CS2

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About the Journal

Our Mission

Neuro Frontiers is a student-run research journal that empowers high school students to delve into the captivating field of neuroscience. One of the key focuses of Neuro Frontiers is to shed light on the critical intersections between neuroscience and mental health, particularly in the context of adolescence. By delving into these complex connections, the journal aims to not only expand our understanding of the brain but also raise awareness about the importance of mental well-being among young individuals.

Upcoming Issues

Spring Seasonal Issue (Vol. 2, Issue 02)

Summer Seasonal Issue (Vol. 2, Issue 03)

Resources

If you wish to continue your reading, here are a few credible resources:

https://pubmed.ncbi.nlm.nih.gov/ https://www.nlm.nih.gov/ https://scholar.google.com/ https://europepmc.org/





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Dear Readers,

The Winter Seasonal Issue of Neuro Frontiers is here! When this journal started in July 2024, the goal was simple: to create a space where students could explore neuroscience in ways that feel relevant and exciting. Since then, Neuro Frontiers has grown beyond what I could have imagined—reaching peers around the world, expanding our team, and continuing to uncover the fascinating ways neuroscience connects to everyday life.

This issue brings a mix of thought-provoking topics. We're diving into how burnout physically changes the brain, why multitasking is worse for you than you think, the incredible phenomenon of synesthesia, and the longterm effects of repeated mild traumatic brain injuries (mTBI). There's no single theme, but every piece ties back to a bigger question: how do our brains shape the way we experience the world?

We're also introducing a Did You Know? section—highlighting quick, surprising facts about the brain. From the shrinking prefrontal cortex in burnout to the way some people can literally "taste" words, these small insights remind us just how complex and remarkable the brain really is.

Thank you for being part of this journey, whether you've been with us since the first issue or are just discovering *Neuro Frontiers* now. This journal is built by students, for students, and I hope this issue leaves you with something new to think about.

-Varsha Senthilkumar



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Scientific Review

The Neuroscience of Burnout and Resilience

Varsha Senthilkumar

ABSTRACT: Chronic stress significantly alters brain structure and function, leading to cognitive decline, emotional dysregulation, and conditions such as burnout. These effects manifest through structural and functional changes in key brain regions, including the hippocampus, prefrontal cortex, and amygdala. The hippocampus, which plays a crucial role in memory consolidation, is particularly vulnerable to stress-induced atrophy. In the prefrontal cortex, stress is shown to yield functional impairments that affect executive control and emotional regulation. Conversely, the amygdala, a critical center for processing emotions, becomes hyperactive, heightening stress sensitivity and increasing susceptibility to anxiety and mood disorders. This review synthesizes current research on the neural mechanisms underlying these changes, with a particular focus on the role of the hypothalamic-pituitary-adrenal (HPA) axis and stress-related neurotransmitter imbalances. Additionally, it explores the impact of chronic stress on the dopaminergic reward system and discusses strategies to enhance resilience. By integrating findings from neuroscience, psychology, and public health, this paper highlights the necessity of targeted interventions to mitigate the detrimental effects of chronic stress.

INTRODUCTION

Stress is an inevitable part of life and, in short bursts, can enhance motivation and cognitive performance. However, chronic stress—prolonged exposure to stressors—can significantly impact the brain, altering neural plasticity, impairing cognitive function, and increasing susceptibility to psychiatric disorders. The hypothalamic-pituitary-adrenal (HPA) axis plays a central role in this process, as prolonged stress leads to excessive cortisol (the primary stress hormone) release. Normally, cortisol helps regulate the stress response by signaling the HPA axis to shut off further production. But under chronic stress, this feedback system becomes dysregulated, leading to persistently high cortisol levels and its damaging effects (McEwen, 2007).

Stressors vary widely, including interpersonal (e.g., conflict, loss), professional (e.g., job pressure, academic demands), and societal (e.g., economic instability, discrimination) stress. Their impact on the brain depends on biopsychosocial factors, which influence whether stress builds resilience or leads to trauma—a concept explored in *What Happened to You?* by Oprah Winfrey and Dr. Bruce Perry. As stress becomes more prevalent in modern society, particularly among students, working professionals, and individuals in high-pressure environments, understanding its effects on the brain is more critical than ever.

