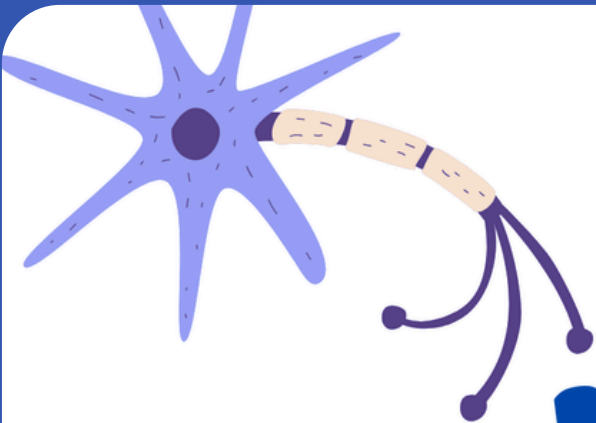




GREY MATTERS



SPRING 2026



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EPILEPSY AND THE LIMITS OF MODERN TREATMENTS

Author: Pooja Karan

Artist: MJ Soares

Of all the neurological disorders, epilepsy is one of the most recognizable — yet, despite decades of research and its commonality, it remains a disorder with treatments that can control symptoms, but no cure. Treatments most commonly consist of anti-seizure medications, but also involve a ketogenic diet or surgery if there is only a specific brain region triggering the seizures. However, part of the reason why it still has no universal cure is that epilepsy is a heterogeneous group of disorders rather than one single disease, and certain forms of epilepsy cannot be controlled by modern treatments. Moreover, although the basic mechanisms of epilepsy are understood, the various manifestations make it difficult for scientists to understand the underlying molecular mechanisms behind this disorder.

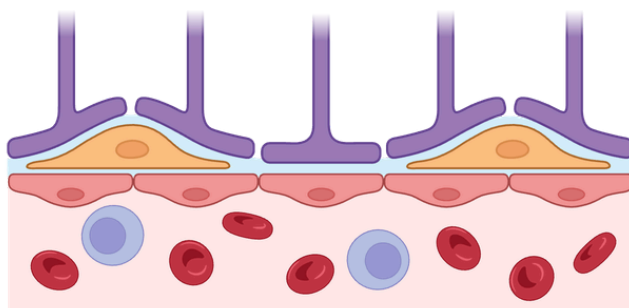
Epilepsy is, at its core, a chronic disorder of abnormal brain signaling. This can trigger an epileptic seizure, which is different from those that may occur in people without epilepsy. Individuals with this disorder generally have a lower seizure threshold and are therefore more susceptible to seizures than those without. Non-epileptic seizures are typically due to temporary trauma, another disease, or substance abuse. During an epileptic seizure, groups of neurons fire rapidly and simultaneously: this rapid burst of electrical activity can involve shaking, falling, muscle

stiffening, lapses of consciousness, involuntary movements, and other symptoms. One difficulty with treating epilepsy stems from the fact that epilepsy exists along a broad spectrum — there are many different types of seizures (such as tonic-clonic seizures, which involve stiffening and jerking phases, or absence seizures, which induce lapses in consciousness) and subtypes of epilepsy (such as generalized epilepsy, where misfiring occurs across the entire brain, or focal epilepsy, which only affects one hemisphere).

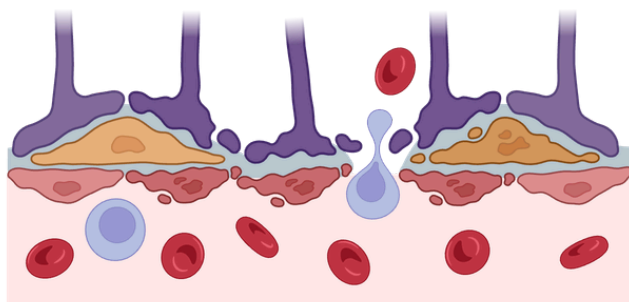
Thus, many factors can trigger an epileptic seizure. A seizure can occur when there is a decrease in inhibitory signaling (a type of signal that decreases neuron firing) or when there is an increase in excitatory signaling (which increases neuron firing), as both of these signaling patterns can rapidly increase electrical activity in the brain. Another mechanism that can result in abnormal brain activity and seizures includes mutations in the ion channels that can alter neuronal excitability and disrupt electrical signaling. Alongside these factors, inflammatory cells can release molecules that impact neuronal signaling, while the presence of IgG antibodies in the brain may also trigger seizures. In addition, several converging genetic and environmental factors are currently not fully understood, but correlate

with epilepsy.

Though there is basic knowledge on the causes of epilepsy, there are still major gaps in the understanding of this disorder. The exact cause of epilepsy and how chemical and structural brain variations can trigger seizures is still not understood in many patients, these cases are classified as idiopathic. Other unclear factors are: the manner in which seizures spread in the brain; why some seizures stay localized while others generalize; long-term brain changes from repeated seizures; the existence of any potential, reliable, and widely usable biomarkers to predict seizure occurrence; and the role of non-neuronal cells such as glial cells and immune cells. Furthermore, some people with epilepsy remain resistant to treatment even after trying two or more anti-seizure medications. In fact, approximately 30% of individuals with epilepsy are drug-resistant epileptics. The lack of understanding around epilepsy and its resistance to medication only further increases the risks associated with this disorder.

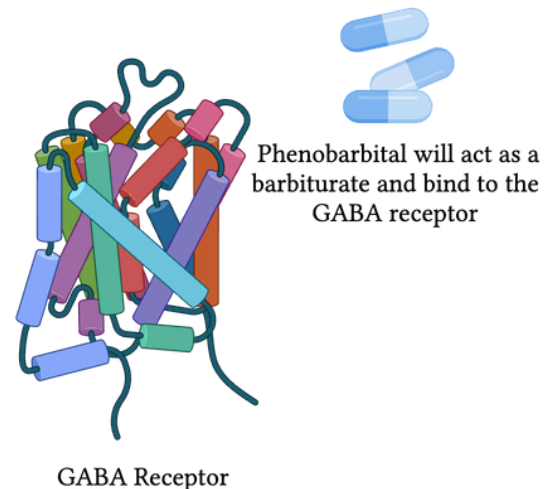


Healthy blood-brain barrier



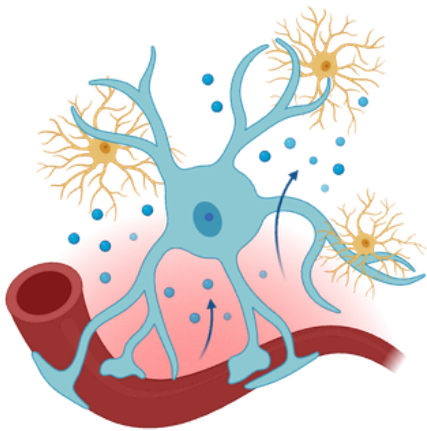
Drug-resistance epilepsy blood-brain barrier

Since the underlying causes of epilepsy are varied and still not fully understood, current treatments focus on controlling seizures rather than curing the disorder. Although there are a multitude of treatments, these include medication, following a ketogenic diet, and/or surgery. However, the focus of this article will be on medication as a treatment and drug-resistant epilepsy.



Anti-epileptic drugs (AEDs) work in a variety of ways: medications like Phenytoin, Oxcarbazepine, Lacosamide, and Carbamazepine act on voltage-gated sodium channels in neurons to block or slow repetitive activation; Phenobarbital acts as a barbiturate and binds to the receptor for the neurotransmitter GABA, allowing neuronal cells to hyperpolarize; Levetiracetam binds to the synaptic vesicle protein 2A; additionally, other anti-epileptic drugs combine several of these modes of action. AEDs can be broad-spectrum, treating a wide variety of seizures, or narrow-spectrum, specifically treating focal or partial seizures. Nevertheless, both have potential side effects, such as headaches, fatigue, dizziness, blurry vision, and more. Although these medications offer a solution to seizures for many epileptics, 30-40% of epileptics still experience drug resistance.

Though the reasoning behind drug-resistance in epileptics remains unknown, there are proposed theories, including the transporter hypothesis, the drug-target hypothesis, the rewiring of neural networks, and inflammatory responses. The transporter hypothesis suggests that drug-resistant epilepsy is caused by the overexpression of efflux transporters at the blood-brain barrier and within epileptic foci of the brain. The overexpression of these transporters reduces the concentration of AEDs in brain tissue, thereby making the medication less effective. The drug-target hypothesis suggests that seizures can induce molecular changes that alter the function of anti-seizure drug targets. Two other factors that may explain drug-resistance in epileptics explore how chronic seizures may rewire neural networks so that even if AEDs suppress individual neurons, the overall network can still generate seizures. Additionally, increased inflammatory signaling in the brain can increase excitatory signals between neurons while also reducing drug responsiveness.



Model of BBB leakage and inflammation

Despite these challenges, the limitations of current treatments for epilepsy may not be as fixed as previously thought. Recent research in animal models shows that targeting particular ion channels, such as TRPC3, can reduce the frequency and severity of seizures. These results suggest a more targeted, mechanism-based therapeutic strategy that focuses on the particular pathways causing seizures. This differs from previous research, which has generally focused on suppressing neural activity and treating all epileptic seizures as having the same mechanisms. Additionally, these treatments mark a significant shift toward more focused and potentially successful interventions, though they are still developing and require more validation in human trials.

New clinical data challenged long-held beliefs regarding drug-resistant epilepsy, suggesting that experimenting with various medications over time occasionally results in a significant decrease in seizure activity. Studies have recently shown that alternating three or more medications over a duration of several months to years decreased the amount of seizures experienced by some drug-resistant epileptics. These findings raise the possibility that the brain's potential for neuroplasticity enables it to gradually react to therapy in novel ways. When considered collectively, these advancements point to an increasingly complex understanding of epilepsy as a dynamic disorder that can be responsive to changing treatments rather than as a static condition. These results demonstrate that epilepsy is not an incurable illness; rather, more potent and long-lasting treatments are achievable with further research and tailored methods.



BORDOM: FRIEND OR FOE?

Author: Krista Proteasa

Artist: Jasmine Smith

You're bored. You turn to your phone, laptop, or the insects crawling on the windowsill to relinquish you from this state of nothingness. However, should you really be squashing boredom at every opportunity? Truth be told, the answer is multifaceted and not as black and white as you would like it to be. To understand this, it is worth examining both the benefits and drawbacks of boredom. Boredom is associated with positive effects such as increased creativity, understanding of the concept of time, and the cerebrospinal

fluid (CSF) cleansing of the brain. On the other hand, boredom is also linked to unhappiness, emotional eating, and feelings of meaninglessness. In short, boredom is complicated, as it is both a biologically necessary process and an interesting catalyst for different social behaviors.

Boredom was once thought to be insignificant to study or address. However, in recent years, it has been found that boredom may play an

adaptive role in cognitive and behavioral regulation alongside its ability to improve one's integration in society and subsequent thriving nature¹. Boredom is defined as the mental state of being without internal or external stimulation². Several models exist for explaining the purpose of boredom through means of attention, homeostasis, and cognitive resources. The Meaning and Attentional Components (MAC) model posits that boredom is a result of the discrepancy between one's actions and goals (meaning), and between cognitive demands and available attention³. It proposes that boredom serves as a deliberate launching pad from which people derive new goals and desires⁴. For example, if one feels frequently bored at their current job, they might daydream of other jobs that would be more interesting to them. This would cause them to pivot directions and explore their other interests more.

