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Greetings wonderful humans!

This edition of The Nerve Journal has been long awaited. Our team has been working long and hard hours to publish this collection of articles related to neuroscience and the world around us. We have had a lot of creative ideas brought to us this year, with 23 pieces being published! Our magazine now includes, for the first time, poetry and creative narratives in addition to our research articles and reviews. STEM and the humanities are often thought of as separate entities. A neuroscientist works in a lab. An artist creates a painting. A writer strings together words to write a poem that taps into our deepest human emotions. The goal of our publication is to blur the lines between these professions. All of our art, layout, and articles come from passionate students at Boston University. Neuroscience is an artistic discipline. The creative human mind stems from connections between each and every cell in our brain. We hope to show, using this journal, that art and science are not separate. The mind is art, and art is the mind. We want to thank our staff for all their hard work and for helping us get this magazine back in shape. So, with that being said, enjoy.

Sincerely,
Noelle Wojciechowski (CAS, KHC ‘21) and Isabelle Pelcher (CAS ‘21)
Co-Editors in Chief
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Addictive Genes

In the late 1970s, a Canadian psychologist named Bruce Alexander conducted a study on addiction known as “Rat Park.” The experiment used rats of both sexes as subjects and was designed to test their willingness to consume morphine, an opiate, under contrasting sets of environmental conditions.

Alexander sought to determine whether drug addiction was simply the inevitable effect of consumption on the brain or the result of adverse environmental components; specifically, he hoped to validate his own hypothesis that addiction stemmed exclusively from the environment, and the drug itself did not possess inherently addictive qualities.

The psychologist divided a group of forty-eight newborn rats into two subgroups, housing a colony of thirty-two in “Rat Park,” a spacious enclosure designed to resemble a woodland environment, meanwhile restricting each of the remaining sixteen rats in its own small metal cage. Each cage, as well as Rat Park, offered two drinking bottles: the first held sugar-water, while the second contained a morphine-laced variant of the sugar-water solution.

Alexander found that the rats housed in isolation compulsively drank the opiate solution in preference to sugar-water, and did so much more often than those occupying Rat Park, who rarely touched it.1 While the rats living in solitude quickly became morphine-dependent, those enjoying the physical and interactive freedom provided by Rat Park were deemed emotionally fulfilled and therefore unmotivated by the prospect of substance intoxication.1 Alexander thus concluded that addiction was not of biochemical origin, but rather resulted entirely from environmental factors.

Bountiful evidence in support of the “Rat Park” model of addiction has been acquired during and since Alexander’s experiment. Take the Vietnam War, for example. According to a study conducted in the 1970s by pioneering American psychiatric epidemiologist Lee N. Robins, only 5% of soldiers addicted to heroin in Vietnam showed symptoms of narcotic dependence in the year following their societal reintegration upon return to the United States.10 This consistent, rapid recovery did not result from extensive drug abuse treatment—researchers concluded that the massive drop in addiction was due simply to a change in setting and, accordingly, posited that drug addiction was a legitimate response to the misery of war and served no purpose at home in the United States; in Robins’ words, “the settings in which [the soldiers] lived and worked [were not] associated in their minds with use or withdrawal symptoms, and therefore [did] not serve as stimuli to relapse.”10 Thus, undoubtedly, social reintegration is positively correlated with a successful recovery.

This study, along with Rat Park, effectively debunked what was arguably the greatest misconception about addiction at the time: the assertion that drug chemistry is the sole factor driving addictive behavior. Unfortunately, however, the conclusions of both studies merely replaced one hegemonic fallacy with another.

Alexander hypothesized and allegedly confirmed that addiction is caused not by the drug itself, but rather the user’s environment—and, as Robins proved, there are certainly cases in which environmental changes lead to recovery. The real flaws in the Rat Park study, though, have less to do with scientific accuracy than with the assumptions derived from its results. While Alexander rightfully opposed the damaging view of drug-induced addiction, his theory that addiction to all drugs occurs when people feel “caged” is supported solely by his experimentation with one type of drug, an opiate.

Although there is surely some truth in Alexander’s conclusion, Rat Park and Robins’ human-subject equivalent cannot explain cases in which emotionally-satisfied, social and successful individuals become addicted to drugs. For instance, a 1997 study involving survey of more than 850 young drug-addicted adults and over 100 comprehensive interviews...
demonstrated that many addicts tend to be active, independent, and exhibit healthy levels of self-esteem, suggesting that while environmental conditions are certainly relevant in determining the root(s) of one's drug dependence, it would be a hasty and erroneous generalization to conclude that the environment is, in all cases, to claim full responsibility.2 There is no “math” when it comes to extrapolating the causes of drug addiction; that is, more often than not, its onset cannot be predicted in the way we know one plus one equals two—the human brain is far too complex.

To clarify, addiction is defined by the National Institute on Drug Abuse (NIDA) as “a chronic, relapsing brain disease that is characterized by compulsive drug seeking and use, despite harmful consequences.”3 It is accurately described as a disease because certain drugs can impose long-lasting changes on the brain that promote destructive behaviors. Thus, while environmental factors often influence one’s decision to begin using a drug, the fundamental cause of addiction cannot accurately be described as exclusively environmental, behavioral, or biological in nature.

According to Abraham Maslow’s hierarchy of needs theory, our “deficiency needs” (i.e., in order of descending urgency: physical survival, safety, love and belongingness, and self-esteem) arise from feelings of deprivation and are typically fulfilled through innate motivation. For example, Maslow regards intrinsic biological drives such as hunger, thirst, and sex as “physiological needs,” while desires such as self-confidence, achievement, and respect for and from others fall under the “esteem needs” category. Maslow’s final and highest stage of growth, “self-actualization,” is comprised of fulfillment needs (i.e. morality, creativity, complex thought and decision-making) rather than those which merely satisfy physical and emotional inadequacies.7 While some individuals begin using drugs out of curiosity, stemming from a desire to somehow access a path to self-actualization, others do so in an attempt to appease deficieny needs; in other words, as a user becomes an addict, behavioral motivation shifts from positive to negative reinforcement, meaning that drugs once used to fulfill “growth” needs are now perceived by the body as life-sustaining.