THE BRAIN UNDER STRESS

When faced with stress, the body activates the hypothalamic-pituitaryadrenal (HPA) axis, a system that helps regulate the stress response. This triggers the release of cortisol, the body's main stress hormone. In short bursts, this response is essential for survival—for example, if you suddenly come face-to-face with a bear, your body goes into "fight-or-flight" mode. Your heart rate spikes, your breathing quickens, and your brain sharpens focus so you can either escape or defend yourself.

However, when stress becomes constant and long-term (chronic stress), this system stays activated for too long. Instead of helping, high cortisol levels can start to harm the brain, making it harder to think clearly, manage emotions, and handle new challenges (McEwen, 2006)

1. Hippocampus

The hippocampus, a brain region critical for learning and memory, is especially vulnerable to stress. Chronic exposure to elevated cortisol, the body's main stress hormone, can shrink the hippocampus, weaken connections between brain cells, and reduce the growth of new neurons, leading to memory deficits and a higher risk of depression (Heuser & Lammers, 2003; McEwen, 2007)



Brain models highlighting the hippocampal regions, with specific areas marked in color to show the distinct subfields, understanding how chronic stress impacts memory and neurogenesis.

Additionally, prolonged stress lowers levels of brain-derived neurotrophic factor, a protein essential for keeping brain cells healthy and promoting growth. This contributes to hippocampal atrophy, a process where the hippocampus physically shrinks over time (de Kloet, 2000). Studies show that when neurogenesis, the formation of new neurons, slows down, individuals become more vulnerable to cognitive decline and mood disorders (Kanatsou et al., 2019).

2. Prefrontal Cortex

The prefrontal cortex (PFC) governs executive functions such as decisionmaking, impulse control, and emotional regulation. Chronic stress weakens prefrontal synaptic connections, leading to deficits in cognitive flexibility, impaired working memory, and increased susceptibility to risk-taking behavior (Heuser & Lammers, 2003). Furthermore, stress-induced reductions in dopamine transmission within the PFC exacerbate cognitive dysfunction and emotional instability (Baik, 2020).



Green arrows in the unstressed brain indicate controlled thoughts, actions, and emotions, while red arrows in the stressed brain represent disrupted cognitive and emotional regulation.

3. Amygdala

The amygdala, a brain region responsible for processing fear and emotional stimuli, undergoes structural and functional changes under chronic stress. Heightened amygdala activity strengthens fear responses and emotional reactivity, increasing the risk of anxiety and mood disorders (McEwen, 2007). At the same time, stress reduces inhibitory control from the prefrontal cortex, amplifying negative emotional responses and making individuals more susceptible to stress-induced psychopathologies (Dirven et al., 2024).

Amygdala activation can be adaptive or maladaptive, depending on the situation. In a high-stakes moment, such as standing at the edge of a cliff before a jump, increased amygdala activity helps sharpen focus and heighten awareness, which is beneficial. However, if the same stress response is triggered every morning before school, it becomes maladaptive, contributing to chronic anxiety and emotional dysregulation. This shift from an appropriate response to an excessive, prolonged one is an indicator of stressinduced brain changes.

BUILDING RESILIENCE

While chronic stress can be harmful, the brain's ability to change, known as neuroplasticity, allows for both adaptation and recovery. However, plasticity can be adaptive or maladaptive: helping the brain build resilience or reinforcing negative stress patterns.

1. Physical Activity

Regular exercise increases the production of brain-derived neurotrophic factor (BDNF), a protein essential for neuronal survival and synaptic plasticity. Research shows that physical activity promotes neurogenesis in the hippocampus, counteracting the effects of chronic stress (McEwen, 2007).

