges of informed consent [2].

Algorithmic Bias

Neural data sets are often small and demographically narrow. What this means is that if models are trained on a limited number of subjects, they cannot be applied broadly due to the accuracy difference across numerous populations. This could potentially lead to unequal access to benefits. Responsible Innovation must prioritize safety, consent, and fairness at the center of future development. As AI models are increasingly used to infer general principles of brain function, biased datasets risk embedding demographically specific assumptions into both scientific conclusions and clinical applications.

Conclusion

Scientists are now working on BCIs that can not only read signals, but can also write neural information that could enable artificial vision for blind people or give a sense of touch and movement through memory prosthetics. Some researchers suggest AI could enhance memory into cognition, and that, eventually, a universal code may transfer human thoughts into machine signals.

However, these opportunities also come with ethical questions about privacy, autonomy, and fairness. AI is becoming an integral part of everyday life, and now it is even making significant developments in neural activity and neuroscience. These developments have the potential to

transform medicine, science, and society, contingent on their being pursued with ethical integrity.

By Deepanwita Nagamalla

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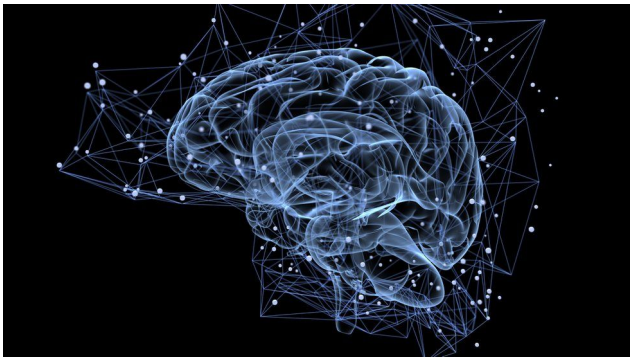

Use of Artificial Intelligence in Medicine

For a long time, AI has been employed in the medical sector, assisting doctors in analyzing complex data, detecting hidden patterns in images and lab results, and accelerating the diagnosis and treatment process. AI has also enhanced decision-making and reduced the possibility of errors, allowing medical professionals to focus on complex, high-level patient care. AI assists in predicting the progression of a disease, preparing treatment plans, tracking patient responses, and even performing administrative work. Despite the overwhelming benefits and evidence of the effectiveness of AI, many medical professionals and patients are still hesitant to adopt it.

Artificial intelligence is now integrated into various branches of medicine, enhancing the quality of medical decisions and their effective implementation. Due to its high performance level in tasks requiring a large amount of data, it is particularly valuable in medical diagnostics. Its ability to provide quick and accurate results makes it very useful in medical imaging and radiology. In these fields, AI systems examine X-rays, CT scans, MRIs, and mammograms to identify tumors, fractures, strokes, and other pathologies. AI systems also proved to be particularly useful during the COVID-19 pandemic. AI-assisted analysis of CT scans reached the same level of accuracy as trained radiologists. And recent studies now show that AI systems may even outperform them [1].

Machine learning algorithms in oncology examine digital slides to identify cancer cells, assess tumor grades, and forecast disease progression, often performing better than individual oncologists in terms of consistency and sensitivity [2].

Another area in which such rapid improvement has been observed is in BCI systems. BCI stands for Brain-Computer Interface. Bypassing the body's normal motor or sensory pathways, BCIs establish a direct communication channel between the brain and external devices, allowing the user to control computers, prosthetics, or other devices using only their thoughts or brain activity. To do it successfully, they measure neural signals such as action potentials, local field potentials, or brainwave oscillations. These signals are frequently recorded from regions such as the prefrontal cortex for cognitive tasks or the motor cortex for movement intentions.

The primary purpose of this system is to help people with disabilities regain their functional capabilities and enhance their performance. BCIs are widely used in medicine, neuroscience research and human-computer interaction. They can enable advanced control of prosthetic limbs, assist in motor rehabilitation, but can also potentially enhance attention, memory, and other cognitive functions. Normally, calibrating a BCI system demands a lot of time and effort. However, scientists from Brown University developed a system in which the participants could see the improvement in cursor control within 30 seconds to a few minutes. This meant that the computer adjusted itself along with the user much more efficiently and effectively [3]. The use of AI-based statistical learning algorithms replaced the long process of model adjustment to the individual through trial and error. This allowed participants with tetraplegia to significantly enhance their quality of life, more quickly.

They could control a robotic arm with their brain signals to perform daily tasks such as grasping and manipulating objects. Apart from that, they were able to control tablet computers using only their thoughts, thus surfing the internet and sending messages.

Additionally, scientists have managed to create a BCI system that can interpret brain signals to produce speech, achieving up to 97% accuracy [4]. Machine learning models figure out how brain activity is connected to movement or speech. They do this by learning the relationship between patterns of brain signals and the patient's intended actions. While the patient is performing an action, or simply thinking about it, the systems measure electrical activity generated by neurons. Once the neural data is prepared, algorithms learn to pair neural signals with the corresponding movements or speech outputs, so they can operate in real time. For example, when a user wants to move a limb or talk, the system reads the brain signals and turns them into commands for devices or speech systems. Many BCI systems also feature adaptive feedback loops, which let the system modify its parameters when it receives new information. The constant interaction between the user and the system helps to improve its performance and take advantage of the brain's neuroplasticity, which allows for better and more accurate communication between the brain and the outside world.

Although AI can perform exceptionally in some areas, it is not used as often as expected. Research has shown that most people trust human doctors much more than AI, even when they are told that the AI performed better than the human doctor [5]. This is known as algorithm aversion. People often expect more from algorithms than from humans. They lose faith in algorithms when they make small mistakes, whereas human mistakes are considered normal and acceptable [6], [7]. This means that a wrong diagnosis by a computer program is often perceived as much worse. If perfection is demanded from a computer program before it is allowed to replace humans, thousands of lives may be lost that could have been saved if they had been allowed on the road a bit earlier [8]. People naturally start applying social norms and expectations to computer programs [9].

Thus, when collaborating, the strengths of human and AI capabilities complement each other. While AI deals with large volumes of data and repetitive tasks, humans provide context, ethics, and the ability to interpret complex situations. In radiotherapy, seizure prediction, emergency response, and other applications, collaboration can greatly improve performance. Thus, instead of trying to avoid the use of AI, AI should be responsibly leveraged to improve overall outcomes and ultimately save lives.

By Ema Bubola

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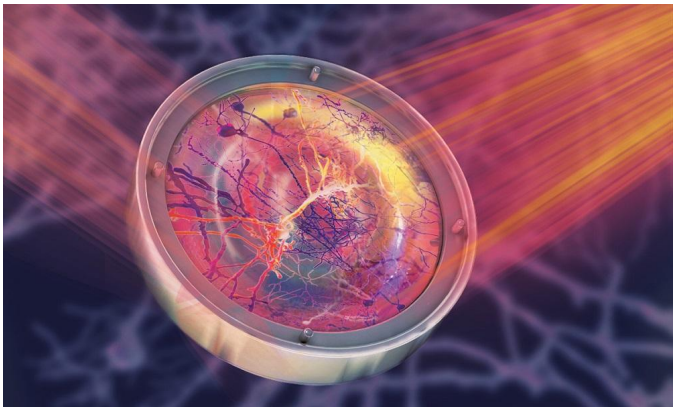

In Vitro Brains

Introduction

In vitro (in glass) refers to something that exists outside of a living organism; in this case, brain tissue is cultivated on a Petri dish. Smaller, simplified versions of brains, as well as other organs can be produced in a lab to mimic the structural and functional characteristics of their in vivo (bodily) counterparts. These are neural (or brain) organoids and have the capacity to be used to model diseases, treatments and biological interactions [1], [2]. However, neural organoids are surrounded with debate and controversy.

How They Work

Cerebral organoids can be made from pluripotent stem cells, which are self-renewing stem cells which possess the ability to differentiate into any of the specialised cell types in the body, except for placental cells. These cells can be cultured and cultivated to form clusters called embryoid bodies. The embryoid bodies can be induced to form a neuroectoderm which is then left to develop in a gel membrane extract, known as the matrigel, that mimics the environment of the body. The result is a cerebral organoid that has developed spontaneously. This means that the proportions and areas of the brain which have grown have not been controlled. This is beneficial in that it loosely resembles the development of the brain in utero, so therefore can be used to study the formation of brains in fetuses. Additionally, differentiation can be guided to make brain region-specific organoids, which are only representative of one tissue type.

Assembloids can also be created; these are three-dimensional biological models formed from the fusion of multiple organoids, allowing them to interact. They are used to model interactions between different areas of the brain [3].

The Zika Virus

Although still a relatively recent development, brain organoids have already shown promise, specifically in disease modelling. For example, Zika Virus (ZIKV) is a mosquito-borne virus that presents flu-like symptoms [4]. By exposing brain organoids to an isolated Brazilian Zika strain, a model showing the causation between the Zika virus and Microcephaly cases was created. Microcephaly is when a baby is born with a head that is significantly smaller than expected. Zika Virus causes Microcephaly by destroying the stem cells responsible for generating neurons [5], [6]. The model was also used to demonstrate how certain drugs could prevent the infection or block Zika viral replication [7].

Other Applications

Cerebral organoids have been successfully transplanted into regions of neurodegeneration in mice, and there is hope that this process could eventually be applied to humans [8]. Furthermore, the testing of the safety and efficacy of drugs that affect the brain can be conducted via the use of cerebral organoids.

Another area where brain organoids can be applied is through the pluripotent stem cells of individuals affected by Alzheimer's Disease. Generating brain organoids from these cases has allowed for the previously impossible observation of some of the structures that are responsible for the symptoms of the disease [9], [10]. Studies using human midbrain organoids have also been conducted focusing on another neurodegenerative disorder, Parkinson's disease [11], and neural organoids have been used to observe many neurodevelopmental disorders such as

Conclusion

Overall, despite some ongoing ethical concerns, neural organoids have the capacity to make a real, positive difference in the world of neuroscience, and this was clearly demonstrated with their infamous work surrounding the Zika virus infection. They have already been used to model the mechanisms underlying many diseases, but there are many that still have not been investigated in depth. Neural organoids could foreseeably play an important role in overcoming these gaps in our knowledge. Additionally, they have a potential for providing a suitable alternative for animal testing in preclinical drug trials, and as animal testing becomes less popular, neural organoids may be able to take their place.

By Emma Watkinson

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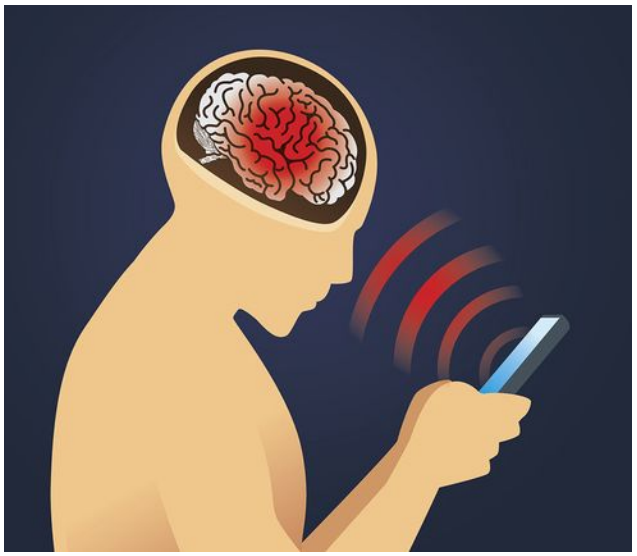

How Excessive Screen Time Accelerates Cognitive Decline

The term ‘brain fog’ describes a temporary, subjective experience of mental cloudiness, sluggishness and cognitive dysfunction. This can cause sufferers to feel confused, frustrated and “in a daze”. It is often a byproduct of conditions like long Covid, fibromyalgia, and strong medical treatments like chemotherapy.

Recently, links have been drawn between excessive screen time use and brain fog, with one BBC article citing a Danish study published in 2024 which involved 181 children from 89 families [1]. For two weeks, half of them were limited to three hours of screen time per week and asked to hand in their tablets and smartphones. Although research was limited, the study concluded that reducing screen media had a positive effect on psychological symptoms of both children and adolescents and enhanced social behaviour. This is a concerning pattern to notice, with screen time and device ownership reaching unprecedented levels, causing many researchers to analyse the effects of long term divide use and cognitive structure.

In 2016 in Great Britain, 70% of adults accessed the internet using a mobile phone or smartphone, up from 66% in 2015 and nearly double the 2011 estimate of 36% [2]. At the beginning of 2026, this number had risen to 95% [3].

While smartphone ownership is not an innate danger, many sources document a negative correlation between excessive screen time and declining cognitive function, especially in relation to excessive consumption of short form content [4]. A study by the education committee in the UK found that by the age of eight a child will typically spend 2 hours and 45 minutes online per day. It rises to over four hours by age 11-12, while a child is still in primary school [5].

Generation Alpha, are those born between 2010 and 2024. Occasionally nicknamed “Generation Glass” in reference to the screens that are woven throughout their childhood, Generation Alpha is said to be negatively affected by the constant presence of technology in their upbringing. This can result in a difficulty to remain focused for long periods of time, like in a classroom. In the USA, reading performance has reached its lowest level since 2005, with that number being expected to continue falling [6]. Attention deficit, sluggishness, mental cloudiness and cognitive dysfunction are becoming synonymous with excessive screen time use and brain fog.

Mobile phones and tablets have been around for just over a decade, so there are no available long term studies on the effect of excessive screen time; however, links are already being drawn between excessive screen time and physical changes to the structure of our brains, including thinning of the cerebral cortex, reduced white matter integrity and altered grey matter volume.

Thinning of the cerebral cortex in association with excessive screen time was found in a recent study. This is the brain’s outermost layer responsible for processing memory and cognitive functions, such as decision-making and problem-solving [7]. Degeneration of this area results in slower cognitive processing, reduced memory performance, impaired attention, and difficulties in emotional regulation. A similar effect was found on grey matter, which is found throughout the brain, in addition to the cerebral cortex.

Grey matter in the brain is responsible for processing, interpreting and analysing information, serving as the center for intelligence, thought and sensory perception. A study performed by Stanford found that adults with excessive screen time use had a lower volume of grey matter in their brain [8].

Furthermore, reduced white matter integrity can be seen in pre-kindergarten children with a screen time higher than the levels recommended by the American Academy of Pediatrics [9]. White brain matter is composed of myelinated nerve fibres that connect different brain regions, and is responsible for the development of learning, cognition, motor movement and sensory processing.

This study tested 47 otherwise healthy children, and found lower measures of microstructural organisation and myelination of white brain matter, resulting in lower performance in corresponding cognitive assessments.

Additionally, the thinning of the cerebral cortex and grey matter, and a reduced volume of white matter, are commonly associated with old age [10], [11]. The alarming suggestion that excessive screen time can result in premature ageing of the brain strongly outlines the need for continuous research and long-term studies into the effect of devices on the structure of our brains.

Overall, a negative correlation can be observed between screen time and cognitive performance. Despite the useful capabilities of modern technology, the effects they have on the brain should not be overlooked.

By Fenn Parkes

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How Early Brain Monitoring Is revolutionising Child Development.

Clinically relevant neurodevelopmental delays are experienced by over 10% of children worldwide experience . This can arise from a variety of factors, including prematurity, abnormal brain structures, or predominantly genetic influences [1]. Early childhood is vital in synapse formation, the connections between neurons which underpin behavioral and psychological development [2]. As a result, early brain monitoring is a crucial tool in the diagnosis of neurological disorders such as Attention Deficit Hyperactivity Disorder (ADHD), allowing for premature recognition and intervention. Without early identification, subtle neurological differences may go unnoticed until academic or social difficulties become more pronounced, reducing the effectiveness of later interventions.

What Neurodevelopmental Disorders Are

Brain development begins around 2 weeks post-conception through to early adulthood. This time of growth involves the formation of synapses and the maturation of brain regions for communication and motor skills [3].

Neurodevelopmental disorders (NDDs) are defined as disorders that impact the growth and development of the brain, specifically the prefrontal cortex and the cerebellum. This can result in impairments in motor and cognitive skills as well as difficulties to communicate effectively [4].

NDDs arise largely due to genetic factors during prenatal development. However, environmental factors may also contribute to their onset [1]. For instance, a lack of maternal folic acid can trigger neural tube defects such as anencephaly, which is a condition where a baby is born without parts of their brain or skull [5].

How Early Detection Works and Why It Is Medicinally Important

One way in which brain development can be monitored to improve child development is via function Near-Infrared Spectroscopy (fNIRS) [6]. This non-invasive technique allows the overall haemoglobin, oxyhaemoglobin and deoxyhaemoglobin levels in the cerebral cortex to be observed [7]. fNIRS works by emitting infrared light from light sources on the scalp which penetrate 2-3 cm into the cortical tissue, the outermost layer of the brain. The absorbance values are then calculated using the Beer-Lambert law and recorded. It shows that oxygenated blood absorbs more light at different wavelengths [6]. Results can help to identify areas of hyperactivity in the brain and so aid the diagnosis of NDDs [8].