Humans experience pleasure when their basic physiological drives are satisfied, this sensation emerging from the action of organic chemicals—primarily, the neurotransmitter dopamine—withiin the nervous system. Dopamine is naturally present in regions of the brain that regulate motivation, emotion, and pleasure. When stimulated at normal levels, the release of dopamine rewards behaviors that satisfy life-sustaining drives, encouraging the individual to repeat them. Certain drugs, however, increase the dopamine levels generated by natural rewards like eating, drinking, and sex by a magnitude of ten; this effect usually occurs almost instantly (typically when the drugs are injected), and the resultant impact on the brain’s reward system far exceeds that provided by naturally-pleasure-inducing behaviors.6 Such powerful feelings of euphoria often motivate users to take drugs repeatedly and in a manner that is instinctive rather than voluntary.

To reiterate, both environmental and biochemical factors typically contribute to an individual’s risk of developing a drug dependence. However, this is not to suggest that any two people living under the same conditions, consuming the same drugs at the same rate and frequency, will theoretically be affected by drug use in exactly the same way. Studies show that addiction to drugs, including nicotine and alcohol, also depends heavily on genetic makeup. According to NIDA, family studies that used identical twins, fraternal twins, adoptees, and siblings as subjects indicate that up to 50% of an individual’s risk of becoming addicted to drugs is genetically determined.5

In 1997, the American Journal of Psychiatry published a study on the etiology of cocaine use among 1,934 female twins between the ages of 22 and 62. For this study, NIDA-supported researchers Kenneth Kendler, M.D. and Carol Prescott, Ph.D. defined drug “use” as involving at least one non-prescribed use of a drug; “abuse” as displaying symptoms such as repeated use in situations where it imposes a physical danger, failure to meet work- or school-related responsibilities, or recurrent social difficulties stemming from the effects of the drug; and “dependence” as presenting with physical symptoms of tolerance or withdrawal, consuming the drug in larger amounts over a longer time period than originally intended, or spending increasingly longer periods seeking, obtaining, and recovering from the effects of the drug.8

Doctors Kendler and Prescott found that, while socio-environmental components are principally influential in determining whether an individual will begin using drugs, the transition from use to abuse or dependence is due primarily to genetic factors. Additionally, the concordance rates for drug use, abuse, and dependence were generally much higher for identical twins than fraternal twins: for cocaine use, concordance was 54% in identical twins and 42% in fraternal twins; for abuse, 47% in identical twins and only 8% in fraternal twins; and for dependence, 35% in identical twins and 0% in fraternal twins.8

Furthermore, extensive research conducted on the human genome has shown that the DNA sequences of any two randomly-selected individuals within the world’s human population are 99.9% identical, while the remaining 0.1% is responsible for universal genetic variation, including visible characteristics (i.e. height, hair color, and eye color) and invisible traits, such as increased vulnerability to or protection from diseases like diabetes and addiction.5 Thus, this seemingly insignificant one-tenth of a percentage determines whether a particular individual is likely to become addicted to a drug upon use.

Some diseases are caused by monogenic mutations, while the majority of known diseases, including addiction, are characterized by variations in multiple genes that contribute to an individual’s overall level of risk or protection.5 While exposure to drugs can alter both gene expression and activity, genes can also influence one’s response to the environment, consequently placing some people at a higher risk for disease than others.

The term epigenetics refers to the study of heritable changes in gene expression that are independent of nucleotide sequence; in other words, this discipline studies how organisms change as a result of modifications to gene function and expression, rather than of modifications to the genetic code itself. Such changes can occur through a mechanism called histone modification, a process by which post-translational changes to histone proteins affect gene expression by altering chromatin structure.5 Exposure to social and environmental conditions can often reconstruct, or “mark,” the structure of DNA at the cellular or organism level, such “marks” consequently impacting the expression of characteristics inherited by one’s offspring. For example, the consumption of cocaine can remodel an individual’s DNA, consequently increasing the production of proteins that correspond to drug-seeking behaviors.5

A person’s social environment can influence epigenetic patterns that, in turn, impact overall health. Researchers at the National Institute on Drug Abuse found that an estimated 22.7 million Americans required treatment for problems stemming from drug and alcohol consumption in 2013, and, in response, a University of Michigan Medical School team sought to understand the genetic and epigenetic foundations of these individuals’ evidently increased propensity for addictive behaviors.8 These scientists became the first to demonstrate a clear difference in susceptibility to addiction on the genetic level following prolonged cocaine consumption by selectively bred rats, and to accordingly attribute this increase to variations in gene expression within a specific brain region.

The UM researchers focused on the nucleus accumbens core, a brain region associated with the reward and reinforcement of life-sustaining stimuli. The study employed selectively bred high-responder (bHR) and low-responder (bLR) rat lines, which differ in several traits
contributing to overall “temperament,” to thoroughly examine the neurobiological antecedents and repercussions of habitual drug-seeking and use. High-responder rats are characterized by a constellation of attributes “reminiscent of human addiction,” including impulsivity, aggression, and novelty-induced locomotor activity, while low-responder rats exhibit addiction-resilient behaviors, most importantly, low levels of activity in a novel environment.4

Both lines of rats were exposed and adapted to a self-administration procedure over a prolonged period and subsequently examined for the diagnostic criteria for addiction, which include the persistence of drug-seeking behaviors and the propensity for relapse following abstinence.4 As anticipated, the high-responder rats were more likely to self-administer cocaine and, accordingly, to seek out the drug when the researchers stopped providing it.4

The group of scientists studied the expression and epigenetic regulation of two transcripts ascribed to major bHR/bLR differences and previously linked to addiction-related behaviors—fibroblast growth factor 2 (FGF2) and the dopamine D2 receptor (D2)—in the nucleus accumbens core.4 Researchers assessed differences in this area of the brain both before and after the rats’ prolonged cocaine exposure. FGF2 mRNA levels in the nucleus accumbens core were higher in bHRs than in bLRs both prior to and following cocaine self-administration; decreased FGF2 levels in the nucleus accumbens core likely serve as a protective ingredient, reducing one’s propensity for “transitioning” to a state of drug dependence, while high FGF2 availability may serve as a neuromolecular antecedent of drug-seeking and use.4 In contrast, the baseline mRNA levels for the dopamine D2 receptor (a so-called “pleasure receptor”, denoted D2R) observed in bHRs were lower than those found in bLRs. However, following consistent cocaine intake, this distinction was no longer observed, both lines of rats exhibiting virtually equal levels.4

The evidently addiction-prone high-responder rats were also more likely to carry a greater association of the repressive histone mark H3K9me3 at the D2R promoter (the DNA region at which gene transcription is initiated), preventing brain cells from “reading” the gene for D2 receptors.4 Low levels of D2 receptors in the striatum (the nucleus accumbens, along with the olfactory tubercle, comprise the ventral striatum) have been recorded in human cocaine abusers and positively correlated with increased rates of cocaine self-administration.4 These studies attribute such findings to previous cocaine exposure, although D2R availability has been recorded at lower levels in “impulsive” rodents that had never consumed cocaine, but were determined
to have an increased propensity for cocaine self-administration.4 These findings indicate that decreased D2R mRNA levels in the nucleus accumbens core, accompanied by epigenetic modifications, largely contribute to an individual’s predisposition to cocaine addiction; meanwhile, decreased FGF2 levels likely serve as a preventative factor.