The cognitive homeostasis model proposes that boredom is evidence of engagement falling outside of the desired range, driving us to seek regulation of our engagement to reestablish the homeostatic range, with chronically bored people having unrealistically high standards of the excitement of their activities⁵.

The Boredom Feedback Model (BFM) states that boredom is a signal that one is not fully utilizing one's cognitive resources, thus causing one to seek further engagement externally. One pursues new tasks either internally through mind-wandering and creative endeavors such as different projects or hobbies, or externally by returning focus towards the original task⁶. This optimal range of engagement is said to be the state in which one is neither under nor overstimulated. Rather, they have the optimal level of stress and engagement to keep them

active and interested in their activities without having them become overwhelmed. This functionality shows how boredom is evolutionarily advantageous. It is a means to push us out of dull and repetitive situations, bolstering our creativity and propelling us in life through exploring different professional, social, or personal goals⁴. With all of these models, it is difficult to arrive at a definitive conclusion on whether boredom is positive or negative.

Negatively speaking, boredom is responsible for various uncomfortable situations. It has been shown to increase the likelihood of sadistic or harmful behaviors under certain experimental conditions. Sadistic tendencies are more pronounced in those with a predisposition for boredom in their daily lives. However, it was demonstrated that artificially increasing boredom without alternatives also increased participants' sadistic tendencies, such as killing worms and destroying other peoples' pay. When alternatives were presented, such as a more interesting video to watch, boredom increased sadistic tendencies more so in those with naturally high sadism⁷.

In the experiment regarding killing worms, participants were asked to watch a 20-minute video of either a waterfall (the boredom paradigm) or a documentary about the Swiss Alps (the control condition) with the opportunity to place a cup containing a worm in a modified coffee grinder; there was a barrier on the blender that would not place the worm in contact with the blades, but participants were not aware of this at the time of the experiment. The overwhelming majority of participants did not elect to grind the worms. However, when boredom was induced through having participants watch the waterfall video, there was a significant increase in the number of participants who placed the worms in the grinder. Similarly to this experiment,

participants in a separate online study were randomly assigned to watch a 5-minute video of stone (boredom condition) or a 5-minute video of two magicians doing two magic tricks (control condition). They were told that they were paired with a real person receiving 50 cents. During the video, they were given periodic choices to either increase or decrease the person's pay by 10-cent increments, a version of the "joy of destruction" game. Between those who watched the stone video and those who watched the video of the magicians, there was a significant increase in the number of participants who elected to destroy the other person's pay, showing that boredom not only correlates with an increase in sadistic behavior, but actively causes it⁷.

Boredom is further thought to increase emotional eating in those with poor interoception (the ability to recognize internal cues)⁸. Additionally, an increase in one's tendency towards boredom has correlated with a decrease in their undergraduate GPA⁹. Moreover, proneness to boredom is linked with an increase in problematic phone use, defined as the inability to control one's phone use and one's phone use subsequently leading to negative impacts on their mental and physical health, causing increases in one's anxiety and depression symptoms¹⁰.

Boredom also makes people think about things that aren't happening with nearly the same frequency as they think about things that are happening, i.e. mind wandering. The researchers used an iPhone app to ask a large number of people what they were doing at random points throughout the day, whether they were thinking of something other than what they were doing, and whether that thing was pleasant, neutral, or unpleasant. They found that people thought of something other than what they were doing (let their mind

wander) during every activity except for when they were making love. These participants were also no happier thinking about other pleasant things than they were thinking about their current task. Ultimately, this pattern of thinking makes people unhappy as it makes them no happier to let their mind wander than it does to think about their current state and activities¹¹. While these are all negative effects, they are largely due to a lack of alternative activities during lapses of boredom. Alternative activities can deter one from resorting to problematic behaviors for quenching boredom, such as excessive phone use and even sadism.

Positively speaking, there are many effects of boredom. Firstly, boredom may be the brain's attempt at recovering lost sleep and its associated byproduct clear-out. It's been found that at the onset of boredom, there is an influx of cerebrospinal fluid (CSF). CSF is produced to cleanse excess waste products from the brain following a period of sleep deprivation¹². Furthermore, boredom is hypothesized to contribute to one's discovery of the concept of time. In life, people are forced to ponder the reasons why they are not receiving the influx of information they may have expected at any given moment, i.e., why they are bored. This leads the mind to ascribe such reasons to the "container" of experiences: time. Thus, one goes from being unaware of time to gaining an awareness of time and its passage, causing further uses and/or abuses of time at one's own discretion¹³. Similarly, boredom and its counterpart, effort, influence how we perceive time by how boring or engaging a task is. It manages this through the insular cortex, the brain region responsible for the awareness of one's internal states, homeostasis, and emotion. This balance of effort and boredom emerges through familiarity with tasks: new, higher-effort tasks seemingly slow time while easy, monotonous tasks seemingly speed up time¹⁴.

Furthermore, boredom is associated with a rise in creativity. When prompted to think meaningfully, participants were either completely unable to, or unable to do so pleasurably¹⁵. The study in question asked participants to think for pleasure and for meaning, the methods for which will be described in that order. For the first portion, the researchers either provided no guidance on what to think about or examples of meaningful things to think about, such as one's first kiss, academic or personal accomplishments, prior achievements, or family events, which have been shown to be meaningful to those who think about them¹⁶. The participants who did not receive examples showed a nominal (but not statistically significant) increase in the extent of meaningfulness they experienced, but those who received examples did find the task of thinking to be both more enjoyable and more meaningful. In the second portion, the participants were asked to specifically think for meaning. This resulted in a decrease in their enjoyment, as it is posited that more meaningful thoughts may be related to more negative events, such as the memorial service for a loved one¹⁵. Across the two studies, we can see that people do enjoy thinking about positive events, and feel even more enjoyment when given prompts of such positive thoughts, although the inverse correlation in enjoyment is found when they were unprompted to think meaningful thoughts. This implies that with exposure to and training in enduring boredom, people can successfully learn how to use their creativity and imagination to form pleasing thoughts.

Through these studies, it has been shown that boredom is a double-edged sword. While boredom causes uncomfortable feelings and a lack of stimulation, it isn't necessarily a bad thing. Boredom can contribute to the understanding and realization of new

concepts, such as time, and can stimulate creativity and imagination. Boredom may also be the brain's defense mechanism for clearing itself of waste during the day if one is sleep-deprived and could not do so during the night. In addition, boredom may also be the catalyst for pleasing thoughts when one is prompted and permitted to think positively. Boredom is a foe if we make it out to be one, but a friend if we accept it for what it is.

FROM VIBRATION TO SENSATION: THE NEUROSCIENCE OF SEXUAL STIMULATION

Author: Mindy Kim

Artist: MJ Soares

After having a phase of “living inside my brain”—getting lost in endless thoughts, memories, “what ifs,” and mental simulations during COVID, stuck in my room for weeks with no physical interaction, I learned how important it is for humans to have environments that provide adequate sensory experiences. After the lockdown, I learned that a good way to stop unregulated thoughts is to consciously focus on external sensory stimuli: the breeze blowing through my hair, the softness of hands, the temperature of water as I soak my feet in a creek. I find senses fascinating—that the same stimulus can be felt differently by different beings, and that every sensory experience starts from receptors at the very end of a nerve.

For the entire semester of the Behavioral and Integrative Neuroscience (BINS for short) class, Professor Anders Nelson jokingly mentioned that the sensory system he was teaching that day was his favorite of the entire course—I found that funny. What is your favorite sensory system, dear readers? Mine is the somatosensory system, which detects and processes bodily sensations including touch, pain, temperature, pressure, and proprioception (position sense). I love it because somatosensory receptors are

distributed all over our body, inside and outside. Out of all the different sensations the somatosensory system can detect, one thing that has captured my attention recently is how specific types of touch, particularly vibration, can trigger dramatically different responses depending on exactly where on the body the stimulus lands. Each type of receptor enables us to sense a different parameter of touch, and they are located in precisely the place where they need to be. For instance, you cannot orgasm from the vibration of an electric toothbrush on your tongue, but you can from a vibrator stimulating your genitals. Why? Why does stimulating certain areas of skin in a certain way, for a certain duration, provoke such strikingly different responses compared to other parts of the body? This question was not covered in the lecture, and I suspect it won't be covered in any other lecture either. So I decided to find answers in the newest research on genital sensory mechanoreceptors and share them with readers of the Grey Matters Journal. This article will first go over how somatosensory inputs are processed, then apply that knowledge to the receptors, nerves, and neural circuits related to the senses of the genital area.

ON THE SOMATOSENSORY MECHANORECEPTOR-FOCUSING ON KRAUSE'S CORPUSCLE

Somatosensory end organs are specialized for the functions of the body region or skin type in which they reside. For example, Meissner corpuscles in fingertip skin underlie light touch and support fine motor dexterity, while structures associated with hair follicles respond to hair deflection (Qi et al., 2024). Despite a great deal being known about touch sensors, the physiological properties and functions of sensory structures in the mammalian genitalia had remained completely unclear—until recently.



Krause corpuscle

The genitalia contain specialized sensory structures called Krause corpuscles, first described by anatomist Wilhelm Krause in the 1850s. He noticed they came in two forms: some coiled like a ball of yarn (complex shape), and others small and cylindrical (simple shape). Despite these detailed anatomical descriptions, nobody ever figured out what they actually did or how they functioned. However, a 2024 paper published in *Nature* by Qi et al. set out to finally answer that question, using mice as a model because they are genetically manipulable and share similar structures with humans.

The first thing Qi et al. (2024) established was where exactly these corpuscles are and how many exist. They took 200-micrometer-

thick slices of genital tissue and stained them with two chemical markers: NF200, which lights up large nerve fibers, and S100, which lights up the Schwann cells that wrap around sensory nerve endings to form each corpuscle. This allowed them to see and count every Krause corpuscle precisely under a microscope. What they found was striking. In female genitalia, corpuscles were densely distributed throughout the clitoris but entirely absent from vaginal tissue. In male genitalia, they were found throughout the glans penis and internal prepuce. Despite the size difference between the two organs, the total number of corpuscles in the clitoris and penis was comparable, which means the much smaller clitoris ends up with a 15-fold higher density than the penis. To put that in perspective, the clitoris has three times more Krause corpuscles per unit volume than your fingertips have Meissner corpuscles (Qi et al., 2024). The clitoris is more densely packed with touch sensors than your fingertips. In terms of shape, 93% of clitoral corpuscles were the complex multi-coiled variety, compared to 70% in the penis. In the penis, corpuscles were concentrated in the erectile tissue and the distal tip, and in erect tissue they sat directly adjacent to the blood-filled cavernous spaces, suggesting they may also respond to pressure changes during erection itself (Qi et al., 2024).

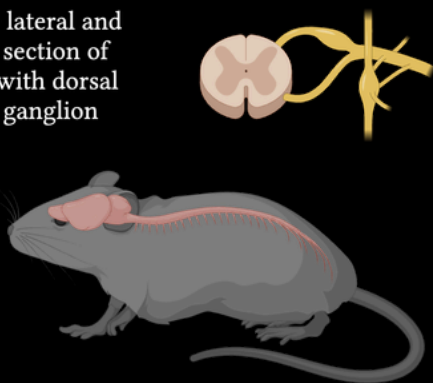


Meissner's corpuscle

ABOUT NEURONAL CONNECTION

Knowing where the corpuscles are is one thing, but to study what they do, the researchers needed to identify which specific neurons supply them, which is not an easy task, since genital tissue contains many different nerve fiber types all mixed together. This is where genetic tools became essential. The researchers used the Cre-lox system, a technique where a gene called Cre is linked to a specific molecular marker, and administering the drug tamoxifen permanently labels only the cells expressing that marker with a fluorescent color, like flipping a light switch exclusively inside those cells. Testing two markers, they found that **TrkB+** neurons (labeled by giving tamoxifen to mouse pups five days after birth) terminated in over 90% of all Krause corpuscles in both organs, and nowhere else in genital tissue. **Ret+** neurons (labeled during fetal development) innervated approximately 70-80% of corpuscles. complex corpuscles receive input from **both TrkB+ and Ret+** neurons simultaneously, while simple corpuscles receive input **only from TrkB+** neurons. The team further showed that TrkB signaling is not just a label but a requirement, which means mice engineered to completely lack it had almost no Krause corpuscles in either organ (Qi et al., 2024). No TrkB, no corpuscles.