2. Mindfulness and Meditation

Mindfulness practices enhance prefrontal cortex function, improving emotional regulation. Neuroimaging studies suggest that meditation increases gray matter density, which refers to the regions of the brain rich in neuron cell bodies that are crucial for processing information, attention, and self-awareness. Greater gray matter density in these areas strengthens cognitive function and helps combat stress-related decline (Dirven et al., 2024).

3. Social Supports

Spending time with friends, family, or a supportive community can help protect the brain from the effects of stress. Social interactions trigger the release of oxytocin, a hormone that promotes bonding and reduces stress. Strong social connections are linked to lower amygdala activity, meaning the brain reacts less intensely to stress, making individuals more resilient to challenges (de Kloet, 2000).

4. Adequate Sleep

Sleep is essential for both thinking clearly and managing emotions. When sleep is insufficient, stress has a stronger impact, impairing memory and increasing emotional reactivity. Research shows that deep sleep is crucial for clearing stress-related toxins from the brain and maintaining the function of the prefrontal cortex (PFC) the part of the brain responsible for decision-making and emotional control (McEwen, 2007).



Sleep disruptions leading to glymphatic system impairment and neurodegeneration

CONCLUSION

Chronic stress reshapes the brain by altering neural plasticity, disrupting neurotransmitter balance, and impairing cognitive and emotional functions. However, the brain's adaptive capacity allows for resilience through lifestyle modifications such as physical activity, mindfulness, strong social connections, and adequate sleep. Research highlights that early interventions and targeted strategies can significantly reduce stress-induced neurodegeneration.

In addition to behavioral approaches, emerging studies highlight the potential of pharmacological treatments and cognitive training programs in strengthening neural resilience. This is a rapidly growing field, with new advancements gaining attention in the media—such as the concept of neural resilience discussed in *Quarterback* on Netflix, where athletes like Kirk Cousins explore techniques to strengthen their brains against stress. A close examination of these techniques could offer valuable insights into how to build resilience in everyday life.

For teenagers, understanding the long-term impact of stress and taking steps now to manage it is critical. The capacity to tolerate biological changes brought on by stress is not unlimited while some may feel they can handle stress for a while, its impact can accumulate and affect health for years to come. Taking care of mental health, learning stress-management techniques, and prioritizing well-being can prevent lasting damage and promote a healthier future.

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Review Article The Negative Effects of Multitasking on our Brains

Rohma Kazmi

ABSTRACT: Multitasking is the ability to handle several tasks at once, and task switching is obtaining the ability to transfer all your focus from one task to the other in a matter of seconds. This is all possible due to the functions in our prefrontal cortex. Though the concept of multitasking may sound time efficient and productive, the effects it leaves on our brains are depleting and oftentimes highly disruptive. Previous research regarding this concern has shown that multitasking makes us more distractible and prone to errors. Additionally, increased blood pressure and high stress levels are oftentimes associated with the juggle of completing multiple tasks at once. Various methods have been implemented to this topic as a way to reveal the underlying and deteriorating outcomes of multitasking. Such as fMRI (Functional Magnetic Resonance Imaging), EEG (Electroencephalography), as well as general behavioural experiments. As a result, key findings include symptoms associated with depression and anxiety.

OVERVIEW

The negative effects of multitasking on our brains as well as being far damaging are quite unknown to people who perform this so-called "balancing of tasks" otherwise known as crosstasking. It is recorded from a study conducted by Mark Carrier, a certified professor of psychology, that nearly 70% of people from all around the world spend a minimum of one hour multitasking per day. This high percentage shows just how popular this detriment act is. This issue highly revolves around the anterior cingulate cortex. his area in our brain is responsible for functions such as

cognitive abilities, emotion regulation, as well as decision making, all being essential elements in the process of multitasking. Due to multitasking, these functions in the anterior cingulate cortex decline at a moderate rate.



In other words, our cognitive abilities such as conflict monitoring and general decision making are weakened. Along with these abilities, our emotional regulation falls, essentially putting our brains at risk for symptoms such as depression and anxiety as mentioned prior. The National Library of Medicine reveals that depression and anxiety is correlated highly with high blood pressure and high stress levels.