With high FGF2 and low D2R levels relative to the low-responder rats, the high-responders were much more likely to expect and consequently seek out cocaine in response to light, a stimulus once associated with cocaine delivery.4 Similarly, such cravings are frequently reported by humans trying recover from addiction following exposure to stimuli they associate with drug use. Hence, the information obtained from this “rat model” can certainly enhance our understanding of drug addiction from a human perspective.

Ideally, treatment and public policy as it pertains to drugs can be improved in response to an understanding of the biological—specifically, the genetic and epigenetic—basis of addiction. Rather than attempting to determine whether the cause of drug addiction should be attributed to either “nature” or “nurture,” one might more accurately argue that both nature and nurture contribute to addictive behaviors. While socio-environmental factors may influence an individual’s likelihood of drug use, biological factors often dictate subsequent addiction, in the same way both genetics and lifestyle choices, such as diet and exercise, contribute to the risk of high blood pressure. For this reason, drug addiction is most accurately defined as a chronic, complex, and relapsing brain disease, and should therefore be treated as such.

**A person’s social environment can influence epigenetic patterns that, in turn, impact overall health.**

**Sources**


BRIEF DESCRIPTION OF SCHIZOPHRENIA

Schizophrenia is a mental disorder that can greatly impact the ability to think, control emotions, and empathize with others. It affects about 1% of the population and is even more rare in children. The most well known symptoms of schizophrenia are hallucinations and delusions, though it can also manifest as symptoms of disorganized thinking and the inability to socialize well with others. As of now, the cause of schizophrenia is unknown; however, past research has hinted that it could be a combination of environment, genetics, and physical factors that could be to blame.

BACKGROUND INFORMATION

Studies of families, twins, and adoptions show that genetics play a bigger role in developing schizophrenia than previously thought. However, despite most of the susceptibility to presenting the mental disorder being caused by genetics, how it is inherited is still unknown. Results from structural imaging studies also show that brain abnormalities contribute to the pathology of schizophrenia. Most studies’ results show that the lateral and third ventricle enlargement is the reason for schizophrenia. Other studies found that the reduction in grey and white matter, and specific regions in the frontal lobe, thalamus, and limbic structures take part in the development of the mental disorder. Studies have also shown that the cerebellum contributes to the cause of schizophrenia.

Genetics and the Brain Structure

In 2000, researchers wanted to investigate how genotypes can cause abnormalities in other parts of the brain, besides the thalamus, that are often seen in schizophrenic patients. Researchers took 32 same-sex siblings discordant for schizophrenia and compared them to another set of 32 normal siblings as the control group. The control group was specifically matched up with the 32 same-sex siblings by age, gender, and handedness. The average age among the healthy siblings were 40.9 years and the comparison subjects were 40.3 years. Researchers measured the volume of multiple different areas of the brain including the cerebellum, amygdala, and hippocampus in the control group and patients who have schizophrenia and a healthy sibling by using magnetic resonance imaging (MRI). Results show that there was no difference in the volume of the third ventricle among schizophrenic patients and their healthy sibling; however, in the control group, the third ventricle volume was much lower in comparison. Schizophrenic patients had a lower volume of the cerebrum compared to their healthy sibling and control group. The cerebrum volume was very similar between the healthy siblings and the control group. Patients with schizophrenia also showed a volume reduction in gray matter in the frontal lobe and an increase in lateral ventricle volume compared to the healthy sibling and the control group. Other parts of the brain that were measured did not show any significant difference in volume in the two groups of siblings.

CONCLUDING REMARKS

Genetics and brain abnormalities could be linked through two traits: third ventricle enlargement and decrease in cerebral volume. The two traits that are shared between the schizophrenic patient and their healthy sibling could be linked to genetic defects that make people more susceptible to schizophrenia. Until there is a breakthrough in how exactly genetics and brain abnormalities cause schizophrenia, the cause of the mental disorder is unknown.

REFERENCES


Alzheimer’s disease is a type of neurodegenerative disease that causes dementia. “It afflicts 4 million Americans and is the fourth leading cause of death in the United States. It is the leading cause of mental impairment in elderly people and it accounts for a large portion of admissions into nursing homes, assisted living homes, and other facilities designated for long term care.”

Hallucinations and delusions (psychotic symptoms) have been reported in a large portion of patients with Alzheimer’s Disease (AD). Hallucinations are defined to be the occurrence of the perception of something that is not actually there. People see and or hear things that are not actually present and generally have a hard time discerning whether it is there or not. A delusion is an outrageous or bizarre belief that a person has and believes very strongly despite it being contradicted by what is accepted as true or a rational argument. One example of a delusion is the belief of being constantly watched. Psychotic symptoms decrease the well-being of the patient and put more of a burden onto the caregiver of the patient. The rapid progression of dementia seen in Alzheimer’s patients has been reported to be in part to these symptoms.

A group of scientists reviewed various studies and discovered that a large portion of patients with Alzheimer’s Disease have delusions at some point after their diagnosis. It was also reported that between 21-49% of patients have suffered from hallucinations at some point during their illness. Psychosis is associated with many factors such as “age, sex, race, education, the duration of illness, and cognitive impairment. There are a few inconsistencies about associations across different studies that must be thought about. These inconsistencies are different diagnostic criteria of AD, the lack of statistical power due to methodological factors, lack of consideration of interactions among risk factors, and different definitions of psychotic symptoms.”

