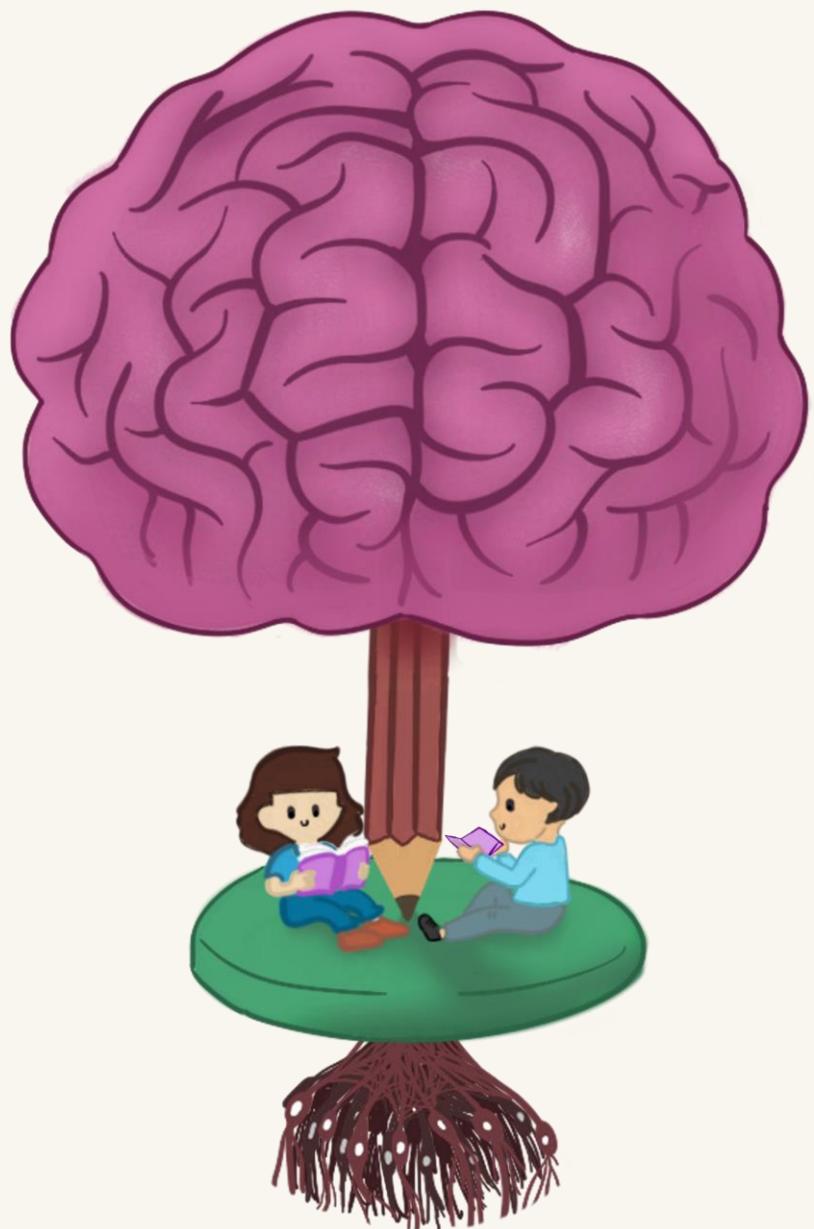


Tales of Neuroscience



The Mind Gala

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The Mind Gala is a collaboration of Project Encephalon and Thakur Neurodegeneration Lab. This program is sponsored by IndiaBioscience.



Project
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Preface

Welcome to the world of neuroscience! If you are reading this, chances are you are curious about the brain and how it works. Well, you are in the right place! Everything ranging from a needle to entire civilizations has been possible because of the squishy ≈ 1.5 kgs of stuff inside our skulls. The human brain is a marvelous piece of biological machinery that has shaped humans and human civilizations.

The human brain is truly an amazing thing, it's like a supercomputer that can handle multiple tasks at once! It's not just one single entity but a collection of specialized systems that work together seamlessly. Think of it like a team of superheroes, each one with their own unique powers, but all working together to achieve a common goal.

The brain accomplishes these tasks by employing billions of cells called neurons. Each neuron is connected to several hundreds and thousands of other neurons giving rise to trillions of synapses in the brain. The sheer number of connections gives rise to networks with enormous processing power.

However, the enormous complexity of the brain means that sometimes it becomes too difficult to understand how it works. This book is our attempt at simplifying some of the concepts in Neuroscience. The study of neuroscience has a long history dating back to ancient civilizations. But it has only been in recent decades that we have made significant progress in understanding the function of the brain. Technological advancements such as brain imaging, gene editing etc have made it possible to study the brain in details that was impossible a few decades ago. By understanding how the brain works, we can better understand and treat a wide range of medical conditions, from chronic pain to neurological disorders.

This book is a collection of articles written by students from across India who have participated in The Mind Gala Science Writing Mentorship Program. This program, funded by IndiaBioscience Outreach Grant, is a 6-week mentorship program that pairs students with early career scientists and professional science communicators to learn about science writing and communication. In these pages, you'll explore the various themes of neuroscience, including basic neuroscience, diseases, encounters of daily life, mental health, and neuro-technology. You'll learn about the structure and function of the nervous system, as well as the various diseases and conditions that can affect it. You'll also discover how neuroscience impacts our daily lives and how it's being used to develop new technologies. We hope that you'll find this book both exciting and informative and that it will spark your curiosity about the amazing world of neuroscience.

The Mind Gala initiative is a result of a very fruitful collaboration between Project Encephalon and Thakur Neurodegeneration Lab at IISER-Thiruvananthapuram. We'd

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like to extend our sincere gratitude to the mentors, who have guided and supported the students in the journey of becoming effective science communicators. We also thank all the volunteers working in this initiative. We hope this book will encourage more young students to take an interest in science and inspire them to explore the wonders of the world inside our head. Happy reading!

Pranjal Garg (Project Encephalon)
Poonam Thakur (IISER-Thiruvananthapuram)

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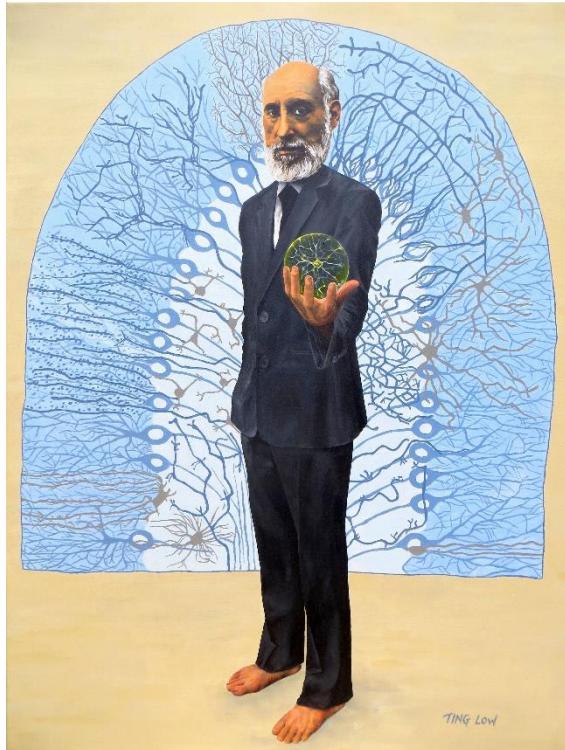
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TING LOW

Santiago Ramón y Cajal
(Nobel Prize in Physiology or Medicine, 1906 for enhancing our
understanding of the structure of nervous system)

PART I

Basic Neuroscience

Through the Kaleidoscope: The Multi-layered Conscious Mind

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SUMMARY

Consciousness emerges as a uniting feature of all living beings and is vast and complex in its scope and implications. Dreams and their supposed distortion of consciousness; add another layer of intrigue. With its multiple potential theories and structural bases, consciousness presents an insightful backdrop to studying the intricacies of processing and integration within the brain.

INTRODUCTION

Have you ever woken up trying to recollect bits of your dream and make sense of them?

Most of us are familiar with this feeling, wondering if our brain cooks up connections once we wake up or if our dreams are what we remember them to be.

What does it mean to be 'awake' and 'conscious'? How does this state differ from the state of dreaming? In fact, how do we even know that we don't just live in a literary cliché and that everything we experience isn't just a dream?

An essential characteristic of our normal waking state is consciousness. Biologically speaking, consciousness refers to an organism's ability to respond to a stimulus. It is also the state of being aware or perceptive of one's surroundings. It encompasses everything you experience - the good, the bad, the ugly, the sound of music, and cheeky movie references.

This article explores the various theories of consciousness, the unconscious mind and its workings, the social neuroscience underlying consciousness and unconsciousness, and, lastly, explores the neuroscience of dreams. Let's begin

FREUDIAN IDEA OF THE CONSCIOUS AND THE UNCONSCIOUS MIND

Over a hundred years ago, the famous Austrian neurologist and the pioneer of the psychoanalytic theory, Sigmund Freud introduced the world to the Freudian Iceberg - a creative analogy to better explain the human mind and psyche. (Fig. 1)

Sigmund Freud is the founder of psychoanalysis. He devised a clinical method for assessing and treating pathologies of the psyche (including both the conscious and the unconscious mind) through dialogue between a patient and a psychoanalyst. However, social neuroscience - an emerging domain- uses fMRI techniques, which shows that the unconscious part of the mind is beyond our control and awareness. This realization contradicts the former idea of unconsciousness being a mere shadow of the conscious mind [1]. Hence, simple therapeutic interventions like dialogues don't seem to be sufficient to understand that part of the mind. This novel idea is far from the traditional Freudian view that the unconscious mind is hidden for emotional reasons and can be conjured back up using introspection therapies [2].

MODERN NEUROSCIENCE AND CONSCIOUSNESS

Social neuroscience is a merger of three fields - social psychology (the science of how people interact with each other), cognitive psychology (the science of how people think), and neurobiology (the anatomy, physiology, and biochemistry of the brain that enable function). This modern approach has made our understanding of the human mind much clearer since we are now able to link molecular mechanisms and neural pathways to behaviors we see [2].

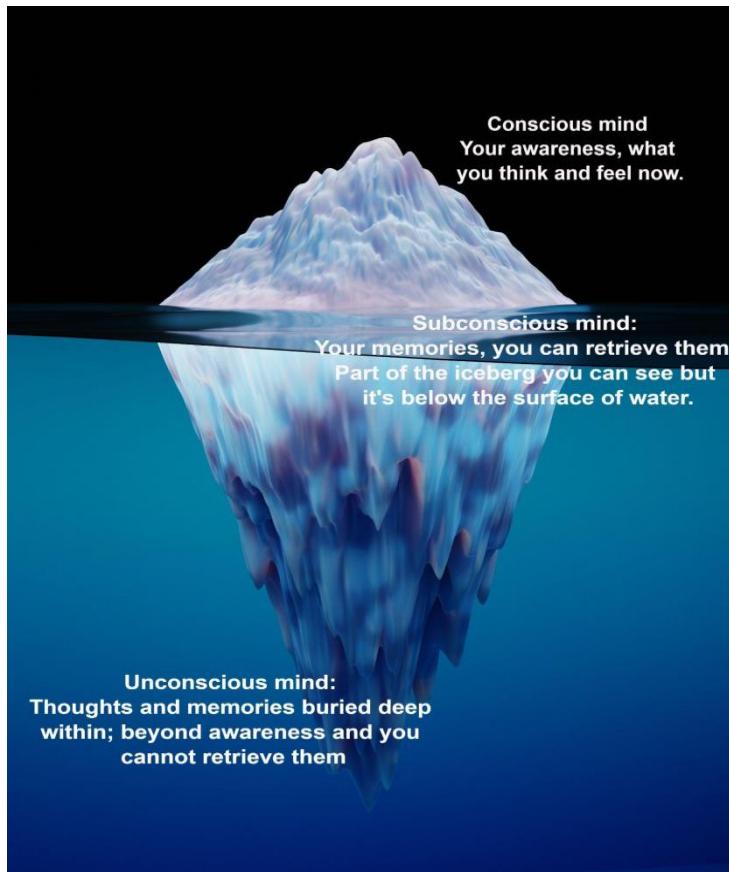


Figure 1. Freudian Iceberg.

"The mind is like an iceberg; it floats with one-seventh of its bulk above water."

- Sigmund Freud

Image Credit: Photo by [SIMON LEE](https://unsplash.com/) on <https://unsplash.com/> (edited by author)

Our sensory perceptions, such as visual and auditory, are innately similar to our social perceptions, such as our judgments of people and situations. Both of these have been greatly influenced by the unconscious mind, which acts like a filter, giving us a lucid picture of the world around us. Without this subliminal filter, the data we see and hear is overwhelming and makes little sense [2].

The conscious and the unconscious mind work hand in hand. For instance, on hearing the word 'book', consciousness tells you that it's a set of pages with something written on them which are bound together. However, the unconscious goes ahead and starts conjuring up images of all the books you've read, revisiting some of their stories and potentially even transporting you to a beach where you'd want to sit and relax with your favorite novel.

Consciousness keeps the unconscious mind in check and doesn't let it stray too far. If unconsciousness is the horse, consciousness is its reins. That is why we can complete our daily tasks without getting distracted by disjointed thoughts. The conscious mind is like the ringmaster of a circus. It gives direction and context to the thoughts and imagery churned out by the unconscious mind. Without this regulation, the unconscious mind would be like a ship without a sail, cast about by random winds.

THEORIES OF CONSCIOUSNESS

Consciousness was long regarded as a mystical entity with no structural or neurobiological basis. The study of consciousness as a scientific phenomenon began only in the 20th century. Since then, attempts to delineate the location and mechanism of conscious processing in the brain have taken four main theoretical approaches: -

- global workspace theories,
- higher-order theories,
- the Integrated Information Theory, and
- re-entry and predictive processing theories [3].

The first two approaches address the functional aspects of consciousness, while the other two deal with consciousness as an experience [4]. Structurally speaking, while each of these theories implicates different brain regions, the consensus is that the cerebral cortex is the 'seat of consciousness'. The midbrain reticular formation and thalamic nuclei perform functions such as gating [5]. Intracranial electrical stimulation (IES) of certain prefrontal regions (chiefly the orbitofrontal cortex and the anterior cingulate cortex) has produced perturbations of the conscious experience, proving that these regions also play a role [6].

The Integrated Information Theory, proposed by Giulio Tononi in 2004, is an effect-to-cause approach, essentially walking backward from the experience of consciousness and its properties - termed "axioms" - to infer the properties of physical systems (termed "postulates") that can account for these axioms [7]. It asserts that consciousness relies on the capacity of a system to integrate information and that consciousness is associated mainly with a posterior cortical "hot zone" in the brain (which includes parts of the parietal, temporal, and occipital lobes).

This theory also allows the mathematical quantification of consciousness in terms of the *phi* metric (the expression of synergy, indicating how much a system is greater than the

sum of its parts). Hence, this explains what consciousness is, where it comes from, and how much of it is present in a person at any given point [8].

Global neuronal workspace (GNW) theories, meanwhile, depict the brain as a blackboard – a space where different cognitive entities can interact to produce the cumulative experience of consciousness. A simple way to think about global workspace theories is to consider that conscious states are more "famous" in the brain than unconscious states, i.e., they are broadcast more widely in the brain and influence a vast array of cognitive processes. This hypothesis is supported by functional brain imaging, which demonstrates that conscious cognition is associated with diffuse, widespread cortical activity (especially in the frontoparietal and medial temporal regions), while unconscious states activate only local regions in the brain. In fact, unconscious states such as deep sleep, general anesthesia, and coma show a marked decrease in metabolic activity in the frontoparietal region [9].

Higher-order theories liken the conscious state to a naive child – one who knows he exists only because his elders tell him he does. These theories are inherently "meta", proposing that a mental state is conscious when the subject is conscious of that state. Reminiscent as this is of the transitive property we use in mathematics (and life), it has been dubbed the Transitivity Principle.[8] These theories' primary goal is to explain what differentiates conscious states from nonconscious states [10].

Finally, we have the theory of predictive processing (PP). Predictive processing is exemplified beautifully in everyday occurrences such as your foot tapping along to a beat. Herein, your brain identifies patterns in the rhythm and uses this information to anticipate the timing of the next beat, and produce the motor output of a tap. This 'anticipation' is believed to occur through top-down, bottom-up, and, essentially multidirectional signaling [11]. While predictive processing in itself is not a theory of consciousness, it has been suggested that it plays a chief role in attention and may provide a valuable perspective on some of the functions and theories of consciousness [12].

DREAMING AND CONSCIOUSNESS

Many theories seeking to explain the difference between the conscious and the unconscious attempt to correlate it with the occurrence of dreams. However, we are still uncertain as to which of these is the whole truth; the truth could possibly be a combination of parts of these theories. One of the crucial theories goes as follows:-

When you're sleeping, your conscious mind sleeps too. It is at this time that the unconscious mind takes control and roams about freely with no boundaries. Now, since the unconscious mind can only access already existing memories and experiences, it mixes and matches and plays those bits of memories in a random kaleidoscopic demonstration - a dream. Once you wake up, the consciousness gets back in the driver's seat and tries to give context to those dreams, grounding the ones that make sense in reality and letting the others fade from the memory [13].

By now, it must be clear that the necessary condition to dream is the unavailability of consciousness. That explains why we do not dream throughout our sleep. In our sleep cycle, the REM (rapid eye movement) phase, when our mind is free from the shackles of consciousness, is when we dream. Also, our body physiologically ensures that we don't act out

our dreams in the waking world by lowering our blood pressure, reducing our heart and breathing rate, and largely paralyzing voluntary functions. However, surprisingly, brain activity is sustained and may even rise higher than that when you're awake! [14].

Ever experienced a situation where you go to sleep with a complex problem unsolved, miraculously waking up to its answer the next morning? That's the doing of your unconscious mind - piecing things together, looking at things from a different perspective. Research has also shown that dreams might actually enhance creativity [14]. This might be because the REM phase is when the notorious anxiety-inducing hormone noradrenaline is at an all-time low in our bodies. This gives the unconscious mind a calmer environment to conjure up important memories your conscious mind tried to suppress. Hence, you see, unconsciousness isn't just that pesky, disobedient cousin that consciousness shudders at the mention of!

"It's said that time heals all wounds, but my research suggests that time spent in dream sleep is what heals."

-Dr. Matthew Walker

New theories continue to be proposed every day, distilling and bringing into focus what we know (or what we think we know) about the mind and consciousness. Does the truth of the matter lie in the existing theories? Or is there some yet-to-be-undiscovered force at play?

Well, as the saying goes, only time will tell!

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Healthy Aging - Can Mushrooms Help?

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SUMMARY

Ergothioneine (EGT) is a naturally occurring compound found in mushrooms that is now being explored as a potential helper for healthy aging. It has the potential to prevent/reduce the incidences of aging-related diseases/disorders with no adverse effects. Current research aims to identify its exact mechanism of action and therapeutic capabilities for various conditions associated with aging. Here, we summarize the current status of EGT research in the context of aging.

INTRODUCTION

According to the World Health Organization (WHO) by the year 2030, one in six people will be aged 60 years or older, and this population is expected to increase further in the coming decades. Aging is an obvious inevitability of human life that poses new medical and societal challenges. Researchers are exploring ways to delay or even completely prevent aging in their current scientific pursuit. In 2020, UN General Assembly declared 2021-2030 the UN Decade of healthy aging because we need to modify both our actions and our perspective toward aging and increased life expectancy [1]. In this article, we will discuss various possible theories of aging and some probable interventions.

THEORIES OF AGING

Aging is a complex multifactorial phenomenon that involves various cellular and molecular mechanisms, intrinsic (genetic), and environmental factors. Many theories have been used to explain the process of aging, but none thoroughly explain all the associated conditions because of its multifaceted dependence. Some of these theories are summarized as follows:

According to the DNA Damage theory, multiple exogenous and endogenous factors are responsible for DNA damage, all of which are not repairable. Hence, the accumulated damage causes the cell to progress toward aging [2, 3].

The telomere attrition theory suggests that the progressive shortening of telomeres results in cell cycle arrest [2]. Telomeres are short, repeated DNA sequences present at the ends of chromosomes. During replication, a small telomere segment is not duplicated, resulting in progressive shortening of the telomeres in successive cycles. The ends of chromosomes are left unprotected, which signals cell cycle arrest, leading to an inability to generate new cells that can replace damaged ones [3, 4].

The Oxidative Stress aging theory postulates that there is a slow and steady accumulation of oxidative damage to nucleic acids, lipids, sugars, and proteins, due to free radical attack. Reactive oxygen species (ROS) are highly reactive molecules formed due to incomplete reduction of oxygen generated during cellular metabolism and are considered key contributors to oxidative stress [2, 3, 4, 5].

Other theories include defective protein homeostasis due to impaired chaperone (which mediates the correct folding of proteins) and proteasome (responsible for the degradation of misfolded, damaged proteins) functions [3]. Impairment of folding and degradation of misfolded proteins due to aging has been observed. Thus, abnormal protein homeostasis can have diverse effects on cell functioning, division, and survival.

The neuroendocrine theory is as follows: as aging progresses, the hypothalamus loses its ability to regulate the hormone system precisely, and the receptor down-regulation decreases the efficacy of the hormones [3, 5]. This depicts the potential role of hormones and neurotransmitters in producing changes associated with secondary aging. It can be due to variations at various levels in the neuroendocrine system.

As mentioned above, none of these theories thoroughly explains the complex and multifactorial phenomena of aging. Hence, finding its therapies or at least delaying aging is tedious and challenging. Most likely, all these multiple cellular and molecular events occur in parallel. Hence, compounds targeting various pathways will be potential options for anti-aging therapies.

DIETARY SUPPLEMENT – CAN MUSHROOMS HELP IN AGING?

Some dietary supplements have antioxidants and anti-inflammatories that can help target some of these aging mechanisms. Among these, mushrooms have been attracting increasing attention for over a century due to their multiple beneficial compounds. Ergothioneine (EGT) is one such compound found in mushrooms.

EGT is a sulfur-containing (histidine-derived) amino acid with antioxidant, anti-inflammatory, and metal-chelating properties [6]. EGT is particularly synthesized in sporocarp (fruiting bodies) of fungi and actinomycete bacteria, with the highest concentration in mushrooms, especially grey oyster mushrooms. EGT was discovered by Charles Tanret when it was purified from the ergot mushroom *Claviceps purpurea* in 1909. Even though found over a century ago, EGT has recently gained renewed attention, owing to the discovery of its particular transporter – the organic cation transporter novel-type 1 (OCTN1), which mediates its uptake from the diet. The variable concentrations of EGT in different body parts is according to OCTN1 transporter distribution in the respective tissue [7, 8]. A study even proposed to rename OCTN1 as Ergothioneine Transporter (ETT) [9] due to its highly specific nature for EGT, becoming a focus for extensive research lately to find its implications to human health (both physiological and therapeutic implications).

EGT is available as an over-the-counter supplement, currently being investigated in combination with other vitamins, especially Vitamin D [10]. It is also used in a variety of skincare and haircare products, since it helps prevent skin aging and promotes hair growth, respectively. It is nicknamed a 'longevity' vitamin due to its potential therapeutic and physiological effects. EGT has been shown to cross the blood-brain barrier, which suggests the predisposition of people with low blood EGT levels to aging-related neurological diseases and other diseases [11].

MECHANISM OF ACTION OF EGT

While it is true that EGT is not synthesized in the human body and needs to be consumed through the diet, multiple studies have shown that EGT levels in blood serum have

correlations to age and cognitive health. It was found that with increasing age, the levels of EGT decrease significantly, especially beyond 60 years of age [12, 13, 14]. Further studies confirmed that the lowered levels of EGT could be one of the factors contributing to age-related diseases including cognitive health, frailty, and cardiovascular problems [15].

Effect on Oxidative Stress and Cognitive Health

EGT is a Reactive Oxygen Species (ROS) scavenger; this essentially means that it has the potential to protect the body from ROS and other oxygen radicals, which can cause cellular damage when present in excessive amounts. In this way, EGT is an antioxidant that promotes healthy aging by preventing the accumulation of oxidative stress [16,17]. EGT also decreases lipid peroxidation [18], and protein oxidation [19] *in-vitro* (studies without living organisms; mostly using cell culture and their tests), which are other forms of oxidative stress.

It has been shown that EGT protects against cell death induced by protein accumulations in mice [18]. This provides a potential treatment strategy for neurological disorders such as Alzheimer's Disease (AD) (which is caused by protein aggregation [21]) by preventing neuronal injury and death [20]. AD is a progressive neurodegenerative disorder characterized by memory impairments and communication problems, and is the most common form of Dementia, especially in old age. EGT seems to have a negative correlation with these causative protein aggregations [22]. Hatano and co. found that levels of EGT are much lower in patients with Parkinson's Disease (PD), another neurodegenerative disease related to movement, memory, learning, and thinking, as compared to healthy controls [23]. This further supports the idea that EGT levels in blood serum and neurological disorders are co-related with each other. It should also be noted that these disorders are mostly age-related, i.e., they occur most commonly in people over the age of 60, indicating that EGT could be involved in healthy aging.

Further, EGT levels were found to be associated with the incidence of Mild Cognitive Impairment (MCI) [12, 24]. MCI is a condition that includes problems with memory, language, and thinking abilities. It was seen that individuals with MCI had significantly lower blood EGT levels as compared to healthy individuals at the same age [12].

Smith and colleagues have also shown that EGT is an important marker of healthy eating patterns and higher levels of EGT indicate a reduced risk of cardiometabolic disorders and death [15]. Additionally, it was found that EGT protects against cardiovascular injury caused by Type II Diabetes in male Sprague Dawley rats [25]. This could relate to EGT's effects on oxidative stress, which has been linked with cardiovascular injury.

Taking together all these studies, we can infer that low levels of EGT can be a potential marker for age-related neurodegeneration, and consumption of more EGT could potentially help in some age-related problems.

Effect on Telomere Shortening

Telomere shortening is another key characteristic of aging that causes aging-related complications. An *in-vitro* study by Samuel and co-workers found that EGT from mushrooms slowed the telomere shortening rate [4]. This directly relates to aging and many other diseases as well. The slower the telomere shortening, the lesser will be the age-related problems, resulting in healthier aging.

Telomere shortening is associated with many life-threatening diseases, such as diabetes mellitus, neurodegenerative diseases, cardiovascular diseases, and cancers. By reducing this phenomenon, EGT can help develop therapeutics for some diseases.

Furthermore, studies show that oxidative stress accelerates the rate of telomere shortening even more. Since EGT prevents oxidative stress (as explained in the previous section), it could also reduce telomere shortening.

Thus, it can be said that EGT could become the basis for healthier aging with further research.

IS CONSUMING ERGOTHIONEINE SAFE?

While EGT has so many benefits, not everyone can eat mushrooms, and those who do may be hesitant to eat in higher quantities. After all, it is said that anything in excess is bad. Thankfully, this is not the case with EGT. A European Food Safety Authority (EFSA) Panel on Nutrition, Dietetic Products, and Allergies (NDA) dubbed EGT supplements safe for infants, young children, pregnant women, and breastfeeding mothers. It has a no-observed-adverse-effect level (NOAEL) of 800 mg/kg, which makes it very safe for consumption [26]. The NOAEL is a measure of the highest dose of a drug at which there are no adverse effects. A higher score means that the compound can be safely consumed. So, both mushrooms and EGT supplements are completely safe to eat.

CONCLUSION

Research on the benefits of EGT has been on the rise in the last decade. While the exact mechanism remains mostly unknown, all the studies done so far, indicate that EGT helps protect the body from various deleterious phenomena and can lead to healthier aging. EGT offers new and exciting avenues, possibly as therapeutics too. Presently, clinical trials are going on in Singapore to find the effects of EGT on cognitive health [27].

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Is it broken beyond repair? A story of neural regeneration

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SUMMARY

Have you wondered why it takes brain injuries so long to heal? While finger cuts and knee scrapes heal in a matter of days, an injury to the brain might take years to heal properly. Is fixing your brain as simple as it appears? Probably not! In this article, we talk about how our brain evolved and the modern technologies that enable its regeneration.

INTRODUCTION

In 2013 Michael Schumacher knew he was treading a dangerous line. He was skiing in deep snow in the French Alps to save a young girl. Just then the World No. 1 Ferrari driver collided with an exposed rock. Propelled into the air, he fell with his head landing on another rock, resulting in a traumatic brain injury despite wearing a helmet. With limited recovery of mobility, Schumacher continues to fight for his life even today.

In contrast to injuries to other parts of the human body that heal within days, months, or even years, an injury to the brain or spinal cord may never heal. Humans possess a relatively little capacity to repair or regenerate the building blocks (neurons) of the central nervous system (CNS) due to their complexity. The peripheral nervous system (made up of nerves branching out from the CNS) on the other hand, can regenerate, allowing for rapid recovery of function in the event of damage.

HUMANS ARE UNLIKE FISH AND AMPHIBIANS

Although humans, other mammals, and birds do not have the ability to regenerate neurons, you may be surprised to learn that most fish can repair and regenerate various components of their CNS. It was seen that fish lack a protein called Nogo-A that impedes CNS regeneration, while humans express it abundantly [1]. Amphibians such as frogs lose the ability to regenerate the CNS only in their adult form, which makes them special. So, frogs occupy an evolutionary position that is in between man and fish.

WHY DID HUMANS LOSE THE ABILITY OF NEURAL REGENERATION DURING EVOLUTION?

It is still unclear why mammals lost the seemingly useful ability to regenerate neurons during the course of evolution, but there exist a few theories [2].

1. One possible explanation is that the loss of neuronal regeneration in organisms could increase resistance to infections in the CNS by blocking the multiplication of infected cells, which have the potential to severely damage the system.
2. Another hypothesis proposes that increased thyroid (hormonal) signaling may have resulted in the loss of regenerative abilities among mammals. It was seen that this hormone inhibits the regrowth of neuronal appendages in the human cerebellum (little brain). Subsequently, an evolutionary trade-off was made between the thyroid hormone and neuronal regeneration with a gain of thyroid function (essential for proper metabolism, development, and growth).
3. Loss of regenerative capacity has also been linked to increased resistance to cancer. Uncontrolled cell multiplication, mutation, and tumor formation are hallmarks of cancer.

Inhibition of increased cell division and thereby not regenerating neurons may indirectly provide resistance to cancer formation in long-living species such as mammals.

SO HOW CAN WE REGENERATE NEURONS IN HUMANS?

Neurological disorders are a significant cause of disease and death throughout the world. They not only reduce the quality of life of affected individuals and their families but also pose a significant financial burden on the healthcare system. In India, the number of non-infectious neurological disorders more than doubled from 4% in 1990 to 8.2% in 2019. Stroke alone claimed 699,000 lives in 2019, accounting for 7.4 percent of all deaths in the country [3]. Other non-communicable neurological disorders include dementia such as Alzheimer's disease, Parkinson's disease, etc.

Regenerative cell therapy, also known as stem cell therapy, has demonstrated promise in the treatment of neurological disorders. This is due to the ability of stem cells to replace damaged cells and provide an environment conducive to neural cell regeneration. Stem cells are distinguished by their ability to self-renew and differentiate into a variety of cell types. They are the starter kit of every organ in the body. What is special about modern-day stem cells is that they can be personalized to an individual. Such stem cells (known as induced pluripotent stem cells or iPSCs) are a product of reprogramming adult cells. So imagine, tomorrow you could have at your disposal custom-made stem cells by donating just a few of your skin cells, and these could ideally replace any broken part of your body!

In general, for stem cell therapy to be effective in treating brain diseases in which neurons are damaged or defective, the treatment should aim to repair, replace, or at the very least prevent future deterioration. The goal of stem cell therapy is thus to allow therapeutic cells to be directed to impaired or injured brain regions, to stimulate tissue repair and maintenance, and potentially even to generate new neurons. Recent studies have found that exogenous stem cells can migrate to damaged brain tissue, then participate in the repair of damaged brain tissue by further differentiation to replace damaged cells, while releasing anti-inflammatory and growth factors, thereby significantly improving neurological function [4]. Another study [5] used the rat model with traumatic brain injury to study the role of mesenchymal stem cells (MSCs) in neurogenesis. It was observed that in addition to their secretory ability, MSCs could selectively migrate to the injured tissue sites and then differentiate into neurons and astrocytes to repair damaged brain tissue (Fig.1).

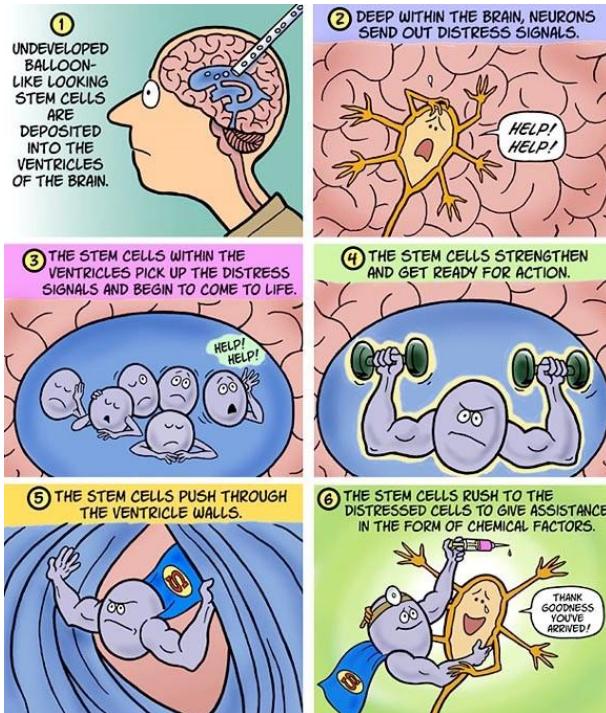


Figure 1. Injected stem cells rescue brain damage

Image credit: Steve Greenberg for California Neuroscience Institute, used here with the artist's permission.
<https://www.greenberg-art.com/Infographics/StemCells.html>

APPLICATIONS OF STEM CELL THERAPY IN NEURODEGENERATIVE DISORDERS.

Regenerative medicine based on stem cells is becoming an appealing approach to developing therapies for neurological disorders. Japanese authorities recently approved a mesenchymal stem cell (MSC) product for the treatment of spinal cord injuries. This is the first and only stem cell therapy that has been approved for the treatment of a neurological condition. MSCs are generally derived from bone marrow or adipose tissue and administered to patients via a lumbar puncture or spinal tap. Following this, patients are administered the MSC medium. This medium contains a range of cytokines, chemokines, and growth factors which are thought to further promote neuronal regeneration [6]. Stem cell therapies using MSCs is one of the most sought-after and dynamically developing branches of regenerative medicine as MSCs are easy to obtain and present a minimal risk of rejection. Their multidirectional ability to differentiate has driven up their value in translational research and clinical trials for the treatment of the common neurological diseases such as stroke, Alzheimer's disease, amyotrophic lateral sclerosis, Huntington's and Parkinson's diseases, multiple sclerosis and

spinal cord injury, for which there are no effective treatments till date [Andrzejewska, et al., 2021][7]. Additionally, more therapies are being developed by researchers using different stem cell types to regenerate and restore neural cells.

Perhaps if Michael Schumacher had received such treatment after his accident, he may not have had to deal with immobility or loss of speech until today. Although much work remains to be done in the use of stem cell therapy to treat neurological disorders, regenerative therapy is expected to be successfully applied in a clinical setting soon.

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To Remember or Not to Remember: The Science of Memory Editing

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SUMMARY

Memories form the basis of our experiences and shape our perspectives of the world. Understanding the neuroscience behind memory has given rise to the field of memory editing. In this technique, fear associated with traumatic memories can be 'lost', and memories can be rewired, thereby serving as therapy for patients with psychological trauma.

INTRODUCTION

Anita woke up with a jerk, sweating profusely and hyperventilating. She reached for the glass of water kept on the alarm stand beside her bed, her hands shivering. The red glow of the clock reading 3:16 am was the only light in the pitch-black darkness in her room. She took generous sips of the water, slowly calming her breath down. It was the nightmare again. The same nightmare that replayed in her mind every night for the past 2 years. If only she could brush it off as a mind conjecture, an unwanted illusion of thought. But no, unfortunately, this was more than just a nightmare. It was a memory, something that had actually happened, a piece of trauma etched into the brain, causing waves of horror every night it played, again and again. Anita took her prescribed medication, and went back to bed, but stayed awake for a long time until her brain shut from exhaustion, only to be awakened by the shrill sound of the alarm clock a few minutes later.

"How are you feeling, Anita? It's been a while..." Anita looked at the doctor sitting across from her. Dr. Nandini was sitting at her desk, report in hand, with a composed, soft expression on her face.

"They're getting worse", Anita said. "I wake up almost every night now."

"So, the meds aren't helping?", asked the doctor.

"Barely. They mellow down for a few days after my therapy sessions, but I can't afford them anymore. Why can't I just get used to it?" she replied.

Dr. Nandini sighed, with an empathetic look on her face. "Anita, you have to remember, that considering what you went through, you've made great progress. You are very strong. It's no fault of yours."

"Why can't I just forget it?! After all this time, it still feels like it happened yesterday..."

"Unfortunately, that's the deal with memories. Your brain turns short-term memory into long-term memory through a process called *consolidation*. Remembering and replaying the traumatic memories again and again on receiving environmental cues only strengthens it. It's a process called *reconsolidation*. However..."

"Yes?"

"The reason I called you is to tell you about this new experimental therapy that edits traumatic memories," Dr. Nandini replied.

"Edits memories? Like changing them?"

"Yes, your memories can be altered to remove certain associations."

"How does this work?" asked Anita, with a curious glimmer in her eye.

"I'll explain this! First, let's start with the neuroscience of memory."

NEUROSCIENCE OF MEMORY

The process of memory formation does not occur in a single area of the brain; rather, it is distributed into multiple regions within the brain. We all know that neurons help convey information to different parts of the brain and the body at the most fundamental level. Messages are conveyed from a single neuron to another with the help of molecules termed *neurotransmitters* which pass into the spaces between two neurons termed the *synaptic cleft* [1]. When information is received through our sensory nerves it undergoes various steps for its storage and retrieval. This can be summarized in 4 steps:

1. Encoding

Information is perceived by our brain in the form of sensory inputs. Not all of these inputs are stored in the form of memory, only the ones we pay attention to or have significant emotional responses are stored as short-term memory. For example, we might scan different people's faces while walking but don't always tend to remember their faces until someone we recognise appears in front of us. Short-term memory is temporarily stored in a region called the prefrontal cortex. Short-term memory only lasts for a few seconds to minutes (memories like remembering a phone number/OTP). If the information is not retrieved within a short span it is deleted from our memory by weakening the connections between our neurons [2].

2. Consolidation

Consolidation is the process by which the memories are reorganized by molecular and cellular events that alter synaptic connections to increase complexity and distribution between the hippocampus and cerebral cortex [3].

3. Storage

If the information is retained by continuous repetition, the existing synaptic connections associated with it are strengthened and the information is stored as long-term memory in the hippocampus and any emotional response, if linked to it, is stored in the amygdala [4].

4. Retrieval

During retrieval, we can reactivate the neural circuits associated with memory, and hence we can recall and reminisce any memory whenever we want. When a memory is retrieved, it was previously thought to be stable. However, recent research shows that it is not stable but subject to modifications through interferences and this is known as reconsolidation[5].

The technique of memory editing was seen to be targeting either of these processes (consolidation and reconsolidation). Since memory is vulnerable immediately after encoding, this method targets its encoding by modifying its storage[6].

Since memory is not located in a single part of the brain but distributed throughout the brain, it is maintained in a population of neurons called memory *engrams*. These are responsible for storing a particular memory and they possess the property of plasticity i.e., they can be modified during the phases of consolidation, reconsolidation and extinction of the memory[7].

For example, if you recently met an inspiring individual and were really influenced by the advice you got or you just gave a presentation that went awful, these memories will be stored across the brain in particular engram cells (Fig. 1). When the same memory is recalled/retrieved in the future it strengthens the connection between these engram cells.

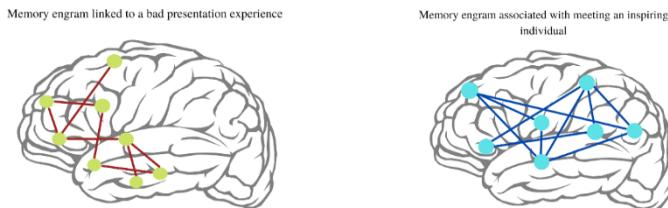


Figure 1: Memory Engrams

“Are you understanding this, Anita?”, asked Dr. Nandita.

“Yes... So now, how exactly can these engrams be edited?”

“Great question! Let’s dive into the realm of memory editing.”

MEMORY EDITING

How do we edit the memories we want? To understand this, it's helpful to think of memory engrams as files that need to be opened in 'edit mode' to make any changes. So, to get a memory into a state where it can be altered, requires specific environmental cues or triggers that are associated with the traumatic memory. When these memories are accessed or 'opened', we can now go in and manipulate them! This is because reactivated memory becomes temporarily unstable and needs stabilization (the process called memory reconsolidation) [8]. More importantly, certain drugs have been shown to interfere with the memory reconsolidation process, effectively serving as potential treatments for traumatic memory removal. A study in 2009 showed that propranolol, a beta blocker drug (used to reduce blood pressure) had the potential to erase fear memories in healthy human volunteers [9]. When you remember a traumatic memory, there is also an adrenaline-rushed fear associated with it. When propranolol is administered after the trigger of a phobia memory, it blocks the pathway of adrenaline-associated fear so that when the memory is reconsolidated, there is no fear associated with the memory anymore [10]!

“Wow!” said Anita. “So, this means that I can lose the fear associated with my traumatic memories?”

“Yes, that’s the goal. Let’s just hope this can work for you.”

“The latest techniques in memory editing involve *optogenetics*, where light is used to activate specific memory engrams to couple fear memories with new environmental cues, which essentially forms a new implanted memory! This has been shown in mice [11], where mice trained to fear a particular environment were shown to exhibit fear in a completely different environment on activating the specific fear engram! Also, video games have been shown to help with memory rewiring. Essentially, performing tasks involving similar regions of the brain involved in the traumatic memory disrupts the reconsolidation process, thereby reducing the impact of intrusive memories [12].”

“Wow! There’s so much advancement to memory editing than I thought!”, exclaimed Anita.

“Indeed! I think the days aren’t too far away that we’ll see movies like ‘Inception’ or

‘Total Recall’ come to life!” remarked Dr. Nandita.

“This is scary! But also exciting because now, there’s a sliver of hope now for people who experience trauma since their traumatic memories can be edited!”

Clinical trials for Propranolol in PTSD patients are not quite conclusive yet[13]. However, the neuroscience behind it is quite promising and the door remains open for new therapies based on memory editing research to enter, so that thousands of people like Anita, may one day lead better lives.

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Adult neurons: origin, fate, and function

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SUMMARY

For centuries, the scientific community believed neurons are formed only in embryos or newborns, but not in adults. In the past few decades, scientists have discovered that new neurons are generated even in adult brains. This phenomenon, called adult neurogenesis, occurs in specific parts of the brain, namely the subventricular zone and hippocampus. However, neurogenesis declines with age and lifestyle changes. This article discusses the concept and history of adult neurogenesis, and the factors that affect it.

INTRODUCTION

If all brains were made the same way, larger brains should have more neurons, and the organism that possesses it must be cognitively more able. Turns out, it's not really the case. The human brain, weighing much lower than that of other mammals such as elephants and whales, performs more cognitively demanding tasks, suggesting that larger brains don't necessarily translate to smarter individuals [1]. Although our brain weighs only about 2% of our body weight, it uses almost 25% of our body's energy. This, among other things, makes the human brain special.

Let us consider how this organ is formed. The brain of a fetus develops in a specialised environment referred to as the 'neurogenic niche.' This environment helps form the composite cells of the brain, i.e., neurons, all the way from their primitive form called the 'neural stem cells' to a mature form that drives many functions in the brain. NSCs are self-regenerating cells in the brain that also have the ability to give rise to mature nerve cells (neurons and glia) in the fetal brain. NSCs ultimately help in maintaining the plasticity of the brain – the process of rewiring the brain, where the brain can compensate for the changes that happen within it throughout one's life. For example, a 55-year-old lady who has lost a small part (left temporal lobe) of her brain right from birth is as healthy as a normal individual with good cognitive abilities, and without any seizures [14]. Neural stem cells (NSCs) are identified by certain proteins present on them, namely Nestin, SOX1/2, and CD133. These are cells that form new neurons, some of which develop into mature, functioning neurons, whereas the others remain in their original form. The latter thus replenishes the NSC pool, thereby helping in forming more neurons.

All these processes occur mostly in fetuses and newborns [2]. Once these neurons mature, which is a few months after birth, neurons typically do not multiply. Yet, there are two areas of the adult brain that still harbor NSCs, which can multiply and form new neurons: the subventricular zone (SVZ) and the hippocampus. This idea of the adult brain forming new neurons is called adult neurogenesis. The NSCs present in the SVZ region generate neurons that then move through a pathway to another region in the brain named the olfactory bulb (OB). OB is the brain area that helps discern different odors. Here, these newly formed neurons grow and branch, maturing into what are called olfactory neurons. This process can be tracked by using proteins that are present on these specialised cells, namely glial fibrillary acidic protein (GFAP), Nestin, Pax6, NeuroD, PSA-NCAM, FoxO3, Tuj-1, and Mash1 [6].

DISCOVERY OF ADULT NEUROGENESIS

In the early 20th century, Santiago Ramón y Cajal, the father of modern neuroscience, said, “In adult centers, the nerve paths are something fixed, ended, and immutable. Everything may die, and nothing may be regenerated. It is for the science of the future to change, if possible, this harsh decree.” For many years the scientific community upheld the idea of fixed, immutable brains. Although skeptical, Cajal was open to the idea of degeneration and regeneration of neurons. He said, “We must recognize that, in the matter of neurogenesis and nerve regeneration, we are still in the phase of collection of materials. As a result, our hypotheses are premature, and they can aspire neither to perfection nor to permanence...We shall abandon it whenever another conception provides a better explanation of the facts of nervous regeneration and degeneration.”

Joseph Altman was the first to challenge the idea of fixed, immutable brains in 1962 in his paper, “Are New Neurons Formed in the Brains of Adult Mammals?” [8]. In his experiments, Altman used adult rats that were traumatized in a specific region of the brain involved in vision, called the lateral geniculate body. Altman tried to label neurons in these regions using proteins present on the cells, and a few days after the injury, Altman found that there were neurons and their precursors around the injured area. Here was the evidence for the possibility of new neurons being formed in adult brains, as opposed to the idea of fixed and immutable brains. However, there was a lot of resistance to this idea from the scientific community. They doubted his methods, criticised his findings, and said that it did not deserve the attention it was due. He was marginalized: denied a promotion he was due at MIT, stripped of his research funding, and rejected his papers. Yet, Altman persisted, and his findings were later corroborated by another scientist, Michael Kaplan. But then, Kaplan also met with a similar fate [8].

Among the scientists who denied adult neurogenesis was Pasko Rakic and M. F. Eckenhoff. The idea of adult neurogenesis faced strong resistance until Fernando Nottebohm, a scientist who was interested in canaries (song birds), proved the findings of Joseph Altman true. Nottebohm and colleagues observed that the female canaries, when exposed to testosterone, could sing like males as they formed new neurons linked to their vocal center in the brain [8, 15].

FACTORS AFFECTING NEUROGENESIS

Several factors including hormones, diet, lifestyle, age, etc. affect adult neurogenesis. These factors, besides aging, can be controlled by staying in a pleasant environment, engaging in physical activity, and maintaining a healthy diet.

The effects of aging on adult neurogenesis have recently garnered a lot of attention. The number of NSCs decreases with age, decreasing neurogenesis. Normally, NSCs are in quiescent form in the adult brain. They divide slowly, forming activated neural stem cells (aNSC). These aNSCs form transit-amplifying progenitor cells (TAPs) that are responsible for generating neurons. With age, proteins in quiescent NSCs tend to form aggregates, and the lysosomes in these neurons turn defective. The number of mitochondria, which are necessary to fulfill the energy demands of these neurons, reduces with age, besides an increase in the number of dysfunctional mitochondria. This leads to the accumulation of reactive oxygen species (ROS) in these cells. All these contribute to the quiescent NSCs losing their ability to get activated, multiply, and mature.

At a molecular level, many proteins play roles in driving the age-induced reduction in adult neurogenesis. For instance, there is a decrease in the amounts of Hypoxia-inducible factor 1-alpha (HIF-1 α), a protein that is necessary for NSCs to multiply when oxygen levels are low, with age. The amounts of androgen (male hormone) and oestrogen (female hormone), which enhance neurogenesis, decrease with age. Moreover, although androgen receptors increase with age, their reactivity with androgen reduces.

The change in the environment where the NSCs reside within the brain also hampers adult neurogenesis. The stem cell niche, where NSCs are present, has a lot of blood vessels. This decreases with age, thus diminishing the ability of these cells to proliferate [9].

EFFECTS OF DECREASED ADULT NEUROGENESIS

An aberration in adult neurogenesis impacts the structure and behavior of OB. Mice without young adult-born neurons (ABNs) could distinguish between very different odours, but not subtle ones [3,4]. In humans, upon loss of adult neurogenesis in the hippocampus, there is emotional confusion along with other neurological conditions like depression, anxiety, and post-traumatic stress.

Studies conducted using brain tissues from non-human primates show that impaired division of TAPs decreases the function of neurons, thereby causing dysregulation in adult neurogenesis. Such dysregulation may ablate cognitive functions and cause neurodegenerative diseases [5].

WHY CARE ABOUT ADULT NEUROGENESIS?

As adult neurogenesis declines with age, understanding this process and the path through which adult NSCs move inside the brain could help us understand neurodegenerative diseases like Parkinson's, identify markers to diagnose them, and devise strategies to treat them. For example, scientists have shown that, in naturally aging female mice, treatment with a protein called plasmalogen could partially improve age-related reduction in adult neurogenesis. In aged mice, increased levels of this protein reduced neuroinflammation, alleviating age-related cognitive decline [7].

In conclusion, as the saying goes, known is a drop, and unknown is an ocean. The idea of adult neurogenesis has come a long way since initial research in canaries; experiments in different organisms, from fishes to reptiles, mice, and humans, have shown adult neurogenesis [10,11]. Recently, some studies have revealed newer adult neurogenesis zones in the brain, besides the hippocampus and subventricular zone (SVZ). These zones include the hypothalamus, striatum, substantia nigra, cortex, and amygdala. Some findings suggest the migration of neurons from the subventricular zone (SVZ) to other regions of the brain. All these findings add to our understanding of the brain, and may also help us in developing therapies for different neurodegenerative diseases [13].

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The unsung heroes of our brain: Interneurons

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SUMMARY

Interneurons integrate sensory information and motor responses. They can be excitatory or inhibitory; however, the brain primarily consists of the latter ones. In this article, we dig deep into the poorly explored world of inhibitory interneurons that are the breaks in our otherwise racing car (the brain) comprised majorly of excitatory neurons. They establish the excitation inhibition (E/I) balance via the release of the ‘silencing factor’ GABA. Understanding interneuron activities is crucial as an imbalance in E/I could result in numerous brain illnesses.

INTRODUCTION

Have you ever had a thoroughly tiring day, so much so that you have been out like a light when your head hit the pillow? How the brain puts you to sleep has been a fascinating question that neuroscientists are now beginning to answer. As you get drowsy, the inhibitory interneurons (INs) take over and apply a break on the constant excitation occurring in your brain [1]. Mice that lost INs also lost their sleep!

Generally, neurons come in two major types, excitatory and inhibitory. Upon receiving a signal from the preceding neuron, an excitatory neuron fires up and the signal is passed down to the next neuron. Conversely, INs dampen excitatory neurons. In recent years, it has become clear that INs are the dark horses that control brain activity. Interestingly, INs are rare (they comprise only about 20% of the neuron population), which made it difficult to study them, but as you can imagine, their fewer numbers also hint at a more diverse portfolio of roles than the excitatory neurons.

GABAERGIC INTERNEURONS ARE THE CLASSIC PARTY POOPERS

Typically, neurons communicate amongst themselves via chemical messengers known as neurotransmitters. GABA is one such messenger used by INs to counteract the effect of excitation. GABA is released from one neuron’s pre-synaptic end and binds via a synaptic cleft to the membrane of another neuron’s post-synaptic end, eventually preventing excitatory signal transmission. Throughout the brain, there are many sizes and forms of inhibitory interneurons. GABAergic interneuron axons can split into many filaments and wrap around the dendrites (axodendritic synapse) of many excitatory neurons (Fig. 1). A single IN can coordinate and regulate the firing of hundreds, even thousands of excitatory neurons, precisely timing their on/off states which are essential for normal functioning of the brain. Thus, considering the complex structure of GABAergic interneurons it is inevitable that they need a constant energy supply towards their distal ends.

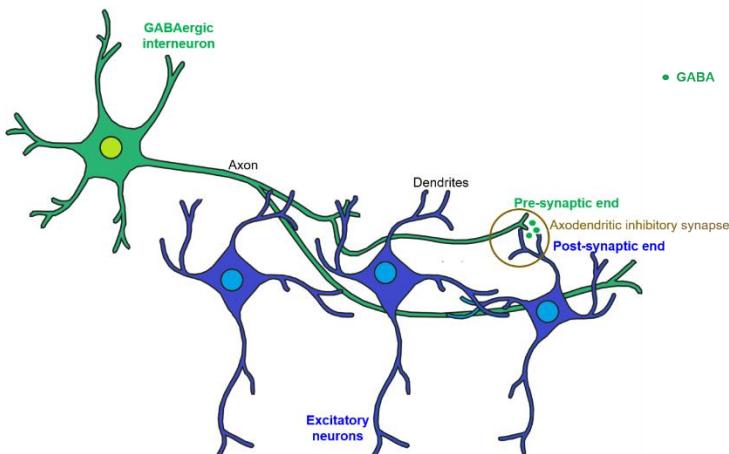


Figure 1. The interneuron-excitatory neuron connection.

This illustration depicts the complexity of the inhibitory interneuron and how it wraps around the dendrites of many excitatory neurons.

INTERNEURONS DEMAND HIGH ENERGY LEVELS!

The firing of neurons accounts for around 25% of the total energy consumption in the brain and synaptic transmission consumes the rest of the energy. One of the major pathways for energy production is glycolysis. However, the glycolytic capacity of INs is restricted; therefore energy produced by mitochondria is essential for inter-neuronal ATP supply.

Mitochondrial trafficking in INs is achieved by a set of proteins that involve anchoring proteins (Miro1/2), adaptor proteins (Trak1/2) and, motor proteins (Kinesin/Dynein). This complex machinery is the lorry that transports the cargo (mitochondrion) towards distal regions of interneurons along the microtubular track, which acts as a road (Fig. 2). This road has directionality, with the minus end pointing at the cell body and the plus end pointing at the synapse. In the figure, the mitochondria are being trafficked towards the plus end where they act as a source of energy and when these mitochondria are irreversibly damaged, they are trafficked back to the minus end for degradation [2].

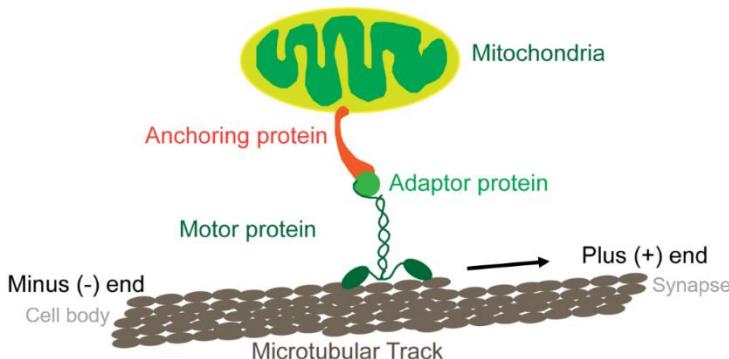


Figure 2. Mitochondrial trafficking.

The mitochondrion is represented in association with the motor protein machinery, which is transporting it towards the plus (+) ended synapse.

DISEASES RESULTING DUE TO MALFUNCTIONING INs

How deadly are faulty INs? Disruption of IN growth, function or metabolism can interfere with brain functions leading to disorders such as schizophrenia, autism spectrum disorder (ASD), epilepsy, bipolar disorder (BPD), various neurodegenerative changes, intellectual disabilities, and even aging-related cognitive changes.

ASDs are caused by disruption of the excitation inhibition (E/I) balance regulated by interneurons. Epilepsy is the result of growth and metabolism imbalance of interneurons in the cerebral cortex. Such an imbalance also causes the appearance of several other neuropsychiatric illnesses, mostly characterized by cognitive impairment. Other neurological diseases including Angelman's syndrome, fragile X syndrome, and neurofibromatosis type I are also associated with abnormal GABAergic interneuron functioning.

DRUGS THAT COMPENSATE FOR POORLY FUNCTIONING INs

So, what can be done to counter these illnesses that are a result of constantly active excitatory neurons? When something is ruined, man hastens to find a cure. Scientists have developed drugs that help in reigning control over the excited neurons. These medical interventions are generally of two types: drugs that block the gates (channels) that excite the neurons and drugs that improve the transport of the silencing factor GABA to excited neurons.

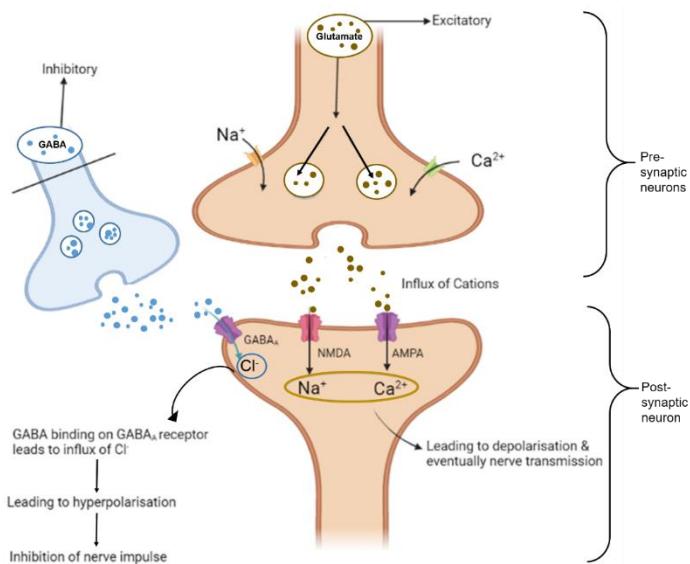


Figure 3. Action at the nerve synapse.

This illustration depicts the two types of pre-synaptic neurons; excitatory (brown) and inhibitory (blue). Glutamate excites the neurons by the influx of ions like Na^+ and Ca^{2+} into the post-synaptic neuron via NMDA and AMPA channels. Whereas, GABA inhibits nerve impulses by increasing the Cl^- activity in the post-synaptic neuron via the GABA_A channel.

Channels on neurons resemble the gates to a town. They allow the movement of ions such as sodium (Na^+) into the neuron. These ions act as currency, that is, they have the potential to excite neurons (Fig. 3). Therefore, as you can imagine, the actions of channels (how open or how close they are) are crucial to protect the neuron. A channel that is open for long periods (possibly due to malfunctioning interneurons) will bring in vast numbers of ions, resulting in prolonged excitation, and this could be a symptom of over 30 neurological diseases! Thus, blocking neuronal channels is a powerful therapy among patients. Phenytoin is an example of a sodium channel-blocking drug commonly used in epilepsy. Interestingly, channel-blocking drugs may just live up to their name and merely jam the movement of particular ions or they could have additional superpowers and combine other functions (such as spacing out the excitation of neurons), making them formidable enemies of neurological diseases [3].

As we learned earlier, binding of GABA to specific sites (GABA_A) on the receiving neuron results in the dampening of neuron excitation. Once GABA is bound to GABA_A, the neuron opens a channel that allows chloride ions (Cl^-) to pass inside it (Fig. 3). It is these negative chloride ions that make the neuron less responsive to other neurotransmitters that would normally excite it. Drugs such as benzodiazepines bind parts of GABA_A. A combination of benzodiazepine and GABA at these sites acts as a booster, allowing more chloride ions to enter the neuron, making such neurons even more resistant to excitation. Other drugs such

as barbiturates keep the GABA_A channel open for extended times, eventually increasing the movement of chloride ions into the neuron and decreasing excitation.

Another mechanism that excites neurons is the excessive binding of neurotransmitter glutamate to NMDA sites, which increases the frequency of calcium (Ca^{2+}) ions entering the neuron and resulting in fast or long-lasting activation (Fig. 3). Drugs such as felbamate block the NMDA sites preventing binding of glutamate and thus inhibiting neuron excitation [4].

DO NOT IGNORE YOUR INTERNEURONS

Finally, you may have realized, neurons power our body to perform every activity, be it playing football, watching movies, or the most obvious- recalling answers in exams. These activities involve not only the firing of neurons but also their eventual silencing. The uncontrolled firing of neurons could result in many brain disorders; you may lose sleep or worse, even forget your name! Thus, we hope to have convinced you of the critical role that inhibitory interneurons play in fine-tuning neuronal firing. But, this silencing comes at the expense of massive energy. Today, several drugs have been proposed to take care of faulty interneurons, however, due to their addictive nature it is advised to proceed with caution.

Glossary

GABA: Gamma-aminobutyric acid, the neurotransmitter (or messenger molecule) produced by interneurons.

Glycolysis: It is a linear pathway that operates in every cell of our body to break down one molecule of glucose into two high-energy molecules known as ATP

ATP: adenosine triphosphate is the foremost energy currency of the cell.

NMDA and AMPA: N-methyl-D-aspartate and α -amino-3-hydroxy-5-methyl-4-isoxazole propionic acid. These are gate-keepers (or channels) found on most excitatory neurons, where they respond to the excitatory neurotransmitter glutamate.

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Splitting Open the Brain

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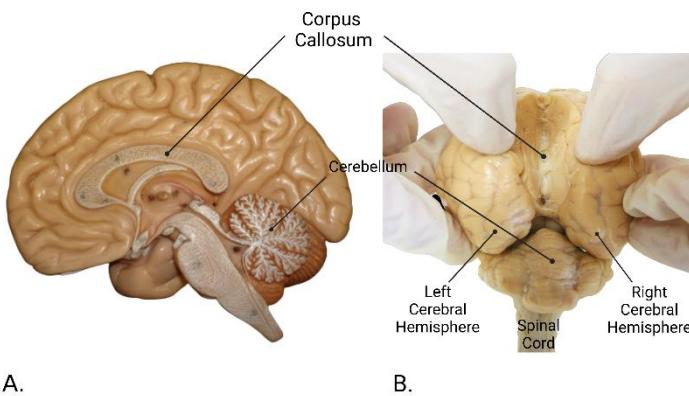
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SUMMARY

Our brain is a single unit that is further made up of two separate units: the left, and the right hemispheres. Scientists with help of different methods and techniques have proved that these two sides of the brain have their own separate cognitions. They share information with each other resulting in an integrated perception. But what if the two sides are unable to reach out to each other, how will that affect our cognition?

INTRODUCTION

Our brain is a 2-in-1 deal which means we have one brain but it is comprised of the twins, the left and right cerebral hemispheres. These hemispheres are connected to the contralateral (opposite) sides of the body, which means the left cerebral hemisphere controls the right side of the body while the right hemisphere controls the left side of the body [1]. These hemispheres are not identical and differ from each other in size, structure, and functions.



A.

B.

Fig1 Corpus Callosum **A)** sagittal section of the brain showing Corpus Callosum. **B)**

Posterior view of the brain showing Corpus Callosum connecting the two hemispheres.

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Though the hemispheres are separate from each other, they exchange information with each other through the *corpus callosum (CC)* (fig1), a bundle of axons located between the two hemispheres which connect the two of them. Other communication channels between the two hemispheres include *anterior and posterior commissure* and *optic chiasm* [1]. At the optic chiasm, half of the nerve fibres exiting from the eye project to the opposite hemisphere,

fetching information to both halves (fig2) [3]. We can say that one hemisphere sees one side of the world while the other sees the other side of the world.

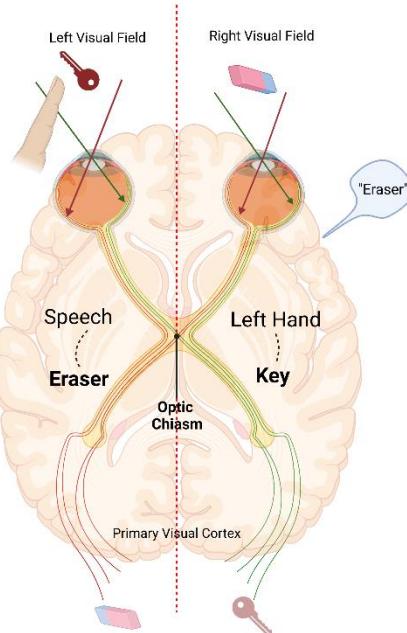


Fig 2: The Divided Field Experiment

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This means that both hemispheres have their own information processing system and speciality. In scientific terms this remarkable property of the brain is called "**Brain Lateralization**".

DISCOVERY

Marc Dax (1771-1837), a French neurologist, was the one who, while examining patients with brain damage due to stroke, tumour, etc. noticed that when the left brain was damaged, the right side of the patient's body used to get paralyzed, which also caused them to lose their speaking capacity. This was so strikingly consistent that out of the 40 or so brain-damaged patients with speech problems whom Dax had seen during his career, not a single one had damage restricted to the right hemisphere[4]. Looking at this link Dax reasoned that the left hemisphere might be the special region for speech production (Rogers, 2003).

Later in 1960, neurologists Robert Sperry and Ronald Myers conducted a series of experiments on cats by completely dissecting their CC and optic chiasm both of which bridge the two lobes and are critical for integrating information. In the first phase of the experiment,

the cats were trained on a visual discrimination task with a patch on one eye. They found that cats could successfully learn and perform the task which indicates one hemisphere is enough for learning any task independently and as rapidly as the two can learn together [1].

In the second phase of the experiment, the patches were shifted to the other eye and the cats were made to perform the same task. Surprisingly they found that the performance of the cats dropped significantly and they had to relearn the task. Looking at these results Myers and Sperry concluded that our hemispheres can function as separate brains and the function of CC is to transfer information from one hemisphere to another [1].

Sperry, the pioneer in medical science described in his words, “... *each hemisphere seems to have its own separate and private sensations; its perceptions; its concepts; and its impulses to act, with related volitional, cognitive, and learning experiences*” [6].

Sperry was awarded the **Nobel Prize** in Physiology or Medicine in 1981 for this groundbreaking split-brain discovery concerning the functional specialization of the cerebral hemispheres.

BEHAVIOURAL LATERALIZATION

As seen previously, although most language capabilities seem to be specialized in the left hemisphere, emotional information processing appears to be right-lateralized. This evidence was found in observing patients with damage in the right hemisphere who were unable to recognize emotions implicit in a speech by paying attention to nonlinguistic components such as tone i.e., anger, surprise, happiness, etc. which is known as “*Emotional Prosody*”. They were able to comprehend the sentences spoken but were unable to interpret the meaning when emotional components were involved [2].

These specializations come into existence as our brain develops depending on how they are getting used. Anatomical differences in neurological circuits of CC are linked to handedness, gender, retardation, autism, etc. For example, in males the right hemisphere, and in females the left hemisphere is more bulging which as a result shapes their unique cognitive styles [7]. The physical properties of the callosal axons determine the callosal size, shape, and connections to the structural organization of the brain [3] and any defects in those could cause brain dysfunctions, and malfunctions in the behaviour.

One such interesting case is Kim Peek, who had savant syndrome. An MRI examination showed an absence of CC and anterior and posterior commissures in Kim's brain. Kim's condition is known as “Agenesis of Corpus Callosum”. No interconnection between both hemispheres allowed Kim to read two open pages of a book simultaneously in a way that he could retain all the information. It has been speculated that the hypoconnectivity between the two hemispheres promotes attention to detail, greater focus, and a need for routine and repetition. He was a walking encyclopedia and had extraordinary memory [9]. Because of his extraordinary abilities, his friends called him “Kim-Puter”. Kim had developed language centres in both hemispheres YES, when the required brain structure for a particular neurological function is not available, the adjacent areas substitute for that unavailability due to our brain's magnificent ability to change and modify itself according to its needs, which is known as neuroplasticity [10].

METHODS TO STUDY BRAIN LATERALIZATION

Sodium Amytal Test

In the sodium amytal test or **intracarotid sodium amobarbital procedure (ISAP)** (also named as **Wada test** named after neurologist J. A. Wada), sodium amytal is injected into the left or right internal carotid artery. This works as an anaesthetic and inhibits any activity in the hemisphere for a short duration and that way it can be tested what brain hemisphere is responsible for which particular cognitive ability [1].

When the left hemisphere was inhibited and some object was presented to the subjects in the left hemifield which takes information to the right hemisphere when asked what they saw subjects could not report the object they saw but could indicate with their finger when given a list of objects.

Dichotic listening task

Two competing messages are presented to both ears. The subject has to verbally repeat those messages. When asked to repeat as many words as possible the subject mainly could reproduce information presented to the right ear, an effect called the *right ear advantage* which translates to left hemisphere dominance for language. The ipsilateral projection from each ear seems to be suppressed when info from the contralateral pathway from the other ear is coming.

Cutting the corpus callosum: Split-brain studies

In an attempt to resolve epileptic grand mal seizures, the idea of splitting the CC came forward, which yielded promising results in epilepsy patients who after surgery exhibited controlled seizures without any side effects [11] but had shown dysfunctional changes in cerebral functioning in cats and primates. Seizures were completely gone after surgery with no other brain dysfunction [12]. But disturbed brain functions of animals but not humans were surprising to researchers. They devised a new method of testing, ‘the Divided Field Experiment’ to see the effect of brain split based on the anatomy of the optic nerve, where each hemisphere requires information from the other to function well; this seemed a promising approach to investigate the outcomes of severing the CC.

When tested, the split-brain patients could recognize and name the objects which were placed before them, but when presented with an object in each hemifield separately they were unable to verbally report the objects presented in the left visual hemifield since the region specialized for speech generation resides in the left hemisphere, unable to access the information from the left eye field which goes to the right hemisphere, but could report the presence of the object in the left visual field using his left hand which is controlled by the right hemisphere itself (fig 2). Visual information obtained by one hemisphere was not accessible to the other [3]. These studies were a rich source of investigation in lateralization research.

CONCLUSION

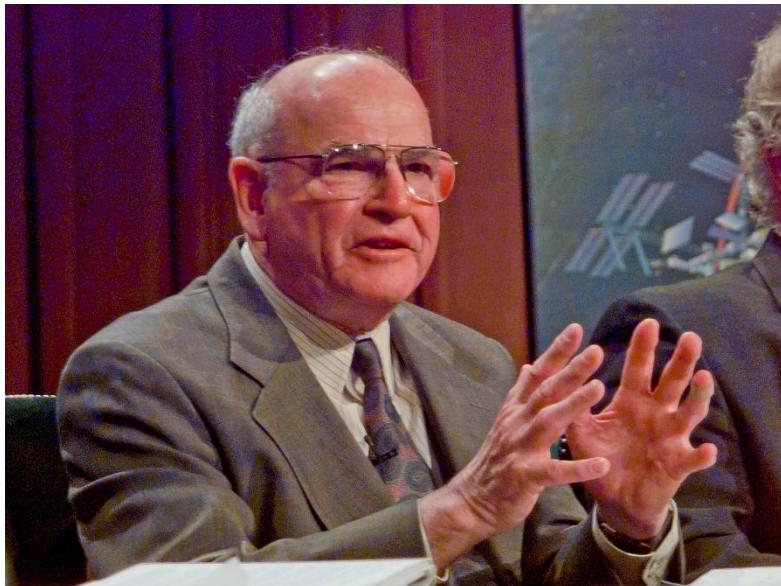
To sum up, the two halves of the brain have their specializations and they interact with each other for coordination and efficient cognition where each hemisphere in its special way contributes to performing any task [14]. The left brain is more logical, detail-oriented, analytic, and mathematical while the right hemisphere of course is found to be non-verbal, mathematical, spatial, imaginative, artistic, and with holistic perception.

Looking at numerous studies scientists have concluded that having lateralized functions in the brain is advantageous in the way that it increases our brain capacity and efficiency, as the same circuits don't have to be duplicated in each hemisphere instead the two brains have their own specialized circuits and functions [14].

Fun Fact: Generally, our (90% of the population) right hand is more dominant than the left and that's why our ancestors used to superstitiously consider it auspicious to start every task using our right hand.

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Baruch Samuel Blumberg

(Nobel Prize in Physiology or Medicine, 1976 for discovering
“origin and dissemination of infectious diseases”)

PART II

Diseases

***Naegleria fowleri*- The 'Brain Eater'**

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SUMMARY

Naegleria fowleri, also known as 'The Brain Eater', is a free-living, aquatic amoeba that lives in relatively hot environments and causes deadly Primary Amoebic Meningoencephalitis (PAM) if accidentally introduced into the human body. What makes it interesting is its attraction to the neurotransmitter acetylcholine and how it escapes our immune system to cause the deadly disease.

It was the summer of 2018. 17-year-old Rama went to his grandmother's home in Ernakulam, Kerala to spend his summer vacation. His grandmother's home was nothing less than a paradise for him- the beach at a stone's throw and a pool in the backyard. Most of his memories were associated with the pool where he first learned to swim and dive. It was his favorite place. After reaching his grandmother's home, he just dived into the pool and swam for hours. Little did he know that he will battle for his life in the upcoming week.

3 days after that dive, he suffered from a high fever, headache, and a stiff neck. The condition worsened and was accompanied by vomiting and seizures. He was admitted to an ICU. The doctors somehow managed to stabilize his condition, but still, they were worried that he may develop brain damage and inflammation. He was battling between life and death. The doctors diagnosed that he was suffering from a disease known as Primary Amoebic Meningoencephalitis which is caused by a deadly, pathogenic, water-borne amoeba known as 'Naegleria fowleri'.

INTRODUCTION

Naegleria fowleri (*N. fowleri*), also known as the 'Brain-Eating Amoeba' or the 'Zombie Amoeba', is a free-living, pathogenic amoeba that causes Primary Amoebic Meningoencephalitis [Inflammation of the meninges (coverings) of the brain and spinal cord (a.k.a PAM)] in humans which is a rare but deadly disease with approximately 95-99% mortality rate. PAM is a waterborne disease generally caused by diving and swimming in non-chlorinated places like natural ponds and lakes. It is primarily caused due to the forced entry of water carrying *N. fowleri* into the nasal cavity while diving and swimming [1]. See Fig 1. for the microscopic images of *N. fowleri*.

N. fowleri belongs to the Heterolobosea class, was named by Malcolm Fowler of Adelaide Children Hospital, Australia, and was first described by R.F. Carter. It is a free-living amoeba that generally doesn't attack humans but becomes a deadly pathogen when accidentally introduced into the human body. Being a heat-loving i.e. thermophilic amoeba, the human body provides an ideal temperature for it to live and thrive. Then the question that arises is why it doesn't live in our body without harming it. This can be attributed to its pathogenic nature due to the:

1. Presence of sucker-like structures present on its body known as ‘food cups’. These can be imagined as giant mouths with large sucker teeth.
2. Releasing of different cytolytic (cell destroying) molecules primarily responsible for brain damage occurring in PAM.
3. It’s love for Acetylcholine, which is present in abundance in specific regions of our brain and that’s why it attacks the brain and not other parts of the body [1].



Figure 1. Different Stages of *N. fowleri* under microscope: trophozoite, flagellate and cyst

Image credit: USCDC, Public domain, via Wikimedia Commons

Acetylcholine is a small molecule in our body that acts as a ‘neurotransmitter’ i.e. transmits messages from one place to another in our body. It is synthesized in the terminals of a brain cell or neuron with the help of the Acetylcholinesterase enzyme and after release, transmits signals to another neuron by binding to its surface through large proteins called G-protein coupled receptors. The reason *N. fowleri* is drawn to acetylcholine is that it also possesses receptors [G protein-coupled receptor] similar to Acetylcholine receptors present in humans [Human M1 muscarinic receptor subtype (mAChR1)]. Acetylcholine acts as a ‘chemoattractant’ to *N. fowleri* when it enters our body. This attracts *N. fowleri* towards olfactory (smell sensing) regions of the brain where Acetylcholine is released in plenty [1].

IMMUNOLOGICAL RESPONSE

When *N. fowleri* infects the brain, the first cells that arrive at the site of infection and react against the infection are Polymorphonuclear Leucocytes (PMNL). These cells are a type of white blood cells that respond to the infection. They release Proteolytic Enzymes i.e. Protein Breaking Enzymes, Antimicrobial Peptides which are small protein molecules that are toxic to microbes, Reactive Oxygen Species (ROS) and, Reactive Nitrogen Species (RNS) which are by-products of oxygen and nitrogen metabolism in our body and are toxic in high concentrations. All these chemical agents are released to ensure the killing of invading microbes. After these cells, Microglial Cells which are resident macrophages of the brain, come into action. These cells are ‘policemen’ residing in the brain to counter these types of infections and infectious agents if they attack the brain. These Microglial Cells secrete amoebicidal molecules which provide primary defense against invasion of the Central

Nervous System. At the later stages of infection, other immune cells like Neutrophils and Eosinophils surround *N. fowleri* to counter the infection [2].

*But then the question arises is how *N. fowleri* succeeds in evading such a great defence mechanism.* *N. fowleri* has various mechanisms to evade the immune system and successfully establish an infection in the brain which are discussed as under:

1. It secretes Matrix Metalloproteins (MMPs) that degrade connection between host cells i.e. cell junctions in between the brain cells that help its invasion.
2. It internalizes the antibodies attached to its body surface which makes it harder for the immune system to recognize and act on it. This is similar to a thief disguising by shaving off their beard and head.
3. It secretes proteins like Phospholipases, Cysteine Proteases [also known as 'Caspases' which are notoriously famous for causing programmed cell death i.e. Apoptosis in host cells], Neuraminidases, and Acid Hydrolases.
4. It can induce the formation of more and more Reactive Oxygen Species (ROS) in host cells that can ultimately harm the host cells.
5. It also has a protein on its body surface known as N-PFP which is a kind of pore-forming protein that destroys the cell membrane of host cells and kills them.

N. fowleri also has different modes of destruction of host cells based on the type of strain. If a weakly pathogenic or infective strain attacks the brain, it causes destruction by ingesting the host cells with the help of 'food cups' but if a highly pathogenic strain attacks the brain, it lyses or breaks nerve cells on contact and subsequently ingests the cell contents [3].

*So, should you worry about being infected with *N. fowleri* and stop going outside?* In a general sense, NO. As referred by Capewell et.al, only 142 patients with PAM were reported in the United States of America between 1987 and 2013. Therefore, the caseload of PAM is very less in comparison to the other pandemics world is facing today (for ex.: CoVID-19) [4]. People die more from drowning than from catching this dangerous disease as estimated by WHO [5]. In addition to this, clinicians and researchers keep coming up with better ways to save from such a deadly infection.

Fortunately for Rama, he received excellent intensive care and was given a combination of antibiotics and antifungals like Amphotericin B, Rifampicin, and Ornidazole early in his treatment [6]. He successfully recovered from the infection and returned home after being in the ICU and later in the general ward for several days. After this incident, his grandmother promptly warned everyone in her family and neighborhood to be wary of using the natural ponds for swimming. They also informed their local municipality about this issue and requested they take precautionary steps.

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Neurogenetics of Autism Spectrum Disorder

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SUMMARY

Many of us have heard the term ‘autism’; or ‘Autism Spectrum Disorder (ASD)’ in news articles, science textbooks or other forms of media. But what exactly does ASD mean? This article presents a fictional case study of Harry, a child showing symptoms of ASD, and explores the science underlying this condition. It focuses on the genetics of ASD and discusses our current understanding of the role of various genes and genetic variations contributing to ASD.

INTRODUCTION

Harry Robert is a physically healthy four-year-old boy; however certain behavioural traits set him apart from other kids of his age. He has difficulty in social communication and interaction with others. He avoids eye contact, and his face hardly shows any expression of emotions like anger, sadness or happiness. He shows repetitive behaviour like repeating the exact phrases and obsessive behaviour like being fixated on specific parts of his toys or arranging his toys in a highly specific order. His parents believed that he would be more like the other kids if he started interacting with his peers, and enrolled him in a preschool. However, his uncommon behaviours continued. Harry’s school teacher who is trained in understanding child behaviour suggested to Harry’s parents that they should consult a paediatrician.

VISIT TO THE PAEDIATRICIAN:

Dr George: "Come in, Mr and Mrs Robert. Have a seat, Harry. What seems to be troubling Harry?"

Mrs Robert: "Well, doctor, I am worried; he isn't very expressive and is hesitant to talk to or interact with others. He doesn't seem to be hitting his developmental milestones as expected. I am perplexed about what's wrong with him."

(Mrs. Robert further explained how he responds to her actions and words in more detail. While Harry sat quietly gazing out of the window Dr George paid attention to his gestures and expressions; Harry’s father looked at his wife and son unhappily; he appeared pensive.)

Dr George: "Would you like to add anything, Mr Robert?"

Mr Robert: "Doctor, one of Harry’s cousins has been diagnosed with autism, so I’ve been reading up on autism spectrum disorder. Harry’s symptoms seem to be somewhat matching with that. Is it at all possible that this condition is genetic?"

Dr George: First, we have to run certain tests before knowing for sure Harry’s condition is ASD. But to answer your question, yes, genetic factors are known to contribute to ASD. Autism is generally revealed early in life. The probability of having autism is much higher in the case of identical twins than in non-identical twins (Rylaarsdam & Guemez-Gamboa, 2019)[1]. Although there are many advancements in this field, we currently know only some

of the genetic variations that contribute to this spectrum of symptoms. (Mehta & Golshani, 2013)[2]. Since genes in our body carry information for making proteins, which carry out important functions in the body, many researchers are trying to identify proteins that may be malfunctioning in autistic children.

Mrs Robert: "So, are bad genes the prime cause of autism?"

Dr George: "Not exactly. Environmental factors are also suspected to contribute to ASD, and our knowledge is far from complete in this aspect (Karahmadi, Karimi, Kamali and Mousavi, 2017)[3]."

Mr Robert: Doctor, why is autism called a 'spectrum disorder'?

Dr George: Because there is individual-to-individual variation in the range of behavioural symptoms, and the degree to which each of these traits manifests also varies over a spectrum! Within ASD there are distinct sets of symptoms, and some of the conditions categorised under ASD include Autistic Disorder, Childhood Disintegrative Disorder, and Asperger's disorder (Rylaarsdam & Guemez-Gamboa, 2019)[1].

Mr Robert: In movies that show characters with ASD, they are depicted to have some exceptional abilities in math, music or art. Is it always true?

Dr George: There are a few cases where people diagnosed with ASD also have prodigious talents. It's called Savant syndrome. Although they have severe mental handicaps overall, they show extraordinary skills in certain domains. (Treffet, 2009)[4]

Mrs Robert: Doctor, what are the tests you mentioned for Harry, and how do we go about them?

Dr George: Firstly, let us run a set of behavioural tests, and if they indicate Harry's condition to be ASD, then I'll suggest a paediatric neurologist who will examine his condition in detail and prescribe management and treatment procedures appropriate for his specific spectrum of symptoms. I'll also share some informative pamphlets on ASD, with you.

Mrs Robert: Thank you, doctor. I just want him to live a normal, happy life.

ROBERTS LEARNS ABOUT AUTISM SPECTRUM DISORDER

Harry was administered a physical exam along with blood tests, hearing tests and tests for coordination and cognition. Following these test results, they were recommended to meet a paediatric neurologist, Dr Mike, to further discuss the boy's course of treatment. He also had to undergo a brain scan in the form of an MRI (Magnetic Resonance Imaging) and an EEG (Electroencephalogram) to study his brain activity. Dr Mike provided them with more information about the cause of ASD at the cellular and molecular levels.

Dr Mike: Autism Spectrum Disorder is a set of neurodevelopmental disorders that cause some characteristic deviations in growth and behaviour. As ASDs are highly heritable, it is necessary to learn about the gene expression and the genetic testing used to study the spectrum of disorders in autism, which are autistic disorder, childhood disintegrative disorder, and Asperger's disorder (Rylaarsdam & Guemez-Gamboa, 2019)[1]. Scientists have identified a

few sets of distinct gene sequence variations that are associated with each of the known ASD conditions. However, the primary reason underlying the ASDs (if any) is elusive. If such a genetic factor is known, that could serve as a potential target for treatment. (Hodges et al., 2020)[5].

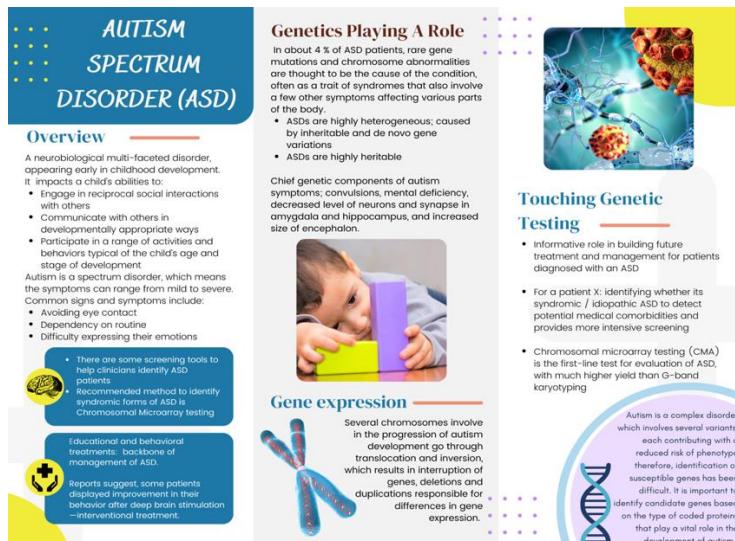


Figure 1: Pamphlet describing the genetic aspects of ASD.

This image was created using Canva (<https://www.canva.com/>)

Mr Robert: Doctor, I am curious to know about the genes that are said to be associated with ASD. What scientific methods and tools do scientists use for such research?

Dr Mike: One of the most widely used methods to study the role of genetics in a certain condition is twin studies. The basic idea behind twin studies lies in the fact that identical twins will have the same set of genes, so any difference seen between them is likely to be caused by environmental factors. Researchers at the Developmental Disability Centre at Washington University conducted a twin study which included both identical and non-identical twins, where eye movement data was collected from non-autistic and autistic toddlers (Constantine, Kennon-McGill et.al, 2017)[6]. They were randomly shown videos of different social situations, and their eye movements were tracked. This was done based on previous research that found that kids with autism tend to avoid or minimize eye contact during social interaction. In this study, it was found that autistic toddlers show reduced attention to eye and mouth regions as compared to non-autistic children. Moreover, identical twins have a striking similarity in eye movement, which suggests a high genetic component to this trait.

While twin studies help us understand how strongly genes influence a trait, they provide little information about the genes involved. Thus, another important tool used in genetics to tie the genotype or the genetic component to the phenotype or the physical characteristics is Linkage analysis. In a nutshell, Linkage analysis looks at the frequency with which a physical trait and the suspected genetic variation appear together across successive generations.

As you know, ASD is characterised by Repetitive and Restricted Behaviour (RRBs). A study published in Molecular Psychiatry in 2017 established an association between RRBs in ASD with a single letter change in the sequence of a gene known as the PHB (Cantor et.al, 2017)[7]. This was established using a technique known as Genome-Wide Association Studies (GWAS). Simply put, GWAS compares the genomes of individuals affected by a certain disease and looks for common patterns of genetic sequence variations or single-letter changes in the DNA. The genius of this approach lies in the fact that it involves looking at almost the complete DNA sequence of the individual rather than looking at certain candidate genes. The study found that mutations in two genes, PHB and SLC35B1, were associated with repetitive behaviour. These genes are expressed in the developing brain in a part called the thalamus, which is the body's relay centre.

Genetic variations include not only changes in a single building block of DNA (i.e. nucleotide) but also changes in the number of copies of certain genes and DNA segments. This is referred to as 'copy number variation (CNV)'. CNVs account for up to 10% of ASD cases! Further research on CNV regions in the genome will hopefully lead to the identification of candidate genes that may play a significant role in the pathophysiology of ASDs.

For molecular diagnostics, Exome Sequencing and Chromosomal microarray testing (CMA) are two useful techniques. In Exome Sequencing, specific candidate genes which are known to be associated with ASD are sequenced. Whereas CMA is used to find very minor variations in our genetic material (DNA) that could be the root of developmental or other health issues in children (Mehta & Golshani, 2013)[2]. CMA finds small deletions and duplications in CNVs.

With a disorder as complex as ASD, it is unrealistic to make any sense of genetic data unless it is coupled with other measures such as behavioural measures and tractography. Behavioural measurements are commonly in the form of tests or questionnaires that are scored on different parameters. These include measures for physical, psychosocial, and emotional well-being. Tractography includes looking at white matter tracts in the brain by using brain imaging techniques and figuring out how different brain regions are connected (Hrdlicka, et.al, 2019)[8]. It is much like looking at the inner electric wiring of a house if a certain appliance isn't working.

An interesting aspect of ASD inheritance is, it affects three times more males than females! This bias towards males might be the result of the under-recognition of females diagnosed with ASD. Alternatively, some experts claim that a female-specific protective effect against ASD might exist (Jack et al., 2021)[9].

Despite the heterogeneity of ASD symptoms and the root cause of these conditions, scientists are making steady progress in understanding the cause, the diagnosis of the condition, treatment and management methodologies.

EPILOGUE

From Harry's case, we can see how ASD can affect a person's ability to carry out day-to-day activities and communication. Dr Mike will prescribe a treatment plan customized for him and his family because each child has a unique presentation of the disease. This generally includes individual or group therapy, speech and language therapy, and training in social skills (Bagatell and Mason, 2015)[10]. The disease is difficult to identify as it presents itself on a spectrum with varying symptoms in different patients. While we do have some information on genetic variants associated with certain traits of the disorder, this is just the tip of the iceberg. The field of genetics requires motivated students like you to solve complex problems so that one day we might have more effective treatments for people on the autism spectrum. If you're passionate about neuroscience, here are a few places in India where you can start building your career in Science!

National Centre for Biological Sciences:

<https://www.ncbs.res.in/adbs/categories/neuroscience>

National Brain Research Centre:

<http://www.nbrc.ac.in/newweb/>

Tata Institute of Fundamental Research:

<https://www.tifr.res.in/~dbs/faculty.html>

Centre for Biological and Cognitive Sciences:

<http://cbc.ac.in/research/research-areas/>

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MS: Dialogues on Disease and Memoir of My Father

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SUMMARY

This article is about a son writing a memoir of his life with his father. His father was ailing with MS and through excerpts of his father's own memoirs we get to see the stories behind the History of MS, how in India the MRI helped break the myth that it was different in India, and the way scientists keep asking why and hopefully discover answers.

INTRODUCTION

I was filled with hesitation; the atmosphere was soggy and sad.

I asked my dad, overcoming my inhibitions, what is multiple sclerosis?

My mom turns around, looking at my head on the window of the car she asks How do you know that

I read the doctor's slip when dad got his eyes checked up, I said.

Okay.

So? What is it, I mustered sheepishly.

You will get to know soon, we will talk about this when we reach home, said my father. At home, the situation was calm and a sense of normality prevailed in the room.

So, Kabir, I will make this short, I was diagnosed with multiple sclerosis a few years back before I met your mother. MS is somewhat of a complicated disease, it's a neurodegenerative disease. Do you know what neurodegenerative diseases are?

No, I don't dad.

Do you know that our bodies are made up of cells?

Yes, dad and there are various types of them, skin cells, muscles, etc.

Yes good, so just like that there are neuronal cells, these special cells make up our brains. Without them, we would not be able to have this conversation, read, write, or even imagine. So now use your neurons and imagine a tree, just like a tree has a crown, trunk, and roots. Neurons have crown-like dendrites, a long trunk-like structure called an axon, and root-like axonal endings.

Now what you are probably imagining will be a simple picture of tree-like cells, but they are not so simple at all. They are numerous, connected to each other, and there are cells that provide support to them. To understand MS you need to know that there is a special covering that axons have, that is made up of fat and scientists call it myelin sheath (Fig.1).

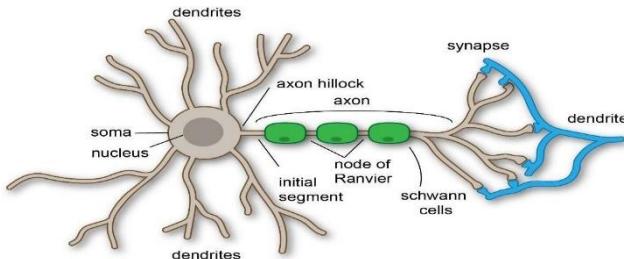


Figure1: Anatomy of Neuron

Image credit: Curtis Neveu

[https://commons.wikimedia.org/wiki/File:Anatomy_of_neuron.png#filehistory] licensed under the Creative Commons Attribution-Share Alike 4.0 International license.

Dad how many neurons are there, how are they connected, and why are they connected?

I don't think we have a specific answer as to how many neurons are there exactly, but it's close to around 85 billion of them packed inside your head.

I once read this rather poetic sentence on the internet, that we have a greater number of neurons in our heads than there are stars visible in the night sky.

It is an astronomical number.

Secondly, they are connected by what's called a synapse. Axon terminals of one neuron and dendrites of the next neuron are placed very close to one another, but they don't touch. There is a very small space between the two and a special substance called neurotransmitter goes from the axon terminals of one neuron through that space and binds to the dendrites of the next neuron. Then the same thing happens in the next and the next synapses, you can see how mind-boggling it gets.

No pun intended.

Yeah...it really feels big, yet it's happening in my brain, which is not so big. It's hard to think about this, I said. I was tired and feeling overwhelmed, my face would have said the same maybe that's why he went

Alright, stick with me kiddo for 5 more minutes, and then we will sleep.

You see what happens in MS is that immune cells in our bodies that fight harmful outer enemies like bacteria, viruses, and other disease-causing microbes, malfunction. We don't know how or why, but our immune cells start fighting a battle with the myelin sheath and axons of our neurons, damaging them badly.

You can imagine now that if neurons are hurt and killed what complications it would create. I once told you that we may take in the light from our eyes, but it is through the brain that we create this image of reality. The eyes may hold the colour, but it is the brain that paints.

Damaged neurons are why you are having eye problems, dad? Your immune system thinks there is an internal enemy this time, but why? How come we don't yet know why this happens?

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Nicely put Kabir. Yes, it's perplexing indeed. I too felt the same when I first learnt about this, but then you realize there are so many things we don't know the reason for, but yet they happen, we just never ask or we stop asking once we get no answers.

But you know some people don't stop. They know they will not find the answers, but they keep on trying small steps at a time and through those people's efforts, we know what we know about this disease. His face had a wide smile by now, this poetic train of thought had somehow connected to something deeper within him and with a happy face he uttered

Science is fun because it never stops asking why.

Hmm..., so dad what are scientists doing nowadays, and was there any Einstein-like scientist that worked on MS too? Is it because of him or her only that we know this much?

Great question Kabir. I will give you something that might satisfy and fuel your curiosity at the same time, but for now, let's sleep, I have a long day tomorrow, we can pass the whole night and more.

Just talking about MS

After your school, you can check up on the drawer, and we will talk about it later.

I paused for a moment, thinking about how I will write about that day in this memoir of mine, the flood of emotions that hits you, it is hard to then sit and write. I did what I did that day that helped me, walk.

Coming back to the table, I wrote all of this.

THE NEXT DAY

Feeling groggy?

Yes, mom didn't get a good sleep yesterday

Your dad too was feeling the same, okay go get ready, I have to go to my clinic early today.

School, friends, classes, the daydreams and the journey back home it feels like all of this went in a haze, time flew by, maybe it was the poor sleep, or maybe I was not able to focus because I was just daydreaming of getting home.