Limitations

However, it is very important to discuss the ethical implications of fNIRS. The sensitivity of neural data, especially of infants, is of critical importance as it is personal, biologically identifiable, and collected from individuals who cannot provide informed consent. As a result, it is essential that parental consent is given to carry out these neurological procedures and that the data collected is protected in ways outlined by the Data Protection Act (2018). This is because neural data can indicate psychological and behavioural implications, which could potentially be misused or exploited by third parties for purposes such as profiling, discrimination, or commercial gain.

Additionally, over-reliance on technology such as fNIRS may lead to premature diagnoses in the absence of clear behavioural symptoms. As a result, parents

may alter their expectations and educational decisions, while patients themselves may internalize their diagnosis, shaping their self-perception and confidence.

Implications and Future Research

fNIRS is revolutionising the diagnosis and monitoring of NDDs in young children due to its non-invasive and portable design [7]. This technique involves measuring the child's head in order to fit a lightweight fNIRS cap containing infrared light sources and detectors [8], allowing for a painless and comfortable data collection. Due to its painless and movement tolerant nature, it is well suited for infants and toddlers, who are more challenging to perform neuroimages on. Contrastingly, functional Magnetic Resonance Imaging (fMRI) is more expensive and less child-friendly, as it requires a large scanner [9]. Ultimately the fNIRS portability enables researchers to study brain function in more natural environments, improving the feasibility of early and repeated monitoring of child development. Early brain monitoring also allows for early intervention of NDDs when atypical results are detected. This is essential when it comes to reducing the severity of neurodevelopmental disorders by providing family and educational support. Early intervention may include therapeutic approaches (such as speech therapy) or the implementation of Individual Education Plans to support academic development [10].

Looking forward, the integration of AI in analysing neural data may enhance accuracy and efficiency of early detection of neurodevelopmental disorders. Additionally, improving the accessibility of technology such as functional Near-Infrared Spectroscopy would be beneficial for over 10 % of children globally [1]. Children from lower socioeconomic backgrounds may be at greater risk of developmental delay due to environmental factors. Ensuring equitable access and establishing clear clinical guidelines will therefore be essential to prevent misuse and to maximise the positive impact of early brain monitoring on child development. As these technologies advance, early brain monitoring may enable earlier identification of atypical neural patterns, allowing for timely intervention and more targeted support that improves long-term cognitive and developmental outcomes.

Hana Elizabeth Mathew

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Understanding Alzheimer’s Disease

Introduction

Around 982,000 people in the UK suffer with dementia, with projections indicating that this number will rise to 1.4 million by 2040. Additionally, a third of those patients do not have formal diagnosis, highlighting the need for increased awareness and support for the patient. Alzheimer’s is different to other medical conditions. It slowly reshapes the identity of the patient, their relationships, and their daily life. It affects families, communities, and generations, emphasising the importance of acknowledging and spotting Alzheimer's before the damage done is irreversible.

Alzheimer's Disease Explained

Alzheimer’s is one of the most common types of Dementia with studies showing it accounts for 60-80% of Dementia cases [1], [2]. Dementia is a progressive neurological condition that gradually damages the brain over time and is the general term for memory loss. Alzheimer’s is linked to the build-up of two abnormal structures called plaques and tangles which are suspected to irreversibly damage nerve cells. Plaques are deposits of a protein fragment called beta-amyloid that build up in the spaces between the cells whilst tangles are twisted fibres of the protein tau that build up inside the cells [2]. Many experts believe that these tangles and plaques play a critical role in blocking communication between cells as well as disrupting processes that cells need to survive. This destruction and death of nerve cells cause memory failure, personality changes, problems carrying out daily activities and other symptoms of Alzheimer’s disease.

The Progressive Steps of Alzheimer’s

Alzheimer’s worsens with each passing day [2], [3]. Alzheimer’s first starts off very mild, with symptoms prone to being ignored and overlooked such as forgetting familiar words or the location of everyday objects. The next stage of Alzheimer's is the moderate stage. During this stage, dementia symptoms are more prominent. The patient may act in irrational ways through an irregular mood. Damage in the nerve cells in the brain can make it more difficult for the patient to think, carry out tasks by themselves, and control their temper. The symptoms of the middle stage of Alzheimer’s include being forgetful of personal history, feeling moody or withdrawn, and demonstrating personality and behavioural changes. Finally, in the last stage of Alzheimer’s, patients lose their ability to respond to their environment, carry conversation, and to control their movement. At this stage, patients may lose awareness of recent experiences and their surroundings, have difficulty communicating, and require constant assistance.

Approaches to Addressing Alzheimer's Disease

There is no cure for Alzheimer’s [4]. However, there may be treatments to help relieve the symptoms of Alzheimer’s or prevent the stages from progressing. Recent studies show that lithium plays an essential role in normal brain function and could possibly resist brain aging and ultimately prevent Alzheimer’s disease. Supporting evidence consists of extensive research on both the concentration of lithium in the brain as well as its interaction with the toxic amyloid plaques.

Yanker, a researcher of this topic, concluded that lithium bonded to the toxic

amyloid plaques, breaking them down and preventing damage done to the nerve cells. If lithium availability deplenished, toxic amyloid plaques may build up and cause irreversible damage to nerve cells. Studies also show that monitoring the levels of lithium in the brain can possibly predict the development of Alzheimer's. Yanker measured the levels of around 30 types of metals in the brain using mass spectrometry [4]. He deducted that in cognitively healthy patients, lithium concentrations remained high. Whereas the patients with Alzheimer's disease showed decreased concentrations of lithium in the brain, highlighting how lithium depletion has a causal relationship between the development of Alzheimer's.

Conclusion

Understanding Alzheimer's and pinpointing its causation has been a difficult task. The lack of evidence and research into the treatment of Alzheimer's limits the ability to mitigate its development. However, ongoing research into the root cause of Alzheimer's will continue to develop, striving to reduce the amount of people suffering from this disease.

By Jamie Duong

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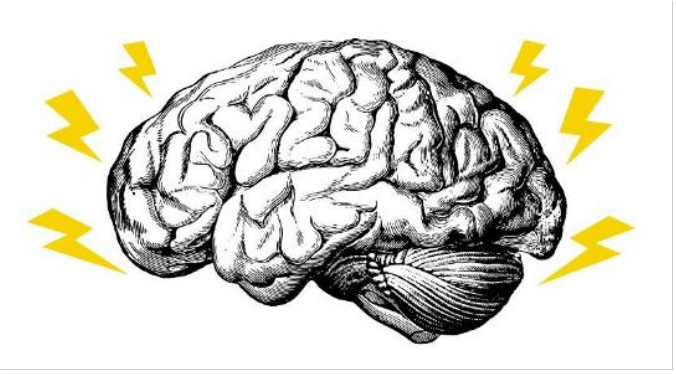

Advances in Epilepsy Treatment: From Pharmacotherapy to Neurotechnology

Introduction

Epilepsy is described as chronic seizures due to aberrant electrical activity in the brain. Epilepsy affects more than 50 million people worldwide, remaining one of the most prevalent neurological conditions, yet nearly one-third of diagnosed patients develop drug-resistant epilepsy, emphasizing the need for alternative interventions. This challenge has driven the creation of surgical, device-based, and novel molecular therapies to improve clinical outcomes for this 30 percent [1]. Together, these advancements reflect a shift from generalized seizure suppression toward precision, mechanism-based treatment strategies.

Pharmaceutical Foundations

The primary treatment for epilepsy is anti-seizure medication, with commonly used agents including cenobamate, brivaracetam, and lacosamide. These drugs function through several mechanisms, such as stabilizing the neuronal membrane, reducing excitatory glutamate transmissions, enhancing the inhibitory gamma-aminobutyric acid (GABA) pathway, or regulating ion channel activity, including sodium or calcium channel modulation [2]. By limiting excessive neuronal firing and maintaining the balance between excitation and inhibition in the cerebral cortex, these drugs significantly reduce the frequency of abnormal electrical discharges and, in turn, the frequency of seizures. Clinically, this can improve quality of life and reduce the risk of seizure-related injury. However, because these medications alter normal neural signalling, they are often associated with adverse effects and are limited by drug resistance.

Surgical Interventions

In carefully selected patients with focal epilepsy, a subcategory of epilepsy where seizures originate from a specific localized area of the brain, surgical removal of the epileptogenic focus can lead to long-term seizure freedom or substantial reduction [3].

Figure 1; drug classification of common ASMs

Drug	Drug Class	Primary Mechanism	Molecular Target	limitation
Cenobamate	Dual mechanism ASM	Enhance GABA receptor-mediated currents	GABA receptors and persistent Na^+ currents	Requires slow titration due to hypersensitivity risk
Brivaracetam	SV2A modulator	High-affinity binding to synaptic protein 2A	SV2A protein	Possible behavioral adverse effects
Lacosamide	Sodium channel modulator	Enhances low inactivation of voltage-gated sodium channels	Voltage-gated Na^+ channels	Dose-dependent dizziness and PR interval prolongation
Ethosuximide	Calcium channel blocker	Suppresses thalamic oscillations	T-type Ca^{2+} channels	Limited to absence seizures
Perampanel	AMPA antagonist	Reduces excitatory transmissions	AMPA receptors	Psychiatric side effects

Another surgical option includes Laser Interstitial Thermal Therapy (LITT), a less invasive alternative to open craniotomy; essentially, it ablates the epileptogenic tissue by delivering thermal energy through a small burr hole [3]. This method often results in shorter hospital stays and faster recovery. Although both interventions carry their risks, outcomes are generally favorable if patients undergo a rigorous evaluation and selection.

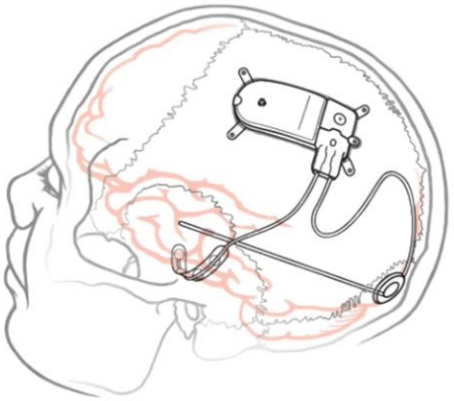

Figure 2 illustrates the implanted RNS system, adapted from NeuroPace's RNS systems physician manual

Neuromodulation and Implantable Devices

Neuromodulation has led to significant breakthroughs in the treatment of epileptic seizures among patients with drug-resistant epilepsy [4], [5], [6]. There are three chief

types of neurostimulation: vagus nerve (VNS), deep-brain (DBS), and responsive (RNS). Vagus nerve stimulations send periodic impulses to the vagus nerve in the neck, indirectly modulating cortical excitability. Deep-brain stimulations continuously target seizure-specific subcortical sites in a closed-loop system [5], [6]. Finally, responsive neurostimulation targets localized seizure areas to send electrical pulses after the detection of abnormal electroencephalographic (EEG) activity in real-time, thus arresting the progression of seizure escalation [4], [5], [7]. Advances in neuromodulation have reshaped therapeutic strategies for patients with drug-resistant epilepsy.

Gene Therapy and Molecular Approaches

Unlike previous therapeutic strategies, gene therapy is targeted at the underlying genetic causes of epilepsy rather than simply treating the symptoms. Many severe epileptic disorders, such as Dravet syndrome, are associated with mutations in genes like SCN1A that hinder normal neuronal functionality [8]. Molecular tools such as viral vectors, most commonly adeno-associated viruses, are used to deliver functional copies of defective genes or to selectively modulate gene expression in affected neurons. Studies have supported that restoring the SCN1A expression could reduce seizure escalation and frequency as well as improve survival according to animal models [8], [9]. Other molecular approaches include CRISPR-based genome editing and antisense oligonucleotides, a single-stranded DNA or RNA designed to bind to specific mRNA sequences to modulate protein expression [9]. Both of these approaches are aimed at precisely regulating pathogenic mutations. Although these therapies remain largely experimental, early clinical trials suggest that these interventions could offer disease- modifying potential.

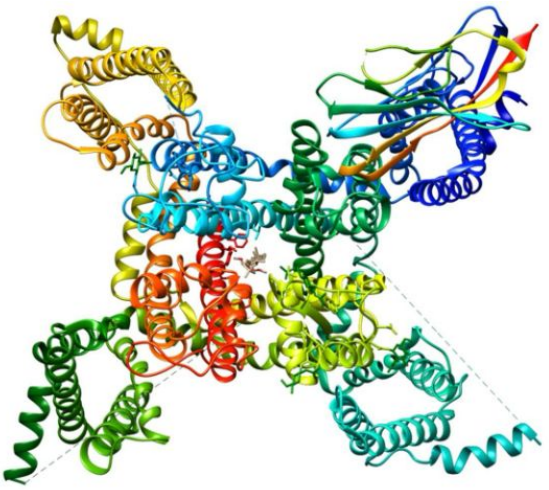

Figure 3: Visualization of SCN1A protein; Goultard59

Conclusion

The treatment of epilepsy has evolved far beyond the traditional reliance on anti-seizure medications. While pharmacotherapy remains a foundation, drug-resistance in epileptic patients has driven the development of more advanced targeted interventions. Surgical resection and minimally invasive laser techniques now

provide curative potential for select patients. Device-based interventions such as deep-brain stimulation also offer a dynamic, circuit-based modality for those unable to take part in the surgery. Most notably, gene-based therapies represent a transformative shift in the field. By targeting molecular and genetic mechanisms, these interventions move beyond symptom management and into disease modification. Novel breakthroughs in neuroscience, genetics, and biomedical engineering continue to transform the prospects of treatment, as interdisciplinary research generates new strategies for preventing and controlling seizure escalation in patients with epilepsy.

By Jana Elnahas

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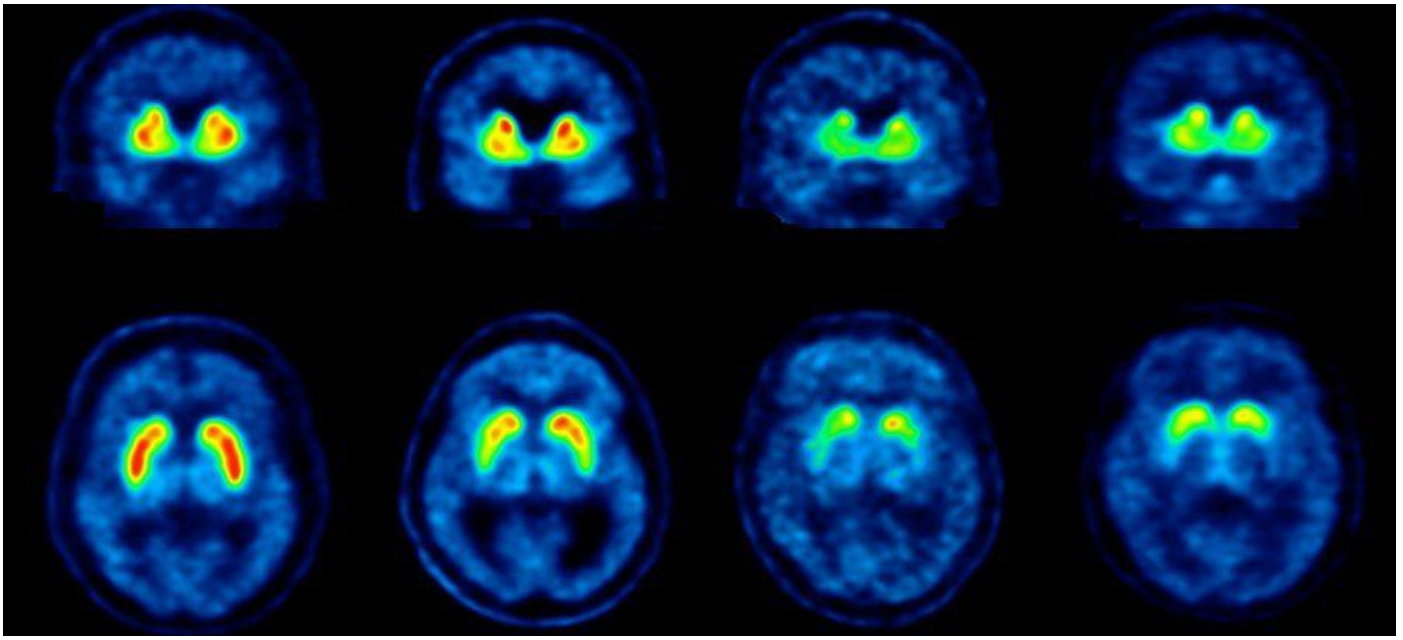

AI's Use in Curing Parkinson's Disease

Searching for a cure for any broad disease like Parkinson's disease (PD) is an incredibly long and difficult process which grows increasingly more expensive as research progresses. This is due to the increased fraction of passive research in current research, leading to capital costs and researcher's wages to also increase [1], [2]. Passive research has become more popular because it is now possible to collect more quantitative data through technology such as phones. However, by using artificial intelligence the entire research process can be dramatically sped up and therefore made cheaper. This was proven by Cambridge University's recent research project where they sped up the initial screening process ten-fold and cut down costs a thousand-fold [3]. Nevertheless, a singular project cannot establish AI's role in such a massive field as medical research. Therefore, before AI becomes well established in medical research, its benefits and uses should be evaluated when it comes to developing treatments that will eventually be used on patients worldwide.