Mouse lateral and cross section of spine with dorsal root ganglion



With access to these specific neurons established, the central question could finally be asked: what do Krause corpuscles actually sense? Are they touch sensors, vibration sensors, heat sensors, pain sensors? To find out, the researchers developed an experimental setup to apply precise mechanical and thermal stimuli directly to the external genitalia of living, anesthetized mice while recording individual neuron activity in real time. They surgically exposed the L6 dorsal root ganglion, the cluster of sensory neurons that receives signals from genital tissue and lowered a multielectrode array (MEA) onto it. An MEA is a tiny grid of electrodes that picks up the electrical spikes, called action potentials, that neurons fire when activated. But many different neuron types from many body regions are packed into the same ganglion.

So how do you know **which signal belongs to which neuron?** The researchers solved this using optogenetics: TrkB+ neurons were additionally engineered to express a light-sensitive protein called an opsin. Shining light onto the penis activated *only* TrkB+ neurons, and any electrode that responded to that light pulse was confirmed as recording a TrkB+ Krause corpuscle neuron, which is a process called **optotagging**. With the right neurons identified, the researchers vibrated the genitalia at different frequencies and measured firing rates. The answer was unambiguous. Both TrkB+ and Ret+ neurons responded most strongly to vibrations between 40 and 80 Hz, dropping off at lower and higher frequencies (Qi et al., 2024). They **confirmed this with a second independent method**, which was calcium imaging, where neurons engineered to express a fluorescent calcium indicator (GCaMP6) glow brighter when they fire. Watching neurons glow at different vibration frequencies produced the same peak: 40-80 Hz. Two completely different methods, one result.

ON VIBRATION

So why 40–80 Hz specifically? What is happening inside the corpuscle when it vibrates? The answer operates at two levels: 1) the **mechanical structure** of the capsule, 2) and the **molecular machinery** inside it. Each Krause corpuscle is not a bare nerve ending sitting in open tissue. The axon terminal is wrapped concentrically in layers of cells called lamellar cells, forming a capsule made of collagen, water, and cell membranes. These materials give the capsule viscoelastic properties, meaning it behaves like both a fluid and an elastic solid depending on how fast force is applied. Under slow, sustained pressure, the capsule layers gradually flow and redistribute internally (a process called stress relaxation), so the inner axon terminal stops being stretched even though the pressure is still present. The neuron fires briefly at the start and goes silent. Under **rapid vibration**, each push-and-release cycle happens too fast for that relaxation to occur, so the capsule snaps back and forth elastically, faithfully transmitting each deformation inward. The result is a mechanical filter: sustained pressure gets absorbed; vibrations in the right frequency window get passed through. The 40–80 Hz range is where this transmission is most efficient.

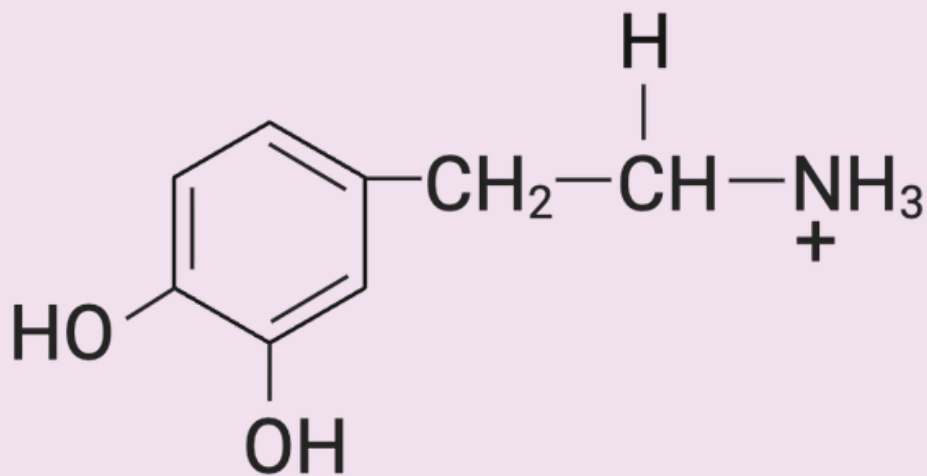
At the molecular level, the protein that **converts each membrane deformation into an electrical signal** is **Piezo2**, which is an ion channel that opens when the cell membrane is physically stretched. When Piezo2 opens, sodium and calcium ions rush in, depolarizing the membrane and triggering an action potential that travels to the spinal cord. Critically, Qi et al. (2024) found that Piezo2 is concentrated **only** inside the corpuscle on the **axon terminal**, not along the rest of the axon fiber. The neuron is mechanosensitive only at this one precise

location, which is why the receptor has such a specific, well-defined receptive field.

This also explains **why** clitoral TrkB+ neurons are more sensitive than penile ones, meaning they respond to lower forces, a difference the researchers measured by applying stimuli of increasing force in small steps and recording exactly when each neuron first fired. The clitoris has 93% complex, multi-coiled corpuscles, which pack far more axon membrane surface area inside the capsule than simpler ones. More surface area means more Piezo2 channels available to open per deformation, generating a larger initial electrical signal that reaches firing threshold at a smaller applied force (Qi et al., 2024). The clitoris is more sensitive at every level: denser corpuscles, more complex architecture per corpuscle, and lower individual neuron thresholds, all compounding on each other.

CONCLUSION

Taken together, these findings reveal that Krause corpuscles are vibration detectors, and that the clitoris is arguably one of the most densely sensory-innervated structures in the body, more so than the fingertips. Qi et al. (2024) also noted that the vibrations naturally produced by skin-on-skin contact during intercourse fall in the same 40–80 Hz range, suggesting the sensors are tuned precisely to the physical input they evolved to detect. That the clitoris and penis begin development from the same embryonic tissue with the same number of sensors installed and then diverge in size while the sensor count stays fixed, may explain why the density difference exists at all. The penis grows larger; the sensors stay concentrated in the small clitoris. What began as Krause's anatomical curiosity in the 1850s turns out to be the answer to why a vibrator works where it does, and why an electric toothbrush does not.



Dopamine structure

REWIRED: ADDICTION AND HOW IT REWIRES THE BRAIN

Author: MJ Soares

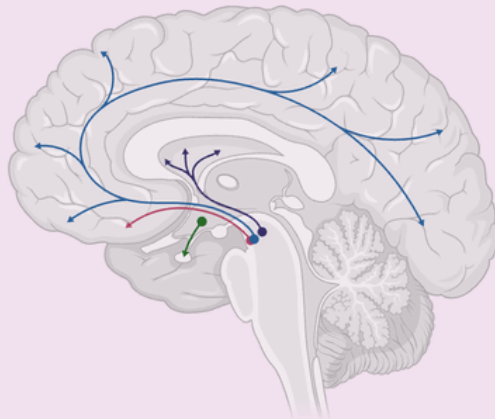
Artist: MJ Soares

What is addiction? What do we consider a state of addiction? Of course, the first things that come to mind are hard substances such as drugs and alcohol. But activities like gambling are also often classified as addictive acts. You could even get addicted to adrenaline! As a neurological condition, addiction is defined as “a chronic and relapsing disorder marked by specific neuroadaptations predisposing an individual to pursue substances irrespective of potential consequences,” (*Neurobiology of Addiction*, Semaan, Khan). This definition emphasizes that addiction is a chronic, relapsing condition

that is shaped by specific neuroadaptations. This paper navigates the way addiction rewires the brain by exploring how different drug dependencies change the brain's reward system, how these habits form, and the role neuroplasticity plays in all of this.

Most addictions target the dopaminergic (DA) reward system. It is first important to understand that most goal-directed motivations are learned. According to studies, “it is primarily through its role in the selective reinforcement of associations between rewards and otherwise neutral stimuli that DA

is important for such motivation,” (*Dopaminergic reward system: a short integrative review*, Oscar Arias-Carrion). Key parts of the dopaminergic reward system include specific pathways that connect to the ventral tegmental area (VTA), nucleus accumbens, and prefrontal cortex.



Dopamine pathways

How do natural amounts of the neurotransmitter contribute to pleasurable feelings? According to the Cleveland Clinic, “Dopamine is part of your reward system..designed, from an evolutionary standpoint, to reward you when you’re doing the things you need to do to survive. As humans, our brains are hard-wired to seek out behaviors that release dopamine in our system,” (*Cleveland Clinic*). This balanced amount of dopamine in our system evolved to keep us alive, as it serves to release pleasure from activities necessary for survival. However, high levels of the neurotransmitter are usually characterized by feelings of euphoria and increased energy, as well as “poor impulse control” and “being more aggressive” (*Cleveland Clinic*). Addiction is usually characterized by this higher dopamine level.

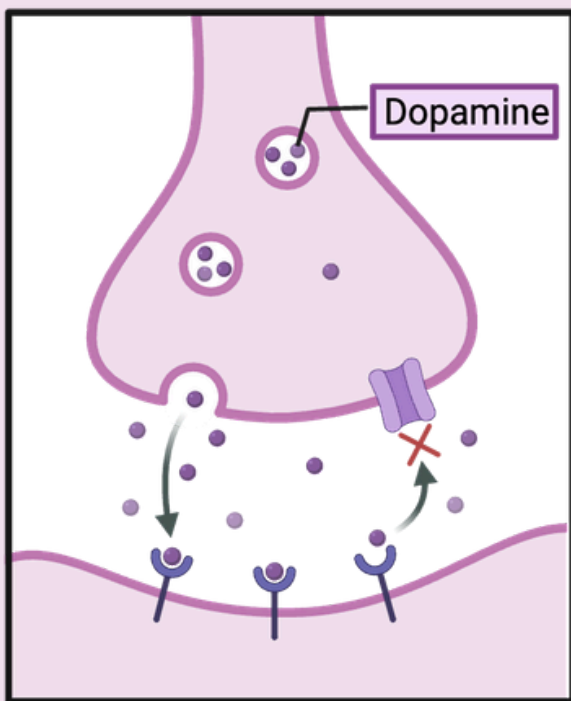
The role that dopamine plays in reward circuitry is often referenced in how “addictive drugs share the common property of

enhancing the effect of midbrain DA function...” (*Dopaminergic reward system: a short integrative review*, Oscar Arias-Carrion). Because most addictive drugs are characterized by their ability to enhance the effects of dopamine, the feelings usually connected with a reward are further enhanced when drugs come into the system. Many addictive drugs can fall into the dopamine reuptake inhibitor category. These prevent dopamine from re-entering and being reabsorbed by the nerve cell that released it. This makes more dopamine available to more neurons in your brain.” (*Cleveland Clinic*). Dopamine reuptake inhibitors are very useful in treating depression and narcolepsy. (*Cleveland Clinic*). When prescribed and monitored by a practicing and licensed physician or psychiatrist, these inhibitors can be very helpful to patients and are a good medical tool when used correctly.