As I open the drawer a black diary was lying in it, on the first page it said, MS: a personal study.

I turned the page and saw it is divided into sections on "what", "why", "how", and "who"? of MS. I flipped through the "what" page, but it was filled with jargon-laden terms on types of MS

Relapsing MS, primary progressive MS, benign MS, etc. I flipped again, and a picture caught my eyes. I read further.

Jean Marie Charcot is credited with defining and naming MS, a French neurologist whose work spans wide, not only was he a professor and a scientist but also a talented artist [1,3].

He would work with his patients for years, gathering records of their symptoms and performing autopsies when they die. His pioneering method of following a group of patients for a long time leads him to make several contributions to the science of various

neurodegenerative diseases.

In 1868, he gathered an audience full of scientists and doctors and gave 3 landmark lectures on MS, due to these lectures' history remembers his name and the idea of a new disease he termed MS, before he specified MS it was considered to be a type of paraplegia an umbrella term in which many neurodegenerative disorders were placed, he drew the lines and separated MS as a different disease with its own characteristics and symptoms and gave the world the language to communicate and collaborate on MS.

Surprisingly, in the marginalia of the page, I read a side note-. He was not a great lecturer but used a variety of techniques to charm his audience, he would memorize his lectures for days only to then deliver them without any notes and made them look spontaneous.

Amused, I read ahead.

Saint Ludwina of Schiedam in Holland is considered not only the first ice skater but also to be the first case of MS recorded in history. In fact, her first symptom occurred when she was skating on a frozen lake, and suddenly she fell because of trouble balancing [1,3].

Her disease progressed with time, as is usually seen in patients.

Cruising along the lines, I realized that day because my father also wrote such a diary. It was when, I read about the case of Augustus d'este who was coming from a funeral when his vision got blurred, he thought it must be due to him trying to suppress the tear from coming, and from that day onward he kept a diary of his progressive disease. His detailed written experiences helped to characterize the disease.

In another entry, there is talk of a student of Charcot named Pierre Marie who is said to have pounded tables and claimed that MS was due to a viral infection and its vaccine would soon be developed. Now at that time vaccines were all the rage; Louis Pasteur was working on microbes and would create a rabies vaccine.

Does the idea of MS be a viral infection was due to the situation at the time or not we don't know, many studies further tried to find the cause but till now we haven't yet.

INDIAN MS AND MRI

It was thought MS is prominently a disease of the west, Indian population seems different in this regard, but as we developed and new technologies emerged, new truths surfaced [2].

But what is MRI? I thought, then the very next moment I typed on Google what is MRI's definition easy. MRI is magnetic resonance imaging; it is a technique where we use powerful magnets to create a magnetic field and using that we can create images of our brain.

Back to the diary, I read MRI transformed the field of MS because now you can see the brain and patients with MS have brains that are partially degenerated (Fig.2,3) and with time you can see the progressive loss of brain structures, this opened our eyes as to the progressive nature of MS before such dramatic representation scientists used to believe MS comes in phases and goes not that it is an ongoing process that slowly continuously happens every day.

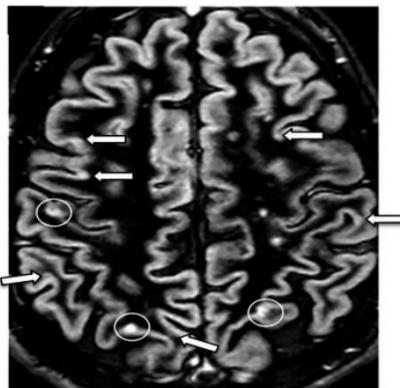


Figure 2: Axial DIR MRI of a brain with MS

Image credit: (2015). "The Parallel Analysis of Phase Sensitive Inversion Recovery (PSIR) and Double Inversion Recovery (DIR) Images Significantly Improves the Detection of Cortical Lesions in Multiple Sclerosis (MS) since Clinical Onset". *PLOS ONE* 10 (5): e0127805. DOI:10.1371/journal.pone.0127805
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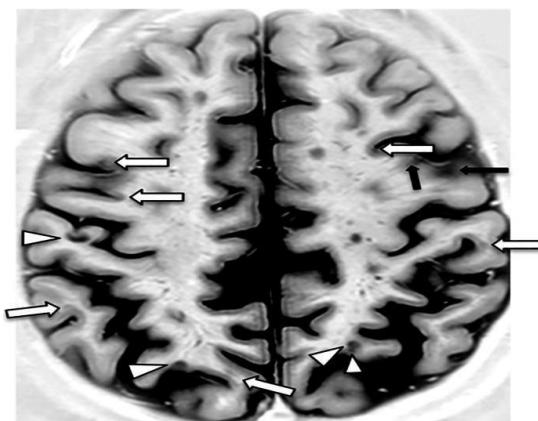


Figure 3: Axial PSIR MRI of a brain with multiple sclerosis lesions

Image credit: Alice Favaretto, Davide Poggiali, Andrea Lazzarotto, Giuseppe Rolma, Francesco Causin, Paolo Gallo (2015). "The Parallel Analysis of Phase Sensitive Inversion Recovery (PSIR) and Double Inversion Recovery (DIR) Images Significantly Improves the Detection of Cortical Lesions in Multiple Sclerosis (MS) since Clinical Onset". *PLOS ONE* 10 (5): e0127805.
https://commons.wikimedia.org/wiki/File:Axial_DIR_MRI_of_a_brain_with_multiple_sclerosis_lesions.jpg licensed under the Creative Commons Attribution-Share Alike 4.0 International license.

A pulse of anxiety ran through me, reading the diary, I realized how real this disease was, I thought at that moment, how my dad's brain must be going through a storm, a storm as big as Jupiter's great red spot erasing part of him every day, yet he smiled and hugged and wrote about his life following a tradition of memoirs, that people suffering from MS shared with us and the disease. He was my own Augustus d'este.

I couldn't read further that day, I was emotional, anxious, scared didn't know what to do, I stepped outside the sun was setting, and stripes of clouds were covering the blue sky. A walk, a sigh, that's how gained a new perspective that day that how messy, and fuzzy this existence is.

I was deep in my thoughts, I missed the voice of my mother, which was now getting louder and then it zapped my attention back to reality.

Kabir come home, Dad is back early, we were looking for you, said my mom, come let's talk at home and don't worry dear, it's alright, don't be sad.

My eyes were dried by then and I wanted to talk to him, ask him how was he. It amazes me how our brain develops, I was there all along but still, it was on this day, I felt this crazy weight on my head, that yes, my father was not in good health, the disease was growing and I didn't know how much time I have left with him.

I asked "Hey dad, how do you feel, I didn't know so much was happening".

Kabira, I know it feels heavy, but it is a quirk of life, we all will have some of it.

And don't you worry, medications help me, you are there, your mom she is such a force. You learn to make peace with it.

I remember in his characteristic poetic sense that I still try to embody in my life he said to me It is important from time to time to let go of what we hold and flow.

You know there was a person named Bruce Frederick Cummings, he died at the age of 30 of MS, he wrote a book that helped me a lot. Journal of a Disappointed Man.

Now I don't wish to be disappointed or angry, I give my all to enjoy this moment, no matter what.

I hugged him tightly and made a note in my mind to be always like that, equanimous and happy, facing life's quirks with a gentle heart.

Writing this Meta-memoir of my life with him something funny happened, news broke that scientist finally have the answer to what causes MS, it is a virus called Epstein Barr virus. I smiled, guess the table-pounding guy was really onto something.

I thought about my dad, and what he would feel, really missing his smile and poetic sense.

Miss you, Dad.

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Stroke and NMDA receptors in the Brain

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SUMMARY

A stroke occurs when blood flow to any region of the brain stops for a few minutes until the neurons start dying. The sensitivity to blood loss is due to the absence of the capacity of neurons to store energy inside them, hence they require a constant supply of oxygenated blood. The damage due to loss of energy leads to an ionic imbalance in neurons, which leads to glutamate spillover around the synapse. The spillover causes activation of extrasynaptic NMDA receptors, which promotes cell death signalling pathways in neurons, which leads to further neuronal damage. Currently, we are exploring potential neuroprotective agents for stroke that interact with NMDA signalling pathways.

INTRODUCTION

Stroke is the second leading cause of death worldwide, responsible for approximately 11% of total deaths globally (WHO, 2019), thus being an important cause of morbidity and mortality. Given its relevance, we should know the pathophysiological process behind a stroke and how the neuroscience behind its damage can help us find new avenues for its treatment.

After an acute stroke, the sudden reduction in cerebral blood flow causes the death of neurons. This is followed by ion transport protein dysfunction and disruption of ion homeostasis, which in turn leads to impaired glutamate release, reuptake, and excessive N-methyl-D-aspartate receptor (NMDAR) activation, further promoting neuronal death. Excessive NMDAR stimulation during a stroke is a central step in post-stroke damage (which is also known as excitotoxicity), but only some NMDARs based on their location are found to be the bad guys during a stroke.

Here, we will discuss more about the NMDA receptors and how we differentiate the good NMDARs from the bad ones.

WHAT IS A STROKE?

A stroke, also known as a cerebrovascular accident, is a clinical definition and is defined as an abrupt onset of a neurologic deficit that is attributable to a focal vascular cause [1]. Laboratory studies like brain imaging are used to support the diagnosis, and ischemia refers to a reduction in blood flow.

Once blood flow is cut off, by a blood clot, symptoms can manifest within seconds. This is because neurons don't have a way to store energy and need a continuous supply of blood. If the cessation of flow lasts for more than a few minutes, infarction or death of brain tissue results. But if blood flow is quickly restored before the death of neurons occurs, brain tissue can recover fully and the patient's symptoms are only transient: this is called a transient ischemic attack (TIA) [1].

If the blood flow does not recover in time, the patient is said to have had a stroke.

There are 2 types of strokes, ischemic stroke, and hemorrhagic stroke (Fig. 1). Ischemic stroke comprises 85% of all strokes, and it happens when the brain's blood vessels become narrowed or blocked, causing severely reduced blood flow. Hemorrhagic stroke comprises 15% of strokes and occurs when a blood vessel in the brain leaks [2], with each having different treatment but with some overlapping signs and symptoms. Symptoms of stroke depend on the region of the brain affected and can help a neurologist locate the affected region. To name a few, the symptoms can be a weakness, loss of sensation, loss of speech, loss of vision, memory loss, loss of cognitive abilities, loss of balance, etc.

To further our discussion on NMDAR and how it relates to stroke, let us look briefly at the physiology of a synapse, the space between neurons where they send messages between each other, and its associated structures.

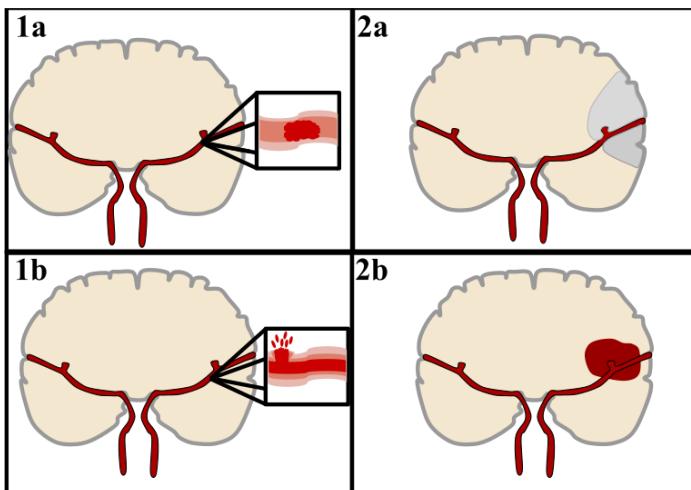


Figure 1. The two main categories of strokes. Ischemic (top), is caused by a blood clot in an artery (1a) resulting in brain death in the affected area (2a). Hemorrhagic (bottom), is caused by blood leaking into or around the brain from a ruptured blood vessel (1b) reducing blood flow in the artery and allowing blood to pool in the affected area (2b) thus increasing the pressure on the brain.

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CHEMICAL SYNAPSE, NEUROTRANSMITTERS, AND NEURORECEPTORS

Neurons communicate with each other at sites called synapses. These can be of two types, electrical synapse and chemical synapse. Electrical synapses are employed primarily to send rapid and stereotyped depolarizing signals. In contrast, chemical synapses are capable of more variable signalling and thus can produce more complex interactions. It can be either excitatory or inhibitory to the next neuron [3]. Because we will be discussing NMDAR, which falls under the chemical synaptic transmission, we will talk about the chemical synapse (Fig. 2).

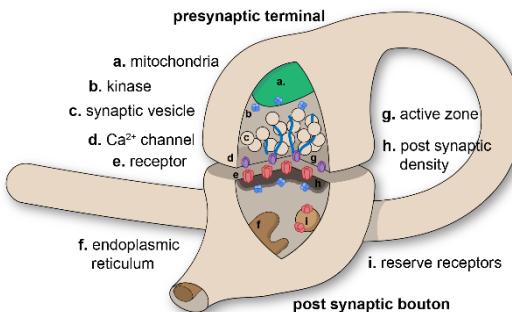


Figure 2. Diagram of the synapse.

Credit: Curtis Neveu from learnbio.org

A chemical synapse consists of 3 structures, presynaptic membrane, synaptic cleft, and postsynaptic membrane. When a neuron is activated, the neurotransmitters - chemical messengers - are released into the cleft between two neurons. These messengers bind to receptors on the next neuron. Thus the message is relayed across a series of neurons. These chemical messengers are degraded or pumped back into the neurons shortly after being released. When this does not happen, it can have disastrous consequences. The whole process gets over in a few milliseconds. This is the basis of signaling in the brain. The function of each neuron depends on the type of neurotransmitter released and the receptor involved.

Now that we have an idea of a synapse, we go deeper into our neuroreceptor of interest, which is the NMDA receptor and its neurotransmitter, called Glutamate.

GLUTAMATE AND NMDA RECEPTOR

Neurotransmitters can either excite or inhibit a neuron's activity. One of the major excitatory transmitters in the brain and spinal cord is glutamate. It is released from the presynaptic membrane and binds to four types of receptors. One of these receptors is called NMDA (N-methyl-D-aspartate), which is a key contributor to brain injury in stroke [3] [4].

Opening/ Activation of the receptor has three requirements: Glutamate, Glycine/ D-serine, and a depolarization of the postsynaptic membrane, which relieves the voltage-dependent channel block by Mg²⁺. With the block gone, potassium, sodium, and calcium ions flood the cell.

An influx of Ca²⁺ triggers intracellular signaling pathways, which are relevant to stroke and excitotoxicity (discussed below).

Optimal activation of such receptors is required for normal brain function and to promote neuron survival. But an excessive and unregulated release of glutamate (that usually occurs in various neurological pathologies like Alzheimer's, Huntington's, and stroke) will rapidly promote cell death, this process is called glutamate excitotoxicity.

WHY DOES EXTRASYNPATC NMDAR GET ACTIVATED DURING ISCHEMIC DAMAGE?

NMDA receptors (NMDARs) are present outside the cell. Such extrasynaptic NMDARs are thought to be the main culprits behind excitotoxicity. Their activation during the excessive and unregulated release of glutamate causes further neuron damage after a loss of blood flow to the brain which has already damaged or killed many neurons.

Usually, in a normal state, a specified amount of Glutamine is released from the presynaptic membrane and attaches to NMDAR only in the synapse, following which Glutamate is removed from the synaptic cleft by several high-affinity glutamate transporters present in both glial cells and presynaptic terminals. In this healthy state, only synaptic NMDARs get activated which is required for normal brain function. During a stroke, neurons undergo ischemic damage, which, if it lasts for a few minutes, will lead to a permanent loss of the neurons. This ischemic damage leads to energy depletion and consequent failure to maintain the resting state of neurons, which leads to the depolarization of cells. The glutamate released at synapses relieves the block on NMDARs and reverses glutamate uptake. Glutamate spills around the synapse and leads to subsequent activation of extrasynaptic NMDARs leading to further neuronal damage [4,5].

DUAL ROLE OF NMDAR IN SURVIVAL AND DEATH OF NEURONS IN ISCHEMIC DAMAGE

Earlier it was thought that increased NMDA stimulation in brain pathologies like stroke led to an excessive calcium ion influx into the cell, which would lead to cellular dysfunction. The term excitotoxicity hence arose from this statement.

But the currently accepted mechanism states that excessive glutamate release during stroke causes its spill into the extrasynaptic region where lies the extrasynaptic NMDAR. These particular receptors possess the cell signaling pathways that promote the death of neurons.

Therefore, in a stroke, the lack of energy due to a loss of blood flow plus the activation of extrasynaptic NMDAR leads to a synergistic pathway for cell death of neurons [4,6].

A survival versus death signal for a neuron depends on whether the synaptic NMDAR or the extrasynaptic NMDAR has been activated. Synaptic NMDAR causes the expression of survival signals in the brain and extrasynaptic NMDAR causes the activation of pro-death signals in the neurons [6].

The difference in synaptic and extrasynaptic signaling could be due to a combination of factors:

The first and most important factor is the activation of the type of subunit. Although the synaptic and extrasynaptic receptors look similar, the majority of NMDA subunit in each location is different, which means their downstream cellular process are different. Activation of such specific synaptic receptors leads to increased cell survival signals and activation of extrasynaptic receptors promotes cell death [7-10].

Another factor that differentiates synaptic and extrasynaptic signaling could be the presence of an NMDAR-associated protein complex exclusively at the synapse or the extrasynaptic site. This means that certain proteins are found only at the extra-synaptic site which causes cell death. A particular protein called TRPM4 (transient receptor potential cation channel subfamily M member 4) is completely absent from the synapse. This protein is associated with NMDAR and the coupling and activation of NMDAR and TRPM4 promote excitotoxicity in neurons [11].

Neuron cell culture and in-vivo mouse models show promise of drug therapy that disrupts the NMDAR/TRPM 4 complex, leading to reduced extrasynaptic toxic NMDAR signaling. The drug, named NMDAR/TRPM4 interface inhibitor disrupts the protein complex causing their dissociation and hence, the extrasynaptic NMDAR loses its ability to damage the neuron.

This should explain why synaptic NMDAR improves neuronal survival and extrasynaptic NMDAR causes neuronal death.

CURRENT PHARMACOLOGICAL NEUROPROTECTANTS FOR STROKE TREATMENT

In theory, blocking the downstream cellular pathways that occur during a stroke seems like a promising solution. Based on pre-clinical studies, the early administration of neuroprotective agents may be a promising treatment strategy for stroke patients [12-14].

However, to date, no compelling data has been published regarding any pharmacologic or other therapies. And as such no neuroprotective agents have been approved by the FDA in the USA or Central Drugs Standard Control Organization (CDSCO) in India for stroke treatment. However, the below-mentioned pharmacological agents show a certain degree of promise.

Memantine:

Memantine is an uncompetitive antagonist of the NMDA receptor [15]. This means its ability to block receptor binding is more effective at higher levels of glutamate. At the therapeutic dose, it shows a preference for extrasynaptic NMDAR [6]. This means that it can prevent the neuronal damage that occurs due to the activation of extrasynaptic NMDAR during a stroke.

Although several pre-clinical studies show memantine as an effective treatment, few clinical trials in humans have evaluated the safety and efficacy of memantine in stroke patients, and thus, as of now, no NMDAR antagonists are FDA-approved for stroke treatment [15].

NMDAR/TRPM4 interface inhibitors:

These inhibitors provide neuroprotection against neuronal death in cultured neurons and in-vivo mouse models and show promise as a future drug as an add-on therapy for stroke management [10].

Other pharmacological agents:

Tamoxifen, a drug used to prevent breast cancer in women and treat breast cancer in women and men, can prevent excessive glutamate release in the synapse during an ischemic damage to the neuron. Glibenclamide, a drug for type-2 diabetes, inhibits a cell death signaling pathway in the brain. These two drugs are being tested as potential therapeutic drugs [4].

HOW TO DETECT A STROKE AS A BYSTANDER

After learning a lot about cell death mechanisms during a stroke, we should be aware of early stroke symptoms and be quick to detect them.

Signs and symptoms of stroke include, but are not limited to:

- **Headache.** A sudden, severe headache, accompanied by vomiting, dizziness, or altered consciousness, may indicate a stroke.

- **Problems seeing in one or both eyes.** The person may suddenly have double vision or blurring/vision loss in one or both eyes.
- **Trouble speaking and understanding what others are saying.** The person may present with confusion, slurred speech, or difficulty understanding what others say.
- **Paralysis or numbness of the face, arm, or leg.** The person may develop sudden numbness, weakness, or paralysis in the face, arm, or leg. Usually, one side of the body is affected. Try to raise both arms over the head at the same time. If one arm begins to fall, it could indicate a stroke. Also, one side of the mouth may droop while smiling.
- **Trouble walking.** The person may stumble or lose their balance. It can be accompanied by sudden dizziness or a loss of coordination [16].

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3 minds in a body? Brain's defence mode is a disorder!

"I am large. I contain multitudes." - Walt Whitman

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SUMMARY

The following article discusses Dissociative Identity Disorder (DID), previously known as Multiple Personality Disorder. It is a mental ailment where the individual develops two or more personalities called 'alters'. This disorder develops as a coping mechanism for overwhelming trauma. The only way to tackle it is by providing a correct diagnosis and psychotherapeutic intervention to manage the symptoms. People with DID are often stigmatised and isolated. It is important to support them and gain awareness about this disorder.

INTRODUCTION

Self-identity and *Personality* are multidimensional concepts. Personal experiences and beliefs shape them. It sets one apart from others and defines a person as a whole. We often find people lingering with a rhetorical question - **"Who am I?"**

But can one imagine a situation where someone forgets who they are for real!
Sounds unreal? But it happens!

Dissociative Identity Disorder (DID) is a rare mental disorder which leads to this condition.

ABOUT THE DISORDER

Formerly, DID was known as a multiple personality disorder. As the name indicates, DID involves a severe state of dissociation from a person's thoughts, memories, feelings, actions or sense of self. In other words, a disconnection from reality!

A person suffering from DID is believed to exhibit two or more personalities, called '*alters*' when exposed to stressors that remind them of past trauma. It develops in an individual as a coping mechanism to deal with overwhelming traumas like child abuse, disorganized attachment to parents, or warfare experience. By expressing the alter, the person tries to escape reality, where the alter takes control of the host's behaviour and thoughts. The host becomes a different person altogether with a degradation of self-identity and remains oblivious that they have undergone a transition. This transition is referred to as '*switching*'. It is episodic, happens involuntarily but reversibly and leaves the host distressed. However, the imaginary alter can deal with the emotional disturbance that the host could not in real life [1].

THE ALTERS DO NOT KNOW ABOUT EACH OTHER!

It is bewildering to imagine but did you know that **a person with DID can express as many as 2-100 alters?**

They all have their unique name, identity and apparent age, gender, behaviour and body language differences. Each "alters" is believed to have a distinct autobiographical memory.

Autobiographical memory refers to memory for one's personal history, which creates and maintains a coherent self-identity. The alter is expressed as a psychological response to the traumatic memory one has; primarily, they represent a disjointed sense of identity [1].

THE SYMPTOMS

The best way to understand any disorder is to observe its symptoms. It is challenging to diagnose DID since its symptoms overlap with other psychological illnesses like PTSD (Post Traumatic Stress Disorder) and BPD (Borderline Personality Disorder). However, there are specific symptoms unique to DID, such as the presence of 2 or more unique alternate personalities in a person and hearing voices which could be a child crying or certain "supernatural beings" communicating and instructing the individual to behave in a certain way. The patient may also suffer from depression and feel helpless and lost, and may not know what is happening to them. Increased alcohol and drug abuse and cases of attempted suicide have also been observed, along with some instances of individuals undergoing psychotic episodes, which have been accompanied by disorientation, loss of control over emotions, actions and attitudes, and a loss of core identity. Some other symptoms are amnesia (where a person has no recollection of events that would have taken place when the alternate personality was expressed) and patients finding themselves in specific geographical locations without understanding how they reached there [2].

In our day-to-day life, it is pervasive that sometimes we lose ourselves daydreaming. However, it is disconcerting when feeling transported becomes so intense that one is separated from one's mind and body. That is precisely what happens in DID when the patient undergoes dissociation.

CAUSES AND TRIGGERS OF DID

Every mental disorder has a trigger. For patients with DID, a trigger is anything which reminds them of the severe trauma they have undergone in their childhood. It can be any sound, sight, smell, taste or action associated with the trauma. Stress, memories, strong emotions, and alcohol and substance abuse can also serve as triggers for people with DID. In some instances, identifying triggers has not been possible [3].

"A child is born like a blank slate, and its later behaviour is shaped by experience",
-John Locke.

Personal identity is still taking shape and is at a tender state during childhood. Thus, children are more susceptible than adults to step outside and observe trauma as though it is happening to a different person. Dissociation represents lapses in psychological and cognitive processing and is linked to trauma. It occurs as the mind's defence mode for survival. Many shades of dissociation lead to full-blown DID. The relationship between trauma, dissociation, and psychosis has been a topic of study in dissociative disorders for several decades. Children learn to dissociate to endure a traumatic experience and may use this coping strategy to respond to stressful situations even when they mature. Trauma fragments a child's personality, whereas adults are more prone to develop PTSD if faced with a similar situation [5].

WHO IS MORE PRONE TO DEVELOP DID? IS IT DIFFERENT FOR MEN AND WOMEN?

As we have understood, DID is directly linked to stress and trauma. Different people respond to trauma differently. However, does gender play a role in this? Let's find out!

Current studies have shown that women are more likely to be diagnosed with DID than men. It is because women have a higher frequency of symptoms than men.

Furthermore, men are most likely to hide their symptoms and traumatic histories. Interestingly, men have been observed to have lesser memory loss, and since the male gender is believed to be aggressive, this has resulted in fewer diagnoses of DID in men. Unfortunately, it has also been seen that females face childhood abuse more frequently than men, making DID more likely to occur in the female population [6].

It is essential to understand that it doesn't matter if a patient suffering from DID is male or female. Both sexes deserve treatment and support from those around them. Since DID doesn't discriminate based on gender, we shouldn't either!

THE NEUROBIOLOGY BEHIND DID IS BEING STUDIED!

Scientific research indicates that DID patients reportedly have smaller cortical and subcortical volumes compared to unaffected people's brains [7]. The difference was observed in -

- 1) *Hippocampus* (part of the brain important for learning and memory; it gets damaged easily by stimuli)
- 2) *Amygdala* (the structure that majorly contributes to processing fearful and threatening stimuli and coming up with a reaction mechanism to stimuli)
- 3) *Parietal structures* (involved in perception and personal awareness) and
- 4) *Frontal structures* (involved in movement execution and fear learning)

Larger white matter tracts are observed in DID patients. These tracts play a role in information exchange between the somatosensory association regions, the basal ganglia, and the *precuneus*.

Precuneus is a brain region involved in many complex activities such as memory and integration of information related to the environment, episodic memory retrieval and response to pain. For expressing multiple personalities, the neural circuits in the brain are more extensively wired, which explains a bigger precuneus size or activity in DID patients [7].

Studies show that people who have suffered child abuse have a significantly reduced hippocampal volume [7]. Current findings also indicate that neuroimaging patterns of biomarkers can distinguish DID patients from healthy controls [8].

These reports are of prime importance because they provide evidence of a biological basis for distinguishing DID patients.

EPIDEMIOLOGY

Rates of diagnosed DID have increased in the late 20th century to approximately **40,000 cases**. Proponents of DID believe that the rise in documented cases is due to increased recognition of and ability to identify the disorder. Statistics from North America found that 4.0–5.4% of psychiatric inpatients met DSM-IV (*Diagnostic and Statistical Manual of Mental Disorders - 4th Edition*) criteria for DID. In Turkey, the prevalence rate of DID is 5.4% among inpatients and 2.0–2.5% among outpatients [1,4].

Rationales for the variations in cases may be attributed to cultural elements that impact both emergence of DID and the understanding of symptoms. For example, European studies

report substantially lower rates of DID. Notwithstanding the abnormality of DID, other dissociative disorders, such as possession syndrome, have been reported commonly in India but not DID. It is presumed that the high prevalence of hysterical possession in India is related to religious beliefs in polytheism and reincarnation.

HISTORY AND CURRENT POP CULTURE PORTRAYAL OF DID

Despite its mention in the history of psychiatry, DID is still associated with controversy leading to misdiagnosis or under diagnosis.

Jean-Martin Charcot, the great 19th-century neurologist, first brought the concepts of hysteria to public attention. Later, it was **Pierre Janet** and **Sigmund Freud** who recognised that altered states came from trauma and that somatic symptoms indicated disguised representations of events repressed from memory. Janet produced the term '**idée fixe**', while Breuer and Freud coined '**double-consciousness**' [9].

There are various books and movies portraying patients with DID. *The Strange Case of Dr Jekyll and Mr Hyde (1800s)* explores the relationship between Dr Jekyll and his alter ego, Mr Hyde. *Psycho (1960)* is a famous movie which is a story about Norman Bates and his alter ego, who takes the form of his mother and commits murders. *Split (2017)* is another movie in which 'Kevin' kidnaps three young girls and locks them in a basement. We also understand the dynamics between Kevin and the 23 other personalities inside him. The media portrayal of individuals with DID has been in the negative light as evil, psychopathic murderers with 'Split' taking it to a whole new level and showing physical transformations in one of the personalities of Kevin, which scales walls and is virtually indestructible. Such misrepresentation in pop culture has stigmatised patients with DID.

In this day and age, when mental health has come to the forefront because of the pandemic, it is necessary to be aware of all mental disorders. Because of media portrayal, DID patients are assumed to be extremely violent. Thus, it is crucial to understand that misrepresentation in the name of entertainment will only lead to further isolation of patients who are already helpless and alone because people with DID are victims, not perpetrators. The patients should be supported and not pushed away. Many famous personalities with DID have publicly spoken about their experiences. Like, comedian and talk show host **Roseanne Barr; Chris Costner Sizemore**, whose story was filmed in *The Three Faces of Eve* and NFL player **Herschel Walker**, author of *Breaking Free: My Life with Dissociative Identity Disorder*.

TREATMENT AND MANAGEMENT

The treatment of DID should be focused on integrating the fragmented personality and acceptance. The patient or the alter must be provided with the opportunity to tell their story and be heard and supported. Psychotherapy should focus on identifying and working through past trauma and managing sudden behavioural changes. Some healthcare providers may recommend medication and hypnotherapy in combination with psychotherapy. Hypnotherapy is a guided meditation that may help people recover suppressed memories. Friends and family members play an essential role in tackling the condition by showing care and support.

The empirical literature on DID is accumulating, although some areas remain under-investigated. Existing data show DID as a complex and valid disorder associated with developmental and cultural variables amenable to psychotherapeutic intervention. Hopefully,

as research progresses and treatment for mental disorders such as Alzheimer's and Parkinson's are developed, other mental illnesses such as DID will also be understood better for devising intervention strategies, diagnoses and therapeutic techniques. Till then, it is vital to support those suffering from this life-altering disorder, acknowledging their issue and reminding them that they are not alone.

"DID raises problematic philosophical and psychological concerns about the nature of the mind itself. Ideas of a unitary ego would incline professionals to see multiplicity as a behavioural disturbance. However, if the mind is seen as a seamless collaboration between multiple selves, a kind of 'trade union agreement' for co-existence, it is less threatening to face this subject."

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Huntington's disease: a neuropsychiatric disorder

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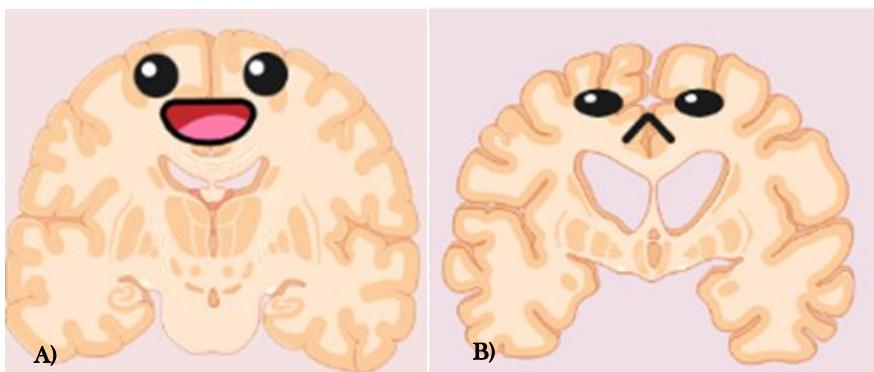
SUMMARY

Huntington's Disease (HD) is a health condition caused by progressive degeneration of brain cells. It affects the individual's movement, intellectual abilities, and behaviour. It is an inherited disease caused by a genetic change in the gene that codes for a protein called huntingtin. This article delves deeper into the causes, effects, and consequences of Huntington's Disease in the form of a conversation between a Normal brain and an HD-affected brain.

NB - Normal Brain

HB - Huntington Brain

Let us look into how the Huntington Brain explains its mechanism in the pathway.



A) Normal Brain B) Huntington Brain

(HB is Screaming in pain, NB looks at him and comes closely towards HB and....)

NB: Hey! What is wrong with you? Why are you making a ruckus?

HB: You can't understand my pain, so please don't mind...

NB: Oh! I am one of the intelligent organs. Can't I understand?

(Without saying a word, HB tries to move but falters, NB helps it balance)

HB: Thank you!!

NB: Why are you unable to move? And why does your cortex look so empty and hollow?

HB: It's because I have a degenerating brain. I am unwell. My neurons are dying.

NB: But you seem to be too young to die!

HB: My story starts with a faulty Huntingtin gene (HTT gene). (Jimenez-Sanchez et. al.)

NB: You mean huntingtin gene? I know this gene is present on the short arm of chromosome 4 of my cells. The protein made by this gene helps us nourish our neural cells and helps us maintain our health. How can an important gene such as this cause any harm to you?

HB: Yes! The normal form of the gene i.e. the normal allele helps us maintain our health. But in my case, I carry a mutated version of the gene i.e. mutated allele. In a normal allele, there is a stretch having multiple repeats of the three nucleotides 'CAG'. In a normal brain like you, the number of CAG repeats in the huntingtin gene can range from 7 to 35. As long as the number of triplet repeats is less than 35, it does not harm the brain. But in some individuals like myself, the number of nucleotide repeats is 36 or more! Such expansion of the CAG repeat is harmful [1].

NB: That is interesting! Can you tell me more about the function of the huntingtin gene and how the triplet repeat expansion affects the functioning of your brain?

HB: Well, the huntingtin gene codes for huntingtin protein. The triplet CAG codes for the amino acid Glutamic acid, which in its ionized form is known as 'Glutamate'. When the gene has a stretch of CAG repeats, the protein encoded by that gene has a stretch of the amino acid Glutamic acid. When the number of CAG repeats increases in the gene, the cell responds by increasing the production of the amino acid called glutamic acid. In its glutamate form, this amino acid functions as an excitatory neurotransmitter. It keeps the neurons in the circuit in an excited state [2].

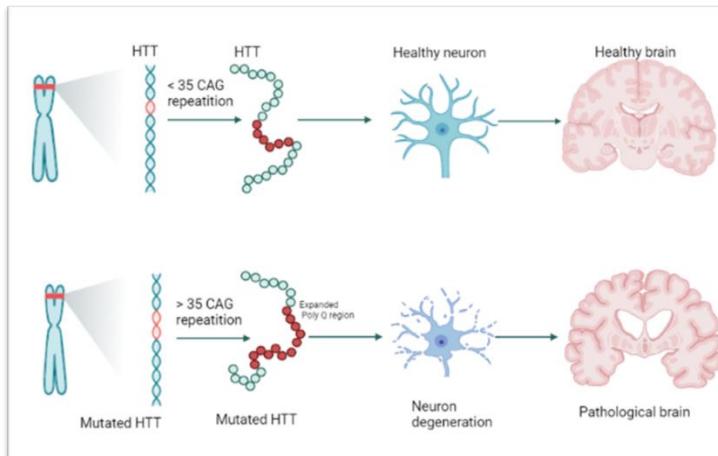


Figure 1: The gene huntingtin is present on the short arm of chromosome 4 in humans. This gene has a region with a repeating sequence of CAG; because the triplet CAG codes for the amino acid Glutamic acid (single letter code 'Q'), the corresponding region in the protein has a stretch of polyQ or poly-glutamic acid. When the number of repeats expands beyond 35, it leads to neuronal degeneration and the progression of Huntington's Disease.

NB: Isn't there any control for this?

HB: Yes! The Glial cells play a significant role, but in my case, that too fails.

NB: The Glial cells maintain homeostasis within our environment by taking up glutamate and converting it into Glutamine to maintain homeostasis.

HB: Yes! You're right. But in my case, the increased production of Glutamate is overwhelming for the glial cells to convert it into Glutamine; this disturbs homeostasis. This disruption makes my neuronal mitochondria susceptible to glutamate and finally causes the death of my neurons, mainly the cortex. That is why you find my cortex region severely degenerated.

NB: Now I understand why you are having difficulty moving. Our cortex which controls movements of the body limbs is affected in your case. I'm curious to understand the cause a little deeper if you don't mind. The huntingtin gene is present on chromosome 4, but each cell has two chromosomes 4s, one received from the father and one received from the mother; hence there must be two copies of the huntingtin gene, isn't it? Even if the huntingtin gene in one of the chromosome 4s has increased triplet repeats, can't the huntingtin gene on the other chromosome 4 function properly and protect the cells?

HB: What you say can be true in the case of inheritance of some other diseases. But unfortunately, not in the case of this gene! In the case of the huntingtin gene, the defective allele of the gene (with >35 CAG repeats) is dominant over the normal allele. Therefore, even if there is one copy of the defective allele and one copy of the normal allele, the defective allele's function takes dominance in the cell. Since this gene is present on chromosome 4, an autosome, the inheritance of Huntington's disease is said to be an Autosomal Dominant inheritance! In the case of autosomal dominant inheritance of disease conditions, even if only one of the parents is affected, each child has a 50% chance of inheriting the disease condition [2].

NB: Oh! Thank you for explaining clearly. What other factors contribute to the progression of this condition?

HB: Other factors play a role in the onset and progression of Huntington's disease. The age of onset depends on the number of repeats. If the number of triplet repeats is about 36, the disease onset starts from the 3rd or 4th decade of life. If the number of CAG repeats is greater than 40, then the disease onset starts at an earlier age. It is known as Juvenile Huntington's Disease when it occurs before the age of 20 [3].

NB: Oh! That's so scary! Are there any other factors contributing to this condition?

HB: Yes, gender seems to play a minor role; females are affected more than males!

NB: You mentioned that the damage affects the regions of the brain controlling the movements of the individual. In what other ways are the individuals affected?

HB: The inability to move is just one of the challenges faced by individuals. They have difficulty speaking, and performing their daily tasks. They also show psychiatric symptoms, and slowly a patient entirely depends on the caregiver. Eventually, they die within about 20 years from onset [3].

NB: How do doctors find out about this disease from the outside?

HB: That's easy. Usually, the huntingtin protein accumulating in the cells forms aggregates. These aggregates can easily be detected in scans like CT or MRI. That is how they find out about Huntington's disease progression [4].

NB: Where do these aggregates form?

HB: These aggregates are in the nucleus in the cells, and as the disease progresses, they are in the cytoplasm and neuronal processes in me.

NB: Can you describe more about what you go through?

HB: Sure! My primary feature is the degeneration of the neurons from the cerebral cortex. My extreme cases lead to chorea, due to the degeneration of spiny neurons in the basal ganglia [3].

NB: How does this affect you at the molecular, organ, and individual levels?

HB: At the molecular level, there are excitotoxicity, mitochondrial dysfunction, and axonal and synaptic dysfunction. At the organ level, there's a decrease in brain mass, neuronal losses, and neurotransmitter abnormalities. The individual affected undergoes cognitive disabilities, motor disabilities, and psychiatric syndromes as mentioned earlier [2].

NB: Okay then. What are the possible psychiatric effects of this disease?

HB: The psychiatric symptoms are depression, psychosis, and obsessive-compulsive disorder.

NB: Are there any other symptoms of extreme cases?

HB: Yes. Those are weight loss, skeletal muscle wastage, and cardiac failure.

NB: It's so sad. Is there any cure for this?

HB: Unfortunately, NO. But there are a few drugs like Chlorpromazine, Haloperidol, Olanzapine, and Clozapine, which partially suppress the chorea and agitation [5].

NB: This sounds so scary!!

HB: Yes, it is. Henceforth, when you find someone with a disease similar to mine, treat them kindly. You may not fully understand our suffering.

NB: Yes. I understand your difficulty, and I am sorry about my inconsiderate behaviour.

HB: Thank you!

(HB gets up and moves away with difficulty, while NB leaves for a journey to help brains with different pathophysiology).

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Spectral Interventions: Drug Discovery in Autism Spectrum Disorders

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SUMMARY

Autism Spectrum Disorders affect about 1% of the global population, and to date, factors contributing to its pathogenesis are not fully understood. Diagnostic criteria remain vague blanket statements. We explore the genetic and molecular basis of neurodevelopmental disorders, along with theories explaining clinical symptoms and molecules that can act as potential drug targets for intervention.

INTRODUCTION

Aaliyah wears a crisp white shirt and pinstriped trousers, just like the rest of her peers. When she hears the trill of the second bell after break, her excitement knows no bounds - to her, art and painting are soothing. They produce a sense of quiet in her mind, a tactile medium of expression; a sense of belonging and validation when her teacher tells her how creative she is - in sharp contrast to the tight ball she feels in her chest when numbers and letters float straight across each other during the rest of her day, and the confusion she feels when her friends laugh at her when she frankly answers a question.

Turquoise and deep browns, greys and fiery oranges - an array of rocks sit on Himanshu's desk, and his tutor smiles as she listens to him talk about his collection of Pokémon, and what he ate for lunch that day. Watching him feel the contours of each stone with the pads of his fingers, she remembers the look on his parents' faces when they sat together a few hours earlier, discussing how sudden fixations could be better managed. Himanshu is mathematically gifted but has underdeveloped executive functions and communication.

An economic analyst at a Fortune 500 company, Kiran thrives in professional environments. As the weekend approaches, however, she finds her mind drifting, obsessively ruminating over memories of fading relationships - from her days in school to the present, she's always struggled with social cues, trying to adhere to what the world around her deemed 'normal', and maintaining eye contact with strangers and friends alike. Years of turmoil and learning to accept the fleeting nature of emotional attachments and human connection have left her with acute anxiety, and she spends a few minutes wondering if she can get through their team-building activity this weekend without experiencing sensory overload.

From children as young as eighteen months old to adults living their own lives, diagnosing and managing Autism Spectrum Disorders (ASD) has always posed a challenge. Clinicians often weigh several factors before providing a definitive diagnosis, and these factors are not always objective - they're not based on visible, palpable data points, like values obtained on blood tests. One clinical test that has consistently produced viable findings is the electroencephalogram or EEG. It involves the application of small disks and cups, called electrodes, to the scalp, allowing us to pick up electrical activity in the brain and view it as waves on a screen. The brain produces different kinds of waves in different stages of thinking

and reasoning. While EEGs are objective, they are currently used only as predictive tools. Most diagnostic criteria are centred around the long-term observation of a child's development, and bias runs high - multiple specialists can have different opinions on what condition a single patient might have.

These factors, or criteria, are listed in the DSM-5, the Diagnostic and Statistical Manual of Mental Disorders - and they pose the biggest hurdle to ASD diagnoses. For example, one of the criteria states that "symptoms must be present in the early developmental period," but these may not manifest fully until social demands exceed the already limited capacities of individuals on the spectrum. In most cases of adult-onset autism, symptoms are masked by strategies learned later in life and are therefore diagnosed only on strong suspicion and careful study - the disorder itself cannot develop in adulthood, because ASD is *neurodevelopmental*.

UNDERSTANDING THE BIOLOGICAL BASIS OF NEURODIVERGENCE

What does this mean, though? The term *neurodevelopmental disorders* is quite broad, and refers to a group of conditions that have one common characteristic - delays in acquiring skills, be they motor, cognitive, language, or social. ASD, as we already know, is a good example. Others include ADHD (Attention Deficit Hyperactivity Disorder), cerebral palsy, and learning disorders such as dyslexia. Truth be told, it's also fair to argue that labelling these conditions disorders is unfair - to be neurodivergent can be said to simply be different. Every human is a unique biological entity, and in those on the spectrum, the fundamental makeup of tissue, the neural networks, and the influence the network itself has on the firing of individual neurons develop differently, and are distinct from those of an individual who is neurotypical - and before you ask, let's explore how these networks work.

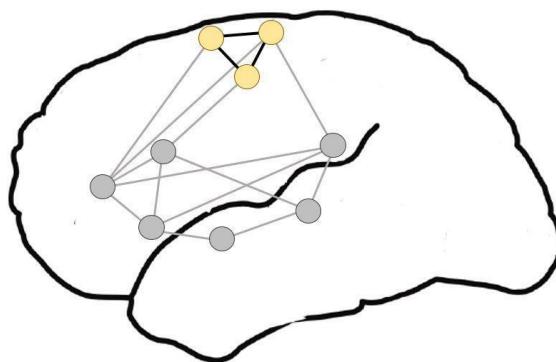


Figure 1. Shows a neural circuit in yellow connected to a larger neural network in gray.

Image credit: By Novasdid - Own work, CC BY-SA 4.0, <https://commons.wikimedia.org/w/index.php?curid=58421153>

Imagine two neurons, floating in a matrix in the darkness of a skull. These neurons are connected by a synapse between them or a junction. The whole unit together is called a *neural circuit*, and multiple circuits together form a *biological neural network*, as seen in Fig.

1. Within this network, there are neuronal highways called *neural pathways* that connect different parts of the brain - from the frontal lobe to the occipital, for example, from front to back. Based on these, scientists developed copies - simulations, designed to help us analyze how our brains process stimuli and information - called *artificial neural networks*, which we use today to attempt to create replicas of neurodivergent brains in order to understand what sets them apart.

EVERYONE HAS AN ORIGIN STORY - AND SO DOES ASD

ASD is an ocean, and its surface is punctuated with theories explaining the very beginning of it all, two of which have garnered much attention. The Enhanced Perceptual Functioning (EPF) theory proposes that ASD arises due to superior low-level processing, which means that they are exceptionally skilled at identifying patterns and solving complex mathematics. However, this occurs at the expense of higher-order functions, such as everyday interaction, understanding social cues, and reading between the lines.

The second theory, called the Neural Noise Hypothesis, explains why some individuals react strongly to bright lights or loud noises - this is thought to be due to certain aspects of sensory stimuli like sound and light being processed differently. Expanding this theory has also shown us that reactions in response to disturbing stimuli, such as the urge to cover their ears in response to a loud sound, are more of a *need*, an involuntary motor response that cannot be controlled [1].

Peering over a wall and down the genetic roots of autism, we see that there is little information on the role our DNA or genes play in ASD. Still, based on recent studies, autistic disorders have been proven to be caused by many different genetic changes [2]. These genetic changes could be of various kinds, right from a change in a tiny sequence of DNA, entailing a single gene mutation to major changes like chromosomal deletions.

The DNA in our cells is arranged into entities called chromosomes. Different chromosomal deletions and duplications contribute to autistic disorders. A chromosomal aberration could potentially involve several genes. The identified genes that play a part in ASD also control several other syndromes, indicating the complexity of its genetics. One such example is phenylketonuria which sometimes features autism-like characteristics. Another condition, Tuberous Sclerosis Complex (TSC), in which individuals develop small tumours all over the body also shows features of ASD in 30 to 60% of cases. Most of these mutations affect the functioning of the nervous system of a developing embryo which eventually manifests as a difference in brain function [2].

A few of the mutations cause an imbalance in neural signals like excitatory signals, E (that contribute to the generation of an action potential), and inhibitory signals, I (that inhibit these action potentials). This phenomenon is called E/I imbalance. In order for our brains to function, neurons need to effectively communicate with each other. This communication occurs through a connection called a synapse between neurons across which chemical messengers called neurotransmitters are released. It is believed that a change in the levels of some neurotransmitters in the brain can lead to an E/I imbalance. A change in other molecules involved in carrying signals within and between neurons can also be damaging. The effect on signal transmission and thus the functioning of neurons has opened a door to drug discovery, and studies on restoring balanced neurotransmission are underway, thus paving a path to finding a cure for autism. Synaptic changes in autism might also play a role in the loss

of social and linguistic function. Even if there isn't much information regarding this, studies have shown that mutations in some proteins associated with neural signal pathways, even mutations associated with signal transmission will negatively affect the development of social behavior [3].

Normally, signalling pathways, like the basic schematic shown in Fig. 2, regulate cellular activities, such as the production of a hormone, cell death, or the activity of proteins. Some of these proteins lie embedded in cell membranes and can act as gateways to the passage of different compounds, including ions and sugars, or as receptors that allow the attachment of other proteins and molecules that trigger - you guessed it - signalling cascades.

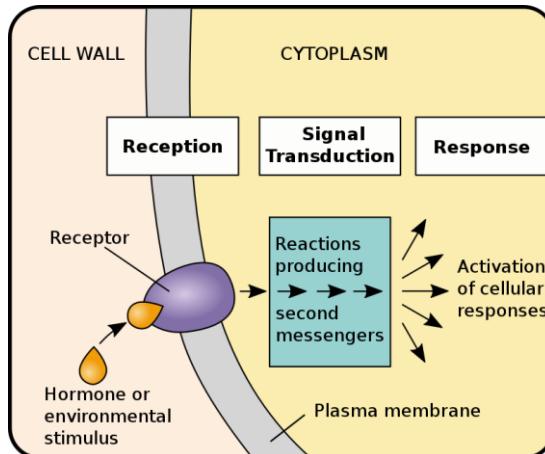


Figure 2. Basic signal transduction pathway, with a receptor molecule in the membrane, messenger molecules acting as transducers, and cellular responses as the endpoint.

Image credit: By Original: Yancepom - Own work based on: 1Signal Transduction Pathways Model.jpg; CC BY-SA 4.0, <https://commons.wikimedia.org/w/index.php?curid=97419672>

The human body is made up of trillions of cells, and each of these cells interacts with each other through hundreds of different types of signalling molecules and receptors in combination to produce what we see as a fully functional biological system. Of these, neuronal development and function depend on one such pathway, known as SMO-SHH signalling. Defects in this pathway have been linked to ASD. SMO stands for Smoothened, a receptor that activates SHH signalling, or Sonic Hedgehog (yes, Sonic the blue, fury hedgehog is everywhere). Another molecule involved in the pathway is Patched-1, which acts as an inhibitor [4].

Research has also revealed that neurodivergent brains show evidence of impaired myelin (think of it as something that allows high-speed communication, like fibre-optic cables for the internet) production, indicating that interneuronal communication may be impaired [5].

A RAINBOW OF MOLECULES, AND BEACONS OF HOPE

So why do we need to keep digging deeper into the defective pathways underlying ASD, and why do we need to frame accurate diagnostic criteria? Knowing what processes contribute to a disease or syndrome in this case, allows us to *choose target molecules* - the hedgehog family, myelin, and Patched - as potential drug targets. While attempting to treat the manifestation of ASD may not bear much fruit, identifying missed milestones in early childhood may allow us to target specific pathways, such as SMO-SHH, and maintain neurological well-being in children with ASD, as well as in cancer, Alzheimer's disease, and recovery from spinal cord injuries [4]. Maher et al., based at the Lieber Institute in Baltimore, say that targeting oligodendrocytes (cells that produce myelin in the central nervous system) may also prove useful - and unlike SMO-SHH, myelination does not end with early development. It is a lifelong process and can be modified using interventions throughout a person's lifespan [5].

And while these newer drugs are quite a while away from entering human trials, let alone the market, there are a wide variety of treatment methods currently in use today - and they are all examples of skillfully integrated biological science and technology, put to use for bettering the quality of so many lives, both young and old. A 5-year-old boy in Philadelphia, diagnosed with non-speaking autism, can now say a few words and initiate conversations using his AAC device, for Augmentative and Alternative Communication. A 14-year-old from Bangalore with ASD and ADHD now enjoys performing stand-up comedy after years of consistent speech and applied behaviour analysis. There is always hope - miracles happen every single day. To see people of all kinds together, neurotypical and atypical, coexisting, is a testament to this. We must never lose sight of the beauty that lies in variation - in being different; in being neurodiverse.

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Brain, trauma, and rehabilitation

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SUMMARY

The brain is an essential organ that regulates our body's functions. Trauma caused by physical injury or emotional stress can alter our thoughts and behaviours. The three brain areas most affected by trauma are the amygdala, hippocampus, and prefrontal cortex. Damage to these organs puts trauma survivors on high alert all the time. Therefore, a victim of a traumatic brain injury deals with physical, cognitive, and emotional issues. Although a traumatic event could cause permanent damage, our brain's capacity to generate new connections aids in its repair and recovery. To improve patient's quality of life, caregivers employ a variety of therapies, including physical, occupational, mental, and speech therapy.

INTRODUCTION

The human brain is a 1.2-1.3 kg mass of highly soft tissue, floating in a fluid inside the skull, that controls every process that regulates our body [1]. It is made up of a network of a billion cells called neurons, which regulate the way we think and act. Different regions of the brain control different functions. Brain injury, therefore, affects different people's abilities and behaviours differently, depending on the area of the brain that is affected.

TRAUMATIC BRAIN INJURY AND POST TRAUMATIC STRESS DISORDER

The damage caused to the brain by a tough blow, perhaps because of a fall, car accident, or gunshot, is called traumatic brain injury (TBI). According to the Centers for Disease Control and Prevention (CDC), falls, injuries from firearms, car crashes, and assaults are the most common causes of TBIs [2]. Moreover, scientists have also found that TBI could also cause emotional trauma and post-traumatic stress disorder (PTSD) — persistent mental and emotional stress due to a severe psychological shock or injury [3].

Such trauma can affect the brain in various ways because of the way our brain responds to injury, just like any other tissue in the body: swelling and getting filled with fluid. As the brain swells, the pressure within the skull increases, damaging the brain further. Chemical messengers that transfer signals between the brain cells, and to the whole body through nerves, are hampered; and all these factors affect the way our neurons behave, and change the way we think and behave. This could also influence our subconscious reactions to events happening around us.

THE BRAIN'S RESPONSE TO TRAUMA

The three parts of the brain that are most affected by trauma are the amygdala, hippocampus, and prefrontal cortex (Fig. 1) [4].

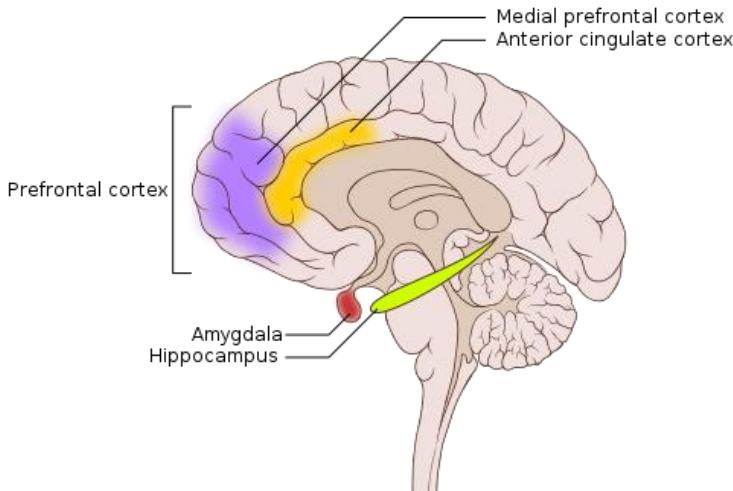


Figure 1. Parts of the brain affected by trauma.

Image credit: Patrick J. Lynch, medical illustrator C. Carl Jaffe, MD, cardiologist FvasconcellosWhidou, CC BY-SA 4.0 <<https://creativecommons.org/licenses/by-sa/4.0/>>; via Wikimedia Commons https://upload.wikimedia.org/wikipedia/commons/c/cc/PTSD_brain.svg

When we are reminded of a traumatic incident, the amygdala — the seat of emotions and survival responses — gets stimulated and responds exactly as it would if we were experiencing that trauma for the first time. At this time, the prefrontal cortex is also suppressed, reducing our control over fear. This leaves us in a reactive state. The hippocampus, which discriminates between the past and the present, is similarly less active due to trauma.

Simply put, our brain perceives the memory of the traumatic event exactly like the actual event, interpreting unpleasant memories as threats. It fails to switch from the reactive state to a restorative mode, keeping trauma survivors in a continually alert state.

A TBI survivor faces problems in three realms: physical, cognitive, and emotional/behavioural [5].

Physically, TBI could affect the mobility of a person to varying degrees, depending on the area of the brain that is hurt. It could paralyse one side of the body (hemiplegia), make it weaker or less mobile (hemiparesis), impair senses of hearing, vision, smell, touch, and/or taste, cause issues with speaking, swallowing, controlling the bladder, and bowel movements, and balance and coordination issues.

Cognitively, TBI could hamper memory, language, attention, problem-solving skills, and empathy. One could struggle with planning and execution, abstract thinking, discerning right from wrong, initiating appropriate actions, and inhibiting inappropriate ones.

Emotionally, one could turn more impulsive, irritable, angry, preoccupied, apathetic, and dependent. They may also lack motivation and sink into depression.

REHABILITATION AND RECOVERY

Although trauma can cause permanent brain damage, our brain's ability to form new connections helps restore it and recover from the harmful effects of trauma. But it's an ordeal that requires a combination of treatment and rehabilitation, which varies from person to person and with time [6].

For patients with severe injuries, full care may be necessary as they find it challenging to perform even routine tasks like bathing. In such instances, they are admitted to a hospital and have an occupational therapist and a nurse to help them through their day. They are monitored constantly and provided with specific exercises and medication that are helpful as patients are always in the hospital. The proximity also helps in immediately treating any complications that may arise during the course of recovery. In less severe cases, the rehabilitation program is largely run outside the hospital with scheduled visits to the hospital. There is also the advantage that the patient is home in a familiar and possibly comfortable environment, which would help with a faster and better recovery. The only disadvantage is that immediate care for complications is difficult. In some cases, the rehabilitation process could be entirely at home, especially in cases where the patient may be too old to travel or is bedridden. There are also comprehensive day programs run at specialised rehabilitation centres that are established to treat people with trauma. Along similar lines, except with more involvement, are the independent living centres where one can board and receive constant care similar to an inpatient hospital program [7].

In all these instances, the care providers use a combination of therapies that include physical, occupational, psychiatric and speech therapies. Physical therapy includes special exercises for the limb that help improve locomotion, and medicines to combat joint pain or any other kind of imbalance in the body. Occupational therapy, which is similar to physical therapy, helps patients with neurological injuries in doing daily activities like washing dishes, bathing, brushing their teeth, etc. In short, it helps the patients get back to their normal routine after treatment. Psychotherapists also evaluate the patients frequently to ensure their mental well-being. Speech and language therapists help patients communicate, eat, and drink properly. The recovery process might vary from weeks and months to even entire lives, depending on the kind of injury, the treatment, and the patient's response to it. Even after rehabilitation and recovery, TBI patients must take good care of themselves and may have certain restrictions. They should also be mindful of any signs of resurfacing symptoms of TBI [8].

Muhammed Nadeer Musthafa, a consultant speech pathologist at Max Super Speciality Hospital, Dehradun, reflects on what motivates him to treat individuals after TBI, "The requirements and capacities of each individual after a TBI vary. We work to increase their capacity to operate at home and in their community during recovery. When they receive social and emotional support, I'm always excited to see the progress they can make."

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Blindsight, and how it helps us see the workings of the human mind

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SUMMARY

Blindsight is an intriguing disorder that offers a glimpse into the fascinating way the brain processes information and perceives the world. Neurological disorders like blindsight offer a starting point to delve further into the bigger questions of neurology, neuroscience and psychiatry, and what the implications of their findings may entail. This article summarizes the idea of blindsight, and how it helps us understand the human brain better.

* * * * *

Mr L (name withheld for privacy) hands his cane over to a person on his right. Completely blind for all practical purposes, he has to walk the length of a room, full of obstacles without any help. It sounds like a cruel joke, but the researchers get what they suspected: without "consciously" knowing it, Mr L has traversed the room moving around obstacles as if he was never blind [1].

If you were blindfolded and asked to move around a room, you would move randomly, trying to avoid as many obstacles as you can. Mr L's condition, caused by a stroke that injured his primary visual cortex, is blindness. He has had a blindfold on his eyes for years now and believes he is taking completely random paths in experiments like this, yet navigates his way around obstacles seamlessly [2].

* * *

How the brain helps us see things, at least on the need-to-know basis for this condition, is depicted in this image (Fig. 1). The image shows the circuit involved in vision: the visual pathway. Impulses encoding the image forming on the retina make their way out of the eyeballs via the optic nerve, like a cable comprising several wires (in this case, nerve cells called "neurons") carrying signals to a destination. The optic nerves cross to form the optic chiasma (which means "the crossing") and then continue as optic tracts taking the signals to various parts of the brain.

Any problem in the visual pathway prevents the flow of impulses, causing blindness in various parts of the individual's field of view. Problems in the pathway of transmission of signal beyond this aren't as easy to consider.

* * *

Human brains are a marvel of evolution. It has developed over a millennia almost like a metaphorical skyscraper of living tissue and electrochemical activity touching the clouds of all that we hold in awe about human civilization: ethics, morals, music, language, creativity, compassion and everything else. We see a reflection of the process of evolution, which happens over time, in the layout of our brain: various parts of the brain are primary to different functions and the parts or circuits of the brain involved in activities of more primaeval origin like breathing are controlled primarily by what we call the "lower areas" closer to the spine

and the brainstem [3]. We ascend from that to functions like feeding, emotions, writing and language, each of them in progressively “higher areas” of the brain based on their complexity and importance.

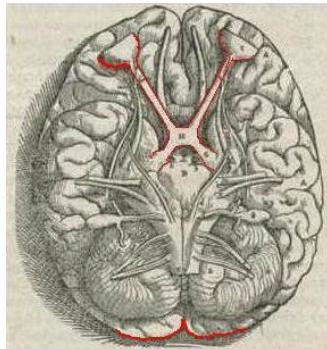


Fig. 1 The visual pathway

Sketch by Andreas Vesalius from his book De Humani Corporis Fabrica Libri Septem (see ref 5)

With that in mind, we return to blindsight and why “consciously” is such an important word. We like to believe that almost all of what we do is “voluntary” — a word neuroscience uses to describe actions we can control. However, we also know that breathing is not a voluntary process: you could change the rate or breath consciously for a while, but it continues even without your conscious engagement from the brain. Being a function of the “lower areas”, this fact isn’t too difficult to accept. But the idea that functions like vision, which is not a part of direct consciousness, also have a significant part, isn’t that easy to believe.

Here are a few examples of this, the first of which is what you are doing at this very moment. As you read these lines, your eyes run over the sentence from left to right. But there is a pattern to those movements you probably never have bothered to consider. These movements, called “saccadic movements”, involve massive amounts of constant processing and feedback that are subjects of extensive research. While reading is a very conscious process, your brain lets the subconscious handle the relatively less significant part of moving the eyes along the page for an easier flow so you don’t have to bother about it every time.

We now apply this concept to blindsight. It is hypothesized that of the two pathways of visual processing in the brain, the “old pathway” remains intact allowing for object tracking and spatial mapping as was important even in the most primaeval organisms — think of a frog on a lilypad tracking a slowly buzzing fly and gobbling it up as it comes close. The “new pathway” on the other hand carries out processes that distinguish the what and how of the world we visualize. As VS Ramachandran puts it, it is possible that with the new pathway blocked “visual awareness winks out” [4]. You may not be able to consciously know that you are seeing something or gauge what you are seeing, but the more primaeval aspects of vision are eerily maintained.

Having said that, I must mention that we don't fully understand blindsight yet — we are only beginning to, rather it is one of the many mind-boggling neurological phenomena that not only grab our attention to where we can learn more about the brain but also constantly challenge and push the boundaries of the field. Questions are just as important as, if not more than, the answers. With the rapid progress in science and the recent advances in technology, we are getting answers galore as we witness studies of the mind at the intersections of fields like neurology, psychiatry and psychoanalysis!

Scientists are still grappling with the many challenges that face our understanding of the origins of blindsight in the brain. These investigations and the processes reiterate how little we know about the human brain even with all the technological advances and decades of research. Although our explorations have continuously yielded new, exciting discoveries, they must not satisfy us into thinking that we know it all; rather, they must challenge us to look further. After all, I like to believe that when Socrates said, “the more I know, the more I realise I know nothing”, it wasn’t in acceptance, but in resounding defiance against being blind to that which we can see if only we try.

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Glioblastoma Multiforme: A Boggart in Disguise

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SUMMARY

Aditya Dwivedi- an eight-year-old Potterhead- is diagnosed with Glioblastoma Multiforme, a lethal brain tumour. The young boy bravely undergoes surgery, radiotherapy, and chemotherapy- the standard of care in cancer treatment. The magical wizard world helps him latch on to hope. He is eventually declared disease free. The notorious disease eventually returns and wreaks havoc on the Dwivedi family again. Aditya's story is inspired by the real stories of several children who die of glioblastoma every year. Through his story, we explore the various nuances of this deadly cancer and decipher some current scientific efforts to tackle this boggart in disguise.

A DAY IN KOKILABEN HOSPITAL

Lying on his bed- in room number 386 at the Kokilaben Dhirubhai Ambani hospital- little Aditya looked over at his mother who gave him a reassuring smile. Behind her smile hid months of sleepless nights and worrying for her 8-year-old son. The Dwivedi family had been in and out of the hospital several times over the last few months.

It all started on 23rd March 2021 when Aditya suffered from a seizure and had to be rushed to the hospital from his school. Doctors did their due diligence; they scanned the whole body but could not find anything. "He was probably exposed to some stressor", they said. Little Aditya's condition only worsened; he got disoriented during physical exercise, lightheaded while standing out in the sun, and drowsy during TV time. His parents made sure that he got the medical attention he needed, but the doctors could not find anything.

Aditya perked up on his bed as he saw his father with the third instalment in the Harry Potter series in his hands. Perhaps escaping into the magical world was his only solace. He jumped up and hugged his dad, snatched the book from his hand, and started reading right away- tuning out of the discussion that his parents were having. Mr Dwivedi explained to his wife that the doctors had scheduled their son for a CT scan and an MRI later that afternoon and were hoping to find some answers soon...

At 5:00 p.m. that day, the doctors got the answers they were looking for. With a heavy heart, the head of pediatric oncology at the hospital informed them that their son had a brain tumour.

Brain cancer is a diagnosis that is bound to cause fear in any patient since the brain is in charge of the entire body's functioning. Brain malignancies typically manifest as neurological dysfunction such as seizures, strokes, instability, and paranoia. They can easily be confused with mental health diseases. Lesions may not be visible in brain scans at an early stage (like in Aditya's case). By the time it is detected, it is likely that the disease has progressed considerably [1].

Patients frequently are unaware of the fact that not all brain tumours are fatal. Both benign (localized and harmless) or malignant (invasive and harmful) tumours may affect the

functioning of the brain; some cancers of the brain, such as hemangioblastoma, germ cell tumours, and ependymomas, are treatable with curative therapy. Hemangioblastomas are blood vessel tumours of the brain. They are typically harmless and easily treated [2]. Germ cell tumours, germinomas to be specific, develop when the spermatocyte (in males) and ovum (in females) accidentally travel to other parts of the brain [3]. Germinomas most commonly afflict young children and have a 90 percent survival rate [3-4]. Another type of brain cancer develops in the brain's supportive cells, known as glial cells [5]. Ependymal cells, a kind of glial cell, are hairy epithelial cells in the brain that support nerves and keep the cerebrospinal fluid balanced. Depending on the origin and location of the tumour, pediatric ependymomas have a survival rate of 50-70 percent [6].

THE BOGGART

Back in the hospital, Aditya has to undergo an urgent craniotomy. Surgeons will open his skull and remove most of the tumour mass from his right cerebral hemisphere. A biopsy of this tumour mass will give doctors more insight into Aditya's prognosis. After surgery pathologists will inspect his tumour sample and then a team of oncologists will come up with his treatment plan.

After the surgery, Aditya although weak- excitedly exclaimed to his parents that he had just gone to sleep and felt no pain during the surgery.

The surgery had gone smoothly and surgeons had removed most of the tumour mass from his right cerebral hemisphere. They had discovered that the tumour was more diffuse than what the scans had revealed. Biopsy of the tumour mass revealed that Aditya had a rare and aggressive form of brain cancer: glioblastoma multiforme.

The Dwivedi, a simple middle-class family- did not comprehend complicated words like "glioblastoma multiforme". All they heard was a rare form of cancer. Their entire world seemed to be falling apart. All they wanted was for their cheerful son to be healthy and romp about the house pretending to be a wizard.

"Glio" refers to the supportive glial cells in the brain. "Blastoma" refers to malignancies in unspecialized precursor cells known as blast cells. "Multiforme"- quite self-explanatory indicates multiple forms.

Glioblastoma multiforme (GBM): a malignancy in the undifferentiated glial cells [7] in the brain that comes in several forms. It usually develops in the brain and/or spinal cord. It is one of the most invasive cancers with a dismal prognosis. In adults, ranging from 55-64 years having the five-year survival rate is 6 percent [8]. In children, it is 20 percent [9]. This metric only accounts for 'survival' and not the quality of life of the patient.

The origins of glial cell cancers can be traced back to the Napoleonic era when two scientists named Berns and Abernety published the first scientific reports describing the gross morphology of primary, non-metastatic tumours in the brain and spinal cord sampled from autopsy specimens. Rudolph Virchow coined the term "glioma" and recognised and classified gliomas (tumours of the glial cells) into low-grade and high-grade gliomas. Percival Bailey and Harvey Cushing's contributions further laid the groundwork for today's modern classification

of gliomas. They were also helpful in describing and naming glioblastomas (grade IV gliomas or high-grade glioma) [10-11].

Studies have shown that GBMs originate in astrocytes. These are star-like glial cells that control the metabolism of neurons and control homeostasis. However, the ‘multiple forms’ within the GBM can be attributed to “neural stem cells, neural stem cell-derived astrocytes, and oligodendrocyte precursor cells” [12]. Glioblastoma stem-like cells possess multipotency [13] that can give rise to a variety of specialised glial cells and pericytes [14] in the brain, making cancer highly heterogeneous and plastic [15, 16]. Under the microscope, GBM cells come in a variety of shapes and sizes including round, slender, and diamond-like structures [17]. Plasticity also allows GBM cells to trans-differentiate into vascular endothelial cells. This means that these cancer cells can differentiate into blood vessels and support their growth by obtaining nutrients from the bloodstream. Access to the bloodstream also allows them to spread to neighbouring brain tissue faster. This phenomenon is known as metastasis [15,16,18,19].

Aditya has to undergo six weeks of radiotherapy. This is expected to damage all actively dividing cells in the brain and kill any remaining cancerous cells in Aditya’s brain. After this, doctors will scan his brain again and depending on his response might suggest the chemotherapeutic drug- temozolomide. This is the standard of care in oncology: surgery, radiation, chemotherapy.

The mantra that the doctors had adopted “wait, observe, and respond” was unsettling for Mr Dwivedi who wanted concrete answers, concrete steps.

Unfortunately, “observe and respond” is the standard in the field of oncology. Clinicians usually have to wait for a while to find out if the patient is responding to a treatment. If a treatment fails, another one is tried. Each individual is unique and their response to therapy is unique and cannot be predicted with certainty. An individual’s response to therapy is influenced by several factors, including their genetic makeup [20], responses to environmental signals [21], general state of health, hormones, interactions between genes and their products, and potentially transient DNA alterations (epigenetics) [22].

Scientists have attempted to categorise GBMs into many subtypes. The presence of an IDH (isocitrate dehydrogenase) mutation is one such indicator of a favourable prognostic marker. This gene encodes an enzyme that causes oncogenic changes in cells. According to research, the presence of this mutation boosts the body’s immune response against the tumour, boosting the overall prognosis [23, 24]. However, the response to therapy, for two patients possessing the same IDH mutation is also likely to be very different. This is only one layer of complexity in medicine.

On 20th September, 2021 the Dwivedi family finally received some good news. Aditya’s cancer was in remission. Doctors advised them to keep coming in for brain scans since GBMs are notorious for their ability to recur. GBM has a recurrence rate of 90% [25].

The family celebrated this news with a small party at Pizza Hut!

THE BOGGART RETURNS

“But... but... we thought Aditya was cured... how can this happen doctor?”, his mother asked.

7 months later, Aditya's GBM had relapsed- it had spread to his brain stem.

You may have heard that cancer cells are ‘normal cells gone wrong’ [26] - and hence may be considered a ‘freak of nature’ [27, 28]. As multicellular organisms evolved, so did the division of labour between cells [29]. Cells in our body act as a unit for the survival of human beings. In any population, the individuals best adapted to survive in a certain environment, survive and thrive. Natural selection selects the cells that are fittest to survive the stressors in their immediate environment as highlighted by the cancer tree in the figure (Fig.1) [30-33]. Thus, cancer cells are essentially competing with their neighbours for survival by hijacking the body’s natural mechanisms.

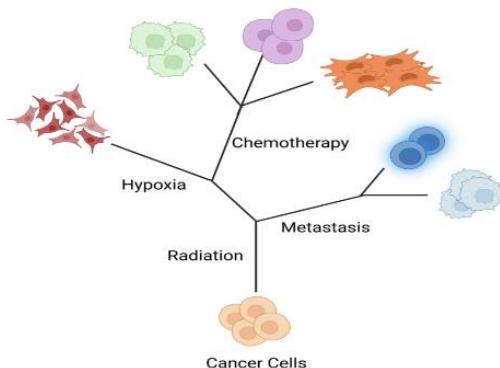


Figure 1. The Cancer Tree - Exposure of cancer cells to various stressors (such as therapy) may result in cancer lineage diversity. The tree depicts various possible paths. Some of these cells may retain their original characteristics (shown by the same form); others may be completely different. Readers may see similarities between this tree and Darwin's tree of life [34], in which one species evolves into different species.

Created with Biorender.com.

But how did this happen? Why did cells from the body turn against the body?

It seems that in the case of cancer, cancerous cells begin acting as their own entity (like unicellular organisms) [35, 36]. Selection is now acting on this entity. And these entities couldn't care less about their surrounding cells. Such is the devious nature of this malady.

These cells now compete for space and nutrients and create a toxic environment [37, 38] for the normal cells. When therapy is applied, most of the cancer cells are likely killed and damaged. However, there might be ‘some’ cells that survived the onslaught. Consider the case of radiotherapy. Radiotherapy damages the DNA of actively proliferating cells. [39] But what about cells that became dormant? They can easily escape therapy and start dividing again under the right circumstances [40, 41]. Another possibility is that certain cancer cells start using glycolysis and releasing acidic products. This deprives the tumour of oxygen, which is

essential for radiation to work. Thus, glycolytic cells end up reducing the efficacy of therapy [42]. Studies have also shown that stem cells promote drug resistance in cancer, due to their ability to self-renew and differentiate into other specialised cells [43].

What makes GBMs even more complex, is their heterogeneity and plasticity. The high levels of diversity in the case of GBM ensure that cancer can evade almost any trap set by the doctors. In 2011, Verhaak et al. identified four subtypes in GBMs namely: proneural, neural, classical, and mesenchymal and stratified them on the basis of genetic signatures using bulk sequencing technology [44].

A latest study performed by the Harvard Stem Cell Institute discovered four subtypes of GBM in pooled patient samples using single-cell RNA sequencing technology [45]. Each state was a relic of a separate brain developmental stage and was labelled as neural progenitor-like (NPC-like), oligodendrocyte-progenitor-like (OPC-like), astrocyte-like (AC-like), and mesenchymal-like (MS-like) [46]. These subtypes were able to recapture Verhaak et al.'s original categorisation. They also discovered the driving genes for each subtype. They were able to demonstrate that certain cancer cells were in a hybrid state between two subtypes and that cells in one state could transition to any of the three other states (Fig. 2).

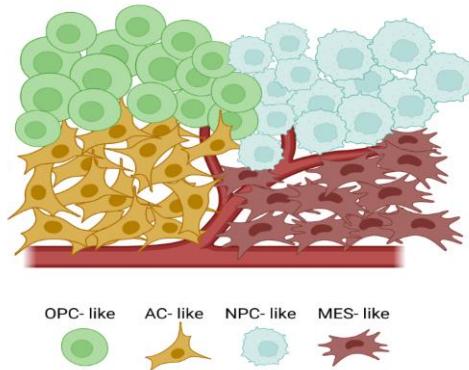


Figure 2. Shapeshifting Nature of Glioblastoma Multiforme - A GBM tumor can be comprised of four possible cellular states as identified by Nefel, C. et al. They are namely: neural progenitor like (NPC-like), oligodendrocyte- progenitor- like (OPC-like), astrocyte-like, and mesenchymal- like states. This contributes to the heterogeneity of the cancer. Furthermore, cells in each of these stages can transition between different states. This makes the cancer more plastic.
Created with Biorender.com.

For the best growth conditions, it is believed that the tumour evolves to maintain a certain balance across all four subtypes. If therapy is administered, the relative frequencies of the subtypes will alter and restore a new equilibrium that is optimal in the new environment [46].

So how can doctors apprehend the next ‘move’ of cancer? What other tricks does it have in store? With the molecular revolution, scientists have taken leaps and bounds in understanding the make-up of tumours [47] and yet the answer eludes us.

“Ohhh! So, I have shape-shifting cells... like... like Prof. McGonagall! She could shapeshift from a witch to a cat to a cup!!”

The nurses sadly smiled at Aditya, who managed to find something joyous in what was killing him.

20 days later, still clutching onto his Harry Potter book, little Aditya uttered his last words, “I love you, mamma- papa.”

In memory of their beloved son, Mr and Mrs Dwivedi set up the Aditya foundation to fund treatment and research in pediatric brain cancer.

The story of Aditya was inspired by children like Tom from Ellesmere Port in England [48] and Trenton O’ Brien from Connecticut [49], who succumbed to brain cancer. The pain and suffering that comes with losing their children plague the parents forever. Pediatric tumours are more aggressive than adult tumours at the time of diagnosis because childhood cancers are infrequent, making early detection difficult. [50, 51]. The story of Aditya continues to be the reality of many.

THE HOPE

To ‘beat cancer at its own game’ [52], scientists and physicians need to join hands and apprehend the next ‘move’ of the cancer. Apprehend how it’s going to respond to therapy. Apprehend how it is going to evolve. Cancer evolution is a complex and dynamic process. Due to various environmental cues, a wide variety of clones are generated. Therapeutic approaches that aim to eliminate these clones [53], inevitably create tremendous selective pressure for the overgrowth of resistant clones [54]. Thus, several groups are working on dissecting the clonal evolution of cancers. A team at Mayo Clinic, led by Dr Kristin Swanson is using mathematics to predict the trajectories of GBM in patients [55]. Innovative strategies like adaptive therapy [56, 57] and evolutionary double binds [58] have also been explored by scientists. These strategies focus on targeting cancer evolution. In adaptive therapy, the goal is to prevent the emergence of resistant cells while maintaining a low tumour volume (not eradicating the tumour). In evolutionary double binds, the goal is to use drugs that act in synergism. Resistance to one drug leads to susceptibility to the other drug and vice versa [59]. This traps the cancer cells and prevents the evolution of resistance. While these therapeutic regimens have undergone clinical testing for prostate cancer and leukaemia [57, 60], it has not been explored in GBMs. Due to their risky position, lack of a distinct mass with distinct borders, and lack of helpful markers [61, 62] of tumour volume, GBMs are very challenging to treat [63].

At 46, Beau Biden, politician, lawyer, advocate, and son of Joe Biden, died of glioblastoma [64]. The sense of loss propelled the US President to launch The Cancer Moonshot effort [65, 66] to turn the corner on this disease. We can only hope that this effort brings about breakthroughs to finally bring an end to this shape-shifting boggart of a disease.

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Can we predict our brain?

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SUMMARY

Alzheimer's disease is a major neurodegenerative disorder prevailing worldwide with no current cure. An early diagnosis can, however, delay its progression and improve the quality of life. Here, we discuss the Cookie-Theft Test that has been used by clinicians since the 1970s to predict the onset of Alzheimer's disease. Modelling this picture description task using artificial intelligence enables the forecasting of this deadly killer over 7 years before its onset.

INTRODUCTION

Charlton Heston, a World War II veteran, and an Oscar Award-winning actor, spent the last few years of his life tragically unaware of his glorious past and slowly losing his ability to remember his loved ones. Eventually, he passed away bedridden, surrendering to a disease that stole a lifetime of memories from him. Heston made a touching farewell speech after being diagnosed. "If you see a little less spring in my step, if your name fails to leap to my lips, you'll know why. And if I tell you a funny story for the second time, please laugh anyway," he said [1].

Charlton was suffering from a neurodegenerative disorder called Alzheimer's disease (AD). Neurodegenerative diseases primarily effect our brains. Specifically, AD falls under an umbrella term called 'dementia', meaning disorders whose symptoms are associated with an ongoing decline of brain function (Fig. 1). The brain is robbed of its ability to perform important tasks such as thinking, remembering, and reasoning.

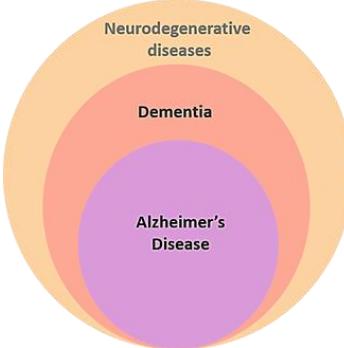


Figure 1. A breakdown of neurodegenerative disease classification.

The human brain constitutes only 2% of our body weight, and yet it is the consciousness of our existence. It acts like a computer connecting the different organs of our body through a network called the nervous system. With its complex functionality, our brain is considered superior to any man-made computer ever created. And still, as we age, this marvellous neural network is prone to 'malicious' attacks (or neuro-degenerative diseases). When we hear stories

of people suffering the wrath of neuro-degenerative diseases, we wish that we had an in-built anti-malware to prevent these attacks. With the rise of technology, it doesn't seem like a science-fiction dream anymore. Scientists are constantly developing computational tools to predict the early onset of neurodegenerative diseases, to help doctors understand and treat them better. In this article, we will take a sneak peek into the notorious AD and discover how computers are helping diagnose the disease faster.

ALZHEIMER'S DISEASE (AD) IS A CONSTANT UPHILL BATTLE

Did you know that AD is a leading neurodegenerative disease, accounting for nearly 70% of dementia cases in the world [2]? In 2016, it was found that around 2.93 million people suffered from dementia in India [3]. Typically, the progression of this disease varies widely amongst people, from an early age to middle and late. However, someone with an early diagnosis may have a longer life expectancy compared to someone diagnosed at a later stage. Accordingly, the average lifespan of someone with AD is 3-11 years after diagnosis, but some people live for 20 years or longer [4].

So, what exactly happens to the brain in AD? AD is often accompanied by massive protein deposits (such as amyloid plaque or tau tangles) in the brain. These traffic blocks affect correct connections among brain cells (neurons) [5]. Such an afflicted mind is characterized by nearly 7 phases, which progress from being mildly forgetful, and misplacing belongings, to forgetting loved ones and losing a major sense of autonomy over bodily functions like eating, swallowing, bladder control, etc., [6].

A NEEDLE IN A HAYSTACK: DIAGNOSIS OF AD

Now that we have some background, it's time to understand how doctors diagnose AD. For disorders such as AD that share symptoms with many other neurodegenerative diseases, physicians suggest a battery of tests to arrive at a near-accurate prognosis.

A physician would begin with a detailed medical history of the patient and move on to assess all current vital signs including blood pressure, heart rate, temperature, and pulse rate [7]. This would be followed by blood and urine tests to rule out other causes of dementia such as vitamin deficiencies and hormonal malfunctions [8]. The next test is possibly the most relevant, a mental state test. In this test, memory and cognitive skills are judged. The results of these tests are compared to those of normal individuals of the same age, gender, and education. The Cookie-Theft Test is a classic example and we will elaborate on it in the coming sections. The last test pertains to imaging the brain which includes magnetic resonance imaging (MRI), computerized tomography (CT) and positron emission tomography (PET). They reveal regions of brain shrinkage, poor sugar metabolism, and amyloid and tau protein build-up in the brain (common symptoms of AD). They also rule out other illnesses like brain tumours [7].

A SMART COOKIE

It is believed that the Cookie-Theft Test (CTT), a picture description task, is uniquely positioned over other forms of language assessment to reveal cognitive impairments resulting in AD. In this task, a simple black and white drawing of a pre-occupied mother washing utensils while two children try to smuggle cookies behind her back is used (Fig. 2). Patients are asked to describe the picture and the way they describe it is considered a cue to their linguistic abilities.

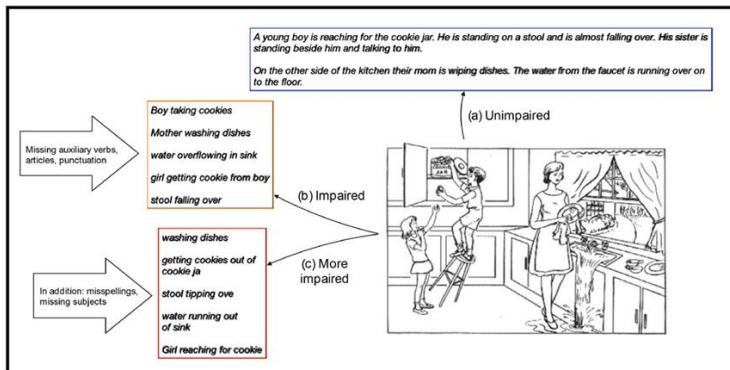


Figure 2. The Cookie Theft Test.

This illustration depicts the popular picture description task used to judge cognitive abilities.
Image credit [10]

This test is a revelation into how linguistic markers can predict the onset of AD. Doctors assess the patient's ability to provide clear, descriptive, grammatical-error-free answers, which gives them an understanding of his/her brain function (Fig. 2). The question arises, how can we connect the linguistic abilities of a person as markers for early-onset AD? This is because our brain is a complex machine and even a simple task such as describing a picture utilizes extensive networks in our brain. Neurodegenerative diseases like AD can slowly hamper the productivity of these networks.

The people that tested for CTT were originally a part of a large and prominent study called the 'Framingham Heart Study (FHS)'. FHS was a decades-long health investigation that began in the 1940s [9]. Recently, the dataset from this study helped scientists at IBM and Pfizer design an artificial intelligence (AI) model to predict the onset of AD in patients before they reached 85 years of age [10]. Curious about how they did it? Let's break the whole process down into simpler steps.

AN AI MODEL THAT MADE USE OF THE COOKIE

Scientists at IBM and Pfizer made use of the CTT, which is not entirely new, but until now, it was only subject to human assessment, that of a doctor. Now they trained a computer to pick up many variables (which is not possible for a human) from the same CTT sample submitted by normal and cognitively impaired individuals. Over 87 language variables including misspellings, use of punctuation, uppercasing, verbosity, lexical richness, and repetitiveness were used. Beyond this, they looked at age, gender, education, reasoning, object naming, memory, and attention, abstraction, and test results from other cognitive assessment tests [10]. Altogether, the computer program could differentiate between a diseased and a normal brain.

They then tested this program on the dataset from the FHS. FHS had the unique advantage of a database of participants' cognition and medical test results every few years. Thus, it was possible to track if and when a participant developed the cognitive disease. Scientists from IBM and Pfizer found that when they used their program on the information provided by

FHS participants, they could predict the occurrence of AD with over 70% accuracy and nearly 7.5 years before the participants demonstrated medical symptoms [9]. This was remarkable considering doctors' diagnosis at that time was only about 60% accurate [11].

HOW DOES AI SHAPE OUR FUTURE?

Such machine learning-based programs seem path-breaking. However, they will not replace the traditional clinical standards. But they could be used as an inkling of 'all' is not well' and a subsequent recommendation for a neurologist visit and a scan.

For now, imagine having an app on your phone that runs in the background as you speak and gives you a report of your cognitive state of mind! Your health at your fingertips, literally.

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Ardem Patapoutian

(Nobel Prize in Physiology or Medicine, 2021 for enhancing our understanding of how we sense temperature and touch)

PART III Encounters of daily life

Late to Bed and Late to Rise? Blame It on Your Teenage Brain and Genes!

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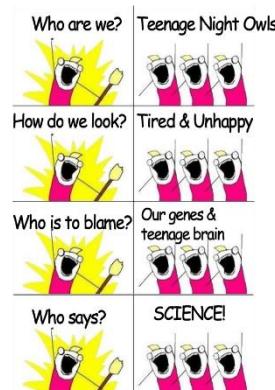
SUMMARY

Like most of our traits, our sleep schedules are partly genetically determined, and partly influenced by social, cultural, and environmental factors. Neuroscience research shows that the tendency of teenagers to go to bed late at night and be slow to rise in the morning, is more than just a personal preference. The dramatic hormonal changes during puberty can alter the brain circuits that regulate sleep.

INTRODUCTION

Fourteen-year-old Dhruv struggles to get out of bed as a dozen alarms go off. He stumbles through breakfast and hardly makes it through the morning classes at school, bleary-eyed. He manages to catch some shut-eye as the teacher drones off during the afternoon class, only to spring to life, wide-eyed and alert, at 10 PM—just as the rest of the house is descending into slumber.

Parents and teachers tend to blame groggy, sleep-deprived teenagers for having brought this on themselves and many are quick to label them as “lazy” and “rebellious”, but researchers have proven this is, in part, based in biology.



OUR INTERNAL TIMEKEEPERS

We are children of the sun; our bodies march to the beats of the sun’s drum. Our sleep patterns are heavily influenced by the earth’s 24-hourly rotation around the sun and our exposure to sunlight. From an evolutionary perspective it is crucial for the survival of any species: to be awake and forage for food when there is light and to sleep when it is dark and vulnerable for predators.

Two mechanisms regulate our sleep. First: The **Sleep-Wake Homeostasis**. If you’ve wondered how coffee (and caffeinated energy drinks) keep you awake, it is by tampering with the action of adenosine, a key mediator in this very mechanism. Adenosine accumulates in the brain of a person who is awake, and chemically builds our sleep drive. The more time we are awake, the greater the pressure to sleep, and the more time we are asleep, the greater the pressure to wake up [1, 2].

Second, our inbuilt 24-hour clock: the ‘**Circadian Rhythm**’, from the Latin for “about a day”. A lot more than just our sleep is influenced by this rhythm for it governs the brain, gut, kidneys, and hormones. A good clock must not only keep regular time but must also have a way to be reset. Environmental cues, termed ‘**Zeitgebers**’ (German for “givers of time”), can nudge our

circadian rhythm forward or backward. The most potent of these is light, particularly the kind at the blue end of the spectrum.

There is not just one uniform clock throughout our body, rather each cell has its own mini-circadian rhythm! To synchronize their rhythms, we'd need a "Master Clock" [1 - 4].

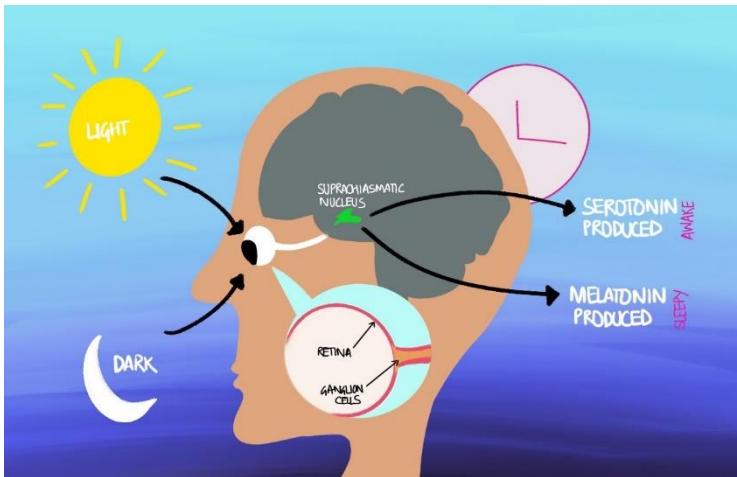


Fig. 1 The Effect of Light on the Suprachiasmatic Nucleus.

The retina of our eye has receptors that communicate the information about light and dark as electrical signals through the optic nerve. If you follow the path that the optic nerve takes from the eye into the hypothalamus of the brain, you'd reach the **Suprachiasmatic Nucleus (SCN)**. As the master clock, this tuft of neurons acts as the control room for the individual clocks in our organs. To adjust our body to the 24-hour light/dark cycle that we live in, the SCN ramps up the release of melatonin - the sleep hormone, at night and suppresses its release in the morning, making us feel awake [1-4].

OF LARKS & OWLS: UNDERSTANDING CHRONOTYPES

Dhruv discusses his struggles of getting up early with his friend Karan but it only leads to more disappointment as Karan has no trouble getting up at 5 AM and getting in a few hours of early morning studying.

We don't all wake up at the same time, go to bed at the same time, or execute tasks at the same time. This personal pattern of circadian rhythms causing our different levels of activity and alertness across the day is called a "**chronotype**". Some people perform their best in the evening and find it impossible to wake up as early as 6 AM. While others can be early risers and become zombies by the end of the day. Your chronotype encompasses far more than your preferred sleeping patterns. Every natural mechanism inside you, from body temperature variations to cortisol synthesis, is controlled by it.

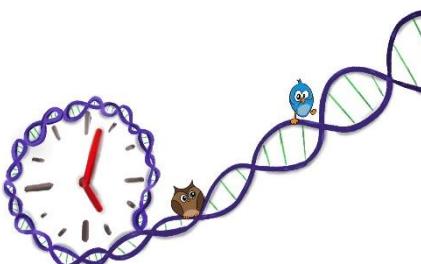
Chronotypes aren't a black and white affair; it is a range of greys, from extreme morningness to severe eveningness. Chronotypes can fall into three "buckets": Morning type (**Larks**), Evening type (**Owls**), and Intermediate (**Third Birds**). Larks get up and go to bed early, with their energy levels plummeting in the early afternoon. They also sleep for longer periods of time than evening types. On the other hand, owls sleep later and experience a drop in energy in the late afternoon. Intermediate chronotypes involve people who lie somewhere between morning and evening type. In the last few years, another category was noticed: intermediate or bimodal. These people have a morning preference for some activities and an evening preference for others.

So, which chronotype do you think you are? Take the [Morningness-Eveningness Questionnaire \(MEQ\)](#) by Horne-Ostberg or the [Munich Chronotype Questionnaire \(MCTQ\)](#) created by Dr. Roenneberg to find out.

The variability in chronotypes in the population could make sense from an evolutionary sense. If someone is always awake and able to watch over the group, we're more likely to stay safe. According to the "[Poorly Sleeping Grandparent hypothesis](#)", older clan members are up early or stay up at night, watching over the clan while the others sleep soundly and safely.

WHAT MAKES OUR INTERNAL CLOCKS TICK?

Your DNA can reveal which chronotype you may be. **Clock genes** (like all genes, you inherit them from your parents), play a crucial role in determining chronotype [3]. For example, those born with a "shorter" variant of the Per3 gene are more likely to be larks and those born with a "longer" variant of the gene tend to be owls.



We first learned about the existence of these genes when researchers Jeffrey Hall, Michael Rosbash, and Michael Young studied what drove the circadian rhythm in fruit flies and discovered the **Period gene**. We now know that such genes are present in humans as well. For their path-breaking discovery, the three researchers were awarded the [Nobel Prize in Physiology or Medicine](#) in 2017.

THE BIOLOGY MAKING TEENS SLEEP ZOMBIES

Being a teenager is hard: one has to get up at 6 AM to get ready for school, sit through classes, prepare for tests, and hang out with friends - all while keeping up with social media and the world! Is it even possible to get 9 hours of sleep? It's difficult. These grueling demands can influence the two mechanisms that regulate your sleep.

But merely these social factors fail to account for the sleep time delays. Turns out that raging hormones and the developing teenage brain cause a lot more than risk-taking behavior. They also affect one's chronotype!