In the study at hand, 342 patients who lived in the community around Johns Hopkins University with Alzheimer’s disease took part in the study. All of the patients needed to meet the criteria of the Neurological and Communicative Disorders and Stroke and Alzheimer’s Disease and Related Disorders Association (NINCDS/ADRDA). The NINCDS/ADRDA criteria is the most used in the diagnosis of AD; it requires a presence...
of dementia and cognitive impairment, which must both be confirmed by neuropsychological testing. "All participants had comprehensive neuropsychiatric examinations using previously published methods, which included the patient's history, neurological examination, mental status examination, brain imaging, ECG, chest X-ray, and laboratory assessment. The laboratory assessments included chemistries, blood count, liver tests, thyroid tests, rapid plasma reagin test for syphilis, urinalysis, and measurement of electrolytes, serum B12, serum folate, and sedimentation rate." This study was cross sectional and case controlled. Each patient's psychiatric diagnosis as to whether or not they suffered from hallucinations and or delusions was made by a geriatric psychiatrist using the DSM-IV criteria. DSM-IV is the Diagnostic and Statistical Manual of Mental Disorders, fourth edition. The DSM-IV is a book published by the American Psychiatric Association that contains every single mental disorder for both children and adults, as well as how to diagnose them.

The study focused on determining the estimate of delusions and hallucinations in the patients throughout the past two weeks, as well as compare the different groups of AD patients on a set of variables. The patients were classified into four different groups: those who have hallucinations only (Group H), those who have delusions only (Group D), those who have both delusions and hallucinations (Group DH), and those who have neither delusions nor hallucinations (Group NN). "The different groups were compared upon the following variables: sociodemographic characteristics (such as age, gender, race, education), disease variables (such as severity of cognitive impairment, falls, general health status), other non-cognitive disturbances (such as behavioral disturbance, depressive symptoms, aggression, wandering), prior history (personal history of mental illness, family history of dementia), and current medication (such as antidepressants, anxiolytics, antihypertensives)."

The results of the study determined the various factors that caused and did not cause the psychotic symptoms in patients with Alzheimer's disease. It was found that patients who had delusions were older than those who did not have delusions. The patients in Group H were less well educated than those in Group NN and were more likely to be of African American race. They also had more falls and received a lower MMSE score than those in the other groups, which means they had more cognitive impairment. The symptoms of having hallucinations were not associated with marital status, gender, or the location where the patient lives. It was also shown that patients in Groups H and NN both had better general health than those in Groups D and DH. Patients in Groups D, H, and DH had more severe behavioral disturbances, such as bursts of aggression (especially in Group D), wandering and sleep disorders, and more severe depressive symptoms than those in Group NN. There was no correlation between family members having mental illness and the patient having psychotic symptoms. Finally, patients in Groups D and DH were both more likely to be on antihypertensives, and those in Group H were more likely to be on anxiolytics. Antihypertensives are used to regulate high blood pressure and Anxiolytics are used to relieve anxiety.

Overall, about one-third of the patients exhibited some sort of psychotic symptom. Delusions and hallucinations have different patterns of associations. Delusions generally were found in people of older age, who had worse health, depression, aggression, and used antihypertensives. Hallucinations generally were found in those who had less education, severe dementia, were African American, had fallen, and used anxiolytics. Patients who had both hallucinations and delusions had similar associations to patients who only had delusions.

**SOURCE**

INTRODUCTION
There have been numerous cases of patients who still felt the presence of a limb that had been amputated. These so-called “phantom limb” sensations feel identical to the stimulations that people normally feel with their limbs, such as the stroking of their arm by an object. In some cases, these phantom limbs may cause great discomfort for the patients, because their spectral appendage can be twisted into awkward position, and they are not able to reorient it to relieve the pain. This phenomenon is not connected to any sort of brain damage, which has led to several theories regarding its origin, ranging from Freudian explanations to soul-based philosophies. In recent years, a new causation theory has been proposed, which links neural plasticity to this phenomenon.

BACKGROUND
Phantom limbs were first observed in the sixteenth century by Ambrose Pare, a French military surgeon. [1] However, it was in the nineteenth century that an American surgeon by the name of Silas Weir Mitchell, coined the term “phantom limb.” At a time when the localization of brain function was currently being debated, Weir Mitchell often associated this phenomenon to the brain’s representation of the parts of the body (called a homunculus), especially with sensation. [2] Eventually, however, researchers began to classify this condition as a psychiatric illness, and this was the prevailing theory for a number of years. But recently, new explanations regarding the neural mechanisms in the brain and spinal cord have been proposed and offer some empirical evidence to support their claims.

Neuroscientist V. S. Ramachandran’s research and observations laid the groundwork for these new theories. In his book The Tell-Tale Brain, he recounts the insights he gained when he encountered a patient named Victor who felt the sensations of a phantom hand after being amputated below his elbow. [3] Ramachandran was initially quite puzzled at this case, since Victor showed no signs of physical brain damage or mental problems. Acting on intuition, he began poking several regions of the patient’s body with Q-tip, asking a blind-folded Victor to identify where he was being poked. This proceeded as one might expect until Ramachandran came to the left side of Victor’s face, when Victor reported that he felt a sensation on his phantom hand. Repeating this process using various objects on different locations on Victor’s face, Ramachandran created an entire sensory map of Victor’s phantom hand on his face, with each phantom finger having clearly-defined boundaries from one another.

FURTHER EXPLANATION
Since Ramachandran’s research, other explanations that also rely on neurological mechanisms were proposed. [1] One such explanation focuses on the damage done to the peripheral neurons during amputations, which are the nerves located in the limbs and appendages (as opposed to the central nervous system nerves, which are found in the brain and spinal cord). Following amputation, the severed area can form neuromas (usually non-malignant tumors) and within these neuromas, molecules that enhance the opening of sodium channels can be found. These sodium channels are key to neurons’ ability to send signals to one another, and it is thought that these hyperactive sodium channels are responsible for the spectral sensations that are under discussion. Trials that have blocked these sodium channels seem to support this possible explanation.

Another explanation targets hyperactivity in the spinal cord. After amputation some neurons found in this area that are not responsible for pain signaling form connections with areas that do signal pain. When this action is coupled with extreme excitability of other regions (often through an increase in neurotransmitter activity) as well as an expansion of the neuronal
receptive field, it is thought that this process can also fuel phantom limb sensations.

**TREATMENT OPTIONS**

Phantom limbs are also quite unusual in that some patients who have this condition can also appear to move their spectral appendage. Ramachandran explains this phenomenon by comparing it to the difference between imagining a movement and actually performing it. Although a person can imagine moving their arm, the motor neurons in their arm testify that their arm is not actually being moved; however, after amputation, these motor neurons are not there to verify the lack of movement. Thus, phantom limb patients are able to move their spectral appendage by merely thinking about it.