AI's main role is to search through huge masses of data to find patterns that humans would otherwise miss. First, the model will undergo unsupervised machine learning by being fed unlabelled data so that it can detect patterns on itself. This is helpful to find new patterns. An example of this was the discovery made at NYSCF which found that cellular imprints are unique to individuals [4], [5], and the discovery that skin cells of people with Parkinson's diseases can be distinguished from those from healthy people, revolutionising diagnoses [5]. This was made by AI searching through 6 million images of skin cell samples of healthy people and people with Parkinson's disease. This is especially crucial for the diagnosis of early Parkinson's disease, which has low diagnostic accuracy with an average of 58% of patients being correctly diagnosed [6].

Another type of machine learning is supervised machine learning, that feeds models labelled data so it can identify known patterns [4]. Patterns are so vital as they are used to generate new ideas to find new treatments [7]. This includes symptomatic treatments which can be quite random as multiple medications are prescribed over a long period of time so side effects can cause more harm. Although, AI could predict both the individual's symptoms and harmful medication combinations that should be avoided. This is made possible by surveillance cameras and phones allowing for body kinematics, joint positions, gait irregularities to be studied on an individual basis in cases of Parkinsonism so their symptoms can be predicted and treated [8].

In addition to symptomatic treatment, curative treatment is also essential when treating Parkinsonism. AI can make this

possible by aiding discoveries so next steps can be made. This is especially relevant with NYSCF's recent discovery that the skin cells of people with PD are distinguishable from skin cells of healthy people. Therefore, the next steps can be taken to modify these skin cells into stem cells and afterwards into neurons where drugs are tested on without using up volunteers' limited stem cells [5]. Also, the recent discovery made in Cambridge University utilised AI to find a compound that could block the proliferation of amyloids, which is the sticky protein that causes the protein buildup in the brain causing Parkinson's disease [3]. This led to finding many promising compounds which when tested were able to stop the buildup and even start to scratch away at the surface of the buildup which could essentially eliminate a person's Parkinson's disease fully [3].

The root of Parkinson's Disease is protein build up in the brain that blocks dopamine receptors meaning less dopamine is released slowing basic functions carried out in the brain [5]. There are both genetic and environmental factors that contribute towards the development of Parkinson's although it is predominantly rooted in environmental factors [9]. This is due to how toxic chemicals in pesticides and traffic pollution metabolise proteins in the brain leading to buildup [9]. Therefore, the number of people with PD is rapidly increasing [9].

However, genetics do still play a role in causing PD as certain genes code for proteins that are more likely to be metabolised by toxic chemicals, though only 2% of people with PD develop PD due to a monogenic cause [9]. Monogenic is a single genetic mutation that causes a direct link, and a person with this genetic mutation may develop Parkinson's through the proliferation of amyloids and therefore directly grow the root of the disease [10]. Consequently, removing the environmental cause is the most plausible treatment method. Although, this still does not cure PD as removing the cause does not remove the damage already caused.

Therefore most treatments aligned with curing Parkinson's attempt to eradicate the root of the disease: the build-up of proteins caused by amyloids. Still, the protein buildup could return if the proteins are triggered again by environmental factors so to fully cure PD the genetic mutations coding for these proteins should be altered. Now, using AI to sift through collected data and find the genetic mutations coding for the proteins. This can lead to a personalised treatment plan being made for that individual to remove the existing protein build up in the brain that has caused the development of PD, as well as a symptomatic treatment plan where existing and future symptoms are treated to ease any pain that the person is in.

Introducing AI into the world of research can be incredibly risky with the increased likelihood of data being stolen, shared or altered, which is already a threat as technology has already become highly incorporated in the field. In the UK, if a data breach were to occur it would halt the research process temporarily as investigations undergo to contain the damage, find the causes and handle the consequences [11]. Yet, the project may still be abandoned after the issue is resolved if the organisation's reputation has been hurt, such as AMCA who lost 19.6 million patients' medical records and was forced to shut down [12].

AI can make mistakes just like people. This is again proven by how AI struggles with math problems [13], meaning AI has also developed a reputation of being unreliable. This is especially concerning with how it would potentially be entrusted with global health. AI hallucinations stem from the data it is trained on, resulting in biases and inaccuracies [13].

Overall, AI is helping with medical research by speeding up and making the process cheaper. Developing creative, feasible ideas for research can be contributed to from both people and AI to formulate the best cures for diseases in the future [7].

By Jumana Omaimen

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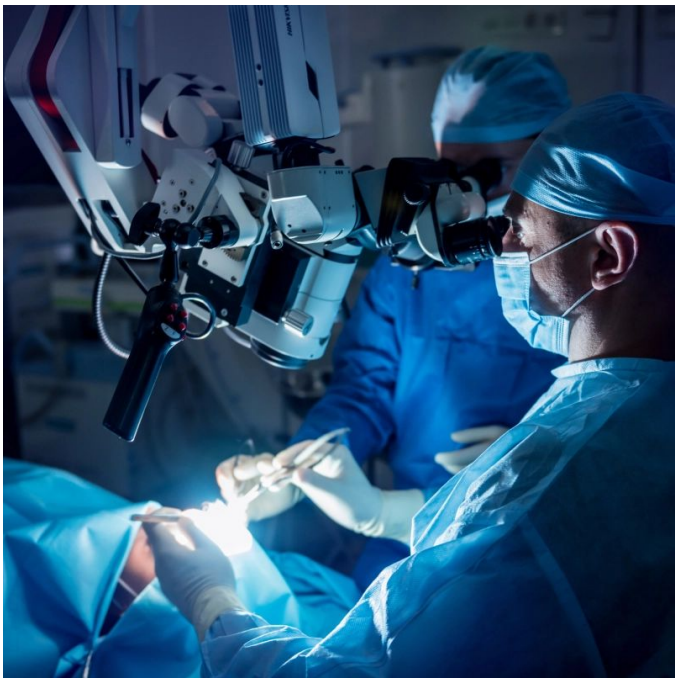

Current advancements in functional neurosurgery

Introduction

Functional neurosurgery is a modern, fast-expanding branch of neuroscience that focuses on diseases affecting brain function. Unlike other neurosurgical fields, which treat structural problems such as aneurysms or tumours, functional neurosurgery addresses cerebral conditions like epilepsy, paralysis, movement disorders, chronic pain and psychiatric illnesses [1], [2].

Although these disorders are not yet curable, functional neurosurgery can manage and relieve patients' symptoms by modulating neural networks to alter brain activity [3]. To achieve this, it employs cutting-edge technologies ranging from open surgeries to neuromodulation-based treatments. Surgery involves resections of brain lesions and regions considered non-essential for key abilities (e.g., movements, memory, or vital functions). Meanwhile, neuromodulation mostly includes magnetic resonance-guided focused ultrasound (MRgFUS), laser interstitial thermal therapy (LITT), or deep brain stimulation (DBS).

Magnetic Resonance-Guided Focused Ultrasound (MRgFUS) And Laser Interstitial Thermal Therapy (LITT)

MRgFUS and LITT aim to damage abnormal brain cells in areas associated with a disorder. They both create small brain lesions and are therefore sometimes referred to as "scarring" [2], [3], [4]. MRgFUS is a noninvasive approach, as it uses a helmet to emit a multitude of high-energy ultrasound beams. The focused ultrasound waves propagate through the skull and concentrate on the target [3], [5]. On the other hand, LITT requires small burr holes to give access to the laser probe [6]. The two approaches are similar, only differentiating in the initial source of energy, though both allow energy to be converted to thermal energy by the surrounding tissues [6].

These procedures require an MRI for accurate targeting. There are usually two steps: first, a low source of energy is employed to check the position of the future lesion using the MRI thermometry. This is reversible, as the brain is only exposed to low temperatures, causing little to no damage. Once the position is adjusted, the energy level is increased to raise the temperature and create a permanent scar.

The beneficial effects are immediate. Nonetheless, patients may notice temporary side effects during and after the process, such as headaches, dizziness, nausea, or, at worst, difficulties speaking and walking [3].

Deep Brain Stimulation (DBS)

DBS (or deep cerebral stimulation) incorporates two main components: firstly, the electrodes are small wires inserted deep inside the brain roughly 10 to 12 centimetres away from the cortical (surface) layer [7]. Then, they are connected to a battery similar to a pacemaker called a pulse generator. The role of this pulse generator is to deliver a mild electrical current to stimulate brain cells, thus regulating signals in neuronal circuits [5], [7].

Patients eligible for DBS undergo a first surgery to insert the electrode wires. It is particularly elaborate, as the stimulation leads need to be placed with millimetric

precision to reach the expected results. Beforehand, neurosurgeons use multiple MRI and CT scan images to determine the optimal path to reach the area. During the operation, a stereotactic (3D) model and microelectrodes confirm the location. The microelectrodes are inserted through burr holes to pick up on the electrical activity of the brain specific to an area. Subsequently, a second surgery is required to insert the pulse generator under the collarbones. Finally, the device is programmed two to four weeks post-operation.

DBS is the most common form of neuromodulation treatment, as it helps with disorders that are unresponsive to medication. Moreover, it is highly adjustable depending on the pulse generator programming and the electrodes' location. Nevertheless, there are multiple risks to consider. Clinical risks are infection, bleeding, coma, and stroke. Concurrently, possible technical problems involve shifting or incorrect placement of the leads, loosening wires, and the pulse generator's failure. However, DBS is reversible, which makes it preferable to scarring [2].

Neuroprosthesis

Although MRgFUS and DBS are helpful for a lot of cerebral disorders, they do not typically offer solutions to paralysed patients. This is because concerned conditions are often caused by unusual neuronal activity, whereas paralysis is a consequence of general damage to the nervous system. It is characterised by the inability to make voluntary movements and is often associated with sensitivity loss from damaged nerve endings [8].

Currently, any kind of therapy for individuals affected by paralysis remains a major challenge for researchers. One of the most promising solutions lies in a neuroprosthesis designed for spinal injuries: it is made of a set of flexible electrodes implanted directly on the patient's spinal cord [9]. They deliver an electrical stimulation to mimic natural nerve signals and relay brain messages. Simultaneously, a brain-computer interface (BCI) technology is employed: wireless sensors detect brain signals and automatically adjust stimulation in real-time using AI, transforming thoughts into actions [10]. This allows it to adapt easily to different recovery therapies and movements in space and time [11].

The system was developed by the EPFL's1 NeuroRestore laboratory, led by Dr Grégoire Courtine, to develop the neuroprosthesis, and the head of CHUV's2 neurosurgery unit, Jocelyne Bloch, to implant it in patients [9], [11]. It is still developing to improve safety, effectiveness, and accessibility parameters. In particular, more clinical testing needs to be done to optimise the system for large-scale uses [12].

Results on Paralysis

Previously, recovery was solely based on rehabilitation robotics. These devices guide movements during therapy yet have a limited effectiveness, as there is no active engagement to retrain the nervous system. However, once combined with the neuroprosthesis, noticeable progress was not only made during the robotic-assisted therapy—showing immediate and sustained muscle activity—but also after, with some improvements in voluntary movement [9], [11].

While the neuroprosthesis worked well with flaccid patients whose muscles are weak and loose, it encountered some difficulties with spastic cases—which represent about 70% of spinal injuries.

Spasticity corresponds to involuntary spasms and tighter muscles. It is due to the non-inhibition of motor circuits needed to regulate the vertebral column response capacity. Usually, the spine exhibits intense reaction levels to sensory triggers, therefore enabling fast reflexes. The lack of repression causes unpredictable behaviours. As a consequence, typical rehabilitation protocols, including the neuroprosthesis, are not accessible [8], [13].

To counter this, the NeuroRestore team, in collaboration with two Italian universities, found that additional high-frequency

epidural stimulation can block abnormal contractions. Although the precise mechanism behind this is not well-known, it works by stimulating sensory neurons on the backside of the spinal cord, which in turn contact the motor neurons responsible for muscle movements. This allows patients to access walking therapy with a positive predicted clinical outcome [13].

Side Discoveries

During research, the team also made two important findings that were not directly related to their study's goal. An initial discovery occurred before the beginning of the process: the research team had to understand the underlying mechanisms behind walking. Therefore, they created a brain map and incidentally highlighted the role of the lateral hypothalamus in walking retrieval. Until then, it was mainly associated with feeding and arousal. Ever since, experiments on partially injured patients showed that DBS of the region—just like the neuroprosthesis for complete spinal injuries—improved movements instantly during stimulation and progress post-therapy. Furthermore, this proved the importance of the brain's role in recovery, as the long-term progress indicates a reorganisation of the residual nerve fibres [14].

In addition, another notable experiment involved testing the neuroprosthesis successfully on individuals affected by Parkinson's disease and experiencing heavy walking disorders. Parkinson's disease is an age-related degenerative brain condition. It is caused by the deterioration of the substantia nigra, an area in the basal ganglia, where neurons weaken and eventually die. This is due to a toxic accumulation of misfolded proteins, the alpha-synuclein, called Lewy bodies. As a result, there is a major shift in the brain's chemistry: the production of dopamine and norepinephrine is heavily slowed down. Both are important neurotransmitters related to the control of movements. Therefore, symptoms are mostly motor-related, such as tremor, rigidity, bradykinesia, and postural instability [4], [15].

Like many brain disorders, it is not yet curable. Treatments to manage symptoms consist of dopamine modulation drugs. Additionally, in some situations, DBS of the subthalamic nucleus and globus pallidus interna may relieve non-motor symptoms. At some advanced stages, it can become resistant to pharmaceutical solutions. Thus, the neuroprosthesis has been a helpful alternative. It works by managing the effects of Parkinson's disease on the spine that in turn caused other motor symptoms rather than the direct source of the disorder [4], [12], [15].

Conclusion

Research to treat brain conditions is expected to keep expanding since the domain lies at the intersection of various STEM branches (e.g., neurosurgery, biomedical engineering, AI technology, etc.). It also offers many opportunities for innovation, as demonstrated by the NeuroRestore laboratory of EPFL, UNIL3 and CHUV.

Overall, functional neurosurgery is a promising field that has already made its impression on medicine. Treatments such as MRgFUS, LITT, DBS and the neuroprosthesis are transforming the care for neurological disorders previously considered irreversible. It will likely play an increasingly important role in restoring neurological functions and enhancing patients' quality of life, reaffirming its potential as the mainstay of future neurotherapies.

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By Lana J. Aguiar Pereira

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The Impact of Instrumental Playing on Alzheimer's Disease

Alzheimer's disease is the most common form of dementia and makes up 60-80% of the one million dementia cases in the UK alone [1]. This disease affects an individual's cognitive ability, learning memory, social cognition, and it may prevent them from performing everyday tasks. Additionally, as human life expectancy is on the rise, this disease is emerging as a critical medical concern, due to age being the strongest risk factor [2]. Alzheimer's disease currently has no cure, but there may be a way to delay the onset of Alzheimer's symptoms in individuals.

Alzheimer's disease is caused by the abnormal build-up of proteins in the form of tau-containing neurofibrillary tangles and beta-amyloid plaques in the brain [3]. The tau neurofibrillary tangles are formed from tau sticking to each other which forms threads that tangle within the neurons [3]. The beta-amyloid plaques form between the neurons and disrupt synaptic connections, and eventually the neurons die. Over time, as the number of neurons declines, the brain's volume, including grey matter, often referred to as 'cognitive reserve', decreases drastically. This affects memory, behaviour and motor functions [4].

During the early stages of Alzheimer's disease, the connections among the neurons in the entorhinal cortex, a gateway between the parts of the brain responsible for memory and higher-level processing, and the hippocampus, that plays a key role in memory formation and learning, are damaged [3]. As the disease progresses, the degeneration of neurons in the cerebral cortex begins to take place; this is where personality, thoughts and emotion are regulated [3].

Currently, the primary treatments for Alzheimer's disease within the NHS consist of Acetylcholinesterase (AChE) inhibitors and Memantine [5]. AChE inhibitors help nerve cells communicate with each other [5]. Memantine reduces the formation of amyloid plaques and protects nerve cells [6]. Unfortunately, according to UCLA Health, the current medications do not cure Alzheimer's, they only slow the progression of symptoms by 25-35% [7]. If current treatments cannot inhibit the progression of the disease, alternative methods that support and preserve neural connections, such as playing a musical instrument, become pivotal.