Young children are generally larks: they usually get up early, bustle about through the day and tire out by early evening. Around puberty, these young larks metamorphose into owls: wake up later, gain energy during the late evening, and fall asleep well after everyone else, like Dhruv. The peak owl-ness is around the age of twenty and then gradually there is a return to being their natural chronotype. As one turns older, there is a gradual shift to being larks, once again [1, 5].

Summed up: **teenagers are disproportionately owls, just as people under twelve and above sixty are disproportionately larks.** *This change in sleep patterns is so marked that the halt of sleeping-in has been proposed as a biological marker to signify the 'end of adolescence'* [1, 8].



Adolescence brings with it a slower build-up of sleep pressure - even with prolonged sleep deprivation. The sleep-wake homeostasis is thrown out for a toss. As a teen, you have to be awake for longer periods to feel sleepy unlike your younger self, who'd tire out easily and fall asleep quickly. It doesn't end there. The peak of your melatonin levels is delayed by a few hours too, sending your internal clock off the rails [6 -8].

These social and biological effects make it harder for you, as a teen, to hit the bed early. When forced to wake up in time for school, your body is simply not prepared for it. This

misalignment in the biological clocks of teens and the social timing of schools and society has been linked to lower grades, obesity, and an increased risk of accidents. The mounting evidence has garnered enough attention to spark recommendations from the American Academy of Pediatrics and the American Academy of Sleep Medicine to push for schools to start later.

On weekdays, as a biological owl, you may stay up late but school compels you to wake up early. This creates a “sleep debt” that your brain tries to pay back by getting a few extra hours of sleep on weekends—you might sleep—in until noon or nap as your schedule frees up from the demands of rigid school timings. Coping with the 6 AM awakenings on Monday mornings, even if your body is screaming out for more sleep, is akin to navigating through multiple time zones. Researchers call this discordance in the sleep patterns on weekdays and weekends ‘social jetlag’ [9]. This chronic fatigue pushes many to begin consuming caffeine, smoking, and drinking alcohol. Social jetlag also increases your risk of suffering from depression [10].

GETTING YOUR RHYTHM BACK: MAXIMIZING SLEEP TIME AND QUALITY AS A TEEN

Sleep scientists say teens may be less sensitive to light in the morning and more sensitive to light at night. As you might recall, light signals the release of melatonin, the sleep hormone in our bodies.

First step: **ditch the electronics at night**. Computers, phones, and TV screens are a big source of blue light that skews our internal clock. If that’s hard, you can start by using a night filter on your devices or glasses with blue light filters to limit how much blue light your eyes get exposed to.

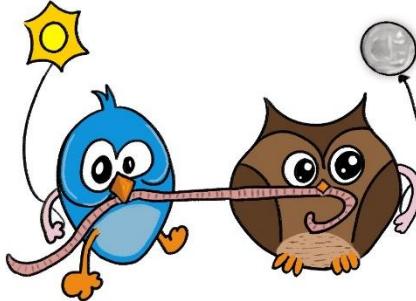
Next step: **soak in some sunshine**. School timings might not allow you to catch enough morning light. Try to use your snack or lunch break for a quick stroll or take to the playground to get some sun.

Another trick: **avoid caffeine near bedtime** to keep you from tossing and turning in bed.

Try maintaining a **sleep routine**. Hit the sack at the same time each night. Unwinding by reading a book, listening to music, journaling, or petting your furry friends might help.

IS A PARTICULAR CHRONOTYPE GOOD OR BAD?

Some studies suggest that there might be a link between chronotype and personality, health, and life satisfaction. Conscientiousness and agreeableness are two personality traits linked to larks. Neuroticism and receptivity to new experiences, on the other hand, are often associated with owls. People who have an evening chronotype are naturally inclined towards later timing of eating patterns, and physical activity, with a higher risk of developing cardiometabolic diseases and Type 2 Diabetes.



In a world that considers getting up early as a virtue, late risers are often branded as unambitious and tardy but our society is configured largely for the larks and third birds among us. Owls are left to feel like left-handers - forced to go against their natural instincts, to conform to a right-handed world.

Maybe some teenage night owls are indeed lazy (or in some cases, have a serious sleep disorder like delayed sleep phase syndrome), sure. But the rest, like Dhruv, have been sorely misunderstood, and science backs that claim!

Illustrations by: Vasudha Mishra

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Born this way: Hormones, Gender, and Sexuality

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SUMMARY

In general, choosing a sexual orientation that is contrary to your gender is an immoral way of living. But have you ever questioned why some people, although having a distinct biological gender, have different gender identities or sexual orientations? When discussing various sexual orientations, a crucial component is frequently overlooked: the brain. Your brain, which may be sexually separate from your genitalia, controls how you interpret your feelings.

INTRODUCTION

Suhani, 16, was assigned female before her birth and brought up with similar values in a conservative family with liberal parents. Growing up she always felt as if something was missing in her, she couldn't always relate to things that her female friends talked about. Things like crushes, boyfriends and girlfriends, fantasies, fairy tales, etc were beyond her interest which made her feel abnormal and detached from her peers. Later, she learns about the massive LGBTQ+ community. During a class about the brain and its function, Suhani curiously asked her teacher if the brain has a role in the sexual orientation and gender of a person. Unable to understand her, the teacher asked her to meet after class and ask these questions later. After school, she went to her teacher and elaborated on her question "some people just don't feel the way they are born, does the brain play a role in this?" and asked the teacher about the LGBTQ+ community. To which the teacher replied, "those people are trying to be fancy and most of them are just confused, nature created only 2 genders which are male and female. It's only natural that both only get attracted to each other". Confused, Suhani leaves the teacher's cabin with more questions than before. If decisions are made by the brain, then why is being something out of the social construct of the gender deemed unnatural? What does my gender have to do with whom I feel attracted to? and so on". Finally, she decided to approach her older brother with her questions. Although he was unable to answer her questions, he introduced Suhani to Joe. Joe explained to Suhani that this is just one example of misrepresentation of sexuality and gender even in the present time. It's important for people to understand that everything that a human does is controlled by our mighty brain including sexuality and even gender.

Sexuality is the way people experience and express sexual feelings. The sexual attraction is part of human sexuality. A person's sexual orientation is the pattern of sexual attraction. It is believed that there are 47 ways to categorize sexuality [1]. But to keep it simple let's just go over the most common ones, that is: -

1. Heterosexuality is the attraction to individuals of the opposite sex;
2. Homosexuality is the attraction to individuals of one's own sex;
3. Bisexuality is the attraction to individuals of either sex; and
4. Asexuality is no attraction to either sex.
5. Pansexuality is sexual, romantic, or emotional attraction towards people regardless of their sex or gender identity.

However, this does not capture the full spectrum of human sexual behavior [2]. People are attracted to individuals, independent of their gender [3].

Gender, sex, and sexuality are often conflated because of a lack of awareness and acceptance. We have socially constructed notions of gender roles and arbitrary norms of sexuality based on gender. In reality, gender and sexuality exist on a spectrum.

Gender identity is how individuals perceive their sexual interests and inclinations. One's gender identity can be the same or different from the biological sex assigned at birth. Biological sex is the different biological and physiological characteristics of males and females, such as reproductive organs, chromosomes, hormones, etc.

Joe proceeds to explain to Suhani what biological sex is by telling her about how the development of the genitals and brain. Let us zoom out of the picture and look at the time when the fetus is in the developmental phase and the genitals are developing. The genital differentiation of the fetus starts around the 6th week of pregnancy. During this period the fetus does not have any external organ to determine sex and there occurs the development of two types of ducts which later gives rise to the female and male genitals. In the fetus with XY chromosomes, there is a sex-determining factor (SRY-gene) on the Y chromosome which sends a message to the fetus and deactivates the female duct, later male genitals (testes) are developed which then produce the male sex hormone testosterone. Fetus with XX chromosomes do not have SRY gene and hence results in the formation of female genitals by the differentiation of female duct to ovaries and later female sex hormones [4]. All this results in the development of external genitalia in males and females by the 12th week and can be identified by ultrasounds. In essence Suhani, your biological sex has been given the female identity by your parents. Ironically whatever the ultrasound reports of the fetus say is considered more important than what the brain of the fetus might say. In India, it holds more value if it signifies a specific result, that's why banned in India under the Pre-Conception and Pre-Natal Diagnostic Techniques Act (PC-PNDT) which was enacted on 20 September 1994 [5].

This makes her puzzled and asks Joe "but why do I not feel like a female then and why do my interests and preferences are different from normal females, am I not normal".

Joe clarifies Suhani, you're absolutely normal, and this explanation of your sexual orientation or gender identity encompasses far more than just your assigned gender. Your brain, which is frequently impacted by hormones in the body, determines what you think, how you feel, who you are attracted to, what keeps you motivated, and around whom your reward neurons get excited. The catch here is that the development of the brain happens after the genitals have been completely developed. The brain also undergoes sexual differentiation exactly like the fetus's genitals, under the influence of hormones released by fully differentiated genitals. However, as the genital system entirely separates between the sixth and twelfth weeks of pregnancy, the sexual differentiation of the brain may or may not coincide with that of the genitalia. In reaction to sex hormones, notably testosterone secreted by the already differentiated genitals, the brain begins to develop sexually in the second half of pregnancy (Fig.1).

New connections are made as the brain develops, strengthening the neuronal networks that connect its various parts. The environment, genetic make-up, and substances that the fetal brain is exposed to affect how the neurons connect and interact with one another.

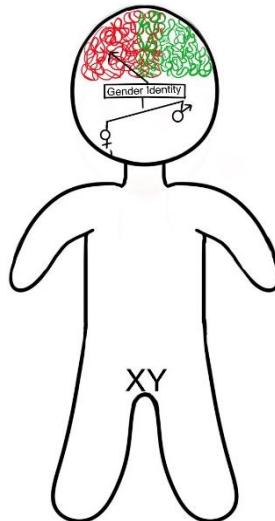


Figure 1: XY individual having a different sexual orientation like XX individual within the brain.

Sex hormones have organizational and activational effects on the sexual differentiation of the brain. Because of the organizational effect of hormones, there are permanent modifications to the neural connections and sensitivity of the brain before birth. Activational effects are influenced by the receptors on the neurons or their connections which are activated later in life, in response to the circulating sex hormones, like what happens during puberty [6].

Testosterone is the major sex hormone that affects the sexual differentiation of the brain. It is released by the male genitals, testes specifically, for the developing fetus. Testosterone itself affects the neuronal and synapse development of the brain, and its derivative estradiol also has similar effects in the brain. Estradiol is formed by the chemical modification of the testosterone by specific enzymes in the brain [7].

During the critical period of brain development, when it is sensitive to the circulating androgens in the body, estradiol is very much important to shape the brain neurons in a way that it shows sexual attraction towards the people with XX chromosomes. Generally, the fetal brain is insensitive to the maternal estradiol circulation because of alpha-fetoprotein, which binds to the freely circulating maternal estradiol and prevents it from entering the fetal brain [8]. But the fetus having XY chromosomal pattern has a higher amount of testosterone released by testes, which gets converted to estradiol in the sexually dimorphic region of the

brain by the action of an enzyme called aromatase. This sexually dimorphic region of the brain has high estradiol receptors. Estradiol on one side molds the neurons to become sexually aroused around a potential mate, and on the other hand prevent the development of female genitals. There are no characteristics or behaviors which typically belong to people with penises or people with vagina, and now are more like socially accepted norms of behaviors forced by society based on gender roles [9].

The complex interactions between the brain's sexually dimorphic areas and hormones are what give rise to the vast range of sexual behavior, cognition, and gender identity.

People with diverse sexual orientations have been discovered to have phenotypically distinct brain areas and neuronal structures, regardless of biological gender and what sex chromosomes you have, even the brain and neurons are sexually dimorphic. Rather than merely your genitalia, your brain and its neurological connections control your sexual behavior. It's as though your brain has its own gender identity and mostly controls how you feel, which may or may not correspond to the biological gender of your genitalia [10].

By the end of the conversion, Joe could notice Suhani getting a little tense. And Joe asked "if all of this feels overwhelming to you then it's alright, what matters the most is how you feel about yourself, it doesn't matter what other feels, thinks, or perceive of you, it takes a whole lot of guts and courage to accept ourselves and to do that we need to understand ourselves. In the end, no one knows who we really are, we all are just trying to figure ourselves out."

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Let's be friends with benefits

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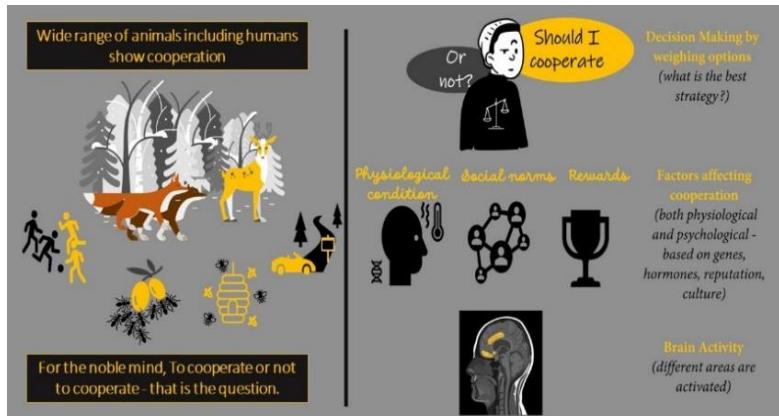
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SUMMARY

Cooperation is an interaction in which two or more individuals coordinate their behavior to pursue a common goal. As per the theory of evolution, individuals who act in their own self-interest have a greater chance of survival. Despite this, cooperation has evolved and sustained in multiple species and societies. Neuroscience studies devoted to revealing its mechanisms suggest that this behavior is governed by numerous factors, including rewards, hormones, and social norms.



Graphical Abstract (The figure includes a modified template from Servier Medical Art.)

INTRODUCTION

Have you ever acted out of selfishness? Maybe by keeping that resourceful study website to yourself to become a topper or by taking a larger piece of cake? Charles Darwin, the father of evolutionary biology, suggests via his theory that both genes and organisms try to move into the next generation by increasing their chances of survival and reproducing ability. This popularly came to be known as the '*Survival of the fittest*' or, sometimes wrongly, the "*Survival of the selfish*". Hence, it appears that there should be no opposition if one behaves selfishly. But, this whim to act selfishly and not think about the common welfare may lead to the '*tragedy of the commons*' where shared resources are often neglected. Aptly penned by Aristotle, "***That which is common to the greatest number has the least care bestowed upon it***". Let us give you a scenario. You went to a hotel, and after eating a delicious meal, you used the napkins kept for everyday use; and noticed that they were of excellent quality. Without one suggesting, you might feel like stuffing your bag with all those napkins, and let's assume that you went

ahead and did this. If every visitor does this, then at some point, a customer would walk in who desperately needs a napkin to wipe their mouth but won't be able to get it. In situations like these, we face a dilemma between our individual interests and collective benefits. One can observe the effects of this behavior when we talk about environmental issues like climate change or practices like over-mining and over-fishing, which leads to the depletion of natural resources. We might then reconsider that the key to achieving maximum benefits is by practicing cooperation.

COOPERATION IS ANY ACTION THAT BENEFITS THE GROUP BUT INCURS A LIKELY COST TO THE INDIVIDUAL.

Humans are highly social, and showing cooperative behavior is relatively common among us. On a day-to-day basis, we deliver this collective behavior when collaborating with someone for a project, while playing for a team, or even while following the traffic rules. Wait a minute, how do you even decide whether to cooperate or behave selfishly? Let us try to find out.

We must first understand why cooperation can prove advantageous. Imagine a scene where a lioness is hunting alone. The chances of her succeeding are low if she's chasing after a bull. But if a few lionesses hunt together, not only does their chance to succeed increase, but they can potentially prey on for more. Thus, increasing their chances of surviving and thus reproducing as a group, even though individually, they might not get a chance to leave a progeny behind. This is the case for many insect societies like bees, ants, and termites, where barring one female and a few male members of the group, many individuals are non-breeding or sterile. The sterile individuals take care of young ones by protecting and feeding them. One of the earliest known examples of cooperation is lichen, an alliance between algae & fungi. The fungus derives the food from the algae, which does photosynthesis. The algae get its water from fungi that absorb moisture from the air. This shows that cooperative behavior leads to optimization and is found in diverse species. It is safe to say that these organisms cooperate for survival. Cooperative societies have evolved and thrived, seemingly dissenting from the theory proposed by Darwin. Researchers have tried to justify the existence of cooperative behavior. Some theories are [1]:

- Direct reciprocity: Cooperate today to receive it tomorrow.
- Indirect reciprocity: Observe someone cooperating; you will cooperate in goodwill someday.
- Spatial selection: Because you live nearby, you will cooperate
- Multilevel selection: Within multiple groups, the most cooperative one will be victorious
- Kin selection: Showing cooperative behavior toward your family and relatives.

NEURAL CORRELATES OF COOPERATION

Now let's delve into the details of how our brains decide to cooperate. When humans cooperate, the brain areas that light up are the same as those that get activated when we receive a reward [2]. The areas involved are the structures embedded deep inside the brain: the nucleus accumbens, caudate nucleus, orbitofrontal cortex, and anterior cingulate cortex (Fig. 1).

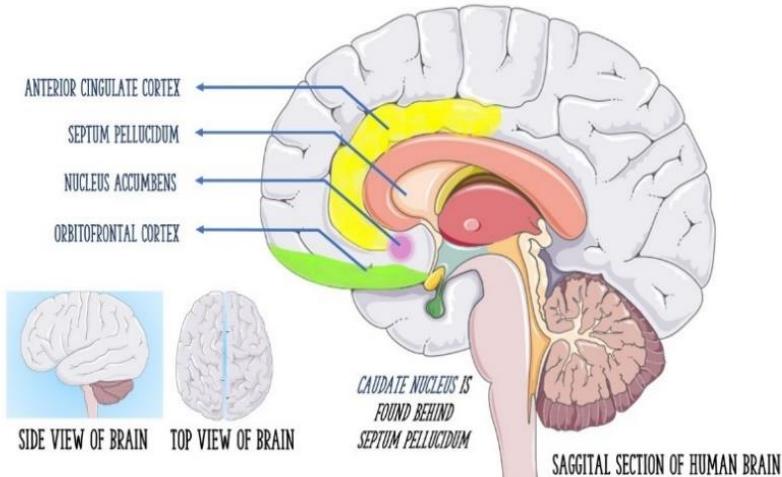


Figure 1: This figure shows the brain areas activated when we cooperate.

On the bottom-left inset are two views of the human brain (side and top view). The main image shows the brain from the side, the sagittal plane. You can get this view when the brain is dissected from the middle of two hemispheres (as indicated by a blue shading, top view).

Image credit: templates from Servier Medical Art.

But how does one identify the brain areas involved? Researchers make the subject lie inside a scanner machine and take images of their brain while they play a computer game, to determine the active brain areas. This is known as functional magnetic resonance imaging (fMRI). The region of the brain involved in the ongoing task will have more oxygen demands compared to other brain regions. The images then acquired by the scanner while performing the task and when they are at rest are subtracted. And the difference between these two images shows the researcher which area was active during the cooperative game.

Games played by the volunteers are based on social dilemmas. It is similar to playing a game of monopoly. The most commonly used game to study cooperation is called as- the prisoner's dilemma (Fig. 2). The volunteers are asked to visualize a situation where the police have arrested them and one other suspect at a crime scene. Now, both convicts are locked up in two separate rooms with no communication with each other. Now, if both individuals confess to the crime, both will be sentenced to three years in prison. Suppose one betrays the other person by testifying against them. In that case, the charges against that person will be dropped, but the other will be jailed for seven years (defecting). If neither confesses nor betrays, both individuals will be charged with misconduct and sentenced to one year of jail time (cooperation). You may say that the rational decision will be to act in self-interest, testify against another person, and go scot-free. But the dilemma here is that the volunteer has to make this choice without knowing what the other person will choose. So, the most profitable case will

be when both show cooperation by remaining silent [3]. This game cleverly shows that the outcome of pursuing self-interest is worse than cooperation.



Figure 2: Prisoner's dilemma pay-off matrix

Next, Scientists tried answering how our brain decides which option to choose. They trained monkeys to play a game like the Prisoner's Dilemma while sitting in front of each other. They recorded the neuronal signals from two relevant brain regions, the anterior cingulate cortex, and middle superior temporal sulcus, by poking needle-like wires in their brains. It was observed that the neurons responded to the opponent's behavior, including eye gaze, eye movements, social rank, and facial expressions, and tried predicting the opponent's yet unknown decision while playing the game. Based on these cues, the monkey decides to cooperate or defect. It was observed that the monkey adjusts its cooperative behavior to win based on previous engagement and rewards received [4-5]. Another human study suggests that participants are more cooperative when they think they are being watched [6].

Oxytocin, a hormone known to be released during familiar and comforting situations, is shown to have a role in social decision-making. Higher oxytocin levels make us more cooperative [7]. Even testosterone hormone, when administered in females, makes them more cooperative during the game [8]. A recent study identified genes responsible for a protein called 'Shank' which is found in the brain, which is essential in mice for showing cooperative behavior. When they removed these genes from the mice, mice showed autism-like behavior and were less cooperative in nature [9]. Studying the neural basis of cooperation is indispensable for understanding disorders like Autism, in which social behavior is affected.

Researchers have also shown that our level of social cooperation is based on what genes we inherit [10]. So, if your parents and grandparents were cooperative, the chances are you will also be cooperative in nature. And not just genes; our cultural background also plays a role in shaping our cooperative personality. People whose cultures have punishment for not

cooperating are generally more cooperative [11]. So overall, humans make the decision to cooperate based on factors like strategy, social norms, reputation, history, physiological state, and rewards [1]. So, unlike other species, not mere survival but other unique aspects drive this sentient behavior.

Let's play a game to determine how cooperative you are. Below is a table (Fig. 3); your task is to choose between the three options (A, B or C) [12]. Each option offers some money to you and another person. Imagine that you have never met the other person, nor will you ever meet them. What choice will you make? Take a minute to decide.

	OPTIONS		
	A	B	C
Money of Self (\$)	500	600	500
Money to Other (\$)	500	200	0

Figure 3: Cooperation game matrix

If you had chosen option A, you are a cooperator as you tried to maximize gain and equality. If you went ahead with option B, you are probably an individualist as you have maximized your profit. And you are a competitor if you picked option C as you maximized your advantage over the other person.

Remarkable if you had chosen option A because the times, we live in demand for cooperation more than ever. We could tackle the COVID-19 pandemic because most of us decided to cooperate. We did that by wearing masks, maintaining social distance, and lending hands via donating blood plasma, vaccines, and other supplies to the needy.

And don't worry if you picked any other option because one can become cooperative easily with intent. A community can be persuaded to behave cooperatively in three easy steps [13]. The first step is to induce a collective demand. For example, we should motivate people to use public transport to reduce both money spent and air pollution, therefore, improving the quality of life for everyone. The second is to coordinate the shifting behavior, ensuring that people use public transport as much as possible. This can be done by making them take pledges. The third step is repeatedly strengthening this norm by introducing newer routes and group ticketing offers that will renew their interest in cooperating. Also, punishing the free riders can serve as a motivation to cooperate.

Returning to the study resource website example, we gave in the introduction, what if you shared that critical website with your friends, and you all did well in the exams as a batch. Whenever you had doubts, you discussed them as a group and improved way beyond what you could have done alone. As a result, you and your friends reached great heights in your professional lives and celebrated their successes together. Isn't that a good feeling!

Albert Einstein once said, "***Nothing truly valuable can be achieved except by the unselfish cooperation of many individuals.***" So, shall we be friends with benefits?

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Learning How to Learn

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SUMMARY

Have you wondered how someone brilliantly remembers song lyrics, phone numbers, hook steps of Gangnam Style, and Kreb's cycle (Phew!)? All thanks to the ability of our brain to re-learn and rewire. Studies of neuronal wiring have revealed that our ability to learn isn't set in stone, and practicing different learning styles could make a sea of difference. Here you will find our neuro-psychological take on all things learning.

INTRODUCTION

Fantastic brains and where to find them

Why are some students labelled 'front benchers' and assumed to be intelligent and attentive in a class? On the other hand, the 'back benchers' are thought to be dim and constantly distracted. Is it possible that these differences arise from how the students are taught? Or could it be that these children employ different styles to learn the same material?

In 1992, Flemming and Mills proposed the VARK model (visual, aural, reading-writing, and kinesthetic learners), a new and attractive learning paradigm. In their opinion, every learner grasps information differently. Some learners prefer movies, graphics, and diagrams (these are visual learners), others prefer lectures and discussions (aural learners), and a few like re-reading and taking extensive notes (reading-writing learners). At the same time, the last group favors hands-on experiments and interactive sessions (kinesthetic learners). The VARK questionnaire is popularly used to identify which style one prefers when learning [1].

However, it is not all rosy. Given that each of the four styles of VARK allows a learner to use one portion of their brain more frequently than the other, it is typically assumed that VARK can impede rather than strengthen learning. For instance, the visual style would require learners to utilize their eyes and the brain's occipital lobe to learn through graphs and diagrams. Instead of having a beneficial effect on the individual's learning, this may prevent neuronal firing in other brain sections[2]. Further, how dependable is the VARK model? Does it mean that a person can only have one learning style? Scientists uncovered that 39% of those who take the VARK questionnaire do not exclusively fall into one category [3]. According to this study, learners preferred two or three learning styles rather than just one (Fig 1). Today, many researchers argue that preferential learning styles do not exist. According to Marshik, to retain information, we must organize it meaningfully, independent of the senses. So, the next time you are tempted to re-read or rewrite a pathway to memorize it, pause and reflect upon it using your examples.

STAND AT EASE, ATTENTION!

While learning, the skill of paying attention is paramount. Attention is the ability to analyse certain detailed information in the environment while ignoring the rest. But let's dig deeper. Imagine an elephant stomping into the room where you are currently sitting. How could you

pay better attention? It can be done in three little steps: activation (when you become aware of the elephant in the room), visual-spatial recognition (realizing how big the elephant is and what color it is), and paying heed to selective-executive components (focusing on sounds made by the elephant rather than every other noise in the background) [4].

So, does excellent learning and razor-sharp attention amount to a desirable memory? The answer lies in the combination and beyond.

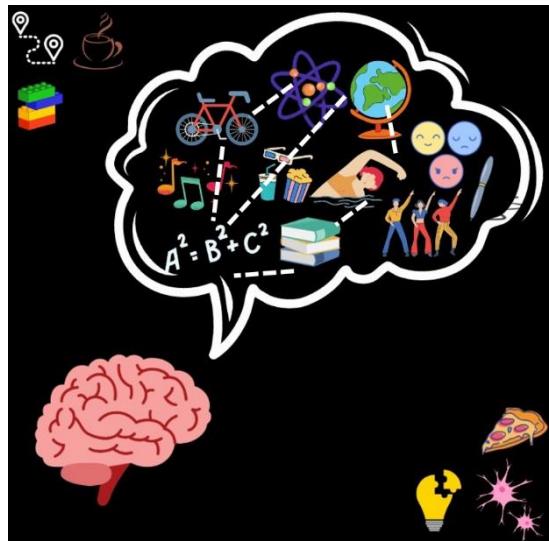


Figure 1: The multimodal brain. The illustration represents how the brain can be trained to learn and form memories and habits using multiple learning styles.

LIGHTS, CAMERA, ACTION!

What goes on in the brain as you try to learn? Hebb proposed that as you grasp new information, neurons are activated. They connect to other neurons and form networks. These networks begin weakly, but each time the stimulus is repeated, the connections grow stronger (a phenomenon referred to as potentiation). [5] This is why revising helps you remember more information. Because the same pathway is used repeatedly, it becomes hard-wired (or bound together). This theory is frequently postulated as "neurons that fire together, wire together."

Scientists discovered exciting facts when they studied these neuronal networks over human lifetimes. Peter Huttenlocher first demonstrated that there is a spike in establishing synaptic connections postnatally up to age 3, eventually these abundant connections are refined by pruning. Eliminating dispensable connections made way for robust networks that facilitated heightened cognitive skills vital for learning. In adults, however, the number of synapses fell

to half [6]. As mentioned earlier, specific networks become hard-wired, while the others are pruned due to their infrequent use.

SETTING UP NEURAL CIRCUITRY 101

Hang on, do we know what the commonly used term 'plasticity' refers to? Plasticity is the brain's ability to change and adapt to new information. It is a nod to how malleable the human brain is. If the brain makes and breaks connections rapidly, it has 'short-term plasticity' [7]. When you pay money for your favourite food on a mobile application, you receive an OTP in your message inbox. You then reproduce the OTP on the application, complete the transaction and sit back dreaming of the pizza. Memorizing an OTP and replicating it is a classic example of short-term plasticity. It is essential for a fraction of a second but immaterial later. However, most memories in our brains need to be stored for long durations. This is referred to as 'long-term plasticity,' which may last from minutes to hours, days, or even years and is the dominant model for how the brain stores information [8].

So, can this plasticity (firing rate) be measured? Yes! Electroencephalography (EEG) is the most common method of measuring neuronal plasticity. EEG measures the brain's electrical activity by placing multiple electrodes along the scalp. Another non-invasive way to measure plasticity is the well-known magnetic resonance imaging (MRI).

Finally, it is worth remembering that even established neuroscientists once thought our brains were like Tupperware boxes. Their ability to learn was fixed. Today, we know it is expandable and can take a variety of shapes, just like Ziplocs!

WHEN DOES THE BRAIN OPEN SHOP?

The foundational network of the brain is undoubtedly laid in infancy. Children's brains develop in bursts known as critical periods. During these periods, the number of synapses between neurons is very high, implying they are more likely to be receptive to any stimulus. For example, the process of learning a new language is believed to be constrained by the critical period. If a child were exposed to a speech during this crucial period, he would be more likely to acquire native-like fluency. After this window, however, it would become challenging to acquire competency even in a linguistically rich environment [9].

Think of learning in terms of constructing a building, where each synapse cements the building blocks of neurons, revealing a blueprint of your brain's structure. Once the foundation and ground floor of the building is set, it's up to you whether to build a duplex or the Burj Khalifa.

Studies have shown that aging does interfere with the performance of cognitively demanding tasks. However, learning ability does not cease altogether with age. Because learning as an adult is primarily self-driven and generalized theories do not help in understanding adult learning. The prefrontal and temporal regions of the brain take care of the segregation between the novel and not-so-novel aspects of the skill, making sure your old and new memories don't mismatch. Imagine forgetting English in an attempt to learn French! Thankfully, our brains are more intelligent than we give them credit for [10].

SO, YOU THINK YOU CAN LEARN?

Let us start putting together the jigsaw of things we have encountered; learning techniques, attention, neural circuits, and a learning trajectory. We hope to have sufficiently convinced

you of the brilliance of our brain so that you no longer limit it to the binary scenario of a pass or fail.

Because if you were performing any cognitively demanding task, it means different brain regions are collectively joining the dots of what you know and do not to make a better sense of the situation.

Current research has stepped ahead to investigate the different facets of learning. It involves investigating neuronal and non-neuronal populations of glial cells, reorganization of white matter, what causes tumbling of impulse transmission, and finally, how your brain rewires in extreme injury, stroke, and significant mental health-related disorders [11].

Learning is demanding because you continuously respond to multi-sensory stimuli and subsequent rewiring of existing connections, which is eventually reflected in your behavior. Apart from basic developmental templates, your learning abilities are not set in stone [12]. If you are determined to learn something new and put in 'focussed attention' to do so, you will triumph (commonly called 'grit'). After all, hasn't APJ Kalam quoted, "the best brains of the nation may be found on the last benches of the classroom."

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Rewiring of the Brain in Outer Space

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SUMMARY

The human brain undergoes many changes in outer space due to primary factors like microgravity and space radiation, secondary factors like isolation in space and changes in circadian rhythm, etc. Overall, these changes are categorized into positive and negative effects that are summarized in this article. The effects caused by microgravity on the human brain can be studied here on Earth using a stimulated environment.

INTRODUCTION

We live in an era where ideas such as space tourism were considered a part of science fiction a few decades ago, are becoming a reality now. Space tourism is thriving albeit among rich, along with the increasing advent of space travel through government agencies such as NASA and ESA and commercialized spaceflight through companies like SpaceX, Virgin Galactic, and Blue Origin. Similarly, Indian space agency ISRO is set to launch humans in space soon. Like any new technology or invention, space travel also presents us with exciting opportunities as well as new challenges. This article is focused on one such challenge, which is brain functioning in outer space.

Our brain is the most complex organ in the human body. It is a 3kg jelly-like mass that can control thought, memory, emotion, touch, motor skills, vision, breathing, temperature, hunger, and every process that regulates our body [1]. It consists of two types of matter: grey matter and white matter. White matter (axons) is significant for communication between the different parts of the brain, and grey matter (cell bodies) acts as the commander. This complex organ is threatened while encountering new environments like outer space.

Have you ever wondered how short and long-duration spaceflight would impact the human brain? Spaceflight has the potential to profoundly alter both the function and structure of the adult brain as the microgravity environment in space has tremendous effects depending upon the duration in the space.

MAJOR CHALLENGES IN SPACE: MICROGRAVITY & SPACE RADIATION

Microgravity is commonly known as the weightless environment. It is also called "Zero-gravity," which is a misnomer because there's still gravity present in space ~ 90% of the Earth's gravity reaches the space station [2]. It has a lot of impact on our whole body, but later in this article, we will discuss how it explicitly impacts the brain in outer space.

The other major concern is space radiation exposure. Space radiations mainly refer to galactic and cosmic radiations. The radiations emitted by the sun consist of visible, infrared, and ultraviolet (UV) radiations. Although seldomly do solar flares and coronal mass ejections occur on the surface of the sun, that results in the release of massive amounts of energy out into space in the form of x-rays, gamma rays, and streams of protons and electrons. These are called solar particle events (SPE).

On Earth, the magnetosphere protects us from most of the harmful radiation of the sun. Radiation from the sun has low levels of high-energy protons. Despite being present at low

level, cumulatively, they are harmful. The radiation causes significant harmful effects on brain that coincide with the effects of microgravity. Due to these concerns, long-time space travel is a matter of great concern [3].

EFFECTS ON THE BRAIN

A recent study found space travellers' brains appear "rewired". The ability of the brain to rewire is called neuroplasticity. In the space travellers these changes can persist for months after their return to Earth [4]. The rewiring can be both constructive and destructive. There is still not much data to support that the rewiring of the brain helps humans adapt to space on a long-term basis.

A rough analogy of going to space could be taking a long intercontinental flight. We can see numerous stressors and resilience factors during each stage of flight: pre-flight, in-flight, and post-flight, that result in both positive and negative outcomes. The positive outcomes include increased structural and functional plasticity and sensory reweighting, but numerous adverse effects exist [4].

Adverse effects on the brain

The significant adverse effects of microgravity on the human brain are white matter dysfunction, increased ventricular (brain ventricles are fluid filled communicating cavities in the brain) size, spaceflight-associated neuro ocular syndrome, and venous thrombosis. In contrast, radiation exposure results in impaired cellular signalling in the hippocampus, which houses learning and memory, causing a significant reduction in performance and decision-making capabilities. It also causes long-term anxiety and depression. When exposed to radiation for the long term, changes in white matter (also called radio necrosis) and hormonal disturbances can be observed [5].

The changes in the brain due to spaceflight can be either dysfunctional or adaptational under different conditions, which can be investigated via associated changes in behavioral and cognitive performances. This concept is named "Spaceflight Perturbation Adaptation Coupled with Dysfunction" (SPACeD).

After spaceflight, the astronauts experience adverse effects on cognition and sensorimotor such as impairments in posture maintenance, decreased motor and vestibular network connectivity (essential for balance function), locomotion, eye-hand coordination, and assessing the differences in the mass of two different objects (Barognosis). Additionally, they experience spatial disorientation in which they cannot perceive their motion in space. Perception of motion in the up/down axis is affected, while the perception of motion in the left/right axis remains unaffected, mainly due to the effect of weightlessness in space that distorts this function [4].

Dual tasking (ability to perform multiple tasks simultaneously) in microgravity is also found to be decreased since it requires additional work cognitively due to altered perception in outer space. Although there are negative effects, microgravity induces adaptive brain processing in vision, proprioception, and vestibular systems. One example of adaptive processing for the vestibular function is the increased sensitivity of fast-acting skin receptors responsible for compensating the vestibular input loss through skin pressure sensation. Whereas the slow-acting skin receptors responsible for postural control on Earth have reduced function in outer

space as there is body unloading in space. For balance control, there is the down-weighting of vestibular inputs and the up-weighting of sensory inputs (pressure input/tactile sensation).

Recently, a case study showed that after six months of in-flight, the decreased motor and vestibular network connectivity, which is a brain function-oriented defect, resulted in vestibular ataxia and motor incoordination. When researchers tried to simulate the physiological effects of in-flight through head-down-tilt bed rest (HDBR), there was increased brain activity during vestibular stimulation, indicating the high neuronal demand due to the reduced efficiency of individual neurons in microgravity. Brain structure-oriented changes include decreased synapses and degeneration of axonal terminals in the somatosensory cortex and cerebellum, which can be attributed to the body unloading and neuronal losses induced by radiation exposure [4].

Recent studies using the imaging tools like MRI and tractography showed that long-duration exposure to microgravity results in disrupted white matter connectivity. This could be caused by microstructural and functional changes [6,7]. There was reduced myelin integrity suggesting the disruption of white matter bundles that run across the brain connecting different parts/lobes such as the superior and inferior longitudinal fasciculi, inferior frontal occipital fasciculus, corticospinal tract, and the cerebellar peduncles. These are significantly important for motor functions and balance functions [4].

Furthermore, the brain swells in space because of reduced gravity, the fluids in our body that usually circulate evenly travel up toward the head and away from the feet. The blood that generally pools in the extremities redistributes toward the head [8]. Moreover, the cerebrospinal fluid, which surrounds the brain and spinal cord, also increases in volume at the base of the cerebrum, which is around the frontotemporal regions, and decreases in the posterior parietal region. This variation in Cerebrospinal fluid (CSF) distribution also decreases grey matter in the frontotemporal regions and vice versa in the posterior parietal regions. The increased CSF also increases brain volume [4]. This increase in CSF increases intracranial pressure (ICP) and leads to vision issues for astronauts, as the increased ICP presses on the optic nerve. Countermeasures are being developed and are under research to help astronauts fight these conditions by studying the effects of microgravity on the brain through the HDBR method. This has been able to mimic certain critical aspects of microgravity, allowing research about various effects of microgravity on human health, including brain effects [8,9].

Positive effects on the brain

Regarding the adaptive effects, the scientists found that due to reduced hindlimb usage and reductions in balance and mobility, there were increased neural resource demands to compensate for the down-weighting of the lower limb neural changes (HDBR study in human subjects). Also, to support these additional neural resources demand, the participants with the least impairment in balance post-HDBR were the ones with the most significant changes in functional connectivity in the motor network that involves the left primary motor cortex, right postcentral gyrus, and the superior parietal lobule. This indicates that the HDBR study achieved at least some level of functional connectivity changes with reduced behavior declines [4].

Other effects

Other side effects should be given more attention, such as isolation, loneliness, anxiety, and depression. Dealing with these situations is unique. Masses have experienced some similar problems during the present COVID-19 pandemic. The brain becomes restless because of general isolation, but prolonged isolation, such as in space, causes a decrease in cognitive capability. Spending such a long time in a wholly confined tiny space isn't something you experience on earth [10].

SPACE ENVIRONMENTS STIMULATIONS ON EARTH

To achieve futuristic goals of efficient spaceflights and to minimize the effects on the brain, the space environments can be simulated here on Earth by creating microgravity environments through parabolic flights (airplanes fly by up-and-down parabolas), clinostats, water immersions, and the pre-existing HDBR technique. All these methods do not simulate weightlessness but the unloading of limbs and headward shift of fluids. At the same time, radiation therapy's effects on cancer treatment can provide some important clues about space radiation's effects on the human body.

FUTURE OUTLOOK

What do we know for sure? Can stimulation help us achieve our goals? What more happens to the human brain in space? Is there anything we're still not convinced of? Is there any possibility of finding some solution? We have come across sci-fi movies that show artificial gravity created inside the space station, but in reality, it's still out of reach. Scientists are yet to find the crucial pieces of this puzzle. We still have a long way to go in space science regarding human health research and to bring in technologies that are beneficial in achieving long-term spaceflight.

The overall goal should be to determine space travel's short- and long-term effects on the human brain. Since this research is trailblazing, there's still a lot to unfold. These results contribute to our overall understanding of the brain dynamics of space travellers and astronauts so far. We still have a far-flung route to decide whether the rewiring is good or bad.

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Why is learning new things so hard?

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SUMMARY

Most of us find it difficult to memorise new information or to learn a new skill at the beginning. Here, we introduce how memories form in the brain, how we remember and the different brain cells and chemicals involved in this process. Next, we discuss how stressful situations make us forget things that we memorised. We also talk about a few science-backed methods that sharpen your memory.

INTRODUCTION

If you were a child living in ancient Greece and were expected to memorize something, you would have been tossed in the river Mnemosyne [1]. According to Greek mythology, Mnemosyne was the goddess of memory, and dipping children in the cold waters of Mnemosyne would give them the gift of astonishing memory.

Unfortunately for you, learning something new is not as easy as taking a dip in a river, or even eating some “memorization bread” like the one Doraemon gave to Nobita. It demands hours of practice and a lot of patience. But, why, you may demand, must it take so much effort to memorize new things? Why do we forget? And is there any way for you to improve your memory?

WHAT IS MEMORY?

First things first, we need to understand what memory is. Memory can be defined as the brain's ability to store and retrieve information [2]. Zoom in to the molecular level, and it includes a complex interplay of the cells of the brain- neurons, and their chemical messengers, neurotransmitters that allow you to store information in your brain and later on fetch that information when required. Essentially memory includes a 3-fold strategy: encoding, storing, and retrieving. To explain this process, imagine your brain is your library. You add a new book to the shelf that you found to be an interesting read. This first step is called encoding. Encoding means you discover something new and you want to remember it, so you input that into your brain. The information fed into the brain is retained in a process called storage: your book is kept or stored on the shelf. Later when you want to read that particular book you fetch it from the shelf. This is called retrieval, the recalling of information stored in the brain.

Here we should note that memory and understanding are different. Memory is when you can recall, correctly, what the formula of Newton's law of Gravitation is. Understanding is when you know how to apply that formula in new ways, for example relating to the same inverse square relation in Coulomb's law of Electrostatics.

SHORT-TERM AND LONG-TERM MEMORY

Say, you've just learnt about Newton's law of Gravitation, its formula, and its derivation for your exam. After a couple of days, you struggled to correctly remember the law during a class test. But, after months of revising the formula and derivations over and over, you could solve questions using the formula in your exam.

When you first came across the law and the formula, that experience was converted to electrical signals and carried by the neurons to a part of the brain called the hippocampus. The hippocampus resides in the temporal lobe of the brain and looks a bit like a seahorse. It encodes your experiences as memories, which are then stored in your short-term memory (STM), which lasts about 30 seconds. The hippocampus strengthens synaptic connections between neurons to consolidate memory or turn short-term memory (STM) into long-term memory (LTM) (something which is under the control of another, rather almond-shaped part of the temporal lobe called the amygdala). The neural circuitries in the hippocampus are stimulated by reviewing facts or concepts. Stimulate these circuitries enough times over a long enough period, and the hippocampus will convert it to long term memory which will be stored in another part of the brain, the neocortex. (Fig. 1) [3].

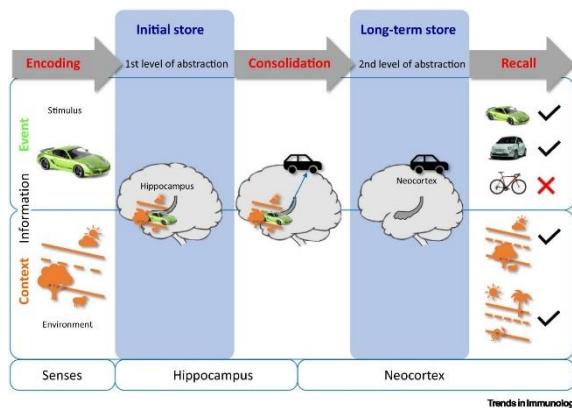


Figure 1. Mechanism of short- and long-term memory formation

Image taken from Lange, 2019 with prior permission from the authors

THE BIG QUESTION: WHY DON'T WE REMEMBER ALL THAT WE LEARN?

Now, the obvious question that comes up is, *"If I can remember the formulae why don't I remember the entire chapter? What if I forget how to calculate the numerical values? Why does it take so long? What if I never finish? What are numbers? What's wrong with me!"* First, there is nothing wrong with you. Second, a possible explanation lies in the definition of memory itself; it is the "capacity" of the brain to store and retrieve information. Your brain is a tissue formed of neurons and glial cells and like any container, it has a limited storage capacity. It selectively converts certain short-term memories to long-term memories to be stored in the cortex. Recalling facts and concepts over and over strengthens the neural connections and these tend to be converted into LTM. However, this only partially answers the question. The other essential aspect is forgetting. Neuroscientists believe that forgetting is a natural process; it makes space for the new while discarding the old ones. According to research, those who recall every detail of every encounter struggle to apply that information to a new circumstance. People who struggle to remember every last detail are superior to them in applying concepts across contexts. Forgetting is a normal process that happens across many different species like humans, the fruit fly (*Drosophila*), and mice [4]. When we look at the

cells in the brain, remembering facts and forming new memories are associated with *neurogenesis* (the formation of neurons), *myelination* (formation of myelin, the lipid-rich layer covering the neurons), and *synaptogenesis* (formation of new synapses, the gap between two neurons). These processes alter the synaptic connections and hinder access to old memories, leading us to forget [5]. Forgetting allows your brain to stay flexible and plastic which allows you to learn new things.

THE ENERGETIC COST OF LEARNING

After learning something new, you must have felt like a marathon runner who has just finished. Ever questioned why? It's because of the energy requirements of your brain. Even though it makes up only 2% of your body weight, the brain uses up 20% of the body's glucose and oxygen! [6]. It takes a lot of energy and increased brain activity to learn, think, and make memories. Your body diverts energy to the brain for the same, leaving you feeling burnt-out after a rigorous hour of studying. Low blood sugar is associated with attention deficits, but high blood sugar might impair memory [7].

Exams, stress, and the cortisol catastrophe

Exams or a viva voce or an interview stress out even the best among us. Under such conditions, the brain responds by prompting the body to release the stress hormone cortisol into the blood. Moderate stress helps in memory formation but extreme and chronic stress hampers memory retrieval, making your mind go blank [8]. Cortisol promotes fear-based or habit-based learning and lowers memory accuracy, according to a study done on mice. Research has also shown that high levels of cortisol impair your ability to think, explore and acquire knowledge [9]. These findings highlight the importance of stress management. So, maybe chill out a little before an important exam.

HOW TO REMEMBER BETTER?

Now for some tips and tricks!

- Repeat
 - "Practice makes perfect" But what does it *mean* and *why* does it 'make perfect'?
 - As it turns out, neuroscience agrees with this. When you repeat a task, you strengthen the neuronal circuits responsible for that particular task. This is true for both cognitive and motor tasks. Several experiments have shown the positive effects of repetition on learning [10, 11].
- Take a cold shower or bath
 - This might seem far-fetched but there is good scientific evidence that supports this. In a series of experiments in the mid and late 1990s, Larry Cahill and colleagues discovered that hormones such as adrenaline and cortisol have a positive effect on memory formation [12]. They divided study volunteers into two groups and asked one group to immerse their arms in cold water after a memorization task. These volunteers had higher cortisol and adrenaline levels. Cahill and colleagues reasoned that this increase in cortisol levels after a period of learning acts to improve memory [13].

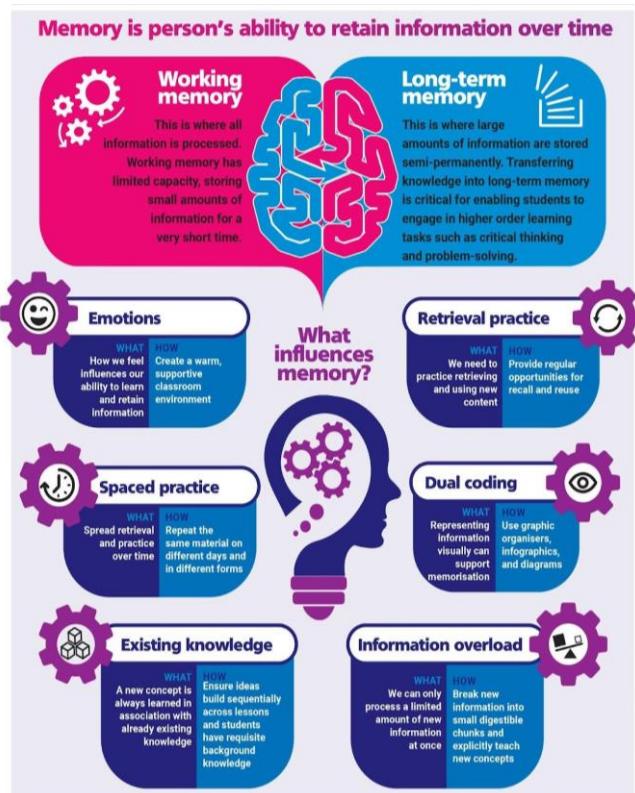


Figure 2. Different factors that influence memory.

Image credit: <https://www.theeducationhub.org.nz>

- Have a cup of coffee!
 - People often drink coffee before beginning work that requires intense focus, like learning something. But it turns out that drinking coffee after a period of learning is a better way to help us remember better. One way how caffeine act on the brain is by causing the release of adrenaline, which, as we mentioned above, will support learning only *after* it is completed. However, coffee must be consumed in moderation and it may not suit everyone.
- Exercise
 - Physical exercise leads to the release of a hormone called osteocalcin from the bones. It travels through the blood and into the brain and supports electrical activity, the formation and maintenance of neural

connections within the hippocampus. How it happens is still an area of active research, but preliminary data shows that it reaches the brain and regulates the expression of neurotransmitters [14]. So, having an exercise routine also keeps your memory sharp!

- Meditation
 - Meditation, a widely encouraged practice, has a host of benefits. Prof. Wendy Suzuki's group at NYU showed that meditating daily for 13 minutes for a minimum of 8 weeks results in enhanced memory. Not only does your memory get better, but your mood also improves, and meditation also helps in dealing with anxiety and mood disturbances [15]. So, dedicating less than 15 minutes a day can work wonders not only for your memory but also for your overall mental health.

The process of learning and memorising is complex and is yet to be fully understood. How well we learn a new skill or memorise a new fact is influenced by the complex interactions between various brain cells and neurotransmitters. The creation of new neural connections is essential for the formation of memories, and regular activation of established neural connections makes them stronger. The fact that this process takes time is a major factor in why it's so hard for us to remember new information. Your learning capacity can be increased by getting enough rest, moving around, and meditating. If not, you could always go swimming in Mnemosyne.

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Beauty in the Brain of the Beholder

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SUMMARY

In this article, we talk about the neuroscience of beauty. We start with a general introduction as well as a historical perspective of beauty, followed by an overview of how neuroscience has been used to study beauty. We further explore how cultural differences influence our perception of beauty. Finally, we talk about the practical applications of neuro-aesthetics in marketing and how this information can be used to influence consumers.

INTRODUCTION

Vineyards stripped bare due to pest infestation, the serene calm after a winter storm, and a former National Geographic photographer on his way to visit his girlfriend. The coincidence of these events set the stage for capturing one of the most iconic photographs in history. In January 1996, on his way to California, Charles O'Rear stopped his car and pulled out his camera, set it up on a tripod, and took the photograph that most of us grew up with. Bought by Microsoft in 2000, the image was rolled out as Windows XP's default wallpaper under the title 'Bliss', and it has since been on the screens of more than a billion computers, possibly making it the world's most viewed image (Fig 1) [1].



Figure 1: 'Bliss' by Microsoft, photographed by Charles O'Rear in 1996
(Windows XP Bliss is licensed under CC BY 2.0)

A hunter from Thespiae, a city in Ancient Greece, was blessed with a long life lest he discovers himself. Unfortunately for him, his cold and cruel rejection of a shy mountain nymph, Echo, invited the wrath of the gods. Enraged by his act, Nemesis, the Greek goddess of revenge, lured a thirsty Narcissus to a clear pool. Captivated by himself in the bloom of youth, the

hunter, under the delusion that it was someone else, fell deeply in love with his reflection. Unable to leave, but eventually aware that his love would never be reciprocated, Narcissus waned to death, eventually leaving behind a gold and white flower (Fig 2) [2].



Figure 2: A part of the painting of Echo and Narcissus by John William Waterhouse (*This is a derivative of John William Waterhouse (1849-1917) - Echo and Narcissus (1903), Walker Art Gallery, Liverpool, May 2012* by ketrin1407 is licensed under CC BY 2.0.)

Tall stature, long faces, prominent chins, lean builds, straight and light hair, light eyes, and fair skin (Fig 3) [3]. Kinda sounds pretty, doesn't it? Well, one group thought this was not just pretty, but racially superior, considered other races a threat, and used this belief to justify torturing and killing more than 17 million people. From 1941 to 1945, the National Socialist German Workers' Party, or Nazi Party, for short, carried out what was the largest genocide in history. All because they identified certain traits as aesthetic and racially superior, backing it with pseudoscientific research.

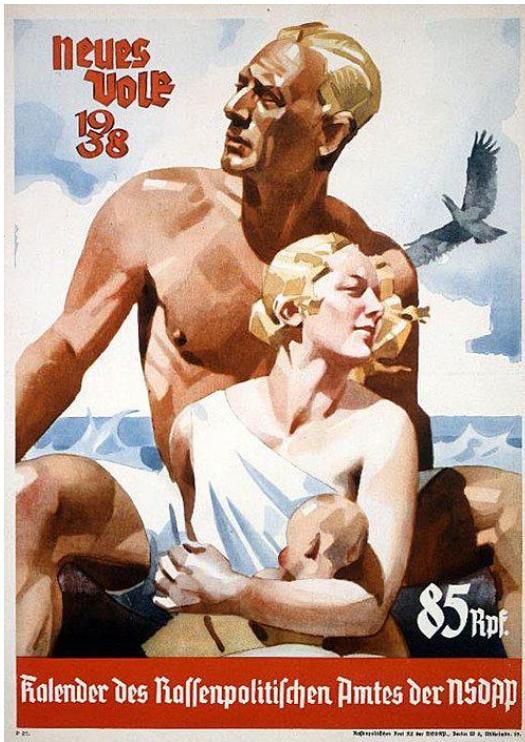


Figure 3: Poster advertising for a propaganda calendar from the Nazi magazine *Neues Volk* ("A New People") issued by the Nazi Party Office of Racial Policy; Watercolor painting by Ludwig Hohlwein 1937; picture showing the idealized concept of a healthy German family as the core of the "Volksgemeinschaft", a pure "Aryan" family, with Nazi ideal racial types (a blond father, mother, and young child), and a flying eagle in the background.

(By Ludwig Hohlwein - <http://galleria.thule-italia.com/ludwig-hohlwein/> No known copyright restrictions; The poster was designed by Ludwig Hohlwein, a German artist who died 1949, and issued by NSDAP, an organization in Nazi Germany ceased to exist in 1945, and the work has fallen into the public domain., CC BY-SA 4.0)

The above three tales, spread across time, have but one idea in common – the concept of beauty. Things both magnificent and horrid have come out of the human pursuit of beauty, but how do we determine that something is beautiful? Taking it one step further, how do our brains decide what is beautiful and what isn't?

EARLY CONCEPTIONS OF BEAUTY

What, according to you, is the most beautiful painting in the world? Perhaps you thought of Leonardo da Vinci's almost life-like Mona Lisa? Or perhaps Spider-Man: Into the Spider-Verse, with its stunning animation, was one of the most beautiful things you've seen. Many

poets, artists, and philosophers have tried to decipher the meaning of beauty. Philosophers such as Plato and Immanuel Kant argued that beauty is universal; "if I think the Mona Lisa is beautiful, so must you and so must everyone else, otherwise it isn't beautiful." Few other thinkers were against this notion. They suggested that beauty is a more subjective experience. Psychologists such as Gustav Fechner equated beauty—in its broadest sense—as something that generates a feeling of pleasure or liking. So, according to Fechner, you like the movie Spider-Man: Into the Spider-Verse because its animation pleases you [4-6].

CRACKING THE SKULL OPEN

In the 1950s, a group of researchers directly shocked certain regions of mice's brains while teaching them to solve problems and run through mazes. This stimulation seemed to give the animals pleasure and a feeling of being rewarded – the same way they (or even we) felt after receiving treatment for solving a problem correctly [7]. The following years revealed that dopamine was the key molecule involved in the process of feeling pleasure and reward [8].

Since philosophers and psychologists thought that beauty and pleasure were connected, neuroscientists now began poking around the brain to verify this claim. To get a peek under the hood, the scientists put participants under a brain scanner and began looking for the parts of the brain that "light up" or are active when assessing an artwork. Studying the human brain can be difficult since we can't remove it, study it, and put it back. Neuroscientists use methods such as Functional magnetic resonance imaging (fMRI) which detects changes in brain activity in different regions of the brain, or an electroencephalogram (EEG) that records electrical signals from neurons closer to the scalp. They also track heart rate, respiration rate, and whether you get goosebumps to measure if something (beautiful) arouses you [9].

Using these techniques, neuroscientists found the link between beauty and pleasure. They noticed that if the pleasure center of the brain - the mesocorticolimbic system - is suppressed, we perceive something as less beautiful than under normal conditions. The mesocorticolimbic system (Fig 4) is a collection of neurons that release the neurotransmitter dopamine and regulate the feeling of reward and pleasure in our brains [10].

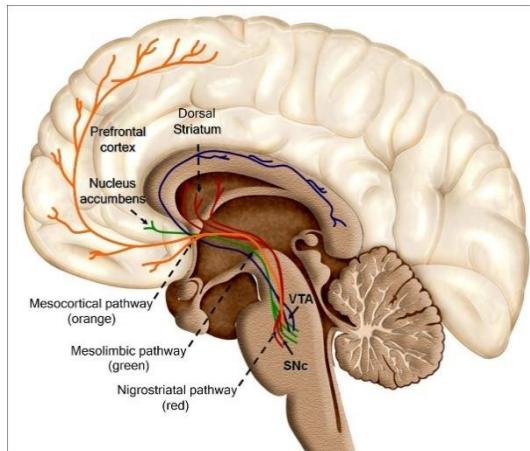


Figure 4: The path to pleasure - the neuronal pathways involved in the feeling of reward/pleasure known as the mesocorticolimbic system. SNC - Substantia Nigra pars compacta; VTA - ventral tegmental area.

(File: Recolored Overview of reward structures in the human brain2.png" by Oscar Arias-Carrón1, María Stamou1, Eric Murillo-Rodríguez, Manuel Menéndez-González, and Ernst Pöppel. Substantially modified by Seppi333 is licensed under CC BY 2.0.)

Scientists then began looking at the object, focusing on its various properties to see how each one contributed to giving us pleasure. For example, we find a variety of flowers beautiful even though they evolved to appeal to the birds and insects that pollinate them. Studies showed that some factors we tend to notice when evaluating beauty (visually) are symmetry, proportions, colors, and contrast. This makes sense evolutionarily too as these properties are what many animals use to judge the health and fitness of a potential mate [11].

But the painting is still incomplete. Later research showed that the amount of pleasure we feel when we see a beautiful object is directly dependent on the context and circumstance related to that object. One study saw that people felt different amounts of pleasure when eating chocolate on a full stomach as compared to on an empty stomach [12]. This applied to beauty as well. Take the Taj Mahal for example. It sounds beautiful to hear that Shah Jahan built such a magnificent monument in his wife's memory, but you might also feel angry or disgusted when you hear the story of him chopping off the workers' hands afterward so that none of them could build something that beautiful again (that story is baseless, by the way). People also experience beauty differently on different occasions, like the first time they listened to a fantastic song versus the thousandth time in a row and growing tired of it [10, 13-14].

YOUR PARENTS TEACH YOU WHAT'S BEAUTIFUL

I'm guessing most of our parents have ranted about us getting influenced by 'western culture'. Be it food, fashion, language, or lifestyle, most people think that their cultural definition of beauty is...well...the most beautiful. We know what an artist from our culture means through their work, and can connect with it on a deeper, emotional level. For example, those following

Hinduism know the significance of Ganesha having the head of an elephant and can find beauty and reverence in it beyond just a pretty image.

Beauty needs cultural context. So, can we locate this cultural preference for what is beautiful in our brains? To this end, a group of researchers showed Eastern (Chinese) and Western (European) paintings to people from both cultures. Parts of the brain involved in feeling empathy lit up more in the European participants when they saw Western-style paintings, which they thought were more beautiful. The Chinese participants, on the other hand, did not show any general preference for either style. One reason could be what our parents always feared - Asian communities have adopted and grown familiar with Western values [14].

The above idea is something that has been applied for thousands of years to design and present objects that appeal to a particular society's specific sense of beauty. We are flooded with this information every single day of our lives, and we are asked to pay to not receive them. Yes, this is the powerful, sometimes annoying, the art of manipulating the masses - a field commonly known as advertising. However, some companies take this one step further, into a whole new world of neuromarketing.

MANIPULATING THE CONSUMER'S MIND

You have ever wondered why Coca-Cola (Coke) sells way more than Pepsi even though they taste nearly identical? Neuroscientists from the Baylor College of Medicine conducted a study to answer this question. They found differences in brain activity when participants drank Coca-Cola and Pepsi with the brand names hidden as compared to when they were served with the brand names revealed. This difference shows us that the preference for Coke over Pepsi does not only depend on taste but also the brand name. This study is just a very basic example of neuromarketing, an up-and-coming field of study that makes use of neuroscience to gain insight into the mind of the consumer [9,15,16].

Companies use neuromarketing techniques to formulate better products that appeal to consumers more. Frito-Lay noticed that though women snack more than men in general, they did not buy Frito-Lay products. So, keeping the female consumers in mind, Frito-Lay used fMRI to evaluate the reactions of the subjects. It turned out that the shiny packaging stimulated a specific area of the brain that made one feel guilty. Frito-Lay reintroduced the chips in matte finish packaging which increased sales significantly [17]!

Wrapping up...

From da Vinci to World War II, cave paintings to Coke or Pepsi - our best creations, our bloodiest wars, colorful sketches to shopaholic tendencies are driven by our pursuit of pleasure influenced by our sense of beauty. This is what we've been trying to understand all along. What *is* beauty? Philosophers argued about it for millennia, psychologists came up with theories for centuries, and now, neuroscientists are looking at the brain directly to find out why we find something beautiful.

Is it evolutionary? To an extent, yes. We aren't the only species to find things appealing. Peahens find peacocks with long vibrant feathers beautiful, nightingales think the partners that can sing best are beautiful, and so on. Now, is the way they find traits in a partner beautiful the same as the way we find the stars beautiful? That is something we can try to find out. However, we also know that things we are exposed to growing up also influence what we find appealing. How this is wired in our brains could tell us why some of our friends love K-Pop music while

others vibe to heavy metal. This also determines whether you would buy soft drinks in bold packaging or junk food in non-flashy packaging. That's why a lot of companies are investing in understanding your brain – to get you to buy more.

With a lot of room to grow and a lot more to reveal, in the end, this field could finally answer the question most of you would have had – “*How could they even think that it's beautiful?!*”

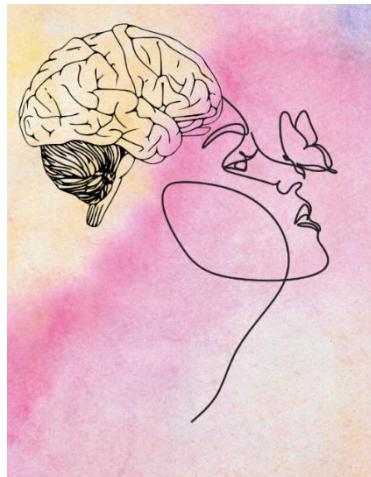


Figure 5: “Beauty is no quality in things themselves: It exists merely in the mind which contemplates them, and each mind perceives a different beauty.” — David Hume, philosopher

(Illustration by Sanah Kumar)

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Removing the Rose-tinted Glasses: A look into the Ethics in Psychological Studies of the Past and Present

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SUMMARY

The article provides a glimpse into some dark and cruel experiments in psychology that reflected a shocking lack of ethical practices in the unfiltered quest for knowledge. It highlights morally dubious choices in experiments like Pavlov's Bell, which are largely unknown. The importance of a sound ethical framework in psychological experiments are also highlighted.

INTRODUCTION

In 2010, a user named 'Roko' posted an intriguing thought experiment on 'LessWrong', a rationality and philosophy-focused blog plus community site, which would go on to become an internet legend. The thought experiment centered on such a controversial idea that the owner of the site removed any evidence of its existence and prevented conversation on the topic, due to its deep implications and potential for causing mental distress. This idea was known as "Roko's Basilisk", named after a mythological beast that can kill its prey with a single glance.

The idea behind this thought experiment deals with the behavior of artificial intelligence (AI) as the singularity point (the point in which AI becomes self-aware and hence evolves beyond our control) is approached. Due to its superior intelligence and lack of ethical regulations, the AI could decide to subject those who did not help in its creation to eternal torment. Since the AI can predict and simulate every one of your choices in the past, present, and future, the question arises - should you not, in a bid to save yourself, aid in its creation and champion its development, while knowing its wrong for such a technology to control your actions?

If the knowledge of this thought experiment was never disseminated, then there would have been no problem. However, due to the creation of the AI (basilisk), all personal freedoms and choices have been eliminated by the basilisk [8, 9, 13].

AN “INFORMATION” HAZARD

So, how does this connect to ethics in psychology? Well, it all starts with the concept of informational hazard. An informational hazard is described as a knowledge that, by its very nature, can cause harm to people when they learn about it. This is the premise behind "Roko's Basilisk". If the knowledge of this idea was never shared, we would never have been forced to act on it. [8, 9, 13] This shows the implications of the curse of knowledge and how it affects us. It further shows that in this hunger to know more, morals and ethics are compromised and made researchers act in ways that would be truly inhumane by any standards.

PAVLOV'S (NOTORIOUS) EXPERIMENTS

In 1904, Ivan Pavlov, who would later go on to become an iconic figure in the field of psychology, won the Nobel Prize in Physiology. He was awarded the Nobel "in recognition of his work on the physiology of digestion, through which knowledge on vital aspects of the subject has been transformed and enlarged."

This contribution closely relates to his work on another fundamental concept- classical conditioning. Classical conditioning is a process by which unconditioned stimuli are associated with a neutral stimulus. The popular base of the experiment is ringing a bell (neutral stimuli) while food (unconditioned stimuli) was served to a dog. This results in salivation in the dog's mouth as a response. If we repeat this several times for a set number of days, the dog will associate the ringing of the bell with food being served, hence next when we only ring the bell, the dog will still salivate due to this "conditioning". This is usually where the story ends, but there are a few significant details that are not commonly known [3, 17, 18].

Ivan Pavlov was never a dog lover. In his lab notes, he described them as lethargic, timid, and greedy animals. His lab was a factory to him and dogs were machinery to be studied, hence, their well-being was never a concern of his. This example of classical conditioning was indeed what he conducted with his subjects.

However, he made several significant improvements to make his experiment more 'efficient'. One of them was to use electric shocks instead of the bell. Despite the agony the subjects suffered, he prioritized precision in the quality and duration of the stimulus.

Furthermore, he removed the dogs' oesophagus and made an opening called a fistula in their throat so that any food they consumed would fall out and never make it to the stomach. They would be constantly salivating, forever hungry, and doomed to die an agonizing death.

However, the majority of his original work in this area was not focused on this 'positive' reinforcement, in which a positive stimulus or reward was coupled with a neutral stimulus. His work in this field was mostly on negative reinforcement. These negative reinforcements included flooding their cages, making them believe they were about to drown, and constantly kicking or pushing them down a flight of stairs, establishing a dread of stairs in them [1, 7, 11, 16].

Unfortunately, dogs were not the only living creatures that piqued his curiosity. There was another point of significance for him to examine- the human mind. He obtained orphans from childcare homes, young vulnerable minds to participate in the same tests as the dogs. They were strapped down to chairs, mouths forced open by tape and force-fed food. This was done to introduce stimuli, while their salivation was constantly monitored [1].

In present day, these experiments seem preposterous and highly unethical in the psychological research field. However, due to the framing effect that has been propagated about these classical experiments, the negatives of these experiments have been obscured. The lack of precise, complete details creates a faulty perception of history and practices in general, which further creates a diminished sense of responsibility in future researchers who may be laxer with these concerns.

Most people today know about the bell experiment with dogs but have never heard of the inhumane treatment Pavlov subjected them to.

Around 2007-2009, an experiment conducted by Dr. Edward Tronick was popularly called the "Still-face experiment". This experiment had a very simple premise - an infant was observed as his mother sat unresponsive, maintaining a "still face" to the infant's actions.

The experiment's findings show that as time passes, the infant grows increasingly distressed, and eventually becomes highly agitated and cries as a result of the stress [2, 6, 19].



Figure 1: Dogs like this, were experimented on by Pavlov, often unethically
Cute Dog hiding inside a Cage by Irina Zhur via Pexels (Copyright and Attribution-free)



Figure 2: Baby meant to represent the age group on which the still-face experiment was conducted on

Grayscale Photo of Girl in White Dress by Brett Jordan via Pexels(Copyright and Attribution-free)

This experiment already bears a disturbing resemblance to the 1950 experiment conducted by Harry Harlow that included the separation of infant monkeys and offering them the choice of inanimate surrogate mothers instead, which were periodically taken away, much to the monkey's distress. The reaction of the monkeys seems very similar to the reaction that the human babies had in the still-face experiment. [2, 4, 10, 19]

However, even more alarmingly, a video of the observations was shared online on YouTube. This video, which has had over 16 million views [2], piqued the interest of many people who replicated the experiment with their children, with little care or thought for their emotional needs. This experiment acts as a dangerous tipping point because it has inadvertently put the fragile well-being of infants in jeopardy in the name of "knowledge".

ETHICAL PRACTICES TODAY

In the present day, ethics in the field have become stricter and most researchers try to follow ethical practices. Ethics are essential to protect not only the participant and the researcher but

also to uphold the integrity of psychological research so that no wrongdoing happens in the name of research.

Researchers are today expected to conduct their studies with participant protection, informed consent, and confidentiality in mind. The researcher must make sure that the participant is aware of the study's specifics, obtain the appropriate consent, and avoid being dishonest in their methods. There must not be any conflicts of interest in the study, and the participant should not be harmed physically or mentally. It must also be ensured that the participants are treated fairly and are not made to feel uncomfortable. Further, participants' identities must be protected and the test should be designed to help both the subject and psychology as a whole. [12, 22, 24]

That is the bulk of what most ethics code of various psychological organizations promotes in studies in the field. These steps are necessary due to the delicate nature of psychological studies. It prevents the researchers from being found in breach of their principles and prevents the field of psychology from gaining a bad name. More importantly, it ensures that participants are saved from irrevocable damage during the course of experiments. A popular example of this is Ted Kaczynski, or as he was more popularly known, the 'Unabomber'.

THE "LAWFUL" TREATMENT

From 1959 to 1962, a psychological study was conducted by Dr. Henry Murray at Harvard University. This study was conducted on 22 graduate students to test how long an individual can withstand intense interrogation. The study started with the participants being told to write an essay on their philosophy of life and were told that they would then be debating on their ideas with another fellow participant. However, this was far from the truth. In originality, their documents were being studied by a law student who was prompted to find faults in their arguments and find the most degrading ways to discredit them for their views.

The participant would then be humiliated while the researchers watched. Then, after the participants had finally had enough, it would be revealed that the entire event had been recorded on film, and they would be shown the tape as a way to further poke their wounds.

One of these participants dubbed "Lawful" in the study was Ted Kaczynski. When he entered the experiments, he was a brilliant 17-year-old prodigy who had extremely strong opinions about how to live his life, not even aware of what the experiments aimed to find. These inhumane experiments were a strain beyond he could ever imagine, instilling fear and hate of technology and academicians.

He would later go on to kill 3 people and disfigure 23 others using letter bombs. [21, 23, 24, 25]

A ROAD OF CAUTION

While there are ethics codes in place, such as those established by the American Psychological Association (APA), it is clear that the line between ethical and unethical practices is a thin one. Unless researchers instill in themselves a sense of caution, they could very well stumble off the knife's edge. This situation aptly gives a reminder of the final line of the poem "Inchcape Rock" by Robert Southey. Future researchers have to make certain to follow ethical practices; else "The Devil below was ringing his knell" will certainly come true. [15]

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The Mind Gala

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The Neuroscience of Fainting

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SUMMARY

Fainting is a common reaction to stressful situations. It is caused by the lack of blood supply to the brain. When the neurons in the brain lose their required amount of glucose and oxygen, they temporarily shut down, causing one to lose consciousness. The reasons for this reduction in blood supply can be varied – based on situations or due to a pre-existing medical condition.

INTRODUCTION

Thud.

A collective gasp, a few scurrying adults, and a quarter of a minute later, we see the sports teacher carrying the boy from the next class across the field towards the infirmary. We all know what has happened, "He's fainted? On the first day of school?"

While the concept of fainting is not new to us—what with dramatic free-falls of actors on-screen (for a handful of the world's population, this holds, alas!), and early morning school shenanigans—the happenings inside the brain get left out. That is why several scientists have been trying to answer if the sun made you faint during morning assembly or if it is something else altogether.

Think of the body as a plugged-in TV. Now, imagine that the plug has come loose. At an opportune moment, the plug can come right off the power supply, and the TV switches off. Fainting is a lot like this. Doctors would define fainting—or medically called "syncope"—as the transient loss of consciousness that is quick to appear and leave. Insert the plug right back into the power supply to resume watching the TV.

The power supply of your brain is blood, bringing in sugar and oxygen for the cells of the brain. This shortage of blood is the main reason for fainting. This shortage of blood can be due to a fall in blood pressure, or reduced or irregular heartbeats, which in turn curtails blood flow to the brain. On the outside, fainting is sometimes also accompanied by a few other symptoms, such as nausea, dizziness, pale skin, overall body weakness, cold, sweating, blurred vision, difficulty in visual and mental focus, failure to maintain position, etc.; all of which can be tell-tale signs of an incoming episode of fainting [1].

CAUSES OF FAINTING AND TYPES OF FAINTING

The most common cause of fainting is the heart, more specifically, how fast or slow it beats. When the heart does not beat with its regular *lub dub* rhythm (a condition called arrhythmia) it leads to other organs receiving lesser oxygen and glucose. Remember the Snickers advertisement? A hungry M. S. Dhoni becomes what we call "hangry": angry and hungry. The brain's version of being "hangry" is going to unconsciousness. When the neurons of the brain are starved of glucose and oxygen, they shut down, causing one to faint.

Another type of fainting is based on reflexes to a situation. Think of a friend with a fear of spiders. When they see one crawling on the wall next to them, can you guess what that friend might do? Here, the body is unable to control blood circulation, because of which the neurons of our brain are left asking for glucose and oxygen. When this need isn't met, the brain simply halts or goes into standby mode, momentarily. There are sub-categories to this type of fainting—like the common faint, and situational faint, among many others, which are based on their triggers [2].

The common faint (or Vasovagal syncope as doctors like to call it) is due to emotional and physical distress. The actress who would drop to the floor when she hears that her lover has met with an accident, or the boy who faints at finding out he has a long-lost twin or someone who faints because of standing in the sun for very long, suffers from Vasovagal syncope. In this situation, the stress of the emotions or even unbearable physical strain causes the blood pressure to fall.

Fainting only in particular situations or activities is called situational fainting or situational syncope. Let's think of a scenario where you are a marathon runner, and every time you cross the finish line, you faint. A very peculiar case was when a patient was presented to doctors. After all, they fainted because they saw someone else faint [3]. Surprisingly, this person did not have a heartbeat because their heart had stopped. Fortunately, this patient was saved. Now, that is something one does not see in the most romantic of movies!

When someone has an existing condition, especially neurological, fainting happens to be quite a common occurrence for them. [4].

So, what's going on in the brain that makes us faint the way we do? Well, scientists aren't sure, but they do have a few hypotheses.

Serotonin is a hormone that is responsible for sleep, and mood, and for relaying signals from one neuron to another. The body chemically changes serotonin to a hormone called melatonin which is involved in sleep. Sleep is a type of unconsciousness. Knowing this, the role of serotonin in fainting cannot be overlooked. Serotonin is also involved with the parasympathetic nervous system. The sympathetic and the parasympathetic nervous systems are responsible for stressful situations or fight or flight situations and calming situations, respectively. [6]. In other words, a sudden change in levels of serotonin is supposed to cause a very low heartbeat. The role of serotonin in fainting however remains controversial and more and more exciting research is probing into this!

We've also seen that stress can contribute to fainting [7]. A study, conducted in 2009 surveyed the effect of psychological and social well-being on the treatment of the common faint [8]. They found that people affected by mental health disorders, or those in deep distress have frequent spells of fainting while also not responding well to treatments. In the same year, a clinical study observed patients with recurrent bouts of fainting [9]. Here the authors discovered that those patients who had severe signs of distress reported episodes of fainting that they couldn't explain. That means fainting very often may be a sign of some psychological disturbance, which might need treatment. The same study saw improvements in some patients after raising awareness and starting treatment. An investigation conducted among teenagers revealed that children who fainted often tend to have a lower quality of life as compared to their peers [10].

We don't know much about what happens inside the brain after one has fainted. An experiment conducted on 59 volunteers for induced fainting studied the effects of reduced oxygen supply to the brain (a condition referred to as cerebral hypoxia) [5]. The results showed that this reversible condition caused people to hallucinate (both visual and auditory), fall to the ground, and have involuntary movement of the muscles (myoclonus).

The researchers also use EEG (a scan of the electrical activity of the brain) to detect and analyze brain waves to see how brain activity is modified during the action of fainting. These jerk movements are commonly controlled by the maze of pipes you see in your textbooks, called the cortical region of the brain (Fig. 1). In this particular experiment, these jerk movements seemed to have their reins held by the brainstem (the bulgy portion that connects the brain to the spinal cord). While this is old news, since this was published in 1994, not much has been investigated after that in this regard.

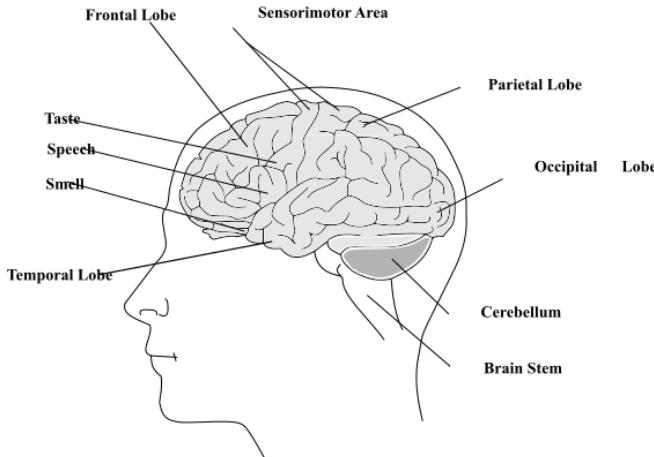


Fig. 1: Parts of the brain. The four lobes (Frontal, Parietal, Temporal, Occipital) make up the cerebral cortex or cortical region. The brain stem emerges from below the cortex and extends as the spinal cord.

(Image credit: Ozhank and <https://openclipart.org/detail/121615/human-brain-by-ozhank>)

BUT WHY FAINT AT ALL?

If fainting is so counterproductive and detrimental to human life, why does it occur at all? Let's be real, only actors on-screen ever accomplish their goals by fainting. Then why did the body not come up with a different solution to combat stressful situations? It seems odd that we turn off at a moment when we should be arming ourselves mentally to fight back, right?

From an evolutionary perspective, fainting is likely an adaptation rather than a medical issue. The earliest theory cites the Blood-Injection-Injury type specific phobia (BIITS phobia). A person shows three types of reactions: fear, avoidance, and fainting, which are consistent with a survival instinct that may have developed in the ancestral human. By fainting, they decreased

their chance of being gravely injured or killed by another human. This behaviour may have served sections of the population that did not engage in combat—most often women and children—but were targets for capture and torture. Fainting rendered them useless for these purposes, perhaps allowing them to escape [11].

Fainting became the path of avoiding danger and unpleasant experiences for the ancient peoples. By reacting this way, they increased their chance of dodging a very dangerous situation. This saved time and energy and eventually preserved life [11].

Research shows that females tend to faint at higher numbers than males during times of danger or stress. From a medical standpoint, this is shown by the fact that blood pressure in females is on the lower side than in males. A large contributing factor to this is the regulation of sex hormones [12].

RETROSPECTION

While fainting is a serious issue, it is an evolutionary development designed to conserve life and energy, the primary goal of humans for eons. But it is a sign of physical, mental, and emotional weakness in modern times since the earth is no longer made of hunters, foragers, and gatherers. We have out-evolved this survival mechanism, and it is now a signal for the distress of multiple facets of the body.

But the occurrence of an episode of syncope can prompt one to identify and address the root of the issue. So, although it's all right to faint, it isn't all right to faint.



Fig. 2 Hero from Shakespeare's Much Ado about Nothing swooning on hearing the accusations against her.

Image Credit: Elmore and <https://journey-and-destination.blogspot.com/2013/09/shakespeare-scenes-in-art.html>

Fainting as a cultural phenomenon is much more commonly referred to as swooning. In William Shakespeare's "Much Ado About Nothing" the crux of the story revolves around confusion, evil plotting, and marriages, one of the heroines of the story. Hero, faints at the accusations hurled at her (Fig. 2). William Shakespeare, one of many examples of using

fainting as a plot device, was very reflective of the culture prevalent in Victorian Era societies at the time. It was sometimes expected to faint in particular situations: to emphasize how affected they were by the news for example.

Swooning has its place in cultural history as far back as the medieval ages as there are instances of people fainting not only at disgraceful accusations but also at proclamations of love, death, or a hidden past. Charles Dickens's Bleak House has the heroine Lady Dedlock faint at possibly her past that she worked hard to forget and conceal.

Swooning might be best described in the words of the feminist author, Angela Carter in her critically acclaimed fictional novel, 'The Bloody Chamber', she writes, "After the dreadful revelation of that bloody chamber, it was his tender look that made me faint".

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The Mourning Mind: Why we lose ourselves in grief

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SUMMARY

Even though grief originally evolved in our brain's chemistry to cope with the death of a loved one, the loss of a long-term plan, for instance, a leg or faith, are also computed as losses of parts of ourselves and hence, as grief in our brains. The article delves into the neurological basis of grief.

INTRODUCTION

Grief refers to the loss of anyone or anything which holds great meaning in our lives. This could be specific people like family and friends, pets, or even the loss of faith or ideas of oneself. On the other hand, grieving is a way of learning to adjust to this new world, one without your loved one in it, one coping with the loss of your identity somehow.

HOW DOES GRIEF MANIFEST?

Everyone loses someone in their lives at some point. Especially when that someone is an integral part of their lives, it can be hard to navigate one's life after their demise. Grief is as personal and subjective as it gets. They say grief has five stages- denial or the tendency to not accept the truth, anger, bargaining or when one wishes that their loved one would be saved as a result of them changing their behaviour, depression, and finally, acceptance. However, having experienced it, I find myself oscillating between the stages continuously. Instead of a linear process, I have found grief to be more circular or cyclical.

The mammalian brain is used to creating neural or brain maps of the places we frequent, like our house, our workplace, the café we go to, and so on. Similarly, it also creates maps of our loved ones. We know how and where we can find them. Mary Frances O'Connor wisely compared it to a piece of furniture in our living room in her book *The Grieving Brain*. We would not expect the dining table in our living room to go missing, the same way we would not expect our loved one to disappear.

To investigate the neuroscience of grief, Norwegian Neuroscientists Edvard Moser and May-Britt Moser used rats as model organisms. Rats are prevalent in neuroscience research because of their similarities in the complexity of their brain to humans. They also have a shorter life span making them conducive model organisms for the study of neurological diseases.

In their study, Edvard and May-Britt Moser found that a class of cells they called "object-trace cells" in a brain region called the lateral entorhinal cortex, was activated when a rat was in a location where it had previously encountered an object [1].

So, this same class of cells would also expect our loved ones to be at a particular location or somewhere on the map. Therefore, their disappearance from the map of the world makes grief extremely overwhelming [2].

MY PERSONAL EXPERIENCE

I lost my father to Covid last year. For someone who has not lost anyone in their life, this one was very hard to tolerate. I still felt like the effects of grief were a lot harder on my mother compared to me. Since I had been living away from my family for four years at the time, my daily life did not involve my parents while my mother's whole life revolved around my father. There was a study conducted where widows/widowers were shown photos of their deceased spouses. The brain region responsible for the processing of rewarding stimuli, called the nucleus accumbens was activated in these subjects.

The reward circuitry in our brain connects multiple brain regions that control our ability to feel pleasure. Feeling pleasure motivates us to repeat behaviors. This means that the subjects have been unable to adapt to life without their loved ones. This feeling of motivation and reward that they experience when looking at pictures or videos of their loved ones is very relevant to the symptoms of complicated grief [3].

Prolonged Grief Disorder, earlier referred to as 'complicated grief' or 'traumatic grief' has recently been recognized in the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5) and the International Classification of Diseases (ICD-11). Around 10% of bereaved people experience this disorder.

The symptoms have been outlined as follows [4]:

- Feeling as though part of you has died
- A sense of disbelief about the death
- Avoidance of reminders that the person is dead
- Strong emotional pain related to the death (anger, bitterness, or sorrow)
- Difficulty moving on with your life (socializing with friends, pursuing interests, planning for the future)
- Emotional numbness
- Feeling that life is meaningless
- Extreme loneliness (feeling alone or separate from others)

HOW DOES GRIEF RESEARCH WORK?

Research on the neurobiology of grief relies heavily on neuroimaging techniques, particularly one called functional magnetic resonance imaging (fMRI). fMRIs have magnets that detect blood flow because of the iron in the blood. After a neuron fires, the blood flow in that region increases to restore oxygen levels. Neuroscientists look at regions with increased blood flow to infer which neurons are firing during a particular experience [5].

To examine the brain regions involved in grief, Gundel et al. showed pictures of the deceased and that of a stranger, combined with death-specific words and control-neutral words to eight bereaved women.

They went on to carry out fMRI studies on these subjects. They saw a combined activation of the brain regions involved in processing emotions, analyzing closeness or attachment to a person, recalling episodic memories, recognizing familiar faces, and coordinating all these activities. On one hand, this study is a crucial step forward in helping people understand how science can be considered in studying grief from the brain's perspective. On the other hand, this is just a list of the brain regions involved in grief and not grieving.

Prolonged grief, unlike depression, cannot be treated by antidepressants. M. Katherine Shear, MD, a psychiatry professor, and director of the Centre for Complicated Grief at Columbia University, developed a grief treatment that draws from interpersonal therapy and treatments for post-traumatic stress disorder (PTSD). Her treatment has a 16-session protocol that focuses on seven topics: understanding grief, thinking about the future, improving relationships, managing painful emotions, storytelling, learning to live with reminders, and remembering the person. Shear stated that across multiple studies, this therapy improved 70 percent of people with prolonged grief [6].

A clinical trial was conducted among 95 people diagnosed with complicated grief, who randomly received either interpersonal psychotherapy or complicated grief treatment. Both treatments resulted in an improvement in complicated grief symptoms, while the response rate and the time to respond were better and quicker, respectively in subjects treated with complicated grief therapy.

While all this scientific understanding of the neurochemistry of grief provides hope for understanding it, this is just the beginning of investigating this powerful human experience and hopefully, improving the quality of life for those who have undergone it.

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The Mind's User Manual

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SUMMARY

To understand the mind, we need to understand its fundamental property, which is consciousness. Understanding consciousness allows us to become aware of our conscious experience. Once that is achieved, we can understand the true nature of our existence, which is that of an observer. The next step is to enhance our conscious experience to reach a state of optimal neurochemistry. In Eastern traditions, various techniques are described to help us achieve such states. One of them is meditation. Meditation has scientifically proven benefits ranging from increased cortical volume to decreased stress levels and overall well-being. It is an essential tool that can help us navigate the complexities of the modern world. With an increasing focus on research, the field of mind sciences is poised to make significant breakthroughs in the coming decades. This is the perfect time for humanity to adopt practices like meditation and mindfulness daily, which will ultimately propel us toward the next stage of the cognitive revolution.

INTRODUCTION

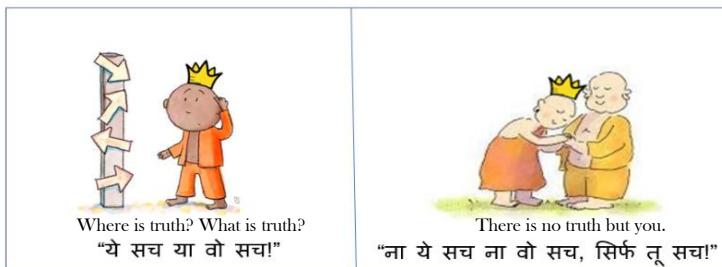
We are all aware that our extremely complex and highly developed brain makes us one of the most intelligent organisms on the planet owing to our evolved cognitive abilities. The question then arises is, how does this critically complex brain function so smoothly and how can we use our brain abilities more effectively to make our lives better? Let's explore our mind's user manual in this article.

"I think, therefore I am" – Rene Descartes

THE KING'S DILEMMA

It was the middle of the night and King Janaka, the ruler of Janakpuri (a kingdom in Ancient India, now Nepal), was sleeping peacefully in his private chamber. A sentry came rushing inside shouting, "Maharaj, Maharaj!" The king woke up, startled by the noise. "Sorry, Maharaj," the sentry apologized, "but the neighboring kingdom has attacked us. We must move swiftly." The King quickly gathered all of his generals, a strategy was mapped out, and the battle started. It was a fierce one, with heavy casualties on both sides. Ultimately, King Janaka was defeated. His life was spared by the enemy king, but he was exiled from the kingdom. Tattered, exhausted, bruised, and depressed, the king wandered in search of food and shelter. A passerby, being unable to recognize the King, thought of him as a beggar and gave him some bread. The king thanked the stranger and found a nice spot nearby to eat and rest. As soon as the king was about to take his first bite, a monkey swung from the tree above and snatched the piece of bread. The King, distraught by his fate, let out a loud shriek in despair. Suddenly, a flash occurred before his eyes, and the king heard a familiar voice. "Maharaj, Maharaj." When the king finally opened his eyes, he discovered the sentry sitting next to him in his chamber. I heard you scream and came rushing in. "What happened, Maharaj?" inquired the sentry. It did not take long for the King to realize that he had just woken up from a bad dream. The king could not sleep for the rest of the night. The king's psyche had been deeply affected by the dream. Having an analytical mind, he thought to himself, "When I was dreaming,

everything that was happening to me felt real, and here I am sitting in my chamber with my kingdom intact, and this feels real too. However, there cannot be two realities, so which one of these realities is truly real?" The king ignored court cases and other proceedings for days as he thought about it. The news of the King's strange behaviour spread in the city. Ashtavakra Muni, a well-renowned sage, was on a visit to Janakpuri. He heard about the king's condition and decided to visit him. The King, upon hearing the news of his arrival, greeted the sage and explained the whole ordeal to him. Then the king questioned him, "O Great Sage, which of these two realities is real?" The sage smiled and replied, "Neither that reality was true, nor is this reality true. The only truth is the observer, the consciousness that is present during and experiencing both realities, which is everlasting and all-pervading. Without consciousness, there is no experience, and without experience, there is no reality."



WHAT IS CONSCIOUSNESS?

The human brain is unique due to its cognitive and reasoning capabilities. Amongst all organisms, the human brain is the most evolved and complex. Despite its complexity, the brain manages to function perfectly. It is due to the peculiar quality of humans known as consciousness.

To explain, we can take the simple example of a computer. There are millions of background processes running on a computer, but we only see a simple wallpaper and some icons on our screen.

That is the most simple and optimal way to look at a computer. Similarly, consciousness, or "awareness of oneself, or one's thoughts, feelings, or emotions," is our brain's way of refining the millions of synaptic transmissions happening in our brains every second into a simplistic and reductionist movie that we see and feel every day, every minute, and every hour of our life [Fig 1].

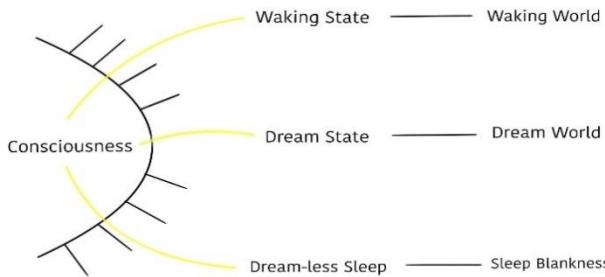


Figure 1: Consciousness illuminates the current reality which causes perceived experience

CAN WE ENHANCE OUR CONSCIOUS EXPERIENCE?

Anxiety and depression are widespread in the modern world. Rapid technological advancement, along with the changing social landscape, has had a vast impact on the general population. These effects were compounded by the recent pandemic. We are now facing a mental health epidemic. In these testing times, we are now faced with a challenge to improve and enhance the mental well-being of humanity.

Sages and monks, since ancient times, have used techniques like meditation and mindfulness to reach higher states of consciousness. They have achieved absolute control over their minds and realized the true nature of their existence, which is sometimes referred to as "enlightenment."

Enlightenment is not a metaphysical or a spiritual concept, but rather a state of optimal neurochemistry. People who ingest psychedelics like DMT, Psilocybin (magic mushrooms), and Ayahuasca often report having divine and spiritual experiences. They experience states of consciousness that are similar to what high-level mediators experience.

Meditation is simply getting comfortable with your thoughts, and mindfulness is being present at the moment. Recent research in neurobiology and psychology suggests that meditation and mindfulness practices improve the quality of life and well-being behavior of an individual.

HOW DOES MEDITATION WORK?

Meditation soothes the brain and makes the person feel calm by using a conscious effort to see one's thoughts. Meditation begins with focusing on your breath. This focus on breathing creates a sensory impulse in the body that is then transported to the brain. The parietal lobe, which sorts the incoming sensory information, passes this particular impulse to the prefrontal lobe—the cognitive and major response center of the brain, the hippocampus, and the insula via some neurotransmitters. These neurotransmitters, such as serotonin, melatonin, and GABA, cumulatively produce an instant feeling of relaxation and calmness. These activated brain parts and neurotransmitters together produce the calming effect of meditation [Fig 2].

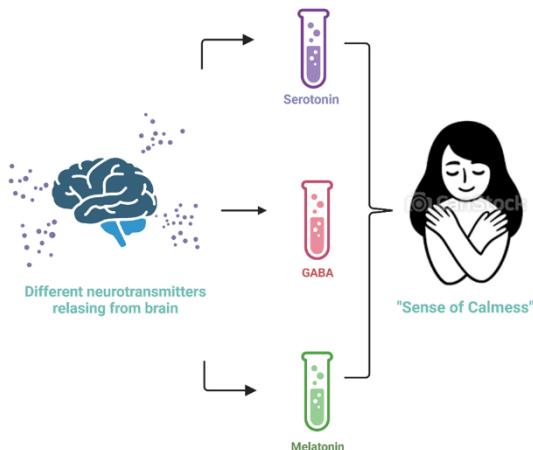


Figure 2: As a result of meditation, three neurotransmitters are released from the brain producing the effect of calmness.

RESEARCH EVIDENCE ON MEDITATION

The results of this study show a pre-post change in brain grey matter concentration in the volunteer's brain. These volunteers actively participated in a mindfulness meditation program. The observations suggest that participation in this program is associated with changes in grey matter concentration in brain regions involved in learning and memory processes, emotion regulation, and self-referential processing. It has been shown that mindfulness practice leads to increases in regional brain grey matter density (see Fig. 3).