On the other hand, when these dopamine reuptake inhibitors are used incorrectly, it intensifies the reward system. It does so by artificially increasing the amount of dopamine the brain senses through signaling. This creates the heightened sense of reward that usually comes from a more basic natural place. The brain starts to associate this euphoric sense with the drug, and this association leads to the formation of habits and dependency on the drug.

While the initial feeling has already been discussed, addiction is ultimately a chronic, recurring behavior that can be explained by synaptic plasticity.: Plasticity is the brain’s ability to strengthen or weaken certain connections between neurons, which is key to learning and memory. There has been a focus on how most drugs target the mesocorticolimbic dopamine (DA) system, but it is important to note how this also impacts other areas of the brain. For example, the glutamatergic and GABAergic synapse

transmissions that occur around these areas are heavily involved in the DA system. Studies show that drug-evoked synaptic plasticity “outlasts the presence of the drug in the brain and contributes to the reorganization of neural circuits...with repetitive drug exposure, they may add up and cause addictive behavior.” (*Drug-evoked synaptic plasticity in addiction: from molecular changes to circuit remodeling*, Christian Luscher). It is this repeated exposure and strengthening of these specific pathways that are key when it comes to understanding how addictions form.



To delve further into the mechanism of addiction, cocaine has been the focal point of most literature. Cocaine is a dopamine reuptake inhibitor, which causes a build-up of dopamine and a perceived sense of euphoria. Over time, with chronic cocaine abuse, the brain starts to rely on a high amount of dopamine. This means that, over time, the user needs more of the drug to achieve the same euphoric high felt during the first use.

Addiction is often described as “hijacking the brain”, but this is not the most accurate definition of what is occurring. Although behaviorally, it makes sense to call it a “hijack” due to the way it compromises decision-making, the biology behind it goes a bit further. Rather than actually changing the anatomy of the brain, “what happens in addiction is..., natural processes involved in all learning, the brain prunes nerve pathways of attention and motivation to preferentially notice, focus on, desire and seek the substance.” (*Addiction and the Brain*, Psychology Today).

In short, addiction does not physically reshape the brain so much as it rewires its pathways, altering patterns of attention, motivation, and behavior. Research is still ongoing, but at the end of the day, addiction is not simple but rather a summation of many experiences, sensations being encoded all at once, and signals getting crossed. In theory, if neuroplasticity allows the brain to change in ways that support addiction, it may also be the mechanism that helps the brain recover and regain control. By leveraging the brain's ability to adapt, advances in treatment aim to restore balanced signaling and rebuild healthy neural pathways.

THE MIND'S HIDDEN COMPASS: DREAMS, DEEP THOUGHT AND THE ART OF INTUITION



Author: Adya Jhunjunwala
Artist: Abby Betts

Have you ever had a gut feeling about something that turned out to be right? Or solved a problem almost out of nowhere after “sleeping on it”? Maybe you’ve had a vivid dream that felt strangely insightful, or walked out of a test or presentation feeling like you just knew what to do. These experiences, often called intuition, the “sixth sense,” or subconscious insight, aren’t mystical. They’re actually reflections of your brain doing what it does best: processing information, recognizing patterns, and making connections without you even realizing it.

The brain is never truly at rest. Even when we’re asleep or feel like we’re doing nothing, it’s constantly working by piecing together experiences and emotions in new ways. Dreams, deep thinking, and intuition give us small glimpses into these hidden processes. Each represents a different side of the mind’s ability to organize and make sense of information beyond conscious awareness. At the same time, they’re closely connected: intuitive insights can take root in dreams, develop during reflection, and eventually show up as sudden clarity or instinct.

DREAMS: NIGHTTIME EXPERIMENTS OF THE MIND

Dreams are strange. They mix fragments of memory and imagination in ways that often don’t seem to follow logic. Neuroscience helps explain part of this. During REM sleep, the stage most closely tied to vivid dreaming, the limbic system, which controls emotions, becomes highly active, while the prefrontal cortex, responsible for logical reasoning, becomes less active (Hobson, 2009). This is why dreams can feel emotionally intense but not always make sense.

Yet dreams serve functions beyond the bizarre. REM sleep helps consolidate memories, regulate emotions, and process

experiences (Walker & Stickgold, 2006). In a sense, dreams are the brain’s internal laboratory, a space where it experiments with ideas, connections, and scenarios. Creative problem-solving often emerges here: artists, writers, and scientists alike have reported breakthroughs inspired by dreams. For example, the structure of the benzene molecule supposedly came to chemist August Kekulé in a dream of a serpent biting its tail, illustrating the subconscious as synthesizing patterns from memory.

Dreaming is also a mechanism for emotional regulation. Traumatic, joyful, or unresolved experiences often appear in dreams, allowing the mind to process and integrate them. Think of it as a form of emotional rehearsal, as it gives us a chance to simulate responses, navigate scenarios, and even confront fears in a safe, imaginative space. In this way, dreams serve as an internal guidance system, quietly preparing us for challenges, social interactions, and creative tasks.

DEEP THOUGHT AND THE UNSEEN CURRENTS OF COGNITION

We dream while we sleep and think while we’re awake, but much of both remains out of sight. Our conscious mind is only a small part of a much larger system. Our conscious mind is only the tip of a vast cognitive iceberg. Cognitive psychology describes two modes of thought: System 1, fast and automatic, and System 2, slow and deliberate (Kahneman, 2011). System 1 runs continuously in the background, detecting patterns, integrating experience, and flagging potential solutions before conscious thought catches up. Whereas System 2 engages more intentionally by analyzing information and working through decisions step by step when conscious effort is required.

This is why stepping away from a problem can actually help solve it. The idea of incubation in



creativity suggests that taking a break allows the subconscious to keep working without interruption (Dijksterhuis & Nordgren, 2006). Those sudden “aha” moments often come from this hidden processing. Even when you’re focused on something completely different, your mind is still sorting through information, comparing possibilities, and building connections.

Deep thought also connects to emotional and bodily states. Sometimes, clarity emerges not from deliberate reasoning but from an alignment of emotional readiness, bodily calm, and subconscious synthesis. These moments feel effortless because the mind has already done the heavy lifting invisibly, like an orchestra tuning itself before the conductor signals the start.

INTUITION: THE BRAIN’S RAPID PATTERN DETECTOR

Intuition is what all of this processing looks like when it surfaces. It’s what happens when the brain recognizes patterns so quickly that it feels like there’s no reasoning involved. Often described as a gut feeling or instinct, intuition brings together emotional memory, physical sensations, and past experiences into fast judgments.

The somatic marker hypothesis helps explain this. According to it, bodily signals, like a sense of tension or ease, act as markers based on past emotional experiences, guiding decisions without conscious thought (Damasio, 1994). Brain studies also show that intuitive decisions involve areas like the ventromedial prefrontal cortex and the amygdala, which are linked to emotion and risk evaluation (Bechara et al., 1997). In simple terms, intuition is the brain compressing a huge amount of past information into a quick sense of what feels right.

Experience strengthens this ability. The more familiar someone is with a situation, the better their brain becomes at picking up patterns (Klein, 1998). That’s why a firefighter can sense danger instantly, and a chess player can anticipate moves of the opponent. Intuition is not magic; it’s the brain operating at peak efficiency, honed by experience and pattern recognition.

WHY SOME PEOPLE SEEM TO HAVE A “SIXTH SENSE”

Intuition isn’t the same for everyone. Some people seem naturally more in tune with it, but that difference isn’t mystical. It often comes down to awareness. People who pay closer attention to their emotions and bodily signals are more likely to notice subtle cues. Those who reflect on past experiences are better at recognizing patterns and refining their instincts.

On the other hand, stress, distraction, or limited experience can weaken intuitive signals. When your attention is scattered, it's harder to pick up on what your brain is already processing.



Practices like mindfulness, reflection, and exposure to varied experiences can sharpen awareness and make these signals easier to interpret. The key isn't creating intuition, it's learning to notice what's already there.

INTERWEAVING DREAMS, THOUGHT, AND INTUITION

Dreams, deep thinking, and intuition aren't separate processes; they're part of the same system. Dreams provide the raw material: emotions, memories, and fragments of problems. Deep thought allows these pieces to settle, combine, and reorganize. Intuition then appears as the final result: a quick insight that feels sudden but is actually built on all that prior processing.

Think about creativity. A painter might struggle with composition, dream about unusual forms, reflect on them later, and

suddenly feel drawn to the right arrangement. A scientist might wrestle with a problem, sleep on it, and wake up with a clear idea. The final insight feels immediate, but it's the product of ongoing work beneath the surface.

Even everyday decisions follow this pattern. Whether it's trusting someone, choosing how to study, or answering a question instinctively, the brain is constantly pulling together past experiences and knowledge to guide us, often without us noticing how.

CULTIVATING AWARENESS WITHOUT FORCING IT

Although subconscious processing happens automatically, there are ways to support it. Mindfulness and meditation can make you more aware of internal signals, helping you notice intuitive cues more clearly (Kabat-Zinn, 2003). Sleep plays a key role in strengthening memory and insight (Walker, 2017). Reflective habits like journaling or thinking through experiences can also help align intuition with conscious reasoning.

Yet the magic is not in trying to control intuition. It's in noticing, listening, and trusting the subtle interplay of memory, emotion, and pattern recognition. These processes are reminders that our minds are constantly at work, even when we feel passive. They show that insight is not sudden or inexplicable; it is the natural product of a brain constantly synthesizing its world.

CONCLUSION: THE MIND'S SUBTLE SYMPHONY

Dreams, deep thought, and intuition reveal how much of the mind operates beneath conscious awareness. What we often experience as gut feelings or sudden insights aren't mystical gifts, but the result of continuous processing shaped by memory, emotion, experience, and bodily awareness. By noticing, reflecting, and giving

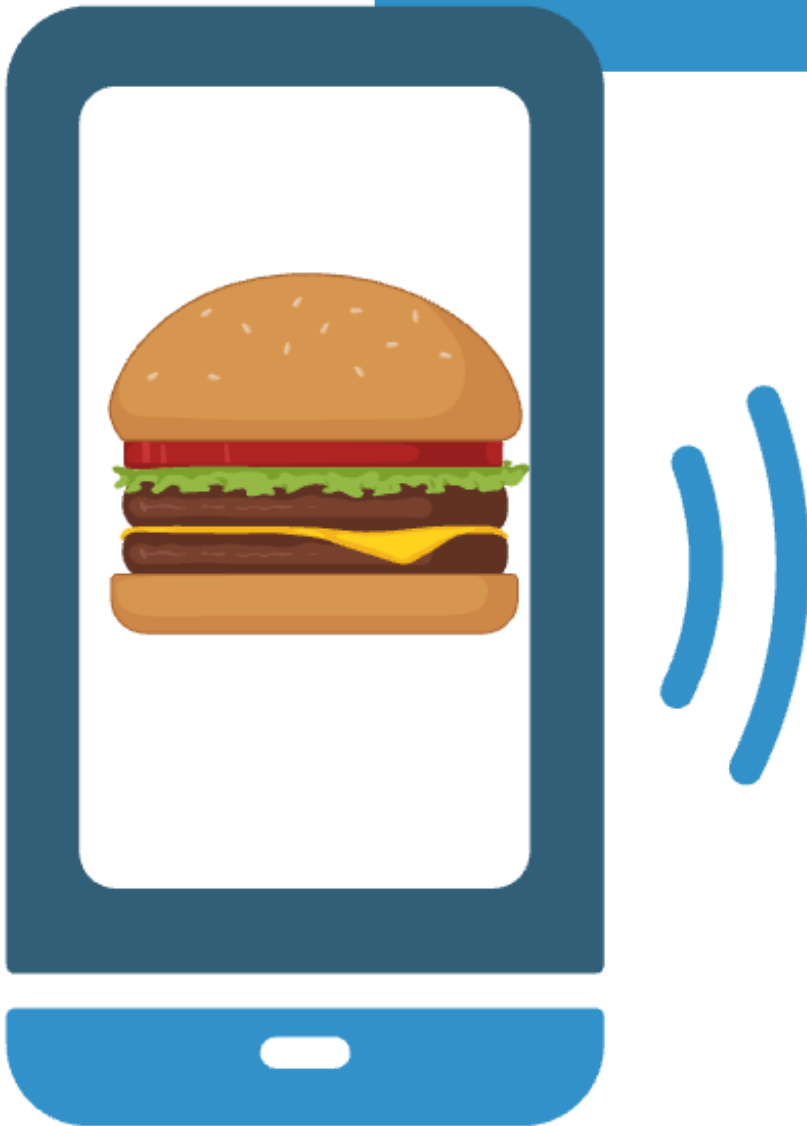
space to these processes, we can become more in tune with the subtle signals our brains are already generating.