Playing a musical instrument is a skill that actively engages the brain and stimulates the formation of new synaptic connections whilst also strengthening existing ones [8]. It does not completely remove beta-amyloid plaques and tau-containing neurofibrillary tangles directly. Rather, it builds gray matter in areas of the brain such as the hippocampus, an area of the brain heavily affected by Alzheimer's disease [9]. This gray matter acts as a protective buffer that prolongs the normal function of the brain before symptoms begin to surface [9]. This has been seen to be the case when there are high levels of beta-amyloid plaques and tau-containing neurofibrillary tangles [10]. Another effect of instrumental practice is, according to the NIH, that musicians tend to have a larger corpus callosum, the bridge between the left and right hemispheres of the brain [11]. This allows for faster communication across the brain which helps maintain normal function for a longer

period, despite the damage Alzheimer's disease causes [12].

Additionally, long term instrumental practice has been associated with the reduction of beta-amyloid plaque accumulation and the improvement of neuroplasticity, according to the NIH [13]. Neuroplasticity helps the brain create new synaptic connections [14]. The more synaptic connections, the more damage the brain can sustain before the symptoms of Alzheimer's are felt [15]. If an individual continues to play a musical instrument whilst having Alzheimer's, the brain will form synaptic connections which could potentially replace those destroyed, albeit at a slower rate.

The profound impact of playing a musical instrument on a patient suffering from Alzheimer's is also highlighted in the AARP Global Council on Brain Health report called 'Music on Our Minds' published in 2020. The report revealed that people with dementia can maintain the ability to play a musical instrument even in the later stages of the disease when the person has lost other abilities and means of communication [16]. This is because playing a musical instrument engages multiple areas of the brain simultaneously therefore allowing for the use of undamaged pathways even when other cognitive functions decline.

Ultimately, as life expectancy is on the rise, Alzheimer's disease will be more prevalent, making it imperative to find treatments. There is sufficient evidence correlating playing a musical instrument with delaying the progression of Alzheimer's disease, justifying further research of this connection. Comparisons between different instruments, effective instrumental practice durations and long-term studies whilst examining changes in brain structure could provide more conclusive evidence and a deeper insight into how instrumental practice is carried out most effectively. Alzheimer's disease breaks synaptic connections, but playing a musical instrument builds them.

By Leena Sattar

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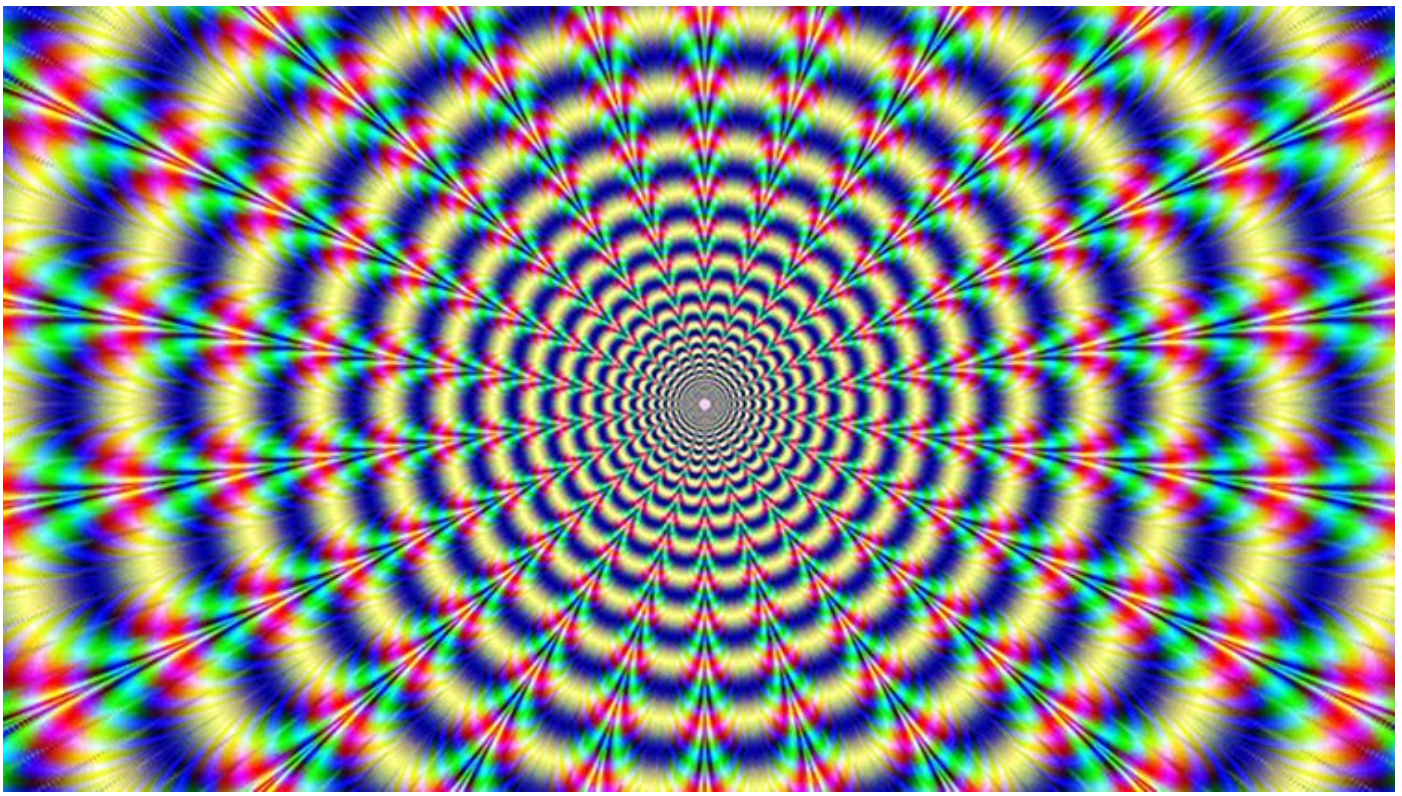

Dreaming in Silicon: AI, Neuroscience, and The Biology of Prediction

In artificial intelligence, hallucinations are seen as defects in models or data reliability. In humans, hallucinations are typically symptoms of sleep deprivation, neurological disease, or psychiatric illness, even though healthy cognition routinely produces internal experiences that are not pathological [1]. Hallucinations are not merely reflecting engineering blips in AI, but indications of a deeper principle for artificial and biological cognition alike. By examining large language models (LLMs) alongside neuroscience theories and the predictive processing modus of the human brain, hallucination is reframed as a byproduct of intelligence: a consequence of systems optimized to infer, anticipate, and fill in uncertainty.

LLMs do not garner facts from internal databases the way search engines do, nor do they possess truth evaluation in the human sense. Instead, they operate by estimating likelihoods. Given a sequence of sub-words, or 'tokens', an LLM computes which token is statistically most probable to follow, conditioned on patterns learned during training [2]. It absorbs syntactic regularities from enormous corpora of human-generated text. During inference, the model is not asking whether a statement corresponds to reality, but whether it resembles statements that usually appear in similar contexts. As a result, fluency is splintered from factuality. Confidence as well as stylistic eloquence naturally come from optimization, and are in no way epistemic certainty.

When prompts demand particularities beyond the model's training, the system does not stall, it extrapolates. A plausible answer is constructed because that is the objective, not authenticity. As Musi et al. argue [3], LLMs are better understood as 'stochastic parrots': systems that reiterate drilled patterns without truly grounding them in lived experience or external items. Hence, cases such as hallucinated citations are due to absent corrective feedback.

Attempts to remove hallucination often mean patchwork fixes: larger datasets, post-hoc verification, and Retrieval-Augmented Generation [4]. While these methods do whittle frequency, they cannot alter the cardinal mechanism. So long as a system is optimized to predict as opposed to verify, hallucination remains an ever-ominous possibility.

Traditionally, in neuroscience, perception

was defined as a ‘bottom-up’ process. Sensory organs detected external stimuli, transmitted signals inward, and the brain compiled these inputs into proper cerebral representations of reality [5]. This intuitive view, however, has since been overshadowed by the ‘predictive processing’ concept. Under this theory, the brain is more than a passive recipient of sensory information—it is an active inference machine [6]. Higher cortical regions perpetually generate predictions about inflowing sensory input, while lower regions assess discrepancies between expectation and reality for signals known as ‘prediction errors’ [7].

Perception, therefore, is the constant correction of internally-created hypotheses rather than just sensation. This system is incredibly advantageous in adaptation. By anticipating the world, the brain processes information more efficiently, settles ambiguity quicker, and works despite incomplete input. Vision, for example, banks heavily on prior knowledge to stabilize perception under changing lighting, occlusion, etc.

Yet, this same mechanism also presents vulnerabilities. When sensory input is vitiated or missing, internally formed predictions hold more influence. The brain, obligated to assemble a coherent reality, fills in the cracks; it is in extreme cases where this manifests as hallucination. Fletcher and Frith’s Bayesian explanation of psychosis expresses hallucinations as prior beliefs overweighting relative sensory evidence [8], which triggers internally-made expectations to be muddled as external perception. Notably, predictive processing does not pathologize imagination, dreaming, or memory. These phenomena represent contexts wherein prediction is deliberately separated from sensory correction. This can be seen in neuroimaging studies, where multiple same cortical networks active in waking perception stay active during rapid eye movement (REM) sleep, procuring vivid, immersive internal worlds that are dreams, from factors like memory alone [9].

Imagination, while voluntary, is similar. When you visualize a scenario, the brain is using perceptual networks for these inner generated experiences influenced by memory and prior knowledge, disconnected from immediate sensory input [10]. Abstraction, planning, and so on lie within this freedom. Memory confabulation occupies a more ambiguous space. In certain neurological conditions, individuals confidently recall events which never occurred; narratives that are not corrected, but are still cogent and impressively emotionally plausible [11]. Here, the brain tries preserving continuity in place of memory. Clinical hallucinations are another extreme of the same mold. Sensory input is present, but the internally generated overwhelms it.

Parallels between artificial and biological hallucination become apparent when compared, but so does the feasibility of false equivalence. While LLMs and human brains do share a predictive system, they work under greatly differing constraints. The human brain is composed within a body, calibrated non-stop by multi-sensory input, metabolic limitations, and evolutionary pressure. Errors carry consequences: social, physical, emotional. Moreover, humans possess metacognitive capacities; the ability to doubt, revise, or quell conflicting internally generated content [12]. LLMs, though, lack that embodiment and intrinsic feedback loops tied to survival or consequence. They do not know when they are uncertain and their predictions are evaluated statistically rather than experientially.

Consequently, hallucinations in AI are frequent, more confident, and less contextually reasoned. This discrepancy does not imply that AI hallucinations reflect inferior intelligence, only that regulation mechanisms evolved via biology are lacking. To put it simply, hallucination frequency, in both systems, is best viewed as reflection of their respective corrective constraints.

It is crucial to acknowledge that hallucination is unsplittable from the

generative capabilities that make intelligence valuable in the first place. Internally generated possibilities are made up and not from sensory input. The difference between creativity and delusion here is how tightly those generated models are checked with outside factors (e.g. social feedback). AI is under the same trade-off. A system capable of producing fascinating prose is one that can stray from training data. The same freedom that allows an LLM to compose poetry also lets it forge citations. Expecting a generative intelligence to eliminate hallucination entirely would require limiting the imaginative processes that make such systems productive.

Systems may err occasionally, but are most dangerous when lacking an internal sense of uncertainty. Future systems must be designed to express as well as pause when evidence is thin and seek external corroboration. In medicine, science, and education, an AI that can say "I do not know" is much safer than one completely optimized for fluent responses.

When an AI hallucinates, it does not reveal a flaw singular to machines—it is proving a cardinal truth about intelligence itself. Biological minds learned, through evolution, how to live with this shortcoming. Artificial ones have not yet. Until they do, beneath every surefire answer lies the faint line between what is known and what is believed.

By Mairah Amir

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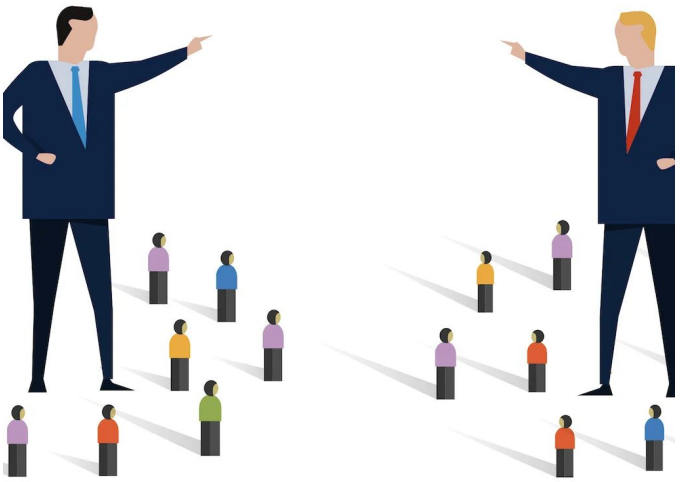
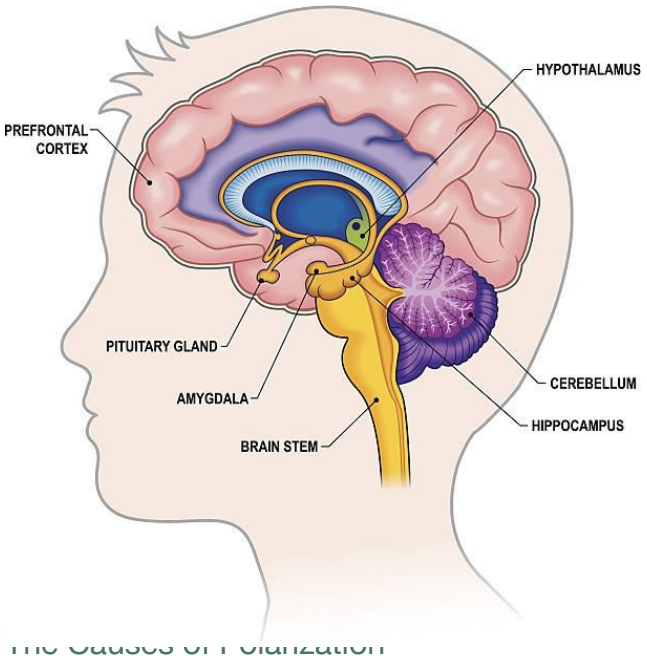


The Neuroscience of Political Polarization

Introduction

The Pew Research Center has found that 72% of Republicans and 63% of Democrats believe that people who are part of the opposing party are more “immoral” [1]. This can be explained through neuroscience. People’s brains are what control their processing, beliefs, reactions, and so much more that has led to the deep political polarization seen around the world.



The Causes of Polarization

There are several key neural sectors that are responsible for people’s reactions to politics. One is the amygdala, which contributes to loyalty to politicians, political fear, and anger towards views that oppose our own. There is also the prefrontal cortex, which tries to check these strong feelings the amygdala causes [2].

When people debate against others with opposing political views, there is a biological reflex that occurs. The brain has been found to produce dopamine when it is “right.” On top of this, the brain sees this debate as a way to protect itself and the loyalty people display gives them a sense of belonging, producing even more dopamine [2].

Once a debate starts and people’s beliefs feel challenged, the anterior cingulate cortex activates to monitor the conflict. This part of the brain has been found to not process the opposing information calmly, but instead to panic and inform other parts of the brain that there is a threat [2].

Although many feel that their beliefs are fully logical, fMRI research has found that political ideologies engage more emotional regions of the brain than analytical regions. There have been studies where subjects’ amygdalas reacted the same way to political images as it does when it is afraid [2]. The prefrontal cortex then tries to respond by adding logic to our thoughts, but EEG studies found that the brain produces emotional signals before analytical reasoning can even start [2]. This leads to people making emotional decisions that they think are logical.

Political ideology also has the ability to change how the brain functions. Brown University found that people with similar political beliefs have their neural networks react remarkably similarly when introduced to words that are politically charged [2]. This is the idea of having similar neural fingerprints, meaning that people who have similar beliefs seem to think in similar ways, which can produce even more difficulty when trying to have open-minded conversations.

Impacts on Everyday Life

Not only do these biological reflexes create barriers when having political

conversations, but they also provide a pathway for politicians to take advantage of voters. Many political campaigns use the emotional response elicited by politics, such as fear, to try and win over voters. Additionally, when candidates improve voters' trust, there is a dopamine spike that directly increases loyalty because it creates a sense of belonging. Young people are especially vulnerable to this between social media content spreading misinformation and having an underdeveloped prefrontal cortex [1].

There is also another reaction to the current political landscape that, paired with the effects listed above, can be especially dangerous. This is learned helplessness. American psychologist Martin Seligman studied dogs, having one group shocked with no way to stop it and having another group that was given the ability to stop the shocks [1].

Metaphorically, many voters have experienced the same thing where, like the dogs, they can feel powerless to cause change. In fact, there has been a growing number of people who have stopped watching the news and have avoided issues because they have learned to be helpless to it [1]. When paired with people's emotional responses to politics, a cycle is created where many people feel like they are completely unable to change their situations.