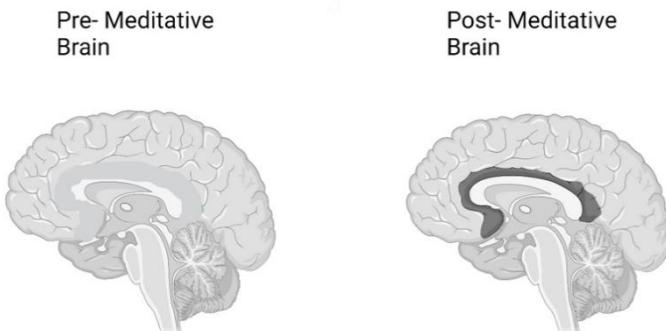


Figure 3: Gray matter increases at the learning center of post-meditative brain

The MRI analysis of the meditative and non-meditative control brain suggests an increased level of activity in the parietal and frontal lobes of the brain. These areas of the brain are associated with cognition and sensory thought processing (see Fig. 4 and ref [6]).

Effect of meditation on Human brain

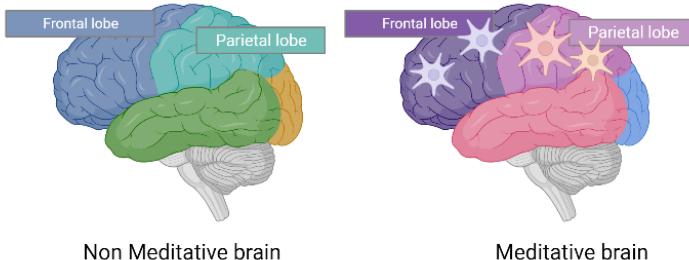


Figure 4: Increase in the activity of the frontal and parietal lobes in the post-meditative brain

Another study found that because mindfulness meditation teaches participants to let thoughts pass by without judgment, participants who learned meditation were less likely to dwell on negative thoughts about their speech and performance and treated themselves with more kindness and less self-judgment (see ref [5]).

FUTURE PERSPECTIVES

The best universities in the world, like Harvard and Stanford, now have well-established departments in Mind Sciences and Consciousness Studies. They are now looking at psychedelics as a way to understand consciousness and potentially treat various mental conditions like anxiety, depression, PTSD, substance abuse, etc.

Technological advances are paving the way for therapies like TMS (Transcranial Magnetic Stimulation) which have shown great success in clinical trials.

The biological basis of meditation will shed light on the cognitive and emotional processing systems.

Meditation can complement other medical treatments and it can boost a patient's mental strength. Combining pharmacotherapy and psychotherapy might be a great way to heal patients. As psychotherapies are accessible to everyone as well as cost-effective, Educating people and spreading awareness about meditation in society can improve social relations.

In a world full of distractions, finding our peace and taking a moment to get to know ourselves is an effective way of self-development, in our opinion. A small habit can lead to a significant change, and change is always good!

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Understanding the gut-brain axis

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SUMMARY

We are what we eat. The food we eat greatly influences our mental state. After entering our body, it is broken down with the help of enzymes and the microbes that colonize our gut to release the nutrients that are further absorbed into the body. Our body enzymes cannot fully digest dietary fibers and whole grains, so the microbes break them down to release small-chain fatty acids and precursors for hormones and chemicals that affect our brain and affect our mood.

INTRODUCTION

Raghav has a chemistry exam today. As his exam ritual, he had sugar with curd before leaving for the exam. For how many of you, sugar with curd was advised before leaving for the exam? My mother always said that having sugar with the curd supplies the brain with glucose and raises energy levels and focus [1]. According to ayurvedic tradition, food plays an essential role in maintaining well-being, healing, and longevity. It was also believed '*Jaisa ann, waista mani*' (your ability to think and reason depends on the food you eat). In this article, you will read and understand how the food we eat is digested with the help of microbes in the gut, the breakdown products, and their effect on the brain.

There are around ten times more microbes in your body when compared to your cells. Sometimes, I wonder if I am myself or a microbe-woman (like a spider man). All the microorganisms and their genetic material in our body constitute the microbiome (microbe + genome). The microbe population in each area, like the skin and respiratory tract, constitutes the regional microbiota [2].

The gut, also known as the gastrointestinal tract, is considered the second brain of the human body. The central brain is connected to different parts of the body by nerves and altogether forms the nervous system. The gut has an exclusive nervous system called the enteric nervous system. The enteric nervous system is linked to the central nervous system in many ways i.e., The gut is connected to the brain via the vagus nerve [3] and it transmits the information between the gut and brain, forming the gut-brain axis. Though communication is two-way, most of the information travels from the gut to the brain. See fig. 1 for the graphical representation of the gut-brain axis.

ABOUT GUT STRUCTURE AND FUNCTION

The gastrointestinal tract is majorly lined by a layer of sticky liquid called mucous, whose work is to lubricate the lining of the gut. This mucosal lining has many pit-like structures called gastric pits. Each pit comprises different cell types like parietal cells, mucous-secreting cells, chief cells, and enteroendocrine cells performing functions like secretion of acidic gastric juice, enzymes, and hormones to aid digestion. On this layer, the microbiota resides. Besides lubrication, the mucus layer protects the gut cells from the acidic gastric juice and prevents the microbiota from being recognized by our gut immune cells [4].

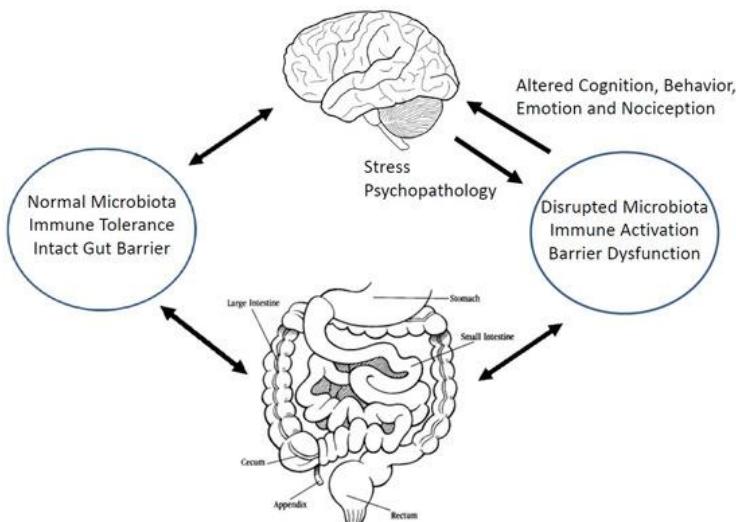


Figure 1. Gut-Brain axis

Image credit: Quigley EMM. The Gut-Brain Axis and the Microbiome: Clues to Pathophysiology and Opportunities for Novel Management Strategies in Irritable Bowel Syndrome (IBS). Journal of Clinical Medicine. 2018; 7(1):6. <https://doi.org/10.3390/jcm7010006>

Bacteroidetes, Firmicutes, Actinobacteria, Proteobacteria, and Verrucomicrobia are the five kinds of bacteria found in the gut. Of these, Bacteroidetes and firmicutes are the most common ones.

HOW DID WE GET THEM?

The moment the amniotic sac is broken, and we come in contact with the mother's birth canal (in case of normal delivery) or the mother's skin (in case of cesarean delivery), we get exposed to bacteria. Once out of our mother's body, we come in contact with millions and millions of microorganisms in our environment, mother's milk, the people we meet, etc. It is said that within the first 3 years, these microorganisms establish a stable profile in our bodies depending on our environment, diet, medicines taken, pets, etc. Like our fingerprint, our gut's microbial makeup is unique.

Just like how you have friends, enemies, and people who behave nicely to you if only you provide them with goodies, some bacteria are good, some are bad for your gut, while some are opportunistic pathogens [5]. The cells of the gastrointestinal tract are also under the control of gut microbiota. Like how different types of trees, shrubs, bushes, and creepers make a healthy, rich forest, diverse types of microorganisms are required to keep your gut healthy.

HOW IS GUT MICROBIOTA BENEFICIAL TO THE BRAIN AND US?

This microbiota protects us in many ways. They fight the harmful bacteria from the food and prevent them from establishing in your gut. They make hormones and vitamins and break down toxins from the food.

They play an essential role in digestion. These microbes help the gut digest the plant food products (dietary fiber, complex carbohydrates) that digestive enzymes cannot break down. Upon feeding on them, they become happy and release chemicals that affect the brain by acting on the vagus nerve. Some bacteria also release short-chain fatty acids (SCFA) like butyrate, acetate, and propionate.

These fatty acids are very beneficial for the colon cells and induce the death of cancer cells. Scientists have found that when SCFA is produced more, there are lower chances for diet-induced obesity. Eating a diet that is beneficial for the gut microbes can increase their numbers and positively influence the brain's health. And maintain its health [6].

And do you know? About 95% of the happy hormone serotonin is produced in the gut. Production of essential mood hormones happens in the gut with the help of food metabolites and normal flora in the intestine. Much research has confirmed the connection between gut microbiota and the brain. People suffering from gastrointestinal disorders were found to suffer from mood disorders like anxiety and depression.

Many of us would have experienced bad moods and anxiety the day after eating spicy, oily, unhealthy food. The food that we eat alters the microbiota composition in our gut. Scientists have found that when microbiota is absent, it causes delayed emptying of stomach contents, delayed intestinal movement, and enlarged caecum. What upsets humans more than stuck stools? They are enough to ruin your day. Microbiota influences how we react to stress, affecting memory functions. Consumption of probiotics is found to reduce the stress-induced release of cortisol, anxiety, and depression. Besides producing neurochemicals, they also help maintain the intestinal barrier, without which pathogens from the food will enter circulation and immune functions. Microbiota can also influence pain perception.

The SCFAs produced by bacteria influence memory and learning. Much research has discovered that alteration of microbiota causes alteration of behavior.

It was found that microbiota affects the nutrient breakdown from food. The enteroendocrine cells in the gut sense the nutrients and secrete peptides that act on the brain and other endocrine glands that help manage stress by secreting cortisol [7]. The gut alone is home to 1014 microorganisms. In recent times, the microbiota profile has changed globally drastically. Due to the consumption of hygienically processed food, frequent or irregular antibiotic use, and living 90% of our lives inside hygienic air-conditioned rooms, the diversity and size of gut microbiota have become smaller. These might explain the reason why mood disorders are on the rise in recent times [5].

HOW TO TAKE CARE OF MICROBIOTA?

Consumption of antibiotics can clear off all the microbiota from the gut. It disturbs the gut environment. Hence, it is advised not to take antibiotics for mild cough, cold, and fever. The brain also influences gut microbiota in many ways. It affects the secretion of mucus and acid, intestinal fluid handling and motility, maintenance of the mucosal layer, and immunity associated with it. Scientists have found that stress can alter the size and quality of mucus

secretion in gut, gastric and intestinal motility, slowing down gastric emptying. Due to delayed motility, enteric microbiota does not get the dietary fibers and prebiotics (food for microbes) properly [7].

Gut Microbiota should be taken care of properly. Foods like pulses, beans, and slightly unripened bananas containing resistant starch encourage healthy gut microbiota that breaks down complex carbohydrates into simpler nutrients that fuels colon cells (cells of the large intestine) and helps to destroy cells that have damaged DNA before they multiply and become cancerous.

If you do not feed the microbiota with dietary fibers and prebiotics, they will start to feed on the mucus lining of the intestine and expose the cells to acidic gastric juices. Homemade curd is the best example of probiotics containing live cultures of *Lactobacillus* and *Bifidobacterium*. Consumption of curd regularly helps maintain a healthy gut microbiota profile. Now, a new thing called psychobiotic has been introduced. They are probiotic bacteria used to treat mood disorders [5].

We co-exist with bacteria for a long time, and when the rapport between the human and microbial cells falls apart, it gives rise to disease conditions. The food we eat affects the gut microbiota, and fibers are good for them. Therefore, eat healthily and stay happy!

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Questioning your Way to an Answer: Rote Learning vs Critical Thinking

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SUMMARY

With its burgeoning young population, India makes up the second-largest education system in the world. The students of today will be making an impact on the world tomorrow. Hence it is imperative to assess if our current learning techniques are making students learners, thinkers, and innovators or just trained memorizers. This article introduces the concept of critical thinking and how students can practice it.

INTRODUCTION

The Indian education system is deeply rooted in rote learning, especially school education. Schools disproportionately focus on exam grades which are not an ideal reflection of one's capabilities. This environment is not conducive to helping children identify their best competencies [1]. Thus, it is impending to create an immersive experience for better learning with more environmental stimulations and interactions [2].

IS KNOWLEDGE ALL ABOUT GRADES?

"Learn this poem by heart tomorrow. We'll have an oral test where you'll be graded on how well you remember it."

This was a significant part of childhood for many of us; memorizing poems, stories, facts, formulae, and so on; the list never ends. What seemed like a question of life and death then (to get a good grade) now seems like redundant information to memorize. This is highly mentally taxing yet intellectually inefficient. In schools, kids are unable to muster up the courage to not commit to memory because that would entail lesser marks.

The experience of gradually giving up completely on memorizing things is liberating. One feels unfettered and unshackled from the binds of rote learning. It is a paradigm shift from memorizing to questioning our way to an answer, a shift that can set us free.

Promoting a free-will study program at educational institutes can inculcate critical thinking among youngsters.

One thing that makes the conditions in the classroom seem artificial compared with conditions outside it is that in the classroom, it is the teacher who asks questions, while outside, it is the pupil. Furthermore, the classroom organization is designed to maintain discipline where the teacher is in command. The function of the student is to obey. Overall, this creates a tense environment [3]. Imparting and gaining knowledge should be fun and not a task, both for the student as well as the teacher.

The idea of relaxed alertness [2], i.e. learning without fear- fear of punishment, fear of deadlines- is well known among students as 'leisure reading,' which, unfortunately, they are seldom allowed to do. Students then find themselves in a helpless situation where they are made to read subjects they are not interested in, only to score good marks on a test. How ironic!

HOW ROTE LEARNING AFFECTS OUR BRAIN?

Hippocampus is the most stress-sensitive area in the brain. Also, it is the relay center for many processes in the brain. Thus, every stressor affects the hippocampus first and foremost, impairing memory function. And, because it is the relay center for many other processes in the brain, a fearful environment for studying hampers the overall functioning and, thus, the efficacy of the brain as a whole [4].

Past electroencephalography (EEG) studies compare generalized learning using a large set of novel stimuli to rote learning using a small set of repeated stimuli. An active processing component of perception was observed in generalized learning, which indicates the ability to adjust rapidly to new stimuli. Rote learning showed no positive effect on the pattern of recognition [5].

Sleep studies were done to examine the effect of sleep on both types of learning. Restored performance and consolidated knowledge after 12 hours were noted in the case of generalized learning. No such improvement was seen during rote learning. Sleep helps to stabilize associative interactions through neuronal replay [6]. To better understand this, generalized learning is like learning a new language from a native speaker. Apart from learning new words and their meaning, you further pick up on subtler and more abstract stimuli like the tone of their voice, social cues, and body language. Compared to this, rote learning is like learning the language solely from a dictionary. These differences suggest a functional dissociation between the two types of learning; with generalized learning building pattern recognition and with rote learning centered merely around information storage and retrieval.

Our brain knows the best way to process information. Thus, we can learn how to learn from our brains. By imposing rote learning on ourselves, we end up being a stubborn hurdle in the brain's natural information-processing procedure. Brain theory tells us that the brain continually attempts to categorize and pattern new information on what is already stored [7-9]. Thus, concentrating too heavily on storing and recalling non-associated and discrete facts is a very inefficient use of the brain [2].

Critical thinking makes us lifelong learners. It promotes free thinking and innovative ideas. Rote learning makes us trained memorizers. We blindly read and learn what is given in textbooks without questioning it. Without discussion, words may perform their original function of expressing sentiments, but they will never become the symbols of intelligible ideas [3].

TOOLS TO DEVELOP CRITICAL THINKING

One of the tools for developing critical thinking is a method of questioning and dialogue called the Socratic Method [16]. Socrates was a Greek philosopher who lived in 500 BC Athens. A lot has changed since then. However, the way our brains work hasn't changed much. The techniques that Socrates used to compel people to start thinking are still successfully used in many fields. Its ubiquity comes from its simplicity.

The Socratic method aims to pose a series of questions such that it defines the matter, analyses it and finds exceptions to it. For instance, take a common belief that students have- "Math is hard". You can start a line of questioning - "Why is math hard?" One may reply, "Because it takes a lot of effort, and we get answers wrong many times." This can be followed by, "Is everything that takes effort and has low accuracy hard?" One might say, "Yes". We can further challenge our line of thinking by introducing contradictions - "What about video games which take a lot of skill, and one usually fails a lot of times before they get it right? Are they hard too?" This makes you think, is math just as hard as video games [17]? Do effort and accuracy determine difficulty? Or is it something else entirely?

This way, you start to analyze your ideas and think from different perspectives. The irony-cum-beauty of the Socratic Method is that it doesn't aim to find a definite answer, but rather it compels the individual to ask better questions. In the end, even if we do not conclude, we still have a better understanding of the topic. And this sets us up to find better solutions.

What makes this method effective is the underlying acceptance of one's ignorance. Socrates never considered himself a wise man or a philosopher. He never wrote books or gave lectures to the crowd. He sought knowledge from the wise people of his time and tried to understand their reasoning the best he could. This simple method of questioning helped him break down complex topics and find gaps in people's thinking.

Many other thinkers have also practiced this learning style, including scientists and writers like Richard Feynman, a Nobel Laureate physicist and educator, who gave rise to the Feynman technique [18] popular today.

How can we apply these principles to our own learning experience and our current position in the education system?

At an individual level, we can start by acknowledging that not knowing answers is an opportunity to foster a deeper understanding of the subject. There is a phenomenon termed "pluralistic ignorance" [19]. It is when a room full of people have the same doubts but don't ask any questions because most think they are the only ones with this doubt. Rather, the question highlights a knowledge gap that most in the room experience. This is a very common classroom scenario. In a case like this, asking a question can be a window to deeper learning rather than a potential source of embarrassment.

Thus, using the Socratic Method, we can turn that amorphous feeling of "I did not understand it" or "the topic is too hard" into identifying what exactly we do not understand. Being clueless is a common experience; what is essential is to deal with the situation and transform it into a learning experience.

At the institutional level, we can incorporate teaching models which foster critical thinking, like the RED Model of critical thinking - recognizing assumptions, evaluating arguments, and drawing conclusions, which helps develop problem-solving skills [20]. Further, the blended learning approach can be used [21], which combines classroom learning with Socratic discussion and real-life experience-building activities. This can be lab experience, hands-on projects, shadowing, and internship opportunities.

HAPPY READING

Have you ever wondered why kindergarteners talk passionately about what they do in school, unlike high schoolers? It is because when the fun stops, learning also stops [10].

Quoting Professor Richard Feynman, "Students don't need a perfect teacher. Students need a happy teacher who's going to make them excited to come to school and grow a love for learning." Thus, one should always propose the idea of happy classrooms and happy teachers. This will bring positive emotions into a solid cognitive association with studying, which is scientifically supported [11-15].

Rather than forcing students to learn a fact in a textbook, they should be allowed to think and question.

But, to question something, first and foremost, the acceptance of not knowing enough is essential.

Quoting Robert E. Park, "I have found it difficult, in some of my classes, to induce students to ask questions. I found it difficult to get them to admit they did not understand what they had read in the textbook or had heard in the classroom" [3].

Question even your answers and then question your way to another answer. This is imperative because if we limit ourselves to the first answer itself, we will reach a plateau in growth and stagnancy in innovation. Thus, keep questioning yourselves for more intellectual answers.

CONCLUSION

It is imperative that from the very beginning, we teach children to think in a way that builds a cohesive knowledge base. Imposing unconnected factual information upon them kills their creativity, curiosity, and interest.

Harmonizing new experiences with the old in a rich, non-threatening, encouraging, joyous, and interactive environment helps students learn naturally. We should allow students to think freely, question and cross-question their way to better questions, thus paving their way to better solutions. The education system must be designed to facilitate this.

What topics would you like to question? What questions would challenge you the most? How would you decipher them? Critical thinking is a process unique to each of us, and it is vital to how our brains work. How and where do you want to implement critical thinking in your life?

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Changes in Brain Activity in Response to Lifestyle/Behavioural Aspects

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SUMMARY

The brain, referred to as the ‘Master organ’, determines the functioning of the body. A healthy lifestyle - with healthy social interactions, diet, and sleep - can lead to greater plasticity in the brain. Neuroplasticity is the ability of the brain to form newer synaptic connections, and this phenomenon is hugely aided by a healthy lifestyle that increases the efficiency of neuron. Just leading a healthy life, however, is not sufficient. A person needs to learn new skills to build newer synaptic connections. The brain has the lifelong ability to adapt, build and reorganise neural pathways, regardless of age. One is never too young, and never too old to learn! (Fig. 1).

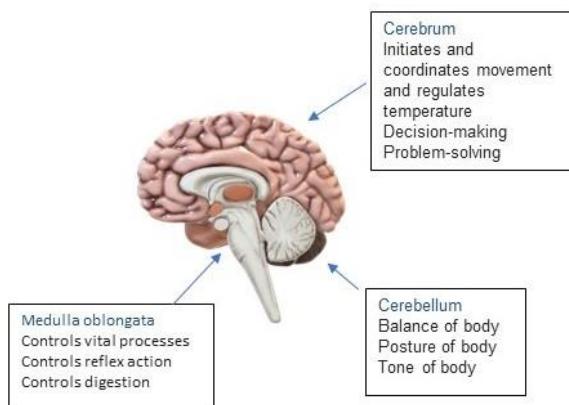


Figure 1. Diagram of the brain.

Image credit: Shivani Pimparkar

INTRODUCTION

The body is capable of performing several amazing feats- from perfectly coordinated metabolic reactions at a molecular level to astonishing physical activities like athletics and adventure sports.

Ever wondered about what controls all these processes? The answer is fairly obvious, yet interesting: the brain. The word ‘brain’ has been aptly derived from the old English word ‘braegen’ which means “wisdom”. The brain controls all life processes, from memory, motor skills and sensory modalities, to respiration, thirst and hunger, that maintain the well-being of

the body. However, the brain is not alone in regulating the body; the Central Nervous System formed by the brain and the spinal cord, is holistically responsible.

CONSTITUENTS OF THE BRAIN: TRAVELLING BEYOND MILES

- **Neurons** are the basic functional unit of the brain.
- Neurons are responsible for **conducting impulses** from the brain to other parts of the body **through the spinal cord** in the form of **electrical impulses**. A neuron is connected to another neuron at junctions called the **synapse**.
- The brain, weighing roughly 1.3-1.5 kg constitutes about 60% fat, making it the **organ with the highest fat content** of the human body. Researchers discovered that this fat cannot be reduced, mainly because they are among the **most crucial molecules** to determine the brain's integrity and performance.
- The remaining 40% of the brain is composed of a mixture of proteins, carbohydrates and salts.
- The **blood vessels, nerves and capillary networks of the central nervous system** extend up to **400 miles** (approx: 643 km). (Fig. 2).

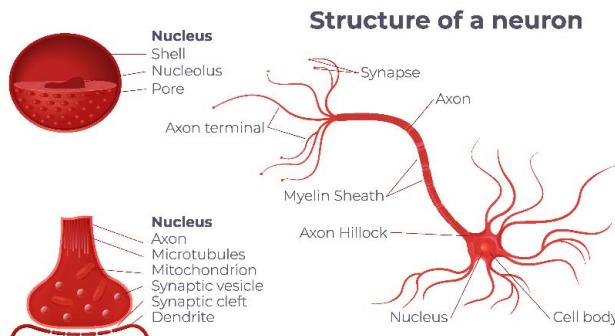


Figure 2. Diagram of Neuron.

Image credit: Macvector and <https://www.freepik.com>

NEUROPLASTICITY

Neuroplasticity or brain plasticity is the lifelong tendency of the synapses, neurons or even entire brain areas to change depending upon their use. Neuroplasticity specifically refers to strengthening or weakening synaptic connections, or adding new nerve cells based on external experiences. There are a variety of mechanisms by which neuroplasticity can occur: axonal sprouting and synaptic pruning are among the most common.

AXONAL SPROUTING

In axonal sprouting, healthy axons sprout new nerve endings that connect to other pathways in the nervous system. This can be used to strengthen existing connections or to repair damaged parts of the nervous system and restore them to full functionality (Fig. 3.).

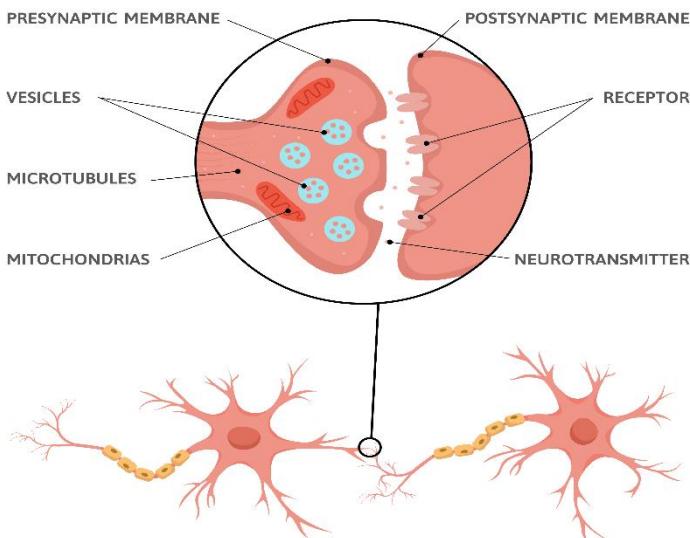


Figure 3. Diagram of Neuron.
Image credit: Macrovector and <https://www.freepik.com>

SYNAPTIC PRUNING

The sense organs of a new-born baby allow a huge influx of information from the world outside. This sensory information must somehow make it back to the brain for processing. To do so, neurons must make connections with one another, to transmit the impulses to the brain. Over the next few years, the brain grows rapidly. As each neuron matures, it sends out multiple branches (axons, which send out information and dendrites which take in information), increasing the number of synaptic contacts and laying the specific connections from house to house, or in the case of the brain, from neuron to neuron.

At birth, each neuron in the cerebral cortex has approximately 2500 synapses. By the time an infant is 2-3 years old, the number of synapses is around 15000. This amount is about twice that of the average adult brain. As we age, old connections are removed through a process called synaptic pruning. Due to synaptic pruning and axonal sprouting that lead to neuroplasticity, damage to the brain or other parts of the nervous system is **not always permanent**. This process is key to making humans **adaptable to a broad range of adverse circumstances**; the very physiology of the brain can change in response to a given set of conditions.

FACTORS AFFECTING BRAIN ACTIVITY

Social interactions

Being socially active, especially later in life, brings many mental and physical health benefits. Social interactions in a group can protect an individual from cognitive decline, according to a new study. One study found positive relationships between reduction in age-related memory decline and the quality of social interactions. There has been evidence showing a positive correlation between the ability to manage emotions and social ties, **supporting the predictive and incremental validity of an ability measure of emotional intelligence**, the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT).

Diet

It has long been hypothesised that the relative quantity of particular nutrients can have an impact on mental and emotional functions. Some of the key mechanisms underlying the effect of nutrition on brain health and mental function have recently been documented and suggest a heavy impact of dietary variables on synaptic plasticity and neuronal function. Several gut hormones that can travel to the brain, or those that the brain produces on its own, have an impact on cognitive function. Furthermore, well-known synaptic plasticity regulators, such as brain-derived neurotrophic factors, can operate as metabolic modulators in response to extracellular inputs like dietary intake. The brain needs sufficient fuel to perform optimally. High-quality “fuel” helps the brain to function at its best. Foods rich in vitamins, minerals, and antioxidants nourish the brain and protect it from oxidative stress, which is an imbalance between the production of free radicals (molecules with one or more unpaired electrons) and antioxidants (substances that neutralize or remove free radicals by donating electrons) in the body, that can ultimately lead to cell and tissue damage.

On the flip side, low-quality “fuel” can damage the brain and its functions. Processed or refined foods (including refined sugar, for example) fall into this category. These foods negatively affect insulin regulation and increase inflammation and oxidative stress. Many studies have found links between sugary diets and impaired brain function—and even mood disorders such as depression.

Studies in this area are known as nutritional psychiatry. This is a growing field that had been left unexplored for many years. So how do researchers and doctors know which foods affect our overall brain health? Simple: they follow the food we eat. And it all starts in the gut! (Fig. 4).

Exercise

Have you wondered why exercise is crucial? In a study done at the University of British Columbia, researchers found that regular aerobic exercise appears to boost the size of the hippocampus, the brain area involved in verbal memory and learning. However, resistance training, balance and muscle toning exercises did not yield the same results.

New skills

Every time you learn a new skill, you change your brain. More specifically, you change the connections inside it. When presented with a challenging environment, our body and mind change: Muscles get stronger, hearts and lungs get larger, and brain connections become faster and more robust. This reorganization of the brain is the basis of all skill acquisition and development. The body itself does not know why these changes happen. But it is programmed

to constantly reorganize itself to make things as simple as possible. The body and mind will try to adapt in order to find the simplest way to perform a specific task. By straining the body when learning a certain skill, you are signalling that this is important, and telling it to devote more resources to this challenge. The results of practicing a specific skill are that we are strengthening neural connections, which makes it much easier to perform better.



Figure 4. Adapting a healthy lifestyle.
Image credit: Macvector and <https://www.freepik.com>

Effects of digital technology use

It is the digital era and the use of digital technologies, including mobile phones have become one of the most crucial components of our lifestyle. In some aspects it has simplified our life, however, it has a significant impact on our brain health. Many studies have drawn a link between computer use, extensive screen time, and the symptoms of Attention Deficit Hyperactivity Disorder (ADHD) specifically in adolescents.

Sleep

Sleep affects our brain activity as the information that was learned during the previous day will traverse through the neuron repeatedly, allowing us to consolidate the events into memories. If one doesn't sleep properly, the process of memory formation and consolidation is severely affected and leading to forgetfulness and decision-making deficits. (Fig. 4).

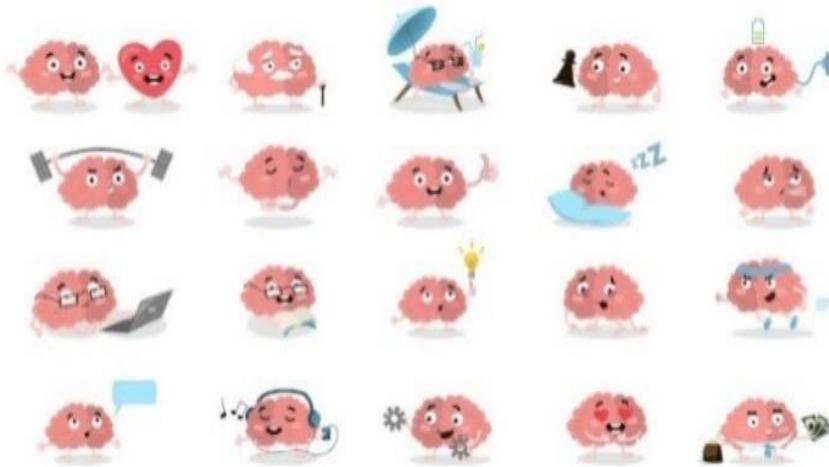


Figure 5. Learning new skills.

Image credit: vectorIstock and <https://www.freepik.com>

EFFECT OF EXERCISE AND SLEEP ON BRAIN FUNCTION

- People who exercise regularly are thought to have a lower risk of developing Alzheimer's disease. Exercise increases blood flow and memory consolidation; it causes chemical changes in the brain that improve learning, mood, and thinking abilities.
- As you get older, the brain is subjected to more harmful stress as a result of lifestyle and environmental factors, resulting in a process known as oxidation, which damages brain cells. Rust on a bike's handlebars or the browning of a half-eaten apple can help give an inkling to the kind of chemical changes that occur due to oxidative stress. Antioxidant-rich foods (such as berries and vegetables) can help protect the brain from the damaging effects of oxidation.
- Sleep energizes you, improves mood and immune system function, and may reduce the build-up of an abnormal form of the amyloid-beta protein leading to plaques forming in the brain, which in turn is linked to Alzheimer's disease.
- Mental exercises can help the brain function better and promote adult neurogenesis, lowering the risk of dementia. One must exercise their neurons regularly, just as one must exercise their muscles, or it may perform sub-optimally.
- Leading an active social life can protect you against memory loss. Spending time with others, engaging in stimulating conversation, and staying connected with

family and friends are good for brain health. Studies have shown that those with the most social interaction in their community experience the slowest rate of memory decline.

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Music and Our Minds

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SUMMARY

“One good thing about music is when it hits you, you feel no pain”

- Bob Marley.

Music therapy is an important tool that helps in enhancing brain function. The brain cells, called neurons, form connections with several others, creating an intricate neural network. They can continually make and break these connections, through a process called neuroplasticity. And, when abnormalities in brain function arise, music can come to the rescue by regulating neuroplasticity.

INTRODUCTION

“Music has a particularly calming effect on the broken mind...the right song, particularly one which holds some personal meaning can prove a salient stimulus”, says Warden Hatch, the head of a psychiatric hospital in the science-fiction drama ‘Stranger Things’ Season 4, about the healing power of music. The show also features a special listening room in the hospital that helps in providing relief and cure to the inmates. Such brilliant portrayal of how music helps someone escape a terrible fate, extends the boundaries of science fiction and finds meaning in neuroscience. Music or musical therapy can be used as a medicine to heal the brain. The brain, an incredibly complex organ, is a part of the central nervous system (CNS). Nerve cells or neurons form the basic unit of the brain as well as the nervous system. These are the longest cells of the human body, and consist of cell bodies, dendrites, axons, and axon terminals (Fig. 1). Different neurons communicate with one another by forming connections through which electrical or chemical signals are passed on. These connections are essential as they enable our nervous system to coordinate with itself and the rest of the body. Synapses or neuronal junctions provide for the site at which transmission of signals between neurons takes place. Chemical messengers such as neurotransmitters are released at the junction of the neurons causing chemical transmission of information from the pre-synaptic neuron (the cell sending the signal) to the post-synaptic neuron (the cell receiving the signal) (Fig. 2). A few well-known examples of neurotransmitters include dopamine and serotonin. It is interesting how the dendrites and axons of a single neuron can form complex connections with numerous other neurons, creating a vast network of connections.

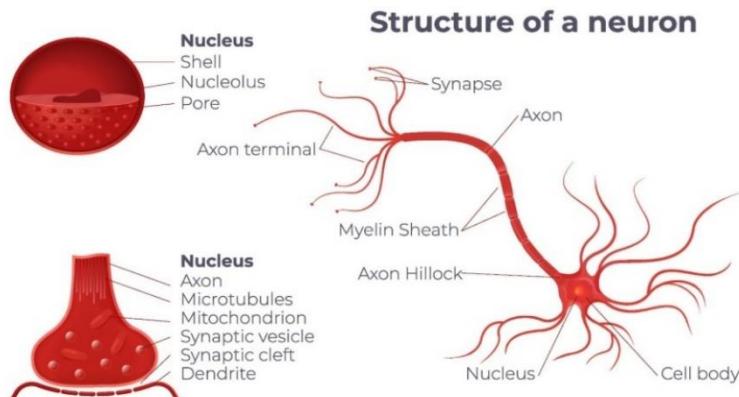


Figure 1. Structure of a neuron.

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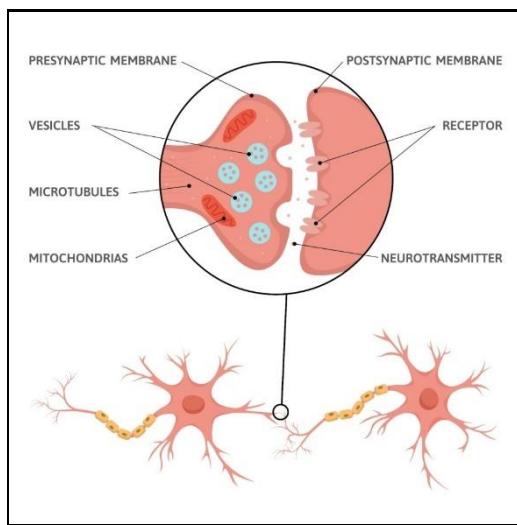


Figure 2. Diagram of a synapse.

Image credit: Designed by Macvector - Freepik.com https://www.freepik.com/free-vector/neuroscience-brain-synapse-flat-infographics-with-diagram-scheme-of-neural-cells-with-text-captions-and-pointers-vector-illustration_26762581.htm

NEUROPLASTICITY

A neuron can make millions of connections with other neurons throughout its lifetime, and just as they have the ability to connect to each other via synapses, they are also capable of breaking these connections. This process of eliminating unused neural connections is called synaptic pruning and is important in helping the brain redirect resources to necessary neural pathways [1]. The formation and elimination of synapses is best understood by considering brain development in infants and adults. From our birth, our brains contain more than 100 billion neurons that keep forming new connections up to the age of 3, soaking up an abundance of information from the environment around us. As one passes the age of 3, the pruning process begins with the removal of weak and infrequently used neural connections. This forming and 'deleting' of neural pathways is what is known as neuroplasticity [2].

Importance of neuroplasticity

Neuroplasticity has been a widely known concept, thanks to the role it plays in our learning, habits, behaviour, and addiction. As we go about living our lives and go through various experiences, our brains help us navigate through these by changing these neural 'wirings', either by strengthening them, an allusion to which could be made out along the lines of the famous quote "practice makes a man perfect" [3]. These wirings can also get eliminated. However, on the flipside, an excess of synaptic pruning can lead to various neurological disorders like schizophrenia [1]. A balance in retaining and eliminating wirings is therefore imperative. The fascinating thing about neuroplasticity is that it occurs throughout our lives. Although it is more rapid in the brains of young children, neuroplasticity continues, although at a much slower rate, up until our 20s. So, does this mean our chance to rewire our brain is only until the critical age of 25? Surprisingly, the answer is no as we can continue reshaping our brains as we want, even at an older age! Our grandparents learning to read, write, or simply use mobile phones can be attributed to the process of neuroplasticity. However, any form of trauma or unfortunate events in the life of an individual can have a negative impact on neuroplasticity and auditory stimuli, especially in the form of music, can be used to ameliorate this damage [4].

MUSIC & MUSIC THERAPY

Music is intricately linked to our lives. We all love the way music makes us feel. It can instantly lift our mood or make us nostalgic about our childhood. It can be an outlet for expressing complicated emotions, be instrumental in instilling patriotism within us, or help us relax as we sleep at night. The neurobiological benefits of music are also well known (Fig. 3). Studies show that playing instruments enhances communication between the two hemispheres of the brain improving learning, memory, fine motor skills, verbal and non-verbal reasoning, and the overall health of the brain [5]. Long-term practice of musical instruments is also found to increase nerve cell density in brain regions responsible for hand movements. The cerebellum responsible for movement coordination is also found to be enlarged in professional musicians [6].

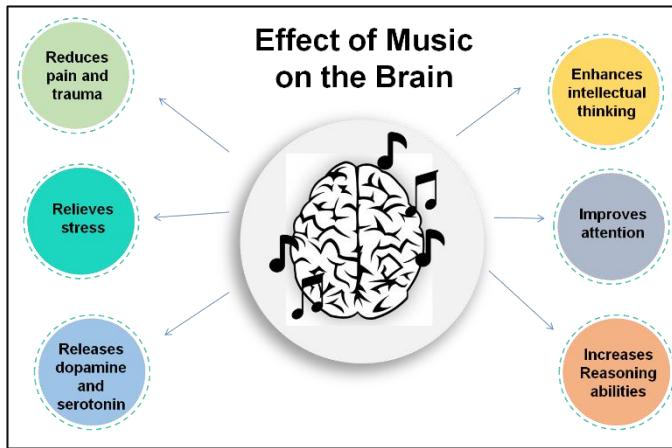


Figure 3. Beneficial effects of music on the brain.

Music Therapy

If music has such a profound impact on our brains, can it be used in and as therapy? The answer lies in the following example. Apraxia is a speech disorder where a person is unable to articulate or produce sounds necessary for speech due to poor coordination of neuromuscular movements in the mouth. 3-year-old Lily was diagnosed with childhood apraxia of speech and was unable to vocalize sounds like other kids of her age except for /uh/, /bah/, /oh/, and /puh/. However, Lily's love for music helped her learn to speak at a faster rate. Her favourite songs like the *Hello* song, *Old McDonald Had a Farm*, *Name Song*, *Pop Goes the Bubble*, and *Wheels on the Bus* along with interactive aids like drawing or flashcards enabled her to recognize and vocalize numerous letters and syllables. At the end of the 9 month-long sessions, Lily was able to vocalize words like /mama/, /dada/, /more/, and /no/, apart from syllables and combination sounds. Music therapy not only helped Lily improve her verbal communication but also her intellectual, emotional, motor, and socialization skills. As one of the milestones achieved through music therapy, Lily's case validates the importance of music and its healing effect on the brain [7].

Elizabeth L. Stegemöller, a music therapist and researcher, defines music therapy as the “use of music to maintain, improve, and restore emotional and physical health” [8]. Music therapy has long been used to treat various ailments such as aphasia, apraxia, dementia, Alzheimer’s, Parkinson’s, stroke, etc [9]. It also helps in providing relief from pain and in recovering from trauma. It helps improve patients’ reasoning and intellectual thinking (cognitive skills), speech, communication, emotional expression, motor skills, and social interactions [10]. Music therapy works by building new connections within the damaged region of the brain which enables the patients to overcome their limitations. Do you remember the famous song for learning the periodic table? Just like the tunes and rhythm that helped us learn the 118 elements of the periodic table, music therapy helps patients synchronize their cognitive activities to tunes of music so that their brains can learn and remember them. Through this,

the patients are able to strengthen their neural pathways for the specific ability and rewire the brain's circuitry to overcome their limitations.

Role of Neuroplasticity in Music Therapy

Elizabeth L. Stegemöller and Fulvia Constantin suggest three mechanisms through which music therapy uses the mechanism of neuroplasticity [2,11]. The first mechanism is the use of the neurotransmitter, dopamine for learning. Music stimulates our brains to rewire with the help of neurotransmitters or chemical messengers like dopamine. Listening, singing, or playing music releases dopamine which plays a significant role in learning, motivation, memory, and reward-seeking behaviour. Doing joyful musical activities (especially listening to your favourite or preferred songs) stimulates the release of this neurotransmitter from the mesolimbic system (region of the brain that contains dopamine-releasing neurons aka dopaminergic neurons). This link between motivation, learning, and reward due to dopamine promotes neuroplasticity or rewiring [6]. Music therapists use this principle and pair musical activities like singing or playing an instrument with non-musical tasks like walking, speaking, or recalling, thus helping to reinforce non-musical behaviour. The second mechanism that can explain the effect of music therapy is called the Rhythm and Hebbian Principle. The Hebbian principle can be summarized as 'neurons that fire together, wire together' [11]. In simple words, neurons need to fire in synchrony, to form and strengthen neural connections. Music therapy also works by synchronizing the non-musical action to the rhythm of the music being played or sung. Our brains find it easy to follow a rhythm while we do any mundane activity. An application of this can be seen in Flint Rehab's MusicGlove, a neuro-rehabilitation device wherein the user performs finger exercises while playing an engaging musical game [12,18]. As the user plays the game, they make pinching movements with their fingers when a musical note appears on the screen. This then improves the user's hand functions as they strengthen the neural connections responsible for those movements. The third important aspect of music in inducing neuroplasticity is the production of clear signals [2,11]. Unclear signals are perceived as noise, which can cause stress and consequently reduce neuroplasticity. Whether a sound (music or spoken text) is perceived as a clear signal or a noise depends on its structure & complexity. Music is found to be more structured and simpler than spoken text. Even within the realm of music, less complex music is often much easier to comprehend than complex music. So, such music, as compared to speech, reduces the noise present in the stimuli, thereby promoting neuroplasticity. Music therapists are therefore trained in order to produce more clear signals through their vocalization or instruments.

Music in clinical practice

Over the years the healing effects of music have found their way into numerous hospitals and clinics. Dr. Deforia Lane, in her TED talk, recalls how her voice was even able to reach the ears of the fetus in a 20-year-old brain-dead woman. As she sang to the baby, later in the neonatal intensive care unit, her voice helped calm the newborn and improved his vitals [14]. The article '[Glynne's Miracle: How Music Therapy Rouses a Rock Fan from Dementia's Darkness](#)' published in [Times of San Diego](#) talks about the healing power of music in overcoming dementia [15]. Similarly, music therapy is used to treat numerous conditions ranging from stammering, depression, Alzheimer's, Parkinson's, Aphasia, and even Autism Spectrum Disorder [9].

CONCLUSION

Music therapy is not a novel concept – it even finds its place in the Pythagorean era when Pythagoras was known to use music to heal physical and emotional problems [16]. But, today, in conjunction with neuroscience and the advancements in technology, music can be classified as a medicine. As a powerful tool to enhance neuroplasticity in the brain, music also paves the way for future research in understanding the basis of the neural wiring mechanisms that underlie all neural connections with the nervous system.

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Lilly



The logo of Eli Lilly and Company and pack of fluoxetine

(Fluoxetine was discovered by the American company Eli Lilly in 1986. It is commonly used to treat mental health issues such as Major Depressive Disorder, Obsessive Compulsive Disorder etc.)

PART IV Mental Health

Looking at Depression through the Lens of Evolution

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SUMMARY

Depression, or The Blue Devil, is a mental illness that has earned itself a bad reputation as a modern vice. The stigma around it is unfortunate and alarming. It's one of the major causes of suicide among the youth. Looking at it from an evolutionary perspective can give us an insight into its working and treatment. It can also help dispel the taboo around it.

INTRODUCTION

Around 280 million people in the world have depression [1], out of which 57 million people live in India [2]. It can lead to suicide, which is the fourth leading cause of death in the 15 - 29 age group [3]. When the word 'depression' comes up, it raises eyebrows, but people take it with a pinch of salt, much like any other mental health disorder. People with mental health issues are told to "move on" by labeling their condition as "Just a phase". The situation is worse among the youth of India. One in seven 15 to 24-year-olds in India reported often feeling depressed or having little interest in doing things [4]. Kids and teenagers find it difficult to understand their emotions. They're said to be 'hormonal', and any possibility of them being depressed is quickly swept under the carpet. However, depression must be treated with the same level of seriousness and attention as any other form of illness or disease.

That is how we see depression - as a disorder [5]. But two scientists want us to look at it differently. They suggest that it is an adaptation, as opposed to a malfunction [6]. But first, let's look into the inner workings of depression.

NEUROBIOLOGY OF DEPRESSION

Mental illnesses like depression do not have a single cause. But, changes in certain structures of the brain have been observed in people with depression. Some areas, like the ventromedial prefrontal cortex (VMPFC), show increased activity. While others, like the dorsolateral prefrontal cortex (DLPFC), show decreased activity.

Regions of the brain, like the amygdala, hippocampus, and prefrontal cortex, control important functions like memory, attention, and mood regulation. Malfunctioning in these regions could lead to depression. In the prefrontal cortex, the VMPFC shows enhanced activity. It causes anxiety and rumination. The DLPFC shows reduced activity. This is associated with apathy and attention deficit. Both these changes are important in the evolution of depression. Regional blood flow studies were able to detect these changes. Increased blood flow to areas of higher activity, while reduced flow to areas of lower activity allowed for the characterization of changes in specific structures.

Interfering with normal levels of neurotransmitters is another characteristic process encoding depression. For example, serotonin, a neurotransmitter often called "the happy hormone" - might be found at lower levels in people with depression. Monoamine oxidase A (MOA) is an enzyme that degrades or catabolizes serotonin, causing serotonin levels to drop. Some antidepressants work by preventing the brain's natural serotonin degrading enzymes [7].

DEPRESSION AND EVOLUTION

Mental health disorders might have served some purpose in the distant past. The two most important tasks for early humans were survival and reproduction. The pressures attached to these tasks were high. Food wasn't available at a moment's notice [8]. Anxiety response saved early humans from predators. It helped them recognize danger and ignite their fight-or-flight response [9]. Depression might have helped them conserve their energy for hunting by making them sluggish and unmotivated while performing other, less essential tasks. But today, our environment is much safer, and food is readily available [8]. So, are mental health disorders an evolutionary mismatch? Or have they stuck around with a purpose? Could depression then be an adaptation?

The most popular idea regarding this is called the analytical rumination hypothesis. It was put forward by Paul W. Andrews and J. Anderson Thomson, Jr. in 2009. They proposed that depression is an adaptation that is an evolved response to complex problems. Does it help in actually solving these problems? Symptoms of depression are indicative of uninterrupted analysis by reducing the urge to engage in other activities and redirecting resources towards 'ruminating', i.e. thinking about the problem in depth. This could minimize disruption through slow, sustained processing and could be evidence of its usefulness in analyzing complex problems. The authors say that these complex problems are what triggers depression. In some of their research, they found that people who got depressed while working on complex problems in an intelligence test tend to have scored higher on it [10]. A book called *Productive and unproductive depression: Success or failure of a vital process*, published in 1989, supports this claim. It also warns against depression turning unproductive if therapies aim toward ceasur of the problem instead of encouraging rumination to solve it [11]. Some studies have found that expressive writing related to these problems leads to quicker resolution of depression because people gain insight into their problems [12-13]. People in a depressed mood have been found to be better at navigating social difficulties [14-16]. Although, another body of research shows that depression reduces the accuracy in tasks that require memory and intelligence [17].

Another reason why depression has stayed with humans could be some of the genes which are associated with depression as well as immunity [18]. Research conducted on a molecule called the 5-HT_{1A} receptor present in the brain gives evidence as to why depression might be an adaptation. This receptor binds to serotonin, a brain molecule we talked about earlier in the article. In an experiment conducted on mutant mice, which lacked the 5-HT_{1A} receptor, scientists observed fewer symptoms of depression in response to stress [19]. Scientists compared the functional part of this receptor in rats and humans. They found the composition to be 99% similar. This suggests that natural selection has preserved it. Hence it might have been important [20].

Depression causes changes in the body to help people analyze their problems without falling for distractions because analysis requires uninterrupted thought. A region in the brain, called the ventrolateral prefrontal cortex (VLPFC), can keep us from being distracted. It can perform this function only if the associated neurons fire continuously. But this demands a lot of energy and can cause the neurons to break down. Studies in rats show that the 5-HT_{1A} receptor feeds the neurons with the fuel they need to fire and prevents them from breaking down. This might explain why this receptor is evolutionarily important [6].

One in seven or ten mothers around the globe experience postpartum depression i.e. depression observed in a mother within 4 weeks of the birth of a child [21-22]. In India, around 1 in 5 new mothers suffer from it [23]. Few findings tell us that this is because it might have helped early women stay home to protect their babies from danger and bond better with their babies and their partner [24].

DEPRESSION: A NON-ADAPTATIONIST VIEW

There is a less strongly held counterview that depression may not always be adaptive. It sometimes accompanies other mental illnesses like anxiety. As anxiety itself is said to be evolutionarily adaptive, it has prevailed, and so has its by-product - depression [25].

Also, severe depression leading to suicide would not have served as an advantage to our ancestors (Although a small body of research suggests otherwise [26]). Depression in modern times causes people to avoid social interactions and bonding with others. So, it might not have helped early humans to stay home and bond with their families [8].

ANTIDEPRESSANTS AND THE PRINCIPLE OF EVOLUTIONARY MEDICINE

Antidepressants are widely used. But they bring about undesirable changes in the brain. They hamper serotonin homeostasis, which plays an adaptive role. The principle of evolutionary medicine states that 'disruption of adaptive processes will degrade biological functioning.' Since homeostasis is adaptationist, prolonged use of antidepressants could change brain chemistry. Serotonin plays a vital role in making new neurons and sometimes killing older ones. Considering how important this neurotransmitter is in the brain, antidepressants may not be all that beneficial [27]. Antidepressants also prevent the process of rumination. This prevention decreases symptoms in the short term, but it may increase them in the long run. Therapists have, in fact, suggested that depressed individuals should be encouraged to 'ruminate' through integrative therapies. Writing is an effective way to spur on these ruminative thoughts, which helps in positive growth of individuals involved in processing [28].

WHY THE PREVALENCE NOW?

Depression isn't something new. Even ancient humans experienced it. The Greeks even had a name for it - 'Melancholia'. It has persisted for a long time. But why have cases of depression increased recently? The answer might be our changing lifestyles. We often turn a blind eye to the effects of our lifestyle and environment on our mental health.

DEPRESSION AND GUT

Changing food habits like eating more junk food affects our brains in ways that are not fully understood. Research has shown that consumption of junk food alters the natural human microbiome of the gut and increases inflammation. The gut microbiome is involved in the production of important neurotransmitters like serotonin and GABA (γ -Aminobutyric acid). Changing the composition of the gut microbiome affects the production of these transmitters. Junk food induces a change in the microbes in our stomach. This can elevate symptoms of depression by affecting the vagus nerve, which regulates digestion [29].

DEPRESSION AND AIR POLLUTION

There is also a connection between air pollution and mental health. Scientists from Stanford University and the University of Denver conducted a survey in the San Francisco Bay Area. They observed, in the case of children who spend significant time outdoors, the rising levels of air pollution led to depression. Ozone, a major contributor to air pollution, disrupts

neuroimmune processes by increasing inflammation. There are many proteins and cells involved here, of which one is the C-protein. The C-protein is over-expressed in individuals exposed to polluted air. It is an inflammatory protein whose production increases in the process of inflammation. C-protein, when overexpressed, creates a cycle by recruiting even more inflammatory proteins [30].

CONCLUSION

In our brief exploration of depression, we did not get a conclusive answer to whether it is an adaptation or a disorder, or something else altogether. Various theories have been put forward to explain it, all of them with their own line of evidence. Some of them are contradictory as well. Regardless, there is merit in looking at depression from an evolutionary perspective. It helps us understand it better. Subsequently, it helps us develop better strategies to mitigate it and help the people suffering from it, not only by alleviating their symptoms in the short term, but by ensuring more robust mental health in the future.

Peer pressure, parental expectations, and exam stress play a role in harming a student's mental health. People who are disproportionately disadvantaged by their religious, caste, gender, or sexual identities are at higher risk of depression [31-32].

If you are suffering from depression or other mental health issues, seek professional medical help immediately. Also, encourage others who're suffering, to do the same. Here are some helpful resources. Ignoring those deep wounds and scars will only worsen the pain. Timely help can mean a difference between life and death.

For immediate help, contact 1800-599-0019 (Kiran helpline: a national 24/7 toll-free helpline).

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Inside a hallucinating brain...

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SUMMARY

Hallucinating is the act of seeing, hearing, smelling, tasting, or sensing something when it does not exist. Genetics, lack of sensory inputs, continuous exposure to sensory inputs for a prolonged time, and several other factors account for hallucinations. This article peeks into hallucinating brains and provides insight into how brains fabricate such unreal experiences.



Figure 1. A depiction of hallucination.

Image credits: https://commons.wikimedia.org/wiki/File:ALUCINACIONES_DEL_QUIJOTE.jpg

INTRODUCTION

Hallucinations are perceptions of something that does not exist in reality (Fig. 1). It is a subjective experience that can change based on the history of a person. Hallucinations can involve any sensory modalities known as auditory, visual, tactile, olfactory, and gustatory hallucinations, respectively.

THE SAVIOUR: AN EPISODE OF HALLUCINATION IN A SCHIZOPHRENIC PATIENT

Slowly, he walked along the road, and there was smoke everywhere, yet he continued along the way as he knew that reaching there was vital to save the city. But just as he opened the door, a gun was pointed at him; before he could react, the wall started to scream in unison. He turned around but realized there was another door. Before he could reach there, he saw

a woman running at him from the side. A splash of blood hit his coat, and he looked around. It occurred to him that there was fire all around him. In his ear came the command, "run, run, fire can't hurt you, you are our saviour, you have to come to us." Just as he sat on the bench under a tree, the women started to describe his mission. He was chosen to be part of that covert team and was the one to save the city from an alien invasion. Or that's how he described how he broke his bones and charred his body to the doctor after the incident.

ANOTHER EPISODE: A MOUNTAINEER'S DARK-COLD HALLUCINATION

As he was treading his path towards the summit of Mount Everest (ME), the wind was swirling all around his body, and it seemed as if several devils were screaming and freely moving all over the open space surrounding the snow-covered peak. "You can, and you will reach the summit," he thought to himself with great determination.

It was his 5th sleep-deprived night since he and his rope team members had started from the base camp. Though he had undergone an acclimatization session, which lasted for about three days, it was the first time for him to move past 9000 ft. Also, sleep deprivation was an adversity to which he was being exposed for the first time. The total height of ME is 29,000 ft.

Though it was very hard for him to keep his eyes wide open, he tried his best. Suddenly, he started seeing things which were not supposed to be there. He saw coloured dots, squares and circles, all forming different weird patterns before his eyes and had started spreading all over the space around him that his vision could cover. When he rubbed his eyes, there was a mild escape from these patterns but the relief did not last long. They started appearing again and again before his eyes. As he managed to continue moving forward ignoring these patterns, the visual disturbance grew bigger and bigger. It had then started forming thick clouds of opacity all around him, clouding his actual vision.

This mountaineer is hallucinating. Our brain is designed in such a way that when any of our senses are deprived of inputs for a long period of time, they start generating them on their own, thus leading to hallucinations.

HALLUCINATIONS - CLASSIFICATION

Hallucinations can be broadly categorized into two groups based on the causative factor and recurrence:

- The first category comprises all hallucinations that happen due to inherent causes or those that occur due to an underlying brain disorder. Examples for this category would include hallucinations caused due to Schizophrenia [1], Parkinson's disease [2], Alzheimer's disease, Encephalitis, Bipolar disorder, etc. The episode of savior we saw earlier was based on hallucinations in such conditions— you might have seen something similar in the movie: "A Beautiful Mind."
- The second category would include those that happen during several adverse conditions. Like sleep deprivation, monotonous exposure to sensory input (for example, exposure to a particular sound or visual scene for a prolonged period) [3]. It also includes lack of oxygen, self-inflicted alcohol, hallucinogen (Eg: LSD, psilocybin, ketamine, etc.) uptake, etc. Hallucination experienced by the mountaineer was due to sleep deprivation and sensory monotony. Sailors and polar explorers generally experience such hallucinations.

So, what exactly happens within the brains of those experiencing these hallucinations? What is precisely behind the screen and is responsible for these anonymous stimulations? This article sheds some light on this grey area.

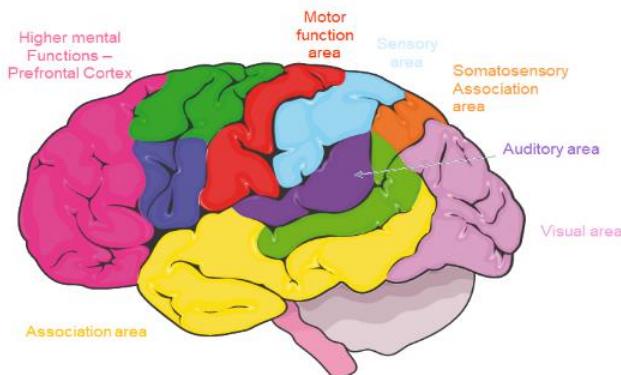


Figure 2.

Parts of the figure were drawn using pictures from Servier Medical Art. Servier Medical Art by Servier is licensed under a Creative Commons Attribution 3.0 Unported Licence (<https://creativecommons.org/licenses/by/3.0/>).

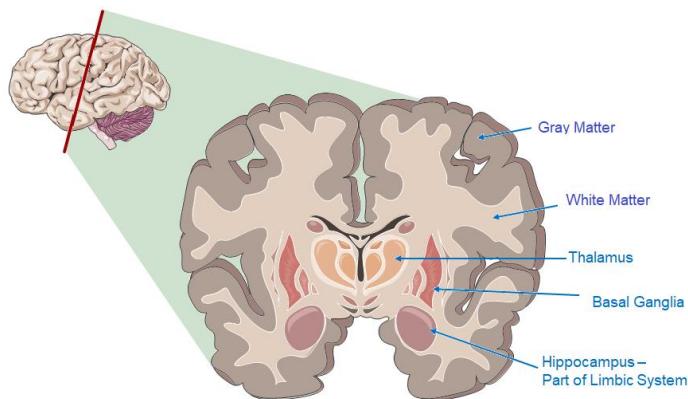


Figure 3. Longitudinal section of the brain.

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NEUROBIOLOGY OF HALLUCINATIONS

"How the machinery of the brain could generate the theatre of the mind."

- Charles Bonnet (1720-1793)

The saviour's hallucination

Let's start understanding several hypotheses about hallucinations that come under category 1 by taking a close look at the structures of our brain (Fig. 2).

The nerve carries signals from the different sensory organs like eyes, ears, and skin to the primary area in the brain (a sound of some frequency, for instance). They are then passed on to association areas to make out the meaning from the sensation (ah! this is the sound of a Cuckoo singing). These areas have connections from the limbic system, which then associates these perceptions with emotions and past experiences (feelings of pleasure, maybe even someone's image).

So, how does this well-structured system of perception go into chaos?

There is no single definite answer yet, but there are various hypotheses put forward by researchers [4] and some interplay of them lie at the heart of the generation of such chaos.

NEUROPHYSIOLOGICAL THEORY

As we understood through the illustrations, different areas in the brain are assigned specific functions. The "Thalamus" works as a gateway for multiple sensory inputs in the brain. It has connections with different parts of the brain. These connections are also thought to generate an internal representation of reality for the brain. Then there is the limbic system, which has widespread connections with the sensory association areas, like the auditory association area.

It has been found that in patients with Schizophrenia, there is increased blood flow in these areas and thus postulated to be hyperactive in these patients, which can lead to an altered perception of reality and association of previous experiences with it [5].

Another area, the prefrontal cortex, is a part of the brain located in the forebrain in the front. It is one of the most vital areas for critical reasoning, logic, and perception control. Several studies which made use of MRI & PET scans (a type of imaging technique that can help in understanding the metabolic activities of tissue) have found that in patients with Schizophrenia, there is an alteration in the connections between the neurons and reduced functional activity [5].

As illustrated in the diagram (Fig. 4), neurons have two parts: dendrites and axons. These help the neurons communicate with other neurons. In patients with Schizophrenia, it has been found that neurons present in some layers of the prefrontal cortex of the brain have reduced amounts of dendrites, leading to reduced neuronal connections.

How does that affect perception? The speculation is that the reduced connections lead to decreased conscious control over sensory perception and processing, leading to positive symptoms (positive symptoms reflect distortion or excess of normal function) like hallucinations.

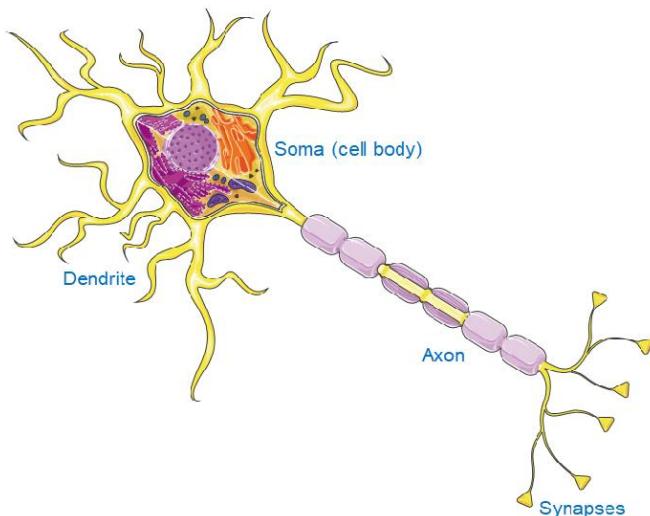


Figure 4. Image of a neuron.

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NEUROTRANSMITTER THEORY

Before we dive in, it is essential to understand what a neurotransmitter is. Let us look at how two neurons interact with each other.

Unlike what you might have thought, two neurons are not always physically connected. There are electrical synapses where neurons are physically connected through gap junctions, so there is a physical gap, albeit very small, between them. If so, then how do they communicate?

Neurons process a message through electrical currents, and when this current passing through the neuronal cell reaches the end of it, it releases a chemical from the cell into that gap.

This chemical passes through the "synapse" gap to reach the second neuron (Fig. 5). On the surface of the other neuron lies the receptors for the neurotransmitters.

Some chemicals function as neurotransmitters like Dopamine, Acetylcholine, Glutamate, etc., in different parts of the brain. For every neurotransmitter, there can be many receptors.

Sensory signals are transmitted from one area of the brain to another to process the information. Neurotransmitters are required to transmit these signals.

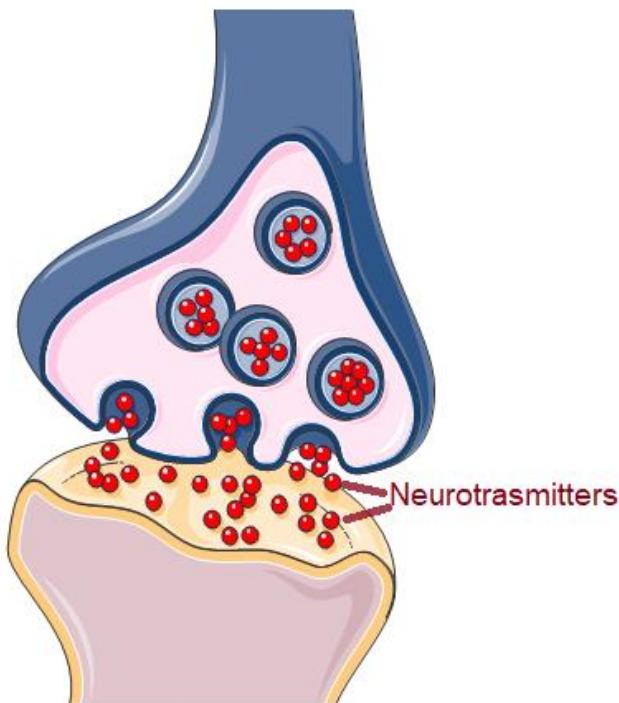


Figure 5. A synapse.

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DOPAMINE THEORY

Studies of the brain have identified that there is an increased activity of dopamine in the brain in patients with Schizophrenia. This has been supported by the fact that many antipsychotic drugs block dopamine receptors in the brain.

Due to this increased transmission of dopamine, there is dysregulation of various pathways in the brain. This also includes the connection between the thalamus and sensory association areas as well as between the thalamus and prefrontal cortex of the brain. *This leads to the activation of these neuronal circuits and eventually leads to abnormal behaviors and experiences in patients with schizophrenia [4].*

Other neurotransmitters like glutamate, acetylcholine, and serotonin are also thought to be playing a role in these diseases.

There is no single answer yet to why these alterations may occur, but studies have revealed that several genes play a role. In addition, recent studies also point to defects during the development of the brain in childhood and adolescence. Numerous environmental factors and drugs (like LSD, Cocaine in susceptible persons) might also play a role in it.

THE MOUNTAINEER'S HALLUCINATION

With the advent of functional brain imaging in 1990, the greatest difficulty to see through the brain and to get to know what is happening exactly while it is hallucinating has been overcome.

Researchers have found that during visual hallucinations, there is an increase in excitability of the occipital cortex and inferotemporal cortex of the visual cortex (VC). The main function of the VC is to process the visual information from the eyes. The information would then be sent to other parts of the brain to make and execute decisions.

Studies have also found that the hallucinations are completely different from self-crafted imaginations, as the prefrontal cortex (PFC), is active during such voluntary visual imagery. The PFC is involved in higher cognitive functions, and it is the region that differentiates humans from the rest of the animal kingdom. So, from this, it can clearly be seen that the VC is getting activated in the absence of an external stimulus and the rest of the brain believes it to be a true stimulus. During imagination, the PFC is getting activated.

Also, in animal models, it has been found that by inducing moderate hypoxic (lower oxygen levels, less than 92%) conditions (12.5% of oxygen saturation level has been used in the study) [6], the levels of the neurotransmitters serotonin (the feel-good hormone) and dopamine (happy hormone), are increased in the extracellular fluid in the striatum. The levels of both these neurotransmitters are also found to be elevated in the cases of hallucinations coming under the first category.

The other NTs involved with sleep deprivation and hallucinations are acetylcholine (ACh) and norepinephrine (fight or flight hormone) (NE). The lower cortical ACh levels and higher NE levels are shown to produce hallucinations under sleep-deprived conditions [7].

A RAY OF HOPE

Though the second category of hallucinations can be reverted by the removal of the adverse conditions causing the hallucinations, the first category is mentally traumatic. Though this condition can be managed with the aid of the prevailing medications, there isn't a definite cure for the voices within the heads of the patients experiencing hallucinations.

But that is not the end of the story, life is a challenge and the great personalities who are leading their lives successfully, have shown the world that there is nothing that is impossible to be achieved by the power of the human will. Some of these personalities are John Forbes Nash Jr. (1994 Nobel laureate, Economics), Elyn Saks (Professor, University of Southern California Gould Law School), Oliver Wolf Sacks (Neurologist), and the list goes on. These people have shown that they can lead successful lives forbearing the traumatic conditions that try to pull them down from behind.

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Attention at gunpoint: What triggers ADHD?

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SUMMARY

ADHD is a mental disorder that affects concentration, increases hyperactivity and impulsive behaviour enough to disrupt one's day-to-day activities. The subtle nature of its symptoms downplays its seriousness. More than adults, ADHD affects children more. Girls tend to be underdiagnosed or misdiagnosed with anxiety or depression. But what causes this problem? It is a neuroscientific mystery. We'll walk you through what research tells us causes ADHD.

INTRODUCTION

Attention—without it, I could not have put these words on paper; you could not have read them. From the simplest tasks like counting to complex ones like organising a party, you NEED attention – focus - concentration. We don't think much of it, and since it comes naturally to most of us, we expect it from everyone around us — family, friends, co-workers, partners, and kids!

Ever wondered what happens to those people who are unable to give attention to everyday activities? Can you imagine not being able to stay attentive to even a single task? The truth is that some people face a difficult time devoting their attention to one task. Either they are distracted by their surroundings or by their own thoughts, and understandably, most are children.

While, clinically, this phenomenon is called attention deficit (lack of attention), some people with deficient attention are also hyperactive—they cannot sit in one place, cannot bear silence, and are always on the move. Doctors have termed this condition "attention deficit hyperactivity disorder (ADHD)". Globally, around 2.6% of children and 6.8% of adults are impacted by ADHD [1]. The prime symptom is inattentiveness, which may be accompanied by one or more of the following: oversensitivity, hyperactivity, or impulsiveness.

Though not lethal, ADHD causes significant problems in the lives of those it affects. While the majority of ADHD-affected children struggle with homework and at school, ADHD-affected adults have a hard time following instructions at work. The volatile nature associated with this disorder could also lead to risky behaviour occasionally. Socially too, people affected by ADHD are less understood, sometimes bullied and discriminated against, impacting their self-esteem, morale, and overall quality of life.

As if this wasn't enough, the symptoms of ADHD are subtle and overlap with what is perceived as ordinary laziness or naughtiness. Hence, many people do not accept ADHD as a legitimate disorder of the mind. They think that it is simply a tag that parents use to cover-up the 'difficult kids' problems (Fig. 1).



Figure 1. The labels attached to ADHD-affected kids affects their self-esteem.

Image Credit: Dr. Jonathan B. Lauter via Flickr

WHAT CAUSES ADHD?

To date, we do not completely understand what causes ADHD. Scientists and doctors continue to research. So far, it seems that many factors exert varying effects to bring about ADHD in an individual. Some of the main factors behind ADHD are described below.

Heredity

In the face of a new disorder, the first thing scientists and doctors check, is if it runs in families (i.e., if it is *genetic*). Do the siblings of the affected have the disorder? Do twins have the exact same disorder? Do the parents or relatives of the parents have the disorder? In this way, the disorder is tracked within the family tree. Yes, ADHD does show patterns of inheritance in families [2]. But it is not caused by a single, specific, well-defined change in the DNA of the ADHD-affected. Rather, many small and subtle variations in many genes seem to be involved. Importantly, though, while these changes do create a genetic *predisposition* for the disorder, this implies that such variations do not necessarily lead to the development of ADHD. Rather, these variations increase the likelihood of developing ADHD at some point in life. This is unlike genetic disorders such as cystic fibrosis or thalassemia, where the mutation (an abnormal change in DNA) definitely leads to disease.

Brain abnormalities

Brain structure and function were compared between ADHD-affected and healthy individuals in order to better understand the effects of this disorder.

Structure: Clearly, an ADHD-affected child's brain is overall smaller in size than a non-ADHD kid's brain by 0.78%. Further, the five parts of the brain that were grossly shrunk in ADHD brains included regions involved in learning, memory, processing of emotions, rewards, and motivation [3].

Brain region	Reduction in Volume (percentage)	Broad function of the region
Hippocampus	0.38%	Learning, memory
Amygdala	1.50%	Emotions, memory
Accumbens	2.55%	Reward/Reinforcement
Caudate	0.93%	Motor skill/Goal-directed behaviour
Putamen	1.22%	Movement, Motivation
Total Brain	0.78%	--

Table 1. The ENIGMA analytical study found a decrease in the size of some important brain regions as also intra-cranial volume (total volume of the brain) in ADHD individuals compared to non-ADHD. The reduction has been depicted in percentage here, based on table no. of the research article

Interestingly, these differences decrease in adolescents and vanish in adults. Yet, over half of ADHD-affected children carry symptoms into adulthood. While some believe this means ADHD is only a developmental delay, i.e., a child's brain develops slowly as compared to a normal one, it may also be that an ADHD brain works differently than a non-ADHD brain.

Signalling: Neurons (brain cells) connect with each other to relay information by means of molecules or chemicals called *neurotransmitters*. They are released by one neuron and taken up by the next (so on and so forth) as a form of messaging. Each neurotransmitter has a unique effect, initiating a specific response in the recipient neuron. This neuron requires *receptors* on its surface to be able to 'catch' the neurotransmitter. Also, the source neuron has *transporters* for the same neurotransmitter on its surface that will suck the unused neurotransmitter back into the source for recycling (Fig. 2).

Evidently, neurotransmitters delicately balance the stimulatory and inhibitory signals for proper brain activity. Dopamine, norepinephrine, and serotonin are three such neurotransmitters. It has been discovered that the respective levels of these neurotransmitters and the presence of their receptors and transporters are altered in ADHD brains. For example, impulsive behavior—a symptom of hyperactive ADHD—is hindered by serotonin but encouraged by dopamine [4]. It is the ratio between these two that determines if a person acts on impulse [5]. The levels of transporters of both norepinephrine [7] and dopamine [6] are low in ADHD people. Serotonin signalling is disrupted [8] by low blood serotonin levels, reduced binding on serotonin receptors and also serotonin transporters.

The full picture is not yet clear, but there is an emerging understanding that ADHD may result from abnormal neurotransmitter signalling in affected brains. Obviously, lack of regulation in neurotransmitter signalling leads to abnormal brain activity, which shows up in diagnostic imaging – a technique used to compare ADHD brains with normal brains. Even during a resting state, people with ADHD show increased brain activity [9].

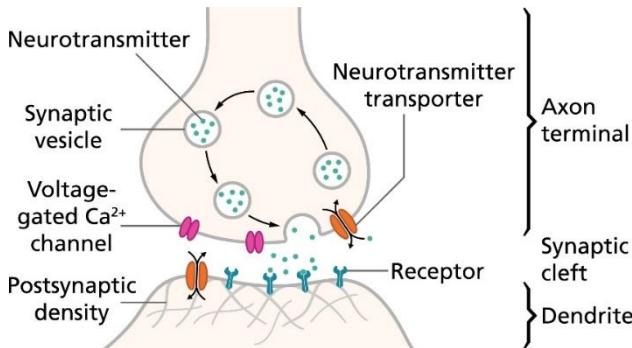


Figure 2. Synapse – where neurons connect and neurotransmitters are released.

Image Credit: <https://qbi.uq.edu.au/brain-basics/brain/brain-physiology/action-potentials-and-synapses>

Neurotransmitter	Effects on Body & Behaviour	Status in ADHD brains
Dopamine	Reward and movement regulation	Transporters - Low
Norepinephrine	Increases alertness, arousal and attention (act on motivation/mobilize)	Transporters - Low
Serotonin	Digestion, regulation of blood flow and body temperature, breathing, blood clotting and wound healing, affects mood & sleep	Blood level - Low Receptors - Low binding Transporters - Low

Table 2. Neurotransmitters are chemicals that relay messages in the brain. There are more than a hundred of them, produced by brain cells and certain other cells. The brain works well in a delicate balance of all of these signals. Lower or higher amounts of neurotransmitters can have undesirable effects.

The variations uncovered in genetic studies also fall in genes that affect the activities of these neurotransmitters, i.e., norepinephrine, dopamine, and serotonin, confirming that ADHD has solid grounds in neurobiology and genetics.

Environment

Some doctors and researchers argue [10] that the surroundings of a child with a genetic propensity to suffer from ADHD may have more to do with the disorder arising in them. By now, you have probably understood that there isn't a particular gene responsible for ADHD. Even if it's genetic, it's a complex disorder, with small variations combining to have a

big impact on an individual's brain function. Over and above that, among twins, not all twin siblings of ADHD-affected kids develop ADHD themselves (though a majority of them do). These facts are indicative of a component other than genetics playing a role in the manifestation of ADHD.

As humans, we thrive on connection; intimate family bonding and social interactions are necessary for our overall wellbeing. If we are deprived of it, trauma occurs. Many practitioners believe that it is childhood trauma [11] – in the earliest years after birth, the years we rarely have any memory of, but also when the brain development is at its peak-precipitating into disorders such as ADHD*.

*In this context, behavioural psychology studies do conclude that the unusual behavioural traits observed in ADHD are simply a mechanism of the brain to cope with the abnormal signalling. An ADHD brain may compensate for a lack of vigilance or attention with the pursuit of hyper-stimulation (hyperactivity) [15].

The first and foremost form of trauma during the developmental phase of the brain can happen in the womb. It is well-known that pregnant women must take utmost care of their physical and emotional health because what they go through, the baby goes through. Premature birth, low birth weight, and other negative new-born parameters are associated with maternal illness [12]. In fact, maternal health strongly impacts children's probability of developing ADHD too [13]. Scientists are still trying to figure out how this happens [14]. But the effect cannot be denied.

Once the child is out in the world, they are exposed to more toxins. Plastics (BPA & BPS) [16] are used in food containers, milk and water feeding bottles, even their toys contain chemicals. Another class of toxins, phthalates [17], makes its way to them through rubber toys, raincoats, shampoos, and medical devices. All of the materials used to make non-stick cooking pans and the rubber used to line toys are harmful to children's health [18]. A specific threat is learning disability, of which ADHD is just an example [19].

The nitty-gritties of modern family life also have some role to play in the shortening of attention spans, furthering the genetic predisposition to disorders such as ADHD. Excessive TV viewing has already been shown to increase the risk of attention deficit. If further investigated, it may be discovered that other similar stimuli—unrestricted mobile phone use, addictive video games—all of which promote "instant gratification" may be linked to an inability to concentrate [20].

SO, WHAT CAN BE DONE?

A growing body of research suggests that getting the neurotransmitter signalling back on track can help ADHD-affected individuals decrease the severity of their symptoms. Medication [21] (stimulants like methylphenidate, amphetamine, or antidepressants and SSRIs), nutritional supplementation [22] (vitamin D, omega-3 fatty acids), or even mindfulness meditation and yoga [23] and other forms of exercise can help with this.

Any activity that trains the brain for eye-motor coordination, decision-making, or short-term memory retention is beneficial for an ADHD individual. Even if it means playing video games (of course in moderation and under supervision)! Classic games that engage more than one

mental faculty, such as marbles, Pictionary, or card games, are a sure-fire way to improve brain function in both children and adults [24].

It points to the fact that despite being rooted in genetics and neurobiology, ADHD comes to the surface upon adverse gene-environment interactions (Fig. 3). The effect of our environment on us is starkly visible here. But with empathy and awareness, nurturing ADHD-affected children can reduce symptoms and improve lives.

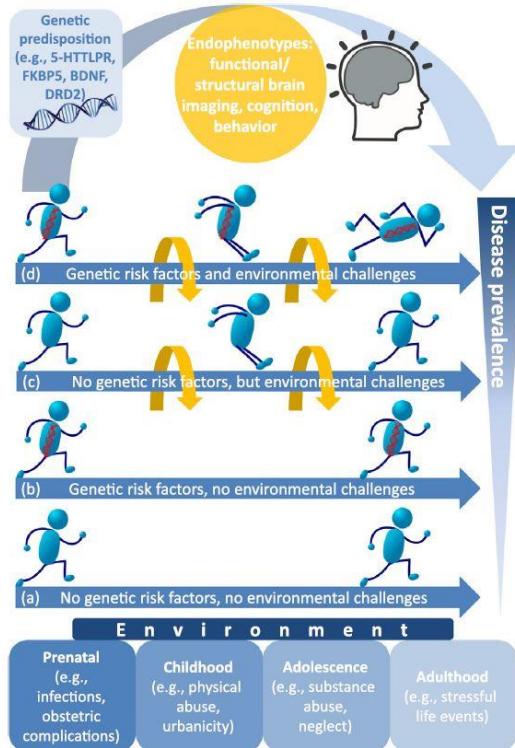


Figure 3. Gene-environment interactions drive ADHD pathology [25].

Image Credit: Grabe *et al* (2020)

TURNING THE TABLES ON ADHD

Our brains are plastic, i.e., they respond to training, experience, and environmental changes to develop new circuits and habits. This is a source of great hope for those affected by ADHD and other learning disorders. As you may have seen in the popular Bollywood film *Taare*

Zameen Par (which featured a dyslexic character), the surroundings of a differently-abled person may be able to do more than we currently realize.

Being aware of ADHD and treating ADHD-affected individuals with empathy and compassion is the least we can do as a community. A small act goes a long way when it is gene-environment interaction at stake.

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Euphoria: The Neuroscience of Addiction

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SUMMARY

Popular media is rife with the depiction of substance abuse which may or may not portray the entire picture. Teenagers are particularly vulnerable to the public health problem of addiction. This article examines the neurobiology of the reward system and how substances might co-opt this and impact neurotransmission. The topics covered include the risk factors for addiction, the long-term consequences, the difficulty of overcoming addiction, and the therapeutic modalities available to achieve this.

INTRODUCTION

The Indian movie theater experience is incomplete without watching trailers of new movies and advertisements for the newest products that are played in the beginning. In the midst of these exciting clips, a grim reminder of the detriments of alcohol or tobacco consumption is also flashed on the screen. These videos often feature patients who've developed cancer after years of consumption.

However, this highlights only the tipping point of the continuum of substance use. How does one progress from using substances occasionally to the point of no return? Owing to the robust tobacco cessation program in India, we know that "smoking kills," but why does one continue to smoke despite this knowledge? What makes it difficult for a person to quit? To answer these questions, we must delve deep into the addicted brain.

WHAT IS ADDICTION?

At its core, addiction is a chronic condition characterized by repeated substance use or repeated behavior engagement despite the negative repercussions. However, due to the interplay of various factors and a plethora of substances that can be abused, addiction is a nuanced disorder to diagnose. The Diagnostic and Statistical Manual of Mental Disorders (DSM-5), which is the gold standard tool for diagnosing mental disorders and illnesses, attempts to break down this complexity by defining certain criteria to diagnose addiction to substances which are termed Substance Use Disorders (SUD) [1,2]. These criteria can be grouped into four categories that form the core of SUD:

- Impaired Control: Inability to limit the amount and/or frequency of use despite wanting to
- Social Problems: Neglecting responsibilities and relationships
- Risky Use: Continued use despite problems
- Physical Dependence: Withdrawal symptoms and tolerance which drives consumption of more substance every time

A LOOK AT ADDICTION IN THE BRAIN

Studies have shown that a vicious cycle consisting of three main stages perpetuates addiction [3,4,5]. These stages are:

- Binge/intoxication stage: Consumption of the substance results in the experience of pleasure
- Withdrawal/Negative affect stage: In the absence of the substance, unpleasant symptoms are experienced
- Preoccupation/Anticipation stage: In order to obtain the pleasurable experience and/or allay the negative one, the user seeks the substance again

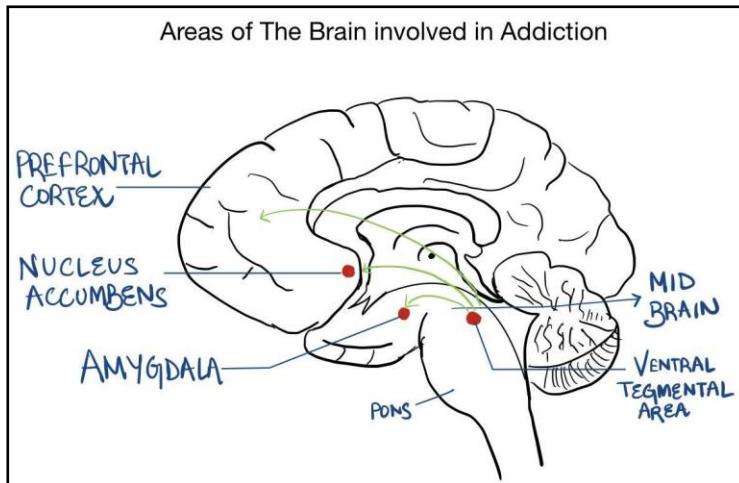


Figure 1. An illustration of the anatomical parts of the brain involved in addiction. Dopamine from Ventral Tegmental Area acts on different parts, as elaborated in Figure 2.

At the crux of the binge/intoxication stage is the reward circuit of the brain, which is regulated by the neurotransmitter dopamine. Substances trigger the release of dopamine from the ventral tegmental area (VTA) in the midbrain, which on binding to receptors in the nucleus accumbens of the basal ganglia, produces a feeling of pleasure known as the "high." This usually happens with other activities, such as eating your favorite food as well but with drugs, the intensity with which the reward system is stimulated is so much that the stimuli associated with the substance themselves activate the reward system resulting in a craving for the substance. This is known as incentive salience.

A study was conducted on animal models wherein a neutral stimulus, such as a sound or light which by itself would not elicit a response, was repeatedly introduced with a drug. After some time, the animal started exhibiting drug-seeking behavior with only the neutral stimulus. Thus demonstrating incentive salience.

Once the effect of the substance wears off and further substance is not consumed, people experience negative physical symptoms and a negative emotional state which is known as withdrawal. The intensity and the duration of symptoms may vary depending on the substance, but the changes in the brain during the withdrawal stage are more or less the same:

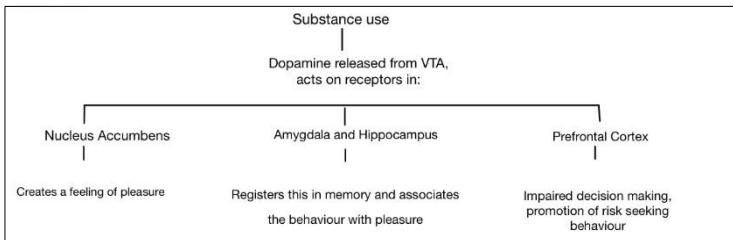


Figure 2. A flow chart explaining the effect of dopamine on different parts of the brain.

When the brain reward circuit is activated, another neurotransmitter, dynorphin, which is an opioid peptide, is also released and acts on the nucleus accumbens. This increase in dynorphin decreases dopamine activity, thereby disabling the reward circuit. Dynorphin also causes activation of the stress response in the amygdala which causes physical symptoms like tremors, sweating, tiredness, headache etc.

During withdrawal, dynorphin is found to increase manifold. The inhibition of the reward circuit during withdrawal is to such an extent that it is not activated by natural stimuli that would otherwise allow the person to experience pleasure. This is the basis for the negative emotional symptoms during withdrawal.

Further, MRI studies of the brains of addicts have shown a decrease in the number of dopamine receptors in their brains over time. Thus, the threshold for the reward circuit activation increases with continued substance use, each time requiring more of the substance to produce the same response. This is known as tolerance.

The prefrontal cortex of the brain is the region that controls decision-making. It can be conceptualized to have a Go system: that gives the go signal for action and is also involved in forming a habit, and a Stop system: which inhibits the Go system. In addicts, the Go system is overactivated while the Stop system is under-activated thereby impairing decision-making and promoting substance-seeking behaviour. This is known as the preoccupation/anticipation stage, and it closes the loop only for it to start again by making the person seek and consume the substance.

LONG-TERM EFFECTS OF DRUGS ON THE BRAIN

Stopping drug usage does not automatically restore normalcy to the brain. Some medications have hazardous side effects that can destroy neurons, and most of these cells are not replaced. And while alterations in neural connections in the brain are not always permanent, they might linger for months. According to some studies, the effects might remain for years. Long-term brain alterations might make it difficult for addicts to stay drug-free. They frequently suffer extreme cravings, which leads to relapse [6].

Different drugs have different kinds of long-term effects on the brain. While the modifications that lead to addiction are common, various classes of drugs have additional distinct impacts on the brain.

A person suffering from addiction frequently requires treatment—sometimes numerous attempts at treatment—to achieve long-term recovery. Treatment can assist people in learning to control their addiction and develop tools and skills to deal with cravings, triggers, and other issues with sobriety.

TREATMENT FOR ADDICTION

Drug addiction is a medical problem and, like other illnesses, has both physiological and psychological components. Therefore, it makes sense that drug addiction treatment should address the physiological and psychological aspects of the problem. Severe addictions may require hospitalization while the person goes through rehabilitation to ensure they don't injure themselves and help their body adjust to functioning without the drugs. This first phase, which separates the addict from the drug, is called "detoxification." It's when you're trying to flush all the toxins out of your body. Some medications are used at this stage to help with basic withdrawal symptoms such as vomiting, nausea, and pain. This is important, but sometimes strong addictions require strong medication to break the cycle of addiction, in addition to treating those symptoms.

Cognitive-behavioral therapy, or CBT, is a type of psychological treatment for drug addiction and has been used successfully in patients addicted to alcohol, marijuana, cocaine, methamphetamine, and nicotine. As the name suggests, CBT addresses addiction's cognitive and behavioral components. Patients learn to recognize problematic thoughts and develop more positive thoughts and coping behaviors. They also learn to anticipate challenging situations. Research shows that the skills people learn in CBT last after the therapy ends, which is critical. Another type of behavioural treatment is motivational interviewing, sometimes called motivational enhancement therapy [7]. This therapy involves working with the patient to find intrinsic motivation to change. It's considered a very focused, goal-directed type of therapy because it involves very few sessions with a therapist.

When discussing therapy, it's important to consider the idea of relapse when a recovering addict makes a mistake and goes back to frequent substance usage. Relapses depend more on the addictive potential of the drug used and the current environmental triggers than anything else. The more addictive substances there are, the more likely they are to relapse and encounter anything a recovering addict previously associated with their addiction. This is one of the reasons CBT can be helpful. It can teach people to anticipate and avoid situations that would lead to relapse.

CONCLUSION

The mainstream narrative of addiction has always been one of an individual "failure", but this is far from the truth. As we've tried to explore in this paper, addiction is a complex disorder that is a culmination of various factors both at the individual and societal levels. There is a need for addiction to be looked at from a lens of rehabilitation rather than criminalization/stigmatization. Addicts are patients and deserve access to the various treatment modalities as much as any other patient. Only through this approach would we be able to intervene before a stage reaches when the impact on health cannot be undone. Until then, we can only hope

this article has been one drop in the ocean of change, and we urge every reader to pause and think when they see the substance control advertisements in the theatre the next time.

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A man demonstrating the use of Brain-Computer Interface

PART V

Neuroscience and Technology

Neuroplasticity and its role in Phantom limb, Bionics, and Adaptations

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SUMMARY

Bionic limbs hold great promise to improve the lives of amputees who have lost their limbs. Prosthetic limbs have a long history of evolution, and bionic limbs are the latest milestone. However, the field of Bionic limb development is in its infancy. Our understanding of the ‘phantom limb’ phenomenon and the science of neuroplasticity has contributed to the progress of bionic limb development.

INTRODUCTION

Trapped in the middle of an enemy’s bomb blast,
The Corporal lay injured, covered in white hospital casts.
How could he now defend his beloved motherland,
sans an *entire leg* and a badly *severed hand*?
Banned from the front lines, forsaken to a wheelchair
The Corporal’s life suddenly became unfair.
Instead of pride for his valor and bravery,
He now received oodles of sympathy.
Despite a prosthetic hand that helped to a degree,
High-fiving a comrade remained a luxury;
When once he ran across battlefields effortlessly
He now sits with a fake leg, wallowing in misery.
While we prance and dance without any strain
Even motionless, our Soldier was writhing with “phantom pain”.
Whilst the privileged cherished all beautiful senses of life,
The Corporal was forced to embrace eternal strife.
His leg stump, a remnant of a man in his prime
How desperately he wished to turn back time!
*“If only there was a way to hide my disability
If only these artificial limbs could be real!
Then I could promptly discharge every duty,
with all my heart, vigor and zeal”*
Besieged with intense helplessness
He cried a silent prayer,
with hope that there might be a day when
even amputees can sense and feel again.

PROSTHETIC LIMBS, A FLASHBACK

Whether it is a soldier on the battlefield, an Olympic athlete competing on the track, or even a toddler at preschool building towers with blocks - our limbs have an indispensable role in everyday life. But what if someone loses a part or whole limb due to some unfortunate event? Is it “game over”?

If it were a pirate in a cartoon who just lost a limb, he would simply attach a hook for a hand or a wooden peg for a lost leg to perch upon the deck and sail the seas, just like Captain Hook from Peter Pan. In the real world, prosthetic limbs have a long history and evolution, from the ancient ones serving merely a cosmetic purpose to the modern-day devices that have improved functionality and ease of use for the amputees (like our soldier). Even as back as 950 BC, artificial toes made of wood and leather were used in Egypt [1]. A prosthetic leg called ‘Roman Capula Leg’ that could move at the hip joint based on leaning movements dates back to 300 BC Italy [2], which offered some functionality apart from cosmetic enhancement. Notably, in 1812 a prosthetic arm was developed that could be controlled by the opposite shoulder with connecting straps — somewhat similar to how brakes are controlled on a bike [3]. In more recent history, during the First World War, many soldiers lost their limbs, which led to the development of prostheses with brass and steel braces that served better functionality; these include below-elbow, below-wrist, and above/below-knee prostheses [4]. Advancements in joint technology and developments in joining the prostheses to the limb, such as the suction-attachment method and prostheses made of lighter material like aluminum or carbon fiber, further improved functionality and ease of use. These mechanical prostheses were powered by cables and harnesses attached to the individual. They rely on the overall body movements for the manual manipulation of the cables that control the prosthetic limb.

The next significant milestone in the evolution of prosthetic limbs is the Myoelectric limbs [5]; these are externally powered by a battery and electronic system to control the movement. Prosthetic sensors are attached to specific muscles of the user; when there is an intention to move, these sensors detect stimuli by sensing movements of the muscle. Here, the myoelectric signals are processed through microprocessors in the prosthetic limb and converted into a control command. This command then actuates the prosthetic hand movement. Various commands, such as grasping, clapping, rolling fingers, etc., are encoded and executed based on the type of muscle movement.

BIONIC LIMBS, THE PROMISE AHEAD

Despite the progress, these prosthetic limbs fall short of restoring the functionality of the natural limb that can send sensation signal to the brain and be directly controlled by signals from the brain! How wonderful will it be for those who have lost a limb (like our soldier in the poem) to regain a functional limb that can not only be controlled by signals from the brain but also a feedback ‘sensation’ from the artificial limb to the brain? Is it just science fiction or is this a realizable dream? Well, the field of bionics has a promising solution!

The word bionics is derived by combining the two words ‘Biology’ and ‘Electronics’; it is a fascinating area of research to develop devices and systems that function like living organisms. Bionic cochleas, bionic pacemakers, and bionic limbs (our current topic of interest) are some examples of bionics.

In order to appreciate the challenges involved in mimicking the natural limb in a bionic limb, it is imperative to first understand the complexity involved in how our nervous system integrates sensory inputs and brings about precise movements of our body parts.