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Scientific Review **A Synopsis on Synesthesia**

Nikhil Bogam

ABSTRACT: Synesthesia is a neurological condition in which individuals report a mixing of senses, meaning they hear sounds, taste colors, or feel sights. There is not a lot known about this condition, and professionals have only theorized how it works and what may cause it. Some of the most popular and widely recognized models on how synesthesia works are the cross-activation model and the disinhibited feedback model. The cross-activation model proposes that individuals with synesthesia have extra connectivity between parts of the brain responsible for processing senses. The disinhibited feedback model proposes that information from more complex functions, like memory or language, influence how the brain processes stimuli from one's senses. There are also many proposed etiologies of synesthesia. There has been research supporting the idea that synesthesia to drug-use or behavioral training. A lot more research must be done about synesthesia in order to fully understand the condition. In this review, we will provide an overview of the different physiological models of synesthesia and potential contributing factors to the development of synesthesia.

INTRODUCTION

Synesthesia is a rare condition where there is a crossover between senses in individuals. For example, one might be able to hear colors or associate sounds with movement. Normally, when processing stimuli, your brain detects what the stimuli are using sensory processes, your neurological networks connect the sensory processes to your brain, and finally, your brain processes and interprets that information. However, in synesthetes, people with synesthesia, the brain processes this information through 2 different areas of the brain, resulting in a primary and secondary effect (Cleveland Clinic,

2023). The primary effect is an accurate understanding of sensory information, but the secondary effect makes the brain believe one of the senses is working when in reality, it is not. With the initial sensation being received by two pathways, the sensory information is processed and interpreted in two different ways. For example, in synesthetes that hear colors, they may accurately interpret the sounds they hear (primary effect), but their brain might believe they are also seeing things (secondary effect).

There are different forms of synesthesia, most commonly including

perceptions of sight, hearing, and touch. This includes auditory-tactile (feeling touch-based stimuli when hearing sounds), day-color (associated colors with days of the week), grapheme-color (associating writing with colors), hearing-motion (associating sounds with movement), mirror-touch (feeling touch-based stimuli when seeing others respond to those stimuli), and timespace synesthesia (visualizing things in a specific way) (Cleveland Clinic, 2023). The phenomenon of synesthesia is so elusive that there is not a concrete agreement between professionals on how synesthesia actually works or what causes it. Below are some of the most popular and recognized theories that scientists have come up with.



An example of grapheme-color synesthesia (Brang & Ramanchandran, 2011).

THE INNER WORKINGS OF SYNESTHESIA

1. Cross-activation and Hyperbinding Model

One theory that experts have come up with is the cross-activation and hyperbinding model. This model was created in order to explain grapheme-color synesthesia, or the ossication between writing and colors. This theory hypothesizes that the part of the brain responsible for processing graphemes, or the posterior temporal grapheme area (PTGA) is physically connected to the color-processing brain areas, the V4 and V8 areas (Science in the net, 2013).

In one study, two test subjects, both with grapheme-color synesthesia, were tested to see if they could recognize the color of a grapheme as the letter/number got further and further away from the participant's central vision. As the grapheme got more into their peripheral view, they stopped associating that grapheme with a color (Ramachandran & Hubbard, 2001). This suggests a cross-wiring between the V4 area and the PTGA as V4 emphasizes central vision processing. In addition, these areas are both in the fusiform gyrus, a section at the base of the brain, and are adjacent to each other. Moreover, in a study looking at synesthetes that "hear colors", researchers found that synesthetes had a higher concentration of white matter in their brain (Zamm, Schlaug, Eagleman, & Loui, 2013). White matter is the tissue in the brain that connects areas together; it allows different parts of the brain to communicate with each other. The researchers measured FA values, a measurement of connectivity in the brain, for both the left and right IFOF. The IFOF, or Inferior Fronto-Occipital Fascicle, is a portion of the brain composed of white matter. These researchers found that synesthetes had larger FA values in the right IFOF than the control group did.