Our internal world is vast, layered, and often out of sight, yet it constantly shapes how we perceive, decide, and create. Even in moments of stillness, the brain remains active, quietly weaving together dreams, insights, and intuition into the fabric of our experience, offering glimpses of understanding that can feel almost magical.

THE DOPAMINE DRIVE-THRU: WHY FAST FOOD FEELS ADDICTIVE

Author: Nafisa Promi

Artist: MJ Soares



On a Friday night, you sit comfortably still under your 3 coziest blankets stacked on top as you scroll endlessly on social media. You remember you haven't had dinner yet, so you can't resist tapping through Grubhub, DoorDash, Uber Eats, or the app of your

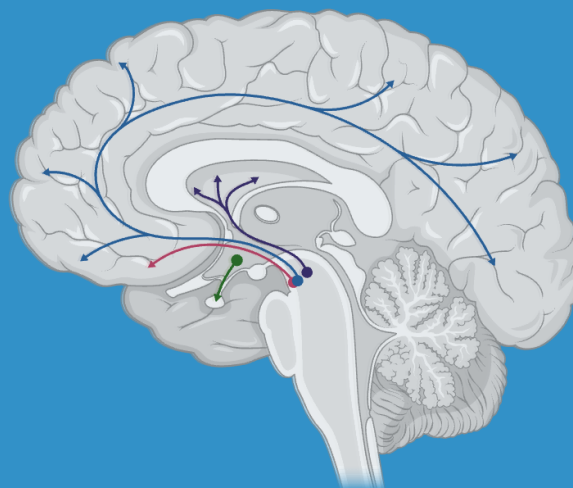
choice. Taco Bell's famous Chicken Cantina wrap, followed by a large nacho fries, makes its way into your bag. Before you confirm your order, you add one more essential item to your bag: the Iconic Baja Blast. What is it about that precise instant that makes placing an order

seem so natural, almost instinctive, as if the choice had already been made before you gave it any thought? Why do some foods suddenly become "necessary" instead of optional? What makes your brain prioritize instant comfort over long-term intention, even when you weren't actively hungry a few minutes ago? And why do apps and images of food seem to intensify that urge the longer you scroll? In a world that carries out our internal psychological behavior to drive our next craving, forcing us to find fast food, specifically with high concentrations of salt and sugar, rewarding, these moments are rooted in deeper brain mechanisms, environmental cues, and evolutionary survival mechanisms to continue to shape our behavior. This reading examines how contemporary food contexts interact with reward circuits, dopamine signaling, and stress-related brain processes to affect eating behavior. Through examining the neuroscience of reward, habit formation, and emotional regulation, we can understand why such fast food begins to feel so compelling and hard to resist. We will delve into the understanding of how our brains become wired to find pleasure in eating more, every time, and how the different regions of our brains play a part without us realizing. It is important to note that our body craves fast food in a continuous, repeated cycle in a similar way drugs exhibit a spark in neural pathways and neurochemical mechanisms during addiction. The brain's executive functioning refines carbs, salt, and sugar in a specific ratio that overstimulates the brain's reward system. This is known as hyperpalatable foods (Hough et al.). On top of that, emotional eating becomes neurologically reinforced. Stress increases preference for high-fat and sugar foods, and has been shown to reduce stress temporarily by releasing hormones like cortisol. These cravings become more desirable and

rewarding. Chronic stress weakens prefrontal cortex control, which leads to impulse eating (Dallman et al.).

REWARD CIRCUITRY IN THE BRAIN

When we experience something rewarding, such as tasty food like Taco Bell, the brain learns from it and tries to predict and repeat that same feeling. This is mostly driven by a pathway called the mesolimbic dopamine system, which connects areas like the ventral tegmental area, the nucleus accumbens, and the prefrontal cortex (Volkow et al.).



When dopamine is released, it sends signals to the brain that convince it to seek the experience as rewarding, increasing the likelihood that the behavior will be repeated in the future. Recent neuroscience research also suggests that they are not just "in the moment" impulses, but also learned responses shaped by repeated cue-reward associations, where even the smallest triggers like food apps or a certain food image can highly activate dopamine signaling and gradually shift behavior from a simple, impulsive decision-making to an automatic, habitual behavior. Our brains are also wired to favor what works in our favor, such as ordering straight from our phone with one simple click, knowing the minimal effort on our part that comes into the arrival of our food. With

repeated exposure to highly palatable foods, the brain undergoes neural sensitization, where long-term stimulation of reward pathways leads to a reduction in D2 receptors, a type of receptor in the brain that responds to the neurotransmitter dopamine, availability in the reward circuit (Johnson and Kenny, 2010). This weakens the brain's ability to regulate reward signaling, making inhibitory control less effective and increasing persistent craving and seeking behavior. This leads to our self-control becoming weaker over time, explaining why eating is not always as simple as responding to hunger. Eating behavior is divided into two systems: hedonic eating and homeostatic eating.



Hedonic v.s. Homeostatic

Hedonic eating refers to eating driven by pleasure, where the brain's reward system can override signals of actual energy needs when highly appealing foods are available. This is why foods like fast food can feel "necessary" even when the body is not truly hungry. In contrast, homeostatic eating is controlled by the body's internal energy regulation system in the hypothalamus, where hormones like leptin, ghrelin, and insulin signal when the body needs fuel or has enough energy (Berthoud 2012). Together, these two systems show how eating behavior is shaped not only by physical need, but also by reward-driven brain processes. While these reward-driven processes provide insight into how the behavior underlying eating operates in the current state, they are rooted in a deep biological system shaped by earlier survival

and evolution.

EVOLUTIONARY SURVIVAL WIRING & NEUROBIOLOGY

From a broader perspective, these reward and eating behaviors are not just shaped by modern environments, but also by deep evolutionary history. Through natural selection, human neural circuits evolved to prioritize high-calorie foods rich in sugar, fat, and sodium, since these nutrients were once scarce but essential for survival. In early environments where food was limited, individuals whose brains strongly responded to energy-dense foods were more likely to survive and pass on these traits. However, in today's world, those same survival-based mechanisms still exist, even though high-calorie foods are now constantly available. As a result, the brain continues to operate as if it is in a scarce environment, even when it is not. This evolutionary mismatch also helps explain the rising rates of obesity in modern environments, where highly palatable foods are constantly available and frequently cued by the surroundings. Although homeostatic mechanisms evolved to maintain energy balance through internal metabolic signals, they are often overwhelmed by strong hedonic drives that promote eating for pleasure rather than need. Now, as ancient survival-based neural systems that once ensured survival in food-scarce environments, contribute to excessive caloric intake in conditions of abundance (Berthoud 2012). Research on habit and reward circuitry suggests that repeated exposure to rewarding food cues can further reinforce these patterns by shifting behavior from conscious, goal-directed control toward more automatic, habit-based responses. This process is largely driven by changes in the basal ganglia, particularly the striatum, where dopamine signaling strengthens learned cue-reward associations over time (Lipton et al.). As these neural pathways become more efficient, eating behavior can become less dependent on hunger

signals and more triggered by environmental cues, making food consumption increasingly habitual rather than intentional. Though evolutionary changes explain why we are drawn to such high-calorie foods, they do not completely consider external factors, such as stress, that are also great contributors to our behavior.

EMOTIONAL REGULATION & STRESS EFFECTS

In animal studies, prolonged stress has been shown to dramatically increase the consumption of highly desirable meals. For instance, stressed mice continuously consumed highly rewarding, high-energy diets in a mouse model subjected to repeated stressors. Neurobiological changes in the brain's reward system, specifically in the nucleus accumbent, a crucial area involved in processing pleasure and reinforcement, are connected to this behavioral shift. Stress can alter mu-opioid receptor expression and dopamine D2 receptor function in this region, making hedonic feeding circuits more sensitive (Wei et al.).



Acute stress has been shown in human studies to reduce self-control when making food-related decisions, causing people to select more harmful, highly rewarding foods

over healthier options following exposure to a standardized stressor. This effect weakens top-down regulatory control over behavior and is associated with stress-induced alterations in functional connections between the prefrontal cortex and limbic areas like the striatum and amygdala (Maier et al.). Dopaminergic alterations brought on by stress thus decrease prefrontal efficiency, leading people to be more motivated by short-term gratification than by long-term goals. In addition to this, external environmental factors further shape eating behaviors through engaging with the brain's reward and decision-making systems.

FOOD MARKETING ALTERING BRAIN ACTIVITY

In an fMRI study, participants made "eat" or "not eat" decisions after seeing food versus non-food ads, demonstrating how food advertisements affect children's food decision-making via changing brain valuation systems. Children showed a shift toward hedonic-driven decision making after being exposed to food advertisements; they prioritized taste and instant reward value over health considerations. Increased activation in the ventromedial prefrontal cortex, a crucial brain area involved in determining subjective value, coincided with this behavioral shift, indicating that food marketing enhances reward-based valuation during decision-making (Bruce et al.). It is no surprise that food advertisements seem to lessen the relative impact of prefrontal control mechanisms. Furthermore, in a study where children viewed different food ads, the children who were overweight or obese displayed increased brain response to high-energy food signals, especially in areas related to reward and sensory processing, according to the study's significant weight status disparities. Food advertising consistently decreased the activation of cognitive control-related prefrontal cortical areas, indicating a transient deterioration of regulatory processing during

subsequent meal judgment (Masterson et al.). It's interesting to note that this brain change did not immediately result in higher overall meal intake, suggesting that the impacts of advertising might be more gradual and long-lasting. Overall, the results imply that food advertisements alter children's neural processing of food signals.