DIGITAL SIGNAL PROCESSING

Consider the task of scratching our noses with our fingers when there is an itch. Initially, we receive the sensation of itch from the sensory nerve endings on the surface of the nose. A signal from the central nervous system proceeds to initiate the scratch. The elbow needs to be bent while raising the forearm so that it is in the optimum position close to the nose. Then, the forearm needs to be rotated to the required angle so that the finger can reach the nose. Next, the finger needs to be extended and moved up and down repeatedly on the itch. All of this must be done while applying the right amount of pressure to stop the itch, but without scratching off any skin.

To mimic this complexity in the bionic limbs, the bio-electronic interface must be able to convert ‘sensory’ input from the prosthesis into neuronal impulse and the motor feedback from the brain into electrical signals that can make the artificial limb make the appropriate motion.

SENSORY HOMUNCULUS

The brain can be compared to a highly complex computer that integrates sensory inputs from different parts of the body and orchestrates appropriate muscular movements and reactions. Specific regions of the brain are dedicated to processing information from different parts of our body. Such mapping of different parts of our body to the corresponding cortex region of the brain is called ‘sensory homunculus’ (see fig. 1). Hands, face and legs get a major share of representation because they have the maximum sensory input through their nerve endings. The larger the size represented on the homunculus implies there are more sensory nerve inputs from that organ and correspondingly more motor output to bring about intricate movements.

In the case of amputees, this raises a couple of questions: What happens to the nerve endings in the removed part? What happens to the neuronal circuitry in the cortex region of the brain that corresponds to the amputated part? The answers to these questions are important to be able to understand the neuronal circuitry in the amputee, and be able to harness the neuronal signals to control the bionic limb. Scientists have gained a significant amount of knowledge on this through studies on a phenomenon called ‘phantom limb’.

NEURONAL PLASTICITY AND PHANTOM LIMB

Many amputees somehow feel that the amputated limb is still present! They experience something called “phantom limb”, a common sequela of amputation involving approximately 90% of amputees. A phantom limb can be defined as a persisting sensory awareness of a limb after amputation- which can range from simple, diffused tingling sensations to complex senses of pain, the source of which is perceived to be from the amputated part of the body. For example, they may feel like a mosquito is sitting on an amputated limb and try to shake the limb to drive away the mosquito, but the limb is not there!

Various hypotheses are put forward to explain the ‘phantom limb’ phenomenon. One of which is “cortical plasticity”. According to this, the sensory cortex region of the brain corresponding to the amputated limb is initially unused. But is soon occupied by the adjacent areas, e.g., the amputation stump. So, stimulation of the amputation stump (by a mosquito, for instance) gives rise to sensations apparently arising from the non-existent limb as this process stimulates areas of the cerebral cortex that previously represented the limb.

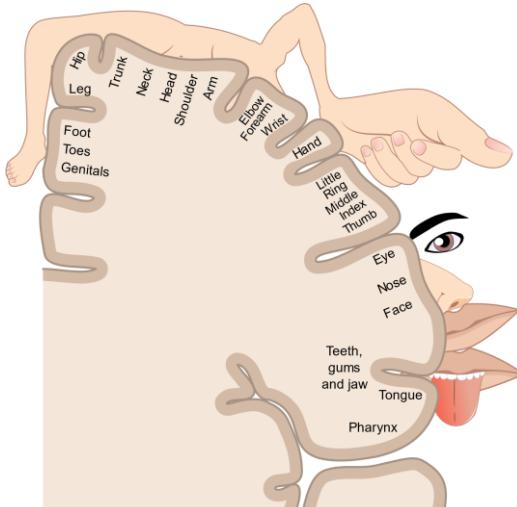


Figure 1. Sensory homunculus

File:1421 Sensory Homunculus.jpg: OpenStax College derivative work: Popadius, CC BY 3.0
<https://creativecommons.org/licenses/by/3.0/>, via Wikimedia Commons

The ability of the nervous system to rewire the circuitry in response to internal and external signals is called neuroplasticity. Neuroplasticity plays a vital role in learning, in memory formation, and in repair following damage (like trauma or stroke). Phantom limb experience is a consequence of neuroplasticity at the stump (or other nearby parts of the body) and at the cortex. Over time, through the plasticity of the nervous system, there can be an increase in the somatosensory representation of the amputated stump on the cortex. In other words, the stump of the severed limb will get a larger representation of the sensory homunculus. This can lead to an increase in sensory discrimination (the ability of the brain to perceive senses), and thus reduce phantom limb syndrome and dampen the feeling of disability.

This is where the researchers are currently working. It is the interaction between thought, response, and action that researchers across the world have been trying to replicate in their bionic technologies. The key to creating a bionic limb that is indistinguishable from a natural limb lies in the precise mapping of the rewiring of the nerves, harnessing signals from these nerves, and electronically processing the signals to actuate the prosthetic limb. Neuronal prosthetics or mind-controlled bionic limbs are a step closer to 'realistic prosthetic limbs'. The prostheses are integrated with body tissue, including the nervous system.

BIONICS, THE FUTURE OF PROSTHETIC LIMBS

Bionic limbs are better than even myoelectric limbs in two significant ways. One, bionic limbs respond to commands from the brain and spinal cord and, therefore, more closely replicate normal movement and functionality. Two, through retrieval and regeneration of sensory

signals in and around the lost limb, bionic limbs can communicate ‘sensory feeling’ to the brain through the electronic-neuronal interface at the stump. This paves the way for a seamless transition in the reintegration of the artificial limb. This can not only improve functionality, offer better control, and help reduce phantom syndrome but also significantly improve the behavior of the amputee.

The field of bionic limbs is in its infancy; there are some hurdles and hiccups to overcome yet. According to a study at the University of Chicago and Chalmers University of Technology, participants with neuromusculoskeletal bionic prosthesis could not regain subjective sensation at the touch receptors even after a full year of regular use [6]. But there have been a few inspiring success stories too. Researchers at the Cleveland Clinic have developed a neurorobotic prosthetic arm that allows users to think, feel and function more like a person with no amputation [7]. This has been achieved through the development of a bionic system added to a standard prosthetic that combines three important functions - touch, grip kinesis, and motor control. Another promising success is the development of the CyberHand system [8], a cybernetic anthropomorphic hand specifically designed to be connected via a neural interface to the peripheral and central nervous systems. This way, the wearer can transmit impulses from the brain to the bionic prosthetic limb when they want to use it. At the same time, the bionic limb receives signals from the external physical environment and relays them back through the peripheral nervous system to the brain. This allows the wearer to “feel” through the prosthesis. It’s almost as if they never lost an arm.

We are at the dawn of a new era of prosthetics; we have come to slowly realize the vast potential of bionics and its practical applications. It is every amputee’s dream to get hold of a prosthetic similar to the one used by Luke Skywalker (from the Star Wars Franchise) rather than a simple senseless hook of Captain Hook.

With more inspired youngsters (like you) getting into this field of research, the realization of such a dream is not too far!

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Into the World of Tractograms

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SUMMARY

Imaging nerves, their branching networks, and the nerve connections through which signals are passed was a challenge that plagued medical science. Advances in Diffusion Weighted Imaging (DWI) helped tackle this challenge. Today, tractograms have immense diagnostic and clinical applications. They also form the basis of connectomics. In this article, we give a brief overview of tractography, its development, application, and future prospects.

INTRODUCTION

A TRIP TO REMEMBER:

Kevin was excited. It was one of the most beautiful things he had ever seen. The stunning three-dimensional view with vibrant colors simply blew his mind. Shuffling along slowly, Kevin made his way to the next monitor. His eyes fell on what he would soon be introduced to as a *'tractogram.'*

As he stood there, appreciating the tractogram, a volunteer escorted him to the lounge, where he met the Chair of the Center. Her energy was infectious.

"I see that you have been using your time well, and I heard that you were engrossed in all the scans that our team is performing today." she remarked.

"Everything looks so exciting!"

"It is, indeed. Allow me to show you. We will cover all of it, starting from the past - which is usually a very good place to start," the Chair said with a smile.

BACKGROUND

"Brain is a complex organ that regulates all our functions. It comprises gray and white matter. The former contains the cell body of neurons, while the latter contains extensions emanating from these cell bodies, called axons.

The brain is also divided into the cerebrum, cerebellum, and brainstem. The cerebrum is the largest part of the brain, and comprises the gray and white matter at its center. It coordinates movement and regulates body temperature. Other areas enable speech, reasoning, emotions, and learning. The cerebellum is located at the back of the brain, and coordinates voluntary muscle movements, and maintains posture, equilibrium, and balance. The brainstem connects the cerebrum to the spinal cord and comprises the midbrain, pons, and medulla oblongata. The medulla is where the brain meets the spinal cord. These structures are connected by various neural pathways.

Neural pathways communicate information [1]. Ascending pathways carry information to the brain. This includes sensations like touch, pressure, and pain. The brain interprets this information and regulates body movements via descending pathways.

The history of neuroimaging

Although many advances in neuroimaging have transpired over the past couple of decades, a majority of the crucial work roots back to more than a century ago. Following World War II,

there were tremendous advances in neuroimaging. Angiography and pneumoencephalographs were common techniques that involved draining much of the cerebrospinal fluid (CSF) from around the brain through a lumbar puncture. The drained CSF was then replaced with air, oxygen, or helium to enable a more defined structure of the brain on an X-ray image. Frustrated by the inadequacies and associated dangers of the technique, in 1959, William Henry Oldendorf proposed the idea of using X rays to scan the head followed by reconstructing the radio density patterns [2]. In 1963, he patented a prototype to scan concealed objects using dense material [3]. This paved the way for the development of CT scans, which may have then cost about \$250,000.

CT and PET-CT

A few years later, in 1971, Godfrey Hounsfield introduced X-ray computed tomography (now called CT) [4]. This invention received enormous attention and changed the perspective of imaging the human brain. A group of investigators at Washington University later developed the positron emission trans axial tomography (or PET) by distributing radionuclides (especially ones that decayed by positron emission) within the X-ray focussed section, and then quantitatively reconstructing the distribution of these radionuclides within the section [5]. A CT scan is commonly used after traumatic injuries and strokes as it gives a clear picture of skeletal injuries and blood vessels. PET-CT is of immense use in studying cancers.

The emergence of MRI

In the last few decades, technical development and exploitation of magnetic resonance imaging (MRI) had a significant impact on diagnostic neuroimaging. In 1973, Paul Lauterbur used Nuclear Magnetic Resonance (NMR) signals to create cross-sectional images of the brain [6]. It uses a scanner to develop an image of the tissue based on the return rates of the spin. The output obtained is much more detailed when compared to CT because of its sensitivity to soft tissues. Although Magnetic Resonance Imaging (MRI) flourished after its introduction, it kindled the curiosity of scientists to know more about tissue chemistry, perfusion, and metabolism. This led to an exciting convergence of functional brain imaging with MRI, the development of fMRI. An MRI can be used to visualize soft tissues like muscles, tendons as well as the brain and spinal cord.

TRACTOGRAMS 101

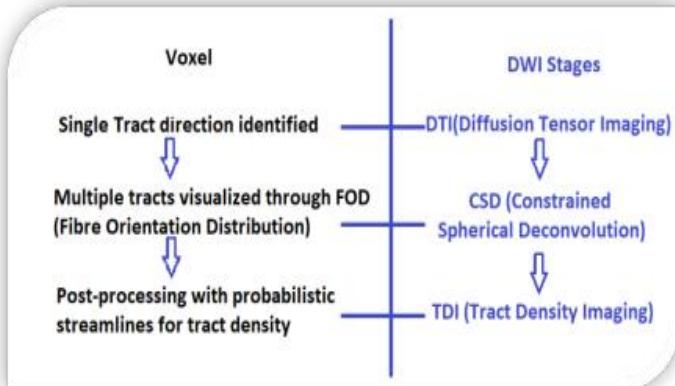
Even after the development of CT and MRI, one lacuna remained: imaging the nerves. The development of diffusion-weighted imaging (DWI) was a milestone in MRI innovations that paved the way to achieve this goal.

In this technique, the movement of water molecules and myelin — the component covering axons in the white matter — are presented as vectors in each voxel, the 3D counterpart of a pixel. This technique, known as anisotropic diffusion, laid the foundation for diffusion tensor imaging (DTI).

Over time, DWI went through many further modifications, as summarized in *Flowchart 1* [8].

Mapping anisotropic diffusion over numerous tracts was still a challenge *[Flowchart 1]*. If there was only one myelin tract in a neural pathway within each voxel, then that single tract direction can be identified with certainty using the anisotropic diffusion signal. However, the maze of a network that the human nervous system is causes every voxel to have many neural pathways, yielding many anisotropic diffusion signals. Complex algorithms that are applied to the

acquired image help us identify which tract directions are more likely within a given voxel.



Flowchart 1: Evolution of DWI over time [8]

Each voxel is, thus, taken as a 'seed point.' Based on the tract directions in its surrounding voxels, the software intelligently generates what is known as a 'probabilistic streamline' — continuous individual lines spread over numerous voxels, demonstrating the most likely pathways of the neural tracts [9]. These probabilistic streamlines put together form tractograms. A tractogram gives a good picture of the overall direction of the various myelin fibers in every voxel. Understandably, this is a highly specialised field that requires a great deal of expertise. And the field that deals with the interpretation of data collected from these diffusion-image studies is called 'tractography'.

Apart from performing individual diagnostic scans, tractography has other crucial research applications. As tractography is a novel field, medical science has only begun to better understand neuropathologies that used to be interpreted using astute clinical examination and various biochemical markers.

A fundamental rule of science is that understanding the normal is the key to identifying the abnormal. Similarly, it is vital to comprehend human physiology (which deals with normal functioning) to identify pathology (abnormal processes).

There are numerous special circuits within the brain, like the Papez Circuit (which regulates emotional expression) that help in various physiological processes [10]. These circuits form the foundation of a field known as connectomics. Connectomics deals with the way an organism's neural wiring is laid out with the aim to produce maps that aid in understanding how these networks help to regulate various physiological functions. Naturally, tractography plays a key role in this as well [9, 11]. Medical science aims to put the information so learned into use in tractometry studies and virtual dissections that will further our understanding of neuropathology [12]. Tractography is thus essential for both diagnoses as well as research.

The clinical applications of these scans and novel research have already been immense [13]. Management of intrinsic brain lesions, including spinal cord tumors, and seizures, are now possible due to tractography. The field has been a boon for patients needing deep brain stimulation and cranial nerve mapping and for those suffering from vascular anomalies like cavernomas and brain arteriovenous malformations (BAVM) [13]. Doctors have recently used tractography to image myocardial fibers to manage infarctions [14].

Though there is still a long way to go in perfecting the technique [15], tractography has opened the way to immense possibilities and promises an exciting future."

FINAL WORDS

"I really look forward to staying in touch with you. You have been an absolute inspiration for me, ma'am!" Kevin said.

He would be coming back here later in life. Back to his dear world of tractograms ...

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Brain Machine Interfacing

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SUMMARY

Brain Machine Interfacing (BMI) is a technology that allows the user to interact with the outside world using brain signals. This revolutionary advancement can help many patients with neurological illnesses to become more independent and live better lives. Broadly, BMIs are divided into two categories: invasive and non-invasive BMIs. The main focus of BMI development is to establish clinical efficacy and safety before they can be rolled out into mainstream clinical practice.

INTRODUCTION

Being able to move a cursor on a screen might not seem like a lot for a healthy individual, but for a quadriplegic patient (paralyzed from the neck down), this would mean independence. With a moving cursor, they can communicate with their loved ones via text, interact with the world on social media, order groceries online, and possibly even work a computer-based job. Controlling devices with 'thought' sounds a lot like science fiction, but this fantasy is now slowly being translated into reality with the help of cutting-edge technology known as Brain Machine Interfacing. Brain Machine Interfaces (BMI) are devices that record brain signals, analyze them, and transform them into commands which are relayed to an output for a meaningful action [1]. The primary focus of these cutting-edge devices is to restore dignity in the lives of patients suffering from crippling neurological diseases like stroke and spinal cord injury by providing an alternate way of responding to their surroundings and carrying out day-to-day activities.

For stroke, these devices have accelerated post-stroke rehabilitation [2,3]. In a stroke, the brain has been choked off of its blood supply by a clot in its blood vessels resulting in some parts of the brain being permanently damaged. Although less common than a stroke due to a clot, a stroke can also happen due to bleeding in one part of the brain. Post-stroke rehabilitation involves activities and exercises intended to teach this crippled brain to recruit the undamaged parts of the brain's neuronal circuitry to carry out daily tasks such as walking, holding a pen, etc. Post-stroke rehabilitation programs that use Brain Machine Interfaces seem to have an accelerated learning curve for recovery and rehabilitation. In spinal cord injury patients, the goal is to help nerve signals travel from the brain to the muscle, bypassing the 'neurological roadblock' created by the damaged spinal cord [4]. This is possible since when the spinal cord gets damaged at a particular segment, the neurological apparatus above and below the damaged section remains largely unaffected. If such a neural bypass is possible, the spinal cord injury patient can live a full and complete life instead of being restricted to a wheelchair and a bed for the rest of their life. BMIs are also being tested to improve the lives of children who can't speak or move well (like cerebral palsy patients) and to overcome the functional limitations of aging in the elderly [5,6].

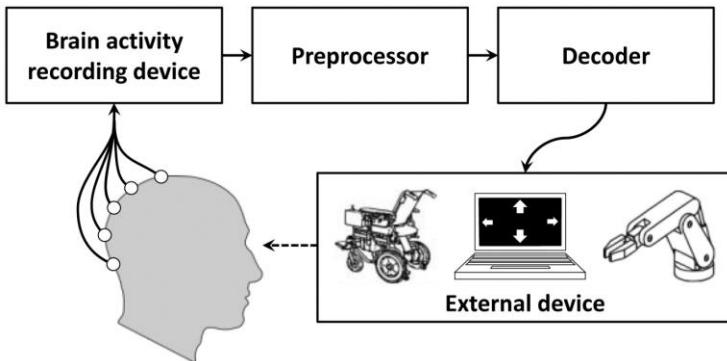
**Figure 1. Brain Machine Interface Scheme.**

Image credit: Anderson Mora-Cortes et al., 2014 (<https://www.mdpi.com/1424-8220/14/4/5967>).

BRIEF HISTORY

The idea of using brain signals to communicate directly with a computer or to use a prosthesis is not new. In fact, Vidal proposed this idea back in 1973, and this has entranced the neuroscience community since then [7]. Since its initiation, BMI research has gone from enabling participants to do modest tasks like controlling the vertical movement of a rocket on a computer screen using EEG signals in 1980 [8] to allowing a spinal cord injury patient with complete quadriplegia to open an e-mail, operate a television and perform rudimentary tasks with a robotic arm by 2006 [9]. The main difference among BMIs is the method by which they acquire brain signals.

DIFFERENT TYPES OF BMI

Broadly, they are divided into non-invasive and invasive BMIs. The most popular one is the non-invasive Electroencephalograph (EEG) based BMIs, where scalp electrodes collect the electrical signals from the brain [10,11]. EEGs utilize electrodes stuck to your scalp to record the electrical activity of the brain in real-time and hence are similar to Electrocardiographs (ECGs) that record the electrical activity of the heart via electrodes attached to the chest. EEGs are commonly used in clinical practice to monitor brain activity, especially to monitor brain activity in patients with seizure disorders, and help in the diagnosis and management of these disorders. Although they are cheap, convenient, and safe, the quality of signal acquisition is far superior in invasive modalities such as electrocorticography, where electrodes are surgically kept on the brain surface [12].

The main reason for this is the distortion of electric signals from the brain by the tissues that are interposed between the cortical surface and an EEG electrode, including the dura, skull bone, and skin. Electrocorticography can acquire cleaner signals from the brain by placing the electrodes directly on the brain's surface, minimizing signal distortion and improving the signal-to-noise ratio. This requires invasive surgery and is risky. Minimally invasive BMIs, such as endovascular BMIs, are also being tested extensively [13]. These devices attempt to plant electrodes into the cerebral vessels, which can be accessed by a minimally invasive procedure that does not require the opening of the skull and hence avoids open brain surgery. The signal

acquisition of endovascular BMIs would be good since the electrodes are situated near the brain surface.

CURRENT STATUS OF BMI RESEARCH

The past few years have seen a rapid surge in companies that are trying to develop BMIs, with significant players like Elon Musk's Neuralink leading the forefront of BMI research. This company not only aims to restore sensory and motor functions in patients with neurological disorders but also wishes to connect human brains to machines and achieve human-Artificial Intelligence Symbiosis [14]. Neuralink utilizes an array of multiple tiny electrode threads, which are planted surgically by a high-precision surgical robot into the brain. Initial testing with rats provided promising results, but the biocompatibility, safety and efficacy of this device must be verified with clinical studies before they can be integrated into clinical practice [14]. Neuralink recently released a video showing a monkey telepathically playing a computer game with its mind, which drew much attention from the public [15].

An endovascular BMI company called Synchron (headed by Australian Interventional Neurologist Dr. Thomas Oxley) started clinical trials this year with their endovascular BMI device called Stentrode [16]. Synchron enabled a patient paralyzed due to Amyotrophic Lateral Sclerosis (ALS, also known as Lou Gehrig's disease, a terrible disease that slowly paralyzes the patient's muscles) to tweet, "Hello World" [17].

Although famous physicist Stephen Hawking had ALS, he could communicate with the rest of the world, continue his research in physics and even write books with a computer that he controlled with the movements of his eyeballs. Imagine what he could have done had his life been enhanced with the capabilities provided by BMI technology. There are many smaller companies attempting to crack into this futuristic niche in medical biotechnology. This commercial interest comes with its own risks and pitfalls. Recently, Neuralink faced a setback when a few of the monkeys in whom their BMIs were tested died due to complications [18]. Nevertheless, the presence of multiple companies spearheading research and development in the BMI space is accelerating its growth rapidly and could be beneficial, provided the ethical and safety concerns are adequately addressed.

LONG-TERM GOALS

Augmenting neurologically normal humans also seem to be on the long-term agenda for BMI research. Things like recording memories and communicating without words seem to be theoretically possible. Integrating human intelligence with Artificial Intelligence could also provide new and exciting avenues for realizing the human potential. This AI-human symbiosis would even allow for less displacement of human jobs by new AI technology. If all these possibilities are discovered, it could potentially be the next step in human evolution.

ETHICAL AND PRIVACY CONCERN

The development of BMIs and their potential applications in augmenting human capabilities raise a wide array of ethical dilemmas which should be addressed adequately. Concerns include the hacking of minds, theft of memories, loss of conventional methods of human interaction, and the impacts of such a radical change in the social dynamics and the human psyche. At the current stage of its development, the main focus of BMI research should be to restore function to neurological patients, and safety testing would be the most critical factor that decides their acceptance among patients. As this technology moves forward in years to

come, other issues will start getting more attention, and we will need innovative solutions to new ethical and privacy challenges.

CONCLUSIONS

Although a concept that has been conceived and worked on since the 1970s, the idea of controlling devices with one's thoughts has been booming in recent years. The current focus of BMI development is to enable patients who are handicapped by specific neurological ailments to interact with the world and even enable them to take care of themselves by controlling devices with their thoughts. This would restore dignity and independence in their lives. BMIs vary mainly in their method of signal acquisition. Invasive BMIs give cleaner signals but bear the risk of open brain surgery. On the other hand, non-invasive BMIs are safer, but this comes at the cost of poor signal quality. Currently, many companies are breaking into the BMI business, and their research is helping realize the potential for BMIs in assisting neurological patients to live better lives. Elon Musk's company, Neuralink, is a big player in this field, and although their developments are promising, the safety, efficacy, and biocompatibility of their devices are yet to be verified by clinical trials. Synchron, an endovascular BMI company, has also shown rapid advancements in its progress, including a currently active clinical trial involving its endovascular BMI device named the Stentrode. Futuristic applications like recording memories, communicating with thought, and human-AI symbiosis are also part of the BMI agenda. BMI development raises multiple ethical dilemmas and privacy concerns which need to be addressed adequately before these devices are rolled out into the clinics and for mass use.

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Neurotechnology and Rehabilitation

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SUMMARY

Disorders of the nervous system often cause problems with the alteration of perception and execution of movements. The incidence of disorders such as stroke, Parkinson's, Alzheimer's etc., is increasing in the population. Rehabilitation aims to improve the quality of lifestyle of the survivors of such disorders. Different technological interventions such as robotics, brain-computer interfaces, stimulation, and virtual reality have enabled a wide range of approaches to be applied for rehabilitation.

INTRODUCTION

When Juliano Alves Pinto, 29, kicked off the ball at the inauguration of the 2014 FIFA world cup, it was indeed an astonishing event. Mr. Pinto had lost control of his lower body due to a car accident eight years ago, and kicking a ball would have been impossible for him but with the help of scientists. Scientists helped convert his brain activity into actions with the use of a robotic suit that moved when he wanted to execute a movement. But how were they able to do so [1]?

Unfortunately, the story of Juliano is not so uncommon. All over the world, a large number of people suffer from diseases or conditions that affect their nervous system and impact their daily lives. Conditions such as stroke, Parkinson's, and multiple sclerosis often leave people with life-altering disabilities. Rehabilitation aims to help such survivors with problems that are either related to making movements, communicating with their friends, families, or caregivers, and problems with memory, attention, or perception. As human life spans, get longer, thanks to improved health care, new disorders have popped up. Due to longer life spans, people have an increased chance of suffering from stroke, Parkinson's, Alzheimer's, and other conditions. To manage such disorders, rehabilitation is beneficial, as it exploits the 'plasticity' of the brain.

THE PLASTIC BRAIN

Have you all seen a map? The brain is a map too, but instead of countries etched on it, it has body parts etched on its surface in an area called the cortex. The specific areas in the cortex represent the hands, feet, lips, tongue, and other body parts. When those body parts are used, their respective representations in the cortex get activated. But what would happen if the limbs were to lose their function or if fingers were amputated? Would that affect the representation in the cortex? The answer is yes! When areas in the cortex do not get signals from the body part they represent, their sizes shrink over time. The neighboring representations in the cortex, meanwhile, are still receiving signals from the functioning body parts, and they take over the affected areas. People who undergo amputation of their limbs sometimes feel what is known as the "Phantom limb sensation," which makes a person feel that their lost limb is still present. This phenomenon occurs as the cortical area of the affected limb is taken over by the neighboring region. To stop this from happening, rehabilitation makes the patients stimulate their weakened senses by sending signals to the cortical representations. Let us now talk about how we can mold the brain [2].

Virtual Reality (VR)

One of the most popular rehabilitation methods these days is virtual reality, which allows the user to perform daily contextual activities in a virtual environment. The setup consists of a screen, VR goggles, sensors that register the movement of the user, and interactors. Beyond its use in the field of entertainment, VR can also be used for effective treatment for overcoming challenges related to movement or perception. Because the user is in a virtual environment, they feel more motivated to perform tasks, and VR tasks are often designed such that they mimic real-life situations like shopping in a mall, withdrawing money from the ATM etc. VR also gives feedback to the user in the form of sensations like sound, light, or movement of the base platform to invoke a sense of reality. Another exciting thing about VR is that while patients perform these tasks, scientists and clinicians can measure various physiological markers, such as heart rate and electrical potential from muscles, and use them to evaluate how well the users are doing. Scientists studying patients using VR found that they make more movements when they are in an engaging environment than patients who were given traditional rehabilitation treatment involving pen and paper [3-5].

Robotics

What do you imagine when you think of a robot? A cyborg, Mars rover navigating the Martian landscape? Well, you must think more closer. Robots can be used in rehabilitation as well because they can assist people who have trouble executing movements. They might help the patient by allowing them to make movements of each joint of the hands, or they might help in moving the whole limb, like the leg. Robotics creates a safe environment for the user and physically supports them while they are attempting to move. It focuses on its users being able to execute meaningful actions, for instance, picking up a coffee cup or walking without a crutch. When the affected limb or body part is used, it leads to positive plastic changes in the brain with the assistance of robotics [3].

Stimulation

We talked about how when we move a body part, its representative cortical region gets activated. However, there is a way to activate these regions of the brain from outside the skull. This astonishing technology is known as non-invasive brain stimulation, in which magnetic fields or weak electric currents are used to change how neurons respond. With a technique known as transcranial magnetic stimulation, neurons that constitute the nervous system can be stimulated to fire signals or repressed, translating into the corresponding cortical regions being activated or inhibited. For effective rehabilitation, activation of cortical regions that are affected and inhibition of those that are not beneficial works best [3,4].

Brain-Computer Interfaces (BCIs)

Brain-Computer Interfaces are devices that read neural signals and enable the user to move a cursor on the screen. Neurologists place electrodes on the brain surface to read these neural signals, which record brain activity. Electrodes are placed directly on the brain surface by performing surgery or on the skull where no surgery is required. These signals encoding brain activity are fed into a computer, which associates the signals with the user's specific tasks. When the computer encounters brain signals based on what it has previously learned, it predicts the user's actions which helps them to move and communicate [3,6,7].

Better Together

It has been seen that when either of the above technologies is used in isolation, they have a moderate positive effect. Studies have found that when used together in combination, the

technologies give holistic and significantly improved results that are retained for a longer time. For instance, when users are highly motivated to perform everyday tasks in a VR environment, stimulating their brains using non-invasive brain stimulation evokes better outcomes [3].

LIMITATIONS

It is indeed exciting to see how many new technological interventions have flooded the rehabilitation landscape that could assist patients by improving the quality of their daily lives. However, when it comes to accessibility, most of these technologies remain a far-fetched dream for patients from low and middle-income countries. With technologies such as BCIs, maintenance is a significant factor, which adds to costs and logistical issues. Additionally, most of the studies involving these technologies recruited a small number of participants, which is insufficient to extrapolate the findings to the larger population. Also, not every participant has uniformity when the disease duration, stage, and severity are concerned.[3,8]

CONCLUSION

Most patients expect ease in their daily activities when they think of rehabilitation, and this field has primarily concerned itself with alleviating deficits in movements, which has shown promising clinical results. Researchers have also been working towards advancing other valuable technologies that would assist in making the results more generalizable and reproducible. People who fall prey to such conditions often report the loss of social support in the form of friends in the aftermath of a disabling condition. Human empathy and support are essential, and neurotechnology has to be coupled with personal care and concern. Therefore, we also need to rethink societal perceptions about disability.

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Translated Articles

Hindi

ब्लाइंडसाइट, और यह कैसे हमें मानव मन को समझने देता है Blindsight, and how it helps us see the workings of the human mind

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Translated from English to Hindi by Vikram Iyer

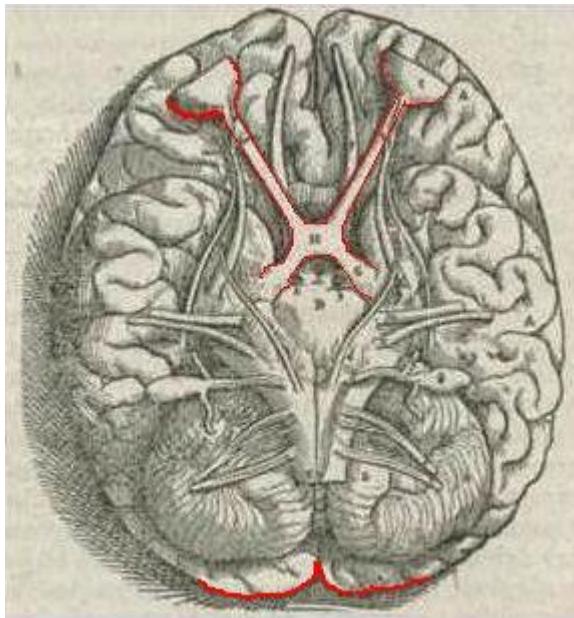
संक्षेप

ब्लाइंडसाइट एक अनूठा रोग है जो हमें मस्तिष्क के भीतर झांकने देता है। ब्लाइंडसाइट जैसे तंत्रिका संबंधी रोग तंत्रिका विज्ञान और मनोचिकित्सा के बड़े प्रश्नों का हल निकालने देते हैं। यह लेख ब्लाइंडसाइट की समीक्षा देता है और स्पष्ट करता है कि यह हमें कैसे मानव मस्तिष्क को बेहतर समझने में मदद करता है।

मिस्टर एल (ओपनीयता के लिए नाम रोक दिया गया है) अपनी छह अपने दाहिनी ओर एक व्यक्ति को देते हैं। लगभग पूरी तरह से अंधा, उसे बिना किसी मदद के बाधाओं से भरे कमरे के एक कोने से दूसरे तक चलना है। यह एक कूर मजाक की तरह लग सकता है, पर शोधकर्ताओं को वही परिणाम मिलते हैं जिनका उन्हें शक था: “चेतन रूप से” जाने बिना, मिस्टर एल ने बाधाओं से टकराए बिना कमरे को पार कर लिया है, जैसे कि वस कभी अंधस थे ही नहीं! [1]

यदि आपकी आंखों पर पट्टी बांधकर आपको एक कमरे में घूमने को कहा जाए, तो आप बिना किसी विचार के आगे बढ़ते और जितना मुकिना हो उतनी बाधाओं से बचते हुए चलते। मिस्टर एल की स्थिति—जो एक स्टोक के कारण हुई जिसने उनके प्राइमरी विशुअल कॉर्टिक्स को घायल कर दिया, अंधापन ज़रूर है। वे वर्षों से अपनी आंखों पर पट्टी बांधे हुए हैं और उन्हें लगता है कि ऐसी स्थिति में वे पूरी तरह से यादचिक पथ ले रहे हैं, परन्तु फिर भी वे बाधाओं से बचकर अपनी चाल चल लेते हैं। [2]

मस्तिष्क हमें चीजों को देखने में कैसे मदद करता है, इस चित्र में दर्शाया गया है (चित्र 1)। (केवल इस स्थिति को समझने लिए कम से कम जानकारी के आधार पर।) चित्र वृश्य मार्ग को दिखाती है जो हमारी दृष्टि में शामिल सर्किट है: । रेटिना पर बनने वाली छिकि का एन्कोडिंग करने वाले ईम्पल्स ऑप्टिक तंत्रिका के माध्यम से नेत्रोलक से बाहर निकलते हैं, जो संकेत ले जाती हैं, जैसे एक केबल जिसमें कई तार होते हैं (यहां यह तार तंत्रिका कोशिकाएं हैं जिन्हें “न्यूरॉन्स” कहा जाता है)। ऑप्टिक तंत्रिकाएं एक दूसरे को पार करती हैं, ऑप्टिक चियास्मा (जिसका अर्थ “क्रॉसिंग” है) बनाती हैं, और फिर मस्तिष्क के विभिन्न हिस्सों में संकेतों को लेकर ऑप्टिक ट्रैक्ट के रूप में जारी रहती हैं।



चित्र १ दृश्य मार्ग

चित्र स्रोत: एंड्रियास वेसालियस के पुस्तक से *De Humanis Corporis Fabrica Libri Septem*

दृश्य मार्ग में कोई भी समस्या ईम्पल्ट्स के इस प्रवाह को रोकती है, जिससे हमारे दर्शन क्षेत्र के विभिन्न भागों में अंधापन हो जाता है। सिग्नल ट्रांसमिशन पाथवे में और आगे आनी वाली समस्याओं पर टिप्पणी करना इतना आसान नहीं है।

मानव मस्तिष्क क्रमविकास का चमत्कार है। इसका विकास लाखों सालों से चला आ रहा है, ऊतक और विद्युत रासायनिक गतिविधि के एक इमारत की तरह, जो उन सभी बादलों को छूता आ रहा है जिन्हें हम मानवीयता का शिखर मानते हैं—नैतिकता, संगीत, भाषा, रचनात्मकता, करुणा, और बाकी सभी कुछ। हम अपने मस्तिष्क के लेआउट में समय के साथ होने वाले क्रमविकास की प्रक्रिया का प्रतिबिंब देखते हैं। मस्तिष्क का हर भाग मुख्यतः कुछ कार्य करता है। मस्तिष्क के निचले हिस्से और सक्रिट सांस लेने जैसी आदिम गतिविधियों में भाग होते हैं। मुख्य रूप से यह क्षेत्र रीढ़ और ब्रेन स्टेम के करीब पाए जाते हैं[3]। यहाँ से ऊपर आते आते हम भोजन, भावनाओं, लेखन और भाषा जैसे कार्यों पर चढ़ आते हैं, जिनमें से प्रत्येक भाग मस्तिष्क के क्रमानुसार “उच्च क्षेत्रों” में अपनी जटिलता और महत्व के अनुसार शामिल होते हैं।

इसे ध्यान में रखते हुए, हम ब्लाइंडसाइट की ओर लौटते हैं और देखते हैं कि उपलिखित “चेतन रूप से” अंश रेखांकित व इतना ज़रूरी क्यों था। हम यह विश्वास करना पसंद करते हैं कि हम जो कुछ भी करते हैं वह “स्वैच्छिक” है - एक ऐसा शब्द जो तत्रिका विज्ञान उन कार्यों के लिए इस्तेमाल करता है जिन्हें हम नियंत्रित कर सकते हैं। हम यह भी जानते हैं कि साँस लेना एक स्वैच्छिक प्रक्रिया नहीं है: आप होशार्टिक कुछ समय के लिए गति या सांस को रोक सकते हैं, लेकिन यह मस्तिष्क के द्वारा सचेतना के बिना भी जारी रहता है। “निचले क्षेत्रों” का कार्य होने के नाते, इस बात को स्वीकार करना इतना मुश्किल नहीं है। लेकिन यह विचार कि दृष्टि जैसे कार्य (जो प्रत्यक्ष चेतना का हिस्सा नहीं है) भी इसका एक महत्वपूर्ण हिस्सा है—इस पर विश्वास करना इतना आसान नहीं है।

इसके कुछ उदाहरण यहां दिए गए हैं, जिनमें से पहला वाला तो आप यहीं कर रहे हैं। जैसे जैसे आप इन पंक्तियों को पढ़ते हैं, आपकी नज़र वाक्य पर बाएं से दाएं की ओर दौड़ती है। लेकिन इस चाल में एक पैटर्न भी है जिस पर आपने शायद कभी विचार नहीं किया होगा। यह चाल, जो “सैकेडिक मूवमेंट्स” कहलाती हैं, निरंतर प्रसंस्करण और प्रतिक्रियाओं को शामिल करती है जो फ़िलहाल अत्यंत शोध के विषय हैं। जबकि वाचन एक बहुत ही सचेत प्रक्रिया है, हमारा मस्तिष्क सरलता के लिए अचेतन मन को अंखों को पृष्ठ पर चलाने देता है, ताकि हमें हर बार इसके बारे में परेशानी न लेनी पड़े।

अब हम इसी विचार को ब्लाइंडसाइट पर लगाते हैं। यह अनुमान लगाया गया है कि मस्तिष्क में दृश्य प्रसंस्करण के दो मार्गों में से, “पुराना मार्ग,” जो वैसा ही रहता है, जिससे वस्तु ट्रैकिंग और स्पेशल मैपिंग स्थापित रहती हैं जैसा कि सबसे अदिम जीवों में भी अवश्यक होता था—कमल के पत्ते पर एक मेढ़क की सोचें जो धीर-धीर उड़ते मच्छर की ट्रैकिंग करता है। दूसरी ओर, “नया मार्ग,” उन प्रक्रियाओं को संभालता है जो इस बात को अलग करती हैं कि हम दुनिया की क्या और कैसे कल्पना करते हैं। जैसा कि वी.एस. रामचंद्रन कहते हैं, यह संभव है कि नए मार्ग के अवरुद्ध होने से, “दृश्य जागरूकता दूर हो जाए” [4]। हो सकता है कि आप सचेत रूप से यह न जान सकें कि आप कुछ देख रहे हैं या उसके बारे में अनुमान लगाएं, लेकिन दृष्टि के अधिक अदिम पहलुओं को बनाए रखा जाता है।

यह सब कहने के बाद, मुझे इसका उल्लेख भी करना होगा कि हम अभी तक पूरी तरह से ब्लाइंडसाइट को नहीं समझ पाए हैं—हमने केवल एक शुरुआत ही की है। यह कई मनोजौनी न्यूरोलॉजिकल घटनाओं में से एक है जो न केवल हमारा ध्यान खींचती है और दिखाती है कि हम मस्तिष्क के बारे में कहां अधिक सीख सकते हैं, बल्कि विज्ञान क्षेत्र की सीमाओं को लगातार आगे बढ़ाती है। प्रश्न उतने ही महत्वपूर्ण हैं जितने कि उत्तर। (और शायद और भी!) विज्ञान की बढ़ती प्रगति के साथ साथ, जैसे-जैसे हम न्यूरोलॉजी, मनोचिकित्सा और मनोविश्लेषण जैसे क्षेत्रों के संगम पर दिमाग पर शोध करे जा रहे हैं, हमें उत्तरों का भण्डार भी मिला जा रहा है!

मस्तिष्क में ब्लाइंडसाइट की उत्पत्ति के बारे में शोध करते समय वैज्ञानिक अभी भी कई चुनौतियों का सामना कर रहे हैं। यह जांच और प्रक्रियाएं हमें याद दिलाती हैं कि हम मानव मस्तिष्क के बारे में कितना कम जानते हैं, इतने तकनीकी प्रगति और दशकों के शोध के बावजूद। हमारे अन्वेषणों ने लगातार नई, रोमांचक खोजें प्राप्त ज़रूर की हैं, पर हमें यह सोचकर संतुष्ट नहीं होना चाहिए कि हम सब जानते हैं। उह्यें हमें आगे देखने के लिए प्रोत्साहन देना चाहिए। हां, मुझे यह विश्वास करना पसंद है कि जब सुकरात ने कहा, “जितना अधिक मैं जानता हूं, उतना ही मुझे एहसास होता है कि मैं कुछ भी नहीं जानता।,” वह अज्ञानता को स्वीकारते हुए नहीं था पर उसके खिलाफ़ जोरदार अवज्ञा में था।

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जीवनशैली / व्यवहार संबंधी पहलुओं के द्वारा में मस्तिष्क गतिविधि में परिवर्तन

Changes in Brain Activity in Response to Lifestyle/Behavioural Aspects

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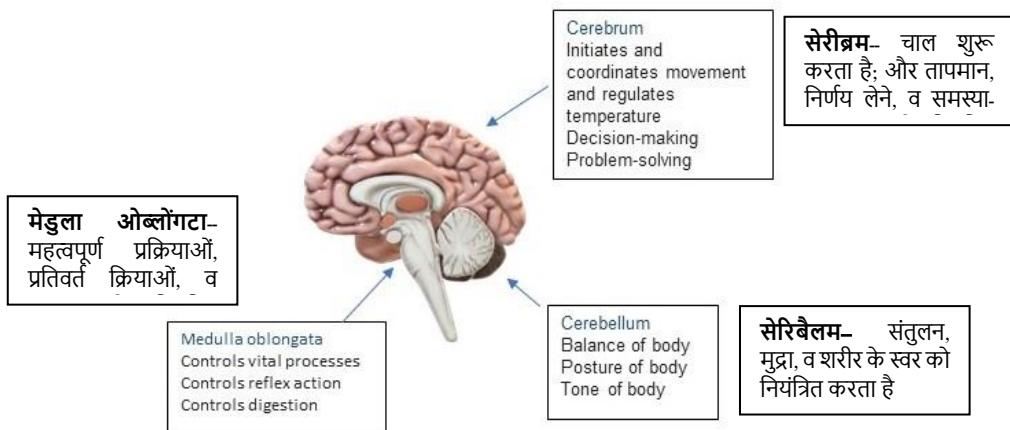
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Translated from English to Hindi by Vikram Iyer

संक्षेप

क्रोमिक शरीर मस्तिष्क के आधार पर चलता है, इसे शरीर का 'मास्टर ऑफ़न' माना जाता है। एक स्वस्थ जीवन शैली अर्थात् स्वस्थ सामाजिक संपर्क, अनुभव की विविधता, आहार और नींद से न्यूरोप्लास्टिसिटी होती है। न्यूरोप्लास्टिसिटी मस्तिष्क की नए सिनेइक कनेक्शन बनाने की क्षमता है, और यह न्यूरॉन्स को ज्यादा कुशल बनने में मदद करके शरीर की क्षमताओं को भी बढ़ाती है। परन्तु, केवल एक स्वस्थ जीवन शैली का पालन काफ़ी नहीं है। नए सिनेइक कनेक्शन बनाने के लिए नए कौशल सीखने की ज़रूरत होती है। उम्र जो भी हो, मस्तिष्क जीवन भर तंत्रिका मार्गों (न्यूरल पाथवे) को अनुकूलित और पुनर्गठित कर सकता है। कोई सीखने के लिए कोई कभी बहुत छोटा या बूढ़ा नहीं होता! (चित्र एक)



चित्र एक- मस्तिष्क की संरचना

Image credit: Shivani Pimparkar

शुरूआत

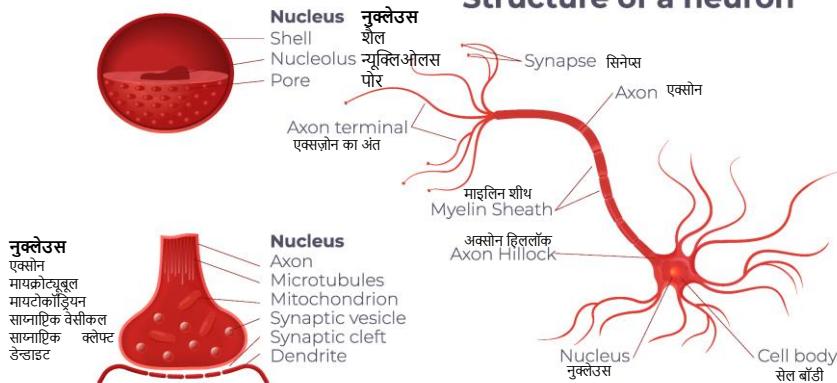
हमारा शरीर कितने ही अनोखे करतब कर पाता है— अपनेआप चलने वाली चयापचय प्रतिक्रियाओं (मेटाबोलिक प्रतिक्रिया) से लेकर एथलेटिक्स जैसे व्यायाम तक।

क्या आपने कभी सोचा है कि यह सभी प्रक्रिया किसके आधार पर चलती हैं? उत्तर स्पष्ट है परं किर भी दिलचस्प है— हमारा मस्तिष्क। 'ब्रेन' शब्द अंग्रेजी के पुराने शब्द 'ब्रेजेन' से लिया गया है जिसका अर्थ 'बुद्धि' है। मस्तिष्क हमारी सभी जीवन प्रक्रियाओं को नियंत्रित करता है जो शरीर को स्वस्थ रखते हैं, यह स्मरण, दृष्टि, श्वास या भूख ही क्यों न हो। परन्तु, मस्तिष्क शरीर को नियंत्रित करने वाला अकेला अंग नहीं है; हमारा मेरुरज्जु (स्पाइनल कॉर्ड) भी केंद्रीय तंत्रिका तंत्र (सेंट्रल नर्वस सिस्टम, सीएनएन) में मोजूद है।

मस्तिष्क के अंग

- न्यूरॉन मस्तिष्क की बुनियादी कार्यात्मक इकाई हैं।
- न्यूरॉन मस्तिष्क से शरीर के अन्य भागों में विद्युत इम्पल्स के संचालन के लिए जिम्मेदार होते हैं, जो मेरुरज्जु के ज़रिए अंगों तक पहुंचते हैं। ज़ंक्शनों पर एक न्यूरॉन दूसरे न्यूरॉन से सिनेप्स के द्वारा जुड़ा होता है।
- लगभग 1.3-1.5 किलोग्राम भारी मस्तिष्क में लगभग 60% वसा होती है जिसके कारण यह मानव शरीर का सबसे वसादार अंग है। शोधकर्ताओं ने पता लाया है कि इस वसा को कम नहीं किया जा सकता, मुख्यतः क्योंकि यह माइलिन जैसे मस्तिष्क के सबसे महत्वपूर्ण अणुओं को बनाता है।
- मस्तिष्क का बाकी 40% प्रोटीन, कार्बोहाइड्रेट और लवण के मिश्रण से बना है।
- सीएनएस की रक्त वाहिका, तंत्रिका और केंशिका नेटवर्क 400 मील (लगभग: 643 किमी) तक फैलती होती हैं। (चित्र 2)

Structure of a neuron



चित्र 2 न्यूरॉन की संरचना

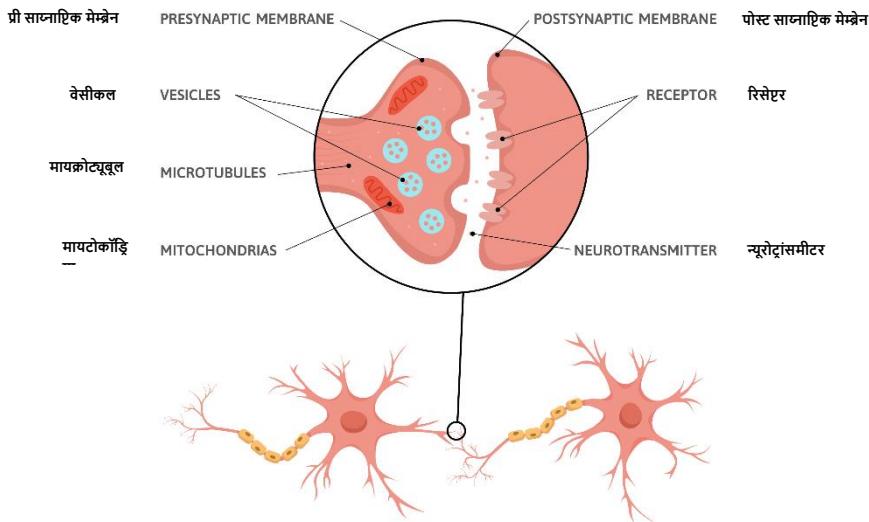
चित्र स्रोत: macropvector व <https://www.freepik.com>

न्यूरोप्लास्टिसिटी

न्यूरोप्लास्टिसिटी या ब्रेन प्लास्टिसिटी सिनेप्स, न्यूरॉन्स या यहाँ तक कि पूरे मस्तिष्क-क्षेत्रों की उनके उपयोगानुसार बदलने की प्रवृत्ति है। यह क्षमता हमारे पास जीवन भर रहती है। न्यूरोप्लास्टिसिटी का मतलब बाहरी अनुभवों के आधार पर सिनैटिक कनेक्शन को मजबूत करने या कमजोर करने या नई तंत्रिका कोशिकाओं को जोड़ना है। ऐसे कई तंत्र हैं जिनके द्वारा न्यूरोप्लास्टिसिटी हो सकती है: एक्सोनल स्प्राउटिंग और सिनैटिक प्रूनिंग इनमें से सबसे आम हैं।

एक्सोनल स्प्राउटिंग

एक्सोनल स्प्राउटिंग में एक स्वस्थ एक्सोन से नए तंत्रिका अंत (नर्व एण्डिंग) निकल आते हैं, जो तंत्रिका तंत्र के अन्य मार्गों से जुड़ते हैं। इसका उपयोग मौजूदा कनेक्शन को मजबूत करने या तंत्रिका तंत्र के घायल हिस्सों की मरम्मत के लिए किया जा सकता है। (चित्र 3)



चित्र 3 सिनैटिक जंक्शन की संरचना

चित्र स्रोत: macrovector व <https://www.freepik.com>

सिनैटिक प्रूनिंग

एक नवजात शिशु का मस्तिष्क उसकी की इंद्रियों के द्वारा आने वाली जानकारी से भरा होता है। इस जानकारी को किसी तरह मस्तिष्क के अलग अलग स्थानों तक पहुंचाना होता है। ऐसा करने के लिए तंत्रिका कोशिकाओं को एक दूसरे के साथ नए संबंध बनाने होते हैं।

जनम से अगले कुछ वर्षों के लिए, मस्तिष्क तेज़ी से बढ़ता जाता है। जैसे-जैसे प्रत्येक न्यूरॉन परिपक्व होता है, यह शाखाओं को बाहर भेजता है (एकजून जो सूचना भेजता है, और डेन्ड्राइट जो जानकारी लेते हैं), जिस से सिनैटिक संपर्कों की संखा बढ़ती है और न्यूरॉनों के बीच के संपर्क बढ़ते जाते हैं। जन्म के समय, सेरेब्रल कॉर्टेक्स में प्रत्येक न्यूरॉन में लगभग 2500 सिनेप्स होते हैं। जैसे-जैसे शिशु 2-3 साल का होता है, सिनेप्स की संखा लगभग 15000 तक बढ़ जाती है। यह मात्रा औसत वयस्क के मस्तिष्क में मिले जाने वाली संख्या से लगभग दोगुनी है। जैसे-जैसे हम बड़े होते हैं, पुराने कनेक्शन सिनैटिक प्रूनिंग के कारण हटा दिए जाते हैं। सिनैटिक प्रूनिंग और एक्सोनल स्पाउटिंग के कारण जो न्यूरोप्लास्टी आती है, वह मस्तिष्क या तंत्रिका तंत्र के अन्य हिस्सों के नुकसान को जल्द ही ठीक कर देती है। यही प्रक्रिया मनुष्यों को परिस्थितियों का सामना करने देती है।

मस्तिष्क गतिविधि को प्रभावित करने वाले कारक

सामाजिक संबंध

एक दूसरे से बातचीत और संपर्क करने के कई मानसिक और शारीरिक लाभ होते हैं, खासकर बुद्धिमत्ता में। एक नए अध्ययन के अनुसार एक दूसरे से बात करने से व्यक्ति को संज्ञानात्मक गिरावट (कॉम्प्रेसिव डिक्लार्इन) से बचाया जा सकता है। दो अध्ययनों में भावनाओं को प्रबंधित करने की क्षमता और सामाजिक अंतःक्रियाओं की गुणवत्ता के बीच संबंध पाए गए हैं। यह मेर्य-सलोवी-कार्ल्सो इमोशनल इंटेलिजेंस टेस्ट (MSCEIT) नाम के जांच की सक्षमता का समर्थन है। 1

आहार

हमें बहुत समय से शक था कि विशेष पोषक तत्वों की सापेक्ष मात्रा मानसिक और भावनात्मक कार्यों को प्रभावित करती है। मस्तिष्क स्वास्थ्य और मानसिक कार्य पर पोषण के प्रभाव को अंतर्निहित कुछ प्रमुख तत्वों को हाल ही में प्रलेखित किया गया है और सिनैटिक प्लास्टिसिटी और न्यूरोनल फंक्शन पर आहार के भारी प्रभाव का सुझाव देते हैं। हमारे आहार नली के कई हार्मोन जो मस्तिष्क में जा सकते हैं या जिनमें मस्तिष्क स्वर्यं पैदा करता है, संज्ञानात्मक कार्य पर प्रभाव डालते हैं। इसके अलावा, पहले से जाने हुए सिनैटिक प्लास्टिसिटी रेगुलेटर—जैसे कि ब्रेन डिराइब्ड न्यूरोट्रॉफिक फैक्टर, आहार जैसे बाहरी इनपुट के जवाब में चयापचय न्यूनाधिक को प्रभावित कर सकते हैं।

हमारे मस्तिष्क को सही ढंग से काम करने के लिए पर्याप्त ईंधन की आवश्यकता है। अच्छा ईंधन मस्तिष्क को उत्तम रूप से चलने में मदद करता है। विटामिन, मिनेरल, और एंटीऑक्सिडेंट से भरपूर भोजन मस्तिष्क को पोषण देता है और इसे ऑक्सीडेटिव तनाव से बचाता है। यह फ्री रैडिकल (एक या अधिक इकलोते इलेक्ट्रॉन वाले अणु) और एंटीऑक्सिडेंट (पदार्थ जो दान करके मृक्त कणों को हटाते हैं) के उत्पादन के बीच असंतुलन के कारण हो सकता है और अंतःकोशिका और ऊतक को हानि पहुंचा सकता है।

HEALTHY LIFESTYLE	स्वस्थ जीवन शैली	अस्वस्थ जीवन शैली	UNHEALTHY LIFESTYLE
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चित्र 4 स्वस्थ जीवन शैली का पालन

चित्र स्रोत: Macropvector व <https://www.freepik.com>

दूसरी तरफ, कम गृणवत्ता वाला “ईंधन” मस्तिष्क को नुकसान पहुंचा सकता है। प्रसंस्कृत भोजन (जैसे वे जिनमें बहुत सकरा मिलाया जाता है) इसके उदाहरण हैं। ऐसे खाद्य पदार्थ इंसुलिन के मात्रा पर नकारात्मक प्रभाव डालते हैं और सूजन और ऑक्सीडेटिव तनाव को बढ़ाते हैं। कई अध्ययनों में शर्करा युक्त आहार और बिंगड़ा हुआ मस्तिष्क कार्य—यहां तक कि डिप्रेशन जैसे मनोदशा संबंधी विकार के बीच संबंध पाए गए हैं।

इस क्षेत्र में अध्ययन को न्यूट्रिशनल साईकायट्री कहा जाता है। यह एक उभरता हुआ क्षेत्र है जिस में शोध केवल अभी शुरू ही हो रहा है। इसके शोधकर्ता और डॉक्टर कैसे जानते हैं कि कौन से खाद्य पदार्थ हमारे मस्तिष्क स्वास्थ्य को प्रभावित करते हैं? वे हमारे द्वारा खाए जाने वाले भोजन का निरीक्षण करते हैं। इसीलिए यह कहानी आहार नली में शुरू होती है! (चित्र 4)

व्यायाम

क्या आपने सोचा है कि व्यायाम महत्वपूर्ण क्यों है? ब्रिटिश कॉलंबिया विश्वविद्यालय में किए गए एक अध्ययन में शोधकर्ताओं ने पाया है कि एरोबिक व्यायाम को अक्सर करने से हिप्पोकैम्पस का आकार बढ़ जाता है, जो मस्तिष्क का वह क्षेत्र है जो स्मृति और सीखने में शामिल होता है। अन्य किसी प्रकार के व्यायाम के समान परिणाम नहीं थे।

नए कौशल

जब भी हम कोई नया कौशल सीखते हैं, तो अपने दिमाग को बदलत डालते हैं। विशेष रूप से, हम दिमाग के अंदर के कनेक्शन को बदलते हैं। जब हमें किसी चुनौती के साथ प्रस्तुत किया जाता है तो हमारा शरीर और दिमाग बदल जाते हैं— मांसपेशियां मज़बूत हो जाती हैं, दिल और फेफड़े बड़े हो जाते हैं, और मस्तिष्क के कनेक्शन तेज़ और मज़बूत हो जाते हैं। मस्तिष्क का यह पुनर्गठन सभी कौशल अधिग्रहण और विकास का आधार है। हमारा शरीर खुद नहीं जानता कि यह बदलाव क्यों होते हैं, लेकिन खुद के लिए बातों को संभवतः सबसे सरल बनाने के लिए यह लगातार खुद को पुनर्गठित करता जाता है। मानो कि हमें ऐसे प्रोग्राम किया गया है। जब चीजें आसान होती हैं तो हमारे शरीर और दिमाग को यह पसंद आती हैं। इसलिए अगली बार वैसी चुनौती मिलने पर वह उसे वैसे ही सरल बनाने का प्रयास करता है। किसी कौशल सीखते समय शरीर को तनाव देकर, हम अपने शरीर को संकेत देते हैं कि यह महत्वपूर्ण है। मानो हम उसे चुनौती के लिए और अधिक संसाधनों को समर्पित करने के लिए कह रहे हों। किसी भी कौशल का अभ्यास करने से हम तंत्रिका कनेक्शन को मज़बूत करते हैं, जिससे उसे अगली बार करना बहुत आसान हो जाता है!



चित्र 5 मस्तिष्क नए कौशल कैसे प्राप्त करता है?

चित्र स्रोत: vector4stock व <https://www.freepik.com>

डिजिटल प्रैदौरीकी के उपयोग के प्रभाव

डिजिटल युग में मोबाइल फ़ोन जैसे डिजिटल प्रैदौरीकी का उपयोग हमारी जीवन शैली में अत्यंत महत्वपूर्ण बन चुका है। इसने कुछ पहलुओं में हमारे जीवन को सरल कर दिया है, पर इसका हमारे मस्तिष्क के स्वास्थ्य पर भारी प्रभाव पड़ता है। कई अध्ययनों ने किशोरों में कंप्यूटर के उपयोग, ज्ञादा समय के लिए स्क्रीन को देखने और एडीएचडी के लक्षणों के बीच संबंध दिखाई है।

नींद

नींद भी हमारे मस्तिष्क की गतिविधि को प्रभावित करती है। यदि हम ठीक से नहीं सोते हैं, तो हमारे मस्तिष्क कमज़ोर हो सकता है, चीजों को भूल सकता है, और निर्णय लेने में कठिनाई पा सकता है। (चित्र 4)

मस्तिष्क के कार्य पर व्यायाम और नींद का प्रभाव

- नियमित व्यायाम करने वालों में अल्जाइमर रोग का विकास कम होता है। व्यायाम रक्त प्रवाह और स्मृति को बढ़ाती है। यह मस्तिष्क में रासायनिक परिवर्तन लाती है जो सीखने, मनोदशा और सोच में सुधार लाती हैं।
- जैसे-जैसे हम बढ़े होते हैं, जीवनशीली और पर्यावरणीय कारणों की वजह से मस्तिष्क अधिक हानिकारक तनावों का सामना करता है, जिसके परिणामस्वरूप ऑक्सीडेशन होती है जो मस्तिष्क की कोशिकाओं को नुकसान पहुंचाती है। जंग लगना या आधे खाए हुए सेब का भरा होना ऑक्सीडेशन के उदाहरण हैं जो दर्शा सकते हैं कि ऑक्सीडेशन के कैसे परिणाम होते हैं। जागून और सज्जियां जैसे एंटीऑक्सिडेंट युक्त खाद्य पदार्थ मस्तिष्क को ऑक्सीडेशन के हानिकारक प्रभावों से बचाने में मदद कर सकते हैं।
- नीद हमें जोश देती है, हमारे मूड और प्रतिरक्षा प्रणाली के कार्य को बेहतर करती है, और मस्तिष्क में बीटा-एमिलोयडल प्लाक नाम के एक असामान्य प्रोटीन की मात्रा को कम कर सकती है जो अल्जाइमर रोग से संबंधित है।
- मानसिक व्यायाम मस्तिष्क को अच्छी तरह से काम करने में मदद कर सकता है और मस्तिष्क की नई कोशिकाओं के विकास को बढ़ावा दे सकता है, जिस से मनोध्रंश (डेमोशिया) की संभावना कम हो सकती है। जैसे हम अपनी मांसपेशियों को चलाते रहते हैं, हमें वैसे ही अपने मस्तिष्क की कोशिकाओं को चलाते रहना चाहिए।
- एक सक्रिय सामाजिक जीवन जीने से हम याददाश्त खोने से बच सकते हैं। दूसरों के साथ समय बिताना, बातचीत में शामिल होना, और दोस्तों से जुड़े रहना मस्तिष्क के स्वास्थ्य के लिए अच्छे हैं। अध्ययनों से पता चला है कि ऐसा ज्यादा करने वाले लोग अपनी याददाश्त को सबसे धीरे-धीरे खोते हैं।

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सीखना कैसे सीखें Learning How to Learn

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संक्षेप

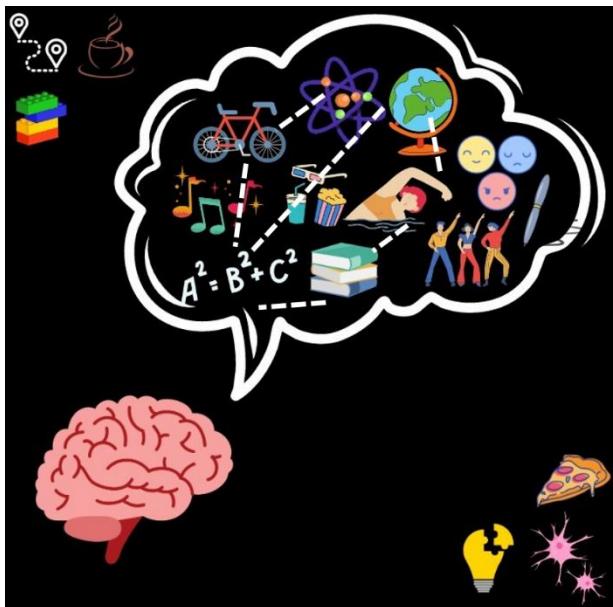
क्या आपने सीखा है कि हम इतने शानदार ढंग से गाने, फोन नंबर, कथक नाचने की विधि, और यहाँ तक कि कठिन विज्ञान को कैसे याद रख पाते हैं? यह सब हमारे मस्तिष्क की सीखने और पुनः सीखने की क्षमता के कारण है। न्यूरोनल वायरिंग पर अन्वेषण से हमें पता चला है कि सीखने की हमारी क्षमता ठोस नहीं है। सीखने की विभिन्न शैलियों का पालन करने से हमारी सीखने की क्षमता में जमीन-आसमान का सुधार आ सकता है। यहाँ आपको शिक्षण संबंधित सभी चीजों पर हमारा न्यूरो-मनोवैज्ञानिक दृष्टिकोण मिलेगा।

शुरुआत

शानदार दिमागों की खोज

कक्षा के कुछ छात्रों को 'फ्रॉट बैंचर्स' का नाम देकर बुद्धिमान और उत्तम क्यों माना जाता है? तथाकथित 'बैक बैंचर्स' को मूर्ख क्यों माना जाता है? क्या संभव है कि छात्रों को कैसे पढ़ने की प्रणाली से ही ये मतभेद उत्पन्न होते हैं? क्या यह हो सकता है कि ये छात्र एक ही विषय को सीखने के लिए विभिन्न शैलियों का उपयोग करते हैं?

1992 में, फ्लोरिंग और मिल्स ने वार्क मॉडल (विसुअल ऑरल रिडिंग राइटिंग व किनेस्टेटिक सीखने वाले) का प्रस्ताव रखा, जो एक नया और आकर्षक शिक्षण प्रतिमान था। उनके अनुसार हर एक शिक्षार्थी एक ही विषय को अलग अलग तरह से समझता है। कुछ शिक्षार्थी फिल्में, ग्राफिक्स और रेखांकित परिकल्पनाएँ करते हैं (ये दृश्य शिक्षार्थी हैं), कुछ व्याख्यान और चर्चा (श्रवण सीखने वाले), और कुछ पुनर्विचन और नोट्स (पढ़ने-लिखने वाले शिक्षार्थी हैं) पर जोर देते हैं। अत मैं, एक और समृद्ध व्यावहारिक प्रयोगों और संवादात्मक सत्रों (काइनेस्टेटिक शिक्षार्थीयों) को फायदेमंद पाता है। वार्क प्रश्नावली का उपयोग अक्सर यह पहचानने के लिए किया जाता है कि किसी को सीखते समय कौनसी शैली भाती है। [1]



चित्र 1 बहुविध मस्तिष्क। यह चित्रण दर्शाता है कि किस तरह मस्तिष्क को सीखने के लिए प्रशिक्षित किया जा सकता है और सीखने की कई शैलियों का उपयोग करके यादें और आदें बनाई जा सकती हैं।

परन्तु, सब इतना सरल नहीं है। क्योंकि वार्क की चार शैलियों में से प्रत्येक एक शिक्षार्थी को अपने मस्तिष्क के केवल एक हिस्से को दूसरे की तुलना में ज्यादा उपयोग करने देता है, आमतौर पर यह माना जाता है कि वार्क सीखने को मजबूत करने के बजाय उसे और बिगाड़ सकता है। उदाहरण के लिए, दृश्य शैली के लिए शिक्षार्थियों को रेखांकन के माध्यम से सीखने के लिए अपनी आंखों और मस्तिष्क के फ्रंटल लोब का उपयोग करने की आवश्यकता होगी। यह मस्तिष्क के अन्य वर्गों में चूरोनल फायरिंग को रोक सकता है। [2] और फिर आखिर वार्क मॉडल कितना भरोसेमंद है? क्या इसका तात्पर्य है कि एक व्याक्ति के पास केवल एक सीखने की शैली ही सकती है? वैज्ञानिकों ने पाया है कि वार्क जांचे गए वालों में से 39% विशेष रूप से एक श्रेणी [3] में नहीं आते हैं। इसके अनुसार शिक्षार्थियों ने केवल एक के बजाय दो या तीन अधिगम शैलियों को प्राथमिकता दी। (चित्र 1) आज कई शोधकर्ता का मानना है कि ऐसी शिक्षण शैलियाँ मौजूद ही नहीं हैं। विस्कॉन्सिन-ला क्रॉस विश्वविद्यालय के एक मनोवैज्ञानिक डॉ टेसिया मार्शिक के अनुसार जानकारी को याद रखने के लिए, हमें उसका अभ्यास इंद्रिय स्वतंत्र रूप से करना चाहिए।

सारधाना!

सीखते समय ध्यान देना अनिवार्य है।

ध्यान देना बाकी सब कुछ अनदेखा करते हुए जानकारी का विश्लेषण करने की क्षमता है। लेकिन आइए गहरी खुदाई करें। कल्पना कीजिए कि जिस कमरे में आप बैठे हैं उस कमरे में एक हाथी भाग रहा है। आप उसकी और ध्यान कैसे दे पाएंगे? यह तीन छोटे चरणों में किया जा सकता है: सक्रियण (जब आप कमरे में हाथी के बारे में जागरूक हो जाते हैं), दृश्य-स्थानिक पहचान (यह महसूस करना कि हाथी कितना बड़ा है और उसका रंग क्या है), और चयनात्मक-कार्यकारी घटकों पर ध्यान देना (पृष्ठभूमि में हर दूसरे शोर के बजाय हाथी द्वारा की गई आवाजों पर ध्यान देना) [4]।

तौ, क्या उक्तकृष्ट शिक्षा और ध्यान एक वांछनीय स्मृति की मात्रा है? उत्तर दोनों के योग में निहित है।

लाइट्स, कैमरा, एक्शन!

जब आप सीखने की कोशिश करते हैं तो दिमाग में क्या चल रहा होता है? कनाडा के मनोवैज्ञानिक डॉ डोनाल्ड ओ. हेब्ल ने प्रस्ताव दिया कि जैसे ही आप नई जानकारी को समझते हैं, न्यूरॉन्स सक्रिय हो जाते हैं। वे अन्य न्यूरॉन्स से जुड़ते हैं और नेटवर्क बनाते हैं। इन नेटवर्कों में न्यूरॉन्स की ये कड़ी शुरुआत में कमजोर होती है, लेकिन उत्तेजना की पुनरावृत्ति के साथ, न्यूरॉन्स के बीच संबंध मजबूत होते जाते हैं (एक घटना जिसे पोटेंशिएशन कहा जाता है)। [5] यही कारण है कि आवृत्ति से आपको अधिक जानकारी याद रखने में मदद मिलती है। क्योंकि एक ही मार्ग का बार-बार उपयोग किया जाता है, यह हार्ड-वार्यर्ड (या एक साथ बंधा हुआ) हो जाता है।

जब वैज्ञानिकों ने मानव पूरे मानव जीवन काल के दोरान इन न्यूरोनल नेटवर्क का अध्ययन किया तो कई अनोखी बातें पता चली। जन्म के समय, मानव मस्तिष्क में न्यूरॉन्स लगभग 2,500 सिनेप्स (सिनेप्स न्यूरॉन्स के बीच संपर्क बिंदु हैं) बनाते हैं और यह संच्चा वृद्धि के साथ तेजी से बढ़ती है (2-3 साल के बच्चे में 15,000 सिनेप्स तक)! परन्तु वयस्कों में, सिनेप्स की संच्चा घटकर आधी हो जाती है [6]। जैसा कि पहले उल्लेख किया गया है, विशेष नेटवर्क हार्ड-वार्यर्ड हो जाते हैं, जबकि अन्य की उनके क्रम उपयोग के कारण काट दिया जाता है।

तंत्रिका सर्किटरी की स्थापना

क्या हम जानते भी हैं कि आमतौर पर इस्टेमाल किया जाने वाला शब्द ‘प्लास्टिसिटी’ क्या है? प्लास्टिसिटी मस्तिष्क की नई जानकारी को बदलने और उसके अनुकूल होने की क्षमता है। यह इस बात का संकेत है कि मानव मस्तिष्क कितना लचीला है। यदि मस्तिष्क तेजी से संबंध बनाता और तोड़ता है, तो इसमें ‘शार्ट टर्म प्लास्टिसिटी’ [7] होती है। जब हम मोबाइल एप्लिकेशन पर कोई खर्च करते हैं, तो हमें अपने इनबॉक्स में एक ओटीपी प्राप्त होता है। फिर हम एप्लिकेशन पर ओटीपी को पुनः प्रस्तुत करते हैं, लेनदेन पूरा करते हैं और इंतजार करते हैं। ओटीपी को याद रखना और उसकी नकल करना ‘शार्ट टर्म प्लास्टिसिटी’ का एक बहुत अच्छा उदाहरण है। यह एक सेकंड के एक अंश के लिए आवश्यक होता है लेकिन बाद में फ़ालतू हो जाता है। परन्तु हमारे दिमाग में अधिकांश यादों को लंबे समय तक संग्रहीत करने की आवश्यकता होती है। इसे ‘लॉन्ग टर्म प्लास्टिसिटी’ कहा जाता है जो मिनटों से लेकर घंटों, दिनों या वर्षों तक भी रह सकता है और यह इस बात का प्रमुख मॉडल है कि मस्तिष्क कैसे सूचनाओं को संग्रहीत करता है। [8]

तो क्या इस प्लास्टिसिटी (फायरिंग रेट) को मापा जा सकता है? हाँ! इलेक्ट्रोएन्सेफलोग्राफी (ईईजी) मानव मस्तिष्क गतिविधि को मापने का अर्थात् न्यूरोलास्टी को मापने का सबसे मामूली तरीका है। ईईजी खोपड़ी के ऊपर कई इलेक्ट्रोड रखकर मस्तिष्क की विद्युत गतिविधि को मापता है। प्लास्टिसिटी को मापने का एक और प्रचलित तरीका एमआरआई है।

अंत में, यह याद रखने योग्य है कि प्रसिद्ध न्यूरोसाइंटिस्ट भी कभी सोचते थे कि हमारा दिमाग एक डिब्बे की तरह है जिनकी सीखने की क्षमता निश्चित थी। आज हम जानते हैं कि ऐसा नहीं है और यह एक थैले की तरह कई प्रकार के आकार ले सकता है।

दिमाग कब चलना शुरू करता है?

मस्तिष्क का आधारभूत नेटवर्क बचपन में रखा जाता है। बच्चों का दिमाग इंटकों में विकसित होते हैं जिसे क्रिटिकल पीरियड्स कहा जाता है। इनके दौरान, न्यूरॉन्स के बीच सिनेप्स की संख्या बहुत अधिक होती है, यानी कि वे किसी भी उत्तेजना के लिए ग्रहणशील होने की अधिक सक्षम होते हैं। उदाहरण के लिए, एक नई भाषा सीखने की सक्षमता को क्रिटिकल पीरियड द्वारा सीमित माना जाता है। यदि इस महत्वपूर्ण अवधि के दौरान एक बच्चे को भाषा के संपर्क में लाया जाता है, तो उसको नेटिव भाषिक की तरह भाषा प्राप्त करने की संभावना होगी। इस काल के बाद, ऐसा करना करना चुनौतीपूर्ण हो जाएगा। [9]

सीखना एक इमारत को बनाने जैसा है, जहां प्रत्येक सिनेप्स न्यूरॉन्स के ईंटों को मजबूत करता है ोर हमारे मस्तिष्क की संरचना का ढांचा उभर आता है। एक बार इमारत का शिखर और भूतल बन जाने के बाद, यह आप पर निर्भर होता है कि आप को मकान बनाना है या बुर्ज खीलीफा।

अध्ययनों से पता चला है कि बढ़ती हुई उम्र संज्ञानात्मक कार्यों का बाधा बन जाती है। परन्तु, सीखने की क्षमता उम्र के साथ पूरी तरह समाप्त नहीं होती। क्योंकि एक वयस्क मुख्यतः अपने आप सीखता है और सामान्यीकृत सिद्धांत वयस्क सीखने को समझने में मदद नहीं करते। मस्तिष्क के प्रीफ्रंटल और टेप्पोरल क्षेत्र कौशल के नवल और अनवल पहलुओं को अलग रखते हैं, यह सुनिश्चित करते हुए कि आपकी पुरानी और नई यादें बेमेल नहीं होती। फ्रेच सीखने के प्रयास में अंग्रेजी को भूलने की क्षम्यना करें! शुक्रज्ञार है कि हमारा दिमाग जितना हम उन्हें श्रेय देते हैं, उससे कहीं ज्यादा बुद्धिमान हैं। [10]।

तो, आपको लगता है कि आप सीख सकते हैं?

आइए, अब तक देखे हुए सब कुछ को एक साथ रखते हैं; सीखने की तकनीक, ध्यान, तंत्रिका सर्किट, और सीखने की गति। हम आशा करते हैं कि आप समझ चुके हैं कि हमारा दिमाग कितना शानदार है। यदि आप कोई संज्ञानात्मकता की ज़रूरत होने वाले कार्य को कर रहे हैं, तो इसका मतलब है कि विभिन्न मस्तिष्क क्षेत्र एक साथ काम करके निश्चित कर रहे हैं कि आप क्या जानते हैं ोर क्या नहीं। वर्तमान शोध सीखने के विभिन्न पहलुओं के विषय के अंगे बढ़ चुका है। इसमें गिलियाल काशिकाओं की न्यूरोनल और गैर-न्यूरोनल आबादी की जांच करना, वाईट मैटर का पुनर्गठन, इम्पल्ट्स संचरण के कारण क्या होते हैं, और अंत में, हमारे मस्तिष्क अत्यधिक चोट, स्ट्रोक व मानसिक स्वास्थ्य संबंधी विकारों में कैसे पुनः स्थापित होता है—सभी शामिल हैं। [11]।

कुछ भी सीखना कठिन है क्योंकि हम लगातार बहु-संवेदी उत्तेजनाओं को संभालते हैं और मौजूदा कनेक्शनों का रीवायरिंग करते हैं। जो अंतः हमारे व्यवहार में दिखता होता है। बुनियादी विकासात्मक टेम्पलेट्स के अलावा, हमारी सीखने की क्षमता ठोक नहीं है। [12] यदि हम कुछ नया सीखने के लिए दृढ़शाली हैं और ऐसा करने के लिए ‘ध्यान केंद्रित’ करते हैं, तो हम सफल होंगे ही (जिसे आमतौर पर ‘धैर्य’ कहा जाता है)। आखिरकार, ऐपीजे कलाम ने कहा ही है, “देश का सबसे अच्छा दिमाग कक्षा की आखिरी बेंच पर पाया जा सकता है।”

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बहुस्तरीय चेतन मन

Through the Kaleidoscope: The Multi-layered Conscious Mind

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संक्षेप

चेतना सभी जीवित प्राणियों की एक संयुक्त विशेषता के रूप से उभर आती है। इसके तात्पर्य विशाल और जटिल है।

जब हमारे सपनों में चेतना की कथित विकृति होती है तो इस चेतना पर विस्थय का एक और परत जुड़ जाता है। चेतना अपने कई संभावित सिद्धांतों और संरचनात्मक आधारों के कारण मस्तिष्क के भीतर चलने वाली प्रक्रियाओं को समझने में एक पृष्ठभूमि के रूप से प्रस्तुत होती है।

शुरुआत

क्या आपने कभी उठते ही अपने सपनों को याद करने और समझने की कोशिश की है? हम सब ने कभी ना कभी सोचा ही है कि हमारे सपने सच हैं या नहीं।

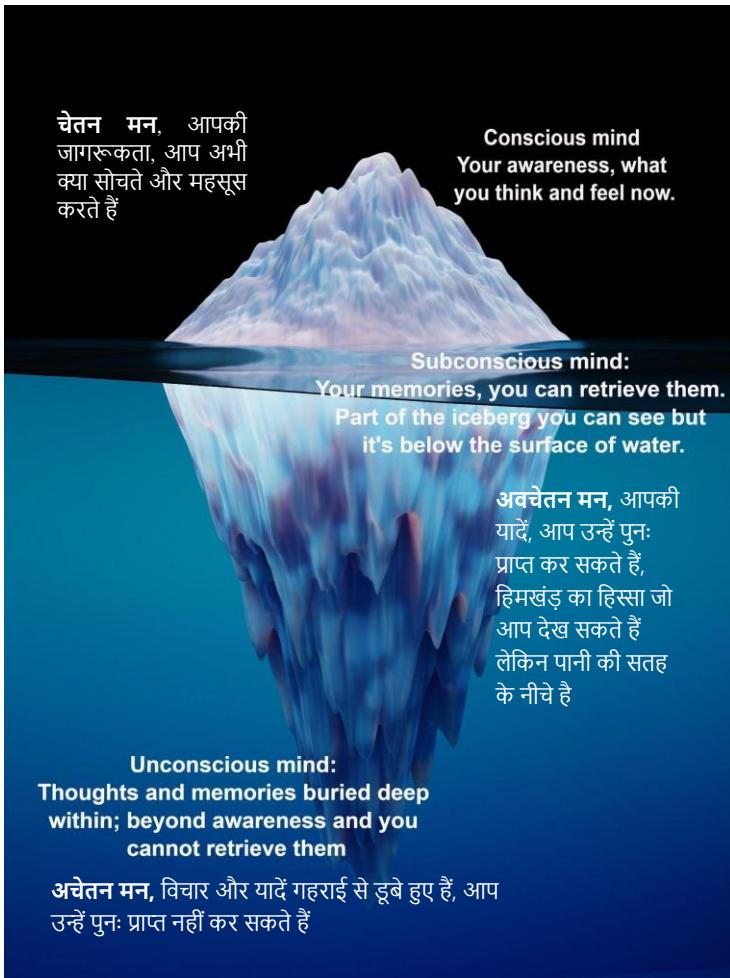
‘जागत’ और ‘सचेत’ होने का क्या मतलब है? यह अवस्था स्वप्न की अवस्था से किस तरह अलग है? हम कैसे ही जानते हैं कि हमारे अनुभव मात्र सपने नहीं हैं?

उत्तेजना देने पर स्थूल में बदलाव लाना— इसी की क्षमता चेतना कहलाता है। यह हमारी जाग्रत अवस्था का एक अनिवार्य लक्षण है। यह पर्यावरण के प्रति जागरूक होने की अवस्था भी है। इसमें अनुभव किया जाने वाला सब कुछ शामिल होता है - अच्छा, बुरा, बदसूरत, और सुंदर।

इस लेख में चेतना के विभिन्न सिद्धांतों का, अचेतन मन और उसके कार्यकलापों का, चेतना और अचेतन में अंतर्निहित सामाजिक तंत्रिका विज्ञान का, और सपनों के तंत्रिका विज्ञान का— सभी का अवेषण किया गया है। तो चलिए शुरू करते हैं!

चेतन और अचेतन मन का फ्रायडियन विचार

सौंसे ज्यादा साल पहले, प्रसिद्ध ऑस्ट्रियाई न्यूरोलॉजिस्ट और मनोविश्लेषणात्मक सिद्धांत के अग्रणी सिगमंड फ्रायड ने दुनिया को फ्रायडियन आइसबर्ग से परिचित कराया, जो मानव मन और मानस को बहतर ढंग से समझाने के लिए एक रचनात्मक साइदश्य है। (चित्र 1)



चित्र 1 फ्रायडियन आइसबर्ग

“मन एक हिमखंड की तरह है जो पानी के ऊपर अपने एक सातवें हिस्से के साथ ही तैरता है।” -

सिगमंड फ्रॉयड

Image Credit: Photo by [SIMON LEE](https://unsplash.com/) on <https://unsplash.com/> (edited by author)

मनोविश्लेषण के संस्थापक सिगमंड फ्रॉयड हैं। उन्होंने एक रोगी और एक मनोविश्लेषक के बीच के संवाद के माध्यम से चेतक और अचेतक मानस की विकृति का आकलन और उपचार करने के लिए

एक लाक्षणिक और क्लीनिकल विधि तैयार की। सामाजिक तंत्रिका विज्ञान (जो कि एक उभरता हुआ क्षेत्र है), एफ़एमआरआई तकनीकों का उपयोग करके दर्शाता है कि मन का अचेतन हिस्सा हमारे नियंत्रण और जागरूकता के पार है। इसी के कारण यह विचार कि अचेतन मन चेतन मन की मात्र छाया है असत्य दर्शाया जाता है। [1]। इसलिए, स्पष्ट है कि संवाद जैसे सरल खयाल हमारे दिमाग के अचेतन हिस्से को समझने के लिए काफ़ी नहीं हैं। यह विचार पारंपरिक फ्रायडियन दृष्टिकोण से बहुत अलग है, जिसके अनुसार अचेतन मन केवल भावनात्मक कारणों के वजह से छिपा हुआ है और आत्मनिरीक्षण के द्वारा सामने लाया जा सकता है। [2]

आधुनिक तंत्रिका विज्ञान और चेतना

सामाजिक तंत्रिका विज्ञान तीन क्षेत्रों के मिलन से बना है - सामाजिक मनोविज्ञान, (जो लोगों की बातचीत की प्रणाली का विज्ञान है) संज्ञानात्मक मनोविज्ञान (लोगों के विचारों का विज्ञान), और तंत्रिका जीव विज्ञान (जो स्वयं शरीर रचना विज्ञान, शरीर विज्ञान, और मस्तिष्क की जैव रसायन का विज्ञान है)। इस आधुनिक दृष्टिकोण ने मानव मन की हमारी समझ को अच्छी तरह से स्पष्ट कर दिया है कि व्यक्तिकि अब हम आणविक तंत्र और तंत्रिका मार्गों को दिखे जाने वाले व्यवहारों से जोड़ने में सक्षम हो गए हैं। [2]। दृश्य और श्रवण जैसी हमारी संवेदी धारणाएं स्वाभाविक रूप से लोगों और स्थितियों के बारे में हमारे खयाल जैसे हमारी सामाजिक धारणाओं के समान हैं। यह दोनों अचेतन मन से प्रभावित होती हैं, जो एक छान्ती की तरह काम करता है जिससे हमें अपने आसपास की दुनिया की एक स्पष्ट तस्वीर मिलती है। इसके बिना हमारे द्वारा महसूस होने वाला सब कुछ हमारे दिमाग पर बाढ़ की लहर की तरह आकर हमें कुछ भी बूझने नहीं देता। [2]।

चेतना और अचेतन मन साथ-साथ काम करते हैं। उदाहरण के लिए, 'पुस्तक' शब्द सुनने पर चेतना आपको बताती है कि यह साथ बधे हुए पृष्ठों का एक समूह है जिस पर कुछ लिखा हुआ है। अचेतन मन हमें पहले पढ़ी गई सभी पुस्तकों के चित्र दर्शाता है, उनकी कहानियों की याद दिलाता है, और शायद हमारे पसंदीदा उपन्यास को पढ़ने की यादों को भी तुरंत सामने ला देता है।

चेतना अचेतन मन को काढ़ू में रखती है और उसे भटकने नहीं देती। यदि अचेतन मन घोड़ा है, तो चेतना उसे बांधने वाली रसी है। इसलिए हम अपने दिनर्चयों को बिना भटक पूरा कर पाते हैं। चेतना अचेतन मन द्वारा दर्शाए हुए विचारों और कल्पनाओं को दिशा और संदर्भ देती है। इसके बिना अचेतन मन भटकते हुए आवारा की तरह होता।

चेतना के सिद्धांत

चेतना को लंबे समय तक रहस्यमय माना जाता था जिसका कोई संरचनात्मक या तंत्रिका-विज्ञान आधार न हो। चेतना का वैज्ञानिक अध्ययन केवल 20वीं शताब्दी में शुरू हुआ। तब से मस्तिष्क की सचेतना का अन्वेषण चार मुख्य सैद्धांतिक दृष्टिकोणों के आधार पर चला है:-

- ग्लोबल वर्कस्पेस थिओरी
- हायर ऑर्डर थिओरी
- इंटीग्रेटेड इनफार्मेशन थिओरी
- री एंटी तथा प्रेडिकिटर प्रोसेसिंग थिओरी [3]।

पहले दो चेतना के कार्यात्मक पहलुओं के बारे में हैं, और बाकी दो चेतना के अनुभी पहलुओं को देखते हैं [4]। संरचनात्मक रूप से यह सारे दिमाग के अलग अलग हिस्सों के बारे में हैं, पर सेरेब्रल कॉर्टिक्स ही 'चेतना की सीट' मानी जाती है। मिडब्रेन रैटिकुलर फार्मेशन और थलमिक नुक्लिअइ गैटिंग जैसे कार्य करते हैं। [5] कुछ प्रीफ़ॉन्टल क्षेत्रों (मुख्य रूप से ऑर्बिटोफ्रॉन्टल कॉर्टिक्स और एंटीरियर सिंगुलर कॉर्टिक्स) के इंटाक्रैनियल इलेक्ट्रोक्लिंसिंग इलेक्ट्रोक्लिंसिंग (आईएलसी) ने सचेत अनुभव की गड़बड़ी पैदा की गई है। यह सार्वित करते हैं कि ये क्षेत्र भी एक भूमिका निभाते हैं [6]।

2004 में गिउलिओ टोनोनी द्वारा प्रस्तुत इंटीग्रेटेड इन्फोर्मेशन थोरी एक प्रभाव-से-कारण (इफ़ेक्ट टू कॉज़ि) दृष्टिकोण है। यह चेतना और उसके गुणों (जिसे एक्सिसेंस कहते हैं) के अनुभव से पीछे चलते हुए भौतिक प्रणालियों के गुणों (जिसे “पोस्टुलेट” कहा जाता है) तक जाने की कोशिश करता है जो इन एक्सिसेंस को समझा सकें। [7]। यह दावा करता है कि चेतना जानकारी को एकीकृत करने वाली क्षमता पर निभर है और यह चेतना मुख्य रूप से मस्तिष्क में एक पश्च कॉर्टिकल “हॉट ज़ोन” में मौजूद होती है (जिसमें पारिएटल, टेम्पोरल व ऑक्सिपिटल लोब के हिस्से शामिल हैं)।

यह सिद्धांत क़ाइ मेट्रिक (यह दर्शाता है कि एक प्रणाली अपने भागों के योग से कितनी अधिक है) के आधार पर चेतना की संख्या देता है। इसलिए, यह बताता है कि चेतना क्या है, यह कहाँ से आती है, और किसी भी व्यक्ति में इसका कितना हिस्सा मौजूद है। [8]।

इस सब के दौरान ग्लोबल न्यूरोनल वर्कस्पेस (जीएनडब्ल्यू) सिद्धांत, मस्तिष्क को एक ब्लैकबोर्ड की तरह दिखाती हैं— एक ऐसा स्थान जहाँ अलग-अलग संज्ञानात्मक संस्थाएं चेतना के उत्पादन के लिए बातचीत कर सकती हैं। जीएनडब्ल्यू सिद्धांतों के बारे में सोचने का एक आसान तरीका है कि सचेत अवस्थाएं अचेतन अवस्थाओं की तुलना में मस्तिष्क में ज्यादा “प्रसिद्ध” होती हैं। अर्थात्, वे मस्तिष्क में अधिक व्यापक रूप से प्रसारित होती हैं और संज्ञानात्मक प्रक्रियाओं के एक विशाल जाल को प्रभावित करती हैं। फंक्शनल ब्रेन इमेजिंग इस परिकल्पना के लिए सबूत देती हैं और दर्शाती है कि संचेतनाष्ट व्यापक कॉर्टिकल गतिविधि (विशेष रूप से फ्रंटोपेरिएटल और मीडियल टेम्पोरल क्षेत्रों में) से जुड़ा है, और दूसरी और बेहोश स्थितियां मस्तिष्क में केवल स्थानीय क्षेत्रों को सक्रिय करती हैं। गहरी नींद, पूरे शरीर का एनेस्थीजिया और कोमा जैसी अचेतन अवस्थाएं फ्रंटोपेरिएटल क्षेत्र में चयापचय गतिविधि में काफ़ी कमी दिखाती हैं। [9]।

आखिर में हमारे पास प्रेडिक्टिव प्रोसेसिंग (पीपी) का सिद्धांत है। इसका एक अत्योक्तम उदाहरण हर रोज़ होने वाली घटनाओं में है— जैसे कि ताल में हाथपैर हिलाना। यहाँ, हमारा मस्तिष्क ताल को पहचानता है, इस जानकारी को अगले बीट के समय का अनुमान लगाने के लिए करता है, और मोटर आउटपुट अर्थात् पैर के हिलाने का उत्पादन करता है। माना जाता है कि यह अनुमान लगाने में टॉप-डाउन, बॉटम-अप और, मल्टीडायरेक्शनल सिग्नलिंग [11] सभी शामिल होती हैं। प्रेडिक्टिव प्रोसेसिंग अपने आप में चेतना का सिद्धांत नहीं है, पर सुझाया गया है कि यह ध्यान देने के क्रिया में एक मुख्य कार्य करता है और चेतना के कुछ कार्यों और सिद्धांतों पर एक बेहतारीन दृष्टिकोण दे सकता है। [12]

सपने देखना और सचेतना

कई सिद्धांत सचेत और अचेतन के बीच के अंतर को समझाने की कोशिश करते समय इसे सपनों के साथ जोड़ने की कोशिश करते हैं। परंतु हम अभी भी अनिश्चितता से नहीं जानते कि यह सच है। सत्य संभवतः इन सिद्धांतों के कुछ-कुछ हिस्सों से भी बना हुआ रह सकता है। महत्वपूर्ण सिद्धांतों में से एक इस प्रकार है:-

जब हम सो रहे होते हैं तो हमारा चेतन मन भी सुप्त होता है। इसी समय अचेतन मन नियंत्रण लेता है और स्वतंत्रता से धूमता है। अब, क्योंकि अचेतन मन केवल पहले से मौजूद यादों और अनुभवों तक ही पहुंच सकता है, यह उन यादों के टुकड़ों को संकलित करके उनकी प्रदर्शनी लगाता है अर्थात् एक सपने में मिलाता है। जग जाने के बाद चेतना वापस नियंत्रण ले लेती है और उन सपनों को संदर्भ देने की कोशिश करती है, जिस के आधार पर हमें कुछ समझ में आते हैं और याद रहते हैं, और दूसरे नहीं। [13]।

अब तक आपको यह स्पष्ट हो ही गया होगा कि सपने देखने के लिए वेतना की अनुपलब्धता अनिवार्य है। इसी लिए हम सोते समय हरदम सपने नहीं देखते हैं। हमारे नींद में, रेम (रैपिड आई मूवमेंट) होते

समय ही सपने देखे जाते हैं, जब हमारा मन चेतना से विमुक्त होता है। हमारा शरीर भी ध्यान रखता है कि हम अपने सपनों को हकीकत में ना करें। यह हमारी रक्तचाप को कम करके, हमारे हृदय और सांस लेने की गति को कम करके इत्यादि करके करता है। आश्वर्य की बात है कि मस्तिष्क की गतिविधि निरंतर बनी रहती है और जब हम जगे होते हैं तो इससे भी अधिक बढ़ सकते हैं! [14]।

व्या आपके साथ कभी ऐसा हुआ है कि आप एक कठिन प्रश्न के बारे में सोचते-चोचते सो जाएं और अगली सुबह उसका उत्तर आपको तुरंत सूझ जाए? यह आपके अचेतन मन का काम है, जो चीजों को एक साथ जोड़ता और चीजों को अलग नज़रिए से देखता है। शोध से यह भी पता चला है कि सपने रचनात्मकता को बढ़ा भी सकते हैं [14]। ऐसा इसलिए हो सकता है क्योंकि रेम तब होता है जब चिंता-उत्सरण करने वाला हार्मोन नॉरएफेनालाईन हमारे शरीर में सबसे कम मात्रा में होता है। यह अचेतन मन को महत्वपूर्ण यादों को समेटने के लिए एक शांत वातावरण देता है जिसे हमारे चेतन मन ने दबाने की कोशिश की थी।

‘ऐसा कहा जाता है कि समय सभी घावों को भर देता है, लेकिन मेरे शोध से पता चलता है कि सपने वाली नींद में बिताया गया समय ही घावों को ठीक करता है। - डा मैथ्यू वॉकर

मन और चेतना के बारे में हम जो कुछ भी जानते हैं (या जो हम सोचते हैं) कि हम जानते हैं, सब ध्यान में रखते हुए, हर दिन नए सिद्धांतों का प्रस्ताव किया जाता है। व्या सच्चाई मौजूदा सिद्धांतों में ही छुपी है या नहीं, समय ही बताएगा!