An example of grapheme-color synesthesia (Brang & Ramanchandran, 2011).

2. Disinhibited Feedback Model

Another model created to explain how synesthesia works in the disinhibited feedback model. This model proposes that, in synesthetes, information from higher-level cortical areas of the brain "bleed" into the information from the senses that the brain is processing (Science in the net, 2013). Higher-level cortical areas are parts of the brain that deal with more complex functions, like memory, language, recognition, etc.

In an interesting case study, a patient who was completely blind at 40 reported seeing things when having tactile sensations at 42. When tested to see how much pressure was needed to elicit visions, researchers found that the amount of pressure needed was different depending on whether the hand they were touching was in front or behind the patient's head (Armel & Ramachandran, 1999). This suggests that the patient's synesthesia might have correlated with higher-level functions of spatial awareness and location/navigation. In addition, when researching a participant's synesthesia of tasting things after hearing words, researchers found that conceptual understanding of words superseded the actual sound of the word when eliciting

a taste (Ward & Simner, 2003). The participant, JIW, generally followed a pattern where a syllable or sound elicited a certain taste. For instance, the s and t sounds in the words past or coast made JIW taste toast. However, when presented with the word cabbage, JIW tasted cabbage, despite the "age" sound being associated with sausage in many other cases. This suggests that higher-level understanding of conceptual ideas influenced JIW's synesthesia, supporting this model of disinhibited feedback.

THE CAUSES OF SYNESTHESIA

1. Genetic

One contributing factor to synesthesia is genetics: some people have a predisposition to developing synesthesia because of their genes. In a study looking at the genetics of synesthesia through genome mapping, they found that the trait for synesthesia was correlated to certain chromosomes (Asher et al., 2009). In this study, they used nonparametric linkage analysis, or NPL analysis, to see if this trait was actually linked to other traits on a chromosome. Nonparametric means they tested their hypothesis without assuming the qualities of the trait of synesthesia, such as if it is dominant or recessive. They found there were significantly high LOD values in the 5th, 6th, and 17th chromosomes. LOD, or logarithm of the odds, is a measurement that looks at the probability that two loci are located close to each other, and therefore, are inherited together. Loci are points on the chromosomes where genes and their subsequent traits are

found. A high LOD means a high probability that the trait of synesthesia is actually inherited through genes. In the same study, the researchers also performed HLOD analysis, or heterogeneous logarithm of the odds. This HLOD analysis showed there was significant linkage on the 2nd chromosome, and suggestive linkage in the 6th, 9th, and 12th chromosome. It is clear that synesthesia in individuals is linked to their genes, though more research must be done on this topic to determine genetic influence.



Loci are parts of chromosomes where genes are found, and these genes code for traits like synesthesia (Genes and Gene Loci)

2. Acquired

Another possibility of the cause of synesthesia is that it could be acquired throughout one's life or in single experiences. For example, researchers looking at drug-induced synesthesia found that multiple drugs can elicit signs or symptoms of synesthesia (Luke, Lungu, Friday, & Terhune, 2022). They found that a control group that are not synesthetes most commonly experienced synesthesia when under the influence of LSD (57% of control group), ayahuasca (49% of control group), and psilocybin (45% of control group). In addition, the researchers found that sound-colour, sound-space, and sound-shape were most commonly experienced, and that the type of synesthesia wasn't necessarily linked to a certain drug.

In addition to drug-induced synesthesia, individuals can learn to become synesthetes. Researchers trying to "train" adults into associating colors with graphemes saw that after a 9 week period of training, participants did show signs of being genuine synesthetes (Bor, Rothen, Schwartzman, Clayton, & Seth, 2014). A test called the "Color Consistency Test" measures the participants' association between letters A-Z, numbers 0-9, and different colors. After training, these participants showed scores low enough (higher consistency) to classify as exhibiting genuine synesthesia. In addition, another test training participants to associate colors with 13 different graphemes resulted in participants having shorter reaction times for recognizing the color associated with the grapheme after training.