However, the main problem lies when patients have phantom limbs that are paralyzed and are thus unmovable. Often, these phantom limbs are in an awkward position that causes the patients immense pain, but since the limb is static, the patient cannot reorient it to relieve themselves. In evaluating his patients’ charts, Ramachandran noticed that many of these patients had paralysis prior to amputation, and he speculated as to whether the brain had “learned” this paralysis prior to amputation and simply carried it over to the phantom limb. In short, learning is performed through the strengthening of connections between neurons, but when the brain does not sense movement from the motor neurons in an appendage (as is true in paralysis), the connections between the brain and muscles becomes weak. Thus, when the patient had their appendage amputated and consequently developed a phantom limb, the learned paralysis was “carried over” to the spectral limb.

Based on this information, Ramachandran proposed that if his hypothesis was correct, the patients’ pain could be relieved by simply strengthening the brain’s connections. He accomplished this by placing the patient in a box that contained a lengthwise mirror. Ramachandran oriented the mirror in such a way that if the patient stood and extended his amputated stump, reached his unamputated hand out and looked at the mirror’s reflection of his hand, it appeared as if his phantom hand was actually there. Using this, Ramachandran instructed his patient to move his unamputated hand to mirror the feeling of his phantom limb. When the patients did this, their brains interpreted this as motion from their phantom hand, based on visual feedback, and thus the patients were able to stretch their phantom limb that had been stuck in awkward position and relieve their pain. In some cases, when the patients took this mirror box setup home and experimented with it for a couple of weeks, their phantom limb had entirely disappeared!

Although this mirror box method is quite ingenious, it is not sufficient for treating all patients. In such cases, other treatment options are available, such as pharmaceutical drugs. Antidepressants that target various pathways (including sodium channels) and acetaminophen have been shown to have mixed success. [1] Calcitonin, which inhibits the rate at which neurons fire, has also been used. [4] Behavioral methods that attempt to relax the brain have also been implemented in some settings. Surgical procedures, unfortunately, have not shown much success.

**CONCLUSION**

In summary, phantom limbs are unusual phenomenon that are common in patients after amputation procedures. Various explanations of their origin have been proposed but further research will be required to address patients whose phantom limbs cannot be treated with the methods that have been described.
You are staring at a calm, tranquil lake. Suddenly you think of how the lake may be filled with harmful germs that could carry disease. You begin imagining what would happen if the water leaked onto the shore and killed everything in its path. What caused you to think so negatively as to turn this peaceful lake into a natural disaster? The answer is, unipolar major depression.

Unipolar major depression, or clinical depression, is a condition of the brain and nervous system that causes one to lose both interest and pleasure in life. Depression isn’t just a fancy way of saying someone is in a bad mood, rather it is recognized by the American Psychiatric Association as a mental illness that can happen to anyone, anywhere. Furthermore, depression has existed for a long time as Hippocrates, the father of modern medicine, identified it as “sleeplessness, despondency, irritability, restlessness, and an aversion to food” back in 400 B.C.E. Major depression affects 18 million Americans every year, around 6% of the national population. The first round of major depression typically occurs when an individual is between 30-40 years of age, but can also develop in early childhood or in the later stages of one’s life. About 15% of people affected by clinical depression contemplate suicide and roughly 15% of those people are successful. Clinical depression is a real threat to the social, mental, and physical well-being of its victims and scientists are constantly searching for ways to better diagnose and treat this disease.

Symptoms of Unipolar Major Depression

The symptoms of depression range through a variety of psychological, biological, and social categories. The most obvious symptom of depressed individuals is “anhedonia”, or a loss of pleasure in the things they used to enjoy. Other common symptoms include insomnia, loss of appetite, over-consumption of junk food, indecisiveness, overwhelming negative thoughts, lack of ambition or motivation, lethargy, fatigue, and lack of focus. Former British Prime Minister Winston Churchill once described his own depression symptoms as preferring to “stand rather than walk and sit rather than stand”. While many of these symptoms are natural and are likely to occur to everyone at some point in their lives, if these symptoms persist for several weeks or causes an individual to contemplate suicide, it can be recognized as unipolar major depression.

Biological Basis of Unipolar Major Depression

While traumatic life events, alcoholism, and drug abuse may cause one to develop depression,
studies have shown that genetics may play a role in clinical depression as well. If one of your parents or siblings have depression then you have a 20% likelihood of developing it as well. If both of your parents were depressed, you have a 50% chance of becoming depressed. So how do genes cause one to become depressed? Our body naturally secretes stress hormones such as adrenaline, as a “flight or fight” response to emergencies. Adrenaline causes an increased heart rate, increased breathing rate, tightened muscles, widened eyes, and the release of large amounts of energy in the form of glucose. These preemptive actions are our body’s evolutionary responses to danger in our environment, and are collectively known as the sympathetic nervous system. However, these responses cannot continue forever as they use up a lot of energy, so the body secretes other hormones to return the body to a normal state. Such hormones include acetylcholine, and they collectively make up what is called the parasympathetic nervous system. When our brain responds to a large amount of stress, some of its cells become damaged or die but can later be healed by BDNF, or the “brain-derived neurotrophic factor”. Patients with genes associated with unipolar major depression such as Serotonin Transporter Gene 5HTT/SLC6A4 and Serotonin Receptor 2A Gene HTR2A do not produce enough BDNF to heal damaged brain cells, causing them to live with a lot of anxiety, negative thoughts, and stress.

The regions associated with depression are the hypothalamus, pituitary gland, and adrenal glands (where adrenaline is secreted). These regions are what regulate the sympathetic and parasympathetic nervous systems, and secrete steroids such as cortisol and neurotransmitters such as norepinephrine in addition to hormones. These steroids and neurotransmitters flood brain regions that are responsible for thoughts and behavior (frontal lobes), emotions (amygdala), memory (hippocampus), and vigilance (the brain stem). The loss of brain cells in these regions due to the flood of neurotransmitters, coupled with the lack of BDNF, accounts for the many side-effects of depression that affect different aspects of our lives. In MRI scans, neurologists also view the depletion of the hippocampus as a common sign of depression.