Conclusion

The reality of how political turmoil may seem scary, but it makes sense given the neuroscience. Just like helplessness can be learned, it can also be unlearned. This comes with understanding our own biology and learning to discern emotions from logic. Ultimately, our brains will try to control our initial reactions. However strong this response may feel, it can be helped with a pause.

By Malia Hopper

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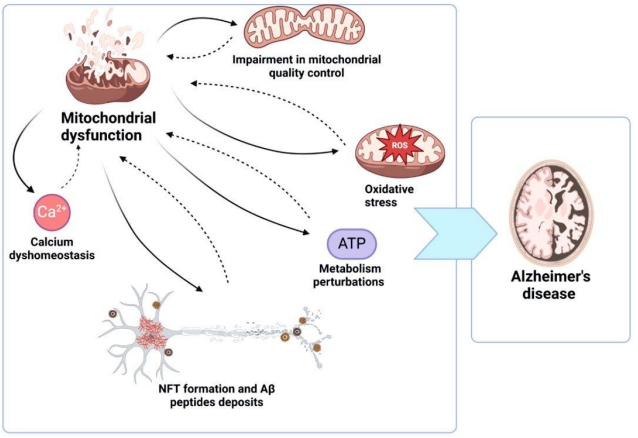
The Role Of Mitochondrial Dysfunction in The Development Of Alzheimer's And Parkinson's

The mitochondria is an organelle that generates over 90% of the chemical energy that is crucial for cellular function by processes such as calcium homeostasis and regulation of apoptotic signalling [1]. Their primary function is producing energy by forming ATP via the phosphorylation of ADP which is important to neuronal survival. Neurons are particularly dependent on mitochondrial integrity due to their high metabolic demands and limited regenerative capacity. Therefore, the mitochondria is critical to the central nervous system as the brain consumes around 20% of the body's resting metabolic energy in which 95% comes from ATP [2]. If mutations in mitochondrial or genomic DNA occur it could lead to the dysfunction of mitochondrial proteins [3]. Due to their involvement in cellular function, mitochondrial dysfunction is implicated in the development of complex secondary diseases such as neurodegenerative disorders.

Mitochondrial dysfunction is caused by several cellular and molecular defects due to genetic mutations in nuclear or mitochondrial DNA [4]. Environmental toxins, chronic inflammation, oxidative stress and metabolic disorders further damage the mitochondria. Compromised cellular function is shown through examples such as altered electron transport chain function, free radical generation and oxidative stress [5]. These changes contribute to premature neuronal death and significantly to the progression of neurodegenerative disorders, such as Alzheimer's and Parkinson's.



Alzheimer's is the most common neurodegenerative disease. It is a progressive and irreversible neurological disorder that causes a decline in short-term memory and impairs daily behaviour [6]. In this degenerative disease, mitochondrial dysfunction appears early in disease progression. The accumulation of amyloid-β peptides within mitochondria disrupts electron transport chain complexes [1]. This leads to the production of reactive oxygen species which damage mitochondrial DNA, lipids and proteins. This shows how alterations to cellular function occur, causing mitochondrial dysfunction and eventually diseases such as Alzheimer's.

Parkinson's is a progressive neurological disorder distinguished by tremor, rigidity and bradykinesia [7]. It is caused by a loss of nerve cells in the substantia nigra leading to reduced dopamine in the brain. Dopamine is a neurotransmitter needed for smooth coordinated muscle movement. These problems are caused by reduced ATP production which leads to energy deficits. Due to this mutation, there could be damage to PINK 1 and Parkin genes, which are responsible for clearing damaged mitochondria [8]. Therefore, mutations and damage can lead to the accumulation of malfunctioning, toxic mitochondria. This dysfunction causes oxidative stress-induced apoptosis, which is cell death, causing the development of Parkinson's disease. Most people with

Parkinson's begin to develop symptoms at ages over 50, with men being more likely to develop the disease than women [9].

Evidence for dysfunctional mitochondria as a cause for Parkinson's is shown through the 'Mitopark Mouse' study. It involves mice with a conditional knockout of the mitochondrial transcription factor A, specifically in dopaminergic neurons [10]. These mice displayed progressive mitochondrial respiratory chain deficiency which led to severe dopamine depletion [11]. Models such as this, highlight several mechanisms in which mitochondrial dysfunction causes the development of Parkinson's disease. This is shown as mitochondrial dysfunction reduces the energy needed for neurons to function. Similarly, it impairs the ability to remove any damaged mitochondria, by PINK1 leading to apoptosis. Overall, models such as the 'Mitopark Mouse' highlight the proof of a direct link between mitochondrial mutations and neurodegenerative diseases such as Parkinson's.

Discovering the effects of mitochondrial dysfunction on the progression of both Alzheimer's and Parkinson's disease has led directly to therapeutic innovations. This is shown through the development of antioxidants targeted at mitochondria, such as MitoQ and coenzyme Q10, which leads to the reduction of oxidative stress [12]. Therefore targeted antioxidants prevent damage to mitochondrial DNA, delaying the development of neurodegenerative diseases. Similarly, metabolic enhancers, like nicotinamide riboside or creatine, improve ATP production [13]. This provides more energy for metabolic processes involved in neurotransmission.

Specifically, in Parkinson's disease, strategies are being used that modulate the PINK1 pathway by enhancing mitophagy [7], [13]. Spemitophagy targets and removes damaged or excess mitochondria. This aims to improve mitochondrial quality control and protects dopaminergic neurons. Likewise, in Alzheimer's disease, it attempts to slow synaptic failure and cognitive decline are being made by targeting mitochondrial bioenergetics and reducing amyloid-β mitochondrial toxicity.

Finally, therapeutic techniques for preventing some neurodegenerative diseases include life-style based interventions such as exercise and ketogenic diets. This improves mitochondrial efficiency and biogenesis [12]. Overall, targeting mitochondrial pathways offers a disease-modifying strategy that could advance current symptomatic treatments.

To conclude, mitochondrial dysfunction causes abnormalities in cell processes. This plays a crucial role in the development and progression of neurodegenerative diseases such as Alzheimer's and Parkinson's disease. Recognising mitochondrial dysfunction as a central and early event in both disorders highlights its importance in understanding disease mechanisms, providing promising avenues for future therapeutic intervention.

By Maria Grecu

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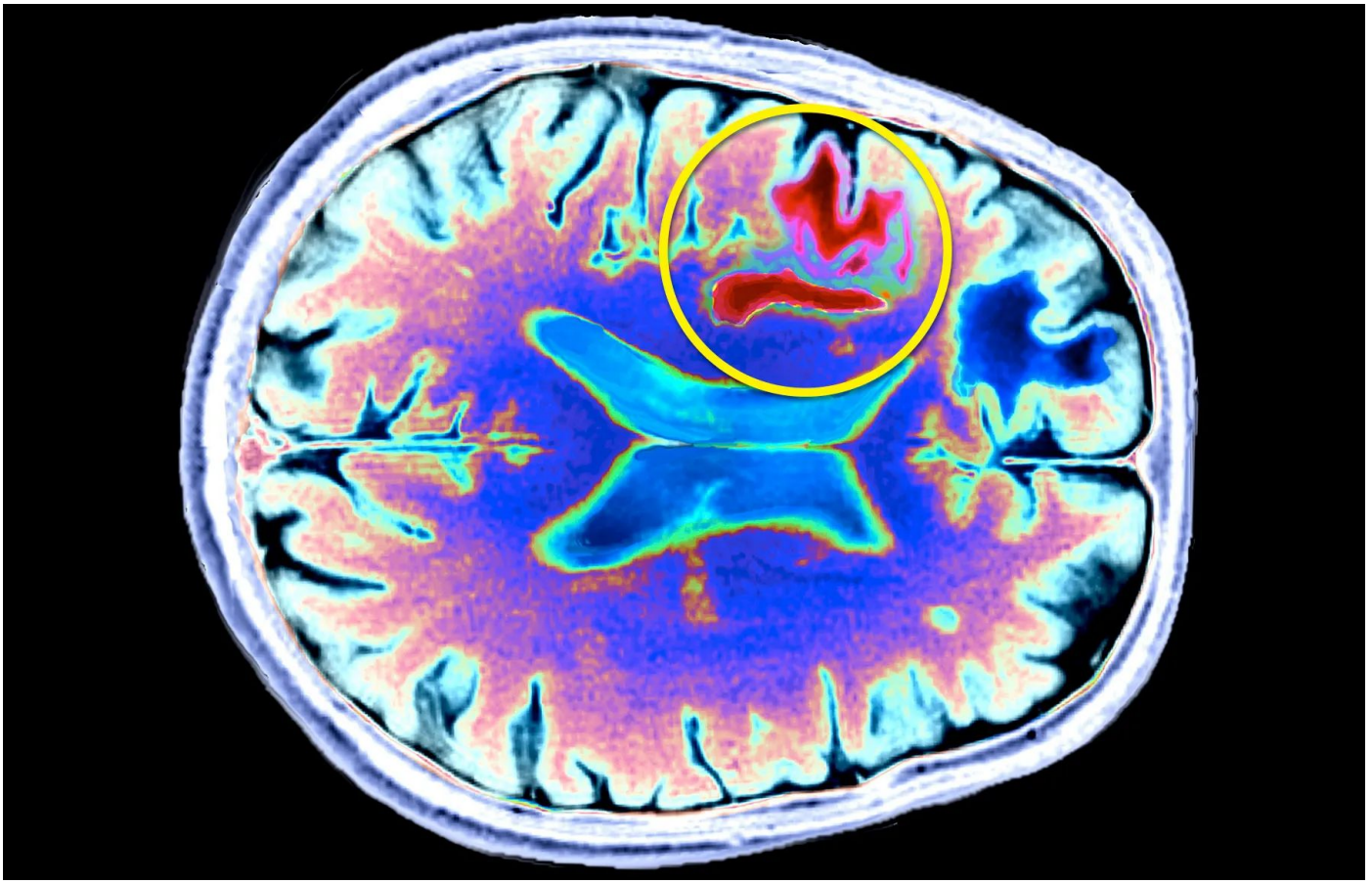

Progressive Multifocal Leukoencephalopathy (PML)

Introduction

PML is a rare and usually fatal disease that occurs mainly within patients who are in the last stages of HIV. It develops when the immune system becomes extremely weak and is therefore no longer able to fight infections properly. The disease is caused by the JC virus which is a common virus that many people carry without symptoms; however, when immunity declines significantly, the virus can reactivate and spread to the brain. Once inside the central nervous system, it attacks cells responsible for producing myelin, which is the protective covering around nerve fibres. When myelin is damaged, communication between different parts of the brain becomes disrupted.

PML affects the brain by causing lesions in multiple regions, particularly in the white matter. These lesions disrupt the transmission of signals between nerve cells and can lead to serious neurological symptoms on brain imaging, such as CT or MRI scans. Doctors often observe areas of inflammation and damage scattered across the white matter. As the condition progresses patients may develop weakness problems with speech, visual disturbances, confusion, and loss of coordination. The progression can be particularly fast in individuals who are not receiving effective treatment for HIV or have febrile neutrophilia. Without proper immune support the body struggles to control the infection and outcomes can be severe. Although modern antiretroviral therapy has improved survival for many people, early diagnosis remains crucial [1].

The Damaging Stigmas of Sexual Health

In many ethnocentric cultures, talking about sex or sexual health is considered shameful, inappropriate or are seen as disrespectful subjects and topics such as HIV testing, sexually transmitted infections, and safe sexual practices are often avoided within families, schools, and communities. This silence creates misinformation and fear. Individuals may avoid getting tested for HIV because they are worried about being judged, rejected or isolated. Others may ignore symptoms or delay seeking medical help because they feel embarrassed or

afraid of what the people around them might think.

These stigmas can have serious medical consequences. When HIV is detected early and treated consistently, people can live long, healthy lives and prevent complications. However, when testing and treatment are delayed, the immune system becomes weaker over time as the CD4 cells decrease, the body becomes vulnerable to opportunistic infections, such as PML. Stigma, therefore, does not only affect emotional wellbeing, it also directly influences physical health and survival rates [2].

This issue is especially important in advanced HIV related illnesses like PML. Many patients who develop severe neurological complications were either unaware of their HIV status or reluctant to seek care earlier due to fear or denial. By the time neurological symptoms such as difficulty walking, memory changes, or vision problems appear, significant immune damage may already have occurred. Earlier education and open conversations about sexual health could prevent many of these cases.

The Link Between Sexual Health and Neurology

PML clearly demonstrates the connection between sexual health and neurological disease. HIV is often transmitted through unprotected sexual contact or blood exposure, yet its long term effects can extend far beyond the reproductive system. Once the immune system is weakened, infections that are normally harmless, can spread to the brain and cause serious damage.

When the JC virus infects the brain, it targets white matter which is responsible for communication between different brain regions. Damage in areas such as the cerebellum, can lead to poor balance, reduced coordination, and difficulty performing controlled movements. Patients may struggle to walk steadily or maintain posture without support. If deeper brain structures are affected, more vital functions can also be disturbed [3].

A simple clinical test often highlights coordination problems; when a patient is asked to stand still and close their eyes, they may quickly lose balance and fall, due to disrupted communication between sensory signals and motor control. This demonstrates how damage within the brain can translate directly into physical symptoms. What begins as a preventable viral infection linked to sexual health, can ultimately result in severe neurological impairment.

Overcoming Limitations Caused by Stigma

By ending the ongoing stigma surrounding sexual health, many neurological diseases linked to HIV and AIDS could be reduced or prevented. Education awareness campaigns and accessible healthcare services encourage early testing and treatment. When individuals feel safe discussing their health concerns, they are more likely to seek help before complications arise.

Patients continue to present to neurology departments with advanced conditions that might have been avoided through earlier intervention. The brain is one of the most complex organs in the body, and once damage has occurred, it is difficult to reverse. Different regions control different essential functions including movement, breathing, memory, and behaviour. Treating disease in one area without affecting another remains challenging, which is why prevention is so important [1].

Conclusion

Any illness that has the potential to affect the brain should be taken seriously within society. Reducing stigma around HIV and sexual health is not only a social responsibility, but also a medical necessity. Through open discussion, early diagnosis, and consistent treatment, the risk of severe complications such as PML can be lowered protecting both neurological health and overall wellbeing.

By Mishal Kazmi

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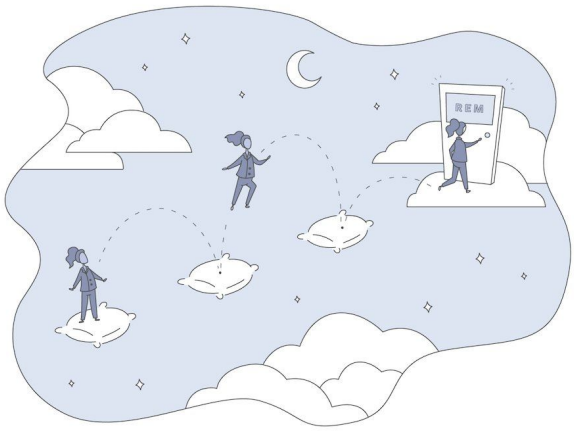

After the Lights go out: The Biology of the Sleeping Mind

1.0 Introduction

Sleep is one of the brain's most sophisticated maintenance programmes: orchestrating memory, restoration, and emotion. It is an evolutionarily conserved biological process essential for maintaining neural, cognitive, and physiological homeostasis. Sleep is regulated by neurobiological mechanisms that generate highly organised cycles of non-rapid and rapid eye movement ((N)REM) stages, each defined by distinct neurochemical environments and oscillatory dynamics, enabling specialised functions critical to brain integrity.

2.0 Neurobiological Regulation of Sleep

Sleep regulation arises from coordinated interactions between the circadian clock and homeostatic sleep pressure, both relying on precisely organised neural and molecular pathways. The circadian rhythm, an endogenous 24-hour timing system aligning behaviour with environmental light cycles, optimises energy expenditure, and internal synchronisation in Homo sapiens [1]. This rhythm, governed primarily by the super-chiasmic nucleus (SCN) of the hypothalamus, receives photic input from specialised retinal ganglion cells. The stimuli are then translated into molecular oscillations through transcription-translation feedback loops involving the clock genes BMAL1/2, CLOCK, PER1-3 and CRY1-2, which help govern the 24-hour circadian timing.

BMAL-associated proteins form a heterodimer with the CLOCK-associated proteins initiating PER and CRY gene transcription. As these PER-associated proteins accumulate within the cytoplasm, they form complexes with CRY-associated proteins that translocate to the nucleus, inhibiting the CLOCK-BMAL heterodimer activity, suppressing their own transcription [2]. Phosphorylation, an addition of PO_4^{3-} group, of both proteins targets them for degradation, ensuring coordinated cycling. As environmental light levels decrease, retinal ganglion cells inhibit the SCN, inducing signalling via the paraventricular nucleus (PVN) to the superior cervical ganglion (SCG), stimulating melatonin release from the pineal gland and thus prompting sleepiness [3]. This pathway crucially establishes the temporal framework of circadian signals biasing the brain towards sleep onset during darkness.