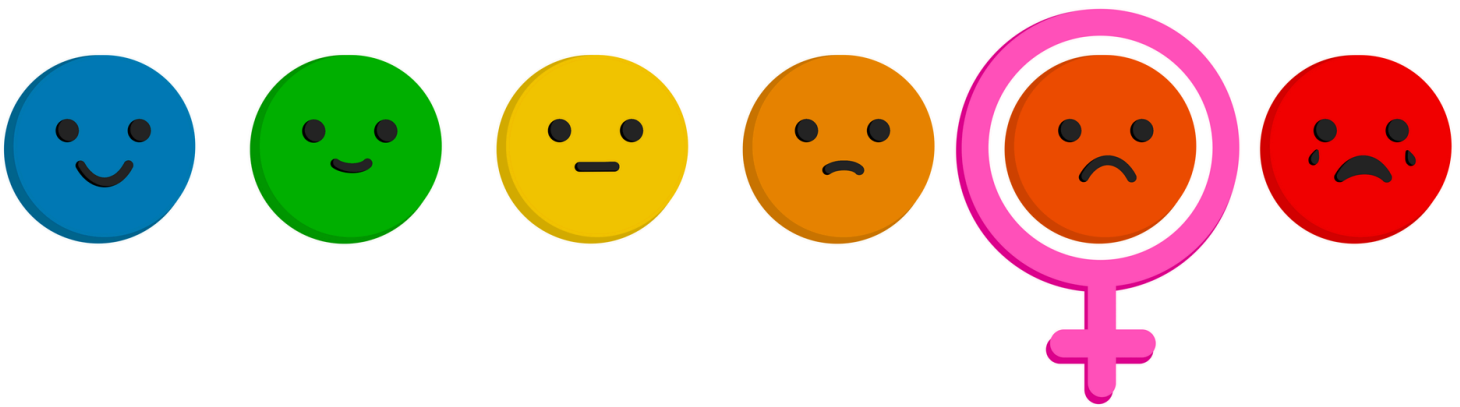
CONCLUSION

All things considered, these interrelated biological and environmental elements demonstrate how deeply ingrained brain systems influenced by both evolution and contemporary culture determine eating behavior rather than being just a question of conscious choice. The balance of decision-making gradually swings toward automatic, hedonistic behavior when reward-driven circuitry is repeatedly triggered, and prefrontal control is regularly tested by stress or outside stimuli. Brain-based systems have a significant influence on determining daily food-related behavior and help explain why overconsumption continues even in the presence of knowledge or intention to eat properly.

THE NEUROSCIENCE BEHIND WHY WOMEN EXPERIENCE CHRONIC PAIN DIFFERENTLY

Author: Krisha Shah

Artist: Abby Betts



While it may seem that biological men and women have similar levels of pain tolerance, women are significantly disproportionately affected by chronic pain compared to men. As awareness of women's health continues to grow, it's vital to consider the neuroscience behind differences in health. Sex differences in the experience of chronic pain arise from enhanced central sensitization and neuroplastic changes to pain-processing networks, which are more pronounced in women due to hormonal modulation and neuroimmune influences. Understanding the basis of these differences is important for developing more targeted and effective treatments in the future.

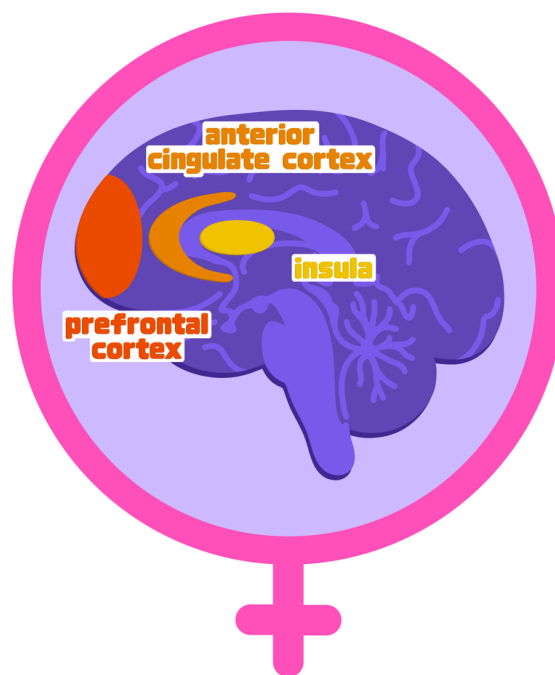
The International Association for the Study of

Pain states that women experience higher prevalence, greater severity, and longer duration of chronic pain, compared to men. In fact, these symptoms are disproportionately seen across many different conditions such as fibromyalgia, endometriosis, irritable bowel syndrome, chronic migraines and more (Blyth, 2024). These listed conditions commonly co-occur and are known as Chronic Overlapping Pain Conditions, which are characterized with symptoms of fatigue, chronic pain, cognitive difficulties, and for irritable bowel syndrome it causes diarrhea and abdominal pain. The overlap of these conditions points to shared psychological pathways, which highlights the importance of examining underlying mechanisms such as hormonal regulation and neural sensitization.

One key mechanism in shared psychological pathways is the hormonal modulation of pain, in which ovarian hormones influence how pain signals are processed and regulated. In simpler terms, fluctuations in hormone levels can alter how sensitive the body and brain are to pain. Ovarian hormones modulate pain pathways through their effects on neurotransmitter systems, opioid receptors, and inflammatory signaling. In a 2015 study, Dr. Victor Chaban found that female primary afferent sensory neurons exhibit increased excitability and responsiveness to pain-related stimuli under the influence of ovarian hormones. These neurons are responsible for detecting and transmitting pain signals and therefore may increase pain signaling in females. Additionally, hormone fluctuations during the female menstrual cycle can lead to variability in pain sensitivity and increase vulnerability to chronic pain conditions (Chaban, 2015). Together, these findings suggest that hormonal regulation provides a foundation for sex-based differences in pain perception.

Besides hormone modulation, central sensitization and neuroplasticity play a large role in chronic pain in women as well. Central sensitization refers to the process where repeated, prolonged pain exposure leads to increased excitability in the central nervous system. There have been studies that suggest that women are more susceptible to these changes, resulting in amplified, persistent pain even in the absence of an injury (Smith, 2019). Sleep disruption is also known to significantly increase pain sensitivity, with increased effects observed in women (Rosen, 2017). According to these findings, both biological susceptibility and external factors can interact to amplify chronic pain.

Sex differences in chronic pain are also evident in brain function and connectivity. A functional MRI study identified differences in



functional connectivity across brain regions involved in pain perception. These areas include the insula, anterior cingulate cortex, and prefrontal cortex and are associated with sensory discrimination, emotional regulation, and pain modulation, which suggests that differences in neural network activity may influence how pain is experienced (Wilcox, 2015). As a result, men and women may process and respond to pain in different ways at the neural level, which signifies how pain is not only a significant physical sensation but also an experience shaped by emotional and cognitive processing.

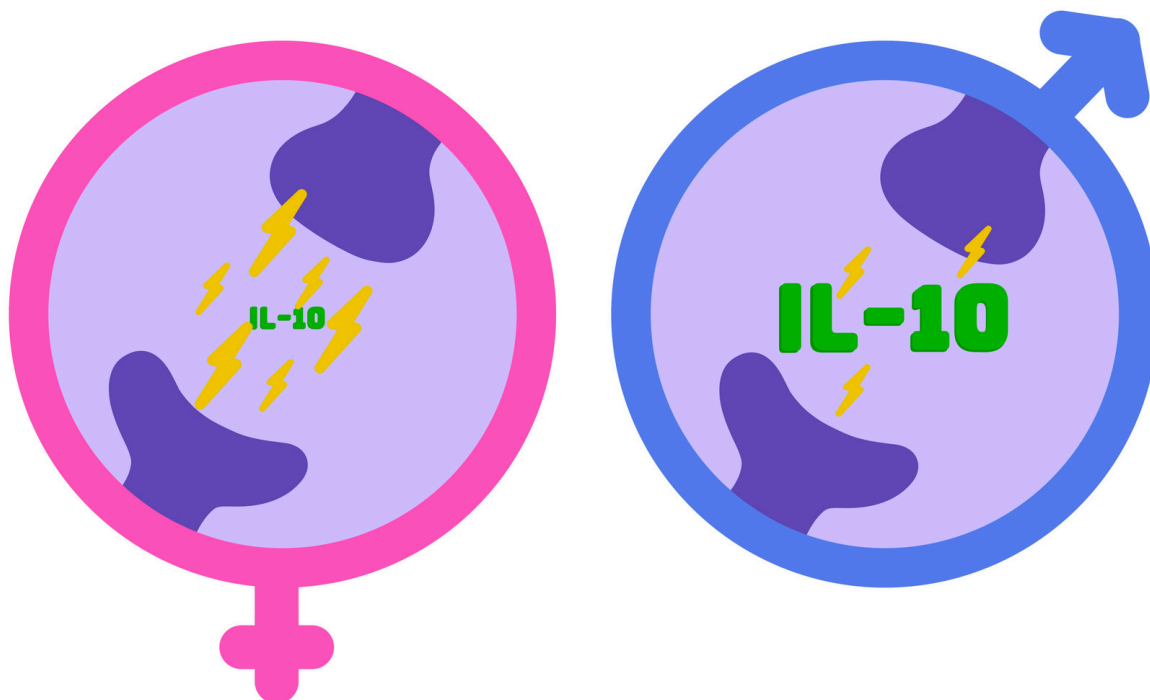
In addition to the differences found at neural levels, research points to the importance of neuroimmune interactions in shaping experiences in chronic pain. The activity of microglia and inflammatory mediators in the immune system plays an important role in modulating pain signaling. Microglia are specialized immune cells in the central nervous system that are important for the brain's defense systems. When these neurons are activated, most likely in response to high

amounts of stress or injury, they release inflammatory mediators such as cytokines and chemokines which increase neuron excitability and pain signaling pathways. This process enhances the communication between the immune and nervous systems of the body. These methods may also vary by sex, as the magnitude and regulation between inflammatory responses of men and women can differ and lead to greater or prolonged pain signaling in women. Research done by Sarah Rosen found that communication between the immune system and nervous system differs between sexes and therefore leads to variations in pain initiation and how it's maintained (Rosen, 2017). Understanding these sources together suggests that chronic pain differentiation does not only stem from a neurological aspect, but an immunological one as well with sex-specific immune responses being large contributors.

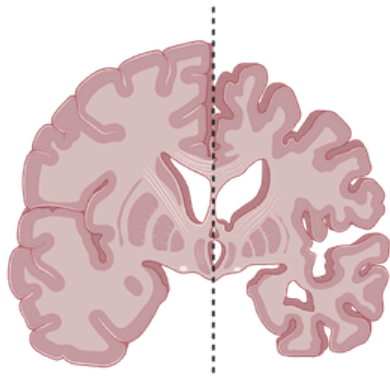
Building on this previous work, a study done in 2026 by Jaewon Sim et al. demonstrated

that more specific immune mechanisms may explain differences in pain duration between sexes. They found that monocyte-derived IL-10, an anti-inflammatory cytokine (signaling protein) that helps suppress pain responses, plays an important role in resolving pain and has notable sex-dependent effects. This study also suggests that testosterone may enhance IL-10 activity and therefore gives a possible explanation for why there is more efficient pain resolution in males (Sim, 2026).

Overall, sex differences in chronic pain arise from the complex inner workings of hormonal, neural, and immune mechanisms. Differences in peripheral sensory processing to variations in brain connectivity and immune signaling show how multiple systems contribute to the increased prevalence of pain in women. It's important to recognize these factors to improve our current knowledge of chronic pain and to apply different approaches for treatment in a clinical setting.



WHEN MEMORIES FADE: THE SLOW EFFECTS OF ALZHEIMER'S ON MEMORY AND IDENTITY



Author: Kaniz Fetema

Artist: MJ Soares

Healthy Brain vs. Alzheimer's

When people think about Alzheimer's disease, they often associate it with memory loss. While memory loss is one of its most recognizable features, Alzheimer's is far more complex than simply forgetting names or misplacing objects. It is a progressive neurological disorder that gradually alters how the brain functions and, in many ways, reshapes who a person is. Over time, it affects memory, cognition, behavior, and a person's sense of identity. What makes Alzheimer's especially difficult is that it not only impacts the individual but also deeply affects families and relationships. Examining both the biological processes behind the disease and the lived experiences of patients reveals that Alzheimer's is not just a medical condition, but a deeply human one.