**Treatment of Unipolar Major Depression**

Short-term depression, as is the case with many mental illnesses, can simply be treated with a good diet, exercise, abstinence from alcohol and drugs, and talking to a psychiatrist. However due to the atrophy caused to the brain by unipolar major depression, it is often important to take medications called antidepressants. Antidepressants typically help the body restore normal levels of BDNF and prevent the reuptake of neurotransmitters in the synapse. These neurotransmitters are typically biogenic amines such as serotonin, dopamine, and norepinephrine. SSRI’s (selective serotonin reuptake inhibitors) such as Celexa, Desyrel, and Lexapro decrease the reuptake of serotonin in the synapse, while SNRI’s (serotonin and norepinephrine reuptake inhibitors) such as Effexor, Prozac, and Luvox decrease the reuptake of both serotonin and norepinephrine. There are also DRI’s (double reuptake inhibitors) and TRI’s (triple reuptake inhibitors) that decrease the reuptake of two or three neurotransmitters respectively. It is important that you are properly diagnosed before you take antidepressants, because if you have a bipolar disorder, for example, these medications can cause you to develop mania and in the past they have also caused misdiagnosed patients to commit suicide. Like most medications, antidepressants may have harmful side-effects such as nausea, drowsiness, migraine, etc. but these symptoms are usually short lasting and may disappear altogether once your body becomes used to the medication. Your doctor can help you figure out which antidepressant is right for you.

**Looking Ahead**

“Clinical depression is the commonest psychiatric disorder and in the U.S. has the greatest impact of all biomedical diseases on disability.” However, scientists have made major strides in our understanding of unipolar major depression in the past decades, and are inching ever so close to developing an antidepressant that can treat depression universally, without the deleterious side-effects. Till then we will need to be there for those who have unipolar major depression and ensure that in the case of a depressive episode, they receive the appropriate medical attention that they need.

**References**

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What do mRNA molecules and ancient Greek and Latin texts have in common? Well, the answer is that both can be translated by Dr. Lucia Pastorino, a neuroscience professor here at Boston University. Having grown up in Lecco, Italy, it should not come as a surprise that ancient classics were a significant part of her childhood. Whether it was Aristotle, Socrates, Plato, or any renowned ancient philosopher, Professor Pastorino could decipher any of their famous texts. Her passion for Greek and Latin, as well as philosophy, was so great that she focused her classes in these areas throughout high school. There was something about understanding the human experience that was too captivating to deny, and it was this curiosity that eventually led her to study neuroscience.

ACHIEVING A GREATER EDUCATION
When Professor Pastorino began her studies at the University of Milan in 1986, she set herself on a mission to understand humans on a scientific level. Medicinal chemistry was her primary focus in college, and it was the interdisciplinary means of approaching this subject that made it so fulfilling to her. Biology, chemistry, organic chemistry, and pharmacology were a few of her favorite classes among the many science courses she enrolled in while at university. During her time at the University of Milan, Professor Pastorino found her niche scientific passion as she began dabbling in neuroscience. Having fallen in love with neuro, she began work on a peripheral marker in Alzheimer’s Disease. Although Alzheimer’s was bound to become the primary focus of her research years later, she embarked on a Ph.D. thesis on the neuropharmacology of memory and learning. Once Professor Pastorino graduated from the University of Milan with a Ph.D., she crossed the Atlantic Ocean to pursue her neuroscience passion stateside, at The Mount Sinai Hospital in New York City.

LIFE AS A RESEARCHER
Having recently obtained her Ph.D., Dr. Pastorino decided to confront a new frontier of research in the United States. She faced a significant culture shock by moving to New York City, but it was a challenge she welcomed with open arms.

“Chi non risica, non rosica”, she would remind herself.

This Italian phrase roughly translates to “if you don’t push yourself out of your comfort zone, you don’t gain much”. At about 30 years old and having recently moved across the Atlantic Ocean, Dr. Pastorino was about to begin her postdoctoral work at Mt. Sinai, New York. To this day Professor Pastorino can exactly recall her first day at Mt. Sinai- October 4th, 1999.

The research project she undertook was focused on the cellular and molecular biology of Alzheimer’s Disease, and the primary goal of this time was identifying the protein gene responsible for cleaving APP in the amyloidogenic pathway of Alzheimer’s Disease. APP is a molecule that can be found throughout the body, but when present in the brain it can be cleaved by proteases which cause a cascade of events that eventually lead to a buildup of amyloid beta plaques (Haass et al. 2012). Such buildup of amyloid beta plaques is considered to be a major symptom of Alzheimer’s Disease, and therefore an understanding of the enzymes that create amyloid beta plaque buildup would be revolutionary for developing potential therapeutic methods.

A BUMP IN THE ROAD
Yet, just as Dr. Pastorino was beginning her work in researching the protein calsenilin, a mere two weeks after her first day at Mount...
Sinai two research companies published their studies of the identification of the BACE gene which encodes for β-secretase, the enzyme that initiates the amyloidogenic processing of amyloid beta plaques. When β-secretase was identified for the first time, Dr. Pastorino switched her studies to focus on studying BACE, specifically on BACE trafficking which questioned how BACE is dispersed throughout the cell. Such a study would be beneficial for understanding a correlation between BACE’s localization in cells and its toxic properties, as BACE’s location could potentially explain how increased or normal amyloid beta levels come about. Professor Pastorino conducted this research for the following two years. However, in 2001 a group of German researchers published a scientific paper outlining BACE’s subcellular localization. Though this hindered her research she decided to still publish her research on this subject in 2002 because her research explored a few aspects that the German group did not (Pastorino et al. 2002). Having completed this research, she undertook further studies in BACE while at Mount Sinai. This time, her research project focused on whether BACE cleaves other molecules and substrates. Such research was important because therapeutic methods could not be developed without comprehension of the potential repercussions of inhibiting BACE, especially if it was involved in spontaneous processes in the cell. By 2004, Dr. Pastorino had completed her research in this and published her second major paper, this time focusing on how BACE cleaves another substrate, APLP2, in addition to APP (Pastorino et al. 2004).