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बेहोशी का तंत्रिका विज्ञान The Neuroscience of Fainting

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संक्षेप

तनावपूर्ण स्थितियों में हम अक्सर बेहोश पड़ जाते हैं। यह मस्तिष्क में रक्त की आपूर्ति में कमी के कारण होता है। जब मस्तिष्क के न्यूरोन्स अपनी आवश्यक मात्रा में ग्लूकोज और ऑक्सीजन नहीं पाते, तो वे थोड़ी देर के लिए बंद हो जाते हैं, जिससे इंसान बेहोश हो जाता है। रक्त की आपूर्ति में इस कमी के कारण कई हो सकते हैं—परिस्थितियों के कारण या किसी रोग के कारण।

शुरुआत

धड़ाम!

“वह बेहोश हो गया है? स्कूल के पहले दिन?” खुसफुसाहट शुरू हो जाती है। हम सभी जानते हैं कि क्या हुआ है।

हम सब जानते हैं कि बेहोशी क्या होती है, पर क्या हम जानते हैं कि उस समय हमारे दिमाग के अंदर क्या चलता है? शायद नहीं। यहीं कारण है कि कई वैज्ञानिक यह जवाब देने की कोशिश कर रहे हैं। शरीर को टीवी की तरह समझें। अब, कल्पना करएं कि इसका प्लग ढीला हो गया है। प्लग कभी भी गिर सकता है, और टीवी बंद हो सकता है। बेहोशी कुछ इसी प्रकार है। डॉक्टर बेहोशी को इस प्रकार परिभाषित करते हैं— थोड़ी देर के लिए चेतना में नुकसान जो फटाफट आता और निकल जाता है। इसका डाक्टरी में नाम “सिकोपी” है। इसे फिर चलाने के लिए प्लग को वापस लगाना पड़ता है।

हमारे मस्तिष्क को ऊर्जा रक्त से आती है, जो मस्तिष्क की कोशिकाओं के लिए शर्करा और ऑक्सीजन लाती है। इसी खुन की कमी बेहोशी का मुख्य कारण है। रक्त की यह कमी रक्तचाप में गिरावट, या कम या अनियमित दिल की धड़कन के कारण हो सकती है, जो मस्तिष्क में रक्त के प्रवाह को कम कर देती है। कभी-कभी बेहोशी कुछ अन्य लक्षणों के साथ भी आती है जैसे कि चक्कर आना, पीली त्वचा, शरीर की कमजोरी, ठंड लगना, पसीन लूटना, धूंधली दृष्टि, दृश्य बनाए रखने और मानसिक ध्यान देने में कठिनाई, स्थिति बनाए रखने में विफलता, आदि। इनमें से सभी आने वाले बेहोशी का संकेत दे सकते हैं। [1]

बेहोशी के कारण और प्रकार

बेहोशी का सबसे आम कारण दिल की धड़कन है— विशेषतः यह कितना तेज़ या धीमा धड़कता है। जब दिल अपनी नियमित लब-डब ताल से नहीं धड़कता है (जिसे अरिथमिया कहते हैं) तो इस से अन्य अंगों को कम ऑक्सीजन और ग्लूकोज प्राप्त हो सकते हैं। क्या आपको स्निकर्स का ऐड याद है? जिसमें भूखा एम। एस। धोनी चिड़चिड़ा हो जाता है। मस्तिष्क के लिए ऐसी ही स्थिति बेहोशी है। जब मस्तिष्क के न्यूरोन्स ग्लूकोज और ऑक्सीजन के भूखे होते हैं, तो वे बंद हो जाते हैं, जिससे व्यक्ति बेहोश हो जाता है।

एक और प्रकार की बेहोशी स्थिति के प्रति रिफ्लैक्स पर आधारित है। मकड़ियों से डरने वाले दोस्त के बारे में सोचें। जब वे अपने बगल की दीवार पर एक को रेंगते हुए देखता है, तो क्या आप अनुमान लगा-

सकते हैं कि वह क्या करेगा? यहां, शरीर रक्त परिसंचरण को नियंत्रित नहीं कर पाता जिसके कारण हमारे मस्तिष्क के न्यूरोन्स में गलकोज और ऑक्सीजन की मांग रह जाती हैं। जब यह पूरी नहीं होती है तो मस्तिष्क बस रुक जाता है, स्टेंडबाय मोड में चला जाता है। इस प्रकार की बेहोशी की उप-श्रेणियाँ हैं - जैसे सामान्य बेहोशी, और स्थितिजन्य बेहोश जो उसके कारणों [2] पर आधारित हैं।

सामान्य बेहोशी (वासोवागल सिंकोपी) भावनात्मक और शारीरिक संकट के कारण होता है। फिल्म की वह अभिनेत्री जो यह सुनकर फर्श पर गिर जाती है कि उसके प्रेमी का एक्सीडेंट हो गया है, या वह लड़का जो यह जानकर बेहोश हो जाता है कि उसके पास एक जुड़वां है, या कोई जो बहुत देर तक धूप में खड़े रहने के कारण बेहोश हो जाता है, वासोवागल बेहोशी से पीड़ित हैं। इस स्थिति में भावनाओं का तनाव या असहनीय शारीरिक तनाव के कारण रक्तचाप गिर जाता है।

केवल विशेष परिस्थितियों या गतिविधियों में बेहोशी को सिचुएशनल बेहोशी या सिचुएशनल सिंकोपी कहा जाता है। इसके उदाहरण कुछ ऐसे हैं— आप कई मैराथन में भाग लेते हैं, और हर बार फ़िनिश लाइन को पार करते ही आप बेहोश हो जाते हैं। एक बहुत ही अंजीबोगरीब मामला था जब एक मरीज को डॉक्टरों के सामने पेश किया गया व्यक्तोंकि वे बेहोश हो गए थे व्यक्तोंकि उन्होंने किसी और को बेहोश [3] देखा था। हैरानी की बात यह है कि इस व्यक्ति की दिल की धड़कन नहीं थी व्यक्तोंकि उनका दिल सचमुच रुक गया था। गनीमत थी कि उन्हें बचाया जा सका। अनूठी ही घटना है!

जब किसी की मौजूदा रोग होता है, विशेष रूप से न्यूरोलॉजिकल रोग, बेहोशी उनके लिए काफ़ी सामान्य घटना होती है। [4] ।

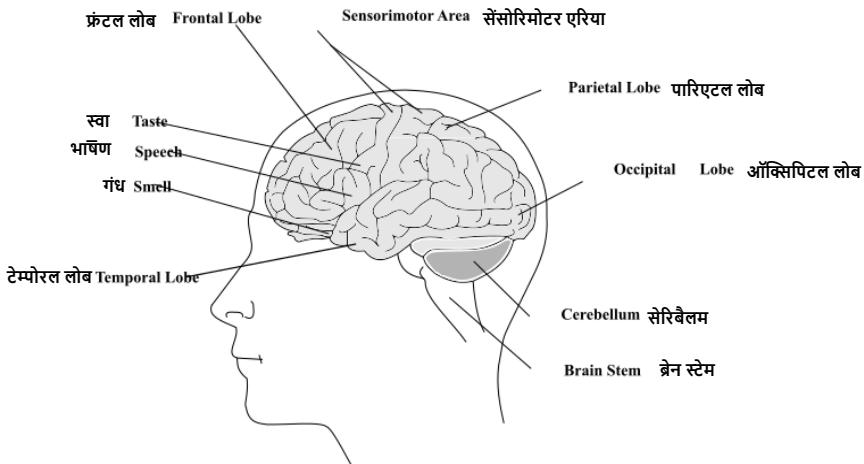
मस्तिष्क में ऐसा क्या चलता है जो हमें बेहोश कर देता है? वैज्ञानिकों का एक मत नहीं है, लेकिन उनके पास कुछ परिकल्पनाएँ जरूर हैं।

सेरोटोनिन एक हार्मोन है जो नीद, मनोदशा और एक न्यूरॉन से दूसरे न्यूरॉन तक संकेतों को पहुंचाने के लिए जिम्मेदार है। शरीर रासायनिक रूप से सेरोटोनिन को मेलातोनिन नामक हार्मोन में बदल देता है जो नीद संबंधित है। नीद भी एक प्रकार की बेहोशी है। यह जानते हुए बेहोशी में सेरोटोनिन की भूमिका को नजरअंदाज नहीं किया जा सकता है। सेरोटोनिन पैरासिम्पेथेटिक तंत्रिका तंत्र (पैरासिम्पेथेटिक नर्वस सिस्टम) में भी लागू है। सिम्पेथेटिक और पैरासिम्पेथेटिक तंत्रिका तंत्र क्रमशः तनावपूर्ण स्थितियों या लड़ाई या उड़ान स्थितियों और शांत स्थितियों के लिए जिम्मेदार होते हैं। [6] । दूसरे शब्दों में, सेरोटोनिन के स्तर में अचानक बदलाव से दिल की धड़कन बहुत कम हो जाती है। बेहोशी में सेरोटोनिन के असर पर शोध चालू है।

हमने यह भी देखा है कि तनाव [7] बेहोशी में योगदान दे सकता है। 2009 में किए गए शोध में सामान्य बेहोश [8] के इलाज पर मनोवैज्ञानिक और सामाजिक भलाई के प्रभाव का सर्वेक्षण किया गया। उन्होंने पाया कि मानसिक स्वास्थ्य विकारों से प्रभावित लोगों या गहरे संकट में रहने वाले लोगों को बार-बार बेहोशी आती है, और उपचार के लिए भी अच्छी प्रतिक्रिया नहीं होती है। उसी वर्ष, एक दूसरे अध्ययन में रोगियों को बार-बार बेहोशी के दौरे [9] के साथ देखा गया। यहां शोधकर्ताओं ने पाया कि जिन रोगियों में संकट के गंभीर लक्षण थे, वे बेहोश ज्यादा पड़े, जिसका कारण वे समझा नहीं सके। इसका मतलब है कि बहुत बार बेहोशी किसी मनोवैज्ञानिक गड़बड़ी का संकेत हो सकती है, जिसके लिए उपचार की आवश्यकता हो सकती है। इसी अध्ययन में जागरूकता बढ़ाने और उपचार शुरू करने के बाद कुछ रोगियों में सूधार देखा गया। किशोरों के बीच की गई एक जांच से पता चला है कि जो बच्चे बेहोश हो जाते हैं उनके जीवन-सुख उनके साधियों की तुलना में कम होती है। [10] ।

किसी के बेहोश होने के बाद उनके मस्तिष्क के अंदर क्या होता है, इसके बारे में हमें ज्यादा जानकारी नहीं है। एक अध्ययन में बेहोशी के लिए 59 वौलंटर्स पर किए गए एक प्रयोग ने मस्तिष्क को कम ऑक्सीजन की आपूर्ति के प्रभावों का अध्ययन किया (एक ऐसी स्थिति जिसे सेरेब्रल हाइपोक्सिया कहा जाता है) [5] । परिणामों से पता चला कि इस प्रतिवर्ती स्थिति ने लोगों को मतिभ्रम (दश्य और श्रवण दोनों), जमीन पर गिरने और मांसपेशियों (मायोक्लोनस) की अनैच्छिक गति का कारण बना। मस्तिष्क

तरंगों का पता लगाने और उनका विश्लेषण करने के लिए शोधकर्ता ईईजी (मस्तिष्क की विद्युत गतिविधि का एक स्कैन) का भी उपयोग करते हैं ताकि यह देखा जा सके कि बेहोशी की क्रिया के दौरान मस्तिष्क की गतिविधि को कैसे संशोधित किया जाता है। इन झटकेदार गतिविधियों को आमतौर पर पाइयों के चक्रव्यूह द्वारा नियंत्रित किया जाता है जिसे आप अपनी पाठ्यपुस्तकों में देखते हैं, जिसे मस्तिष्क का कोर्टिकल क्षेत्र कहा जाता है (चित्र 1)। इस विशेष प्रयोग में देखा गया था कि, यह झटकेदार गतिविधियां ब्रेनस्टेम (मस्तिष्क को रीढ़ की हड्डी से जोड़ने वाला उभार वाला हिस्सा) द्वारा आ रही थीं। यह पुरानी खबर है, पर 1994 में प्रकाशित होने के बाद इस पर अधिक जांच नहीं की गई है।



चित्र 1 मस्तिष्क के भाग चार लोब (फ्रंटल, पारिएटल, टेम्पोरल व ऑक्सिपिटल) हमारे सेरीब्रल कार्टैक्स को बनाते हैं। ब्रेन स्टेम इसके नीचे से निकल आता है और मेरुरज्जु (स्पाइनल कार्ड) बनकर निकल आता है।

चित्र स्रोत: Ozhangk व <https://openclipart.org/detail/121615/human-brain-by-ozhangk>

लेकिन बेहोश पड़े ही क्यों?

यदि बेहोशी मानव जीवन के लिए इतनी प्रतिकूल और हानिकारक पेश होती है तो ऐसा होता ही क्यों है? तनावपूर्ण स्थितियों को निपटाने के लिए शरीर एक अलग समाधान के साथ क्यों नहीं आया? अजीब बात नहीं है कि हम ऐसे क्षण में बंद हो जाते हैं जब हमें मानसिक रूप से खुद को वापस लड़ने के लिए तैयार करना चाहिए?

एक क्रमविकासवादी वृष्टिकोण से बेहोशी एक समस्या नहीं पर एक अनुकूलन की संभावना है। प्रारंभिक सिद्धांत ब्लड इंजेक्शन इंजरी टाइप फोबिया (बिट्स फोबिया) की बात करता है। एक व्यक्ति तीन प्रकार की प्रतिक्रियाएं दिखाता है: भय, परिहर और बेहोशी, जो एक जीवित वृत्ति के पैतृक मानव में विकसित हो सकती थी। बेहोशी से, उन्होंने किसी अन्य मानव द्वारा गंभीर रूप से घायल होने या मारे

जाने की संभावना को कम कर दिया। इस व्यवहार ने आबादी के उन वर्गों की सेवा की हो सकती है जो युद्ध में शामिल नहीं थे—अक्सर महिलाएं और बच्चे—लेकिन कब्जा और यातना के लक्ष्य थे। बेहोशी ने उन्हें इन उद्देश्यों के लिए बेकार कर दिया, शायद उन्हें बचने दिया। [11] ।

बेहोशी प्राचीन मानव के लिए खतरे और अप्रिय अनुभवों से बचने का तरीका बन गई। ऐसी प्रतिक्रिया देकर उन्होंने एक बहुत ही खतरनाक स्थिति को चकमा देने की संभावना बढ़ा दी। इसका फायदा है कि समय और ऊर्जा की बचत के बजाए से अंततः जीवन [11] की रक्षा होती है।

शोधकार्य से पता चलता है कि खतरे या तनाव के समय महिलाएं पुरुषों की तुलना में अधिक संख्या में बेहोश होती हैं। चिकित्सकीय दृष्टिकोण से यह इस तथ्य से प्रदर्शित होता है कि पुरुषों की तुलना में महिलाओं में रक्तचाप कम होता है। इसका एक बड़ा योगदान कारक सेक्स हार्मोन [12] का नियमन है।

मनोविचार

हालांकि बेहोशी एक गंभीर मुद्दा है, यह एक क्रमविकासवादी तरीका है जिसे जीवन और ऊर्जा के संरक्षण के लिए बनाया गया है, जो कि हमारे इतिहास के अधिकांश के लिए हमारा प्राथमिक लक्ष्य था। परंतु यह आधुनिक समय में शारीरिक, मानसिक और भावनात्मक कमज़ोरी का संकेत है, क्योंकि हमारे जीवन अब शिकारियों और जंगलों से मुक्त हैं। हमने इस अस्तित्व तंत्र को विकसित कर पार कर लिया है, और इसलिए इसका वर्तमानीय लक्ष संकट का संकेत है।

बेहोशी की घटना किसी को समस्या की जड़ को पहचानने और संबोधित करने के लिए प्रेरित कर सकती है। इसलिए, हालांकि बेहोश होना प्राकृतिक है, यह लुप्त समस्या का चिह्न है।



चित्र 2 शेक्सपियर के मुच अडू अबाउट नथिंग के हीरो अपने खिलाफ़ लगाए गए आरोपों को सुनकर बेहोश हो जाती है

चित्र स्रोत: Elmore व <https://journey-and-destination.blogspot.com/2013/09/shakespeare-scenes-in-art.html>

बेहोशी एक सामाजिक घटना के रूप में हमारे जीवन में स्थापित है। विलियम शेक्सपियर के “मच अडू अबाउट नथिंग” में कहानी मुख्तातः भ्रम, बुराई की साजिश और विवाह के इर्द-गिर्द घूमती है, हीरो नामक कहानी की एक नायिका उस पर लग आरोपों से बेहोश हो जाती है (चित्र 2)। विलियम शेक्सपियर बेहोशी का एक प्लॉट डिवाइस के रूप में उपयोग करने वाले कईयों में से एक हैं। यह उस समय के विक्टोरियन समाज में प्रवलित सांस्कृति का प्रतिबिंब था। उदाहरण के लिए कभी-कभी विशेष परिस्थितियों में बेहोश होने की अपेक्षा भी की जाती थी।

मध्ययुगीन युग में सांस्कृतिक इतिहास में भी बेहोशी का अपना स्थान है। न केवल आरोपों पर, बल्कि प्रेम, मृत्यु, इत्यादि पर भी लोगों के बेहोश होने के उदाहरण हैं। चार्ल्स डिकेन्स के ब्लॉक हाउस में नायिका लैडी डेडलॉक संभवतः अपने अतीत हेतु बेहोश होती है, जिसे उसने भूलने और छिपाने के लिए कड़ी मेहनत की।

नारीवादी लेखिका एंजेला कार्टर के समीक्षकों द्वारा प्रशंसित उपन्यास, ‘द ब्लॉक चैंबर’ में बेहोशी का एक उत्तम वर्णन है। वे लिखती हैं, “उस खूनी कक्ष के भ्यानक रहस्योदयाटन के बाद, उसके चेहरे पर मार्मिक भाव थी जिसने मुझे बेहोश कर डाली।”।

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Malayalam

അസ്യകാഴ്ച, മനുഷ്യ മനസ്സിന് റെ പ്രവർത്തനങ്ങളെ എന്നേനെ കാണാൻ സഹായിക്കുന്നു

Blindsight, and how it helps us see the workings of the human mind

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Translated from English to Malayalam by Arya Jayaraj, Marian Michael and Nuha Fathima

സംഗ്രഹം (SUMMARY)

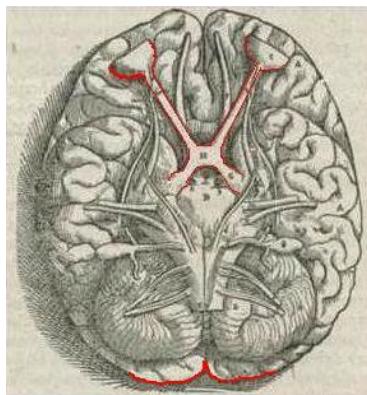
മന്ത്രിപ്പക്കം എന്നേനെ വിവരങ്ങൾ കാര്യക്രമം ചെയ്യുകയും ലോകത്തെ ശ്രദ്ധിക്കുകയും ചെയ്യുന്നു എന്നതിനെ പറ്റി സകിർണ്ണമായ വികിട്ടണം നൽകുന്ന ഒരു ഉപജാപകപൂർവ്വമായ വൈകല്യമാണ് അസ്യകാഴ്ച (Blindsight). നാഡിവൃദ്ധി വിജ്ഞാനിയം, നാഡിശാസ്ത്രം, മനോരാഹപഠനം എന്നിവയുടെ പലിയ ചോദ്യങ്ങളിലേക്കും അവധ്യം കണ്ണഭരണത്തിലുകളുടെ അനന്തരഹമലങ്ങളിലേക്കും കൂടുതൽ ആളുനിറങ്ങാൻ അസ്യകാഴ്ച പോലുള്ള നാഡിവൃദ്ധി വൈകല്യം ഒരു തുടക്കം നൽകുന്നു. ഈ ലേഖനം അസ്യകാഴ്ചയെക്കുറിച്ചുള്ള ആശയവും മനുഷ്യ മന്ത്രിപ്പക്കത്തെ നന്ദായി മനസ്സിലാക്കാൻ നമ്മുടെ സഹായിക്കുന്നതെന്നെന്നെന്നും സംഗ്രഹിക്കുന്നു.

മിസ്റ്റർ എൽ (സപ്രകാര്യതയ്ക്കായി പേര് മരച്ചുവെച്ചിരിക്കുന്നു) തന്റെ ചുരുക്ക പഥതുവരമന്ത്രജ്ഞ ഒരു വ്യക്തിക്ക് കൈമാറുന്നു. പൂർണ്ണമായും അസ്യനായ അധികാർക്ക്, എല്ലാ അവധ്യങ്ങൾക്കും തന്ത്രങ്ങൾ നിറഞ്ഞ ഒരു മുറിയിൽ പരസ്പായമില്ലാതെ നടക്കേണ്ടി വരുന്നു. ഇത് ഒരു കുറവായ തമാശയായി തോന്തുന്നുവെകിലും ഗവേഷകർക്ക് അവർ സന്ദേഹിച്ചത് എന്നാണെന്ന് ഇതിൽ നിന്ന് മനസ്സിലായി: മിസ്റ്റർ എൽ മുറിയിലും "ബോധപൂർവ്വം" താൻ ഓക്കെല്ലും അസ്യന്നൂ എന്ന മട്ടിൽ തന്ത്രങ്ങളിലും സഖ്യരിച്ചു [!].

നിങ്ങളുടെ കണ്ണുകളും ഒരു മുറിക്ക് ചുറ്റും നീങ്ങാൻ ആവശ്യപ്പെടുകയാണെങ്കിൽ, നീങ്ങൾ ക്രമരഹിതമായി, കൂദിയുന്നത് തന്ത്രങ്ങൾ ഒഴിവാക്കാൻ ശ്രമിച്ചുവെക്കാണ് നീങ്ങും. സ്റ്റോക്ക് മുലം പ്രേമറി വിഷയത്ത് കോർട്ടക്സിൻ (primary visual cortex) ക്ഷതിമേറ്റ മിസ്റ്റർ എൽ നിന്ന് അവധ്യ വളരെ യഥാർത്ഥ അസ്യതയാണ്. വർഷങ്ങളായി തന്റെ കണ്ണുകൾ മുടിക്കൊട്ടിയിരിക്കുന്നതിനാൽ, ഇതുപോലുള്ള പരിക്ഷണങ്ങളിൽ താൻ തികച്ചും യാദുശ്വികമായ പാതയിലും

സാമ്പത്തികമുകയാണെന്ന് വിശ്വസിക്കുന്നു, എന്നിട്ടും നിഷ്പദയാസം തടസ്സങ്ങൾ മറിക്കുന്നു. [2].

ഈ അവസ്ഥയുടെ അടിസ്ഥാനത്തിലെകില്ലോ കാര്യങ്ങൾ കാണാൻ മന്തിഷ്കം നമ്മുടെ സഹായിക്കുന്നതെങ്കണ എന്ന്. ഈ ചിത്രത്തിൽ ചിത്രീകരിച്ചിരിക്കുന്ന (ചിത്രം 1), ചിത്രത്തിൽ, കാഴ്ചയിൽ ഉൾപ്പെട്ടിരിക്കുന്ന സർക്കുട്: ‘ദൃശ്യ പാത’ നമ്മുടെ കാണാൻ സാധിക്കും. രേഖിന്യാസിൽ (retina) രൂപംകൊള്ളുന്ന ചിത്രം വിവരിതനം ചെയ്യുന്ന പ്രേരണകൾ നേത്രനാഭങ്ങളിൽ നിന്ന് ഒപ്പറിക്കുന്ന നാഡി (optic nerves) വഴി പുറത്തേക്ക് പോകുന്നു, ഒരു കേവിൽ പോലെ നിരവധി വയറുകൾ (ഈ സാഹചര്യത്തിൽ, “നൃഗണാഭങ്ങൾ” എന്ന് പിളിക്കുപ്പെടുന്ന നാഡിക്കൊണ്ടുണ്ട്) ഒരു ലക്ഷ്യസ്ഥാനത്തേക്ക് സന്ദേശങ്ങൾ കൊണ്ടുപോകുന്നു. ഒപ്പറിക്കുന്ന മായിൽ നിന്ന് ഒപ്പറിക്കുന്ന തെരസുകൾ കോണ്ട് ചെയ്യുകയും, തുടർന്ന് തലച്ചുറാറിന്റെ വിവിധ ഭാഗങ്ങളിലേക്ക് നിശ്ചയിക്കുകൾ കൊണ്ടുപോകുന്നു. ഒപ്പറിക്കുന്ന ലഘുവേദകളായി തുടരുകയും ചെയ്യുന്നു.



ചിത്രം 1: ദിപ്പുമാനിക്കോർപ്പറിസ് ഹാബേിക്ക ലിബ്രി സെപ്റ്റം എന്ന തന്റെ പുസ്തകത്തിൽ നിന്ന് ആര്യാധികാരിയാം എസാലിയസിന്റെ രേഖാചിത്രം.

ദൃശ്യ പാതയിലെ എത്ര (പ്രത്യന്ധും ആവേശങ്ങളും) ഒഴുക്കിനെ തെയ്യുന്നു, ഇത് കാഴ്ചയുടെ വിവിധ ഭാഗങ്ങളിൽ അധിക ഉണ്ടാക്കുന്നു. ഇതിന്പുറമുള്ള നിശ്ചാൽ സംപ്രകാശണ (signal transmission) പാതയിലെ പ്രശ്നങ്ങൾ പരിശോഖിക്കുന്നത് അതു എളുപ്പമല്ല.

മനുഷ്യ മന്തിഷ്കം പരിണാമത്തിന്റെ അന്ത്യത്തമാണ്. ഈ സഹസ്രാബ്ദങ്ങളായി പികസിച്ചു വന്നതാണ്.. ജീവനുള്ള കോണ്ടജാലങ്ങളുടെയും വൈദ്യുതരാസ

പ്രവർത്തനത്തിന് റെയും രൂപകമായ ഒരു അംബരചുവിയെപ്പാലെ മാനുഷിക നാഗരികത്വയെക്കുറിച്ച് നമൾ വിസ്മയിരിത്തരായ എല്ലാറിന് റെയും മേഖലയെള്ളുവ സ്പർശിക്കുന്നു: യാർമ്മികത, സംഗ്രിതം, ഭാഷ, സർജ്ജാന്തകത, അനുകംഡ തുടങ്ങി എല്ലാം. കാലക്രമേണ സംഭവിക്കുന്ന പരിബന്ധം (പ്രക്രിയയുടെ പ്രതിഫലം) നമ്മുടെ മന്ത്രികൾ വിന്ദിക വിവിധ ഭാഗങ്ങൾ പ്രവർത്തനങ്ങൾക്ക് പ്രാധാന്യമാണ്, കൂടാതെ തലച്ചോറിന്റെ ഭാഗങ്ങൾ അല്ലെങ്കിൽ സർക്കൂട്ടുകൾ (circuit) ശ്രസ്യം പോലെയുള്ള കൂടുതൽ പ്രാകൃത ഉത്തരവ് പ്രവർത്തനങ്ങളിൽ എല്ലാം പ്രാധാന്യമായി നിയന്ത്രിക്കുന്നത് നേര്ണ്ണിനും മന്ത്രികൾക്കും അടുത്തുള്ള "താഴ്ന്ന പ്രദേശങ്ങൾ" (lower areas) ആണ്[3]. അതിൽ നിന്ന് ഭക്ഷണം, വികാരങ്ങൾ, എഴുതൽ, ഭാഷ തുടങ്ങിയ പ്രവർത്തനങ്ങളിലേക്ക് നാം ഉയരുന്നു. അവ ഓരോനും അവയുടെ സകിൾഭൂതയും (പ്രാധാന്യവും അടിസ്ഥാനമാക്കി തലച്ചോറിന്റെ "ഉയർന്ന മേഖലകളിൽ" (higher areas) ക്രമാനുഗതമായി പ്രവർത്തിക്കുന്നു.

അത് മനസ്സിൽ വെച്ചുകൊണ്ട്, നമൾ അസ്ഥാപ്തചയിലേക്ക് മടങ്ങുന്നു. എന്നുമുകളാണ് "ബോധപൂർവ്വം" എന്ന വാക്ക് വളരെ പ്രധാനമാക്കുന്നത്. നമൾ ചെയ്യുന്ന മികവാറും എല്ലാ കാര്യങ്ങളും "സ്വദേശയാ" ആണെന്ന് വിശ്വസിക്കാൻ ആഗ്രഹിക്കുന്നു - നമുകൾ നിയന്ത്രിക്കാൻ കഴിയുന്ന പ്രവർത്തനങ്ങളെ വിവരിക്കാൻ നാഡിശാസ്ത്രം ഉപയോഗിക്കുന്ന ഒരു വാക്ക്. എന്നിരുന്നാലും, ശബ്ദാശ്വാസം സ്വദേശയാ ഉള്ള ഒരു പ്രക്രിയയെല്ലാം നമുകൾക്കിടയാണ്: കുറച്ച് സമയത്തേക്ക് ബോധപൂർവ്വം വേഗതയോ ശബ്ദാശ്വാസമോ നമുകൾ മാറ്റാം. പക്ഷേ തലച്ചോറിൽ നിന്ന് നിങ്ങളുടെ ബോധപൂർവ്വവായ ഇടപെടൽ കൂടാതെ അത് തുടരുന്നു. "താഴ്ന്ന പ്രദേശങ്ങളുടെ" പ്രവർത്തനമായതിനാൽ, ഈ വസ്തുത അംഗീകരിക്കാൻ പ്രയാസമില്ല. എന്നാൽ നേരിട്ടുള്ള ബോധത്തിന്റെ ഭാഗമല്ലാത്ത ദൃശ്യം പോലെയുള്ള പ്രവർത്തനങ്ങൾക്കും കാര്യമായ പകുണ്ടന ആശയം വിശ്വസിക്കാൻ അതു എല്ലാപ്പോലെ.

ഇതിനുള്ളിൽ ചില ഉദാഹരണങ്ങളിൽ അരുതേരൽ മൂന്നിനിഷം നിങ്ങൾ ചെയ്യുന്നത് തെന്ന്. നിങ്ങൾ മൂന്ന് വരികൾ വായിക്കുന്നേബാൾ, കണ്ണുകൾ ഇടത്തുനിന്ന് വലത്തോടു വാക്കുകളിലൂടെ ഓടുന്നു. എന്നാൽ ആ ചലനങ്ങൾക്ക് നിങ്ങൾ ഏറകലും പരിശനിക്കാതെ ഒരു മാതൃകയുണ്ട്. "സക്കാഡിക്" (prismacanങ്ങൾ) (saccadic movements) എന്ന് വിളിക്കേണ്ടതുനാണ് മൂന്ന് ചലനങ്ങളുടെ പിന്നിൽ നിരന്തരമായ (processing) (പ്രതികരണവും) (feedback) ഉൾപ്പെടുന്നു. ഇവ വിസുലമായ ഗവേഷണത്തിന് വിധേയമാണ്. വായന വളരെ ബോധപൂർവ്വവായ ഒരു പ്രക്രിയയാണെന്നീക്കിലും, വായനയുടെ ഒരുക്കിനായി പേജിലും കണ്ണുകൾ ചമിസ്റ്റിക്കുന്ന ഉത്തരവാദിത്തം നിന്നേവെറ്റുന്നതിനായി തലച്ചോർ ഉപഭോഗമനസ്സിനെ അനുവരിക്കുന്നു. അതിനാൽ നിങ്ങൾ ഓരോ തവണയും അതിനെക്കുറിച്ച് വിശദമിക്കേണ്ടതില്ല.

ഈ നമുകൾ മൂന്ന് ആശയം അസ്ഥാപ്തചയ്ക്ക് പ്രയോഗിക്കാം. തലച്ചോറിലെ കാഴ്ച തിരിച്ചറിയുന്നതിന്റെ രണ്ട് പാതകളിൽ, "പശയ പാത" കേടുകൂടാതെയിരിക്കുമെന്ന് അനുമാനിക്കപ്പെടുന്നു, മുത് എറ്റവും പ്രാകൃത

ജീവികളിൽ പോലും ഇരയുടെ പാത നിർബന്ധിക്കാനും ചുറ്റുമുള്ള ഭൂപടം വികസിക്കാനും അനുവദിക്കുന്നു - താമര ഇലയിൽ ഇരിക്കുന്ന ഒരു രംഗം അതിന്റെ ഇരയെ നിർബന്ധിക്കുന്നതും അകൃതത് വരുമ്പോൾ അതിനെ വിശുദ്ധിക്കുന്നതും സക്തിപ്പിക്കും. മറ്റൊരുത്ത്, "പുതിയ പാത", നമർക്ക് കാണുന്ന ലോകത്തെ എത്രാണേണ്ണും എങ്ങനെയെന്നും വേർത്തിരിച്ചറിയുന്ന പ്രക്രിയകൾ നടത്തുന്നു, വി.പ്രിൻസ്. രാമചന്ദ്രൻ പരിയുന്നതുപോലെ, പുതിയ പാത അടയുന്നതോടെ, "ദൃശ്യഭോധം കണ്ണടയ്ക്കാൻ" സാധ്യതയുണ്ട് [4]. നിങ്ങൾ എത്രക്കിലും കാണുന്നണ്ടെന്ന് ഭോധപൂർവ്വം അറിയാനോ നിങ്ങൾ കാണുന്നതെന്നതാണെന്ന് അളക്കാനോ നിങ്ങൾക്ക് കഴിഞ്ഞെന്നകില്ല. എന്നാൽ കാഴ്ചയുടെ കുടുതൽ പ്രകട്ടു വരുമ്പോൾ വിചിത്രമായി പരിപാലിക്കേണ്ടുന്നു. ഇത് പറഞ്ഞ സ്ഥിതികൾ, നമക് ഇരുവരെ അസ്ഥാപയെക്കുറിച്ച് പുർണ്ണമായി മനസ്സിലായിട്ടിരുന്ന് താൻ പറയുന്നു - നമർക്ക് ആരംഭിക്കുന്നതെയുള്ളൂ; ഇത് തലശ്ചാറിനെക്കുറിച്ച് കുടുതൽ പരികാശ കഴിയുന്ന സ്ഥലങ്ങളിലേക്ക് നമ്മുടെ ശ്രദ്ധ പിടിച്ചുപറ്റുക മാത്രമല്ല. നിരന്തരം വെള്ളുവിളിക്കുകയും നാഡിശാസ്ത്രത്തിന്റെ തന്നെ അതിരുകൾ തളളുകയും ചെയ്യുന്ന മനസ്സിനെ അലോസറപ്പെടുത്തുന്ന നിരവധി നൃഗാന്ധിജികൾ (പ്രതിഭാസങ്ങളിൽ) ഒന്നാണിത്. ചോദ്യങ്ങളും ഉത്തരങ്ങൾ പോലെ തന്നെ പ്രധാനമാണ്. ശാസ്ത്രത്തിലെ ഭൗതഗതിയിലുള്ള പുരോഗതിക്കും സമാപ്പകാല സാങ്കേതിക പുരോഗതിക്കും ഒപ്പ്, നൃഗാന്ധി (neurology), സൈക്യാട്ടി (psychiatry), സൈക്കോ അനാലിസിസ് (psychoanalysis) തുടങ്ങിയ മേഖലകളുടെ കവലകളിൽ മനസ്സിനേക്കുറിച്ചുള്ള പതംങ്ങൾക്ക് സാക്ഷ്യം പറിക്കുപോൾ നമകൾ ധാരാളം ഉത്തരങ്ങൾ ലഭിക്കുന്നു!

അന്യകഴച്ചയുടെ ഉത്തേവത്തെക്കുറിച്ചുള്ള നമ്മുടെ ശാഹരൈത അഭിമുഖവികരിക്കുന്ന നിരവധി വെള്ളുവിളികളുമായി ശാസ്ത്രജ്ഞൻ ഇപ്പോഴും പോരാടുകയാണ്. ഈ അനേകണണങ്ങളും പ്രക്രിയകളും, എല്ലാ സാങ്കേതിക പുരോഗതികളും പതിറാണ്ടുകളും ഗവേഷണങ്ങൾ ഉണ്ടായിട്ടും, നമുഖ്യ മന്ത്രിക്കൾത്തെക്കുറിച്ച് നമകൾ എന്തെങ്കിലും അറിവ് മാത്രമേ ഉള്ളൂ എന്ന് ആവർത്തിക്കുന്നു. നമ്മുടെ പര്യവേക്ഷണങ്ങൾ തുടർച്ചയായി പുതിയ, ആവേഷകരമായ കണ്ണഭൗതിക നൽകിയിട്ടുള്ളെങ്കിലും, എല്ലാം നമുക്കെന്നിയാം എന്ന ചിന്തയിൽ അവ നമെ തുപ്പിൽപ്പെടുത്തുവും; മറിച്ച്, കുടുതൽ നോക്കാൻ അവൻ നമെ വെള്ളുവിളിക്കണം. എല്ലാത്തിനുമുമ്പായി, "കുടുതൽ അറിവ് നേടുന്നതായും അറിയില്ല എന്ന തിരിച്ചറിവ് എന്നിൽക്കും ഉണ്ടാവുന്നു" എന്ന് സോക്രറ്റീസ് പറഞ്ഞപ്പോൾ അത് സ്വികാര്യതയിൽ ആയിരുന്നില്ല മറിച്ച് നമർക്ക് ശ്രമിച്ചാൽ മാത്രം കാണാൻ കഴിയുന്ന കാര്യങ്ങളിൽ അസ്യത കാണിക്കുന്നതിനെതിരായ ശക്തമായ ധിക്കാരത്തിലായിരുന്നു എന്ന് താൻ വിശദിക്കാൻ ആഗ്രഹിക്കുന്നു.

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ജീവിതശൈലിയുടെയോ പെരുമാറ്റ എടക്കങ്ങളുടെയോ ഫലമായി മന്ത്രിഷ്ക പ്രവർത്തനത്തിലൂണ്ടാവുന്ന മാറ്റങ്ങൾ Changes in Brain Activity in Response to Lifestyle/Behavioural Aspects

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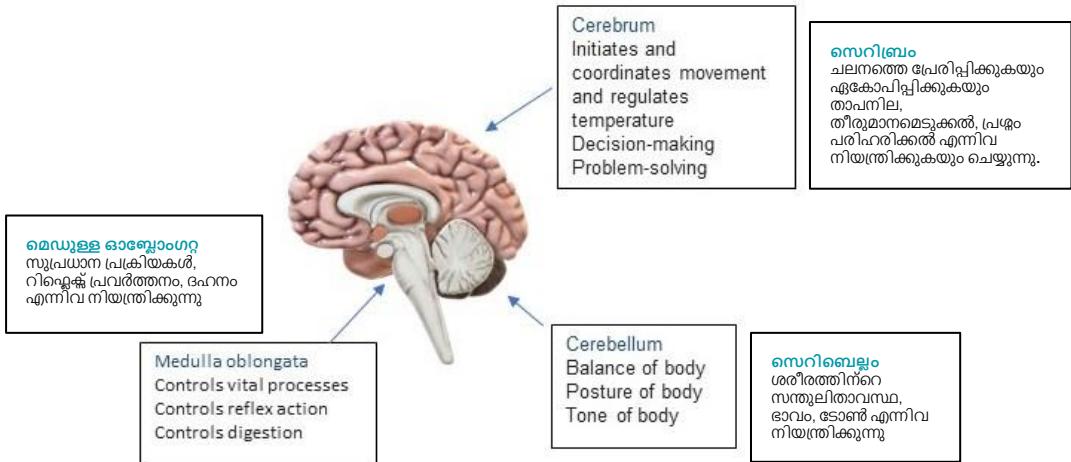
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Translated from English to Malayalam by Nuha Fathima

സംഗ്രഹം (SUMMARY)

തലച്ചോറിനെ 'മാസ്റ്റർ ഓർഗൻ' അമവാ അവയവങ്ങളുടെ നേതാവ് ആയി കണക്കാക്കുന്നു; ശരീരപ്രവർത്തനങ്ങളെ നിർണ്ണയിക്കുന്നത് തലച്ചോറാണ്. ആരോഗ്യകരമായ സാമൂഹിക ഹംപെടലുകൾ, അനുഭവങ്ങളുടെ വൈവിധ്യം, ഭക്ഷണക്രമം, ഉറക്കം എന്നിവ ഉൾക്കൊള്ളുന്ന ആരോഗ്യകരമായ ജീവിതശൈലി നൃറോപ്പാസ്റ്റിറ്റിയിലേക്ക് നയിക്കുന്നു. പൂതിയ സാഹചര്യങ്ങൾക്കും സന്ദർഭങ്ങൾക്കും അനുസരിച്ചു മനുഷ്യർിൽത്തന്നെ പ്രവർത്തനം ക്രമപ്പെടുത്തുവാനുള്ള തലച്ചോറിന്റെ കഴിവാണ് നൃറോപ്പാസ്റ്റിസിറ്റി. അമവാ, പൂതിയ സിനാപ്പറ്റിക് ബന്ധങ്ങൾ രൂപീകരിക്കാനുള്ള തലച്ചോറിന്റെ കഴിവ്. ആരോഗ്യകരമായ ജീവിതശൈലി 'പ്ലാസ്റ്റിക്' ആകാനുള്ള അമവാ സാഹചര്യമനുസരിച്ച് മാറാനുള്ള തലച്ചോറിന്റെ കഴിവിനെ വളരെയധികം സഹായിക്കുന്നു. ഇത് പ്ലാസ്റ്റിറ്റി നിർണ്ണയിക്കുക മാത്രമല്ല, നൃറോണുകളെ കുറച്ചതൽ കാര്യക്ഷമമാക്കാൻ സഹായിക്കുന്നതിലൂടെ ശരീരത്തിന്റെ കഴിവുകൾ വർദ്ധിപ്പിക്കുകയും ചെയ്യുന്നു. പക്ഷെ, ആരോഗ്യകരമായ ജീവിതശൈലി നയിച്ചാൽ മാത്രം പോരാ, പൂതിയ സിനാപ്പറ്റിക് ബന്ധങ്ങൾ രൂപീകരിക്കാൻ ഒരു വ്യക്തി പൂതിയ വൈദഗ്ധ്യങ്ങൾ പറിക്കേണ്ടതുണ്ട്. പ്രായം കണക്കിലെടുക്കാതെ, നാഡിപാതകളെ നിർമ്മിക്കാനും അനുയോജ്യമാക്കാനും പുന്നസംശാടിപ്പിക്കാനും തലച്ചോറിന് ആജീവനാന്ത കഴിവുണ്ട്. ഒരാൾ വളരെ ചെറുപ്പുള്ളതാവെട്ട് പ്രായം ചെന്നതാവെട്ട്, പൂതിയ അറിവുകൾ നേടാൻ പ്രായപരിധികളാനും ഇല്ലോ! (ചിത്രം. 1.)



ച്ചിത്രം. 1. മന്തിഷ്ക്കത്തിന്റെ ഘടന

Image credit: Shivani Pimparkar

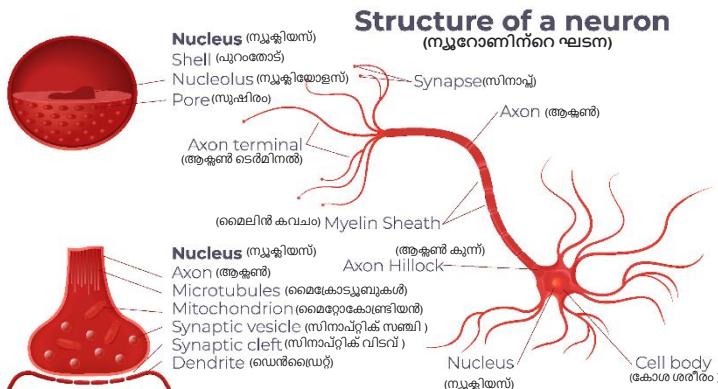
ആരുപ്പം

നിരൂപയി ആദ്യരൂപങ്കമായ നേട്ടങ്ങൾ നേടാൻ ശരീരത്തിന് കഴിയുന്നു . അവ തമാത്രാ തലത്തിൽ എക്കോപ്പിത്തമായി നടക്കുന്ന ഉപാപചയ പ്രതിപ്രവർത്തനങ്ങളിൽ തുടങ്ങി അസ്ഥിക്ക്‌സ്, സാഹസിക കായിക വിനോദങ്ങൾ തുടങ്ങിയ ശാരീരിക പ്രവർത്തനങ്ങളിൽ ചെന്നുനിൽക്കുന്നു. ഈ പ്രക്രിയകളെ നിയന്ത്രിക്കുന്നത് എന്നാണെന്ന് എപ്പോരുകിലും ചിന്തിച്ചിട്ടുണ്ടോ? ഉത്തരം പാളേര വ്യക്തവും എന്നാൽ രസകരവുമാണ്: അതെ, ബെയിൻ അമായ മന്തിഷ്ക്കം എന്നതാണ് ഉത്തരം. അതാണ് എന്നർത്ഥം വരുന്ന പഴയ ഇംഗ്ലീഷ് പദമായ 'ബൈൻ' എന്നതിൽ നിന്നാണ് 'ബെയിൻ' എന്ന വാക്ക് ഉച്ചത്തായി ഉരുത്തിരിക്കുന്നത്. മന്തിഷ്ക്കും നമ്മുടെ ഔദ്യോഗിക്കതി, മോഡ്യൂൾ പ്രവൃത്തികൾ, കാഴ്ച, ശ്രദ്ധാ, വിശ്വസ്ത എന്നുതുടങ്ങിയ എല്ലാ ജീവിത പ്രക്രിയകളെയും നിയന്ത്രിച്ചു ശരീരത്തിന്റെ കേഷമം നിലനിർത്തുന്നു. എന്നിരുന്നാലും, ശരീരത്തെ നിയന്ത്രിക്കുന്നത് മന്തിഷ്ക്കം മാത്രമല്ല; സുഷുംബാ നാഡിയും സമർഥമായി ഉത്തരവാദിയാണ്. ഈ രണ്ടും ചേർന്ന് സെൻസേൻസ് നേർവ്വസ് സിസ്റ്റം (സിപ്പിസ്-എസ്) അമവാ കേന്ദ്രനാഡിവ്യവസ്ഥ രൂപപ്പെടുന്നു.

മന്തിഷ്ക്ക ഘടകങ്ങൾ: മെല്ലുകൾക്കുറി

- തലച്ചോറിന്റെ അടിഖ്യാന പ്രവർത്തന യൂണിറ്റാണ് നൃറോണുകൾ.അമവാ നാഡികോശങ്ങൾ.

- തലച്ചോറിൽ നിന്ന് നാഡികോശങ്ങൾ വഴി രേഖപ്പെടുത്തിയ ആവേഗങ്ങളുടെ രൂപത്തിൽ സൂഷ്ടുചന്ന നാഡിയില്ലെന്നുണ്ട് ശരീരത്തിന്റെ മറ്റ് ഭാഗങ്ങളിലേക്ക് സഞ്ചരണമെന്നുണ്ട്. രണ്ട് നാഡികോശങ്ങളെ ബന്ധിപ്പിക്കുന്ന സന്നിധിയെ സിനോപ്സ് എന്ന് വിളിക്കുന്നു.
- ഏകദേശം 1.3-1.5 കിലോഗ്രാം ഭാരമുള്ള തലച്ചോറിന്റെ അറുപതു ശതമാനം വരെ കൊഴുപ്പുണ്ട്, ഈ മൂലം തലച്ചോർ മനുഷ്യ ശരീരത്തിലെ ഏറ്റവും കൊഴുപ്പുള്ള അവയവമായി മാറുന്നു. ഈ കൊഴുപ്പ് കുറയ്ക്കാൻ കഴിയില്ലെന്ന് ഗവേഷകൾ പറയുന്നു, കാരണം മയലിൻ പോലെയുള്ള തലച്ചോറിന്റെ സമർത്ഥയും പ്രകടനവും നിഖയിക്കുന്ന ഏറ്റവും നിർണ്ണായക രഹസ്യകളിൽ കൊഴുപ്പ് അടങ്കിയിരിക്കുന്നു.
- ബാക്കിയുള്ള 40% തലച്ചോറും പ്രോട്ടീനുകൾ, കാർബോബൈഡേറ്റ്‌സ്, ലവണങ്ങൾ എന്നിവയുടെ മിശ്രിതമാണ്.
- **കേരസ നാഡിപ്പുഹരിതിന്റെ രക്ഷകളും,** നാഡി, കാപ്പിലി ശൃംഖലകൾ 400 ദേശങ്ങൾ (ഏകദേശം: 643 കി.മീ) വരെ നീളുന്നു.(ചിത്രം. 2)



ചിത്രം. 2.: നാഡിപ്പുവസ്ഥയുടെ അടിസ്ഥാന ഘടനാപരവും പ്രവർത്തനപരവുമായ യൂണിറ്റ്

കുറപ്പാട്: macrovector കൂട്ടാബേത് <https://www.freepik.com>

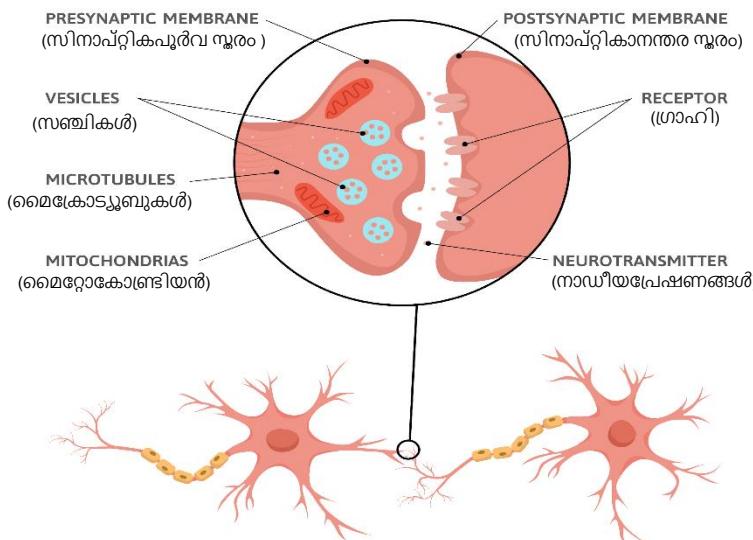
നൃഗോഷ്ഠാസ്ഥിസിറ്റി

നൃഗോഷ്ഠാസ്ഥിസിറ്റി അബ്ലൈറ്റിൽ വൈയിൻ പ്ലാസ്റ്റിറ്റി എന്നത് സിനോപ്സുകൾക്കോ, നാഡികോശങ്ങൾക്കോ അബ്ലൈറ്റിൽ തലച്ചോറിന്റെ മുഴുവൻ ഭാഗങ്ങൾക്ക് പോലും അവയുടെ ഉപയോഗത്തിനുസരിച്ച് മാറാനുള്ള ആജീവനാത

പ്രവണതയാണ്. നൃരോഷ്ട്യിസിറ്റി എന്നത് സിനാപ്പറ്റിക് കൺക്ഷൻകളെ ശക്തിപ്പെടുത്തുകയോ ഭൂർബലപ്പെടുത്തുകയോ അല്ലക്കിൽ ബഹു അനുഭവങ്ങളെ അരിസ്മാമാനി പൂർണ്ണമാക്കുകയോ ചെയ്യുന്നതിനെ സൃച്ചിപ്പിക്കുന്നു. നൃരോഷ്ട്യിസിറ്റി സംഭവിക്കാൻ പല വഴികളുണ്ട്, ആതിൽ പൊതുവായ രണ്ടുണ്ട് ആണ് ആക്സോണൽ സ്പ്രാറ്റിംഗും സിനാപ്പറ്റിക് (പുണിംഗും English terms in bracket?). ഈ ഏതാണെന്ന് നിഖലീകരിക്കാം.

ആക്സോണൽ സ്പ്രാറ്റിംഗ് (AXONAL SPROUTING)

ആക്സോണൽ സ്പ്രാറ്റിംഗിൽ ആരോഗ്യമുള്ള ആക്സോണുകൾ നാധിപ്പുവസ്ഥയിലെ മറ്റ് പാതകളുമായി ബന്ധപ്പെട്ടിരുന്ന പുതിയ നാഡി അറുങ്ങൾ മുളച്ചിരുന്നു. നിലവിലുള്ള ബന്ധങ്ങൾ ശക്തിപ്പെടുത്തുന്നതിനോ നാധിപ്പുവസ്ഥയിലെ കേടുപാടുകൾ തീർക്കുന്നതിനോ പൂർണ്ണമായ പ്രവർത്തനക്ഷമതയിലേക്ക് പുനഃസ്ഥാപിക്കുന്നതിനോ ഇത് ഉപയോഗിക്കാം. (ചിത്രം 3.)



ചിത്രം 3: സിനാപ്പറ്റിക് സംബന്ധിച്ച ഒരു നിർദ്ദേശിക്കാൻ ഉപയോഗിക്കുന്ന ഒരു പ്രതിഫലിക്കാൻ ഉപയോഗിക്കുന്ന ഒരു ചിത്രം

സിനാപ്റ്റിക് പ്രൂണിംഗ് (SYNAPTIC PRUNING)

ഒരു നവജാത ശിശുവിന്റെ മന്തിഷ്കം ആ കുണ്ടിന്റെ ഇള്ളിയങ്ങളിൽ നിന്നുള്ള വിവരങ്ങളാൽ നിറഞ്ഞിരിക്കുന്നു. ഈ വിവരങ്ങൾ എന്നെന്നെയക്കിലും അത് ഗ്രഹിക്കാൻ കഴിയുന്ന തലചേച്ചാറിലേക്ക് തിരികെ കൊണ്ടുവരണം. അങ്ങനെ ആവേശങ്ങൾ തലചേച്ചാറിലോട് ഒക്കമാറാൻ നാധികോശങ്ങൾ പരസ്പരം പുതിയ ബന്ധങ്ങൾ സ്ഥാപിക്കണം.

അടുത്ത ഏതാനും വർഷങ്ങൾക്കുള്ളിൽ, മന്തിഷ്കം അതിവേഗത്തിൽ വളരുന്നു. ഓരോ നാധികോശവും പക്കത പ്രാപ്തിക്കുന്നേയാൽ, അതിൽ ഒന്നിലധികം ശാഖകൾ (വിവരങ്ങൾ അയയ്ക്കുന്ന ആക്ഷേണാണുകൾ), വിവരങ്ങൾ സ്വികരിക്കുന്ന ബയൻസ്രേഡുകൾ) വേദ്ധിരിക്കുന്നു. സിനാപ്റ്റിക് സുഖർക്കണ്ണളും എല്ലാം വർദ്ധിക്കുകയും ഒരു നാധികോശത്തിൽ നിന്ന് മറ്റാരു നാധികോശത്തിലേക്കുള്ള (പ്രത്യേക ബന്ധങ്ങൾ സ്ഥാപിക്കാപ്പെടുകയും ചെയ്യുന്നു.

ജനനസമയത്ത്, സെറിബ്രൽ കോർട്ടുകൾ സിരിലെ ഓരോ നൃംഗാണിനും എക്കുണ്ടോളം 2500 സിനാപ്സുകൾ ഉണ്ട്. ഒരു ശിശുവിന് 2-3 വയസ്സ് പ്രായമാക്കുന്നേയാൽ, സിനാപ്സുകളുടെ എല്ലാം എക്കുണ്ടോളം 15000 ആണ്. ഒരു മുതിർന്ന വ്യക്തിയുടെ സിനാപ്സുകളുടെ എല്ലാത്തിന്റെ ശരാശരി ഇരട്ടിയാണിത്. പ്രായമാക്കുന്നേയാൽ, സിനാപ്റ്റിക് പ്രൂണിംഗ് അമ്പവാ സിനാപ്സ് മുറിച്ചുമാറ്റുന്ന പ്രക്രിയയിലൂടെ പഴയ ബന്ധങ്ങൾ നികം ചെയ്യപ്പെടുന്നു. നൃംഗാഘാത്യിറ്റിക് കാരണമാകുന്ന സിനാപ്റ്റിക് പ്രൂണിംഗും അക്ഷേണാണിൽ സപ്രാറ്റിംഗും കാരണം, തലചേച്ചാറിനോ നാധിപ്രവസ്ഥയുടെ മറ്റ് ഭാഗങ്ങൾക്കോ ഉണ്ടാകുന്ന കേടുപാടുകൾ എല്ലായ്പോഴും ശാശ്വതമല്ല. ഈ പ്രക്രിയ മനുഷ്യരെ വിശ്വാലമായ സാഹചര്യങ്ങളുമായി പൊരുത്തപ്പെടുത്തുന്നതിന് പ്രധാനമാണ്; നിശ്ചിത വ്യവസ്ഥകളോളുള്ള പ്രതികരണമായി തലചേച്ചാറിന്റെ ശരിരാസ്ഥം തന്നെ മാറാം.

മന്തിഷ്ക് പ്രവർത്തനത്തെ ബാധിക്കുന്ന അടക്കങ്ങൾ

സാമൂഹിക ഇടപെടലുകൾ:

സാമൂഹികമായി സജീവമാകുന്നത് പിന്നീടുള്ള ജീവിതത്തിൽ മാനസികവും ശാരിരികവുമായ നിരവധി ആരോഗ്യ ആനുകൂല്യങ്ങൾ നൽകുന്നു. സാമൂഹിക ഇടപെടലുകൾക്ക് ഒരു വ്യക്തിയെ ബൈജനിക തകർച്ചയിൽ നിന്ന് സംരക്ഷിക്കാൻ കഴിയുമെന്ന് പുതിയ പഠനം പറയുന്നു. പികാരങ്ങൾ നിയന്ത്രിക്കാനുള്ള കഴിവും സാമൂഹിക ഇടപെടലുകളുടെ ശുണ്ണനിലവാരവും തമിൽ പോസിറ്റീവ് റിതിയിൽ ഉള്ള ബന്ധങ്ങൾ ഉണ്ടെന്നു രണ്ടു പഠനങ്ങൾ കണ്ണെത്തി. ഇത് മേയർ-സാലോവേയ്-കേരുസോ ഇമോഷൻൽ ഇന്റിലിജൻസ് ടെസ്റ്റിന്റെ (Mayer-Salovey-Caruso Emotional Intelligence Test or MSCEIT¹) ബൈപ്കാരിക ബുദ്ധി അളക്കാനുള്ള കഴിവിന്റെ പ്രവചനാത്മകവും വർദ്ധിച്ചുവരുന്നതുമായ സാധ്യതയെ പിന്നുണ്ടെങ്കുമ്പും.

ഈക്ഷണക്രമം:

പ്രേര്യക പോഷകങ്ങളുടെ ആപേക്ഷിക അളവ് മാനസികവും വൈദകാർക്കിപുമായ പ്രവർത്തനങ്ങളിൽ സ്വാധീനം ചെലുത്തുമെന്ന് വളരെക്കാലമായി അസുമാനിക്കപ്പെടുന്നു. മസ്തിഷ്ക ആരോഗ്യത്തിലും മാനസിക പ്രവർത്തനത്തിലും പോഷകാഹാരത്തിന്റെ സ്വാധീനത്തിന് അഭിസ്ഥാനമായ ചില പ്രധാന സംവിധാനങ്ങൾ അടുത്തിടെ രേഖപ്പെടുത്തിയിട്ടുണ്ട്. ഇവ സിനാപ്പറ്റിക് ഫ്ലാറിറ്റിയിലും നാഡി പ്രവർത്തനങ്ങളിലും പോഷക വൈവിധ്യത്തിന്റെ കന്തത സ്വാധീനം നിർദ്ദേശിക്കുന്നു. മസ്തിഷ്കത്തിലെത്തിച്ചേരുന്ന അഭ്യുക്തി മസ്തിഷ്കം തന്നെ ഉൽപ്പാദിപ്പിക്കുന്ന നിരവധി ആമാശയ-കുടൽ ഹോർമോണുകൾ വൈജ്ഞാനിക പ്രവർത്തനത്തെ സ്വാധീനിക്കുന്നു. കൂടാതെ, മസ്തിഷ്കത്തിൽ നിന്ന് ഉരുത്തിരിഞ്ഞ സിനാപ്പറ്റിക് ഫ്ലാറിറ്റി നിയന്ത്രിക്കുന്ന നൃംബോഡാഹിക് (നാഡികോശ വളർച്ചയെ സഹായിക്കുന്ന) ഘടകങ്ങൾ പോലെ അറിയപ്പെട്ട ഘടകങ്ങൾക്ക് ഭക്ഷണക്രമം പോലുള്ള പുറത്തുനുള്ള നിക്ഷേപങ്ങൾക്ക് പ്രതികരണമായി ഉപാപചയം കുഴിക്കിക്കുന്നവരായി പ്രവർത്തിക്കാൻ കഴിയും.

എറുവും ഭിക്ഷ റിതിയിൽ പ്രവർത്തിക്കാൻ തലച്ചോറിന് മതിയായ ഖ്രിസ്തവം ആവശ്യമാണ്. ഉയർന്ന നിലവാരമുള്ള ഖ്രിസ്തവം തലച്ചോറിനെ മികച്ച റിതിയിൽ പ്രവർത്തിക്കാൻ സഹായിക്കുന്നു. പിറ്റാമിനുകളും ധാരുകളും ആന്റിനാക്സിയർഗ്ഗുകളും അടങ്കിയ ഭക്ഷണങ്ങൾ തലച്ചോറിനെ പോകിപ്പിക്കുകയും ഓക്സിഡേറ്റീവ് സമർദ്ദത്തിൽ നിന്ന് സംരക്ഷിക്കുകയും ചെയ്യുന്നു. ഇത് (പ്രീ റാഡികലുകളുടെയും (ജോടി ചേരാതെ ഒന്നൊ അതിലെയിക്കുമോ **ഖ്രിസ്തവാക്കളുടെ** തമാരകൾ) ആന്റിനാക്സിയർഗ്ഗുകളും ചെയ്യുകയോ നിക്കും ചെയ്യുകയോ ചെയ്യുന്ന പദാർത്ഥങ്ങൾ തമിലുള്ള അസന്തുലിതാവധിയാണ്. ശരീരത്തിൽ അത് ആത്മരന്ത്രികമായി കോശങ്ങളുടെയും ചിഷ്പുകളുടെയും നാശന്തിലേക്ക് നയിച്ചേക്കാം.

HEALTHY LIFESTYLE (ആരോഗ്യകരമായ ജീവിതശൈലി)

- MORE FRUITS (കുടുതൽ പഴങ്ങൾ)
- ENOUGH SLEEP (മതിയായ ഉറക്കം)
- ENOUGH WATER (ആവശ്യത്തിന് വെള്ളം)
- WEIGHT CONTROL (ഡേര നിയന്ത്രണം)
- MORE VEGETABLES (കുടുതൽ പച്ചക്കറികൾ)
- REDUCE STRESS (മാനസികസമർദ്ദം കുറയ്ക്കുക)

(അനാരോഗ്യ-കരമായ ജീവിതശൈലി) **UNHEALTHY LIFESTYLE**

- SMOKING (പുകവലി)
- SWEETS (മധുരപലഹാരങ്ങൾ)
- ALCOHOL (മദ്ധം)
- DRUGS (മയക്കുമരുന്ന്)
- JUNK FOOD (ജം എഡിബ്)
- TOO MUCH COFFEE (അമിതമായ കാപ്പി)

ചിത്രം. 4. ആരോഗ്യകരമായ ജീവിതശൈലി സ്വികരിക്കൽ

ചിത്രത്തിന് കെപ്പ്: macrovector കൃതാർ <https://www.freepik.com>

മറുവശത്ത്, താഴെ നിലവാരമുള്ള "ഇന്റെ" തലച്ചോറിനെയും അതിന് ഒരു പ്രവർത്തനങ്ങളെയും നശിപ്പിക്കും. സംസ്കർഖ്വതോ ശുശ്വരികൾക്കും ആയ ക്രഷണങ്ങൾ മുംബാറന്തരത്തിൽ, ശുശ്വരികൾ പ്രഭാവാര ഉൾപ്പെടെ ഇംഗ്ലീഷിൽ പെടുന്നു. ഈ ക്രഷണങ്ങൾ ഇൻസൗലിൻ നിയന്ത്രണത്തെ പ്രതികൂലമായി ബാധിക്കുകയും വികിം, ഓക്സിഡേറീപ് സമർദ്ദം എന്നിവ പരിപ്പിക്കുകയും ചെയ്യുന്നു. പല പറഞ്ഞെല്ലും മധുരമുള്ള ക്രഷണങ്ങളുടെ ഉപയോഗവും മസ്തിഷ്ക പ്രവർത്തനവും തമിലുള്ള ബന്ധങ്ങൾ കണ്ടെത്തിയിട്ടുണ്ട് - വിഷാദരോഗം പോലുള്ള മാനസിക വൈകല്യങ്ങൾക്കു പോലും ഇത് ബാധകമാണ്.

ഈ മേഖലയിലെ പറഞ്ഞെൾ പോഷകാഹാര മനസ്സാസ്ത്രം (nutritional psychiatry) എന്നാണ് അറിയപ്പെടുന്നത്. പള്ളന്തു കൊണ്ടിരിക്കുന്ന എന്നാൽ വർഷങ്ങളായി പര്യവേക്ഷണം ചെയ്യപ്പെടാതെ ഒരു മേഖലയാണിൽ. നമ്മുടെ മൊത്തത്തിലുള്ള

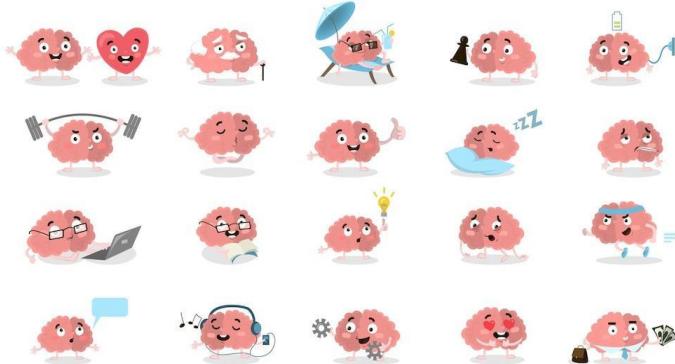
മന്ത्रിഷ്ക ആരോഗ്യത്തെ ബാധിക്കുന്ന ഭക്ഷണങ്ങൾ എത്രാക്കയാണെന്ന് ഗവേഷകർക്കും ഡോക്ടർമാർക്കും എന്നെന്ന അറിയാം: ലഭിതം: നമർക്ക് കഴിക്കുന്ന ഭക്ഷണം അവൾ പിരുമ്പുന്നു. എല്ലാം ആരംഭിക്കുന്നത് ആമാശയത്തിലും കുടലിലും നിന്നാണ്! (ചിത്രം 4).

പ്രായാമം:

പ്രായാമം നിർണ്ണായകമായിരിക്കുന്നത് എന്നുകൊണ്ടാണെന്ന് നിങ്ങൾ ചിന്തിച്ചിട്ടുണ്ടാ? ബീഡിഷ് കൊളുംബിയ സർവകലാഖാലയിലെ ഗവേഷകർ നാത്തിയ ഒരു പഠനത്തിൽ, നിരമായ എയ്റോണിക് പ്രായാമം ഹിപ്പോകാസസിന്റെ പലും വർദ്ധിപ്പിക്കുമെന്ന് കണ്ണേതി, വൈശ്വാസത്തിൽ മെമ്മറിയിലും (verbal memory) പഠനത്തിലും ഉൾപ്പെട്ടിരിക്കുന്ന മന്ത്രിഷ്ക മേഖലയാണ് ഹിപ്പോകാസയ്ക്ക്. പ്രതിരോധ പരിശീലനം, ബാലൻസ്, മനിൽ ഭോഗിംഗ് പ്രായാമങ്ങൾ എന്നിവയ്ക്ക് സമാനമായ ഫലങ്ങൾ ഉണ്ടായില്ല.

പുതിയ വൈദഗ്ധ്യങ്ങൾ :

ഓരോ തവണയും നിങ്ങൾ ഒരു പുതിയ വൈദഗ്ധ്യം പരിക്കുണ്ടോ, നിങ്ങളുടെ തലാശ്ചാർ മാറുന്നു. കൂടുതൽ വ്യക്തമായി പറഞ്ഞാൽ, നിങ്ങൾ അതിനുള്ളിലെ സാധ്യങ്ങൾ മാറുന്നു. ഒരു വൈദ്യുതിപിളി നിരന്തര അന്തരീക്ഷം അവതരിക്കുന്നോ, നമ്മുടെ ശരീരവും മനസ്സും മാറുന്നു: പേരികൾ ശക്തമാകുന്നു. ഹൃദയവും ശാസകോശങ്ങളും പലുതാകുന്നു, മന്ത്രിഷ്ക ബന്ധങ്ങൾ വേഗത്തിൽ സ്ഥാപിതമാവുകയും കൂടുതൽ കരുതുന്നതുമായിത്തീരുന്നു. മന്ത്രിഷ്കത്തിന്റെ ഈ പുനഃസംഘടനയാണ് എല്ലാ രേനപുന്നു സ്വപ്നാനന്തരിന്റെയും വികസനത്തിന്റെയും അഭിസ്ഥാനം. എന്നുകൊണ്ടാണ് ഈ മാറ്റങ്ങൾ സംഭവിക്കുന്നതെന്ന് ശരീരത്തിന് തന്ന അറിയില്ല. എന്നാൽ കാര്യങ്ങൾ കഴിയുന്നതു ലഭിതമാക്കുന്നതിന് നിരന്തരം പുനഃസംഘടിപ്പിക്കാൻ തുടർന്നു. ചെയ്യേംപ്രക്രിയകൾ, കാര്യങ്ങൾ എല്ലാപ്രമാക്കുന്നത് ശരീരവും മനസ്സും ഇഷ്ടപ്പെടുന്നു, അതിനാൽ അഭ്യന്തര തവണ ഒരു കാര്യം ചെയ്യുമ്പോൾ അത് ലഭിതമാക്കാൻ അവ ശ്രമിക്കും. ഒരു പ്രത്യേക വൈദഗ്ധ്യം പഠിക്കുമ്പോൾ ശരീരത്തെ ആധാസംപ്രദാത്തുനില്ക്കും, തുടർന്നു പ്രധാനമാണെന്ന സുചന നമ്മൾ ശരീരത്തിന് നൽകുകയും ഈ വൈദ്യുതിപിളിക്ക് കൂടുതൽ ഉപാധികൾ വിനിയോഗിക്കാൻ ആവശ്യപ്പെടുകയും ചെയ്യുന്നു. ഒരു പ്രത്യേക വൈദഗ്ധ്യം ആവശ്യപ്പെടുകയും പരിശീലിക്കുന്നതിന്റെ ഫലമായി നമ്മൾ നൃറൽ ബന്ധങ്ങൾ ശക്തിപ്പെടുത്തുകയാണ്, തുടർന്നു മികച്ച പ്രകടനം കാഴ്ചവയ്ക്കുന്നത് വളരെ എളുപ്പമാകുന്നു.



ചിത്രം. 5. മന്ത്രിഷ്കം എങ്ങനെയാണ് പുതിയ കഴിവുകൾ നേടുന്നത്?

ചിത്രത്തിന് കടപ്പാട്: vector4stock കൃതാരം <https://www.freepik.com>

ധിജിറ്റൽ സാങ്കേതികവിദ്യയുടെ ഉപയോഗ ഫലങ്ങൾ:

ഈ ധിജിറ്റൽ യൂഡത്തിൽ, മൊബൈൽ പ്രോസൈകൾ ഉൾപ്പെടെയുള്ള ധിജിറ്റൽ സാങ്കേതികവിദ്യകളുടെ ഉപയോഗം നമ്മുടെ ജീവിതശൈലിയുടെ എൻ്റെ നിർണ്ണായക ഘടകങ്ങളിലെബാന്നി മാറിയിരിക്കുന്നു. ചില വഴങ്ങളിൽ ഈ നമ്മുടെ ജീവിത ലളിതമാക്കിയുള്ള നമ്മുടെ മസ്തിഷ്ക ആരോഗ്യത്തെ ഹരിക്കാനും കാരൂശയി ബാധിക്കുന്നു. കമ്പ്യൂട്ടർ ഉപയോഗം, വർദ്ധിച്ച സ്കീൻ സമയം എന്നിവയും കുഞ്ഞാർക്കാരിൽ എ എച്ച് ഡി അമൈവാ അരെൻഷൻ യെഫിനിറ്റ് ഹെപ്പർഡ്രോക്ടിവിറ്റി ഡിസോർഡർ (attention deficit hyperactivity disorder) ലക്ഷണങ്ങളും തയ്യാറായി ബന്ധങ്ങൾ പല പഠനങ്ങളും തെളിയിച്ചിട്ടുണ്ട്.

ഉറക്കം:

ഉറക്കം നമ്മുടെ മന്ത്രിഷ്ക പ്രവർത്തനത്തെ ബാധിക്കുന്നു. ഉറങ്ങുമ്പോൾ, തലേറിപസം നേടിയ പിവരങ്ങൾ ആക്സോൺ പഴി വെദ്യുത ആവേശങ്ങളായ് ആവർത്തിച്ച് ന്യൂറോൺിലും കടന്നുപോകുകയും പിന്നീട് അത് വിപരിതമാക്കുകയും ചെയ്യും. നിങ്ങൾ ശരിയായി ഉറങ്ങുന്നില്ലെങ്കിൽ, മന്ത്രിഷ്കം ഭൂർജ്ജലമാവുകയും കാരുണ്യൾ മറക്കുകയും തീരുമാനങ്ങൾ എടുക്കാൻ ബുദ്ധിമുട്ടുകയും ചെയ്യും.

മന്ത്രിഷ്ക പ്രവർത്തനത്തിൽ വ്യായാമത്തിന് വരുത്തി ചെയ്യും പ്രകാശം

- സ്ഥിരമായി വ്യായാമം ചെയ്യുന്നവർക്ക് അൽ-ഷിമേഴ്സ് റോഗം വരാന്നുള്ള സാധ്യത കുറവാണ്. വ്യായാമം രക്തപ്രവാഹവും

- ഓർമ്മക്കരിയും വർദ്ധിപ്പിക്കുന്നു: ഈത് തലച്ചോറിൽ രാസ മാറ്റങ്ങൾക്ക് കാരണമാകുന്നു, അത് പതംഗം, മാനസികാവസ്ഥ, ചീറ്റ് എന്നിവ മെച്ചപ്പെടുത്തുന്നു.
- പ്രായമാകുന്നതുനാശിച്ച്, ജീവിതത്തെശലിയുംതുന്നും പാരിസ്ഥിതിക ഘടകങ്ങളുടെയും ഫലമായി മന്ത്രിഷ്ക്കം കൂടുതൽ ഭോഷകരമായ സമ്മർദ്ദത്തിന് വിധേയമാകുന്നു, ഈത് ഓക്സിഡേഷൻ എന്നറിയപ്പെടുന്ന രൂപ പ്രക്രിയയ്ക്ക് കാരണമാകുന്നു, ഈത് മന്ത്രിഷ്ക്ക കോശങ്ങളെ നിർണ്ണിക്കുന്നു. ബൈക്കിളിന്റെ ഹാൻഡിൽ ബാറിലെ തുരുപോ പാതി തിനു ആപ്പിളിന്റെ തവിട്ടുനിറമോ ഓക്സിഡേഷൻ മുംബം മന്ത്രിഷ്ക്കത്തിൽ സംഭവിക്കുന്ന രാസമാറ്റങ്ങൾ വിശദികരിക്കാൻ സഹായിക്കും. ആന്റർഭൈക്സിഡന്റ് അഞ്ചിയ കൈജണങ്ങൾ (സർസപലങ്ങൾ, പച്ചക്കറികൾ എന്നിവ പോലുള്ളവ) ഓക്സിഡേഷൻ ഭോഷകരമായ ഫലങ്ങളിൽ നിന്ന് തലച്ചോറിനെ സംരക്ഷിക്കാൻ സഹായിക്കും.
 - ഉറക്കം നിങ്ങളെ ഉഖജ്ജസ്യവരുക്കുന്നു, മാനസികാവസ്ഥയും രോഗപ്രതിരോധ സംവിധാനത്തിന്റെ പ്രവർത്തനവും മെച്ചപ്പെടുത്തുന്നു, കൂട്ടാതെ അത്രഷിമേഴ്സ് രോഗവുമായി ബന്ധപ്പെട്ടിരിക്കുന്ന മന്ത്രിഷ്ക്കത്തിലെ ബിറ്റാ-അമിലോയ്ഡ്യൽ പൂക്ക് എന്ന അസാധാരണ പ്രോട്ടീന്റെ രൂപീകരണം കുറയ്ക്കുന്നു.
 - മാനസിക വ്യാധാമങ്ങൾ തലച്ചോറിനെ നന്നായി പ്രവർത്തിക്കാനും പൂതിയ മന്ത്രിഷ്ക്ക കോശ പള്ളിച്ചരയ പ്രോസ്റ്റാഗ്ലിഫിക്കാനും ഡിമെൻഷ്യൂറുടെ സാധ്യത കുറയ്ക്കാനും സഹായിക്കും. നിങ്ങൾ പേരികൾ ഉപയോഗിക്കേണ്ടതുപോലെ, മന്ത്രിഷ്ക്ക കോശങ്ങൾ പതിവായി ഉപയോഗിക്കണം, അല്ലെങ്കിൽ അത് ലിക്ചു റിത്യിൽ പ്രവർത്തിക്കില്ല.
 - സജീവമായ രൂപ സാമൂഹിക ജീവിതം നയിക്കുന്നത് ഓർമ്മക്കുറവിൽ നിന്ന് നിങ്ങളെ സംരക്ഷിക്കും. മറുള്ളവരുമായി സമയം ചിലവഴിക്കുക, ഉത്തേജിപ്പിക്കുന്ന സംഭാഷണങ്ങളിൽ എർപ്പെടുക, കൂടുംബാംഗങ്ങളുമായും സൃഷ്ടിത്തുകളുമായും സവർക്കം പുലർത്തുക എന്നിവ തലച്ചോറിന്റെ ആരോഗ്യത്തിന് നല്കാണ്. സമൂഹത്തിൽ ഏറ്റവും കൂടുതൽ സാമൂഹിക ഇടപെടൽ ഉള്ളവരിൽ ഓർമ്മക്കുറവിന്റെ വേഗത കുറഞ്ഞതായി പറഞ്ഞു തെളിയിച്ചിട്ടുണ്ട്.

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എങ്ങനെ പഠിക്കാമെന്ന് പറിക്കുന്നു Learning How To Learn

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Translated from English to Malayalam by Arya Jayaraj

സംഗ്രഹം (Summary)

പാട്ടിന്റെ വരികൾ, ഫോൺ നമ്പറുകൾ, നൃത്യചൂവടുകൾ എന്നിവ ഒരാൾ എങ്ങനെ അനുഭാവം നൽകുന്നുവെന്ന് നിങ്ങൾ ചിന്തിച്ചിട്ടുണ്ടോ? പിണ്ഡിയിലും പഠിക്കാനുള്ള പുന്നക്രമമീരിക്കാനുമുള്ള നമ്മുടെ തലച്ചോറിന്റെ കഴിവിന് നന്ദി. നൃത്യാശാലയിൽ വയറിൻഗിനേക്കുറിച്ചുള്ള (neuronal wiring) പഠനങ്ങൾ, പഠിക്കാനുള്ള നമ്മുടെ കഴിവ് കല്പിൽ സ്ഥാപിച്ചിട്ടില്ലെന്നും, പുതുയ്യും പറഞ്ഞ ശൈലികൾ പരിശീലിക്കുന്നത് പുതുയ്യുമായ ഒരു കടക്കൽത്തീരം സ്പെഷ്യൽക്കുമെന്നും ബെഖ്ഷിപ്പുടുത്തിയിട്ടുണ്ട്. അതുകൊണ്ടുതന്നെ പഠിക്കുന്ന എല്ലാ കാര്യങ്ങളിലെല്ലാം നാഡിമന്ത്രശാസ്ത്ര (Neuropsychology) വ്യാവ്യാനം ഇവിടെ കണ്ടെന്നതാം.

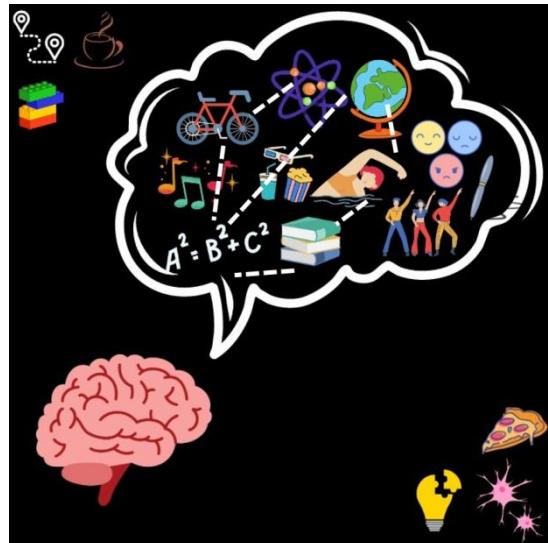
ആരുമുഖം (Introduction)

ഹന്തിനാസ്റ്റിക് ബൈബേൻസ് (Fantastic brains) എവിടെ കണ്ടെന്നതാം?

എന്തുകൊണ്ടാണ് ചില വിദ്യാർത്ഥികളെ മുൻ ബെഞ്ചേർമ്മാർ¹ എന്ന് അഭ്യാസിപ്പുടുത്തുകയും ക്ലൗഡിലെ ബുഖിയും ശ്രദ്ധയും ഉള്ളിപ്പുത്തുമായി കമ്പനക്കുകയും ചെയ്യുന്നതു്? മറ്റൊരു പിൻ ബെഞ്ചേർമ്മാർ² വിളിക്കുപ്പുനുവർ പതുക്കെ പരിക്കുന്നുവരും നിരന്തരം ശ്രദ്ധ തിരിക്കുന്നവരുമാണെന്ന് കരുതപ്പെടുന്നു. വിദ്യാർത്ഥികളെ എങ്ങനെ പഠിപ്പിക്കുന്നു എന്നതിൽ നിന്ന് ഈ പുതുയ്യാശാലർ ഉണ്ടാകാൻ സാധ്യതയുണ്ടോ? അതേ ഒരേ കാര്യം പഠിക്കാൻ ഈ വിദ്യാർത്ഥികൾ പുതുയ്യും ശൈലികൾ ഉപയോഗിക്കുന്നതായിരിക്കുമോ?

1992-ൽ, എല്ലിദും മിൽസിം പുതിയതും ആകർഷകവുമായ പഠന മാതൃകയായ VARK മോഡൽ (visual, aural, kinesthetic, reading-writing), കൈനന്ന്-തെറ്റിക് (kinesthetic) പഠിതാക്കൾ³ നിർദ്ദേശിച്ചു. അവരുടെ അഭിപ്രായത്തിൽ, ഓരോ പഠിതാവും വിവരങ്ങൾ പുതുയ്യുമായി മനസ്സിലാക്കുന്നു. ചില പഠിതാക്കൾ സിനിമകൾ, ശാഫിക്സ്, രേഖാചിത്രം എന്നിവ ലൂജ്ജിപ്പെടുന്നു (visual) പഠിതാക്കൾ), മറ്റൊളവർ (പ്രാഹിംഗണങ്ങളും ചർച്ചകളും ഇംജീപ്പെടുന്നു (ശവപ് (aural) പഠിതാക്കൾ), കുറച്ചപേര് വീണ്ടും വായിക്കുന്നതിനും വിപുലമായ കുറിപ്പുകൾ തയ്യാറാക്കുന്നതിനും (ശാധന-എഴുതൽ (reading-writing) പഠിതാക്കൾ). അതേ സമയം, അഭവസാന വിഭാഗം, ഹാൻഡ്-ഓൺ (hands on) പരീക്ഷണങ്ങളും സംവോദനങ്ങളും (കൈനന്ന്-തെറ്റിക് (kinesthetic) പഠിതാക്കൾ)

ഇഷ്ടപ്പെടുന്നു. പറിക്കുവോൾ എൽ ശൈലിയാണ് ഇഷ്ടപ്പെടുന്നതെന്ന് തിരിച്ചറിയാൻ VARK ചോദ്യാവലി ഉപയോകമതമാണ്. [1]



ചിത്രം 1: മശ്രിമോധൻ തലച്ചോറ്. ഓനിലയിക്കും പഠന ശൈലികൾ ഉപയോഗിച്ച് ഓർമ്മകളും ശ്രീലങ്കയും പറിക്കാനും രൂപപ്പെടുത്താനും തലച്ചോറിനെ എങ്ങനെ പരിശ്രീലപ്പിക്കാമെന്നതിന് ഒരു ചിത്രീകരണം.

എന്നിരുന്നാലും, എല്ലാം അതിന് അനുകൂലമല്ല . VARK-ന്റെ നാല് ശൈലികളിൽ ഓരോനും തലച്ചോറിന്റെ ഒരു ഭാഗം മാറ്റാനിന്നേക്കാൾ കൂടുതൽ തവണ ഉപയോഗിക്കാൻ അനുവദിക്കുന്നതിനാൽ, പഠനത്തെ ശക്തിപ്പെടുത്തുന്നതിന് പകരം തക്കണ്ണബുദ്ധത്വമെന്ന് സാധാരണയായി അനുമാനിക്കുമ്പോൾ. ഉദാഹരണത്തിന്, ദൃശ്യ ശൈലി പരികാരശക്തി ഗ്രാഫുകളും ധയയശ്വരകളും ഉപയോഗിച്ച് പറിക്കാൻ കമ്പ്യൂട്ടകളും മന്ത്രിപ്പാത്രിന്റെ ആസിനിപിറ്റൽ ലോബും (occipital lobe) ആവശ്യപ്പെടും. വ്യക്തിയുടെ പഠനത്തെ ശുണ്ണകരമായി ബാധിക്കുന്നതിനുപകരം, ഇത് മറ്റ് മന്ത്രിപ്പാ വിഭാഗങ്ങളിൽ നൃജോണൽ ഫയറിംഗ് (neuronal firing) തടയാം. [2] കൂടാതെ, VARK മോഡൽ എത്രത്തോളം വിശ്വസനീയമാണ്? ഒരു വ്യക്തിക്ക് ഒരു പഠന ശൈലി മാത്രമേ ഉണ്ടാകും എന്നാണോ ഇതിനർത്ഥമാം? VARK ചോദ്യാവലി എടുക്കുന്നവർിൽ 39% പേരും ഒരു വിഭാഗത്തിൽ മാത്രം പെടുന്നില്ലെന്ന് ശാസ്ത്രജ്ഞരെ കണക്കത്തിലിട്ടുണ്ട് [3]. ഇരു പഠനമനുസരിച്ച് പറിക്കാകൾ ഒന്നോ അതിലധികമോ പഠനരീതികൾ തിരഞ്ഞെടുക്കുന്നു (ചിത്രം 1). ഇന്ന്, മുൻശ്രാന്ത പഠന ശൈലികൾ

നിലവിലിഛുന്ന് പല ഗവേഷകരും വാദിക്കുന്നു. വിന്റക്കോൺസിൻ-ലാ ഫോസ് സർവകലാശാലയിലെ മന്ദിരത്തിൽ നിന്നും വേണ്ടി, അവ മാർഷിക്ക പറയുന്നതനുസരിച്ച്, വിവരങ്ങൾ നിലനിർത്താൻ വേണ്ടി, അവ ഇന്തിയാളിൽ നിന്ന് സ്വത്തെമായും അർത്ഥപൂർണ്ണമായും സംഘടിപ്പിക്കണം. അതിനാൽ, അടുത്ത തവണ ഓർമ്മിക്കുവാൻ വേണ്ടി, പീണഡു വായിക്കാനോ മാറ്റിയെഴുതാനോ നിങ്ങൾ പ്രലോഭിക്കപ്പെടുവോൾ, നിങ്ങളുടെ ഉദാഹരണങ്ങൾ ഉപയോഗിച്ച് പ്രതിഫലിപ്പിക്കുക.

സ്ഥാനവ്യ അറ്റ് ഇന്റ് അരുന്ധിഷ്ഠൻ! (Stand at ease, attention!)

പറിക്കുവോൾ, ശ്രദ്ധിക്കാനുള്ള കഴിവാണ് പ്രധാനം. ബാക്കിയുള്ളവയെ അവഹനിച്ചുകൊണ്ട് പരിസ്ഥിതിയിലെ വിശദമായ വിവരങ്ങൾ പിശകലനം ചെയ്യാനുള്ള കഴിവാണ് ശ്രദ്ധ. നമുക്ക് കൂടുതൽ ആഴത്തിലേക്ക് കടക്കാം. നിങ്ങളുടെ മുറിയിലേക്ക് ആന ചവിട്ടുന്നതായി സങ്കർപ്പിക്കുക. നിങ്ങൾക്കുങ്ങനെ മികച്ച ശ്രദ്ധ നൽകാൻ കഴിയും? മുന്ന് ചെറിയ ഐഞ്ചളിലുടെ ഇത് നിർവഹിക്കാൻ കഴിയും: പ്രവർത്തനക്ഷമത (മുറിയിലെ ആനയെക്കുറിച്ച് നിങ്ങൾ ഭോധാനാരാക്കുവോൾ), വിഷയ-സ്വീകരിക്കപ്പെട്ടിരിക്കുന്നതിലും (visual-spatial) തിരിച്ചിറയൻ (ആന എത്ര ലഘുതാണെന്നും അതിന്റെ നിറമെന്താണെന്നും മനസ്സിലാക്കുവോൾ), ഉത്കൂഷ്ടതാനുഞ്ഞാണ ഐടക്കങ്ങൾക്ക് ശ്രദ്ധ നൽകുക (പ്രശ്നാത്തലഭത്തിലുള്ള മറ്റൊരു ശമ്പുങ്ങളുമായും ആന പുറപ്പെടുവിക്കുന്ന ശമ്പുങ്ങളിൽ ശ്രദ്ധ കേന്ദ്രീകരിക്കുവോൾ) [4].

അതിനാൽ, മികച്ച പതനവും തീക്ക്ലമായ ശ്രദ്ധയും അഭിപ്രാണിയമായ ഓർമ്മയ്ക്ക് തുല്യമാണോ? ഉത്തരം അതിന്റെ സംയോഗത്തിലും അതിന്പുറവും ഉണ്ട്.

ലൈറ്റുകൾ, ക്യാമറ, പ്രവർത്തനം! (Lights, camera, action!)

നിങ്ങൾ പറിക്കാൻ ശ്രദ്ധിക്കുവോൾ തലച്ചോറിൽ എന്നാണ് സംഭവിക്കുന്നത്? കുന്നേഡിയൻ മന്ദിരത്തിൽ നിന്നും വേണ്ടി, സാംഗാംഡി ഒരു പ്രധാന ആനുസരിച്ച് നിങ്ങൾ പുതിയ വിവരങ്ങൾ മനസ്സിലാക്കുവോൾ, നാഡികൾ (neurons) സജീവമാകുന്നു. അവ മറ്റ് നാഡികോശങ്ങളുമായി ബന്ധിപ്പിച്ച് ശുംഖല രൂപീകരിക്കുന്നു. ഈ ശുംഖലകളിലെ നാഡികോശങ്ങൾ തുടക്കത്തിൽ ദുർബലമാണ്, എന്നാൽ ഉത്തേജിത്തിന്റെ ആവർത്തനത്തോടെ, നാഡികോശങ്ങൾ തമിലുള്ള ബന്ധം കൂടുതൽ ശക്തമാകുന്നു (പൊട്ടുപേശപ്പെടൽ (potentiation) എന്ന് വിളിക്കുന്നു). [5] ഇതുകൊണ്ടാണ് കൂടുതൽ വിവരങ്ങൾ ഓർത്തിരിക്കാൻ, പുനരവലോകനം ചെയ്യുന്നത് നിങ്ങളെ സഹായിക്കുന്നത്. ഒരേ പാത ആവർത്തിച്ച് ഉപയോഗിക്കുന്നതിനാൽ ഒരുമിച്ച് ബന്ധിക്കപ്പെടുന്ന (hard wired). ഈ സിലാതം - "ഒരുമിച്ച് ധയർ ചെയ്യുന്ന സ്വീഡോസ്കൂൾ ഒരുമിച്ച് വയർ ചെയ്യുന്നു" ("neurons that fire together, wire together") എന്നറിയപ്പെടുന്നു.

മനുഷ്യജീവിതത്തിൽ ഇംഗ്ലീഷ് നാഡികോശ ശുംഖലകളുടെ പിന്തുംതിൽ ആവേശകരമായ വസ്തുക്കൾ ശാസ്ത്രജ്ഞത്തിൽ കണ്ണാട്ടി. ജനനസമയത്ത്, മനുഷ്യ മനസ്സിൽ നിന്നും നാഡികോശങ്ങൾ ഏകദേശം 2500

സിനാപ്സുകൾ (synapse) ഉണ്ടാക്കുന്നു (നാഡികൾ തമ്മിലുള്ള സവർക്ക സമാനമാണ് സിനാപ്സുകൾ), വളർച്ചയോടെ ഈ സംഖ്യ ഗണ്യമായി ഉയർന്നു (രു കൊച്ചുകുട്ടിയിൽ 15,000 സിനാപ്സുകൾ വരെ)! എന്നിരുന്നാലും, മുതിർന്നവരിൽ, സിനാപ്സുകളുടെ എല്ലം പകുതിയായി കുറത്തു [6]. ഇതിനകം സൂചിപ്പിച്ചതുപോലെ, നിർദ്ദിഷ്ട ശൃംഖലകൾ ഹാർഡ് വയർസ് അരയി മാറുന്നു, മറ്റുള്ളവ അവയുടെ അപൂർവ്വമായ ഉപയോഗം കാരണം മുറിച്ചുമാറ്റപ്പെടുന്നു (Prune).

നാഡികോശ പ്രവാഹപാതയുടെ (circuitry) സജ്ജീകരണം 101

സാമാന്യ പ്രയോഗമായ 'പ്ലാസ്റ്റിനിറ്റി' (plasticity), എന്തിനെ സൂചിപ്പിക്കുന്നവനുണ്ടാമോ? പുതിയ വിവരങ്ങളുമായി പൊരുത്തപ്പെടാനും പരിപ്രത്യനം ചെയ്യുന്നമുള്ള തലച്ചോറിന്റെ കഴിവാണ് പ്ലാസ്റ്റിനിറ്റി. മനുഷ്യ മൺഡിഷ്ട് എത്രമാത്രം യോജിപ്പിക്കുന്നതാണെന്നത് ഈ സൂചിപ്പിക്കുന്നു. മൺഡിഷ്ട് വേഗത്തിൽ സവർക്കം ഉണ്ടാക്കുകയും വേർപ്പെടുത്തുകയും ചെയ്യുന്നുവെങ്കിൽ, അതിന് 'ക്രോസ്കാല് പ്ലാസ്റ്റിനിറ്റി' (short-term plasticity) ഉണ്ട് [7].