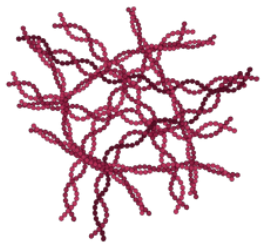
At the biological level, Alzheimer's disease is driven by changes in the brain that disrupt

communication between neurons. One key feature is the accumulation of amyloid-beta proteins, which normally play a role in neuron function and repair.



Amyloid-Beta Plaque

In Alzheimer's, however, these proteins clump together to form plaques between neurons, interfering with cell signaling. Another major factor involves tau proteins, which typically help stabilize the internal structure of neurons. In the disease state, tau proteins become abnormally twisted into tangles, weakening the neuron's structure and transport system. As plaques and tangles accumulate, neurons begin



Tau Protein Tangle

to lose function and eventually die, weakening connections across different brain regions.

This damage is especially significant in the hippocampus, the area of the brain responsible for forming new memories. As a result, memory loss is often one of the earliest noticeable symptoms (Kumar, 2024). As more neurons are affected, the brain gradually loses its ability to function normally, leading to widespread cognitive decline.

One of the most challenging aspects of Alzheimer's is how gradually it develops. The disease begins with subtle changes that can easily be mistaken for normal aging. A person may forget a recent conversation, lose track of time, or struggle to find the right word. At first, these moments may seem harmless. However, as the disease advances, these symptoms become more frequent and begin to interfere with daily life. Tasks that once felt automatic, such as cooking or managing a schedule, become increasingly difficult. Over time, individuals may struggle to recognize familiar faces or navigate places they have known for years. This slow progression makes Alzheimer's particularly unsettling, as the changes build over time until they can no longer be ignored (İscanlı et al., 2024).

Beyond the biological changes, one of the most profound impacts of Alzheimer's is how it affects a person's identity. Memory plays a central role in how we understand ourselves—it connects us to our past, our relationships, and

our sense of continuity, meaning the feeling of being the same person over time. As memories fade, individuals may begin to lose parts of who they once were. This loss can appear as changes in personality, mood, or behavior. Someone who was once outgoing may become withdrawn, while someone who was independent may begin to rely heavily on others. These changes can be confusing and emotionally difficult, not only for the individual but also for those around them. Alzheimer's highlights how closely identity is tied to the brain and the memories we carry (Yang, 2016).

The effects of Alzheimer's also extend beyond the individual to families and caregivers. As the disease progresses, loved ones often take on increasing responsibilities, both emotionally and physically. Communication becomes more difficult, roles within relationships shift, and caregivers may experience significant stress and fatigue. Family members often describe a sense of loss, even while their loved one is still physically present. This emotional strain can deeply impact relationships, emphasizing the need for support not only for patients but also for those who care for them (Blieszner and Shifflett, 1990).

In the end, understanding Alzheimer's disease requires looking at both the science and the human experience. While biological changes explain what is happening in the brain, they cannot fully capture what it feels like to live through the disease. Alzheimer's challenges how we think about memory, identity, and connection. Although continued research is essential for developing better treatments, it is equally important to approach the disease with empathy and awareness. Alzheimer's is not only about losing memories, it is about understanding their significance and recognizing the profound impact their loss has on individuals and the people around them.

ONE CANNOT THINK WELL...IF ONE HAS WATCHED OTHERS DINE

Author: Nicole Gullason

Artist: Jasmine Smith



VIRGINIA WOOLF & CHIPOTLE

“One cannot think well, love well, sleep well, if one has not dined well.” This quote, famously authored by the modern literary mastermind Virginia Woolf, decorates restaurant walls, shopping bags, and more. At first glance, one might assume it means that you cannot think, love, or sleep well if you are not properly nourished. A simple sentiment such as this, however, goes much deeper. In fact, it can be reframed through a neuroscientific lens: optimal cognitive function depends on metabolic stability.

The brain, which consumes approximately 20% of the body’s glucose¹, relies heavily on consistent energy availability. When basic needs are unmet, the body activates stress response systems, such as the hypothalamic-pituitary adrenal (HPA) axis, which raises cortisol levels, can impair executive function, attention, and creativity.

When looking at it from the socio-economic standpoint, the phrase “dined well” truly symbolizes economic needs and security. Without those basic standards being met, both creative and intellectual freedom are hindered. Therefore, in modern terms, brain function depends on glucose and proper nutrition. Stressors from poverty raise cortisol, consequently reducing focus and creativity by forcing individuals to activate survival mode. Essentially, she is saying that before genius, there must be stability.

What if now, for some, stability no longer precedes genius in the same manner it had before? With access to fast-food options, large volumes of calories are consumed daily regardless of their limited nutritional value. Such a diet of overconsumption can lead to a myriad of conditions and, eventually,

instability.

WHAT THE HELL ARE MUKBANGS?

Almost all words can be broken down into their parts. The term “Mukbang” originates from South Korea. It can be loosely translated as “eating broadcast,” as *meongneun* means eating and *bangsong* means broadcast. Together, they form the catchy word “meokbang.” But what does this amusing word look like on the internet, its birthplace?

These videos are widely shared across almost all major media platforms, including YouTube, Instagram, TikTok, and more. But what about them fosters a sense of comfort for those who sit down to eat a meal? To answer this query, we must go to antiquated territory--COVID-19.

According to Delish, although Mukbangs originated in the early 2010s, their popularity surged during the COVID-19 pandemic. This correlation can be attributed to several factors, including the psychological stress of unstable access to food, isolation, and desiring comfort. Driven by the effects of neurobehavioral mechanisms, Mukbangs rose in popularity, expanded to a broader range of food, and became an imperative marketing strategy for trending foods. Most recently, foods such as Dubai chocolate, Crumbl cookies, and Biscoff cheesecake have gained viral popularity, with companies going as far as to financially support creators who film themselves eating their products—not as simple taste tests, but as lavish feasts featuring milk and three different Dubai chocolate bars. What was once a niche form of digital companionship has quietly transformed into one of the most powerful—and lucrative—engines of food marketing in the modern era.

THE BRAIN & THE REWARD SYSTEM

The brain's reward system is a distributed network of cortical and subcortical structures that encode motivation, reinforce behavior, and assign value to stimuli. The principle system is the mesolimbic dopamine pathway, in which dopaminergic neurons project from the ventral tegmental area (VTA) to the nucleus accumbens. From this process, something called incentive salience is signaled, which fosters the "wanting" sensation that drives goal-directed behavior. A simple example is when a child gets punished for misbehaving, causing them to do less "misbehaving" actions. Another example is when someone consumes a candy, enjoys the sweetness, and takes actions to taste more sweetness.

The reasons why the brain is wired to value sugar are quite interesting. For starters, in ancestral environments, calorically dense foods were scarce, so neural systems evolved to prioritize and remember high-reward stimuli. Consequently, sweet tastes activate not only the gustatory pathways—the neural circuit that translates taste from the taste bud in mouth to the specialized receptors in the brain—in the insular cortex but also dopaminergic projections to the nucleus accumbens. However, this reward system is highly intricate and goes well beyond sugar.

Mukbang videos often have saturated colors, and the visual processing of highly appealing foods triggers dopamine release in both the ventral tegmental area and the nucleus accumbens². Even without consuming the food, our brains jump to think: that's energy! Interestingly, mirror neurons are a type of cell that are stimulated when a specific action is observed. Whilst watching a Mukbang video, the brain partially mirrors the experience. As a

result, anticipatory dopamine fires the same way it does when one eats.

The reward for watching Mukbang videos, however, is not just food, but companionship. Parasocial interactions create a sense of eating alongside the content creator, and for the brain, that sense of connection is a reward in its own right. The Mukbanger often makes eye contact with the camera, speaks conversationally, and addresses viewers directly. The brain then processes this as a quasi-social interaction, a type of psychological interaction where an individual develops an emotional bond or connection to a media figure or content. And although the relationship is one-sided, it activates social reward circuitry in the brain, which are the same regions implicated in real human connection. So the brain is no longer just releasing dopamine to respond solely to food cues, but also in response to the feeling of not being alone.

For clarity and deeper scientific understanding, let's broadly define a few areas of the brain: the medial prefrontal cortex is the site of social cognition; the temporoparietal junction is vital to taking perspectives; and the ventral striatum is involved with the reward system. When watching these videos, all of these areas are stimulated, creating the illusion that the viewer is actually with the Mukbanger, sharing those delicious foods. The brain evolved for communal eating, so watching others eat activates those same social reward pathways, recreating the warmth and comfort of a shared meal.

HUNGER, HORMONES, & DIET

The hypothalamus is the brain's metabolic control center and is responsible for managing blood glucose levels as well as appetite hormones like ghrelin, leptin, and insulin. For

context, ghrelin is the hormone responsible for the sensation of hunger, while leptin is the satiety hormone. Visual and sensory food cues can activate hypothalamic circuits, therefore adjusting hormone levels. For example, while watching a Mukbang, ghrelin release can be amplified via hypothalamic activation, enhancing the central striatum's response, which increases subjective cravings. Exposure to Mukbang videos can negatively impact the effectiveness of leptin. Furthermore, leptin, when working adequately, decreases hunger and hinders dopamine response to food. Remember the last Thanksgiving dinner when you ate so much you had to unbutton your jeans, and when Grandma Carol offered you another slice of pumpkin pie, you got queasy? That is due to leptin. However, in leptin resistance, the brain stops responding properly, satiety signals weaken, and reward sensitivity to food may remain high, which is a direct result of watching Mukbang content.

Now it is time to discuss insulin. Insulin regulates blood glucose levels by increasing the cellular absorption of glucose, thereby decreasing its levels in the blood. Insulin also acts on the brain. It promotes a sense of satiety and modulates dopamine activity. Chronically high insulin levels from incredibly refined carb diets can alter dopamine sensitivity and thus change reward thresholds, making content about those high-calorie foods that lack nutritional depth all the more stimulating. This means for frequent Mukbang viewers, who are simultaneously exposed to visually stimulating food content while potentially snacking on the very refined carbohydrates being consumed on screen, the cycle becomes self-reinforcing: Dulled dopamine sensitivity demands ever more stimulating content and ever more indulgent food to feel the same reward.

Diet composition can change hormone dynamics. When viewers begin indulging in the high-calorie foods they are inclined to consume after watching Mukbangs, their diet often spikes in sugar and fat. This leads to rapid insulin spikes, rebound hunger, potential leptin resistance, and increased dopamine reactivity. However, when an individual's diet includes high protein and fiber intake, they have relatively low ghrelin levels, a slower rise in blood glucose, increased satiety hormones, and reduced reward activation. So the moral of this story is: eat well--please.

SIDE SECTION: "THE SKINCARE DINNER"

Dare I say, some influencers do it right. @danicolexx on TikTok has created a platform that promotes the idea of "eating your skincare." This does not mean literally eating your CeraVe facial cleanser--but rather eating foods naturally high in retinol, fiber, and collagen. This ultimately has a positive impact on one's dietary choices and promotes a healthy, balanced lifestyle. In my opinion, it is a breath of fresh air.

SLEEP OR SNACK?