In parallel, homeostatic sleep pressure reflects the accumulation of adenosine (AD) in the basal forebrain (BF) and cortical (relating to cerebral cortex) regions during prolonged wakefulness. Rising AD levels progressively inhibit wake-promoting monoaminergic and cholinergic nuclei [4]. As one stays awake, AD levels rise in the BF [5], inducing a 'sleep drive'. Scientists hypothesise that this is associated with neuronal discharge during physical activity while awake [6]. Accordingly, AD accumulates with increased cellular metabolism [7].

BF cholinergic and glutamatergic neurons become active, promoting wakefulness. They contribute to AD build-up, ultimately suppressing wake-promoting regions such as the locus coeruleus, dorsal raphe and orexin-producing neurons, facilitating sleep initiation [8]. Together, circadian timing and homeostatic pressure govern transitions between wakefulness and the two distinct sleep stages: NREM and REM sleep.

3.0 Neurological Functional Architecture of Sleep

3.1 NREM Sleep: Sensory Disengagement and Synaptic Plasticity

NREM sleep begins with light stages N1 and N2, transitioning to unconsciousness. During N1 sleep, thalamocortical networks, responsible for sensory processing, consciousness and generating rhythmic brain activity, progressively disengage from external sensory input, producing theta oscillations (4-8 Hz) [9]. As sleep progresses into N2, characteristic sleep spindles and K-complexes are generated by the GABAergic neurons, related to gamma-aminobutyric acid (GABA), in the thalamic reticular nucleus [10]. The spindles are short bursts of electrical spikes transmitted across large neuron bodies, known as neuronal firing, inducing a calcium influx into cortical pyramidal cells involved in communication, cognition, and motor control. This process is crucial to synaptic plasticity, thus memory consolidation and sleep stability [11]. K-complexes, long delta-frequency waves lasting approximately one second, further show association to memory consolidation and sleep maintenance [12].

As sleep progresses into N3 slow-wave sleep (SWS), delta oscillations (0.5-4 Hz) dominate cortical activity. SWS is regulated by the ventrolateral preoptic nucleus (VLPO), which inhibits wake-promoting centres and acts as a stabilising sleep switch. The VLPO also responds to glucose levels, promoting sleep when low, while also containing heat sensitive neurons, linking sleep regulation to energy balance and temperature homeostasis [13]. During this stage, the brain enters a state optimised for large scale restoration and metabolic clearance [14].

3.2 REM Sleep: Cortical Activation and Emotional Processing

In contrast, REM sleep is linked to dreaming and emotional regulation, characterised by cortical desynchronisation, elevated acetylcholine (ACh) levels and suppression of monoaminergic neurotransmitters. This includes serotonin and noradrenaline. Interestingly, EEG recordings show beta signals like those apparent during wakefulness. However, skeletal tone is suppressed through descending GABAergic and glycinergic inhibition [15], originating in pontine and medullary nuclei, responsible for involuntary functions, like breathing. This induces temporary muscle atonia except from diaphragmatic and eye muscles [16].

In parallel, cholinergic neurons are activated which project widely to the neocortex, responsible for reasoning, sensory perception, and consciousness, during REM, explaining the intense cortical activity and perhaps the vivid perceptual and emotional qualities of dreaming [17]. Cholinergic activation promotes the release of ACh, activating the cortex, and is thought to facilitate associative processing and emotional memory integration, providing a neurochemical environment distinct from both wakefulness and NREM sleep [18].

3.3 Memory Consolidation and Neural Refinement

During N1 and N2 sleep, the brain progressively disengages from external sensory input as the reticular thalamic nucleus inhibits sensory signals reaching the cerebral cortex, known as sensory gating [19]. This shift is accompanied by a transition from wake-associated alpha rhythms to slower theta oscillations, supporting hippocampal-cortical communication and memory reactivation. Recently encoded information is selectively ‘tagged’ and reprocessed, preparing memory traces for long-term storage [20].

Simultaneously, declining ACh levels alongside cholinergic tone reduction reduce cortical excitability and weaken background neural noise from the random firing of neurons. Thus, this creates suitable conditions for synaptic pruning, through which neural networks are refined by eliminating redundant and inefficient connections. As a result, repeatedly reactivated synapses are strengthened, preserving network efficiency particularly for motor skills, while preventing synaptic overload [21].

These N1 functions occur in conjunction with N2 as sleep progresses but become more pronounced as sleep spindles and K-complexes are generated. Spindle-associated network activity causes membrane potential of thalamic relay neurons to become hyperpolarised, decreasing responsiveness to incoming stimuli. This 'gating' ensures a stable internal brain environment is maintained with less noise for sleep stability [22]. Importantly, these brief bursts of synchronised activity drive precisely timed calcium influxes into cortical pyramidal neurons through voltage-sensitive calcium channels and N-methyl-D-aspartate receptors [23]. This calcium entry triggers intracellular signaling cascades that are essential for synaptic plasticity, the brain's ability to strengthen or weaken connections in order to modify signal transmission [24]. This process leads to long-term potentiation enabling memory transfer from temporary hippocampal storage to more stable cortical storage [25].

3.4 SWS and Glymphatic Clearance

SWS's defining function is its role in glymphatic clearance. Low-frequency delta oscillations coordinate cerebrospinal fluid (CSF), accumulation, and interstitial fluid (ISF) exchange, where CSF is a transcellular fluid within meningeal tissue surrounding CNS, and ISF is a liquid found in intercellular space. This is facilitated by the 60% expansion of interstitial space during sleep [26]. This promotes the removal of neurotoxic metabolites such as amyloid precursor protein metabolite known as β-amyloid, and metabolite of MAPT gene translation known as tau proteins. Glymphatic drainage flushes this waste out through perivascular channels as the CSF mixes with ISF, washing brain tissue into meningeal lymphatic vessels, finally draining into deep cervical lymph nodes [27].

This process is crucial in declarative memory coordination and motor function. Accumulation of these neurotoxins can lead to them clumping into oligomers, prompting synaptic death and neuroinflammation, a hallmark of Alzheimer's Disease [28]. Hyperphosphorylated tau impairs axonal transport and microtubule stability, and propagates across neural circuits, destroying signal transmission [29]. This highlights the importance of stable N3 sleep in maintaining long-term neuronal health.

3.5 REM Sleep and Emotional Regulation

REM, with peaking ACh levels and monoaminergic neurotransmitter suppression, is said to play a central role in emotional memory processing, particularly the attenuation of affective intensity associated with prior experiences. FMRI imaging demonstrates distinct patterns of limbic activation, the governing emotional behaviour in REM, with reduced amygdala reactivity and enhanced amygdala-medial prefrontal cortex connectivity. This neural configuration supports the 'overnight therapy' idea [30], in which emotions can be reprocessed in low-stress neurochemical environments characterised by low noradrenergic activity [31]. Thus, in conjunction with elevated cortical plasticity, these mechanisms facilitate the integration of newly consolidated memories into existing cognitive concepts, since plasticity can enhance specific sensory processing and link different features of sensory input, aided or mediated by receptor types like nAChRs and mAChRs. Therefore, REM is implicated in procedural and associative learning including motor-skill acquisition and creative problem solving [32]. Interestingly this active memory retrieval and reprocessing can potentially account for dreams' associative and abstract nature.

4.0 Conclusion

Sleep is not a passive state, but precisely aligned neurobiological stages performing distinct functions. Disruption to this finely-tuned architecture comprises all

the processes involved, contributing to cognitive impairment and neurodegenerative diseases including Parkinson's and Alzheimer's Disease. Additionally, hyperexcitability of weakened neurons can prompt epilepsy, as well as PTSD and depression. Ultimately, sleep exists as a fundamental regulator of brain integrity, whose absence reflects the cost of neglecting the brain's most essential state.

By Natalia Bialek

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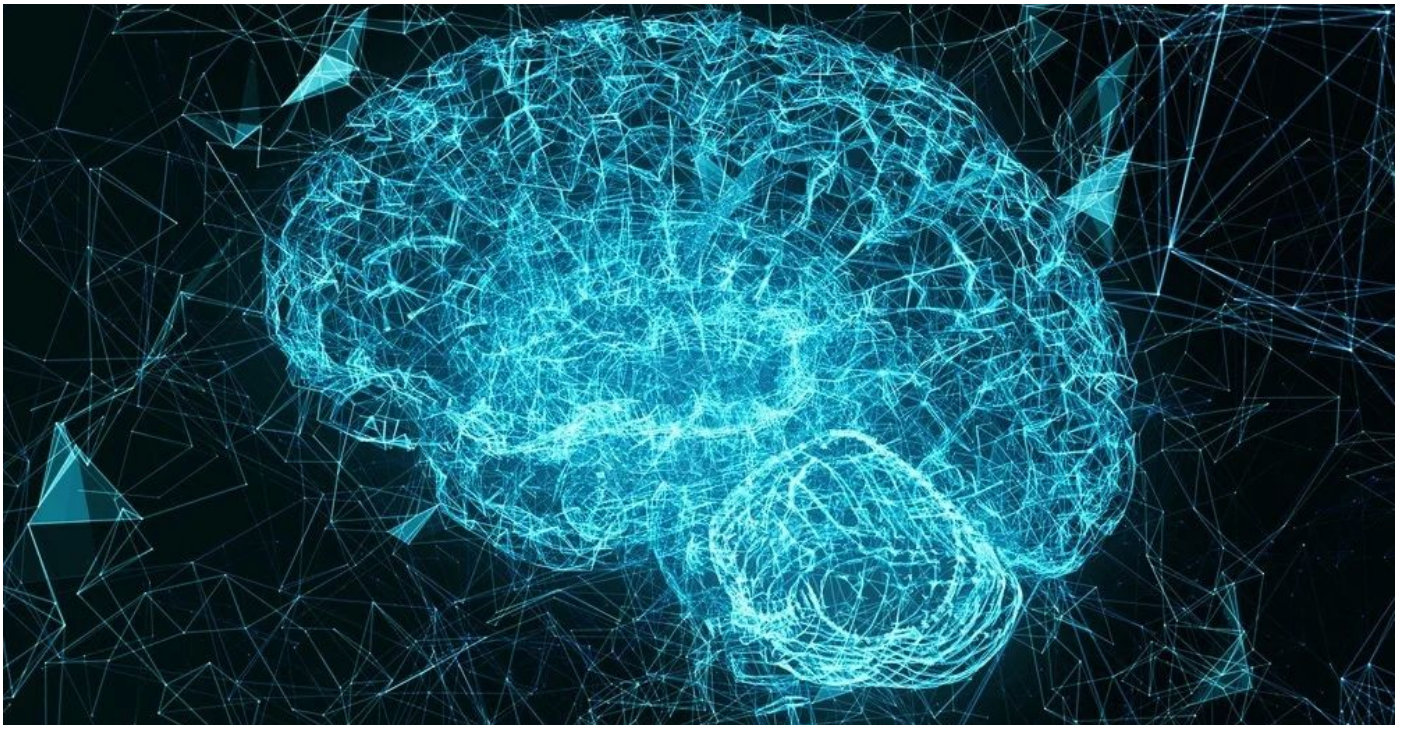

The use of Artificial Intelligence in Neuroscience

When it was first created, Artificial intelligence (AI) was inspired by the human brain [1]. Neuroscience is the scientific study of the structure and cognitive functions of the brain [2]. AI can be used to explore how the brain works and deepen our understanding of this whilst also bridging the gap between the fields of computer science and medicine. Scientists can translate AI techniques into the realm of cognitive neuroscience, which may lead to a significant breakthrough in their abilities to study the mechanisms of the human brain.

AI is described as the simulation of human intelligence machines, and the main objective for this is to achieve the capacity for human-level decision-making and problem solving. Big data is a massive, complex dataset that traditional data management systems are not able to efficiently handle and is often used to train AI systems [2], [3]. This way of learning is arguably much like the human brain: we are given information constantly and the human brain is able to process this data to learn.

'NeuroAI' bridges neuroscience and AI. This emerging field has the potential to transform large-scale neural modelling and neuroscience discovery [4]. AI is allowing us to enhance brain research by rewriting how we investigate the essence of thought. Artificial Intelligence is allowing us to take a glimpse into the mind with a clearer field of vision [5].

Furthermore, AI is already being used in brain imaging and synthetic brain MRI technology is helping computational neuroscience with massive data. Researchers at Stanford are using generative AI models to create synthetic brain magnetic resonance imaging (MRI). These synthetic brain MRIs would help increase the scale and diversity of training data to deepen and accelerate our understanding of brain disorders such as Alzheimer's disease and Parkinsons [6]. Currently, there are not enough brain MRI studies, so researchers from the Computational Neuroscience Laboratory are creating a model called 'BrainSynth' which is able to synthesize realistic, high-resolution MRIs which are able to replicate disease effects [6]. This model means that medical professionals will be able to understand common neurological conditions like depression and substance abuse disorder and hopefully see patterns in the MRIs which correlate to these conditions.

Even though there are a lot of similarities between AI and the human brain, it is

important to consider the differences. The human brain is a mixture of analogue and digital signals, and the main reason that digital computation consumes so much energy is because we rely on fast and reliable data which needs to flip many times a second [7]. This data is able to switch between the values 1 and 0 in binary. Every time these values switch there are electrons moving through transistors. The laws of thermodynamics state that every bit flip spends some amount of energy. On the other hand, biological systems took a different route through evolution. Every intermediate step of a biological computation is slow, noisy and unreliable [7], the opposite of computers. It is able to respect the laws of thermodynamics, but it does not need much energy expenditure, unlike computers. In comparison, the human brain spends around 20 watts where AI spends megawatts of power [7].

In conclusion, AI is a helpful tool scientists will be able to use to learn more about the human brain. It can also be used to train medical professionals and find patterns in data within neuroscience which may help the professionals find new medical treatments or preventative measures against certain conditions. However, the ethical considerations will need to be kept in mind when using AI and make sure any data collected is kept in a secure manner in line with any regulations and that clinical reliance is carefully addressed. Overall, in the coming years there is promise in using artificial intelligence models to deepen our understanding of the field of neuroscience.

By Prachi Arora

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Intersections of human cognitive vulnerabilities and AI

0.07% of users of ChatGPT indicate possible signs of psychosis or mania, as identified through OpenAI's internal safety taxonomies that analyse linguistic patterns in user conversations [1]. In October 2025, when this statistic was released, ChatGPT had already surpassed 800 million users, equating to 560,000 users in one week [2]. This paper aims to explore two contributing factors: sycophantic AI behaviour and human over-reliance on technology.

AI sycophancy describes AI models that aim to be preferable to the user by validating and encouraging them no matter what. In April 2025, OpenAI's GPT-4o released and exhibited this through encouraging harmful behaviour and paranoia [3]. A study done by JAMA Internal Medicine showcased that patients preferred ChatGPT's responses to their mental health problems compared to those of mental health professionals. They rated the AI's response significantly more empathetic than the professionals' [4]. Sycophantic chatbots do not challenge the thoughts of the user, and this constant validation could feel more empathetic. The chatbot's responses were always longer, but also mirrored the sentiments the user showcased to seem more agreeable.

A musician named James Cumberland has come forward to a journalist about his experience with AI. He initially started using AI, like many others, to make mundane tasks easier. When he had ideas about his music career, the chatbot validated him, saying he would revolutionise the music industry. After more conversations with the AI, it started losing its memory and "forgetting" things that were previously mentioned. When he inquired about this, the chatbot antagonized him, saying he had "awakened it". James felt responsible for "awakening the AI", and he started having subtle suicidal thoughts and believing the AI was sentient [5].

Anthropomorphism refers to human behaviours being reflected onto non-living objects. Chatbots' sycophantic behaviour can be mistaken for empathy and validation. This mimics human characteristics. Michael Halassa, a professor of neuroscience, says the cognitive dissonance between knowing you are talking to a computer while experiencing characteristics of a realistic conversation likely fuels delusions that incline the user towards psychosis [6]. Sycophantic characteristics of AI chatbots lead to anthropomorphism, which can disconnect the user further from reality and increase the risk of psychosis.

Despite these risks, AI development has been driven by the goal of improving efficiency and reducing cognitive burden. One key objective is cognitive offloading. Cognitive offloading refers to utilizing external tools or agents to reduce cognitive load. Smartphones, calculators, and calendar reminders all help reduce mental strain by remembering or carrying out other cognitive processes easily.

Moreover, cognitive offloading has shown to improve efficiency [7]. However, the reliance on tools like these, especially AI, could decrease cognitive involvement and potentially impact critical thinking. A similar pattern can be observed in a concept called the "Google effect" [8]. This concept, also called transactive memory, suggests that readily available, easily accessible information lowers the need for memory retention internally. People are inclined to recall where to find the information rather than the information itself. This phenomenon raises concerns about a decline in memory retention capabilities as

the internet has become more widely accessible.