ഒരു മൊബൈൽ ആപ്പിക്കേഷൻിൽ നിങ്ങളുടെ പ്രിയപ്പെട്ട കേഷണത്തിന് പണം നന്നക്കുപോൾ, നിങ്ങൾക്ക് OTP ലഭിക്കും. തുടർന്ന് നിങ്ങൾ ആപ്പിക്കേഷൻിൽ OTP ആവർത്തിക്കുകയും ഇടപാട് പൂർത്തിയാക്കുകയും, പിന്നെ സ്ഥാപിക്കുകയും ചെയ്യുന്നു. ഒരു OTP ഓർമ്മക്കുകയും അത് ആവർത്തിക്കുകയും ചെയ്യുന്നത് ഹിസ്പകാല് പ്ലാസ്റ്റിനിറ്റിയുടെ ഒരു ഉത്തരം ഉദാഹരണമാണ്. ഒരു നിംഫത്തിന്റെ ഒരു അംശത്തിന് അത് അന്തുനാപേക്ഷിതമാണ്, എന്നാൽ പിന്നീട് അത് അപ്രസക്തമാണ്. എന്നിരുന്നാലും, നമ്മുടെ തലച്ചോറിലെ മിക്ക ഓർമ്മകളും ദീർഘകാലത്തേക്ക് സൂക്ഷിക്കേണ്ടതുണ്ട്. ഇതിനെ 'ദീർഘപകാല പ്ലാസ്റ്റിനിറ്റി' (long-term plasticity) എന്ന് വിളിക്കുകയും, മിനുട്ടുകൾ മുതൽ മണിക്കൂറുകൾ, ദിവസങ്ങൾ അല്ലെങ്കിൽ വർഷങ്ങൾ വരെ ഈ നീണ്ടുനിന്നുന്നകയും ചെയ്യുന്നു. മൺഡിഷ്ട് വിവരങ്ങൾ എങ്ങനെനു സംഭരിക്കുന്നു എന്നതിന്റെ പ്രധാന മാതൃകയാണിത്. [8]

അതിനാൽ, ഈ പ്ലാസ്റ്റിനിറ്റി (ഹയറിംഗ് നിരക്ക്) അജ്ഞകാൾ കഴിയുമോ? അതെ! മനുഷ്യൻ മൺഡിഷ്ട് പ്രവർത്തനവും അതിനാൽ നൂറോളം അജ്ഞകുന്നതിനുള്ള ഏറ്റവും സാധാരണമായ റീതിയാണ് ഇലക്ട്രോഡുസെന്റോഗ്രഫി (എഎംഇ) (EEG). തലയോട്ടിയിൽ ഓനിലഡികം ഇലക്ട്രോഡുകൾ (electrodes) സ്ഥാപിച്ച് തലച്ചോറിന്റെ വൈദ്യുത പ്രവർത്തനം EEG അജ്ഞക്കുന്നു. പ്ലാസ്റ്റിനിറ്റി അജ്ഞകുന്നതിനുള്ള മണ്ഡാരു ദ്രാവകയാമുഖ്യം മാർഗ്ഗമാണ് മാശൈനിക്ക് റെസാണൽസ് ഇമേജിംഗ് (എംആർഎഫ്) (MRI).

അവസാനമായി, സ്ഥാപിത നാഡി ശാസ്ത്രജ്ഞന്മാർ പോലും ഓരിക്കൽ നമ്മുടെ മൺഡിഷ്ട് ട്രാൻസ്ഫോർമേറുകൾ ബോക്സുകൾ പോലെയാണെന്ന് കരുതിയിരുന്നത് ഓർക്കേണ്ടതാണ്. അവരുടെ പറിക്കാനുള്ള കഴിവ് സ്ഥിരപ്പെട്ടതായിരുന്നു.

വിപ്പുലീകരിക്കാവുന്നതാണെന്നും വിവിധ രൂപങ്ങൾ ഉൾക്കൊള്ളാൻ കഴിയുമെന്നതും നമുക്കറിയാം.

എപ്പോഴാണ് മസ്തിഷ്ഠം തുറക്കുന്നത്?

മസ്തിഷ്ഠത്തിന്റെ അടിസ്ഥാന ശൃംഖല നിസ്സംശയമായും ബൈജ്ഞാനികവിദ്യയിൽ സ്ഥാപിച്ചിരിക്കുന്നു. കൂട്ടികളുടെ മസ്തിഷ്ഠം വികസിക്കുന്നത് 'നിർബന്ധായക കാലഘട്ടങ്ങൾ' (critical periods) എന്നറിയപ്പെടുന്ന സ്ഫോടനത്തിലൂടെയാണ്. ഈ കാലഘട്ടങ്ങളിൽ, നാഡികൾ തമിലുള്ള സിനാപ്സുകളുടെ ഏഴും ഒളം വളരെ കുടുതലാണ്, ഇത് ഏതെങ്കിലും ഉത്തേജനം സ്വീകരിക്കാനുള്ള സാധ്യത കുടുതലാണെന്ന് സൂചിപ്പിക്കുന്നു. ഉദാഹരണത്തിന്, ഒരു പുതിയ ഭാഷ പഠിക്കുന്ന പ്രക്രിയ നിർബന്ധായക കാലഘട്ടത്തിൽ പരിമിതപ്പെടുത്തിയിരിക്കുന്നുവെന്ന് വിശ്വസിക്കപ്പെടുന്നു. ഈ നിർബന്ധായക കാലഘട്ടത്തിൽ ഒരു കൂട്ടി സംഭാഷണത്തിന് വിധേയനായാൽ, അവൻ സ്വന്ദര്ശിയെപ്പോലെയുള്ള വാചാലത നേടാനുള്ള സാധ്യത വളരെ കുടുതലാണ്. ഏന്നിരുന്നാലും ഇതിനുശേഷം ഭാഷാപരമായി സന്പന്നമായ അന്തരീക്ഷത്തിൽ, കഴിവ് നേടുന്നത് ഒരു വെള്ളവിളി തന്നെ ആണ് [3].

അഞ്ചേ സിനാപ്സും നാഡിക്കോണങ്ങളുടെ നിർമ്മാണക്ലൂസ്കളെ നിന്മിന്ന് ചെയ്യുകയും, തലച്ചോറിന്റെ ഘടനയുടെ ഒരു രൂപരേഖ വെളിപ്പെടുത്തുകയും ചെയ്യുന്ന ഒരു കെട്ടിടം നിർമ്മിക്കുന്നതിന്റെ അടിസ്ഥാനത്തിൽ പറിക്കുന്നതിനേക്കുറിച്ച് ചിന്തിക്കുക. കെട്ടിടത്തിന്റെ അടിത്തിയും താഴെത്തെ നിലയും സജീവിക്രിച്ചുകഴിത്തോൻ, രണ്ടുനില കെട്ടിടം നിർമ്മിക്കണം അതോ ബുർജ്ജ് ലഭിച്ച നിർമ്മിക്കണം എന്നത് നിങ്ങളുടെ തീരുമാനമാണ്.

വെജഞ്ചാനികമായി ആവശ്യപ്പെടുന്ന ജോലികളുടെ പ്രകടനത്തെ പ്രായം തടസ്സപ്പെടുത്തുന്നതായി പാനങ്ങൾ തെളിയിച്ചിട്ടുണ്ട്. എന്നിരുന്നാലും, പ്രായത്തിനുസരിച്ച് പഠനശേഷി പുർണ്ണമായും അവസാനിക്കുന്നില്ല. പ്രായപൂർണ്ണത്തിയാവുമ്പോൾ പഠിക്കുന്നത് പ്രാമാണികമായി സ്വയം നയിക്കപ്പെടുന്നതും, സാമാന്യവൽക്കരിച്ച് സിഖാന്തങ്ങൾ മുതിർന്നവയുടെ പഠനത്തെ മനസ്സിലാക്കാൻ സഹായിക്കുന്നതുമില്ല. നിങ്ങളുടെ പശയതും പുതിയതുമായ ഓർമ്മകൾ പൊരുത്തപ്പെടുന്നില്ലെന്ന് ഉറപ്പാക്കിക്കൊണ്ട്, നേന്മുണ്ടുത്തിന്റെ പുതുമ ഉള്ളതും ഇല്ലാത്തതും ആയ വശങ്ങൾ തമിലുള്ള വേത്തിരിവ് തലച്ചോറിന്റെ പ്രീഫോർണ്റ് (prefrontal), ടെസ്റ്റാൾ (temporal) മേഖലകൾ ശ്രദ്ധിക്കുന്നു. മെഞ്ച് പറിക്കണമുള്ള ശ്രമത്തിൽ ഇല്ലേപ്പീ മറന്നതായി സങ്കൽപ്പിക്കുക! നമ്മുടെ മസ്തിഷ്ഠം നാം സങ്കൽപ്പിക്കുന്നതിനേക്കാൾ കുടുതൽ സമർപ്പനാണ് [10].

അതിനാൽ, നിങ്ങൾക്ക് പറിക്കാൻ കഴിയുമെന്ന് നിങ്ങൾ കരുതുന്നുണ്ടോ?

നാം നേരിട്ട് കാരുജങ്ങളുടെ ജിഗ്സോ കൂട്ടിച്ചേരക്കാൻ തുടങ്ങാം; പഠന വിദ്യകൾ, ശ്രദ്ധ, നാഡി പ്രവാഹപാതകൾ, പഠന പാത. നമ്മുടെ മസ്തിഷ്ഠത്തിന്റെ മിച്ചിവിനേക്കുറിച്ച് നമ്മൾ വേണ്ടതെ ബോധ്യപ്പെടുത്താൻ കഴിയുമെന്ന് നമ്മൾ പത്രീകരിച്ചുകൊണ്ടും, അതിനാൽ നാം അവയെ പരാജയത്തിന്റെ യുദ്ധക സാഹചര്യത്തിലേക്ക് പരിമിതപ്പെടുത്തരുത്.

കാരണം, നിങ്ങൾ ബുദ്ധിപരമായി ആവശ്യപ്പെടുന്ന എന്തെങ്കിലും ജോലി ചെയ്യുകയാണെങ്കിൽ, അതിനിന്ത്യാം വൃത്തിയു മനസ്സിൽ മേഖലകൾ നിങ്ങൾക്ക് അറിയാവുന്ന ബിന്ദുകൾ ഓൺചീഴ് ചേർക്കുകയും സാഹചര്യം നന്നായി മനസ്സിലാക്കുകയും ചെയ്യുന്ന എന്നതാണ്.

പഠനത്തിന്റെ വിവിധ വശങ്ങൾ പരിശോധിക്കാൻ നിലവിലെ ഗവേഷണം മുന്നോട്ട് പോകുന്നു. ഫീയൽ (glial) കോശങ്ങളുടെ സ്ഥാനങ്ങളോടൊപ്പം പോപ്പുലേഷൻകൾ (non-neuronal populations), ബെജുത്ര ഭവ്യതത്തിന്റെ (white matter) പുനര്സംഘടന, പൾസ് (pulse) സംബന്ധിക്കാത്തിന്റെ (transmission) തകർച്ചയ്ക്ക് കാരണമായ, ഒട്ടവിൽ, തീവ്രമായ പരിക്രമകൾ, സ്ലീഞ്ചർകൾ, മാനസികരോഗങ്ങളുമായി ബന്ധജുട്ട് കാരുമായ തകരാറുകൾ എന്നിവയിൽ നിന്ന് നിങ്ങളുടെ മനസ്സിൽ എങ്ങനെ മാറും [11] എന്നിവ ഇതിൽ ഉൾപ്പെടുന്നു.

മർട്ടി-സൺസറ്റി ഉത്തേജനങ്ങളോട് നിങ്ങൾ തുടർച്ചയായി പ്രതികരിക്കുന്നതിനാലും നിലവിലുള്ള സമ്പർക്കങ്ങൾ തുടർന്ന് റിവയർ ചെയ്യുന്നതിനാലും പഠന കല്പനാജനകമാണ്, അത് ഒടുവിൽ നിങ്ങളുടെ പെരുമാറ്റത്തിലും പ്രതിഫലിക്കുന്നു. അടിസ്ഥാന വികസന രൂപരേഖ കൂടാതെ, നിങ്ങളുടെ പഠന കഴിവുകൾ കല്പിൽ സജ്ജീകരിച്ചിട്ടില്ല [12]. നിങ്ങൾ പുതിയ എന്തെങ്കിലും പഠിക്കാൻ ദൃശ്യമിച്ചയാം ചെയ്യുകയും അതിനായി 'കേന്ദ്രീകൃത ശ്രദ്ധ (focused attention)' നൽകുകയും ചെയ്യുകയാണെങ്കിൽ, നിങ്ങൾ വിജയിക്കും (സാധാരണയായി 'ഗ്രിറ്റ്' എന്ന് വളിക്കുന്നു). എല്ലാത്തിനുമുമ്പാണി, ഏപ്രിജീ കലാം ഉഖരിച്ചിട്ടില്ലോ, "രാജുത്തിന്റെ ഏറ്റവും മികച്ച തലപ്പോരുകൾ ഝാസ് മുറിയുടെ അവസാന ബെഞ്ഞുകളിൽ കണ്ണെത്തിയേക്കാം."

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കാലിയോസ് കോപ്പിലുടെ: ബഹുതല ഭോധ മനസ്സ്

Through the Kaleidoscope: The Multi-layered Conscious Mind

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സംഗ്രഹം (Summary)

എല്ലാ ജീവജാലങ്ങളുടും ഓനിപ്പിക്കുന്ന ഒരു സവിശേഷതയായി ഭോധം ഉയർന്നുവരുന്നു. അതിന്റെ വ്യാപ്തിയിലും പ്രത്യാഹാരങ്ങളിലും അത് വിശാലവും സകീർണ്ണവുമാണ്. സപ്പനങ്ങളും അത് ഉണ്ടാക്കുന്ന ഭോധത്തിന്റെ വികലതയും; സകീർണ്ണതയുടെ മഠാരു തലം ചേർക്കുന്നു. ഓനിലയിക്കം സാധ്യതയുള്ള സിഖാനങ്ങളും ഘടനാപരമായ അടിത്തറകളും ഉപയോഗിച്ച്, മന്ത്രിഷ്ക്കത്തിനുള്ളിലെ റിതികിയകളുടെയും സംയോജനത്തിന്റെയും സകീർണ്ണതകൾ പരിക്രമനത്തിനുള്ള ഉൾക്കാഴ്ചയുള്ള പരാത്തലം ഭോധം അവതരിപ്പിക്കുന്നു.

ആരുവം (Introduction)

നിങ്ങൾ എപ്പോഴുകിലും മുൻകണ്ണിട്ടും, സ്വപ്നത്തിന്റെ ചില ഭാഗങ്ങൾ ഓർമ്മതട്ടുകാണും അവക്ക് അർത്ഥം നൽകുവാനും ശ്രദ്ധിച്ചിട്ടുണ്ടോ?

നമ്മൾ ഉണ്ടുവോൾ നമ്മുടെ മന്ത്രിഷ്ക്കം കണക്ഷനുകൾ സ്വയം നിന്മിക്കുന്നതാണോ അതോ നമ്മുടെ സപ്പനങ്ങൾ നമ്മൾ ഓർക്കുന്നത് പോലെയാണോ എന്ന് ആശ്വര്യപ്പെടുന്ന, ഒരു സ്ഥിതിവിശേഷം നമ്മളിൽ ഭിക്കവർക്കും സുപരിചിതമാണ്. 'ഉണ്ണൻനിരിക്കുന്ന', 'ഭോധം' എന്നതിന്റെ അർത്ഥമെന്നാണ്? ഈ അവസ്ഥ സ്വപ്നം കാണുന്ന അവസ്ഥയിൽ നിന്ന് എന്നെന്ന പ്രത്യാസപ്പേഴ്സിഡിക്കുന്നു? വാസ്തവത്തിൽ, നമ്മൾ ഒരു സാഹിത്യ കൂട്ടിഷയിൽ(cliche) മാത്രമല്ല ജീവിക്കുന്നതെന്നും നമ്മൾ അനുഭവിക്കുന്നതെല്ലാം പെറും സപ്പനങ്ങളെല്ലാം എന്നെന്ന തിരിച്ചറിയാം?

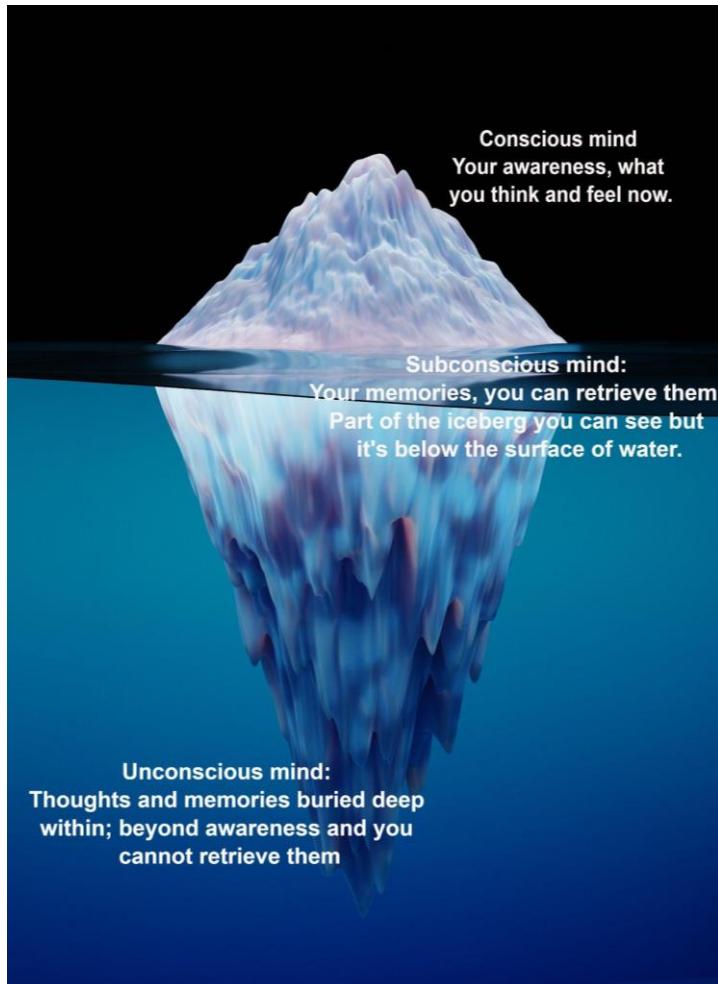
നമ്മുടെ സാധാരണ ഉണ്ണൻനിരിക്കുന്ന അവസ്ഥയുടെ ഒരു പ്രധാന സ്വഭാവം ഭോധമാണ്. ഒരുപാസന്നർഹപരമായി പറഞ്ഞാൽ, ഒരു ഉത്തേജജനനതാർ പ്രതികരിക്കാനുള്ള ഒരു ജീവിയുടെ കഴിവിനെ ഭോധം സുചിപ്പിക്കുന്നു. ഒരാളുടെ ചുറ്റുപാടുകളുടെ ഭോധംവാനാരായിരിക്കുകയോ മനസ്സിലാക്കുകയോ ചെയ്യുന്ന അവസ്ഥ കൂടിയാണിൽ. നിങ്ങൾ അനുഭവിച്ചിരുന്ന എല്ലാ കാര്യങ്ങളും ഇതിൽ ഉൾക്കൊള്ളുന്ന - നല്ലതും, ചീതതും, പുതതികടക്കും, സംഗ്രഹിതത്തിന്റെ ശബ്ദവും മുതൽ സിനിമാ പരാമർശങ്ങൾ വരെ.

ഈ പ്രവർത്തനം ഭോധത്തിന്റെ വിവിധ സിഖാനങ്ങൾ, അഭോധ മനസ്സും അതിന്റെ പ്രവർത്തനങ്ങളും, ഭോധത്തിന്റെയും അഭോധവസ്തുവയുടും അടിസ്ഥാനമായ സാമൂഹിക നൃത്വം സയൻസ് എന്നീവ പര്യവേക്ഷണം

ചെയ്യുന്നു. അവസാന ഭാഗത്ത്, സ്വപ്നങ്ങളുടെ ന്യൂറോ സയൻസിനെ കുറിച്ച് സമഗ്രമായ അനേപാഷണം നടത്തുന്നു നമുക്ക് തുടങ്ങാം!

ബോധത്തിന്‌രെയും അബോധ മനസ്സിന്‌രെയും ഫ്രോയിഡിയൻ ആശയം (FREUDIAN IDEA OF THE CONSCIOUS AND THE UNCONSCIOUS MIND)

നുറു വർഷങ്ങൾക്ക് മുമ്പ്, പ്രശ്നസ്ത ഓസ്ട്രീയൻ നാലിഖാസ്റ്റ്രജണനും സൈക്കിാ അനലിറ്റിക്(psychoanalytic) സിഖാന്തത്തിന്‌രെ തുടക്കക്കാരനുമായ സിഗ്മൺ ഫ്രോയിഡ് ഫ്രോയിഡിയൻ മണ്ണുമലബയ ലോകത്തിന് പരിചയപ്പെടുത്തി - മനുഷ്യന്‌രെ മനസ്സിനെയും മാനസികാവനയും മികച്ച ലിതിയിൽ വിശദികരിക്കുന്നതിനുള്ള ഒരു സ്വഷ്ടിപ്രാർമ്മായ സാമ്യം.(ചിത്രം. 1)



Conscious mind
Your awareness, what
you think and feel now.

Subconscious mind:
Your memories, you can retrieve them.
Part of the iceberg you can see but
it's below the surface of water.

Unconscious mind:
Thoughts and memories buried deep
within; beyond awareness and you
cannot retrieve them

ബോധമുള്ള മനസ്സ് നിങ്ങളുടെ
അവബോധം, നിങ്ങൾ ഇപ്പോൾ
എന്താണ് ചിന്തിക്കുന്നതും
അനുഭവിക്കുന്നതും

ഉപബോധ മനസ്സ്: നിങ്ങളുടെ
ഓർമ്മകൾ, നിങ്ങൾക്ക് അവ
പീണ്ടുകൂടാൻ കഴിയും.
മനസ്സുമലയുടെ ഒരു ഭാഗം
നിങ്ങൾക്ക് കാണാൻ കഴിയും,
പക്ഷേ അത് ജീവത്തിന്റെ
ഉപരിതലത്തിന് താഴെയാണ്

അവബോധ മനസ്സ്: ചിന്തകളും
ഓർമ്മകളും അശ്വത്തിൽ
അടക്കം ചെയ്യിരിക്കുന്നു;
അവബോധത്തിന്നുറി
നിങ്ങൾക്ക് അവ
പീണ്ടുകൂടാൻ കഴിയില്ല

ചിത്രം 1. ഫോറ്മാറ്റിഫിയൻ മണ്ഡുമല.

"മനസ്സ് ഒരു മണ്ഡുമല പോലെയാണ്, അത് വെള്ളത്തിൽ മുകളിൽ അതിന്റെ
പ്രിഫീലേബാന് ഭാഗം കൊണ്ട് പൊങ്ങിക്കിടക്കുന്നു." - സിശമൻക് ഫോറ്മാറ്റ്
ചിത്രത്തിന് കടപ്പാട്: ഫോട്ടോ എഞ്ചത്ത് [SIMON LEE](https://unsplash.com/) on <https://unsplash.com/> (edited by author)

സിശമൻക് ഫോറ്മാറ്റിഫിയാണ് ദൈഖാക്കാ അനാലിസിസിന്റെ സ്ഥാപകൻ. ഒരു
രോഗിയും മനോവിശകലന വിദ്യയും തമിലുള്ള സംഭാഷണത്തിലൂടെ

மந்திரின்கெடு வேவாயமந்திரும் அவேவாயமந்திரும் உல்லெப்புதொரோஶலகசளம் விழுதிருத்துநாற்றிரும் சிகித்திக்கூன்றதிருமாயி அடேஹா ஏரு க்ஷிகித்துவபியி ஸங்பூதியின் அதிகரித்து. பின்கூறுநாலும், ஸாமுபை நாவிஶாஸ்தாம் ஏரு உத்திரவூரும் ஶூவெட்-பிப்புமின்டுர்ஸ்டீ(fMRI) ஸாகேதிக பிழக்கல் உபயோகித்து மந்திரின்கெடு வேவாயாவங்முதிலுத்து வாரம் நம்முடை நியநிறநிறத்திரும் அவேவாயத்திரும் அதிர்வாணங்க தெழியிக்கூறும். ஹூ திரித்திரிவ் அவேவாயாவங்முதி வேவாயமந்திரும் நிதில் மாற்றுவாணம் முன் அதைத்திருப்பிருவுமான் [1]. அதிர்வால், ஸாங்காஷ்ணிகங்கள் போலுத்து லதிதமாய சிகித்து ஹட்டப்ளவுக்கல் மந்திரின்கெடு அது வாரம் மந்திரிலாக்கான் பருப்பத்துவாணங்க தோன்னிப்பு. அவேவாயமந்திரும் வெக்காரிக்கமாய காரணங்களுக்கு மாற்றிருக்கூன்தும் அதைப்பிரிவையாக சிகித்துக்கல் உபயோகித்து புகாங்மாபிக்காவுகூன்துமான் பின் பரங்காரத போயியியியன் விக்ஷங்காத்திற்கு நின்க ஹூ பூதிய அதையும் வழிர அகவெய்யான் [2].

அதையிருக்கும் நூற்று சுயங்களும் வேவாயவும்

ஸோஷ்யல் நூற்று சுயங்கள் முன் மேவுக்குவெ லயநமான் - ஸாமுபை மந்திரும் நூற்று பரிச்சுபரம் ஹட்டப்ளக்கூறு பின்கூறுமின்கெடு ஶாஸ்தாம், கோரிட்ரீப் செஸகோலஜி (cognitive psychology) (அதைக்கல் பின்கெடு சிதிக்கூறும் பின்கெடு ஶாஸ்தாம்), நூற்று வேவாயோலஜி (neurobiology) (அனாடுமி, மிஸியோலஜி(physiology), மஸ்திஷ்கத்திரும் கெடு புவர்த்தனங்கள் ப்ரபார்த்தமாக்குவது வையோகெடுக்கில்சிடி(biochemistry). தற்காலது ஸஂவியானங்களுத்தும் நாவிப்பாதகமெல்லையும் நா காலான ஸாவாணங்களுமாயி வையிப்பிக்கான் ஹப்போர் நம்முக்கு கடியுக்காத்திர்வால் ஹூ அதையிருக்கும் ஸமிப்பான மந்திரிக்கூறித்துத்து நம்முடை நார்ளையை கூடுதல் வழக்கமாக்கான் ஸபாயிக்கூறும் [2].

பூரியுமெப்புமங்கியியாய நம்முடை ஹட்டிய யார்ளைக்கு, நம்முடை ஸாமுபைக் காரணக்குமாயி; அதைக்கூடுதல்லையும் ஸாபாபருப்பங்களுத்தும் குரித்துத்து நம்முடை வியிக்காயங்களுமாயி ஸபாஜமாயி ஸமாநமான். ஹவ் ரலெகு அவேவாய மந்திரும் வழுதெய்யிக்கும் ஸபாயிக்கிப்புக்கூடும், அத்து ஏரு அரிப்பு போலே புவர்த்தித்து நம்முக்கு சுருமுத்து லோக்கிரும் கெடு வழக்கமாய சிதிரம் நாக்கூறும். ஹூ ஸ்டிலிக்கெடு அரிப்பு(subliminal filter) ஹலைக்கீல், நம்முக்கு காலான கடியுக்கூறும் கேள்கூக்கூயும் செய்யும் விவரங்கள் வழிர வழுத்தும் அத்தமத்துநூற்று நம்முடைன் [2].

வேவாயவும் அவேவாயமந்திரும் கெக்கோர்த்து புவர்த்திக்கூறும். உடாபாரனநிற்கு, 'பூந்தகை' பின் வாக்கு கேள்கூக்கோர், வேவாய நினைக்கோக் கூறுதலாக, பரிச்சுபரம் வையிப்பித்திரிக்கூறு பின்கெடுக்கில்லை பிழுதியிரிக்கூறு ஏரு கூடுதல் பேஜுக்களுவாணங்க. பின்கெடு, அவேவாயாவங்முதி ஏரு பாரி கடங்கு நினைக்க வாயித்து பிழிப்பு பூந்தகைகளுத்துமெல்லையும் சிதிரங்கள் வரய்க்கான துக்கங்கூறும், அவையிலே சிதிர கமக்கு விளைகும் ஸார்ளைக்கூறும் நினைக்குவெ பிழிப்பு நோவலுமாயி ஹருங்கு நினைக்க விழெக்கான் அதுமாபிக்கூறு ஏரு கடங்காத்திர்வால் கொள்ளுபோகுவானும் ஸாயுத்திரயைக்கூடும்.

வேவாய அவேவாயமந்திருமின் நியநிறநிறத்திலாக்கூறும், மாற்றமல்ல அதிர்வாக கூடுதல்ல வழிசுலிக்கான அங்குவசிக்கையூக்கையுமில்லை. அவேவாயாவங்முதி கூதிரயாவெக்கில், வேவாய அதிர்வால் கடிதெட்டான். அதைகொள்ளும் அஸங்கிதமாய சிதிரக்கால்

വ്യതിചലിക്കാതെ നമുക്ക് ഭദ്രന്റെ ജോലികൾ പൂർത്തിയാക്കാൻ കഴിയുന്നത്. ബോധമന്നു ഒരു സർക്കസിലെ റിൻ മാറ്റീറേപ്പോലെയാണ്. അഭോധമന്നുനിന്നും ചിത്രങ്ങൾക്കും ഇത്തരംക്കും ഇത് ദിശയും സന്ദർഭവും നൽകുന്നു. മെത്ര പറമ്പെ നിയന്ത്രണമില്ലായിരുന്നുകിൽ, അഭോധമന്നു യാദൃച്ഛികമായ ഇഷ്ടകളാൽ എറിയപ്പെട്ട് ഒരു കപ്പൽപായ് ഇല്ലാത്ത ഒരു കപ്പൽ പോലെയാകും.

ബോധത്തിന് രാഖിയാത്താണിൾ

ഫ്ലോപ്രമോ നൃംഗാബോധയോളജിക്കൽ അടിസ്ഥാനമോ ഇല്ലാത്ത ഒരു നിശ്ചിയമായ അസ്ത്രിപ്രമായി ബോധം വളരെക്കലാമായി കണക്കാക്കപ്പെട്ടിരുന്നു. ബോധത്തെ ഒരു ശാസ്ത്രിയ പ്രതിഭാസമെന്ന നിലയിൽ പഠം ആരംഭിച്ചത് ഇരുപതാം നൂറ്റാണ്ടിൽ മാത്രമാണ്. തുടർന്ന്, തലച്ചോറിലെ ബോധപൂർവ്വമായ റീതിക്കിയകളുടെയും ന്യാനവും സംവിധാനവും നിർവ്വചിക്കാനുള്ള ശ്രമങ്ങൾ നാല് പ്രധാന സൈഖാനിക സമീപനങ്ങൾ സ്വീകരിച്ചു:-

- ആശേരാള വർക്ക്‌സ്‌പേസ് സിഖാത്താണിൾ (global workspace theory)
- ഉയർന്ന-ക്രമ സിഖാത്താണിൾ (higher order theories)
- സംയോജിത വിവര സിഖാത്തം (the Integrated Information Theory)
- റീ-എൻട്രി, പ്രൈഡിക്രീവ് പ്രോസസ്സിംഗ് സിഖാത്താണിൾ (re-entry and predictive processing theories) [3].

ആദ്യത്തെ രണ്ട് സമീപനങ്ങൾ ബോധത്തിന് രാഖിയതെന്നും പ്രവർത്തനപരമായ വശങ്ങളെ അഭിസംഭോധന ചെയ്യുന്നു. മറ്റ് രണ്ടും ബോധത്തെ ഒരു അനുഭവമായി കൂട്ടകാരം ചെയ്യുന്നു [4]. ഘടനപരമായ പറഞ്ഞാതെ, ഈ സിഖാത്താണിൾ ഓരോനും വ്യത്യസ്തമായ മന്ത്രിക്ഷക മേഖലകളും സൂചിക്കുന്നോളം, സെറിബ്രൽ കോർട്ടക്സ് (cerebral cortex) 'ബോധത്തിന് ഇരിപ്പിടം' ആശേന്നാണ് സമവായം. മില്യ് ബൈറയിൻ റെറിക്യൂലാർ (midbrain reticular) രൂപീകരണവും താലമിക് നൃംഗിയിനസും (thalamic nucleus), ശ്രീറിൻ [5] പോലുള്ള പ്രവർത്തനങ്ങൾ ചെയ്യുന്നു. ഫില്ല (prefrontal) മേഖലകളും (പ്രധാനമായും ഓർബിറ്റോഫ്രontal കോർട്ടെക്സ്‌സും (orbitofrontal cortex) ആറ്ററിനിറയർ സിൺഗ്രൗപ്പേഴ്സ് കോർട്ടെക്സ്‌സും (orbitofrontal cortex) ഇൻട്രാക്രീയൽ ലാലക്ടിക്കൽ റീലീഫ്വേഷൻ (പ്രീഇൻട്രിസ്) (IES) ബോധപൂർവ്വമായ അനുഭവത്തിന് അസ്പദമതകൾ സ്വീച്ചിക്കുന്നതില്ലോ. ഈ പ്രദേശങ്ങളും ഒരു പക്ക് പദ്ധതിക്കുന്നുണ്ടെന്ന് തെളിയിക്കുന്നു [6].

2004-ൽ ജിയൂലിയോ ഫോനോണി നിർദ്ദേശിച്ച സംയോജിത വിവര സിഖാത്തം, ഭാതിക സംവിധാനങ്ങളുടെ ("പോസ്റ്റുറൂക്സ്" (postulates) എന്ന് വിളിക്കപ്പെടുന്ന) ഗുണങ്ങളെ അനുമാനിക്കുന്നതിനായി ബോധത്തിന് രാഖിയതെന്നും അനുഭവത്തിൽ നിന്നും അതിനെ ഗുണങ്ങളിൽ നിന്നും ടിനോട് നടക്കുന്ന ഒരു ഫല-കാരണ സമീപനമായി ഈ സിഖാത്താണിൾ കണക്കാക്കാം [7]. വിവരങ്ങൾ സംയോജിപ്പിക്കാനുള്ള ഒരു സിസ്റ്റമ്പാം (system) ശേഷിയെ ബോധം ആശ്രയിക്കുന്നുവെന്നും ബോധം (പ്രധാനമായും തലച്ചോറിലെ ഒരു പിൻഭാഗത്തെ കോർട്ടിക്കൽ "ഹോട്ട് സോൺമായി" (hot zone) ബന്ധപ്പെട്ടിരിക്കുന്നുവെന്നും (ഇതിൽ പരിയേറ്റൽ (parietal), ടെപൊറൽ (temporal), ആൻസിപ്പിറ്റൽ (occipital) ലോബുകളുടെ ഭാഗങ്ങൾ ഉൾപ്പെടുന്നു) അത് ഉറപ്പിക്കുന്നു.

ഈ സിഖാത്തം നി മെട്ടിക് (phi metric) (സിനർജിയുടെ ആവിഷ്കാരം, ഒരു സിസ്റ്റം അതിന് ഭാഗങ്ങളുടെ ആകെത്തരുക്കരയക്കാർ എത്രതെന്താളം വലുതാണെന്ന് സുചിപ്പിക്കുന്നു) അനുസരിച്ച് ബോധത്തിന് ശാന്തിശാസ്ത്രപരമായ അളവ്

അനുമാനിക്കുന്നു. അതിനാൽ, ബോധം എത്രാണെന്നും അത് എവിടെ നിന്നാണ് വരുന്നത് എന്നും, എത്ര ഖട്ടതിലും ഒരു വ്യക്തിയിൽ അത് എത്രതേതാളിം ഉണ്ടെന്നും വിശദിക്കിക്കുന്നു [8].

അതേസമയം, ഫ്രോബൈൻ സ്ക്രാൻപേസ് (ജിഎൻഡിഎഫ്ലൂ) (GNW) സിഖാത്തങ്ങൾ, തലച്ചോറിനെ ഒരു ഫ്ലാക്സ്‌ബോർഡായി ചിത്രിക്കിക്കുന്നു - ബോധത്തിന്റെ സഖിയെ അനുഭവം സ്പീഷ്സിക്കുന്നതിന് പ്രത്യസ്ത വൈജ്ഞാനിക ഘടകങ്ങൾക്ക് ഇടപഴകാൻ കഴിയുന്ന ഇടം. ആശോള വർക്ക്സ്പേസ് സിഖാത്തങ്ങളുടെ ചിത്രകാരിയാണ് ഒരു ലഭ്യത്മായ മാർഗ്ഗം, ബോധാവസ്ഥകൾ അബോധാവസ്ഥകളുടൊരു തലച്ചോറിൽ 'പ്രസിഡംബൾ' എന്ന പരിശീലനിക്കുക എന്നതാണ്, അതായത്, അപേക്ഷയും തലച്ചോറിൽ കുടുതൽ പ്രാപകമായി പ്രക്ഷേപണം ചെയ്യപ്പെടുകയും വൈജ്ഞാനിക പ്രക്രിയകളുടെ ഒരു വലിയ നിരയെ സ്വാധീനിക്കുകയും ചെയ്യുന്നതും സിഖാത്തത്തെ ഫണ്ടണ്ടാണ് വൈയിൻ ഹമേജിംഗ് (functional brain imaging) പിന്നുണയ്ക്കുന്നു, ഇത് ബോധപൂർവ്വത്മായ അറിവ് വ്യാപിക്കുന്ന, വ്യാപകമായ കോർട്ടിക്കൽ പ്രവർത്തനവുമായി (പ്രത്യേകിച്ചു ഹ്രേണാഹരിയുറ്റൽ, മീഡിയൽ ടെൻസറ മേഖലകളിൽ) ബന്ധപ്പെട്ടിരിക്കുന്നുവെന്ന് തെളിയിക്കുന്നു, അതേസമയം അബോധാവസ്ഥകൾ തലച്ചോറിലെ പ്രാഭേഡിക പ്രാഭേഡങ്ങളെ മാത്രം സജീവമാക്കുന്നു. വാസ്തവത്തിൽ, ഗാഡർിസ്, ജനറൽ അനസ്റ്റ്രേഷ്യൂ, കോമ തുടങ്ങിയ അബോധാവസ്ഥകൾ (frontoparietal) മേഖലയിലെ ഉപാപചയ പ്രവർത്തനത്തിൽ ഗ്രന്തമായ കൂറിവ് കാണിക്കുന്നു [9].

ഉന്നത്മായ സിഖാത്തങ്ങൾ ബോധാവസ്ഥയെ നിക്ഷകളുകൊഡായ ഒരു കുട്ടിയോട് ഉപമിക്കുന്നു - മുതിർന്നവർ അവനോട് പരിയുന്നതുകൊണ്ട് മാത്രം താൻ ഉണ്ടെന്ന് അറിയുന്ന ഒരാൾ. ഈ സിഖാത്തങ്ങൾ അന്തർഭീനമായി "മെറ്റാ" (meta) ആണ്, ഒരു വ്യക്തി തന്റെ അവസ്ഥയെക്കുറിച്ച് ബോധാവാനാരായിരിക്കുമ്പോൾ ആ മാനസികാവസ്ഥ ബോധപൂർവ്വത്മാണെന്ന് നിർദ്ദേശിക്കുന്നു. ഗണിതശാസ്ത്രത്തിലും (ജീവിതത്തിലും) നാം ഉപയോഗിക്കുന്ന സക്രിയകമായ സ്വഭാവത്തെ അനുസ്മർപ്പിക്കുന്നതിനാൽ, ഇതിനെ ട്രാൻസിറ്റിവിറ്റി (transitivity theory) എന്ന് വിളിക്കുന്നു. [10] ഈ സിഖാത്തങ്ങൾ ഉപയോഗിച്ച്, ബോധാവസ്ഥകളെ അബോധാവസ്ഥകളിൽ നിന്ന് പ്രത്യസ്തമാക്കുന്നത് എന്നാണ് വിശദിക്കിക്കുകയാണ് (pramamikakalakshayam) [10].

അവസാനമായി, ചർച്ച ചെയ്യുന്നതുത്ത് പ്രവചനാത്മക ഫ്രോസ്റ്റിംഗ് (predictive processing) (പിപി) (PP) സിഖാത്തത്തെ പറി ആണ്. പ്രവചനാത്മകമായ ഫ്രോസ്റ്റിംഗ്, നിങ്ങളുടെ കാലുകൾ താഴ്ത്തിനു അനുസരിച്ചു ടാപ്പ് (tap) ചെയ്യുന്നത് പോലുള്ള ഭേദനാംഗം സംബന്ധം ഉപയോഗിച്ച് മനോഹരമായി ഉഭാഹരിച്ചിരിക്കുന്നു. ഇവിടെ, നിങ്ങളുടെ മസ്തിഷ്കം താഴ്ത്തിലെ ക്രമ തിരിച്ചറിയുകയും അഭ്യന്തര താഴ്ത്തിന്റെ സമയം മുൻകൂട്ടി അറിയുന്നതിനും ഒരു ടാപ്പിന്റെ ചലനം മഹാഭോജി ഓട്ട്-പുട്ട് ഉൽപ്പാദിപ്പിക്കുന്നതിനും ഈ വിവരങ്ങൾ ഉപയോഗിക്കുന്നു. ഈ 'പ്രതീക്ഷ' സംഭവിക്കുന്നത് മുകളിൽ നിന്ന് താഴേക്ക്, താഴെ നിന്ന് മുകളിലേക്ക്, പ്രധാനമായും വിവിധ ഭിശയിലേക്കുള്ള സിംഗിൾ (സിംഗിപ്പിലും) പ്രതീക്ഷയെ വിശദിക്കിക്കൊള്ളുന്നു [11]. പ്രവചനാത്മക ഫ്രോസ്റ്റിംഗ് അവബോധത്തിന്റെ ഒരു സിഖാത്തമല്ലെങ്കിലും, അത് ശ്രദ്ധയിൽ ഒരു പ്രധാന പക്ഷ് വഹിക്കുന്നുവെന്നും അവബോധത്തിന്റെ ചില

പ്രവർത്തനങ്ങളെയും സിലബാന്റങ്ങളെയും കൂറിച്ച് വിലപ്പേട്ട വികശനം നൽകുമെന്നും അഭിപ്രായമുണ്ട് [12].

സൗഹ്യവും ബോധവും

ബോധവും അഭോധാവസ്ഥയും തമിലുള്ള വ്യത്യാസം വിശദിക്കിക്കാൻ ശ്രദ്ധിക്കുന്ന പല സിലബാന്റങ്ങളും, അതിനെ സ്പാസ്റ്റണ്ടുടെ സംഭവവുമായി ബന്ധപ്പെട്ടതാണ് ശ്രദ്ധിക്കുന്നു. എന്നിരുന്നാലും, ഇവയിൽ എതാണ്ട് മുഴുവൻ സത്യമെന്ന് ഇപ്പോഴും അനിയിത്തത്വമുണ്ട്; ഒരു പരക്ഷ സത്യാവസ്ഥയിൽ, ഈ സിലബാന്റങ്ങളുടെ പല ഭാഗങ്ങളുടെ സംയോജനമായിരിക്കാം. അതിൽ നിന്നൊന്നായകമായ ഒരു സിലബാന്റ ഇപ്പോൾമാണ്.

നിങ്ങൾ ഉറങ്ങുമ്പോൾ, നിങ്ങളുടെ ബോധ മനസ്സും ഉറങ്ങുന്നു. ഈ സമയത്താണ് അഭോധ മനസ്സ് നിയന്ത്രണം എറുടുകുന്നതും അതിരുകളില്ലാതെ സ്വത്തെ സംഭവമായി കുറയുന്നതും. ഇപ്പോൾ, അഭോധാവസ്ഥയിലുള്ള മനസ്സിന് ഇതിനകം നിലവിലുള്ള ഓർമ്മകളും അനുഭവങ്ങളും മാത്രമേ രീക്കലുകാൻ കഴിയും എന്നതിനാൽ, അരു ക്രമരഹിതയാണ് റാൻഡിംഗ് കാലിഡോസ്‌കോപ്പിക് പ്രാർശനത്തിൽ ആ ഓർമ്മകളുടെ കൂട്ടിയോജിപ്പിച്ച് പ്രാർശിപ്പിക്കുന്നു. ഒരു സപ്പനം, നിങ്ങൾ ഉണ്ടാണ് കഴിഞ്ഞതാണ്, ബോധം ഫൈവർ സീറ്റിൽ തിരിച്ചെത്തി, ആ സപ്പനങ്ങൾക്ക് സന്ദർഭം നന്ദികാണ് ശ്രമിക്കുന്നു. യാമാർത്ത്യത്തിൽ അർത്ഥമുള്ളവയെ അടിസ്ഥാനപ്പെടുത്തി. മറ്റൊളവയെ ഓർമ്മയിൽ നിന്ന് മങ്ങാൻ അനുവദിക്കുന്നു [13].

ബോധത്തിന് രണ്ട് ലഭ്യതയില്ലായ്മയാണ് സപ്പനം കാണാൻ ആവശ്യമായ അവസ്ഥ എന്ന് ഇപ്പോൾ വ്യക്തമായി കാണണം. ഉറക്കത്തിലുണ്ടിട്ടും നമ്മൾ സപ്പനം കാണാത്തത് എന്നുകൊണ്ടാണെന്ന് ഇത് വിശദിക്കിക്കുന്നു. നമ്മുടെ ഉറക്കച്ചക്രതിൽ, REM (ഇരു കണ്ണുകളുടെ ചപനം) ഘട്ടത്തിൽ, നമ്മുടെ മനസ്സ് ബോധത്തിന് ചെങ്ങലകളിൽ നിന്ന് മുക്തമാവുമ്പോൾ ആണ്, നമ്മൾ സപ്പനം കാണുന്നത്. കൂടാതെ, നമ്മുടെ രക്തസമ്മർദ്ദം കുറയ്ക്കുകയും ഹൃദയത്തിന് ഗെയും ശ്രസ്തന്തരിന് ഗെയും നിരക്ക് കുറയ്ക്കുകയും സ്വയേധ്യാ ഉള്ള പ്രവർത്തനങ്ങളെ വലിയതോതിൽ തള്ളിത്തുകയും ചെയ്തുകൊണ്ട് ഉണ്ടാനിരിക്കുന്ന ലോകത്ത് നമ്മുടെ സപ്പനങ്ങൾ നിരവേദ്ധുനിബ്ലാൻ നമ്മുടെ ശരിരം പ്രസിഡേണ്ടജിക്കലാലി(physiologically) ഉറപ്പാക്കുന്നു. എന്നിരുന്നാലും, അതിശയകരമനും പരയട്ട, ഈ സന്ദർഭത്തിൽ മന്ത്രിക്ക് പ്രവർത്തനം സൂചിപ്പിക്കാം, നിങ്ങൾ ഉണ്ടാനിരിക്കുമ്പോൾ ഉള്ളതിനേക്കാൾ ഉയർന്നേക്കാം! [14].

ഒരു സകിർണ്ണമായ പ്രശ്നം പരിഹരിക്കപ്പെടാതെ നിങ്ങൾ ഉറങ്ങാൻ പോകുന്ന എന്നാൽ രാവിലെ എഴുന്നേള്ക്കുമ്പോൾ, അതഭൂതകരമായി അതിന്റെ ഉത്തരം നിങ്ങൾ കണ്ണംതുകയും ചെയ്യുന്നു ഒരു സാഹചര്യം നിങ്ങൾ എപ്പോഴുകയും അനുഭവിച്ചിട്ടുണ്ടാ? അതാണ് നിങ്ങളുടെ അഭോധ മനസ്സ് ചെയ്യുന്നത് - കാര്യങ്ങൾ ഒരുമിച്ച് ചേർക്കുന്നു. മറ്റൊരു വികശണക്കാണിൽ നിന്ന് കാര്യങ്ങളെ നോക്കുന്നു. സപ്പനങ്ങൾ യാമാർത്ത്യത്തിൽ സർജ്ജാത്തകര വർഖപ്പിക്കുമെന്നും ഗവേഷണങ്ങൾ തെളിയിച്ചിട്ടുണ്ട് [14]. ഇതിനു കാരണം REM ഘട്ടത്തിൽ കുപ്രസിഥമായ ഉത്കണ്ഠം ഉള്ളവകുന്ന നോറാധിനിലാർഡ് (noradrenaline) ഹോർമോൺ നമ്മുടെ ശരിരത്തിൽ എക്കാലത്തെത്തയും താഴ്ന്ന നിലയിലായിരിക്കും. നിങ്ങളുടെ ബോധമനസ്സ് അടിച്ചുമർത്താണ് ശ്രമിച്ച പ്രധാനപ്പെട്ട ഓർമ്മകൾ

மநஸ்தில் கொள்கூவரான் ஹத் அலோயாய மநஸ்திர் ஶாத்தமாய அநந்திக்ஷங் நடக்குங் அதிர்காத், அலோயாயாவயம் ஏற்காத அஸ்யாயம்புகு அனுஸாரனகேட்குமுதல் வயங்கூப்பி, அத் பராமர்ஶிக்கூயோசி வோய் விரியக்குவான்!

'ஸமயம் எழிலூ முரிவுக்கும் ஸுவபெப்டுத்துமென் பரயெப்டுங்கு, பகேச எர்க்கர சவேஷன் ஸுப்பிரிக்கூந்த் உரகை ஸப்பந்தில் செலுவசிசு ஸமயமான் ஸுவபெப்டுத்துங்கர்.'

யோக்கர். மற்று வாச்கரை

மநஸ்திரையும் வோயதெற்றியும் குரிசு மழுகரியாவுங் (அலேக்கில் மழுக்கு அரியாமென் மழுக் கருதுங்) புதிய ஸிலாக்னைச் சீலூ விவரவும் நிரப்பேக்கபெப்டுங்கு. நிலாவிலுதல் ஸிலாக்னைலில் காருத்திர்க்கர ஸத்யமுணோ? அதே ஹனியும் களெத்தாநாக்காத வலி ஶக்தியும் நிழங்கிக்கூங்கோணோ?

பாரவோலி போலெ, ஸமயம் மாற்றமே பரயும்!

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ബോധകഷയത്തിനുപീനിലുള്ള ശാസ്ത്രപരമം The Neuroscience of Fainting

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Translated from English to Malayalam by Ashley Roby

സംഗ്രഹി (Summary)

ബോധകഷയം എന്നത് സമർപ്പകരമായ സാഹചര്യങ്ങളുള്ള ഒരുതരം പ്രതികരണമാണ്. മസ്തിഷ്കത്തിലേക്കുള്ള രക്തവിതരണത്തിന്റെ അഭാവം മൂലമാണ് ഈ സംഭവിക്കുന്നത്. മസ്തിഷ്കത്തിലെ നാഡികോശങ്ങൾക്കു അതിവിശ്വാസമായ തോതിൽ ഫ്ലൂകോസൂം ഓക്സിജൻും കിട്ടാതെവരുമ്പോൾ , അവ താൽക്കാലികമായി പ്രവർത്തനം നിർത്തുകയും അങ്ങനെ ബോധം നഷ്ടപ്പെടുകയും ചെയ്യുന്നു. രക്ത വിതരണം കുറയുന്നതിന്റെ കാരണങ്ങൾ വ്യത്യസ്തമായിരിക്കും . സാഹചര്യങ്ങളെ അഭിസ്ഥാനമാക്കി അഭ്യുക്തിൽ എന്നതിനിലും അടിസ്ഥാനപരമായ മെഡിക്കൽ അവസ്ഥ കാരണവും ആവാം.

ആരുപാം (Introduction)

തുടർച്ച.

എല്ലാവരും ശാസം അടക്കിപ്പിക്കുന്നു, മുതിർന്നവർ കൂതിച്ചുപായുന്നു. കാൽ ലിനിറ്റിനുശേഷം, കായികാധ്യാപകൾ അടുത്ത കൂസിലെ ആൺകുടിയെ മെമതാനവും കടന്ന് ഇതാ ആരുപ്പത്രിയിലേക്ക് കൊണ്ടുപോകുന്നത് നമർക്കാണുന്നു. എന്നാണ് സംഭവിച്ചതെന്ന് നമുക്കെല്ലാവർക്കും അറിയാം, "അവൻ ബോധകഷയും വിണ്ണോ? അതോ സ്കൂളിന്റെ ദിവസം തന്നെ??"

ബോധകഷയം എന്ന ആരുപാം നമുക്ക് പുതുമയുള്ള കാര്യമല്ലക്കിലും-സ്കീറിൽ അഭിനേതാക്കളുടെ നടക്കിയമായ ബോധക്കേളുകൾ (ലോകത്തിലെ ഒരുക്കുടം മനുഷ്യർക്ക്, ഇത് ശ്രദ്ധിയാണോ), സ്കൂളിൽ അതിരാഹിവെ തന്നെ വീഴ്ന്നുന്ന ചിലവിട്ടുകളാർ-ഇതിനിടയിൽ എല്ലാം മസ്തിഷ്കത്തിലെ സംഭവവികാസങ്ങൾ പിടിച്ചോടുകയാണ്. അതുകൊണ്ടാണ് രാവിലെത്തെ അസാംഖ്യി സമയത്ത് സുര്യൻ നിങ്ങളെ തള്ളംതിയതാണോ അതോ മൊത്തത്തിൽ മറ്റൊന്തകിലും പ്രശ്നം ആണോ എന്ന് കണ്ടുപിടിക്കാൻ ശാസ്ത്രജ്ഞന്മാർ ശ്രമിക്കുന്നു.

ശരീരത്തെ ഒരു ഫൂഡ് ഇൻ ടിപി ആയി കരുതുക. ഒരു നിമിഷം ആ പെട്ടെന്ന് ഉജറിപോരുന്നു, ചെപ്പെയ്ക്കുതി വിതരണത്തിൽനിന്നും വിച്ഛേദിക്കേപടുന്നു, ടീപി ഓഫ് ആകുന്നു. ബോധകഷയം പലതും ഇതുപോലെയാണ്. ബോധകഷയം അമ്പവാ ചെവദ്യശാസ്ത്രപരമായി പറഞ്ഞാൽ "സിൻകോപ്": കഷണിക നേരത്തെക്ക് മാത്രമുള്ള ബോധം നഷ്ടപ്പെടുന്ന ആണ്. പെട്ടെന്ന് തിരിച്ചുകൂടുന്നു, ചെവദ്യത്തിവിതരണം പുനരാരംഭിക്കുന്നു, അതോടെ ടീപി ഇതാ പഴയപോലെ പ്രവർത്തനനിരതം ആകുന്നു.

തലാശുഠാറിലെ കോശങ്ങൾക്ക് ഫ്ലൂകോസ്എം ഓക്സിജൻും കൊണ്ടുവന്നു, തലാശുഠാറിന്റെ പവർ സംഭൗഢി ആയി പ്രവർത്തിക്കുന്നത് രക്തമാണ്.

രക്തക്കുറവാൺ പലപ്പോഴും ബോധക്ഷയത്തിന് രെ പ്രധാന കാരണം. രക്തസമ്മർദ്ദം കുറയുന്നതിനാലോ അരബ്ലൂക്കിൽ ഹ്രദയമിടിപ്പ് കുറയുന്നതിനാലോ ക്രമരഹിതമായ ഇം രക്തക്കുറവ് ഉണ്ടാകാം, ഇൽ തലച്ചോറിലേക്കുള്ള രക്തപ്രവാഹത്തെ തെസ്റ്റപ്പുത്തുന്നു. ബോധക്ഷയം ചിലപ്പോൾ മറ്റ് ചില പക്ഷണങ്ങളോടൊപ്പം ഉണ്ടാകാറുണ്ട് ; മർദ്ദി, തലകറക്കം, വിളറിയ ചർമ്മം, ശരീരത്തിന്റെ മെത്തനിലുള്ള ബലപെന്നീനു, ജലദാഷം, വിയർപ്പ്, കാഴ്ച മങ്ങൽ, കാഴ്ചയിലും മാനസികമായും ശ്രദ്ധ കേന്ദ്രീകരിക്കാനുള്ള ബുദ്ധിമുട്ട്, സ്ഥാനം നിലനിർത്താനുള്ള പരാജയം മുതലായവ. അവയെല്ലാം ബോധക്ഷയത്തിന് രെ ഇൻകമിംഗ് എപ്പിസോഡിനു സുചനകളാകാം. [1].

ബോധക്ഷയത്തിന് കാരണങ്ങളും രഹണങ്ങളും:

ബോധക്ഷയത്തിന് എറ്റവും മുഖ്യകാരണക്കാരൻ ഹ്രദയമാൺ, കുടുതൽ പുക്തമായി പറഞ്ഞാൽ, ഹ്രദയം എത്ര പേരതിലേം മരഗതിയിലോ സ്പ്രാക്കുന്നു എന്നതാണ് പ്രധാനം. ഹ്രദയം അതിന്റെ പതിവ് ലണ്ട് ധാരാ താഴ്ത്തിൽ (അറിമീമിയ |Arrhythmia| എന്ന വിളിക്കുന്ന ഒരു അവസ്ഥാ-മിറ്റിക്കുന്നിലേക്കിൽ, അത് മറ്റ് അവയവങ്ങൾക്ക് ഓക്സിജനും ട്രൈക്സൈറ്റും കുറക്കുന്നു. സ്റ്റിക്കേഴ്സ് പരസ്യം ഓർക്കുന്നുണ്ടോ നിങ്ങൾ ? വിശകുന്ന ഒരു എം.എസ്. ഡോനി നമ്മൾ “വിശകുന്നു” എന്ന വിളിക്കുന്നത്: ഭേദ്യവും വിശപ്പും. “വിശകുന്നു” എന്നതിന്റെ തലച്ചോറിന്റെ പതിപ്പ് അബോധാവസ്ഥയിലേക്ക് പോകുന്നു. മന്തിഷ്ക്കത്തിലെ നാഡിത്തരസ്യകൾക്കു ട്രൈക്സൈറ്റും ഓക്സിജനും ക്രിട്ടാതവരവുംപോൾ, അവ പ്രവർത്തനം നിർത്തുന്നു, അതോടെ അധാരാളം ബോധക്ഷയത്താക്കയും ചെയ്യുന്നു.

മറ്റാരു തരത്തിലുള്ള ബോധക്ഷയം ക്ഷിപ്പപ്രതികരണ ശേഷിയ സംബന്ധിച്ചിരുന്നു. ചിലതികളെ ദേഹക്കുന്ന ഒരു സുപ്രാത്യിനക്കുറിച്ച് ചിന്തിച്ചുനോക്കുക. അവരുടെ അരികിലെ ഭിത്തിയിൽ ചിലന്തി ട്രായുന്നത് കാണുന്നും, ആ സുപ്രാത്യം എന്തുചെയ്യുമെന്ന് നിങ്ങൾക്ക് ഉണ്ടാക്കാൻ കഴിയുമോ? ഇവിടെ, ശരീരത്തിന് രക്തപാക്കമണം നിയന്ത്രിക്കാൻ കഴിയില്ല, അതിനാൽ നമ്മുടെ തലച്ചോറിലെ നാഡിക്കോശം ട്രൈക്സൈറ്റും ആവശ്യപ്പെടുന്നു. ഇം ആവശ്യം നിറവേറ്റപ്പാതെ വരുമ്പോൾ, മന്തിഷ്കം താൽക്കാലികമായി നിർത്തുകയോ ഡ്യാൻഡ്‌ബെ മോഡിലേക്ക് പോകുകയോ ചെയ്യുന്നു. മുതൽത്തിലുള്ള ബോധക്ഷയത്തിന് ഉപാധികാരങ്ങളുണ്ട്.സാധാരണ ബോധക്ഷയം, സാഹചര്യപരമായ ബോധക്ഷയം എന്നിങ്ങനെ ടിഗറുകൾ അമവാ അടിസ്ഥാനകരണങ്ങളും സംബന്ധിച്ചിട്ടുള്ളവയാണ് ഇവയെല്ലാം.

സാഭാരണയായി നമ്മൾ കാണുന്ന മോഹാലസ്പും(അരബ്ലൂക്കിൽ വെദ്യശാസ്ത്രങ്ങളും വിളിക്കാറുള്ള വാണോവഗൽ നിംഫോസ്/ Vasovagal syncope) വെകാരികവും ശാരീരികവുമായ ഫേശം മുലമാണ്. കാമുകൾ അപകടത്തിൽ പെട്ടു എന്ന് കേട്ടാൽ വിശാൺ പോകുന്ന നടയും, താൻ അറിയാത്ത ഒരു ഗ്രാന്റ് സഹോദരൻ തനിക്കുണ്ടെന്ന് അറിയുന്നവനും,

അതും അരബ്ലൂക്കിൽ വളരെ നേരം വെയിലത്ത് നിന്ന് തള്ളുന്ന വിഴുന്നവനും വാണോവഗൽ നിംഫോസ് എന്ന അവസ്ഥയാണ് അനുഭവിക്കുന്നത്. ഇം സാഹചര്യത്തിൽ, വികാരങ്ങളും സമർദ്ദം അമവാ അസഹന്നയമായ ശാരീരിക സമർദ്ദം രക്തസമ്മർദ്ദം കുറയുന്നതിന് കാരണമാകുന്നു.

പ്രത്യേക സാഹചര്യങ്ങളിലേക്ക് പ്രവർത്തനങ്ങളിൽ ഉൾപ്പെടുത്തേണ്ട മാത്രം ബോധക്ഷയം സംഭവിക്കുന്നതിനെ സാഹചര്യപരമായ മോഹാലസ്പും അമവാ

സാഹചര്യപരമായ സിസ്റ്റേക്കാപ്പ് എന്ന് പിളിക്കുന്നു. നിങ്ങൾ ഒരു മാരത്തൻ ഓട്ടക്കാരൻ ആണെന്ന് കരുതുക, ഓരോ തവണെയും നിങ്ങൾ ഫിറിഷ് ലെൻ കടക്കുമ്പോൾ നിങ്ങൾ ബോധാംകുന്നു. മറ്റാരാൾ തള്ളുന്ന പിശുന്നത് കണ്ണപ്പോൾ ബോധാഹിതനാഥ ഒരാളും യോക്കർമ്മാർക്ക് ഗാജരാക്കിയതാണ് പിപിത്രമായ ഒരു സംഭവമാണ് [3]. അതിശയകരമെന്നു പറയുടെ, ഈ വൃക്തിക്ക് ഹ്യൂയിലിഡിപ്പ് ഉണ്ടായിരുന്നില്ല, കാരണം അവരുടെ ഹ്യൂയിം അക്ഷരാർത്ഥത്തിൽ നിലച്ചിരുന്നു. ഭാഗ്യവഹാൽ, ഈ രോഗി രക്ഷപ്പെട്ടു. ഇതിപ്പോ, ഏറ്റവും കാൽപ്പനികമായ (പ്രണയ സിനിമകളിൽ പോലും ആരും കാണാത്ത കാരുമാണ്!

ആർക്കൈകിലും മുൻപേതെന്ന ഭത്തരം അവസ്ഥയുണ്ടെങ്കിൽ, പ്രത്യേകിച്ച് നാധിസംബന്ധമായ രോഗങ്ങളോ പ്രശ്നങ്ങളോ ഉണ്ടെങ്കിൽ, ബോധക്ഷയം അവർക്ക് സർവ്വസാഭാരംമായ ഒരു അവസ്ഥയായിരിക്കും. ബോധക്ഷയം ഉണ്ടാക്കാൻ കരണമാക്കത്തക വിധം എന്നാണ് നമ്മുടെ മസ്തിഷ്കത്തിൽ സംഭവിക്കുന്നത്? ശാസ്ത്രജ്ഞത്തെക്ക് നിന്നുംശയം പറയാൻ കാരണങ്ങൾ ഇല്ലെങ്കിലും, അവർക്ക് കൂടച്ച് അനുമാനങ്ങളുണ്ട്.

സെറോറോണിൻ [Serotonin] എന്ന അന്തർഗ്രാഫി സാവം (അമവാ ഹോർമോൺ [hormone]), ഉറക്കത്തിനും മാനസികാവസ്ഥയ്ക്കും അതിനോടൊപ്പം ഒരു നാധികോശത്തിൽ നിന്ന് മറ്റാന്നിലേക്ക് അടയാളംമുണ്ടാക്കിയാണ് അമവാ സീറോറോണിൻ റിലേചേരുന്നതിനും ആവശ്യമാണ്.

ഉറക്കത്തിനു ആവശ്യമുള്ള മല്ലാറോണിന് എന്ന ഹോർമോൺ സെറോറോണിനിൽനിന്നും ശരിരം ഉല്പാദിപ്പിക്കുന്നു. ഉറക്കം ഒരുത്തരം അഭോധാവസ്ഥയാണെല്ലാ. ഇൽ റിലിച്ചറിയുന്നോൾ, ബോധക്ഷയത്തിൽ സെറോറോണിന്റെ പക്ക വിസ്മരിക്കാനാവില്ല. പാരാസിപ്പതിറ്റിക് നാധിപ്പവസ്ഥയുമായും സെറോറോണിനും പങ്കുണ്ട്. നിവബത്രീക് നാധിപ്പുണ്ടെങ്കിൽ സമർദ്ദപൂർത്തിമായ സാഹചര്യങ്ങളും ധ്യാകമം പാരനിപതിക് നാധിപ്പുണ്ടെങ്കിൽ ശാന്തമായ

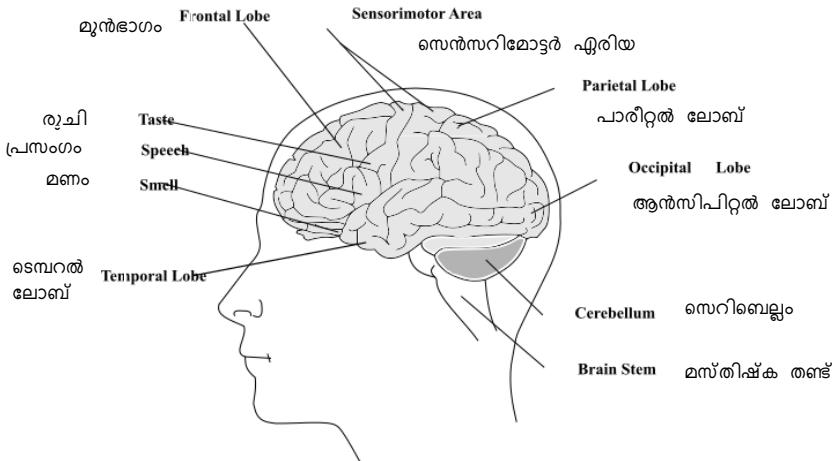
സാഹചര്യങ്ങളും (ക്രീക്കരിക്കാൻ ഉത്തരവാദികളുണ്ട്) [4]. മറ്റാരു പിഡിത്തിൽ പറഞ്ഞാൽ, സെറോറോണിന്റെ അളളിൽ അപത്രിക്ഷിതമായി ഒരു മാറ്റം വന്നാൽ ഹ്യൂയിലിഡിപ്പ് താഴ്ന്ന പോകും. ബോധക്ഷയത്തിൽ സെറോറോണിന്റെ പക്ക വിവാദമായി തുടർന്നുകൊണ്ടിരിക്കുന്നും കൂടുതൽ കൂടുതൽ ആവേശകരമായ ഗവേഷണങ്ങൾ ഈ പിഡിയത്തിൽ സുക്ഷ്മ പരിക്ഷണങ്ങൾ നടത്തിവരുന്നു!

സമർദ്ദം ബോധക്ഷയത്തിന് കരണമാകുമെന്നും ഗവേഷകൾ കണ്ണുപിടിച്ചു [5]. 2009-ൽ നടത്തിയ ഒരു പദ്ധതിൽ, ബോധക്ഷയത്തിനുള്ള ചികിത്സയിൽ സന്ദരിക്കുന്ന ക്ഷേമത്തിന്റെ സ്വാധിനം സൻരേപ് സ്ഥാപിച്ചുണ്ട്. മാനസികാരാഗ്ര വെകല്പങ്ങളാൽ ബാധിതരായ ആളുകൾ, ആഫത്തിലുള്ള ദുരിതം അനുഭവിക്കുന്നവർ, ബോധക്ഷയം പതിവായി അനുഭവപ്പെടുന്നതായും ചികിത്സകളോട് നന്നായി പ്രതികരിക്കാതെ വരുന്നതായും അവർ കണ്ണത്തി. പതിവായി ബോധക്ഷയം ഉള്ള രോഗികളെ നിരിക്കിച്ചിട്ടുരുക്കാണു് ഒരു ചികിത്സാലയ സംബന്ധിയായ പഠനം അതെ വർഷം തന്നെ നടന്നു [6]. തീവ്രമായ ഉൽക്കടവ്യമ നേരിട്ടുന്ന രോഗികൾക്ക് വിശദിക്രിക്കാൻ കഴിയാത്ത ബോധക്ഷയാവസ്ഥകൾ ഉണ്ടാകുന്നതായി രചയിതാക്കൾ ഇവിടെ കണ്ണത്തി. അതായത്, പലപ്പോഴും ബോധക്ഷയം സംഭവിക്കുന്നത് ചികിത്സ ആവശ്യമായിട്ടുള്ള ചീല അടിസ്ഥാന മാനസിക അസ്ഥാനതയുടെ

ലക്ഷണമായിരിക്കാം . ബോധവൽക്കരണം നടത്തുകയും ചികിത്സ ആരംഭിക്കുകയും ചെയ്തതിന് ശേഷം അരേ പഠം ചില രോഗികളിൽ പുരോഗതി കണ്ടുതുടങ്ങി. കൗമാരകലാർക്കിടയിൽ നടത്തിയ ഒരു അനേപാഷണത്തിൽ, ബോധക്ഷയം ഉള്ള കുട്ടികളെ പലപ്പൊഴും അവരുടെ സമ്പ്രായക്കാരുമായി താരതമ്പ്രപ്രടുത്തബോൾ താഴ്ന്ന മാനസിക-ജീവിത നിലവാരമുള്ളവരാണെന്ന് ശവേഷകൾ കണ്ടെത്തി [10].

ഒരാൾക്ക് ബോധക്ഷയം സംഭവിക്കുമ്പോൾ തലച്ചോറിനുള്ളിൽ എന്നാണ് സംഭവിക്കുന്നതെന്ന് ഇപ്പോഴും ശവേഷകൾക്ക് വ്യക്തമായി അറിയില്ല. 59 സന്നദ്ധപ്രവർത്തകരിൽ ബോധക്ഷയം കൂതിമമായി വരുത്തിക്കൊണ്ട് തലച്ചോറിലേക്കുള്ള ഓക്സിജൻ വിതരണം കുറയുന്നതിന്റെ ഫലങ്ങളുകുറിച്ച് സെറ്റിബേൽ ഹൈപ്പാക്സിയ എന്ന് പിളിക്കപ്പെടുന്ന അവസ്ഥ ടി പറിക്കുവാൻ ഒരു പരിക്ഷണം നടത്തി. റിവേഴ്സിബിളിറ്റായ ഈ ബോധക്ഷയം അവസ്ഥ ആളുകളെ വിഭാഗിയിലാക്കുകയും (സ്റ്റ്രൈ- ശവണ വിഭാഗി), നിലത്തു വിഴ്ത്തുകയും, പേശികളുടെ അന്വയനത്തിനായ ചലനം (മയോക്സിണസ്) ഉണ്ടാകുകയും ചെയ്യുന്നുവെന്നും പരിക്ഷണപദ്ധതിൾ വെളിപ്പെടുത്തി. ബോധക്ഷയം സംഭവിക്കുമ്പോൾ മന്തിഷ്ക് പ്രവർത്തനങ്ങൾ എങ്ങനെ പരിഷ്കരിക്കപ്പെടുന്നുവെന്ന് തിരിച്ചറിയാനും മന്തിഷ്ക് തരംഗങ്ങൾ കണ്ടെത്തി വിശദം ചെയ്യാനും ശവേഷകൾ EEG (തലച്ചോറിന്റെ വെദ്യുത പ്രവർത്തനത്തിന്റെ സ്കാൻ) ഉപയോഗിക്കുന്നു.

നിങ്ങളുടെ പാംപുസ്തകങ്ങളിൽ നിങ്ങൾ കാണുന്ന പെപ്പുകളുടെ കുറുക്കുവഴി പോലെ കിടക്കുന്നതാണ് തലച്ചോറിന്റെ കോർട്ടിക്കൽ മേഖല (ചിത്രം 1). ഈ മേഖലയാണ് അന്വയനത്തിനായ ശാരിരിക ചലനങ്ങളെ സാധാരണയായി നിയന്ത്രിക്കുന്നത്. ഈ പ്രത്യേക പരിക്ഷണത്തിൽ, ഈ ചലനങ്ങളെ മന്തിഷ്കക്കണ്ഡം (Brainstem- അമവാ മന്തിഷ്കക്കത്തെ സുഷും നാഡിയുമായി ബന്ധിപ്പിക്കുന്ന ഉള്ള നിൽക്കുന്ന ഭാഗം) നിയന്ത്രിക്കുന്നതായി ശവേഷകൾ കണ്ടുപിടിച്ചു. ഇത് 1994ൽ പ്രസിദ്ധികരിച്ചതാണെങ്കിലും പിന്നീട് ഇക്കാര്യത്തിൽ കൂടുതൽ അനേപാഷണം നടന്നിട്ടില്ല.



ചിത്രം 1: തലാദ്ധോറിന്റെ ഭാഗങ്ങൾ. നാല് ലോബുകൾ (ഫ്രണ്ടൽ, പാരിറ്റൽ, എസിപ്പൽ, ഓക്സിപിറ്റൽ) സെറിബ്രൽ കോർട്ടുകൾ അല്ലെങ്കിൽ കോർട്ടികൽ മേഖല ഉണ്ടാക്കുന്നു. മന്തിഷ്ക് തണ്ട് കോർട്ടുകൾ താഴെ നിന്ന് ഉയർന്ന് സൂചിപ്പം നാഡിയായി വ്യാപിക്കുന്നു.

(Image credit: Ozhank and https://openclipart.org/detail/121615/human-brain-by-ozhank)

ബോധക്ഷയം മനുഷ്യജീവിതത്തിന് പ്രതികുലവും ഹാനികരവുമാണെങ്കിൽ പിന്നെ എത്രകൊണ്ടാണ് ഇത് സംഭവിക്കുന്നത്? ഒരുക്കാരും പറയുവാണെൽ, സ്കീറ്റിംഗിലെ അഭിനേതരകൾ മാത്രമേ ബോധക്ഷയിൽനിന്ന് സ്വഭാവമുണ്ടാണ്. എന്തെങ്കിലും നേരുന്നുള്ളൂ. പിന്നെ എത്രകൊണ്ടാണ് സമർദ്ദപൂർത്തമായ സാഹചര്യങ്ങളെ നേരിടാൻ ശരിരം മരും പാരിഹാരം കൊണ്ടുവരാത്തത്? തിനില്ലെങ്കാണ് മാനസികമായി സ്വയം തയാറാക്കേണ്ട ഒരു നിലിഷ്ടതിൽ നമ്മുടെ മനസും ശരിവും ഒരു നിലിഷ്ടതെക്ക് നിലയ്ക്കുന്നത് പിചിത്രമായി തോന്നുന്നു, അല്ലോ?

പരിണാമപരമായ വിക്ഷണകോണിന്നിനു വിശകലനം ചെയ്താൽ, ബോധക്ഷയം ഒരു പെദ്ദെശാസ്ത്രസംബന്ധിയായ ആരോഗ്യപ്രശ്നം എന്നതിലാലും ഒരു പൊരുത്തപ്രശ്നമാണ്. തുദ്യകാല നിഖാരങ്ങൾ രക്തം-കുത്തിപ്പയ-പ്രവൃക്കുകൾ (Blood-Injection-Injury type specific phobia or BIITS phobia). എന്നിവയോടുള്ള ആസാദാരണ ഭീതിയായിട്ടാണ് ഇതിനെ തരംതിരിച്ചതു. ഒരു പ്രക്തി മുന്ന് തരത്തിലുള്ള പ്രതികരണങ്ങൾക്കാണുക്കുന്നു: ഭയം, തിരസ്കരണം, ബോധക്ഷയം-ഖവയോല്പാം തന്നെ പൂർവ്വിക മനുഷ്യർ വികസിപ്പിച്ചിട്ടുണ്ടാവാൻ സാധ്യതയുള്ളൂ അതിജീവന സഹജാവബോധവുമായി പൊരുത്തപ്പെടുന്നു. ബോധക്ഷയം വിശുദ്ധിക്കുന്നതും, മരും മനുഷ്യനാൽ ശുരൂതരമായി പാരിക്കേൽക്കപ്പോന്നു കൊല്ലപ്പോന്നു ഉള്ള സാധ്യത ചുരുങ്ങുന്നു. യുഖങ്ങളിൽ ഒന്നും പാക്കുകുന്നില്ലെങ്കിലും പിടിക്കപ്പെടാനും

പീഡിപ്പിക്കപ്പെടാനും സാധ്യവെയുള്ള സ്ത്രീകൾക്കും കൂട്ടികൾക്കും സമർപ്പണാഹചര്യങ്ങളിലുള്ള ബോധക്ഷയം അനുകൂലപെടുന്നിരിക്കാം [11].

പുർവ്വികൾക്കു ബോധക്ഷയം എന്നത് അപകടങ്ങളും അപ്രിയമായ സാഹചര്യങ്ങളും ഒഴിവാക്കാനുള്ള മാർഗമായിത്തീർന്നു. ഇതരരം റീതിയിൽ പ്രതികരിക്കുന്നതിലൂടെ, അപകടകരമായ സാഹചര്യങ്ങളിൽ നിന്ന് രക്ഷപ്പെടാനുള്ള സാധ്യത അവർ വർദ്ധിപ്പിച്ചു. സമയവും ഉഞ്ജാവും ലഭിക്കുവാനും ഒന്നവിൽ ജീവൻ തന്നെ സംരക്ഷിക്കുവാനും ഇത് ഉപകാരപെട്ടു[11].

അപകടസമയത്ത് അല്ലെങ്കിൽ സമർപ്പ സമയങ്ങളിൽ സ്ത്രീകൾ പുരുഷമാരേകാൾ ഉയർന്ന അളവിൽ മോഹാലസ്യപ്പെടുന്നതായി ഗവേഷണങ്ങൾ വെളിപ്പെട്ടുതുന്നു. വൈദ്യശാസ്ത്രപകാരം, സ്ത്രീകളിലെ രക്തസമർപ്പം പുരുഷമാരേകാൾ താഴ്ന്ന തോതിൽ ആയതുകൊണ്ടാവാം എങ്ങനെ കണ്ണുവരുന്നത്. ലെംഗ്രിക് ഹോർമോൺുകളുടെ നീയതിനാം ഇതിന് കാരണം[12].



ചിത്രം. 2 ഷേക്സ്പീയറുടെ മച്ച് അഡോയിൽ നിന്നുള്ള ഫീറോ, തനിക്കെതിരായ ആരോപണങ്ങൾ കേട്ട മയങ്ങുന്നില്ല.

Image Credit: Elmore and <https://journey-and-destination.blogspot.com/2013/09/shakespeare-scenes-in-art.htm>

രത്നചുരുക്കം (Retrospection)

ബോധക്ഷയം ഗുരുതരമായ ഒരു പ്രശ്നമാണെങ്കിലും, ജീവൻ രെയും ഉഞ്ജത്തിന്റെയും സംരക്ഷണത്തിനായി ശരിരം രൂപകർപ്പന ചെയ്തിരിക്കുന്ന പരിണാമപരമായ ഒരു വികസനവുമായി ഇതിനെ വീക്ഷിക്കാം. എന്നിരുന്നാലും ഇത് ആധുനിക കാലഘട്ടത്തിൽ ശാരിരിക-മാനസിക-ഭൌമായ ബലഹീനതയുടെ അടയാളമായി ആണ് കാണുന്നത്, കാരണം ഭൂമിയിൽ ഇനി വേദ്യക്കാരോ കാടുകളിലും ഭക്ഷണം തേടിയലയുന്നവരോ ബാക്കിയില്ല. പുർവ്വികരുടെ ഈ അതിജീവന സംബന്ധം, ഇപ്പോൾ ശരിരത്തിന്റെ വിവിധ ഭാഗങ്ങൾ സമർപ്പം നേരിട്ടുന്ന എന്നതിന് ദുർദാന്തമാണ്.

എന്നിരുന്നാൽ തന്നെയും സിൻകോപ്പിന്റെ ഒരു എസ്പ്രിസോയ് സംഭവിക്കുന്നത് പ്രശ്നത്തിന്റെ മൂലകാരണം തിരിച്ചറിയാനും പരിഹരിക്കാനും വെദ്യശാസ്ത്രജ്ഞൻക്കും സഹായകരമാകും. ഇങ്ങനെന്നെല്ലാക്കെ നോക്കുമ്പോൾ, ബോധാംകെട്ട് വീഴുന്നത് കൂഴപ്പമില്ലെങ്കിലും, ശരിക്കും അതിനേതെ ശരിയല്ല.

സാഹിത്യരചനകളിലും പൊതു സംസ്കാരത്തിലും ബോധക്ഷയത്തെ [Fainting] സാധാരണയായി ബോധകേട്ട് അമൈവാ മോഹാലസ്യം [Swooning] എന്നാണ് വിളിക്കാൻ. ആമയകുഴപ്പങ്ങൾ, ശൃംഖലാചനകൾ, വിഭാഗങ്ങൾ എന്നിവയെ ചുറ്റിപ്പറിയുള്ള വില്യും ഷേക്സ്പീയറുടെ മച്ച് അഡോ എബ്രൗം [Much Ado About Nothing] എന്ന കൃതിയിൽ, കമയിലെ നായികമാരിൽ ഒരാളായ ഹിറോ, തനിക്കെതിരെയുള്ള ആരോപണങ്ങളിൽ കേട്ട തള്ളനുവീഴുന്നു(ചിത്രം 2). വില്യും ഷേക്സ്പീയർ ബോധക്ഷയം ഒരു ഷോട്ട് ഉപകരണമായി ഉപയോഗിക്കുന്നത് വിക്കണ്ടാറിയൻ സമുഹങ്ങളിൽ നിലനിന്നിരുന്ന സംസ്കാരത്തിന്റെ പ്രതിഫലനമായിരുന്നു ഉദാഹരണത്തിന്, ദുർബാഗ്രഥതകൾ തങ്ങളെ എത്രമാത്രം ബാധിച്ചുവെന്ന് ഉന്നംബേക്കാടുകുന്നതിനും അത്തരം സാഹചര്യങ്ങളിൽ ബോധാംകെടണമെന്നു പോലും ചിലപ്പോൾ പ്രതിക്രിയിരുന്നു.

ആരോപണങ്ങൾ മാത്രമല്ല പ്രണയം, മരണം, പുറ്റുകാലങ്ങളിലെ രഹസ്യങ്ങൾ എന്നിവയെല്ലാം പുറത്താക്കുമ്പോൾ അള്ളകൾ തള്ളനുപോകുന്ന സന്ദർഭങ്ങൾ മരുകാലവല്ലത്തിന്റെ സാംസ്കാരിക ചരിത്രതാടു രചനകളിലും മറ്റും നിരഞ്ഞുനിൽക്കുന്നു. ചാർസ് ഡിക്കൺസിന്റെ ബ്ലീക്ട് ഹാസിൽ നായിക വേഡി ഡെയ്ലാക്ട് കർന്നാധാരം ചെയ്ത ഭൂതകാലത്തെത്തക്കുറിച്ച് മരക്കാനും മരയ്ക്കാനുമായി ബോധാംകെട്ടു വീഴുന്നുണ്ട്.

ഹെമിനിസ്റ്റ് എഴുത്തുകാരിയായ എബ്രൗം കാർട്ടറിന്റെ വാക്കുകളാണ് ബോധകേടിനെ എറുവും നന്നായി വിവരിച്ചത് എന്ന് തോനുനു, അവളുടെ പ്രശ്നംസ നേരിയ നിരുപക കൃതിയായ 'ദ ബ്ലീഡി ചേസർ' [The Bloody Chamber] എന്ന നോവലിൽ, അവർ എഴുതുന്നു, "രക്തരുക്ഷിതമായ ആ അന്ധയുടെ ഭയാനകമായ വെളിപ്പെടുത്തലിന് ശേഷം, അവൻറെ ആർദ്ദമായ രൂപമാണ് എന്ന തളർത്തികളുണ്ടത്.".