Because Mukbang are highly stimulating thanks to visual food cues, ASMR chewing sounds, and social interactions, dopamine is released, as previously mentioned. This high dopamine activity late in the evening can delay sleepiness, increase alertness, and reinforce the "just one more video" mentality, repeatedly, all because the content is generally relaxing and neurologically stimulating. Consequently, the habit of midnight snacking is promoted.

Blue light exposure also leads to melatonin suppression, so while these addictive videos occupy your screen, blue light (roughly 460nm - 480nm) enters your eyes and leads to a delayed circadian rhythm. Circadian rhythm is

your 24-hour clock that is located in a highly important region in the brain, the suprachiasmatic nucleus. Through hormone regulation and messaging, this rhythm controls an individual's entire sleep cycle. When exposed to blue light for an extended period of time, specialized cells, intrinsically photosensitive retinal ganglion cells, become highly sensitive. This stressor sends signals to the SCN, subsequently decreasing the release of melatonin. In addition, there is higher emotional reinforcement during the night. After hours, the amygdala, the emotional processing center within the brain, has higher reactivity, therefore the prefrontal regulation is weaker. As a direct result, loneliness feels stronger, comfort-seeking increases, and parasocial content feels especially soothing. Together, these neurological forces create a perfect storm—one where the brain is simultaneously wired awake, emotionally vulnerable, and deeply primed to seek the kind of comfort that only another Mukbang can provide.

Let's be real, watching one or two Mukbang videos is not the end of the world. However, our bodies are built largely upon our lifestyle, and the effects of Mukbangs may pose a serious challenge to that regulation. Self-control and healthy decisions are incredibly important for our mental health and general wellness. So, next time you catch yourself binge-watching Mukbangs promoting not-so-clean foods, remember: One cannot think well... if one has watched others dine.



HOW WEATHER PATTERNS AFFECT OUR BRAINS

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INTRODUCTION

Most people think weather only matters when it comes to what we wear or how we prepare for the day. However, it has a bigger impact on us than we realize. The environment around us constantly affects how our bodies and minds react, even in ways we don't notice right away. Weather isn't just about what we wear, it can rewire how we think. Grey skies stimulate serotonin, heat slows our reaction times, and the smell of rain triggers a dopamine rush. Bright sunny days light up our brains with clarity and energy, while long periods of cloudy

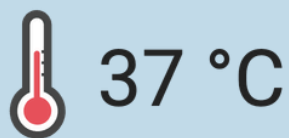
weather can make us feel unmotivated. The sky above acts like a remote control for our mind, pressing our buttons all along, showing us a preview of who you'll be that day.

1. FLUCTUATING TEMPERATURE AFFECTS COGNITIVE ABILITY AND MOOD REGULATION

Cognitive abilities range from critical decision making, analytical thinking, and interpreting incoming information. Our brain is specialized in certain ways that makes our interconnected network of neurons highly functional. For

example, the cerebrum is the largest part of our brain, and can carry out responsibilities such as memory, emotion, and language [2]. Each lobe of the cerebrum is crucial for the well-being of our body. One of the most important lobes is the parietal lobe which is associated with interpreting external stimuli such as temperature, touch, and pain. Changes in weather conditions can have a big role in how we process and act in various situations around us.

When temperatures fluctuate, the brain must work vigorously to keep homeostasis or internal balance. This can cause an additional stress in extreme conditions and alter our cognitive processes. For example, exposure to high temperatures, typically above 90 degrees Fahrenheit has been shown to impair attention, slow reaction time, and reduce overall cognitive performance. Studies have found that individuals exposed to extreme heat often perform worse on tests requiring memory and problem-solving. On the other hand, excessively cold conditions such as 32 degrees Fahrenheit can disrupt cognitive function by reducing alertness and slowing neural processing [4].



However, these effects are not only physical but also psychological. The brain's response to temperature changes is connected to neurotransmitter activity, like serotonin and dopamine which regulate mood, motivation,

and emotional stability [1]. During cooler or darker times, serotonin levels tend to decrease, which can lead to sadness, irritability, and fatigue. This is seen as Seasonal Affective Disorder (SAD), a type of depression that occurs during the fall and winter months when daylight hours are shorter. In contrast, extreme heat has been associated with increased aggression and impulsivity. This explains why people may feel more easily frustrated or mentally drained during periods of unstable or extreme weather.

This is seen in certain cases with neurological conditions such as *Cold Allodynia* and *Heat Hypoalgesia* where our thermal sensations are altered and we experience the cold as painful and heat as nonexistent [5]. These abnormalities highlight how sensitive the brain's sensory systems are to temperature and how disruptions can

Fluctuations in temperature can also influence our hormonal balance within the body. Hormones such as cortisol, the "stress hormone," increase in response to environmental stressors like extreme heat. When the body is exposed to high temperatures, it experiences physical stress, which triggers the release of cortisol as part of the body's natural response to maintain stability. Elevated cortisol levels can impair memory, reduce concentration, and contribute to anxiety. In addition to these effects, prolonged increases in cortisol can also interfere with neurotransmitters in the brain. While hormones travel through the bloodstream, neurotransmitters such as serotonin, dopamine, and melatonin send signals in the brain and affect our sleep patterns and emotional regulation. For example, high stress levels are associated with lower serotonin activity, which can negatively impact mood, as well as disruptions in dopamine, which affects motivation. Cortisol can also interfere with the production of melatonin, the neurotransmitter

responsible for regulating sleep cycles. As a result, individuals may experience difficult sleeping, increased irritability, and more emotions. This shows how strongly weather can shape the way we think, feel, and behave.

Overall, weather patterns demonstrate how closely connected our environment is to brain function. Temperature changes have a dramatic effect in shaping how we think, feel, and behave in our daily lives.

2. RECEIVING ADEQUATE SUNLIGHT DECREASES THE RISK OF NEUROLOGICAL DISORDERS

Just as fluctuating temperatures can disrupt cognitive performance and emotional stability, exposure to sunlight plays a crucial role in maintaining brain health and regulating neurological function. Sunlight not only supports our physical well-being, but also influences brain chemistry, structure, and daily activities. One of the most important systems affected by sunlight is the circadian rhythm, which controls our sleep cycle and energy levels, mood, and cognitive performance.

When sunlight enters the eyes, it sends signals to the hypothalamus, targeting the suprachiasmatic nucleus (SCN), which acts as the body's "internal clock." This helps regulate the release of melatonin, the hormone responsible for sleep [9]. The hypothalamus is a critical part of the brain's structure that maintains homeostasis with the endocrine and nervous systems. During the day, sunlight suppresses melatonin, allowing us to feel alert and focused, while reduced light in the evening increases melatonin to let us rest. Without enough sunlight, this cycle can become disrupted, leading to fatigue, irregular sleep patterns, and decreased function.

Beyond mood and sleep, sunlight has been linked to long-term neurological health. Studies have shown that limited sunlight exposure and seasonal changes are associated with the progression of disorders such as Parkinson's, Alzheimer's disease, and multiple sclerosis [8]. One explanation for this is the role of environmental factors like light exposure in terms of regulating brain volume and neural activity [6,7]. Studies have shown that sunlight contributes to maintaining healthy brain function.

Moreover, sunlight contributes to the production of vitamin D, which plays an important role in brain development and neuroprotection. Vitamin D receptors are found throughout the brain, and receiving enough levels are associated with improved cognitive function and reduced risk for neurodegenerative diseases. However, if sunlight exposure is limited, this leads to a vitamin D deficiency which is linked to cognitive decline, dementia, and Alzheimer's disease. This is due to disruptions in speed processing, "brain fog," and a reduced hippocampal volume, the area largely associated with memory.

Furthermore, it isn't just sunlight but also increased air pollution and seasonal changes in radiation exposure that impacts our brain health. These factors are also very significant in our body regulation and function. Any disruption can lead to higher susceptibility to a neurological condition.

3. SEVERE WEATHER EVENTS CAN TRIGGER CHANGES IN OUR BRAIN

Many weather events such as floods, hurricanes, and wildfires have high impacts on our brain function and overall mental health. These events can place the body under intense stress, activating the brain's hypothalamic-pituitary-adrenal (HPA) axis. This is our body's stress-response system that leads to the release of

cortisol to manage stress, regulate metabolism, and suppress inflammation. While cortisol is helpful short-term in “fight or flight” situations, a high amount can negatively impact memory, concentration, and emotional regulation.

During and after severe weather events, individuals often experience heightened anxiety, fear, and emotional distress. The brain’s amygdala, which is responsible for processing fear, becomes more active, while the prefrontal cortex, which helps with decision-making, can become less effective. This imbalance explains why people feel overwhelmed, and have difficulty thinking clearly or struggle to make decisions in high-stress situations.

In addition to psychological effects, extreme weather can also worsen neurological conditions. For example, individuals with epilepsy can experience an increase in seizure activity due to sleep disruption and environmental changes [11]. On the other hand, people with existing neurological disorders can notice worsening symptoms as their brain struggles to handle various external stressors.

These changes can cause long-term effects and can be just as impactful as the immediate response. For example, individuals can develop chronic stress, post-traumatic stress disorder (PTSD), and multiple sleep disturbances [13]. This can further lead to brain dysfunction and emotional instability. This can eventually decrease cognitive performance and mental fatigue.

4. RISING TEMPERATURES AND EXTREME HEAT ARE LINKED TO HIGHER RATES OF VIOLENCE

Long exposure to high temperatures can have significant effects on both brain function and

behavior, specifically increasing impulsivity and emotional instability. As temperatures rise, the body experiences heat stress, which places restraint on the brain’s ability to regulate emotions and maintain cognitive control. Over time, high cortisol can disrupt normal brain function, particularly in areas that regulate mood and decision-making. In addition, this leads to heightened irritability, reduced patience, and difficulty managing stress, all of which can contribute to more impulsive and aggressive behavior [10].



One of the main reasons for this is the effect of heat on neurotransmitters and brain activity. High temperatures can disrupt the balance of serotonin and lower it, leading to increased aggression and a reduced ability to manage emotions effectively. Heat stress elevates cortisol levels and contributes to frustration, anxiety, and mental fatigue. A Los Angeles study found that the risk of violent crime on hot days was higher than moderate days. This points to the fact that cities with higher latitudes experience hotter and more disruptive days. Greenspaces also are beneficial in reducing the rate of heat-induced violence because they mitigate air pollution and provide psychological ease [12].

Furthermore, prolonged exposure to heat can also contribute to more serious outcomes, such as suicidal ideation and attempts, especially in individuals already experiencing mental health challenges. These effects are more significant

because these individuals often rely on consistent routines, supportive environments, and coping strategies to manage their symptoms. For example, those with depression may already struggle with motivation and energy, leading to further withdrawal and inactivity. In addition, people managing mental health conditions may have fewer emotional or cognitive resources available to adapt to added stressors, making them more vulnerable to feeling overwhelmed. Heat can intensify feelings of hopelessness, exhaustion, and emotional distress, making it more difficult for individuals to cope [12]. Likewise, extreme heat creates a cycle in which heat not only affects the brain but also indirectly worsens behavior through sleep deprivation.

All in all, rising temperatures come at a dangerous risk in that it has a high probability of inducing violent thoughts and behavior in individuals. It increases crime rate, suicides, and other dangerous activities [12].

CONCLUSION

Although weather is very unpredictable and unstable in many situations, it is crucial how we respond to its effects on our brain and body. By understanding these impacts, we gain a sense of agency, allowing us to better manage our emotions, behaviors, and overall well-being. This awareness is especially important in that it will help us navigate uncertain conditions with greater control and confidence.

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