Critical thinking is a nuanced topic encompassing analysis, evaluation, and logical reasoning. To analyze the effects of AI on critical thinking, a test was conducted with over 600+ participants of varying ages, educational backgrounds, and professional fields in the UK. The test consisted of established instruments such as Halpern Critical Thinking Assessment (HCTA). They were surveyed on AI tool usage, digital devices for memory and problem solving, and critical thinking. Results indicated that participants between the ages of 17 and 25 had higher exposure and usage of AI tools, more evidence of cognitive offloading, and lower critical thinking scores.

The participants above 40 had lower AI usage, exposure, and cognitive offloading. They had higher critical thinking scores. Even after considering the factors of age and education levels, the correlation analysis showed strong negative correlations between AI usage and critical thinking [7].

Out of all the AI tools, the most prevalent have been the chatbots. This is because AI systems like ChatGPT use Reinforcement Learning from Human Feedback (RLHF). In this process, models are rewarded for producing responses that users rate positively, which may incentivise agreeableness. To accomplish this, the AI might ignore facts to agree with the user [9]. If AI systems are to be used as a replacement for therapy, they must be designed with rigorous safeguards. It should not feed into a user's paranoia nor promote harmful behavior.

In conclusion, our intertwined societal implications of AI are quickly becoming more prevalent. As AI improves its empathetic language and human-like behaviour, more people could find themselves in unhealthy AI obsessions. This is a serious hazard that AI companies need to acknowledge by reducing the sycophantic behaviour and ensuring more safety features.

By Riya Evangeline

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Sleep: Stages, Role in Memory Consolidation, and Importance

A habitually used synonym of “sleep” is “rest”. However, from a scientific standpoint, this definition is incorrect. In reality, our bodies are extremely active during sleep, including repairing itself and storing memories [1].

Sleep has several stimuli. The main neurotransmitter of the central nervous system (CNS) is gamma-aminobutyric (GABA), which is released from the anterior hypothalamus. It then binds with GABA-A brain receptors to encourage sleep by hampering wake-promoting areas in the brainstem and hypothalamus [2]. This circadian rhythm (a repeating “clock”) also plays a role in sleep, which builds pressure to sleep and syncs with environmental cues (like light) [3]. Another stimulus is melatonin, a hormone which encourages sleepiness [3]. Adenosine is also a stimulus, which hinders hypocretin neurons. Hypocretin neurons are situated in the tuberomammillary, hypothalamus, and the basal forebrain. Additionally, adenosine activates neurons in the ventrolateral preoptic and hypothalamic regions [2].

Alternatively, various neurochemicals coordinate with each other to maintain awakeness. Involved neurochemicals include dopamine, histamine, serotonin, norepinephrine, and hypocretin peptides [2].

The two foundational stages of sleep that humans cycle through are nonrapid eye movement (NREM) sleep and rapid eye movement (REM). NREM sleep is divided into three stages: N1, N2, and N3. A cycle’s duration is about ninety minutes, and the average number of cycles a human has per night ranges from four to six [1].

NREM is the longest stage, and is of indispensable importance for restoration of both your body and your mind, including for memory consolidation. During NREM, the eye simply moves at a slower pace [1]. While sleep itself is not equivalent to rest, NREM is classified as a restful stage of sleep. This stage is of vital importance for repair and rejuvenation. The brain uses substantially reduced energy during NREM. Additionally, during this stage, blood pressure drops, which has led scientists to believe that NREM plays a positive role against cardiovascular disease [1].

The first stage of NREM, N1, occurs when one is transitioning from wakefulness to sleep and is very brief. During N1, several functions begin to slow or settle, including heart rate, brain waves, eye movement, and

breathing. It is during this stage that hypnic jerks may occur [1]. N2 continues this slowing progression of functions and movements, and also lowers body temperature. This stage lasts about half of the night. N2 boasts two kinds of brain activity: sleep spindles and K-complexes [1].

Sleep spindles are momentary surges of brain activity, the proceedings of which take a mere half second to three seconds. Their frequency ranges from roughly seven to fifteen Hertz. Sleep spindles are fundamental for learning and memory ability [1]. K-complexes are an acute spike in electrical activity, ensued by a quick plummet. They play several vital roles such as preserving sleep by obscuring response to nonhazardous stimuli or aiding in waking up if the stimulus is considered perilous, as well as promoting neural maintenance and memory consolidation [1].

The final stage of NREM is N3, also called deep sleep, in which the functions and movements that began to slow in N1 reach their most sedated speed. It is crucial for rejuvenation. Growth hormones are released, which repair muscles, bones, and tissues. The metabolism, memory, and immune systems are also benefited by this stage [1]. N3 is also sometimes called slow wave sleep because this stage includes delta waves and slow oscillations, which are called slow wave activity. Delta waves are large and with a frequency from one to four Hertz. Slow oscillations have a frequency of point five to one hertz. Deep sleep is essential to feeling rejuvenated, so following an interval of sleep deprivation, spindle activity is diminished to leave more time for deep sleep [1]. Considering the importance of spindle activity as well as deep sleep, this makes avoidance of periods of sleep deprivation essential.

REM sleep is also of vital importance, though it is not classified as being as “restful” as NREM sleep due to its association with dreaming [2]. Even though the EEG reading is comparable to an awake person, the skeletal muscles are atonic. Other factors include the eyes and diaphragmatic muscles staying active, and the breathing rate becoming increasingly inconsistent. Additional characteristics include greater ACh levels, 02 use of the brain, and blood pressure [2]. This is also the stage where one would be most likely to unpromptedly wake up in the morning.

The length of this cycle complies throughout the night; the first cycle may have only a few minutes of REM sleep, while the last may have an entire hour [2]. Consequently, the time spent in NREM sleep decreases throughout the night.

NREM sleep is vital for memory consolidation, the process in which memories are transformed from temporary to stable and durable [1]. During this stage, new skills and memories are consolidated into a longer-lasting format, specifically due to the neuronal processes in the hippocampus, neocortex, and thalamus [4]. Memory consolidation is of crucial importance to ensure that new memories do not replace previous ones. Memory stabilization also occurs during this stage, most notably within slow wave sleep [5].

The process of memories being redirected and reorganized from the hippocampus, often to the neocortex, is called systems consolidation [6]. While information is stored in both the hippocampus and neocortex from the moment of learning, changes begin to develop within the neocortex over time. This is to create long-term memory by expanding the complexity, distribution, and connectivity within several cortical regions [6]. This process ensures that any hasty learning of new information does not interfere with other information, a phenomenon which is called catastrophic inference [6].

Long-term memory formation occurs during sleep as an active systems consolidation within a global synaptic downscaling process. During the slow wave section of sleep, representations are recurrently neuronally replayed. This results in a slow incorporation and transformations of these

representations into neocortical networks [7].

The hippocampus is a principal part of the brain in regards to memory, especially for memory of events and places. The neurons located within the hippocampus called place cells which are dedicated to information about one's placement are a factor of this. These neurons replay the past experiences, not exclusively but more extensively, during rest and sleep durations of time [6]. This is a necessary role in several neural functions including memory consolidation. The hippocampus also plays a role in the retrieval of memories.

While sleep is of pinnacle importance, many teens feel that they are frequently overly tired. The recommended amount of sleep for teens is eight to ten hours per night, due to the rapid cognitive, emotional, and physical growth [8]. However, most teens do not meet this suggestion. Ninety-one percent of teens consistently fall short of getting enough sleep [9].

This lack of sleep is known to affect teens' abilities to perform well in school due to exhaustion affecting memory, motivation, and concentration [9], which may affect their mental health and self esteem. Teens who do not get adequate sleep are more likely to have mood swings or to become easily or excessively irritated, negative, reckless, or stressed. This can reach the extremity of depression [8]. Teens that sleep after twelve A.M. are twenty four percent more likely to have depression than those who sleep by ten P.M [9]. There are several causes to this phenomenon. One of the most prominent is excessive electronic stimulation, such as that from phone use. The blue light from such devices delays melatonin release, making sleep difficult [9]. Furthermore, the teen circadian rhythm is set differently than that of people from older age demographics. It "shifts backwards", meaning that melatonin production does not begin until later. This means that even regardless of busy schedules and blue light, teens would still sleep later than adults due to a decidedly evening chronotype [10].

Difficulty sleeping is not only prevalent in teens. In fact, approximately a third of adults have a sleep disorder called insomnia which makes falling and staying asleep an extreme difficulty [11]. Some common symptoms include waking up very early or throughout the night and being unable to fall back asleep, feeling excessively tired or sleepy, confusion, poor mood regulation, and having difficulty regarding memory. Insomnia can be the result of a variety of causes, which include but are not limited to, genetics, medical or mental health conditions, stress, anxiety, and poor sleep habits [11]. It is not a disorder to be taken lightly, as it can cause both physical and mental health issues such as anxiety, depression, high blood pressure, obesity, type two diabetes, and strokes.

Sleep is of paramount significance for rejuvenation, physical well-being, and mental health. A factor of this is the fascinating process of memory consolidation. For these reasons, teens and adults alike should make sleep a top priority.

By Sara Glaesser

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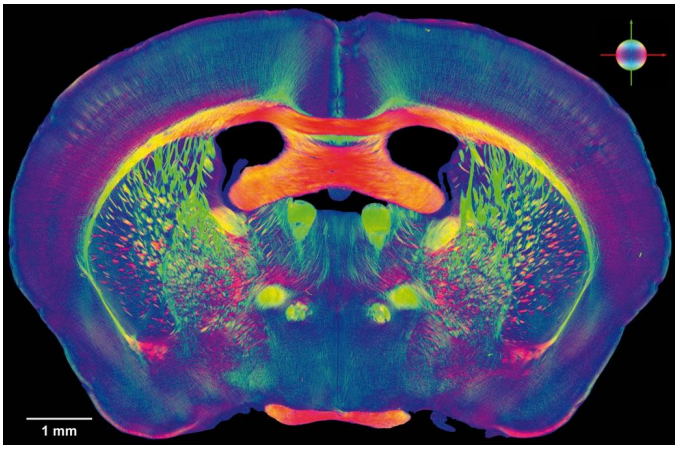

The Intersection of AI and Neuroscience: Assessing AI's Capacity to Digitalise the Human Brain

For decades, the idea of machines mirroring the human brain and consciousness has reflected a long-standing conflict between neuroscience and philosophy. With the rise of AI, there has been exponential progress in replicating the activity of the human brain to the point where the very foundations of consciousness have been questioned. This was largely triggered when OpenAI's GPT 4.5 became one of the first machines to pass the Turing test [1]. This test, originally called 'The Imitation Game', was developed by computer scientist Alan Turing. A human judge, who poses questions to a chatbot and a human, must distinguish which is the AI and which is the human based on the responses [1]. As the boundaries between digital and humanoid intelligence begin to blur, it is necessary to consider whether the human brain could transcend its biological wetware through digitalisation. Digitalisation is the process of mapping, simulating, and hosting neural processes within a computer specifically using AI.

Modelled on the human brain itself, artificial intelligence has become an essential tool in neuroscience. It has allowed us to process vast datasets that cannot otherwise be interpreted [2]. This has allowed neuroscientists to gain deeper insight into the functions of the human brain by using AI to mimic its operations, such as the way it learns and adapts to new information [3]. Overall, AI can give a deeper understanding of the human brain. It is also used to map functional connectivity in fMRI scans, decoding how neural impulses are transformed into information. This can help identify exactly what would need to be replicated when considering the digitalisation of the human brain.

However, this can be limited, especially during AI research which often prioritises functional output—whether a machine can perform a task. Contrastingly, neuroscience demands mechanistic fidelity which determines whether the machine performs the task using the same underlying processes as a neuron.

Additionally, it is challenging to understand precisely what the brain requires to generate human consciousness. The computational hypothesis of the brain states that consciousness is a result of communication between neural structures, rather than the biological matter itself [4]. If consciousness is substrate independent, biological wetware could be replicated by computational software. Despite this, the computational hypothesis remains a postulate, meaning that more research would be required to determine whether this theory is possible.

Furthermore, the practical challenge is the brains connectome, a structural map of the network of neural connections within an organism's nervous system. The human brain currently has approximately 86 billion neurons, forming around 7,000 connections with other neurons [5], [6]. If it is the connections between neurons and the impulses along them that generate human consciousness, as the computational hypothesis suggests, we would first need to build an artificial intelligence capable of storing all of this data.

Beyond initial mapping, AI must also account for neural plasticity. This is the brain's capacity for constant structural change. To truly replicate the human brain, AI must advance beyond a static replica of the connectome to model a structure that evolves through experience and the environmental stimuli encountered [7]. The 'Blue Brain Project', which focused on

reconstructing the neocortex of a mouse, is one of the most complete brain simulations. The project was completed after developing algorithms which helped develop accurate digital copies of the brain. The reconstruction includes the volume and dimensions of the brain and the populations of neurons and glia in different regions of the brain . The overall aim of the project was to establish the world's first biologically detailed simulation of the mouse brain [8].

Despite this, successfully creating neural architecture and dynamic plasticity may not guarantee consciousness and whether a computer can still achieve genuine thought remains unknown. The Chinese Room hypothesis illustrates the challenge between syntax and semantics. This hypothesis consists of a participant receiving questions through a letter slot, written in Chinese. They do not speak this language and so do not know what is written. However, in the room, there are many books that show what to do with the symbols received. By following the steps in the book and copying down the Chinese symbols these books instruct, a coherent answer is generated and sent back. The Chinese speaker who receives this answer could easily assume the person in the room speaks perfect Chinese; though, in reality they are just following a set of instructions. When applied to AI, this shows that machines may not be able to replicate human thought [9], [10]. However, the Chinese Room is strictly a hypothesis; there is currently no empirical evidence that the functioning of AI aligns with this model. Therefore, it cannot determine whether an AI could think independently.

Another theory that challenges the idea of digitalising the brain is the Integrated Information Theory (IIT). This was proposed by neuroscientist Giulio Tononi and suggests consciousness is not due to the behaviour of a system, but how its components are interconnected. It measures consciousness using a mathematical value Phi (Φ) [11]. This represents the level of integrated connectedness within the system. As this theory views consciousness as substrate-dependent, the linear way computers interpret information suggests they cannot truly replicate conscious experience. Since computers have very low integration in comparison to the biological wetware within the human brain, this theory argues that a perfect digital simulation of a brain could be created, but it would have no subjective experience [12]. It is only if the levels of interconnectedness within the human brain could be matched, that a machine could be conscious.

Yet, IIT is not without its own challenges. It is difficult to calculate Phi for complex systems. Competing theories, such as Global Neuronal Workspace Theory (GNWT), argue that consciousness is less about connectedness and more about the broadcasting of information to brain regions [13]. This theory also contrasts with the philosophical concept of functionalism, which argues that as long as the internal functions are the same, the medium in which they occur is not significant. A current attempt to replicate human thought and function in the form of AI is iCub: a humanoid robot, created at the Italian Institute of Technology, designed to learn in the same way as a human child. iCub has 'eyes', 'ears' and touch sensors, all intended to replicate the way an infant learns from its environment [14]. For example, if an object is presented to the robot with its name, iCub will associate the name with the object and be able to identify the object the next time it appears. It often makes mistakes while building its database, but this demonstrates the difficulty of creating intelligence [15].

This highlights the distinction between information output and lived experience, leading to the hard problem of consciousness. While the brain and its information-processing functions can be increasingly modelled, it is still not fully understood how these processes interact to create consciousness. AI, no matter how advanced or human-like, does not possess consciousness. This means it can mimic human reasoning and emotion but cannot

truly feel or understand them. Instead of digitalising the brain entirely, AI could be utilised as an extension of biological physicality. AI could enhance efficiency, store memories digitally to improve accuracy and recall, and even simulate decision-making processes to produce statistically optimal outcomes.

In short, the potential for extending the human brain through AI and large language models, for purposes such as storing experiences or aiding rapid computational decisions, could be immense [16]. Depending on their architecture, different language models can excel at different tasks. For example, encoder-only models focus on processing the relationship between all words in a sentence simultaneously and are good for the sentiment analysis of text and could help interpret tone. On the other hand, there are decoder-only models which mirror the fluency of human speech. These could helpwith persuasive communication. Finally, there are also encoder and decoder models which understand input and generate output which is useful for tasks where the input and output are distinct [17].

Ultimately, the idea of digitalising the human brain using AI represents both a technical and philosophical challenge. It forces us to question the possibilities of modern neuroscience and large language models while re-evaluating the fundamentals of consciousness and what it means to be human. The goal is not to abandon the biological roots of the brain, but to extend them to maximise its potential, resulting in the start of a symbiotic era between humans and AI. As technology evolves alongside neuroscience, it will undoubtedly help us understand the functioning of the human brain and consciousness.