CONCLUSION

Synesthesia is a very perplexing condition where individuals experience a crossing of the senses, where one sense can influence another. Professionals have only some up with theories on how synesthesia actually functions in the brain, like the cross-activation model and the disinhibited feedback model. Moreover, the causes for synesthesia are also somewhat unknown, with some research claiming synesthesia is genetic while others prove that synesthesia can be acquired throughout one's lifetime.

Although synesthesia is a very interesting condition, very little is actually known about it. There is not a lot of research done about synesthesia in the grand scope of things and a lot of explanations for this phenomena are still theories and aren't concrete. The validity of theories are still up to debate and scientists and experts continue to learn more. There is clearly still a lot to be learned and understood about this condition.

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Scientific Review

The Impact and Pathophysiology of Repeated Mild Traumatic Brain Injuries (mTBI)

Lois Oppong

ABSTRACT: Traumatic Brain Injuries, such as concussions, have a profound impact on everyday life. These injuries can be experienced at any point in life, no matter one's age, and can be experienced more than once throughout a lifetime. In the media, including professional sports, injuries are often sensationalized, with pressure on athletes to get back to their sports as quickly as possible, without respect to what impact these injuries will have on their lives after they retire. Millions of people outside the athletic sphere also experience traumatic brain injuries each year. Many people are at risk of having long-term health problems, such as disabilities, and death from experiencing a TBI. TBIs can occur from a variety of injuries such as motor vehicle crashes, falls, and being hit by an object', and many people can be unaware of a possible TBI diagnosis if it goes unchecked. Traumatic Brain Injuries are rated on different scales, but the impact of concurring TBIs is relatively unknown. It is important to explore the different facets of traumatic brain injuries and their impacts, not only on people who experience them as a workplace commonality, but also for others who experience a traumatic brain injury without ever knowing.

INTRODUCTION

A traumatic brain injury is defined by the US Department of Affairs and the Department of Defense as a "traumatically induced structural injury and/or physiologic disruption of brain function as a result of an external force"². The TBI is also graded based on severity which is calculated using the extent of all of these systems to determine whether or not it is mild, moderate, or severe. This system is known as the Glasgow Coma Scale³. The Glasgow

Coma Scale evaluates the level of consciousness in a patient after a traumatic brain injury that assesses eye opening, verbal response, and motor response⁴. A head injury is classified as a Mild TBI when the GCS is from 13-15, moderate when the scale is from 9-12, and severe when the scale is from 3-8. The complete scale ranges from 3-15. Mild TBIs are common in sports medicine, physical abuse, and head banging, and it is often associated with a complete recovery after the fact.



In this study, there will be a focus on how recovery is affected after a repeated occurrence of a mild traumatic brain injury. The average person may experience one to two traumatic brain injuries during their lifetime, although many TBIs are often unreported, so that number could be far greater⁵. Athletes are most likely to experience traumatic brain injuries, especially within contact sports. Many sports related injuries are unreported as athletes are encouraged to play through the pain, making the incidence of traumatic brain injuries within athletics more difficult to collect accurate data on. This also increases the risk of later impacts of traumatic brain injuries, as many athletes are unaware of what could happen later on in life because they are never diagnosed. It is important to address the impacts of repeated mild traumatic brain injuries so that there is less stigma surrounding the report of injuries, leading to more TBI awareness, as impacts can transcend into the latter stages of life⁶.

PATHOPHYSIOLOGY OF MILD TBI 1. Concussions

Concussive injuries are often caused by acceleration and deceleration forces on the brain, causing the brain to elongate and deform. More simply, they are also known as brain bruises. These impacts on the brain can occur with or without a helmet, given that there is a significant,

rapid change in velocity within the head that could possibly result in a deformation of the skull⁷. These forces on the brain can also cause neurometabolic changes within the brain as there is a release of neurotransmitters, influx of calcium, and other changes that occur to maintain membrane homeostasis. These forces on the brain directly impact the axons, and the severity of axon damage determines the severity of the TBI. A mild TBI produces microscopic axonal damage, or the tearing of the brain's nerve fibers, which can contribute to how severe the symptoms are during recovery from this injury.