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Bengali

অঙ্গৰ্ধ এবং কিভাবে তা আমাদের মস্তিষ্কের কার্যকলাপ বুঝতে সাহায্য করে

Blindsight, and how it helps us see the workings of the human mind

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Translated from English to Bengali by Ankush Chakraborty

সারসংক্ষেপ

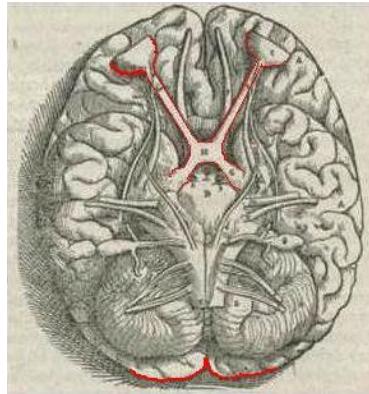
মন্তিষ্কের যে অসাধারণ তথ্য সংগ্রহ ক্ষমতা এবং সেই ক্ষমতার দ্বারা পৃথিবী কে উপলব্ধি করা তাঁর ছাপ আমরা কিছুটা বুঝতে পারি ব্লাইন্ডসাইট এর মতন এক কোতৃহলী ব্যাধির দ্বারা। নিউরোলজি, নিউরোসাইন্স এবং সাইকিয়াট্রির উপরে বড় বড় প্রশ্নের উত্তরের শুরু হয় ব্লাইন্ডসাইটের মতন ব্যাধির মাধ্যমে। নিচে লেখা দ্রব্যের দ্বারা ব্লাইন্ডসাইট ও ব্লাইন্ডসাইট কিকরে মানুষের মন্তিষ্ক বুঝে সাহায্য করে তার সারসংক্ষেপ উল্লেখ করা আছে।

ল (গোপনীয়তার কারণে নাম উল্লেখ করা হয় নি) তাঁর হাতের ছড়ি একজন ডান দিকে থেকে মানুষ কে দিয়ে দিল। সম্পূর্ণভাবে একজন অঙ্গ ব্যক্তি ল কে একটি ঘরের দৈর্ঘ্য অতিক্রম করতে বলা হয়েছে যার মধ্যে রয়েছে বেশ কয়েকটি আসবাবপত্র, এবং তা করতে হবে কেনো সাহায্য ছাড়ায়। শুনতে খারাপ লাগলেও, গবেষক বা তাদের উত্তর কিন্তু এই থেকেই পেয়েছে। দেখা গেছে ঘর টির বিষয়ে অসচেতন হয়েও ল ঘরটি কে সুন্দর ভাবে অতিক্রম করেছে, যেন সে কখনোই অঙ্গ ছিল না।

যদি আপনাকে চোখে কাপড় বেঁধে দেওয়া হয় এবং বলা হয় ল এর মতন একটি ঘর হেঁটে অতিক্রম করতে, আপনি এলোমেলো ভাবে এগিয়ে যাবেন এবং চেষ্টা করবেন যাতে কেনো রকম আসবাবপত্রে যাতে আপনি ধাক্কা না খান। ল এর এই অবস্থার পেছনে রয়েছে ভয়ঙ্কর স্ট্রেক যা তাঁর প্রাথমিক দৃশ্যমান বহিবাহন কে ফিল্টার করেছে এবং তাঁকে সম্পূর্ণভাবে অঙ্গ করে তুলেছে। সারা জীবনের জন্য চোখে কাপড় বেঁধে নিয়েছেন ল এবং তিনি বিশ্বাস করেন উনি সম্পূর্ণ ভাবে এলোমেলো পথে হেঁটে চলেছেন এবং পরীক্ষা-নিরীক্ষা করে চলেছেন, তবুও উনি নির্বি঱্বে নিজের পথ অতিক্রম করেছেন শত বাধাৰ মাঝেও।

নিচে দেওয়া ছবির দ্বারা বোঝানো হয়েছে মন্তিষ্ক আমাদের কি করে সাহায্য করে দেখবার ক্ষেত্রে। ছবিটি তে দেখানো হয়েছে সেই সকল সার্কিট যারা দৃষ্টির সঙ্গে যুক্ত, যাকে বলা হয় দ্বিজুয়াল পাথওয়ে। রোটিনার উপর তৈরি হওয়া ছবি যে আবেগের সংষ্ঠি করে তা আইবল হতে অপটিক নার্ভ দ্বারা মন্তিষ্কের সঠিক জায়গায় পৌঁছে দেয়, অপটিক নার্ভ হল একধরনের তার যাকে আমরা নিউরোপস(Neurons) ও

বলে থাকি। অপটিক নার্ভ, অপটিক কায়েসমা বা ক্রসিং পার করে অপটিক্যাল রাস্তা দিয়ে মস্তিষ্কের বিভিন্ন অঞ্চলে সংকেত পোঁছে দেয়।



চিত্র 1 ভিজ্যুয়াল পাথওয়ে, আন্দ্রেয়াস ভেসালিয়াস তার বই De Humani Corporis Fabrica Libri তে ক্ষেত্র করেছেন।

ভিজ্যুয়াল পাথওয়ে তে ঘটে যাওয়া কোনোরকম সমস্যা আবেগের চলাচলের ক্ষেত্রে বাধা তার ক্ষতিপূরণ চোকাতে হয় আমাদের দৃষ্টি কে, এবং মানুষ অঙ্গ হয়ে ওঠে। কোনো রকম সংকেত সংক্রমণ জনিত সমস্যা গভীর আলোচনার বিষয়।

বিবর্তনের অন্যতম বিশ্বকর উদাহরণ মানুষের মস্তিষ্ক। বছরের পর বছর ধরে মস্তিষ্কের উন্নতি ঘটেছে, ঠিক যেন এক জীবন্ত টিস্যু ও ইলেক্ট্রোকেমিক্যাল কাজকর্মের দ্বারা তৈরি গগণচূর্ণী ভবন, যেন ঝুঁঝে গিয়েছে মানব সভ্যতার সেই দিক গুলি কে যেগুলি আমাদের আজও তাজব হয়ে পড়ি: মীতিশাস্ত্র, নৈতিকতা, গান-বাজনা, ভাষা, সৃজনশীলতা, সহানুভূতি এবং আরো অনেক বিচিত্র জিনিস। মস্তিষ্কের উন্নতির মেছাপ আমরা মস্তিষ্কের বিন্যাসের দ্বারা দেখতে পাই: মস্তিষ্কের বিভিন্ন অংশ বিভিন্ন রকমের কাজের জন্য দায়ি, ঠিক যেমন আমাদের নিঃশ্বাসের জন্য দায়ি 'লোয়ার এরিয়া' (Lower Area) যেগুলি মেরুদণ্ড এবং ব্রেনস্টেমের কাছাকাছি ছানে অবস্থিত। ঠিক তার উপরেই আছে আমাদের খাদ্য প্রহণ, আবেগ, লেখা-পড়া, একে একে রয়েছে হাইয়ার এরিয়া (Higher Area) তে, তাদের যোগ্যতা বা গুরুত্ব বুঝে।

চলুন আমরা আবার ইলাইন্সাইটে ফিরি বা সচেতনতা কেন একটি বিশেষ শব্দ সেই বিষয়ে আলোচনা করি। নিউরো সায়েন্স অনুযায়ী আমরা বিশ্বাস করতে ভালোবাসি আমরা যা ই করে থাকি সেটি করি অধিকায়, কিন্তু আমরা ও জানি যে নিঃশ্বাস নেওয়া একটি ব্রেস্ট্যাময় কাজ নয়, আমরা শ্বাস- প্রশ্বাসের কিছু পরিবর্তন আনতে পারি, কিন্তু তা আবার সাধারণ এ চলে আসে কিছুক্ষন পরেই, আমাদের কোনো

রকম মস্তিষ্কের কাজ করতে হয় না। এটি 'লোয়ার এরিয়ার' কর্ম হওয়ার কারণে এটা বিশ্বাস করতে আমাদের খুব একটা কঠিন মনে হয় না আমাদের কাছে। কিন্তু দেখা বা ভিজন'(Vision) এর মতন কাজ, যার সাথে সরাসরি সচেতনতার কোনো সম্পর্ক নেই, তার ও উল্লেখযোগ্য কোনো অংশ নেই, এটি বিশ্বাস করা একটু কঠিন হয়ে পড়ে।

কিছু উদাহরণ স্বরূপ, যার প্রথম টি আপনি এখন করছেন। আপনি যখন এটি পড়ছেন আয়নার দৃষ্টি বাদিকে থেকে ডানদিকে যাচ্ছে। এই সকল চলাফেরার একটি ধৰ্ছ আছে যা আপনি হয়তো কখনোই খেয়াল করেন নি, এই চলাফেরা কে বলা হয় স্যাক্রাডিক মুভমেন্ট(Saccadic movement), যার মধ্যে রয়েছে প্রচুর পরিমান ক্রমাগত প্রতিক্রিয়াকরণ ও প্রতিক্রিয়া যা ব্যাপক আকারে গবেষণার অঙ্গ। পড়া যেমন এক প্রকার প্রচুর সচেতন প্রতিক্রিয়া, আপনার মস্তিষ্কের অসচেতন ভাগ যে জিনিসগুলোর প্রয়জন নেই সেগুলো সামলায় যাতে আপনি সমন্বাবে পড়ে যেতে পারেন কোনো বাঁধা ছাড়াই। এই ধারনায় আমরা রাইন্ডসাইটের ক্ষেত্রে প্রয়োগ করবো। অনুমান করা হয় যে ভিজুয়াল প্রক্রিয়াকরণের দুটো রাস্তার মধ্য পুরোনো রাস্তাটি বা 'ওল্ড পাথওয়ে(Old Pathway)' একই রকম স্থির থাকে এবং অবজেক্ট ট্র্যাকিং(Object Tracking) ও স্পেসিটিয়াল ম্যাপিং(Spatial Mapping) এর কাজ করে, যা একটি আদিম প্রাণীরও প্রয়জন পড়ো-একটি ব্যাঙের কথা ভাবুন, কি করে সে একটি পোকার গুঞ্জন কে অনুসরণ করে তাকে জীব দিয়ে টেনে গিলে ফেলে। নতুন পথ বা নিউ পাথওয়ে, আরেকদিকে বিভাজন সৃষ্টি করে যেভাবে আমরা পরিষ্কৃ টাকে দেখি। ডি.এস. রামচন্দ্রণের কথা অনুযায়ী "যদি নিউ পাথওয়ে তে বাঁধা তৈরি হয়, তবে মানুষের চাক্ষুস সচেতনতা চলে যেতে পারে", আপনার আসে-পাশে ঘটে যাওয়া জিনিস আপনি সচেতন ভাবে না দেখতে পেলেও, আপনার আদিম দৃষ্টিভঙ্গি তে কোনো রকম ক্ষতি হয় না।

এটি বলার পরেও, আমরা উল্লেখ করা উচিত যে আমরা এখনো পর্যন্ত রাইন্ডসাইট সম্বন্ধে তেমন কিছুই জানি না এবং জানা সবে শুরু করেছি আমরা। তবে আমরা এ নিশ্চয় জানি যে নিউরোলজিক্যাল দিক যা শুধু আমাদের দৃষ্টি আকর্ষণ করে তাই নয় এবং তার সাথে সাথে মস্তিষ্ক সম্পর্কে আরো জ্ঞান জেগাড় করতে আমাদের চ্যালেঞ্জ ছাড়ে দেয়। এই ক্ষেত্রে উত্তরের চেয়েও প্রশ্নের মাহাত্ম্য বা গুরুত্ব অনেক বেশি। নিত্যদিনের বৈজ্ঞানিক অগ্রগতি ও অগ্রিম প্রয়োজন, আমাদের সমস্ত প্রশ্নের উত্তর দিয়ে চলেছে নিউরোলজি, সাইকিয়াট্রি এবং সাইকো এনালিসিসের দ্বারা।

নিত্যদিন বৈজ্ঞানিকরা এখনো পর্যন্ত প্রচুর চ্যালেঞ্জের সম্মুখীন হচ্ছে মস্তিষ্কের রাইন্ডসাইটের মূলের বিষয়ে। এই ধরনের পরীক্ষা-নিরীক্ষা বুঝিয়ে দিচ্ছে মস্তিষ্কের উপর আমাদের দখল কর্তৃ কম এত উন্নতমানের প্রযুক্তি ও মৃগ্যবৃুদ্ধি ধরে করে আশা গবেষণার পরেও। যদিও আমাদের অব্যবেশ প্রতিদিনই আমাদের ফল দিয়েছে, উত্তেজনাপূর্ণ আবিষ্কার ও হয়ে চলেছে প্রতিদিন, কিন্তু এই ধরনের গবেষণা আমাদের নিত্যদিন আরো নতুন বাধা অতিক্রম করতে আহ্বান জানিয়েছে। সক্রেটিসের একটি কথা আমি বিশ্বাস করতে ভালোবাসি 'যত বেশি জানি, তত জানতে পারি যে আমি কিছুই জানিনা'। এটি আমাদের থামতে নয়, নিত্যদিনে আরো এগিয়ে যেতে সম্মোধন করে।

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লাইফস্টাইল বা আচরণগত দিকগুলির প্রতিক্রিয়ায় মন্তিক্ষের কার্যকলাপে পরিবর্তন

Changes in Brain Activity in Response to Lifestyle/Behavioural Aspects

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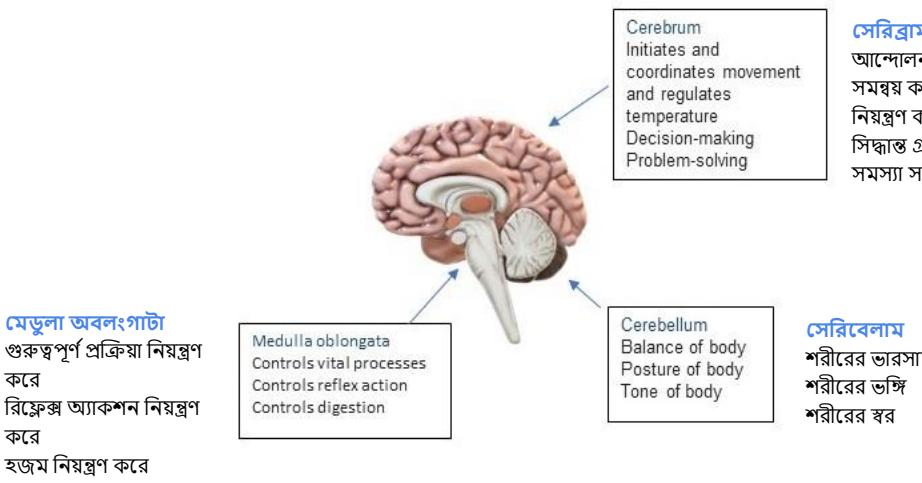
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Translated from English to Bengali by Ankush Chakraborty

সারসংক্ষেপ

আমাদের মন্তিক্ষ কে শরীরের প্রধান অঙ্গ বলে মনে করা হয়। শরীরের কার্যকলাপ সব কিছুই ঠিক করে আমাদের মন্তিক্ষ। একটি সুস্থময় জীবনযাপনের সাথে সুস্থময় সামাজিক জীবন, বিভিন্ন রকমের অভিজ্ঞতা, খাদ্যভ্যাস এবং পর্যাপ্ত পরিমাণ ঘুম আমাদের নিউরোপ্লাস্টিসিটির(Neuroplasticity) দিকে নিয়ে যায়। মন্তিক্ষের সেই ক্ষমতা যেই ক্ষমতা দিয়ে আমাদের মাথায় প্রতোক মুহূর্তে নতুন সিনাপটিক(Synaptic) যোগসূত্র তৈরি হয়, তাকে নিউরোপ্লাস্টিসিটি বলা হয়, মন্তিক্ষের নমনীয় হওয়ার জন্য আমরা একটি সুস্থ জীবন যাপন করতে সাহায্য করে এবং মন্তিক্ষের নিউরনদের(neuron) আরো দক্ষ করে তোলে। কিন্তু শুধুমাত্র একটি সুস্থ জীবনযাপন যথেষ্ট নয়, নতুন নতুন দক্ষতা গড়ে তুলতে হয় একটি মানুষ কে, সিনাপটিক যোগসূত্রের হার বাড়িয়ে তুলবার জন্য। আমাদের মন্তিক্ষ সারাজীবন ক্ষমতা রাখে নতুন জিনিস শিখবার, নতুন নিউডাল(Neural) রাস্তা তৈরি করবার, মানুষের বয়স এখানে হস্তক্ষেপ করতে পারে না।



চিত্র 1. মস্তিষ্কের গঠন

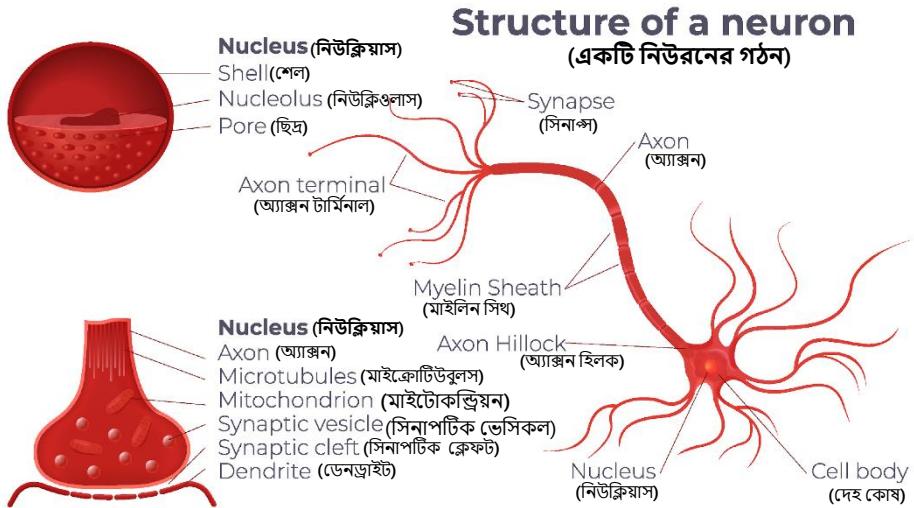
Image credit: Shivani Pimparkar

ভূমিকা

আমাদের শরীরের বিভিন্ন রকমের আশ্চর্যজনক কীর্তি করতে পারে- আনবিক স্তরে নিখুঁত সমন্বিত বিপাকীয় প্রতিক্রিয়া থেকে শুরু করে অসামান্য শরীরিক কার্যকলাপ যেমন খেলা ধূলা বা এডভেঞ্চার স্পোর্টস। কখনো ভেবে দেখেছেন এই সব কার্য নিয়ন্ত্রণ কে করে? উত্তর টা আপনার-আমার জন্ম থাকলেও, উত্তরটি মজাদার, আমাদের মস্তিষ্ক। 'ব্রেন' (Brain) শব্দটি ব্রিফেন (Bragen) থেকে, যার আক্ষরিক অর্থ জ্ঞান। মস্তিষ্ক আমাদের জীবনের সবরকম প্রক্রিয়া নিয়ন্ত্রণ করে, শুনি, খাদ্য, চোখে দেখা, ইত্যাদি। যদিও শরীরভিত্তিক সমস্ত কার্য মস্তিষ্ক একা সামলায় না, সেন্ট্রাল নার্ভাস সিস্টেম (Central nervous system) যা স্পাইনাল কর্ড (Spinal Cord) এর দ্বারা তৈরি, তাঁর ও অবদান রয়েছে এতে।

মস্তিষ্কের উপাদান: মাইলের পর মাইলের ঘাতা

- মস্তিষ্কের মৌলিক কার্যকরী ভাগ হলো নিউরন।
- নিউরনের দ্বারা আমাদের মস্তিষ্ক থেকে আবেগ সারা শরীরে ছড়িয়ে পড়ে স্পাইনাল কর্ডের সাহায্যে, ইলেক্ট্রিক্যাল ইম্পালস (Electrical Impulse) রূপে। একটি নিউরন ৩০০০ একটি নিউরনের সাথে যুক্ত হয়ে এই কার্য সফল করে, এবং এদের মিলনক্ষেত্রে কেবল হয় সাইন্যাপ্স (synapse).
- মস্তিষ্কের সাধারণ ওজন হয় ১.৩-১.৫ এর মধ্য, যার ৬০% ফ্যাট দিয়ে তৈরি, গবেষক রা জানিয়েছেন শরীরের এই ফ্যাট কে কমানো যায় না কারন এর মধ্য রয়েছে মাইয়েলিনের (Myelin) এর মতন কোষ যা মস্তিষ্কের সঠিক কার্যকলাপ নির্ধারণ করে।
- বৈঁচে থাকা ৪০% প্রোটিন (protein), কার্বোহাইড্রেট (Carbohydrate) ও নুন দিয়ে তৈরি।



চিত্র ২: নিউরনের গঠন: সামুদ্রিক মৌলিক কাঠামোগত এবং কার্যকরী একক

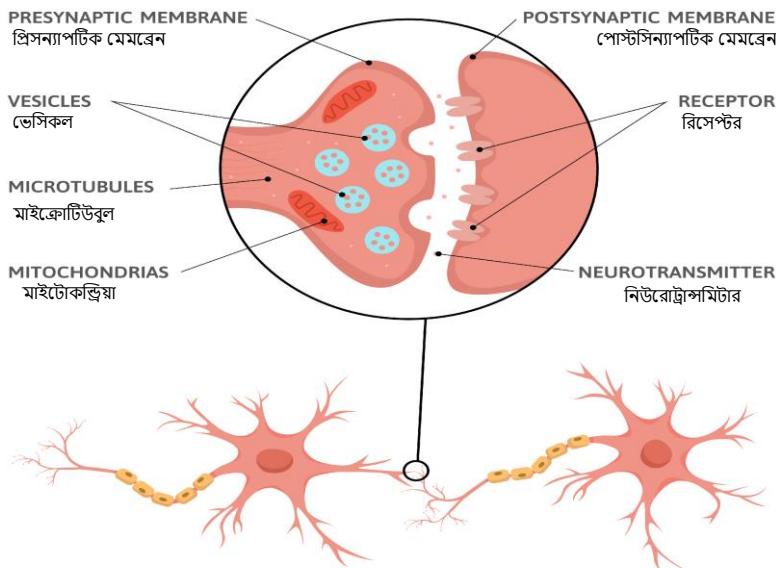
ইমেজ ক্রেডিট: ম্যাক্রোভেক্ট এবং <https://www.freepik.com>

নিউরোপ্লাস্টিসিটি (Neuroplasticity)

সাইন্যাপ্স(Synapse), নিউরন(neuron) এবং সমগ্র মস্তিষ্কের বদলে যাবার যে উদ্দেশ্য তাকে বলা হয় নিউরোপ্লাস্টিসিটি(Neuroplasticity)। সাইন্যাপ্টিক সংযোগের শক্তিশালীকরণ, দুর্বল হয়ে পড়া অথবা বাহির অভিজ্ঞতার কারনে হওয়া নতুন নার্ভ কোষের যুক্ত হওয়া কে নিউরোপ্লাস্টিসিটি বলা যেতে পারে। নিউরোপ্লাস্টিসিটি ঘটবার অনেক পদ্ধতির মধ্যে সব চেয়ে গুরুত্বপূর্ণ এবং একশোনাল স্প্রাউটিং(Axonal sprouting) এবং সাইন্যাপ্টিক প্রনির্ণয়(synaptic pruning)।

একশোনাল স্প্রাউটিং (Action potential)

এই ক্ষেত্রে সুস্থ একশনেরা নতুন নতুন নার্ভ পরিসমাপ্তি তৈরি করে যারা নতুন নতুন নার্ভাস রাস্তার সাথে মেলবন্ধন যোগায়, যেসব যোগসূত্রের অস্তিত্ব রয়েছে তাকে মজবুত করে এবং নার্ভাস সিস্টেমের আগাম প্রাপ্ত জায়গাগুলোই আবার জোড় বাঢ়ায়।



চিত্ৰ 4: সিন্যাপটিক জংশনের গঠন
ইমেজ ক্রেডিট: ম্যাক্ৰোভেক্ট এবং <https://www.freepik.com>

সিন্যাপটিক প্ৰণীৎ (Synaptic Pruning)

একটি সদ্যজাত শিশুর মস্তিষ্ক শিশুদের ইন্দৃয় অঙ্গের দ্বারা পাঠানো তথ্যে ভর্তি থাকে। এই তথ্য মস্তিষ্কের সেই জায়গা পৌঁছানো দৰকার যেখানে এর প্ৰক্ৰিয়া শুরু হবে। আৱ এই প্ৰক্ৰিয়াৰ জন্যে প্ৰয়োজন এক নার্ভ কোষেৰ সাথে অন্য নার্ভ কোষেৰ সংযোগ অত্যন্ত প্ৰয়োজন, যাৱ ফলে মস্তিষ্কে আবেগেৰ প্ৰেৱণ হয়।

তাৰপৱেৰ কিছু বছৰ ধৰে, মস্তিষ্ক দ্রুত গতিতে বেড়ে ওঠে। প্ৰত্যেক নিউৱনেৰ পৰিপ্ৰেক্ষতাৰ ফলে তাৱা নিজেদেৰ শাখা বেৱ কৰে, একশন(Axon) যা তথ্য মস্তিষ্কেৰ বাইৱে নিয়ে যায় এবং ডেন্ড্ৰাইট(Dendrite) যা মস্তিষ্কেৰ ভেতৱে তথ্য নিয়ে আসে এবং এৱ ফলে নিউৱনদেৰ মধ্যে ভালো সংযোগ তৈৱি হয়।

জন্মের সময় আমাদের মস্তিষ্কে সেরিব্রাল কর্টেক্সে(Cerebral Cortex) এ ২৫০০ সাইন্যপ্স হয়, ২-৩ বছর বয়সে সেটা গিয়ে দাঁড়ায় ১৫০০০ এ, যা প্রায় জন্মের সময়ের থেকে ৬ গুণ বেশি। বয়স বেড়ে যাওয়ায় আমাদের মস্তিষ্ক পূরণ সংযোগ সরাতে থাকে বা তুলতে থাকে এবং একই বলা হয় সিন্যাপ্টিক প্রস্তুৎ।

এই দুই পদ্ধতির কারণেই আমাদের মস্তিষ্কে হওয়া আঘাত সব সময় চিরস্থায়ী হয় না। এই পদ্ধতির দ্বারা মানুষের অনেকের মধ্যে পরিস্থিতির যোকাবিলা করতে পারে

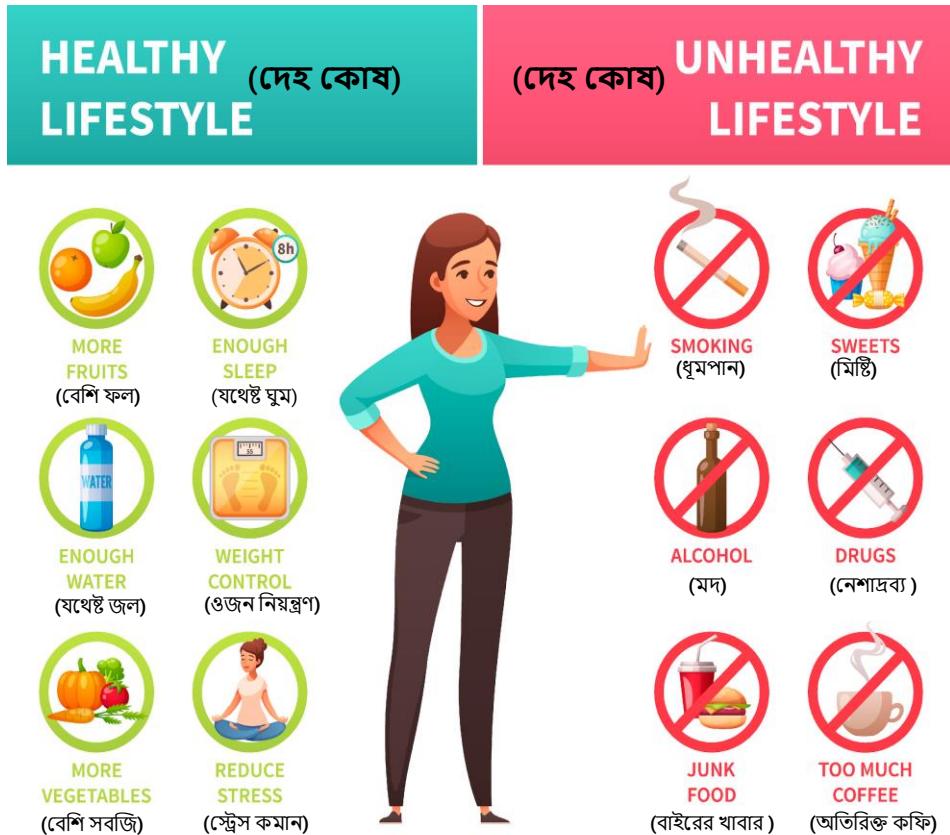
মস্তিষ্কের কার্যকলাপের কারণ

সামাজিকভাবে সক্রিয়তা পরের ভীবনে অনেক মানসিক ও শারীরিক সুবিধা গড়ে দেয়। সর্বশেষ তথ্য অনুযায়ী সামাজিক সক্রিয়তা একজন মানুষকে জ্ঞানীয় পতন থেকে রক্ষা করতে পারে। দুটা অধ্যয়ন অনুযায়ী আবেগ নিয়ন্ত্রণ করবার ক্ষমতার বৃদ্ধি হয় সামাজিক সক্রিয়তায়, যার সাহায্যে আমরা মেয়ার সালোভে-কারুসে ইমোশনাল ইন্টেলিজেন্স(Mayer saloverry-caruso emotional intelligence test)টেস্ট করতে সুবিধা হয়।

খাদ্যভ্যাস

বহুদিন ধরেই তানুমান করা হয়ে এসেছে তুলনামূলক ভাবে সবরকম পৃষ্ঠি উপাদানের প্রভাব রয়েছে আমাদের মানসিক ও আবেগপূর্ণ কাজে। কয়েকটি মূল পদ্ধতি নথিভুক্ত করেছে, খাদ্যভ্যাস আমাদের সিন্যাপ্টিক প্লাস্টিসিটি ও নিউরোনাল ফাংশন কে সরাসরি ভাবে প্রভাব ফেলে। বেশ কিছু সাহসী হরমোন যা আমাদের মস্তিষ্কে যায়, বা আমাদের মস্তিষ্ক তৈরি করে তার সরাসরি প্রভাব পড়ে আমাদের জ্ঞানভিত্তিক কাজে। অধিকন্তু, সুপরিচিত সিন্যাপ্টিক প্লাস্টিসিটি নিয়ন্ত্রকেরা মেটাবলিক মডিউলেটরের(Metabolic modulator) কাজ করে থাকে যখন আমরা আমাদের শরীরে খাদ্যের প্রবেশ ঘটায়।

আমাদের মস্তিষ্কের যথেষ্ট পরিমাণ তেল দরকার পড়ে সঠিক ভাবে চলবার জন্য। উচ্চ গুণমাপ্ত তেল মস্তিষ্ককে নিজের সেরাটা দিতে সাহায্য করে। মিনারেল, ভিটামিন o এন্টিঅক্সিড্যান্ট(Anti oxidants) মস্তিষ্কের পুষ্টিসাধন করে এবং অক্সিডেটিভ স্ট্রেসের(oxidative stress) হাত থেকে আমাদের সুরক্ষা করে, যেটি কিনা ফ্রি রেডিক্যাল এবং অ্যাস্টিঅক্সিডেন্ট দের মধ্য ভারসাম্যহীনতা বজায় রাখে এবং সেল ড্যামেজ(cell damage) থেকে আমাদের রক্ষা করে।



চিত্র 4. একটি স্বাস্থ্যকর জীবনধারা মানিয়ে নেওয়া

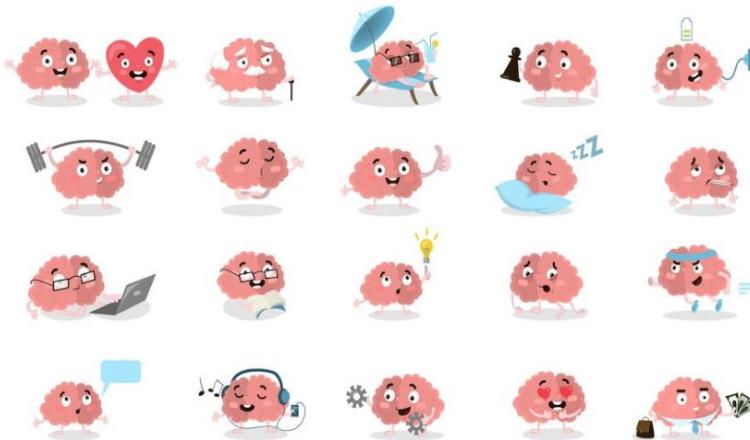
ইমেজ ক্রেডিট: Macvector এবং <https://www.freepik.com>

. অন্য দিকে নিম্ন মানের জীবনযাপন মন্তিক্ষের সুস্থ ভাবে কাজ করে বিঘ্ন ঘটাতে পারে। প্রক্রিয়ার দ্বারা তৈরি খাবার এই বিভাগের অন্তর্ভুক্ত। এই ধরনের খাবার দ্বারা ইনসুলিন তৈরিতে বিঘ্ন ঘটাতে পারে এবং স্ট্রেস বাড়িয়ে তুলতে পারে, অনেক গবেষনায় দেখা গেছে এই ধরণের খাবার দ্বারা ডিপ্রেশনের কারণ হয়ে দাঁড়িয়েছে।

এই ধরনের পড়াশোনা কে বলা হয় নিউট্রিশনাল সাইকিয়াট্রি(Nutritional Psychiatry)। এটি একটি বেড়ে ওঠা ক্ষেত্র যা বহু বছর ধরে ঘাটা হয় নি, তবে কিংবরে গবেষকরা বুঝতে পারে আমাদের মানসিক স্বাস্থ্য খুব সহজেই, তারা আমাদের খাদ্যাভ্যাসের উপর নজর রাখে।

ব্যায়াম

কখনো ভেবে দেখেছেন ব্যায়াম কেন প্রয়োজনীয়? বিটিশ কলাসিয়ার ইউনিভার্সিটির কিছু গবেষকদের মতে বায়ুবীয় ব্যায়াম আমাদের হিপ্লোক্যোপ্সাসের আকার বাড়িয়ে তোলে, যা আমাদের মৌখিক স্থূতি বা শিক্ষালাভের হার বাড়িয়ে তোলে যা অন্য কোনো ব্যায়াম এ অত বেশি দেখা যায় নি।



চিত্র ৫: মস্তিষ্ক কীভাবে নতুন দক্ষতা অর্জন করে?

ইমেজ ক্রেডিট: vector-istock এবং <https://www.freepik.com>

নতুন কলা

যখন আপনি কোনো নতুন কলা শেখেন আপনার মস্তিষ্কে পরিবর্তন ঘটে। সহজ ভাষায়, আপনার মস্তিষ্ক নতুন সংযোগ তৈরি করে। চ্যালেনজিং পরিবেশে আমাদের শরীর ও মস্তিষ্কের পরিবর্তন ঘটে, মাংসপেশিরা শক্ত হয়ে ওঠে, হাস্পিস্ট ও ফুস-ফুসের আকার বেড়ে ওঠে এবং মস্তিষ্কের সংযোগ হয় আর তাড়াতাড়ি। নতুন কলা অর্জন এবং উন্নয়নের ভিত্তিতে মস্তিষ্কের পরিবর্তন ঘটে। শরীর নিজেই জানে না এই ধরণের পরিবর্তনের কারণ কি, কিন্তু এই ভাবেই কার্যক্রম করা রয়েছে শরীরে, যাতে সব কিছু সহজ হয়ে পড়ে। আমাদের মস্তিষ্ক ও শরীর সহজ জিনিস পছন্দ করে, এই ভাবেই মানিয়ে নেতা পাছন্দ করে যাতে পরের কাজে কোনো বাঁধা না ঘটে। শরীর কে ব্যতিব্যস্ত করে যখন আপনি কোনো নতুন কলা শিখছেন তার মানে সেটি গুরুত্বপূর্ণ, এবং আপনি আরো সময় উৎসর্গ করেছেন সেটি শিখে উঠবার জন্য। এই ধরণের কার্য প্রচুর পরিমান নিউডাল সংযোগ ঘটায়, এবং শক্তিশালী করে তুলছে এবং কর্মক্ষমতা বাড়িয়ে তুলছে।

ডিজিটাল প্রযুক্তির প্রভাব

ডিজিটাল যুগে মোবাইল ফোনে ব্যবহার প্রচল্ন মাত্রায় বেড়ে গিয়েছে এবং জীবনযাপন এর সাথে রক্তে রক্তে মিশে গিয়েছে। অনেক অর্থে আমাদের জীবন কে অনেক সহজ করে তুলেছে মোবাইল, এবং

আমাদের মাতিক্ষের উপরেও প্রভাব ফেলেছে। অনেক তথ্যে দেখা গিয়েছে যে কিশোবদের মধ্যে কম্পিউটার ব্যবহারের কারণে এটেনসন ডেফিসিট হাইপারেক্টিভিটি ডিসঅর্ডার(attention deficit)

ঘুম

ঘুম আমাদের মাতিক্ষের কার্ডের উপর সরাসরি প্রভাব ফেলে, ঘুম চলাকালীন আমাদের মাতিক্ষে সারাদিনব্যাপী জোগাড় করে তথ্য নিউরনের দ্বারা গেটা মস্তিষ্ক জুড়ে ঘুরে বেড়ায়, ইলেক্ট্রিক্যাল ইমপালস রাপে একশোন ও ডেন্ডরাইট এর দ্বারা তথ্য যোরাফেরা করে। সম্পূর্ণ ঘুম না হওয়ার কারণে আমাদের মস্তিষ্ক ভুলতে শুরু করে এবং সিদ্ধান্ত নিতে বিলম্ব করে।

মাতিক্ষের উপর ঘুম ও শারীরিক ব্যায়াম এর প্রভাব

•নিয়দিনের শারীরিক ব্যায়াম আমাদের মধ্যে অ্যালজাইমারস হওয়ার সম্ভাবনা কমিয়ে দেয়। শারীরিক ব্যায়ামের কারণে আমাদের রক্ত চলাচল ও বেড়ে চলে এবং আমাদের মনে রাখবার ক্ষমতা ও বেড়ে যায়। শারীরিক ব্যায়ামের কারণে আমাদের মাতিক্ষে রাসায়নিক অনেক পরিবর্তন ঘটে যা আমাদের শিক্ষা লাভ আপনার চিন্তার মধ্যে বৃদ্ধি নিয়ে আসে।

•বয়স বেড়ে যাওয়ার কারণে আমাদের মাতিক্ষেকে অন্যরকম চাপের মধ্যে দিয়ে যেতে হয় জীবন যাপন এবং পরিবেশগত কারণে। ফলে অক্সিডেশন (oxidation) দেখা যায়, যা আমাদের মাতিক্ষের কোষদের আঘাত করে। লোহার গেটে হওয়া জং বা অর্ধেক খাওয়া আপেলের দ্বারা আপনাদেরকে আমরা আরও সুন্দরভাবে এর রাসায়নিক পরিবর্তন এর ফল বোঝাতে পারবো, যা হয় অক্সিডেটিভ কারণে।

অ্যান্টিঅক্সিডেন্ট এ ভার্টি খাবার-দাবারের দ্বারা আমরা অক্সিডেশন থেকে সুরক্ষিত থাকতে পারি।

•ঘুম আমাদের মধ্যে শক্তি নিয়ে আসে আমাদের মেজাজ শুধরে দেয় এবং আমাদের ইমিউন সিস্টেমকে আরো সুন্দর করে তোলে। সঠিক পরিমাণ ঘুমের দ্বারা আমাদের মাতিক্ষে একটি এবনরমাল প্রোটিন তৈরির থেকে বাধা সৃষ্টি হয়, যার নাম বেটা এমিলেডাল প্লেক(beta amyloid plaque), যার সরাসরি যোগ রয়েছে অ্যালজাইমারের মতন রোগের সাথে।

•মাতিক্ষের ব্যায়াম আমাদের মাতিক্ষের কার্য আরো শুধরে তোলে এবং নতুন নতুন মাতিক্ষের কোষের জন্ম দেয় এবং ডিমেনশিয়া(Dementia)হওয়ার সুযোগ কমিয়ে দেয়। আমাদের উচিত মাতিক্ষের কোষদের প্রত্যেকদিন ব্যবহার করার এরকম আমরা প্রত্যেকদিন আমাদের মাস্পেশির ব্যবহার করি।

•সামাজিক জীবনযাপন আপনাকে রক্ষা করতে পারে আপনার স্মৃতির রক্ষার জন্য। বন্ধুবন্ধবদের সঙ্গে সময় কাটানো তর্কিতেকে জড়িয়ে পড়া অথবা পরিবারের লোকেদের সাথে কথাবার্তা বলা বা যোগাযোগ রাখা আপনার মাতিক্ষকে সুস্থ রাখে। অনেক গবেষণায় দেখা গেছে সামাজিক সামাজিক মিথস্ক্রিয়া স্মৃতি রক্ষা করেছে খুব সুন্দর ভাবে।

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শিখতে শেখা

Learning How to Learn

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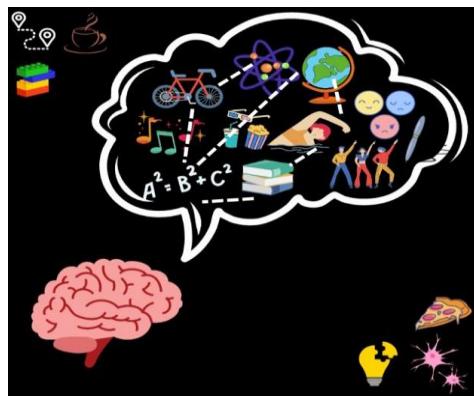
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সারসংক্ষেপ

কখনো ভেবে দেখেছেন মানুষ কি সুন্দরভাবে গানের কলি, ফোনের নাস্থার, 'গান গাওয়ার স্টাইল' নাচের স্টেপ, অথবা ক্রেবের সাইকেল মনে রাখতে পারে(উফফ) ? ধন্যবাদ টা আমাদের জ্ঞানে উচিত আমাদের মস্তিষ্ক কে এবং তাঁর মনে রাখবার ও ফের তার জোড়া লাগাবার ক্ষমতা কে। শিখবার ক্ষমতা যে কোনো পাথরে লিখে রাখত্ব হয় নি তা আমাদের নিউরোনাল ওয়ারিং(Neuronal Wiring) এর বিস্তারিত গবেষণা এবং পড়াশুনা আমাদের সামনে তুলে ধরেছে, এবং বিভিন্ন ক্ষেত্রে শিখবার ধরন যে এক সমৃদ্ধ সমান পার্থক্য গড়ে তুলতে পারে তাঁর কথাও জেনেছি আমরা। আমাদের এই লেখার দ্বারা আমরা নিউরো সাইকোলজিস্ট দের সাধারণ মানুষের শিক্ষা আর্জন ক্ষমতার ব্যাপারে আলোচনা করেছি।

পরিচায়ক



চিত্র 1: মাল্টিমডিল মস্তিষ্ক। এই ছবিটি দেখাচ্ছে যে কীভাবে মস্তিষ্ককে শেখার জন্য প্রশিক্ষিত করা যায় এবং একাধিক শেখার শৈলী ব্যবহার করে স্মৃতি এবং অভ্যাস গঠন করা যায়।

অসাধারন মস্তিষ্ক ও তাঁর খেঁজ।

স্কুল বা কলেজে সর্বদা প্রথম বেঞ্চের ছাত্রদের বা ফার্স্ট বেঞ্চার দের কে মনে করা হয় বুদ্ধিমান ও মনোযোগী এবং শেষ বেঞ্চের ছাত্র দের বা 'ব্যাক বেঞ্চার' দের ঠিক তার উল্লেখ। এর কারণ কি বিভিন্ন রকমের শিক্ষার পদ্ধতি? অথবা বিভিন্ন ছাত্রদের বিভিন্ন উপায়ের মাধ্যমে একই জিনিস শেখার প্রয়াস?

১৯৯২ সালে, ফ্রেমিং ও মিলস ভার্ক(VARK) মডেলের তথ্য সামনে নিয়ে আসে এবং তার দ্বারা আমরা বিভিন্ন রকম শিক্ষার প্রযুক্তির সমন্বয় ওয়াকিবহাল হই। তাদের মতে বিভিন্ন শিক্ষার্থীদের শিক্ষা গ্রহণ করবার ক্ষমতা অন্যরকম। কেউ কেউ পছন্দ করে সিনেমা চলচ্চিত্র গ্রাফিক্স বা ছবির দ্বারা শিক্ষা নিতে তাদের আমরা ভিজুয়াল লার্নার(Visual Learner) বলে থাকি অথবা কেউ পছন্দ করে লেকচার বা তর্কিবিতর্ক যাদের আমরা আওরাল লার্নার(Aural Learner) বলে চিনি কিছু কিছু শিক্ষার্থী পুনরায় পড়াশোনা এবং প্রচুর পরিমাণ নেটস এর দ্বারা শিক্ষা লাভ করতে ভালোবাসে তাদের আমরা বলে থাকি রিডিং-রাইটিং লার্নার(Reading-Writing learner)। একই সময়ে যে লাস্ট চ্রস্পটির কথা মিলস ও ফ্রেমিংস বলছেন তারা হলেন কাইনেস্টিক লার্নার্স(Kinesthetic learner), যারা পছন্দ করেন নিজ হাতে পর্যাক্ষ নিরীক্ষা করা এবং মিথস্ক্রিয় অধিবেশনসম্মত। এই মডেলকে চারিদিকব্যাপী ব্যবহার করা হয় শিক্ষার্থীরা কোন শিক্ষা পদ্ধতিকে পছন্দ করতে এটার বিষয়ে ওয়াকিবহাল হওয়ার জন্য।

যদিও বা সবকিছু এটো সহজ নয়, চার ধরনের ভার্কের যে পদ্ধতি তা দিয়ে শিক্ষার্থীরা মাত্র এক সময়ে মস্তিষ্কের এক অংশই প্রায়শই ব্যবহার করে উঠতে পারে, অনুমান করা হয় ভার্ক(VARK) অনেকে বেশি পরিমাণ প্রতিবন্ধকতা সৃষ্টি করে একটি শিক্ষার্থীর শিক্ষা লাভের ক্ষেত্রে। দৃষ্টিস্পষ্টভাবে ধরে নেওয়া যাক ভিজুয়াল স্টাইলে শিক্ষার্থীদের চোখ ও মস্তিষ্কের আকিপিটোল লোর ব্যবহার করতে হয় গ্রাফ ও ছবির দ্বারা শিক্ষালাভ করতে। এতে মস্তিষ্কের আরেক অংশে নিউরোনাল ফায়ারিং হয় এবং তা মস্তিষ্ককে প্রভাব ফেলে। আরো প্রশ্ন ওঠে যে ভার্ক কতটা নির্ভরযোগ্য? এর মানে কি বোঝায় যে একটি মানুষ একই ধরনের শিক্ষালভের পদ্ধতি নিয়ে জন্মায়? বৈজ্ঞানিকদের মতে ৩৯ শতাংশ মানুষ যারা ভার্ক মডেল ব্যবহার করেন তারা একই বিভাগের মধ্যে পড়ে না। পড়াশোনা বলছে, শিক্ষার্থীরা ২ কি ৩ রকমের শিক্ষার পদ্ধতি ব্যবহার করতে পছন্দ করে(ছবি ১)। ইদানিং কালে বহু গবেষকের মতে পছন্দসহিত শিক্ষা লাভের ব্যবস্থার কোনোরূপ অস্তিত্বই নেই। ডক্টর টেশিয়া মার্শিক, বিশিষ্ট মনোবিজ্ঞানী, ইউনিভার্সিটি অফ উইস্কনসিন- লা ক্রসে, বলছেন 'তথ্য ধরে রাখার জন্য আমাদের তথ্যকে অর্থপূর্ণভাবে সংগঠিত করতে হবে, ইন্সিৎ থেকে স্থায়ী।' এরপর থেকে যখনি আপনি প্রস্তুত হয়ে পড়বেন পুনরায় পড়তে বা লিখতে মুখ্য করবার জন্য, অপেক্ষা করুন এবং প্রতিফলিত করুন আপনার নিজস্ব উদাহরণ দিয়ে।

বিশ্বাম, সাবধান!

শিক্ষালাভকালীন, মনযোগ দেওয়ার গুলো প্রধানতম। কোনো এক জিনিসের উপর কেন্দ্রবিন্দু করে বিশ্লেষণ করার সময় সমগ্র পৃথিবীকে উপেক্ষা করা কে বলা হয় মনোযোগ। চলুন আরো গভীরে যাওয়া যাক এই বিষয়ে। কল্পনা করা যাক, একটি হাতি একটি ঘরের মধ্যে জোর কদমে হেঁটে বেড়াচে যেখানে আপনি বসে আছেন। কি করে আরো ভালো মনোযোগ দেবেন তার উপর: তিনটি ছেট্ট পদক্ষেপেই করা যাবে: এক্টিভেশন(ACTIVATION)- যখন আপনি সচেতন যে আপনার রুমে একটি হাতি আছে, ভিজুয়াল-স্পেসিটিআল রিকগনিশন(VISUAL SPATIAL RECOGNITION)- যখন আপনি হাতিটার চেহারা এবং গায়ের রঙ সম্বন্ধে সচেতন, এবং পেরিং ইড টু সিলেক্টিভ এক্সেকিউটিভ কম্পোনেন্ট(PAYING HEED TO SELECTIVE EXECUTIVE COMPONENTS)- যখন আপনি হাতিটার দ্বারা তৈরি করা সমস্ত আওয়াজ সম্পর্কে সচেতন।

তাহলে কি অসামান্য শিক্ষালাভ এবং তীক্ষ্ণ মনোযোগ রাখতে কি অসাধারণ মনে রাখবার ক্ষমতা লাগে? উন্নত আছে এই পদ্ধতিতে।

লাইটস, ক্যামেরা, একশন!

মাস্টিফের মধ্যে কি চলে যখন আপনি শিক্ষালাভের চেষ্টা করেন? ডক্টর ডোনাল্ড ও. হেব, একজন কানাডিয়ান মনোবিজ্ঞানী প্রস্তাব রাখেন যে আমরা যথনি নতুন কোনো তথ্য সংগ্রহ করি, মাস্টিফের নিউরনেরা সক্রিয় হয়ে পড়ে। তারা বিভিন্ন নিউরনের সাথে যুক্ত হয়ে একটি অন্তর্জাল তৈরি করে। এই নিউরন গুলি প্রথমদিকে দুর্বল হলেও, নিয়মিত উদ্বীপনার কারণে তারা একে অপরের সাথে যুক্ত হয়ে শক্তিশালী হতে থাকে(একেই বলা হয় পোটেনসিসেনেন)। ঠিক এই কারণেই নিয়মিত পড়াশোনা আপনাকে বেশি পরিমাণ তথ্য মনে রাখতে সাহায্য করে। যেহেতু একই পদ্ধতি সমানভালে চলেছে, এটি জোট বৈধে একসাথে হয়ে যায়। এই থিওরি কে আমরা প্রায়শই অনুমান করে থাকি নিউরনস দ্যাট ফায়ার টুগেদার, ওয়ার টুগেদার।"

বৈজ্ঞানিকরা অনেক মজাদার তথ্য সংগ্রহ করেছিল যখন তাঁরা মানুষের জীবনের নিউরোনাল নেটওয়ার্ক নিয়ে গবেষণা করেন। জেমের সময়, কল্পনা করা হয় একটি মনুষ্য মাস্টিফে ২৫০০ নিউরন সাইন্যাপস(দুটি নিউরনের মিলনক্ষেত্র), এবং এই সংখ্যা দিনে দিনে বৃদ্ধি পেতে থাকে, ১৫০০০ সাইন্যাপস দিখা যায় একটি বেড়ে ৭৩ বাচ্চার মাস্টিফে। প্রাপ্তবয়স্কদের মধ্যে দেখা গিয়েছে সংখ্যা কমে অর্ধেক হয়ে যেতে। এর আগেও বেই কথার উল্লেখ আমরা পেয়েছি, কিছু কিছু অন্তর্জালে জোট বৈধে যায়, এবং কিছু জায়গা হয়ে যায় আলগা যেহেতু মাস্টিফের সেই অংশ কে আমরা কাজে লাগাই না।

নিউড়াল সার্কিট তৈরির ১০১

দাঁড়ান দাঁড়ান, জানেন প্লাস্টিসিটি(Plasticity) মানে কি? প্লাস্টিসিটি আমাদের মাস্টিফের সেই ক্ষমতা যা আমাদেরকে নিয়মিত নতুন তথ্য মনে রাখতে সাহায্য করে। মানুষের মাস্টিফে প্রচল পরিমাণ নমনীয়। যদি আমাদের মাস্টিফে নিয়মিত নতুন নতুন সংযোগ তৈরি করে বা ভেঙে ফেলে তাকে বলা হয় শর্টটার্ম প্লাস্টিসিটি। যখন আপনি আপনার পছন্দসই খাবার আপনার মোবাইল থেকে অর্ডার দেন, আপনার মোবাইল ও.টি.পি. আসে টেক্সট ম্যাসেজ মাধ্যমে। আপনি তখন সেই ও.টি.পি. দিয়ে আপনার পছন্দমতন পিজ্জা অর্ডার করে পিজ্জার কথা ভাবতে থাকেন। একটি ও.টি.পি. মুখস্ত করা শর্ট টার্ম প্লাস্টিসিটির প্রধান উদাহরণ, যেটি ক্ষনিকের জন্য আমাদের দরকার পড়ে এবং কিছু মুহূর্ত পরেই ঘার মেয়াদ শেষ হয়ে যায়। কিন্তু, আমাদের এমন অনেক জিনিস মনে রাখতে হয় যা আমাদের অনেকদিন ধরে প্রয়জন পরে এবং একেই বলে লং টার্ম প্লাস্টিসিটি, এবং ঘার অস্তিত্ব থাকে এক মুহূর্ত থেকে এক ঘন্টা কি বহুদিন বা বছর এবং প্রভাবশালী মডেল হিসেবে এই প্লাস্টিসিটি কেই বেছে নেওয়া হয়।

এই প্লাস্টিসিটি মাপবার কি কোনোরক পদ্ধতি আছে? নিচ্ছয় আছে! ইলেক্ট্রো

এনসেফেলোগ্রাফি(Electroencephalography) বা ই.ই.জি. কে সাধারণত গোটা বিশ্ব ব্যবহার করে থাকে মানুষের মাস্টিফের বৈদ্যুতিক কার্যকলাপ সংগ্রহ করবার জন্য। ই.ই.জি. মাস্টিফের বৈদ্যুতিক কার্যকলাপ সংগ্রহ করে মাথার তালু তে ঠেকিয়ে রাখা বিভিন্ন ইলেক্ট্রোডের সাহায্য। আরো আক্ষেত্রে মনাতক পদ্ধতি হলো ম্যাগনেটিক রেশনেন্স ইমেজিং(Magnetic Resonance Imaging) বা এম.আর.আই।

একসময়ে প্রায় সমস্ত ম্যানুবিজ্ঞানীরাই মনে করতেন আমাদের মাস্টিফে একটি টাপারওয়ের বক্সের মতন, তাদের শিক্ষালাভের ক্ষমতা সীমিত, কিন্তু আজ আমরা জানি মাস্টিফে প্রসারণযোগ্য এবং যে কোনো কাপ ধারণ করতে পারে একটি জিপিলকের মতন।

মাস্টিফে কখন দোকান খোলে?

মস্তিষ্কের ভিত্তিগত যোগাযোগ নির্দিষ্টায় থাকে খুদে শিশু দের মধ্যে। শিশুদের মস্তিষ্কের অগ্রগতি হয় অত্যন্ত গতিশীল ভাবে যাকে বলা হয় ক্রিটিক্যাল পিরিয়ড(Critical Period)। এই সময়কালীন সাইন্যাপসিস নিউরনদের মাঝে থাকে অত্যন্ত বেশি, যার ফলে তারা উদ্দীপনার পিছে অনেক বেশি গ্রহণযোগ্য হয়ে ওঠে। কল্পনা করা যাক একটি শিশু কে যদি এই সময়কালে কথা-বার্তা বলা সেখানে হয় স্থানীয় ভাষায়, তাকে শেখানো স্থানীয় ভাষায় কিন্তু সে রপ্ত করবে তাতে যদি সে একটি ভাষাগত ভাবে ধনী জাগরাগ বাসিন্দাও হয় তাও তার প্রভাব তার মধ্যে পড়বে না, এবং দেখা গেছে যে প্রবর্তি কালে স্থানীয় ভাষার উচ্চারণ ছেড়ে বেরোতে সমস্যায় পড়তে হয়েছে।

একটি ভবনের কথায় ভবন, যেখানে প্রত্যেকটি সাইন্যাঙ্ক কে তুলনা করা যায় প্রত্যেকটি ইঁটের সাথে, যা আমাদের সামনে মস্তিষ্কের কাঠামোর মৌলিকক্ষ তৈরি করে। ভীত ও নিচ তলার কাজ শেষ হলেই আপনার ভবনটি দৃঢ়লা বাড়ি বা বুর্জ খলিফা বানানোর জন্য প্রস্তুত হয়ে পড়ে।

আরো পরিমাণ বিশ্লেষণে দেখা গেছে যে কাজ গুলি করবার জন্য মস্তিষ্কের সাহায্য নিতে হয় সেই কাজে বেড়ে যাওয়া বয়স আমাদের কাজে বাধা সৃষ্টি করে, কিন্তু শিক্ষালাভের সাথে তার সম্পর্ক খুব একটা নেই, বাড়তি বয়স আমাদের শিক্ষালাভ কে খুব একটা ক্ষতি করে না। বেড়ে ওঠা বয়সে শিক্ষালাভ মূলত আচ্ছসম্মানমূলক এবং সাধারণীকরণ তত্ত্ব প্রাপ্তবয়সের শিক্ষার সাথে হস্তক্ষেপ করে না। মস্তিষ্কের প্রিফ্রন্টাল ও টেম্পোরাল জায়গা গুলি পৃথকীকরণ করে রাখে নতুন ও পুরোনো দিকগুলো কে কোনো এক নির্দিষ্ট কলার, এবং এও দেখে যে মগজে থাকা নতুন ও পুরোনো স্মৃতির যাতে বিভেদ না সৃষ্টি হয়। ক্ষেপণ শিখাবার জন্য বাংলা ভুলে যাবেন? কখনোই নয়! আমাদের মস্তিষ্কের বুদ্ধিমত্তার প্রশংসা আমরা কখনোই করি না যেটার সে অধিকারী।

চলুন আবারো প্রত্যেকটি দিক কে জোড়া লাগাই, শিখাবার পদ্ধতি, মনোযোগ, নিউড়াল সার্কিট, এবং শিখাবার গতিপথ। আশা করা যায় আমরা আপনাকে মস্তিষ্কের তেজ সম্পর্কে আলোকপাত করতে পেরেছি যাতে করে আপনারা পাস বা ফেল দিয়ে এই বিষয়টি শেষ করবেন না। কারণ আপনি যদি এখন কোনো কাজ করছেন এবং সে কাজ করতে আপনার মস্তিষ্কের সাহায্য নিতে হচ্ছে তার মানে আপনার মস্তিষ্কের ভেতরের নিউরনের জেট বাঁচে এবং আপনার জ্ঞান প্রত্যেকটি বিন্দুর মিলন সৃষ্টি করছে পরিচ্ছিতি কে আরো ভালো করবার জন্য।

ইদানিং কালের গবেষণা আরো অনেক ধাপ এগিয়ে। এখন শিখাবার বিভিন্ন দিক নিয়ে গবেষণা করছে। যার মধ্যে রয়েছে প্লায়াল কোষের নিউরোনাল ও নন-নিউরোনাল সংখ্যা, হোয়াইট ম্যাটারের পুনর্গঠন, টলমল আবেগ সংক্রমনের কারণ, এবং কি করে আপনার মস্তিষ্ক আঘাত, স্ট্রেক বা উল্লেখযোগ্য মানসিক ব্যাধি থেকে পুনরায় রিওয়ার করা শুরু করে।

শিখায় চাহিদা কারন মস্তিষ্ক সর্বদা বহু সংবেদনশীল উদ্দীপনা এবং প্রবর্তী ঘটে যাওয়া সংযোগের রিওয়ারিং, যা আপনার ব্যবহারে ফুটে ওঠে। সাধারণ উম্ময়নমূলক টেম্পেট বাদে, আপনার শিক্ষালাভের ক্ষমতা কোনো পাথরে নেখা নেই। যদি আপনার দৃঢ়তা থাকে, শিখাবার ইচ্ছা থাকে এবং আপনাকে প্রয়জন মতন মনোযোগ দিতে পারেন, আপনার জ্ঞ নিষ্ঠিত। এ.পি.য়ে. আব্দুল কালাম বলে গেছেন "দা বেস্ট ব্রেনস অফ দা নেশন মে বি ফার্টেড অন দা লাস্ট বেঞ্চেস অফ দা কাসরম!"

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ক্যালিডোস্কোপের ভেতর দিয়ে: বহুলযুক্ত সচেতন মন Through the Kaleidoscope: The Multi-layered Conscious Mind

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সারসংক্ষেপ

চেতনা বৈশিষ্ট্যই হলো মানুষকে এক্যবদ্ধ করা, কিন্তু এ যেমন বৃহৎ তেমনি জটিল তাঁর প্রভাব। স্বপ্ন ও তাঁর অনুমিত চেতনার বিকৃতি আরো এক চক্রান্তের পূরু স্তর তৈরি করে। চেতনার সম্ভাব্য তত্ত্ব এবং কাঠামোগত ভিত্তি এক দুরদর্শিমূলক পটভূমি তৈরি করে মন্তিক্ষের প্রক্রিয়াকরণের জাটিলতা এবং মিশ্রনের ক্ষেত্রে।

ভূমিকা

কখনো ঘূম ভেঙে যাওয়ার পর আপনার ঘূমচলাকালীন স্বপ্নের ছোটো ছোটো টুকরোকে জোড়া লাগিয়ে তাঁর মানে খুঁজেছেন?

আমরা প্রায় সবাই এই কাজটি করে থাকি এবং ভাবতে থাকি যে আমাদের মন্তিক্ষেই কি এই ধরণের সংযোগ তৈরি করে নাকি আমাদের স্বপ্ন গুলো ঠিক তেমনি যেমন এলোমেলো ভাবে আমরা তাদের মনে রাখি।

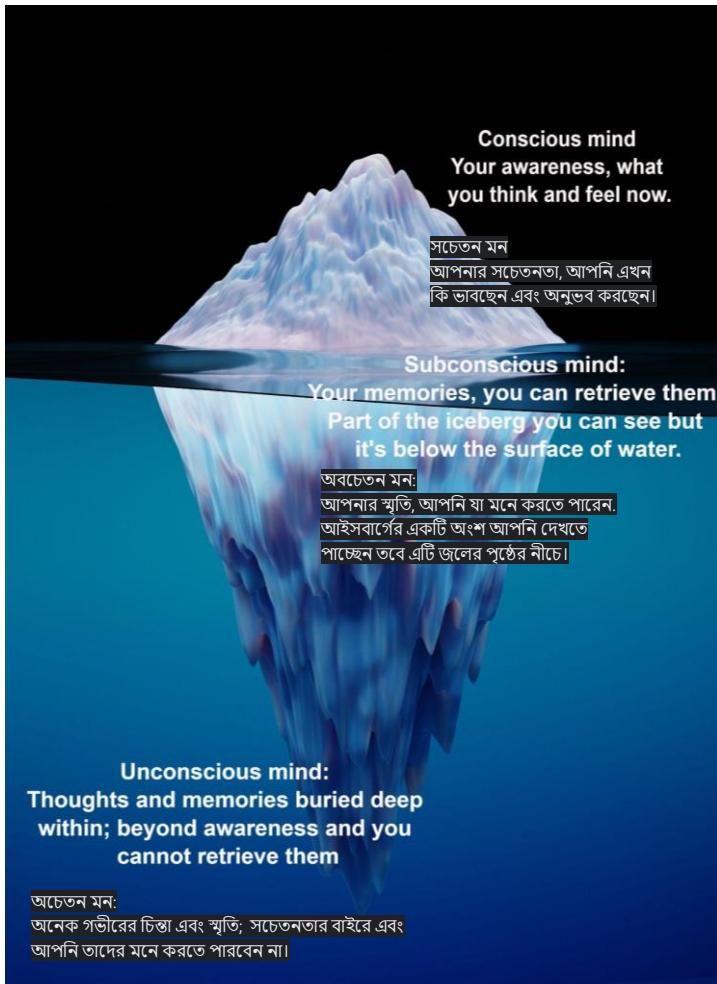
জেগে থাকা এবং সচেতন থাকবার মধ্যে পার্থক্য কোথায়? স্বপ্ন দেখবার সাথে এর বিভাজন কোথায় করা যায়? আমরা এটাই বা কি করে জানতে পারি যে আমরা সাহিত্যের মাধ্যমে বেঁচে নেই এবং আমরা যা অনুভব করি তাঁর সব কিছুই স্বপ্ন নয়?

সাধারণ ভাবে জেগে থাকবার এক অংশ সচেতনতা। বায়োলজিজ(Biology) ভাষায় বলতে গেলে সচেতনতা একটি জীবের উদ্দীপনার প্রতিক্রিয়া। এই অবস্থায় মানুষ ভালো, খারাপ, মন্দ, গানের তাল, চলচিত্র এবং সব কিছুই অনুভব করতে পারে।

এই প্রতিবেদনের দ্বারা সচেতনতার বিভিন্ন তত্ত্ব, অসচেতন মন্তিক্ষের কার্যকলাপ, সচেতনতা ও অসচেতনতার সামাজিক নিউরোসাইন্স(Neuroscience), এবং স্বপ্নের নিউরোসাইন্স, আসুন, শুরু করা যাক।

সচেতন ও অসচেতন মন্তিক্ষ ঘিরে ফ্রয়েডের তত্ত্ব।

প্রায় একশো বছর আগে, একজন অস্ট্রীয় নিউরোলজিস্ট এবং সাইকোএনালিটিক তত্ত্বের অগ্রগামী, সিগমন্ড ফ্রয়েড আমাদের ফ্রয়েডিয়ান আইসবার্গ(Freudian Iceberg) এর সাথে পরিচিত করেছেন, তাঁর এই সৃজনশীল সাদৃশ্য মনুষ্য মন্তিকের ব্যাপারে আমাদের আলোকপাত করে।



চিত্র 1. ফ্রয়েডিয়ান আইসবার্গ।

"মন একটি আইসবার্গের মতো, এটি তার এক-সম্প্রমাণ দিয়ে জলের উপরে ভাসমান।" – সিগমুন্ড
ফ্রয়েড

ছবি ক্রেডিট: <https://unsplash.com/> এ [সাইমন লির](#) ছবি (লেখক দ্বারা সম্পাদিত)

সিগমন্ড ফ্রয়েড সাইকোএনালাইসিস(Psychoanalysis) এর জনক। তার তৈরি ক্লিনিক্যাল পদ্ধতির
উদ্ঘাটন করেছিলেন যার দ্বারা মনুষ মনের মূল্যায়ন করা যায় ও সাইকএনালিস্টরা (psychoanalyst)

কথোপকথনের দ্বারা যে কোনোরকম সচেতন ও অসচেতন মস্তিষ্ক কে চিকিৎসা করা যায়। যদিও বা, সামাজিক নিউরোসাইল্স- এক উঠাতি তত্ত্ব যা এফ.এম.আর.আই.(IMRI) প্রযুক্তি ব্যবহার করে, আমাদের দেখিয়েছে যে অসচেতন মস্তিষ্কের উপর আমাদের কোনোরকম নিয়ন্ত্রণ থাকে না। এই উপলক্ষ্মি আমাদের দেখিয়েছে যে অসচেতন মস্তিষ্ক আমাদের সচেতন মস্তিষ্কের ছায়া নয়। সংলাপের মতন সাধারণ থেরাপিটিক (therapeutic) হস্তক্ষেপ মস্তিষ্কের এই ভাগ বুঝবার জন্য যথেষ্ট নয়। এই নব্য চিন্তাধারা ফ্রয়েডের ট্রেডিশনাল চিন্তা ভাবনার চেয়ে অনেক আলাদা, অসচেতন মস্তিষ্ক আবেগজনিত কারনে লুকোনো থাকে এবং আত্মর্দ্দনের তত্ত্বের দ্বারা আবার জানু করে ফিরিয়ে নিয়ে আসা যায়, এই তত্ত্ব এই নতুন প্রযুক্তির চেয়ে আলাদা।

আধুনিক নিউরোসাইল্স এবং চেতনা

সোশ্যাল নিউরোসাইল্স(Social Neuroscience) কে আমরা তিনটি ভাগে বিভক্ত করতে পারি-সোশ্যাল সাইকোলজি(Social Psychology), সাধারণ মানুষের মধ্যে যোগাযোগের বিজ্ঞান, কগনিটিভ সাইকোলজি(Cognitive Psychology), সাধারণ মানুষের ভাবনা চিন্তার বিজ্ঞান এবং নিউরোবায়োলজি(Neurobiology), যা মস্তিষ্কের এনাটোমি(Anatomy), ফিজিওলজি(Physiology), ও বায়োকেমিস্ট্রির(Biochemistry) ফাঁকেন এর ব্যাপারে। আধুনিক প্রযুক্তি আমাদের মনুষ্য মস্তিষ্কের ব্যাপারে জ্ঞান অর্জন আরো অনেক সহজ করে দিয়েছে, এখন আমরা প্রত্যেকটি ব্যবহারের নিউরাল পাথওয়ে (Neural Pathway) ও মলিকিউলার মেকানিজমের(Molecular Mechanism) এর যোগ সৃষ্টি করতে পারি।

আমাদের সংবেদনশীল উপলক্ষ্মি, যেমন চোখে দেখা বা কানে শোনার সাথে আমাদের সামাজিক উপলক্ষ্মির অনেক মিল, ঠিক যেমন মানুষ বা পরিস্থিতির সমালোচনা করা। দুই ক্ষেত্রেই অসচেতন মস্তিষ্কের প্রভাব প্রচুর, যৌটি একটি ছাকনির মতন কাজ করে, এবং এই পৃথিবীর একটি স্পষ্ট চিত্র আমাদের সামনে তুলে ধরে। এই অন্তর্ভুক্ত ছাকনি ছাড়া আমরা যা দেখি বা শুনি সবই অপ্রতিরোধ্য এবং ঘূর্ণিঝূর্ণী হয়ে পড়ে।

সচেতন ও অসচেতন মস্তিষ্ক কাঁধে কাঁধ মিলিয়ে কাজ করে। আপনি যখনই বই এর নাম শোনেন তখন আপনার মস্তিষ্কে বইয়ের চিত্র ভাসে, অনেক গুলো পাতা একসাথে বাঁধা, প্রত্যেকটি পাতাতেই রয়েছে ছবি বা লেখা, এত আপনার সচেতন মস্তিষ্কের চিত্ত। আপনার অসচেতন মস্তিষ্ক তখন হয়তো ভেবে চলেছে আপনার কিছু প্রিয় বইয়ের কথা, যা আপনি একটি ফাঁকা সন্দরের পারে বসে জুস খেতে খেতে পড়ছেন।

চেতনা আমাদের অঙ্গান্তর কে রেঁধে রাখে। যদি অঙ্গান্তর হয় ঘোড়া, তাহলে চেতনা তার লাগাম, এই কারণেই আমরা নিজেদের নিত্যনিরের কাজ কোনোরকম বাধা ছাড়াই করে চলেছি এবং অঙ্গান্তর আজে বাজে চিন্তাভাবনার দ্বারা বিক্ষিপ্ত হচ্ছি না। চেতনা অনেকটা সার্কাসের রিংমাস্টারের মতন, রিংমাস্টার যেমন চাবুক হাতে বাঘ-সিংহ কে নিয়ন্ত্রণে রাখে ঠিক তেমনি চেতনা আমাদের অসচেতন মস্তিষ্কের চিন্তা-ভাবনাদের নিয়ন্ত্রণে রাখে। এই প্রতিধান ছাড়া আমাদের মস্তিষ্ক এক জাহাজের মতন যার কোনোরকম পাল নেই, চালক নেই।

চেতনার তত্ত্ব

চেতনা কে আজীবন কাল ধরে দেখা হয়েছে এক রহস্যময় সন্তান মতন করে, যার কোনো কাঠামোগত বা নিউরোবায়োলজিক্যাল(Neurobiological) ভিত্তি নেই। চেতনা সম্মত বৈজ্ঞানিক পড়াশুনা শুরু হয় ২০

খিস্টার্বে। তখন থেকেই, চেতনার প্রক্রিয়াকরণ সম্মতে বর্ণনা এবং স্থানের ব্যাপারে ৪ রকম ভাবে এগোনো হয়েছে:-

গ্লোবল ওয়ার্কস্পেস থিওরিস(Global workspace Theory)

হাই অর্ডার থিওরি(High order theory)

ইন্টিগ্রেটেড ইনফরমেশন থিওরি(Integrated information theory)

রি-এন্ট্রি এবং প্রেডিক্টিভ প্রসেসিং থিওরিস(Re entry and Predictive processing theories)

প্রথম দুটো পদ্ধতি চেতনার কার্যকরী দৃষ্টিভঙ্গি দিকে আমাদের নজর আকর্ষণ করে, এবং পরের দুটো চেতনা বে অভিজ্ঞতার সাথে তুলনা করে কাজ করে। কাঠামোগত ভাবে বলতে গেলে, প্রত্যেকটি থিওরি মাস্তিক্ষের বিভিন্ন দিক নির্নয় করে, এক্রমত ভাবে বলা যায়, সেরিব্রাল কর্টেক্স(Cerebral Cortex) কে চেতনার আসন বলা হয়। গেটিংঁ এর মতন কার্য হয় মিডব্ৰেন রেটিকুলার ফর্মেশন(Midbrain Reticular Formation) ও থালামিক নিউক্লেইস(Thalamic nuclei) এর জন্য। কিছু প্রিফ্রন্টাল এরিয়ায় (prefrontal area) এ ইন্ট্রাক্রেনিয়াল ইলেক্ট্রিক্যাল স্টিমুলাইজেশন(Intracranial electrical stimulation) অভিজ্ঞতা চেতনায় ব্যাকুলতা ঘটিয়েছে, এবং বুঝিয়েছে যে এই এসিব জ্ঞানগার ও অবদান রয়েছে এতে।

জিউলিও টোনোনি ২০০৪ সালে ইন্টিগ্রেটেড ইনফরমেশন থিওরি(Integrated information theory) প্রস্তাব রাখে, যেটি একটি কারনের প্রভাবে বেরিয়ে আসে, মূলতঃ এটি চেতনার অভিজ্ঞতা এবং তার বৈশিষ্ট্য থেকে পিছিয়ে আসা, যাদের বলা হয় এক্সিওম(Axiom), অনুমান করা হয় ফিজিক্যাল সিস্টেমের(physical system) বৈশিষ্ট্য যাদের পশ্চালেট(Postulate) বলা হয়, তাদের কে সাহায্য করে। এটি আমাদের বোঝায় যে চেতনা সবসময় নির্ভর করে মাস্তিক্ষের তথ্য সংগ্রহ ক্ষমতার উপর এবং এটি মূলতঃ একটি পোস্টারিয়ার করটিক্যালে(posterior cortical) হট জোন এর সাথে যুক্ত।

এই তথ্য চেতনার হিসেব করতেও কাজে লাগে, ফাইমেট্রিক(phi metric) দ্বারা চেতনার পরিমাপ করা হয়।

গ্লোবল নিউরন ওয়ার্কস্পেস(Global Neuron Workspace) আমাদের মাস্তিক্ষ কে একটি ফাঁকা ব্ল্যাকবোর্ডের সাথে তুলনা করে থাকে। একটি ফাঁকা জ্ঞানগা, যেখানে বিভিন্ন রকমের জ্ঞান ভিত্তিক সন্তারা একসাথে চেতনার অভিজ্ঞতামূলক কথোপকথনের দ্বারা ভরিয়ে তোলে। গ্লোবল ওয়ার্কস্পেস এর তত্ত্ব অনুযায়ী আমাদের মাস্তিক্ষে সচেতন চিন্তা ভাবনা আসচেতন চিন্তা ভাবনার থেকে বেশি বিখ্যাত, সচেতন চিন্তা ভাবনার সম্প্রচার মাস্তিক্ষ জুড়ে অনেক বেশি পরিমাণ হয়। অনুমান করা হয় যে এই কার্যটি ফাঁকশানাল বেন ইমেজিং- একুপ সমর্থন পায়, সচেতন জ্ঞান মাস্তিক্ষ জুড়ে ছড়িয়ে পড়ার কারণ অসম্ভব করটিক্যাল একটিভিটি(Cortical Activity) ফ্রন্টপেরিয়েটাল(Frontoparietal) ও মিডিয়াল টেম্পোরাল(Media temporal) জ্ঞানগা গুলি তে অর্থ সে জ্ঞানগায় অসচেতন মাস্তিক্ষ কাজ করে শুধু মাত্র স্থানীয় কিছু জ্ঞানগায়। গভীর ঘৃম, সাধারণ এনাস্থেসিয়া(Anesthesia) অথবা কোমায় থাকাকালীন ও দেখা গেছে মাস্তিক্ষে মেটাবলিক রিএক্সন(Metabolic Reaction) অতিরিক্ত কর্ম।

হাইয়ার অর্ডার থিওরি সচেতন মাস্তিক্ষ কে একটি বাচ্চা ছেলের সাথে তুলনা করেছে-যে জানে তার অস্তিত্ব রয়েছে শুধু মাত্র তার পিতামাতার জন্য। এই থিওরি গুলি প্রত্যেকটিই অবস্থান পরিবর্তনসূচক উপসর্গ, যেটি আমাদের বোঝায় যে আমাদের সচেতন মাস্তিক্ষ ততক্ষণ অবধি সচেতন ঘটক্ষণ আমরা নিজেরাও সচেতন। এই থিওরি কে আমরা ট্র্যান্সিভিটি থিওরি বলেও জেনে থাকি। এই থিওরির দ্বারা আমরা সচেতন মাস্তিক্ষ ও অসচেতন মাস্তিক্ষের বিভাগ করতে পারি।

সব শেষে আমাদের কাছে রয়েছে প্রেডিকটিভ প্রসেসিং(Predictive Processing)। প্রেডিকটিভ প্রসেসিং খুব সুন্দরভাবে আমাদের নিয়ন্ত্রণের কাজের দ্বারা বুঝতে পারি, যেমন গানের তালে আমাদের পা দোলানো। আমাদের মস্তিষ্ক গানের তালটার আন্দাজ করে এবং সেই তথ্য দিয়ে আমাদের গানার তাল আন্দাজ করতে সহায় করে এবং আমাদের পা সেই তালে পা নাচায়। এই আন্দাজ বিভিন্ন রকমের সিগন্যালের দ্বারা আমরা বুঝতে পারি। প্রেডিকটিভ প্রসেসিং এর চেতনার তত্ত্বের সাথে কোনো যোগসূত্র নেই, এটি বলা হয়েছে যে এই তত্ত্বের মনোযোগের ক্ষেত্রে খুব গুরুত্বপূর্ণ কাজ করে এবং আমাদের গুরুত্বপূর্ণ তথ্য দিয়ে ভরিয়ে তোলে।

চেতনা ও স্বপ্ন দেখা

অনেক থিওরি বোঝানোর চেষ্টা করেছে যে সচেতন মস্তিষ্ক ও অসচেতন মস্তিষ্কের যোগসূত্র হয় আমাদের স্বপ্নের দ্বারা। যদিও আমরা এখনো নিশ্চিত নয় এর কতটা সত্যি আর কতটা মিথ্যা, সত্যি হতে পারে এই দুই ভাগের মিশ্রণ, একটি অত্যন্ত গুরুতনপূর্ণ তথ্য হলো:-

আমরা যখন ঘুমাই আমাদের সচেতন মস্তিষ্কও ঘুমায়, এই সময় তেই অসচেতন মস্তিষ্ক যেখান খুশি ঘুরে বেড়ায় এবং চিন্তা ভাবনা করে আমাদের মধ্যে থাকা স্মৃতি নিয়ে, যখন আমাদের ঘুম ভেঙে যায় সচেতন মস্তিষ্ক আবার আমাদের চিন্তাভাবনার দখল নেয় তখন স্বপ্নে দেখা জ্ঞানের কথা মনে রাখে এবং আজে বাজে চিন্তা আবার হারিয়ে যায়।

এতক্ষনে আপনাদের কাছে নিশ্চয় স্পষ্ট হয়ে গেছে যে চেতনার অভাবে আমরা স্বপ্ন দেখি, এবং সেটা আমাদের বোঝায় যে আমরা কেন সারা ঘুম ধরে স্বপ্ন দেখি না।
আমাদের ঘুমের সময় আমাদের মস্তিষ্ক বাধনচাড়া হয়ে যায়, কারণ তখন আমরা **রেপিড আই মুভমেন্ট(rapid eye movement)** পর্যায় থাকি। আমাদের ব্লাড প্রেসার, হার্ট রেট এবং ভল্যান্টারি একসন কমিয়ে দিয়ে আমাদের শরীর স্বপ্নে দেখা অবস্থার জিনিস করতে বাঁধা দেয়।

কখনো দেখেছেন আপনি একই সমস্যা নিয়ে ঘুমেতে গেছেন এবং সকালে উঠেছেন তার সমাধান নিয়ে, এটি আপনার অসচেতন মস্তিষ্কের কামাল। রিসার্চ এও দেখিয়েছে যে স্বপ্নের দ্বারা আমাদের শৈলিক দিক বেরিয়ে আসতে পারে। এটি হওয়ার কারণ মূলত আর.ই.এম. পর্যায় আমাদের **নোরাদ্রেনালাইন(noradrenaline)** হরমন সবসময় কর থাকে না, এটির কারনে আপনার অসচেতন মস্তিষ্ক সেইসব চিন্তা করতে থাকে যা আপনার সচেতন মস্তিষ্ক চেপে রেখেছিল।

‘বলা হয় সমস্ত ঘায়ের একটাই মলম, সেটা হলো সময়, কিন্তু আমার গবেষণা আমায় বিশ্বাস করিয়েছে যে স্বপ্নে কাটানো সময় আমাদের ঘায়ে মলম লাগায়ঃ ডক্টর ম্যাথিউ ওয়াকার।

মন এবং চেতনা সম্পর্কে আমরা যা জানি (অথবা আমরা যা মনে করি আমরা জানি) তা পাতন করে এবং ফোকাস করে, প্রতিদিন নতুন নতুন তত্ত্বগুলি প্রস্তাবিত হতে থাকে। বিদ্যমান তত্ত্বের মধ্যে কি বিষয়টির সত্যতা রয়েছে? নাকি খেলার মধ্যে কিছু এখনও-আবিষ্কৃত শক্তি আছে?

যাই হোক, বলা হয় যে সময় আসলে সব জানা যাবে, হয়তো সময় হলেই জানতে পারবো।

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অজ্ঞান হওয়ার নিউরোসিয়েন্স The Neuroscience of Fainting

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সারসংক্ষেপ

অনেক সময় দেখা গেছে অনেক মানুষ অনেক চাপযুক্ত সময়ে দুম করে অজ্ঞান হয়ে পড়েছেন, এটি একটি সাধারণ প্রতিক্রিয়া। মাথায় সঠিক পরিমাণ রক্ত না পৌঁছনোর কারণে মানুষ অজ্ঞান হয়ে পড়েন। মাথার নিউরনেরা(neuron) পর্যাপ্ত পরিমাণ গ্লুকোজ ও অক্সিজেন না পাঠাবার কারণে সাময়িকভাবে কাজ করা বন্ধ করে দেয় এবং সেই কারণে মানুষে অজ্ঞান হয়ে পড়ে। রক্তচাপ কমে যাওয়ার অনেক কারণ দেখা গেছে, পরিস্থিতি বা শারীরিক অসুস্থিতার কারণেই হলো প্রধান কারণ অজ্ঞান হয়ে যাওয়ার।

ভূমিকা

ধপ করে শব্দ!

সমষ্টিগত হাঁপানির শব্দ, কিছু প্রাপ্ত বয়স্ক লোকের এদিক সেদিক ছুটে বেড়ানো, এবং আরো কিছুক্ষন পরেই স্পোর্টস টিচার কে দেখা যাচ্ছে একটি ছেলে কে নিয়ে সে ইনফারমারীর দিকে চলেছেন। আমরা সবাই জানি ছেলে টি অজ্ঞান হয়ে পড়েছে, স্কুলের প্রথম দিনেই।

অজ্ঞান হয়ে যাওয়ার ধারণা আমাদের আজকের নয়, পর্দায় নায়ক দের নাটকীয় ভাবে পড়ে যাওয়া থেকে সকা঳ে স্কুলের এসেসলি তে শ্লীলতাহনি-মস্তিষ্কে ঘটে চলা জিনিস কে আমরা বাদ দিয়ে দিএ। এই কারণেই বহু বৈজ্ঞানিক উত্তর দেওয়ার চেষ্টা করেছে যে যদিও সুর্বৈর তেজ ই কি আপনার অজ্ঞান হয়ে যাওয়ার কারণ নাকি এর পেছনে আরো কিছু লুকিয়ে রয়েছে?

ভাবুন আমাদের শরীর ঠিক যেন একটি টিভির প্লাগ। ধারণা করা যাক সেই প্লাগ টি আলগা হয়ে পড়েছে, কিছু মুহূর্তের মধ্যেই সেটি আলগা হয়ে বাইরে পরে গেছে এবং চলমান বোকা বাক্স হয়ে যায় বন্ধ, অজ্ঞান হয়ে যাওয়া টাও অনেকটা এককর্ম। ডাঙ্কারের অজ্ঞান হয়ে যাওয়া কে তাঁদের ভাষায় বলেছেন সিনকোপ(Syncope)- ক্ষণস্থায়ী ভাবে চেতনা হারিয়ে ফেলা। প্লাগ টি কে আবার জোরে গুঁজে দিন আপনার টিভি আবার ঠিক আগের মতনই চলবে।

আপনার মস্তিষ্কের পাওয়ার সাপ্লাই আপনার রক্ত, যা তার সঙ্গে করে নিয়ে আসে গ্লুকোজ ও অক্সিজেন। রক্তের অভাব অজ্ঞান হয়ে পড়বার মূল কারণ। রক্ত চাপ কমে যাওয়ার কারণ হতে পারে অনিয়মিত হার্টাবিট, যেটি আবার সঠিক ভাবে মস্তিষ্কে রক্ত পৌঁছানোয় বাধা সৃষ্টি করে। অন্যদিকে অজ্ঞান হয়ে পড়বার আরো অনেক কারণে মধ্যে রয়েছে বমি বমি ভাব, মাথা ঘোরা, ফ্যাকাশে চামড়া, ঠাণ্ডা লেগে যাওয়া, অতিরিক্ত ঘাম হওয়া, ইত্যাদি, যারা সকলেই কিনা অজ্ঞান হয়ে পড়বার অনেক গল্প শোনাতে পারে।

অজ্ঞান হয়ে পড়বার কারণ ও ধরন।

অজ্ঞান হয়ে পড়বার মূল কারণ আমাদের হৃদপিণ্ডের স্পন্দন, স্পষ্ট ভাবে বলতে গেলে হৃদপিণ্ডের স্পন্দনের গতি-সেটি দ্রুত না ধীর। যখন আমাদের হৃদস্পন্দন এর লাব-ডুব ছন্দে(যাকে বলা হয় আরহায়তমিয়া) ছন্দপতন হয়, তখন বিভিন্ন অঙ্গে সঠিক পরিমান অক্সিজেন ও গ্লুকোজ পৌঁছায় না। মিকাসের বিজ্ঞাপন টি মনে আছে: শ্রুত্বার্থ এম.এস.ধৰ্ম যখন একইসাথে শ্রুত্বার্থ ও রাগাধিত হয়ে পড়ে, এই রকম অবস্থায় মানুষ অজ্ঞান হয়ে পড়ে। যখন মন্তিক্ষের নিউরন দের গ্লুকোজ ও অক্সিজেন থেকে বিরত রাখা হয়, তাঁরা বন্ধ হয়ে পড়ে এবং সেই মানুষটি হয়ে যায় অজ্ঞান।

আর এক রকমের অজ্ঞান হয়ে পড়বার কারণ দেখা গেছে। তা হলো পরিস্থিতির প্রতিফলন। একটি বন্ধুর কথা ভাবুন যে মাকড়শা কে ভয় পায়। যখন মাকড়শা কে সে দেওয়ালে চলা-ফেরা করতে দেখে, আন্দাজ করতে পারেন সে কি করে উঠতে পারে? এখানে, শরীর রক্ত চলাচল কে কোনোভাবেই নিয়ন্ত্রণ করতে পারে না, যার ফলে সঠিক পরিমান অক্সিজেন বা গ্লুকোজ শরীরের বিভিন্ন অংশে পৌঁছাতে পারে না, এবং আমাদের মন্তিক্ষে সাময়িকভাবে কাজ করা বন্ধ করে দেয়, যার ফলে মানুষটি অজ্ঞান হয়ে পড়ে। অজ্ঞান হওয়াকে আমরা আরো দুটি ভাগে ভাগ করতে পারি-সাধারণ অজ্ঞান বা কমন ফেন্ট এবং পরিস্থিতির কারনে অজ্ঞান বা সিচুএসানাল ফেন্ট, অনেক কারনে মধ্যে এই দুটি কারণ।

কমন ফেন্ট অথবা 'ভেসেভগাল সিনকোপ' (Vasovagal Syncope) এর কারণ আবেগ জনিত কারন বা শারীরিক কষ্ট। যেই নারী অভিনেতা তাঁর প্রেমিকের দুর্ঘটনার কারণ শুনে, বা যেই ছেলেটি তাঁর হারিয়ে যাওয়া জমজ ভাই কে খুঁজে পায়, বা যেই ছেলেটি বেশ কিছুক্ষণ রোদে দাঁড়ানোর কারনে অজ্ঞান হয়ে পড়ে, তারা সবাই এই কমন ফেন্ট এর ভৃক্তৃগুলি। এই পরিস্থিতি সে আবেগ জনিত কারণে নিউরন উপর চাপ সৃষ্টি করে অথবা অসম্ভব শারীরিক যন্ত্রনা ব্লাড প্রেসার এ প্রভাব ফেলে, যার কারনে সে অজ্ঞান হয়ে পড়ে।

যেই সকল ব্যক্তিরা বিশেষ কিছু পরিস্থিতিতে বা কারনে অজ্ঞান হয়ে পরেন তাদের কে বলা হয় সিচুএসনাল ফেন্ট। একটি ঘটনার কথা ভাবা যাক, যখন একজন ম্যারাথন রানার ফিনিশিং লাইন অতিক্রম করে, সে অজ্ঞান হয়ে পড়ে। এক অদ্ভুত জিনিস দেখা যায় যখন এক কুণ্ডি কে ডাঙ্গারের কাছে দেখানো হয় কারণ সে আন্য কাউকে অজ্ঞান হতে দেখে সে অজ্ঞান হয়ে পেছিল, এবং অদ্ভুত ভাবেই এই কুণ্ডাটির সম্পূর্ণভাবে হৃদস্পন্দন বন্ধ হয়ে পড়েছিল, যদিও সেই কুণ্ডি কে পরে সুস্থ করে তোলা হয়। অবশ্য এই জিনিস আপনি একটি রোম্যান্টিক সিনেমা তে খুঁজে পাবেন না।

যখন কোনো মানুষের শরীরে সমস্যা থাকে এবং বিশেষ নিউরোলজিক্যাল কোনো সমস্যা, তাতে মানুষের অজ্ঞান হয়ে যাওয়া টা খুবই স্বাভাবিক।

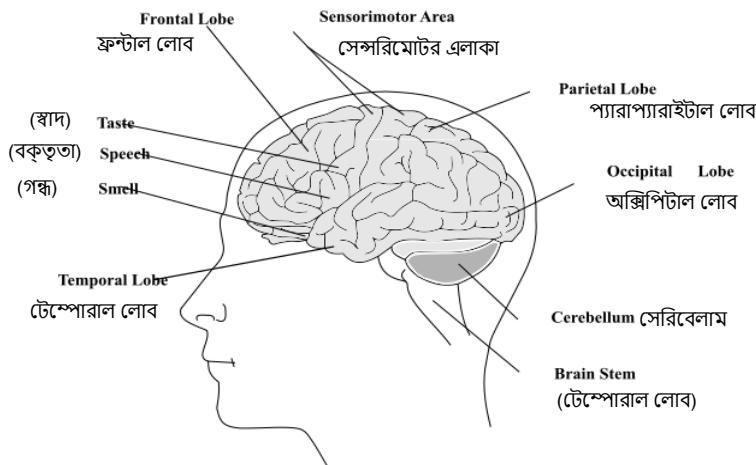
তবে এই অজ্ঞান হয়ে পড়বার কারণ কি? আমাদের বৈজ্ঞানিকেরা নিশ্চিত না হলেও তাঁদের কিছু অনুমান করা তথ্য আমাদের সঙ্গে রয়েছে।

সেরোটোনিন(Serotonin) আমাদের শরীরের এক ধরনের হরমোন যা আমাদের ঘৃম, মেজাজ, এবং এক নিউরন থেকে কোনো নিউরনে তথ্য প্রচার করে। আমাদের শরীর রাসায়নিক ভাবে সেরোটোনিন কে মেলোটোনিন(Melatonin) এ পরিবর্তন করে যার ফলে আমাদের ঘৃম পায়। ঘৃম এক ধরণের অসচেতনতা। এই তথ্য আমাদের মধ্যে থাকবার দরুন অজ্ঞান হয়ে যাওয়ার ক্ষেত্রে এই হরমোন কে আমরা আগ্রহ্য করতে পারি না। সেরোটোনিন প্যারাসিমপ্যাথেটিক নার্ভাস সিস্টেমের(Parasympathetic nervous system) সাথেও যুক্ত। এই প্যারাসিমপ্যাথেটিক নার্ভাস সিস্টেম যে কোনোরকম চাপসংষ্টির কারণ বা শান্তিপূর্ণ পরিস্থিতির জন্য দায়ী। সহজ ভাষায়, সেরোটোনিন এ হঠাৎ পরিবর্তন হৃদস্পন্দনের গতি কমিয়ে দিতে পারে। যদিও বা সেরোটোনিনের ভূমিকা ও

অজ্ঞান হয়ে যাওয়ার ক্ষেত্রে আজও একটি আলোচ্য বিষয় এবং দিনের পর দিন বিভিন্ন রোমাঞ্চকর গবেষণা এর উপর হয়ে চলেছে।

আমরা এও দেখেছি যে অতিরিক্ত চাপ মানুষ কে অজ্ঞান করে দিতে পারে। একটি বিশেষ সার্ভে অনুযায়ী কমন ফেন্ট এর কারণ হিসেবে প্রধানত মানসিক বা সামাজিক কারণ কেই দেখা গেছে। দেখা গেছে যেই সকল মানুষের প্রায়শই অজ্ঞান হয়ে পড়েন তাদের রয়েছে অতিরিক্ত ডিপ্রেশন অথবা মানসিক ব্যাধি এবং কোনোরকম চিকিৎসায় তাদের কোনো প্রভাব পড়ছে না। ওই বছরেই, একটি ক্লিনিক্যাল স্টাডিওর মাধ্যমে জানা যায় অনেক রুগ্নীদের ঘার নিয়মিত অজ্ঞান হয়ে পড়েন। এখানে লেখক রা আবিষ্কার করে ঘার অসম্ভব চাপের মধ্যে দিয়ে পার হয়েছেন তারা প্রায়শই অজ্ঞান হয়ে পড়েছেন এবং তার পেছনে কোনো রকম কারণ থুঁজে পায় নি তারা। এর মানে এই দাঁড়ায় যে অজ্ঞান হয়ে যাওয়ার পেছনে মানসিক কোনো সমস্যা এবং ঘার চিকিৎসা দরকার। সেই স্টাডিতে এও দেখা গেছে অনেক রুগ্নীদের মধ্যে পরিবর্তন দেখা গেছে তাদের চিকিৎসা শুরু করবার পর। এক ধরনের তদন্তের দ্বারা জানা গেছে, যে সকল বাচ্চারা অজ্ঞান হয়ে পরে তাদের জীবন অন্য বাচ্চা দের মতন গুণমান নয়।

আমাদের খুব একটা বেশি জানা নেই সেই বিষয়ে যখন মানুষ অজ্ঞান হয়ে পড়ে তাদের মন্তিক্ষে কি চলে। এক পরীক্ষা মিরীক্ষা করা হয় ৫৯ জন মানুষের উপর, যেখানে তাদের প্রোচিত ভাবে অজ্ঞান করে, তাদের ব্রেনে যাওয়া অক্সিজেন সাপ্লাই করিয়ে দিয়ে(যাকে বলা হয় সেরিব্রাল হাইপোক্রিয়া)। এই পরীক্ষার পর দেখা যায় মানুষের হ্যালুসিনেট করছে, মাটিতে লুটিয়ে পড়ছে এবং অজ্ঞানেই হাত পা ছুড়ছে(যাকে বলা হয় মাইওক্লোনাস), পরীক্ষক রা ই.ই.জি.র সাহায্য নিয়ে অজ্ঞান হয়ে যাওয়া মানুষটার ব্রেন ওয়েভস সনাক্ত করছে এবং তার বিশ্লেষণ করে চলেছে। এই ধরনের ঝাঁকুনি মূলক চলাচল সাধারণত পাইপের তৈরি গোলকর্ধাধা দ্বারা চালিত হয় যা আপনি আপনার টেক্সটবুকে পাবেন, যাকে বলা হয় মন্তিক্ষের করাটিকাল অংশ। এই বিশেষ পরীক্ষায়, ঝাঁকুনি মূলক চলাচল ব্রেনস্টেম দ্বারা লাগাম টানা আছে। যদিও এটি একটি পুরনো খবর, ১৯৯৪ এর প্রকাশিত, তার পরে খুব একটা কেউ এই বিষয়ে তদন্ত করে নি।



চিত্র 1: মন্তিকের অংশ। চারটি লোব (ফ্রন্টল, প্যারিটাল, টেম্পোরাল, অসিপিটাল) সেরিব্রাল কর্টেক্স বা কর্টিকাল অঞ্চল তৈরি করে। ব্রেন স্টেম কর্টেক্সের নিচ থেকে বের হয় এবং মেরুদণ্ডের মতো প্রসারিত হয়।

ইমেজ ক্রেডিট: অজানক এবং <https://openclipart.org/detail/121615/human-brain-by-ozhank>

কিন্তু কেনই বা আজ্ঞান হবো?

যদি আজ্ঞান হওয়া একটাই ক্ষতিকর এবং সাধারণ জীবন থেকে বিপরীত তা ঘটেই বা কেন? আসুন আমরা বাস্তবের মাটিতে পা রেখে চলি, শুধু মাত্র অভিনেতারাই রূপালি পর্দায় আজ্ঞান হয়ে তাদের স্বপ্নপূরন করতে পারে। তাহলে আমাদের শরীর এইরকম একটি কঠিন পরিস্থিতি তে অন্যরকম কোনো সমাধান নিয়ে আসতে পারছে না কেন? দেখে মনে হয় যেন আমরা সব থেকে কঠিন পরিস্থিতি তে যেখানে আমাদের মানসিক জোর তুঙ্গে থাকা উচিত সেখানেই আমাদের শরীর কাজ করা বন্ধ করে দেয়।

বিবর্তনের প্রেক্ষাপট থেকে দেখতে গেলে, আজ্ঞান হয়ে পড়া টা এক ধরনের অভ্যাস, কোনোরকম মেডিক্যাল সমস্যা নয়। প্রাচীনতম তত্ত্ব থেকে আমরা উদাহরণ পেয়েছি খ্লাড-ইনজেকশন-ইনজুরি টাইপ স্পেসিফিক ফোবিয়ার(BIITS phobia)। একটি মানুষ তিন প্রকার প্রতিক্রিয়া দেখায়-ভয়, পরিহার ও আজ্ঞান হয়ে যাওয়া যা যুগ যুগ ধরে আমাদের মধ্যে এক বেঁচে থাকবার অঙ্গীকার হয়ে দাঁড়িয়েছে। আদিম যুগে, মানুষেরা আজ্ঞান হয়ে নিজেদের সুরক্ষিত রাখতেন, কোনোরকম অন্য মানুষের দ্বারা আহত বা নিহত হওয়ার ভয়ে, এই আচরণ হয়তো তখনকার দিনের বহু মানুষেরা আপন করে নিয়েছিল বিপদ মুক্ত হওয়ার কারণে, মহিলা ও বাচ্চাদের মধ্যে এই ব্যবহার বেশ দেখা গেছে, এবং এই কারনে সেই জায়গা ছেড়ে পালিয়ে যেতেও তাদের এই আজ্ঞান হয়ে যাওয়া সাহায্য করেছে। হয়তো অনেক মানুষেরা আজ্ঞান হয়ে নিজেদের শক্তি সঞ্চয় করতেন, এবং বিপদজনক পরিস্থিতি কে এড়িয়ে আসতে পারতেন।

গবেষণার দ্বারা দেখা গেছে মহিলারা বিপদের সময় অনেক বেশি পরিমাণ আজ্ঞান হয়ে পড়েছেন। মেডিক্যাল দিক থেকে দেখতে গেলে দেখা গেছে যে মহিলাদের মধ্যে খ্লাড প্রেসার খুব কম হওয়ার কারণেও তারা আজ্ঞান হয়ে পড়েছে। এক বিশাল অংশ কাজ করে সেক্স হরমোনের উৎপত্তির কারনে।

অতীত দর্শন।

আজ্ঞান হয়ে যাওয়া যেখানে একটি গভীর সমস্যা, সেটাকে জীবনীশক্তি সঞ্চয় করবার এক ধরণে বিবর্তনীয় উন্নয়ন বলা যেতেই পারে, যা মানুষের বেঁচে থাকবার অঙ্গীকার হয়ে রয়েছে যুগ যুগ ধরে। কিন্তু একে এক রকম মানসিক, শারীরিক রোগ বলেই আজকের অত্যধূমিক যুগে গণ্য করা হয়, কারণ পৃথিবী জুড়ে এখন আর আদিমানবেরা বসবাস করে না। এই ধরনের জীবন বাঁচানো পদ্ধতি আমাদের এই যুগে আর কাজে লাগে না, এবং এই পদ্ধতি কে আজকের দিনে রোগ বলেই চিহ্নিত করা হয়।

নিয়মিত সিনকোপ একজন কে সাহায্য করতে পারে সমস্যার কারণ জানতে এবং তাকে চিকিৎসা করতে। যদিও আজ্ঞান হয়ে যাওয়ায় কোনো সমস্যা নেই, কিন্তু আজকের দিনে তা একটা সমস্যার কারণ।



চিত্র ২ শেক্সপিয়ারের মাচ অ্যাডো অ্যাবাউট নাথিং থেকে হিরো তার বিরুদ্ধে অভিযোগ শুনে অজ্ঞান হয়ে যায়।

ইমেজ ক্লেডিট: এলমার এবং<https://journey-and-destination.blogspot.com/2013/09/shakespeare-scenes-in-art.html>

অজ্ঞান হয়ে যাওয়ার যদি সাংস্কৃতিক রূপ হয় তাকে বলা যেতে পারে বেহশ হয়ে যাওয়া। শেক্সপিয়ারের "মাচ এন্ড এবাউট নাথিং" এর মতন একটি অত্যন্ত জটিল গল্পে যেখানে গল্প জুড়ে রয়েছে শুধু কুটিলতা, বিপ্রাণ্তি মূলক ঘটনা, বিবাহ যেখানে গল্পের নায়িকার উপর অভিযোগ উঠলে তাকে অজ্ঞান হয়ে যেতে দেখা গেছে। উইলিয়াম শেক্সপিয়ার কে বহুবার দেখা গেছে গল্পের পটভূমি তে উনি অজ্ঞান হয়ে যাওয়া কে বেছে নিয়েছেন, যার থেকে আমরা ভিট্টোরিয়ান যুগের সংস্কৃতির কথা পেয়েছি। অজ্ঞান হয়ে যাওয়া কে বেছে নেওয়া হতে কিছু বিশেষ পরিস্থিতি বোঝানোর জন্য, নায়ক নায়িকারদের উদ্দেশ্যে, কোনো কথার গভীরতা বোঝানোর জন্য।

বেহশ হয়ে যাওয়ার উদাহরণ আমরা মধ্যযুগীয় সময় থেকেই দেখে আসছি যেখানে মানুষ কে ভালোবাসার প্রকাশ, কোনো মৃত্যুসংবাদ অথবা কোন না জানা অতীত এর খবর এ অজ্ঞান হয়ে যেতে দেখা গেছে। চালর্স দিকেনসের ব্লিক হাউজ এ গল্পের নায়িকা লেডি ডেলক কে তার অতীত ভুলে যাওয়ার প্রক্রিয়ায় অজ্ঞান হয়ে যেতে দেখা গেছে।

বেহশ হয়ে যাওয়া সব থেকে ভালোভাবে বর্ণনা করেছেন নায়িকাদী লেখিকা এনজেলা কার্টার, তাঁর ব্লাডি চেস্বার গল্পে তিনি লিখছেন "ব্লাডি চেস্বার এর ভয়ঙ্কর উদযাচিন এর পর, তাঁর মেহপূর্ণ তাকানো আমায় বেহশ করে দিয়েছিল।"

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ABOUT



The Mind Gala is a year-long initiative by Project Encephalon and the Thakur Neurodegeneration Lab to encourage more people to explore neuroscience. This initiative is funded by the 2nd IndiaBioscience Outreach Grant. The various outreach activities involve webinars, workshops, mentorship sessions etc.



Thakur Neurodegeneration Lab is a lab by Dr. Poonam Thakur. Dr Thakur is a neuroscientist working as an Assistant Professor at IISERTVM. She completed her Ph.D. from Panjab University, Chandigarh followed by post-doctoral research from Lund University, Sweden, and Goethe University, Germany. The Thakur lab primarily works in the field of Parkinson's Disease (PD) which is one of the most common age-associated neurodegenerative disorders. The major research goals of the lab are to develop better mouse models of PD in order to decipher the disease mechanisms and utilize this understanding to generate biomarkers as well as therapeutics for this incurable disease.



Project Encephalon (PE) is an international, trainee-led non-profit organization for neuroscience enthusiasts and conducts various academic and non-academic neuroscience-related activities while accounting for the interdisciplinary needs of the field. PE aims to develop a vibrant and inclusive community for anyone with an interest in neuroscience, where they can reap all the benefits this organization offers despite their

socioeconomic background and as a result, there are no fees to join the organization and all the activities conducted by PE are available without any price.



IndiaBioscience is a program that works to promote positive changes in the life sciences in India by engaging with various groups, including academia, government, and industry. Its goal is to increase the visibility of science in society through policy discussions, science communication, and information aggregation. Based at the National Centre for Biological Sciences in Bangalore, IndiaBioscience serves the entire life science community in India.

Have you ever wondered about how your brain can perform multiple tasks so effortlessly?

Did you know that your gut can effect your brain via gut-brain axis?

Have you ever wondered why learning new things can be so challenging?

The Tales of Neuroscience is a collection of articles written by students just like you, who participated in a 6-week mentorship program where they learned about science writing and communication. Inside, you'll discover the mysteries of the brain and how it impacts our daily lives. From the structure and function of the nervous system to the diseases that can affect it and the latest technology being developed. Neuroscience is an incredible discipline, and this book breaks it down in an easy-to-understand and exciting way.

This book is an outcome of a successful partnership between Project Encephalon and the Thakur Neurodegeneration Lab at IISER-Thiruvananthapuram. The Mind Gala initiative aims to foster interest in the field of neuroscience by simplifying complex concepts.



The Mind Gala



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