MOVING TO BOSTON

Although Dr. Pastorino’s second major paper was not published until 2004, in 2003 she moved to Boston to conduct research at Beth Israel Deaconess Medical Center under the guidance of Dr. Kun Ping Lu. Dr. Lu was able to clone a protein called Pin1 which is found to be a notable cause of cancer. Pin1 serves a role similar to a light switch as it controls how a protein domain “flips”, and therefore controlling its activation in cells. As pivotal as this discovery was for cancer, what may have been more intriguing was its relationship with neurodegenerative disorders. Studies had shown that an absence of Pin1 corresponds to the development of neurodegenerative disorders, such as Alzheimer’s disease. Dr. Ping Lu recognized that Dr. Pastorino was an expert in APP and BACE, and he wanted to understand if there is a relationship between APP and Pin1- does a lack of Pin1 effect APP? Although Pin1 levels were known to affect a protein called tau (another protein involved in Alzheimer’s Disease), Dr. Pastorino undertook a research project to understand the relationship between APP and Pin1. Using genetically modified mice that lacked Pin1, she observed phenotypic traits of increased amyloid beta levels and subsequent increases in amyloid beta deposits in comparison to the mice that expressed normal levels of Pin1. This revelation was the primary research that she published in 2006, that being her third major research paper (Pastorino et al. 2006). Based on her previous finding, Dr. Pastorino conducted another related study about Pin1 and APP, though this time to determine the underlying mechanisms between Pin1 and APP. Dr. Pastorino discovered that Pin1 deficiency results in APP localization in similar areas as BACE, allowing amyloid beta plaque to build up in these parts of the cells. Such buildup leads to neurotoxicity within the cell, which tends to result in cell death. This research was officially published in 2012, making it Dr. Pastorino’s fourth main publication (Pastorino et al. 2012). Given that Dr. Pastorino had undertaken multiple studies as the head researcher, she conducted side studies that she co-published in various neuroscience studies. When asked why she decided to dedicate a significant portion of her life to researching Alzheimer’s Disease and BACE, her answer was simple: “as scientists, we are molding the way to knowledge.”

Having recognized how meaningful science is to not only herself but to the rest of the world, it was a no brainer for Dr. Pastorino to find a new career as a professor. While working at Beth Israel Deaconess Medical Center in 2006, she began teaching at Harvard Extension School, the continuing education program of Harvard University. With years of experience in the Alzheimer’s field, Professor Pastorino taught a course in translation research of Alzheimer’s, as well as a course in neurodegenerative diseases. As she concluded her research at Beth Israel Deaconess Medical Center, Professor Pastorino finished teaching at Harvard Extension School. From 2012 to 2013, Professor Pastorino taught at Brandeis University, her final stop before teaching at Boston University.

A LOVE FOR TEACHING

While Alzheimer’s research was what Professor Pastorino spent multiple years conducting, she realized that she wanted to embrace spreading that knowledge full time. When asked why she decided to focus on teaching instead of research she commented that research is, “beautiful but it’s also a trap. Those three nucleotides become your world and there is so much knowledge that you need to put in to understand the protein”. By the autumn of 2013, she moved on from Brandeis to continue teaching at Boston University, where she currently teaches NE 102, a cell and molecular biology course, NE 525, the biology of neurodegenerative diseases, NE 218, an integrated lab section that is focused on the
“science is awesome, and science at the end of the day is an effort, an effort of a community to study the laws of nature”.

THE TRUE MEANING OF SCIENCE

“Chi non risica, non rosica”. This phrase is what kept Dr. Pastorino going, even through her most frustrating moments. The saying embodies the meaning of science to Dr. Pastorino, that risks are a necessity in order to achieve progress. This idea is manifested in how Dr. Pastorino has been willing to sacrifice in order to get to where she is today. From moving to a foreign country to changing research plans in a matter of weeks, Dr. Pastorino illuminates what it means to be dedicated and courageous. When asked what she would change if she could do it all over again, she explained that she would do everything exactly the same. Through trials and tribulations, such experiences have helped to shape who she is and have helped to perfect her abilities as both a researcher and a professor. Research project after research project, Dr. Pastorino recognized her passion for neuroscience and has wanted nothing less than to share that passion with others. Considering all of the ups and downs, all of the meaningful discoveries and adversities in research, Dr. Pastorino summarized the meaning behind all of her work:

“At the end, it is about believing and enjoying, and not giving up.”

REFERENCES


Images courtesy of: Dr. Pastorino and Emme Enojado
Myth V/s Fact

WRITTEN BY: SARAH JEHLE

We have all been there, we were told one thing in school our whole lives only to be told the complete opposite later on. I never know what to believe anymore. Whether it is related to evolution, learning, or anatomy, there are a lot of myths circulating that were created to ease understanding. Myths about the brain or “Neuromyths” as I like to call them are widely believed simply because the brain is so complex and people aren’t sure what is real and what isn’t. Well, I hate to say it, but in some cases neuroscientists do not have a complete understanding of the brain, however for most common neuromyths, there are corresponding facts that may surprise you.

Myth #1: As we learn more information, new neurons are created. So, the more neurons you have, the more you know.

Though it may be easier to say that as we learn new information, new neurons are created which represent that information, it is simply not true. Although the number of neurons we have does have an impact on the extent of behavioral complexities from animals to humans, such as our ability to plan ahead, the number of neurons from human to human does not tell about their individual intelligence. What actually happens when we learn is that the small space between each neuron, called a synapse, develops stronger connections and the myelin or protective covering around the neuron strengthens allowing signals to move faster. As these signals move faster and become stronger, we develop a better understanding and a faster recall of information. Additionally, learning certain things can allow neurons that have never connected before to make connections. Although you can develop new neurons throughout your life from neural stem cells, this does not correlate to a new neuron developing for each new fact learned, thus debunking this common neuromyth.

Myth #2: Cramming in order to learn material before an exam is the most effective way of studying.

I know, sometimes cramming is all we can do in order to prepare for an exam, but if you are really trying to learn the material, cramming is not the best way to go. In fact, cramming is the exact opposite of what you should do. For best performance, it is suggested to space out learning and memorization over a longer period of time, even if it’s only an hour, this gives your brain more time to process the information and make new connections between neurons. As well as spacing out learning, retrieval practice or quizzing yourself is another great way to effectively learn material. Having a large stack of flash cards integrates both of these methods, because there is enough time between each card to make connections and you ask your brain to retrieve information with each new card. So cram if you have to, but giving yourself time and practice will ultimately allow you to better learn material rather than just placing it in your short term memory only to be lost an hour later.

Myth #3: We only use 10% of our brains on a daily basis; the more you use, the more cognitive abilities you have.