2. Impacts Across Age and Sex

In sports, a larger ratio of female players experience concussions related to male players of the same sport. This was observed in the study of male and female soccer players⁸, one of the only sports where all aspects of play are very similar. This heightened risk in females may be explained by the lack of neck musculature and the larger ball to head size ratios in females relative to males, but the influence of cervical muscle strength on head acceleration is not as clear. On an age spectrum, there have been proposed sources of increased susceptibility to concussions in youth relative to adults, but there is a lack of data to support these claims as there have not been many studies across the spectrum of youth and in sports other than high school and collegiate football.

SYMPTOMS AND RECOVERY OF mTBIS 1. Symptoms

Common symptoms of a mild TBI or a

concussion are headaches, nausea, vomiting, dizziness, double or blurry vision, and light and noise sensitivity. People often have glassy eyes, are confused and forgetful, slowly answer questions, lose consciousness, and have a mood, or behavioral change. These symptoms and signs are often observed right after the concussion occurs, and may be difficult to see because of the underreporting of symptoms in an effort to get back to an activity. Although the symptoms of a concussion might seem unserious, they can lead to many health issues in the future.

3. Recovery

A traumatic brain injury leads to changes within the connectivity of the brain and communication between synapses. The transportation of synaptic vessels may be disrupted, and synaptic changes could show instability and degeneration which could impact recovery. When tested in adult rats, exercise immediately after a TBI decreases synaptic plasticity molecules, or the molecules that are responsible for mediating change at the synapses, while making spatial learning and memory tasks worse. This was contrasted by improvement when exercise was facilitated two weeks after a mTBI⁶. If a mTBI goes undiagnosed and someone tries to exercise immediately after, there could be long term impacts to memory and learning capabilities.

The duration of recovery may increase because of the impact that undetected hormonal changes may have on memory, attention, mood, fatigue, and sleep. There is a window of vulnerability where the brain is vulnerable to another injury, and that is the time when athletes should stay out of play. There are no biological markers for this window of vulnerability, so it is difficult to determine when each individual is ready to return back to play even though there are guidelines in play for different levels of sport. This length of time that the brain is vulnerable has been mainly observed to be the length of time that it takes for the brain to fully recover from a concussion, or 30 days. Within the study, three patients who resumed normal activities directly following the 30 days of initial recovery experienced a second concussion, leading to a prolonged recovery of an additional 45 days. A history of prior concussions is a predictor for an increased risk of future concussions, as people who have previously had a concussion are 2 to 5.8 times more likely to sustain another concussion.

4. Impact of Repetitive mTBIS

Predominantly in young athletes, mild TBIs may result in death through a condition called second impact syndrome, or SIS. SIS happens when an athlete experiences a concussion or another mild head injury, and then has another one before the recovery from the first injury has been completed. This occurrence does not usually include an immediate loss of consciousness when contact has been made, instead the brain deteriorates within minutes of contact as a result of vascular engorgement, or when the blood vessels in the brain swell and become filled excessively with blood, increasing

pressure within the brain. SIS occurs most predominantly in football players from age 10 to 24, but mostly in the high school level. Retired professional football players who had experienced three or more concussions also were subject to more cognitive issues including a three times more likelihood to have significant memory impairment compared to retired players who had never experienced a concussion. Brain trauma has been linked to changes in mood, especially when linked to sports. There is a direct link between repetitive concussions and depressive symptoms, according to another survey done on retired pro-football players. This was also shown in imaging, with many of these players having white matter abnormalities within the brain on structural imaging and increased fractional anisotropy, meaning that water diffusion is restricted in one direction within brain tissue, on diffusion tensor imaging. In sports, repetitive mTBI also directly relates to an increased risk of developing chronic traumatic encephalopathy (CTE), ALS, dementia, and Alzheimer's disease. ALS, or amyotrophic lateral sclerosis, is likely to lead to death in many professional groups, such as professional soccer players in Italy, where the ALS mortality rate is 12 times the average of the regular population.