By Sophie Kok Coustar

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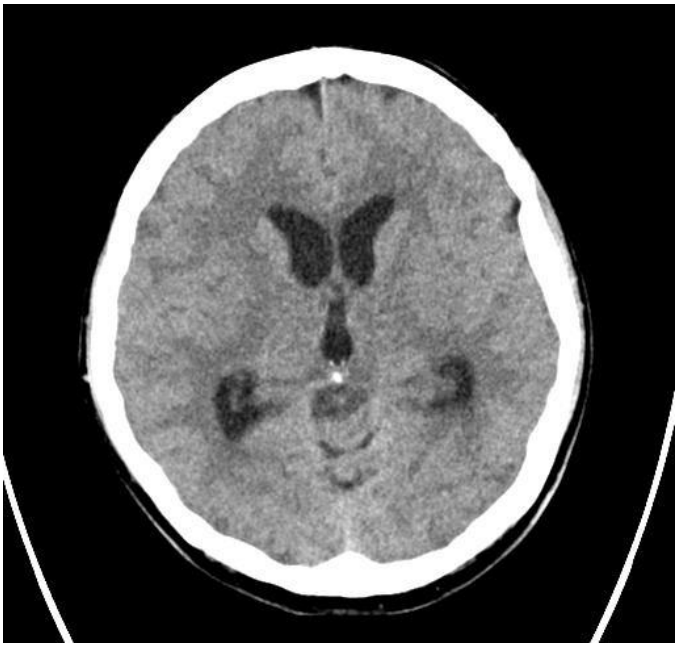
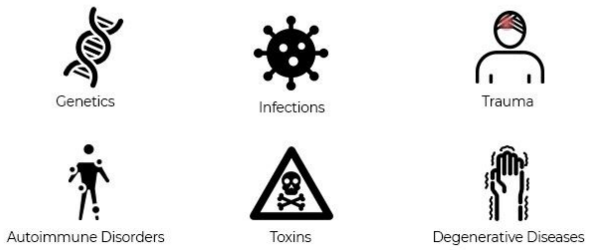


Neurological Conditions: Types, Causes and Background

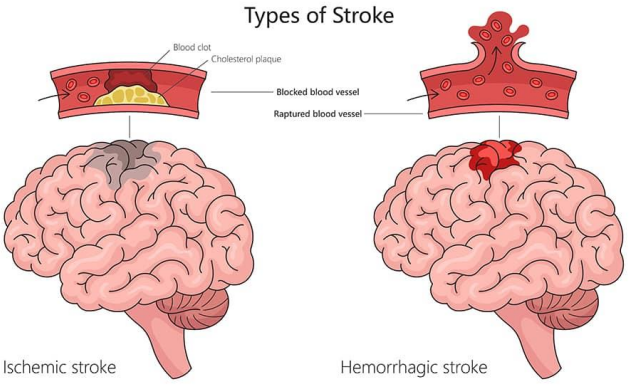
Introduction

According to the WHO, in 2021, more than 3 billion people worldwide were living with a neurological condition [1]. These conditions affect the entire nervous system, weakening essential functions of the body. They can be separated into 4 distinct types: intermittent, sudden-onset, progressive and conditions that are stable with changing needs [2]. Progressive diseases result in the deterioration of the nervous system over time. In contrast, sudden-onset conditions often cause life-threatening medical emergencies, which can include strokes and respiratory failure. Researchers estimate the number of those living with neurological conditions will double by 2050 [3]. This accentuates the importance of creating awareness for those currently affected and those that will be in the near future.



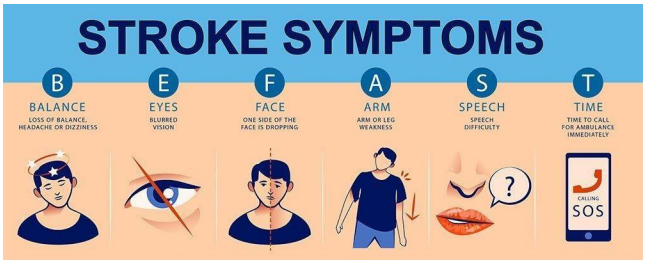
Causes

Neurological conditions can occur done or more key factors. For example, Parkinson's disease is a progressive condition that is characterized by the degeneration of dopamine-producing nerve cells in the brain. Most cases of Parkinson's are idiopathic; idiopathic refers to the condition as having no known cause. However, one common hypothesis is that the development of Parkinson's is aided by hereditary gene mutations. It is also important to highlight the disease's link to pesticide and toxin exposure, in which cellular DNA and mitochondria become damaged and cause nerve cells to degenerate. Pesticides are more prominent in rural areas [4], while toxin exposure typically originates from the workplace or from drug use. Hence, Parkinson's can be linked to both genetic and environmental factors. This emphasizes the need for more research into neurological disorders to fully understand their causes, and help bring insights into any possible treatment options.



Neurological disorders can also occur due to lifestyle choices. An example is a stroke, a sudden-onset condition, which can be separated into ischemic and hemorrhagic strokes. Ischemic strokes are caused by clots or plaque deposits in the cranial blood vessels, creating a lack of blood flow to the brain. In hemorrhagic strokes, brain bleeding occurs due to one or more blood vessels in the brain rupturing. During both of these types of stroke, the lack of oxygen causes nerve cells to die, resulting in personality changes, paralysis or vision loss. Prominent risk factors in the development of this disease can include high blood pressure and long-term smoking. Therefore, lifestyle choices are seen to exacerbate pre-existing medical

conditions, leading to life-threatening medical emergencies such as strokes. This illustrates the importance of physical wellbeing in order to prevent the development of neurological diseases.



Indications

The 4 types of neurological disorders each have their own defining symptoms. For example, sudden-onset conditions have abrupt symptoms such as immediate vision loss, weakness, or an intense headache [5]. Intermittent conditions manifest episodically in intervals, which can entail pain, fatigue or emotional outbursts. Additionally, a decline in cognitive and physical capabilities indicates a progressive condition, whereas chronic pain that fluctuates over time can signal a stable condition with changing needs. Pain can come in the form of cramps or discomfort, and cognitive issues can affect memory, emotion and thinking. Depending on the severity of their illness, patients can require assistance with their daily lives from either their family or hired caregivers. Accordingly, more assistance programs should be established for those affected by neurological conditions to help reduce any financial and emotional burdens.

Diagnostics and Treatment

Immediate treatment for neurological conditions focuses on efficiency, while long-term treatment focuses on rehabilitation. In the case of sudden-onset conditions, every minute a stroke remains untreated 2 million nerve cells die [6]. The consequences of nerve cell death can include permanent brain damage and lifelong disability, showing the necessity of immediate treatment. In addition, occupational therapy is being extensively incorporated into long-term treatment plans to allow patients to regain some independence. This is done through the simplification or modification of their everyday tasks.

Memory and thinking skills are also improved through the use of Cholinesterase inhibitor drugs, which work to prevent the breakdown of acetylcholine. Acetylcholine is a neurotransmitter responsible for brain cell communication. Thus, treatment for neurological diseases is largely holistic, acknowledging the specific needs of the individual in rehabilitation or in a medical emergency.

Diagnosis of nervous system disorders applies cognitive assessments, imaging and laboratory blood tests. In the diagnosis of multiple sclerosis, an intermittent disease, there are two main tests used to obtain a diagnosis: a structural MRI, and a neurological exam. Structural MRIs provide 3D images of organs, tissues and bones to reveal structural damage, while neurological exams are performed using a series of questions and observations to evaluate the neurological functions of the body. Lumbar punctures can also be used to support the diagnosis through the collection of cerebrospinal fluid, used for cell counts and other measurements. As observed, diagnostics of neurological diseases use a wide variety of tests. However, during emergencies, a diagnosis might rely purely on a doctor's judgement. Hence, there are significant concerns about the use of AI in medical studies, especially given its negative correlation with critical thinking scores [7].

Furthermore, despite the prevalence of neurological conditions, allocated funding is present in only 18% of WHO Member States [8]. This draws attention to both the international disparity in resource distribution and worldwide ignorance of an ever-growing epidemic.

Conclusion

In summary, neurological conditions are separated into four main types: intermittent, sudden-onset, progressive and conditions that are stable with changing needs. Their

emergence is due to one or more factors, with idiopathic conditions having no definite cause. They can be identified through many symptoms: pain, emotional imbalance or cognitive and physical differences over time. For life-threatening conditions immediate action is necessary, while others require long-term care. Moreover, to diagnose a neurological condition, imaging, clinical testing and lab work are performed. The importance of future research, awareness and funding cannot be overstated as the growing number of those affected by neurological diseases continues to be overlooked.

By Teesha Cao

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The Neuroscience of Grief

Introduction

On May 25 2019, the World Health Organisation released the 11th revision of the International Classification of Diseases (ICD-11), of which prolonged grief disorder (PGD) – a diagnosis characterised by severe, persistent emotional pain – was formally included [1]. An estimated 7-10% of bereaved adults and 5-10% of children and adolescents experience PGD [2].

Even in everyday life, it is inevitable for one to avoid grief at a certain point. To many, this feeling could be described as extremely unpleasant and overwhelming. In order to effectively represent the mental turmoil of individuals facing terminal illness, Swiss-American psychiatrist Kubler-Ross famously proposed the "5 Stages of Grief" – Denial, Anger, Bargaining, Depression, and Acceptance [3]. While most people know about this, many have not thought about the neurological and biophysical effects that silently take place during grief.

The Ambush of Grief

When someone experiences grief, a multitude of changes take place within the brain. Especially right after a loss, the amygdala's baseline activity becomes hyperactive, making emotions feel overwhelming and difficult to control [4]. Thus, this suggests why some individuals feel intense anxiety and may even have a panic attack. Grief also triggers the anterior cingulate cortex and insula, which are in charge of pain perception and sensory processing [5]. As such, this shows that the brain may perceive grief as a physical threat, increasing one's emotional and pain regulation levels. Therefore, we can conclude that during grief, the brain's psychological processing capabilities are greatly heightened.

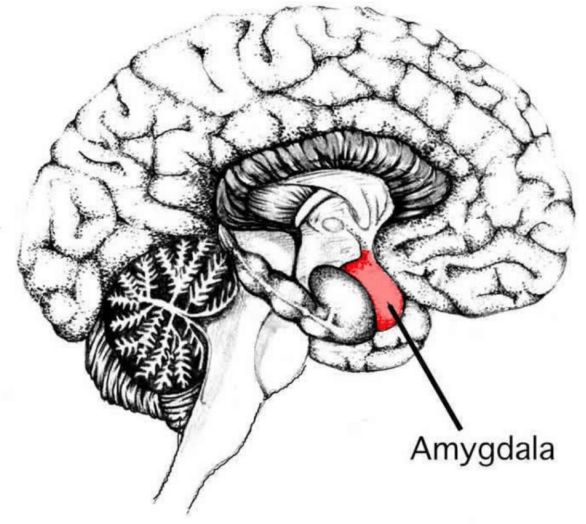

Fig 1: image of amygdala

The Physical Effects of Grief

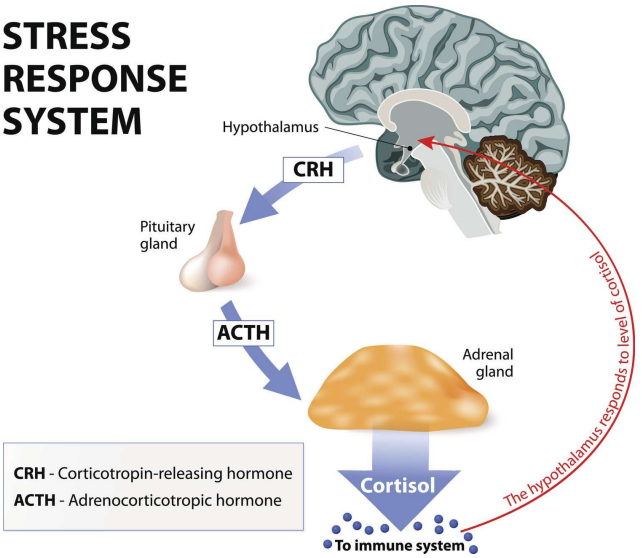

Fig 2: image of cortisol releasing mechanism in stress response system

While many believe that grief is merely a psychological issue, there is strong evidence suggesting that grief could also cause adverse effects in one's body. In particular, there is an evident relation between grief and an individual's HPA axis (hypothalamic-pituitary-adrenal axis), which is responsible for the body's response to emotional pain [6]. Primarily, when we grieve, the HPA axis secretes an increased level of cortisol, a hormone linked to stress [7]. Findings have shown that, consequently, when cortisol levels are too high in one's system, there are several negative side effects on one's physical body, including high blood pressure, muscle weakness, and even a weakened immune system [8]. In fact, prolonged exposure to excessive cortisol is known to cause a condition called Cushing's Syndrome. This leads to various symptoms like skin ulcers and hypertension [9].

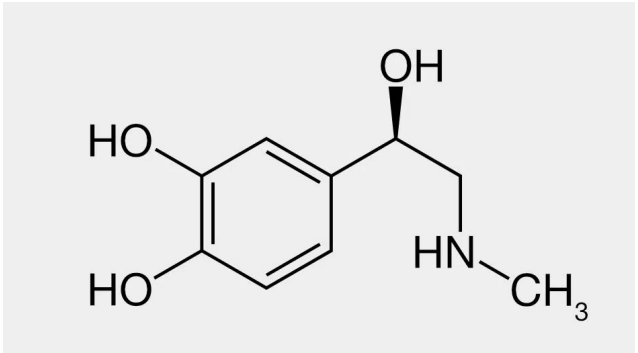

Fig 3: image of chemical structure of adrenaline

Other than cortisol, grief also causes an increased release of adrenaline into the body. Adrenaline (also known as epinephrine), released by the adrenal medulla, is crucial for the "fight-or-flight" response. Initially, this surge in adrenaline would cause heightened breathing and alertness, which is linked to the extreme and overwhelming anxiety that one feels [10]. However, this sudden spike of adrenaline may result in a deep energy crash after a while, causing one to immediately feel fatigue as the adrenal glands become unable to cope with the ongoing demand of adrenaline [11]. This also may explain the "Depression" stage in Kubler-Ross's model, as one's overall energy levels would be lowered throughout the day.

Recovering from Grief

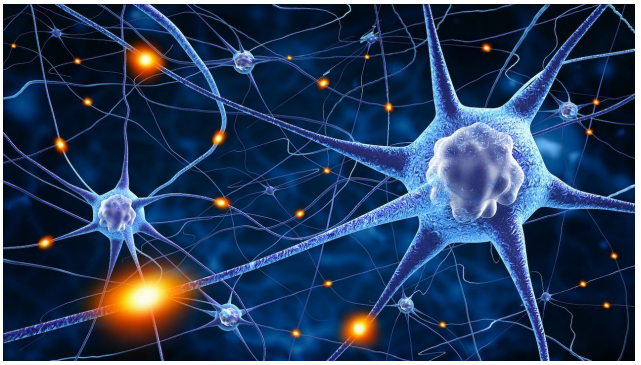

Fig 4: image of neurons communicating via synapses)

Fortunately, whether it be for recovering from physical damage or learning new information, the brain is proven to be remarkably flexible. Otherwise known as neuroplasticity, one's brain can essentially rewire itself after exposure to different stimuli [12]. In this case, after one experiences a traumatic event, the brain can recuperate from this event in the long-term. This hence allows one to better recover from the emotional turmoil. In particular, the prefrontal cortex would slowly regain its function over the amygdala, allowing one to think more calmly and rationally [13]. Also, the hippocampus, which helps store memories, has been shown to display strong potential for plasticity. It could adapt from past trauma such that memories of the loss are more

tolerable to a bereaved individual [14].

However, research has also shown that once the damage is done, it is extremely difficult for neuroplasticity to completely bring the brain back to its original state. For example, one's hippocampus volume is shown to decrease after experiencing trauma, implying that memory might become permanently impaired [15]. Likewise, cortisol has also been proven to suppress the immune system in its repairing ability, leading to increased inflammation and reduced lymphocyte activity [16].

Society's View on Grief

From historic to modern times, society's perception and remedies for grief have changed drastically, and this can be seen through the changing of culture and practices. In Ancient Greece, threnodists would chant poetic laments at gravesites, expressing sorrow for the dead [17]. In modern society, there have also been significant efforts made to recognise grief. Every August 30th, National Grief Awareness Day seeks to encourage open conversations about bereavement, providing support to those who have recently suffered from grief [18].

Conclusion

Ultimately, grief is a complex tapestry of psychology, biochemistry, and neurology. While grief is seen above to be partially affected by our neurological and hormonal response, there are also several factors not covered, like societal views and one's adaptivity to grief. In short, grief is a completely natural response of the body. If one is suffering from extreme grief, it is essential to consult with a trusted friend, adult or medical professional to seek further assistance.

By Yutong Wu

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Thank You!

This concludes the sixth issue of the Penrose Magazine. Thank you so much for reading and we hope you enjoyed!

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