5. Occurrence of Chronic Traumatic Encephalopathy after Repetitive mTBIs

Chronic Traumatic Encephalopathy or CTE is a disease that is found most commonly in people who participate in activities that have a high risk of repetitive mild TBI or concussions. CTE

can only be diagnosed after death using a neuropathologic examination, and there are no criteria or biomarkers that can be used to diagnose CTE prior to death. Some characteristics of CTE include a reduction of brain weight, enlargement of the lateral and third ventricles, scarring of the cerebellar tonsils, and many others.. CTE is associated with the onset of other neurodegenerative diseases such as motor neuron disease, and more specifically ALS. CTE results in behavioral and cognitive deficits as well, characterized clinically by the progressive decline of memory, executive functioning, mood, and behavioral disturbances that lead to dementia over time. There are links from CTE to depression, impulsivity, anger, irritability, a lack of emotion, suicidal behavior, and aggressiveness. These symptoms are often inherited mid-life, and symptoms progress as time passes and the disease progresses. The clinical progression of CTE begins asymptomatic, but there are symptoms that may be present. In early stages, people may experience headaches, loss of attention and concentration, depression, and irritability, but the non specific nature of these systems make it difficult to characterize them as earlystage CTE. In stage II of CTE, there may be short-term memory difficulties, aggressive tendencies, mood swings, difficulties with planning and organization, explosivity, and suicidal tendencies. The advanced stages of CTE are associated with cognitive impairment, memory loss, profound loss of attention and concentration,

language difficulties, paranoia, gait, and aggression. In recent studies it may be suggested that CTE might have two different representations:one with a younger age at onset, with more behavioral disturbances and mood changes, and another with late life cognitive impairment.

CONCLUSION

Mild TBIs are very important in relation to recovery and allowing the brain time to recover before activity continues. If there is a lack of care during the recovery period where the brain is most vulnerable, people become more susceptible to long-term diseases such as CTE, which can only be diagnosed after death. CTE has a multitude of long term impacts, such as high suicidality rates and connections to many neurodegenerative diseases. It is important to consider concussions and mTBIs as serious diseases because of their connections to CTE. Full recovery instead of rushing back to play or another activity must be a serious commitment, helping to lower the risk of a consecutive concussion, and aiding brain health in the long term. The disregard of mTBIs needs to be changed, and there needs to be an abundance of caution when treating mild traumatic brain injuries, just like with any other major injury, especially in sports. There needs to be more regulation and rules related to return to play from youth to professional sports, allowing players to feel safe while playing their sport, and protecting future generations so that CTE isn't as recurrent as it is now.

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Did You Know?

Your brain uses about 20% of your body's energy.

Even though it only makes up about 2% of your body weight, your brain consumes a massive chunk of your energy, constantly powering thoughts, memories, and actions.



24

The brain is more active when you're asleep than when you're awake.

During sleep, especially in deep stages, your brain is busy sorting memories, cleaning out toxins, and forming connections to help you learn and grow.

Humans can process visual information in just 13 milliseconds.

That's faster than the blink of an eye! Our brains are built for lightning-fast responses, especially when it comes to seeing and interpreting the world around us.



As we close out this issue of Neuro Frontiers, we want to take a moment to express our gratitude to everyone who has been part of this journey. From the amazing writers to the dedicated readers and all of you who continue to support us—this issue, and this journal as a whole, wouldn't be the same without you.

Watching Neuro Frontiers grow and evolve has been an incredible experience. What started as a simple idea has blossomed into a platform for high school students to engage with neuroscience, contribute their voices, and learn from one another. And this is just the beginning.

We're so proud of the work we've done so far, but the possibilities are endless. With each issue, we're not just publishing articles, we're building a community that thrives on curiosity, collaboration, and a shared love of learning. So, whether you've contributed, read, or simply followed along, thank you for being a part of something truly special.

We can't wait to see where this journey takes us next. The future is bright, and we're thrilled to have you with us every step of the way.

-Neuro Frontiers Team

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