Wouldn’t it be great if we could access 100% of our brain and have superhuman powers like Lucy (Scarlett Johansson) in the 2014 action thriller Lucy? Well, unfortunately we already use almost 100% of our brain, and so far there are no super human abilities. This myth has been held in discussion for so many years, because it convinces people to work for more “brain power” by creating the desire to access that other 90% of their brain. However, if this were the case then neurosurgery would be much easier than it is now because they would only have to worry about 10% of the volume rather than a full 100%. Additionally, from an evolutionary point of view, we wouldn’t have developed the size of brain we did if there was no advantage to it. So, while it would be more fun to have 90% of our brain untapped, it would not be practical based on the amount of selection that went into forming the complex brain tissue we currently have. Despite this, I urge you to keep striving to access more, but I assure you it will not lead to your ability to read minds or fly. Or at least not right now.

Myth #4: There are left-brained and right-brained people, and this makes each type of person good at different things.

Hopefully, you didn’t choose your occupation or major based on the fact that you were labeled as left brained or right brained in the 4th grade based on a ten question quiz. Although there are correlations to specific attributes in each hemisphere, these attributes are not localized only to the left or right side. This means that no one is left brained or right brained; rather, each person is an equal mixture of both hemispheres. It is true that some people are more analytical and logical while others are more artistic and emotional, but it has nothing to do with how dominant your right or left hemisphere is. For the most part, both sides of the brain normally work together with the help of the corpus callosum. So my best advice is not to treat some quiz you took on Buzzfeed as a self-fulfilling prophecy that you should choose being an artist over being an astronaut. Instead look at what you are actually passionate about and go from there. Let the right and left hemispheres of your brain work together as they should.

Neuromyths like these tend to fill our biology and psychology classes in high school, but don’t believe everything you hear. It is important to debunk these myths so we can more accurately represent the brain and its function to our society. If we continue to tell these tales, we may have another generation full of kids comparing neuron numbers and hemisphere size, or trying to stimulate the other 90% of their brain in order to fly.

References:
This idea, the premise of director Luc Besson’s 2014 sci-fi action-thriller, Lucy, imagines possibilities of extraordinary proportions for the future of humankind if we could somehow access the abundant reserves of untapped gray matter our brains allegedly possess. Such a prospect holds obvious appeal, but seems rather unattainable—even more so when audiences realize it is rooted in falsity. Functional brain imaging technologies have allowed researchers to confirm that activity flows through the entire organ at all times; after all, the adult human brain accounts for approximately 2% of the body’s total weight, but consumes a whopping 20-25% of its total energy intake—hence, it is implausible that any part of it would simply remain inactive, waiting to be discovered and, at last, utilized. Accordingly, extensive research on neuroplasticity and somatosensory reorganization has demonstrated how swiftly the brain employs all available tissue.\(^1\) Consider a 2013 study led by cognitive neuroscientist Tamar Makin. Using magnetic resonance imaging (MRI), Makin illustrated that, following amputation the neural tissue associated with the missing limb is instinctively recruited by neighboring brain regions to represent other body parts.\(^2\)

Like all other organs, the modern human brain is a product of natural selection. As neural tissue is metabolically expensive, it is highly improbable—if not wholly impossible—that any part of the brain was, at any point during the evolutionary process, underutilized or altogether neglected. Electrical stimulation of targeted brain regions during neurosurgery—performed on conscious patients under local anesthetic, as the brain itself has no pain receptors—has yet to discover areas that fail to provoke an emotional, motor, or somatosensory response when stimulated.\(^3\) As Professor Barry L. Beyerstein of the Simon Fraser University Brain Behavior Laboratory explains,

Lost far less than 90 percent of the brain to accident or disease has catastrophic consequences. What is more, observing the effects of head injury reveals that there does not seem to be any area of the brain that can be destroyed by strokes, head trauma, or other manner, without leaving the patient with some kind of functional deficit.\(^1\)

Thus, it seems strange that Besson would strive for a scientifically plausible storyline when, in fact, there is no scientific evidence to support the notion on which his film was based. Despite a vast body of evidence demonstrating that the entire brain of a healthy individual operates at all times, the “Lucy” myth has somehow persevered.

The persistence and prevalence of the 10-percent myth piqued the interest of neuroscientist Suzana Herculano-Houzel, a professor of psychology and biological sciences at Vanderbilt who, in 2002, surveyed over 2000 college-educated individuals to reveal that over 60 percent of this population believed humans use only 10% of their total brain capacity.\(^2\) Herculano-Houzel decided that, in order to ultimately debunk the myth, she must first determine the exact number of neurons the human brain actually contains. So, she turned to scientific literature.

Unfortunately, the neuroscientist’s quest for answers seemed only to reveal an anthology of preponderant misconceptions. Among these was the assertion that the human brain contains approximately 100 billion neurons—an estimate based solely on orders-of-magnitude extrapolated from certain areas of the brain that, in actuality, was supported by virtually no viable scientific evidence.\(^3\) Additionally, many scientists believed that all mammalian brains were built in the same fashion, with a number of neurons directly proportional to the size of the organ. Pursuant to this principle, one could conclude that any two mammalian brains of the same size possess roughly the same number of neurons and therefore retain similar cognitive abilities. Herculano-Houzel references this assumption in her 2013 TED Talk, comparing the brain of a chimp to that of a cow. While these mammalian brains are approximately same-sized, it is quite evident that chimps are capable of far more complex behaviors. Herculano-Houzel’s example therefore disproves this theory.

In response to her discovery of these alarmingly prevalent scientific fallacies, Herculano-Houzel developed her own method of counting neurons. After stumbling upon roughly 40-year-old papers describing efforts made by researchers to dissolve brains into a liquid “soup” to measure their DNA concentration, she speculated that this method could be modified to liquefy cell membranes while leaving the nuclei intact. This meant that a scientist could theoretically count the nuclei in a small tissue sample, then multiply that value by the total volume of liquefied
brain to determine neuron density.\(^3\) If all brains were identical in composition, larger brains would, as a rule, contain more cells than smaller brains—and, accordingly, cognitive ability would increase indefinitely with increasing brain size.\(^3\) In such a case, the world’s largest brain should be the most cognitively able. While the average human brain weighs between 1.2 and 1.5 kilograms (2.6 and 3.3 pounds), the average elephant brain typically weighs 4 to 5 kilograms (8.8 to 11 pounds), and whale brains often weigh up to 9 kilograms (19.8 pounds).\(^3\) For this reason, scientists at some point concluded that the human brain, with its uniquely enhanced cognitive abilities, was simply yet inexplicably an exception to the putative “brain size dictates intelligence” rule.

Furthermore, the human brain-to-body ratio exceeds that of other primates; while gorillas tend to be two to three times larger than the average human, the human brain is roughly three times the size of the gorilla brain. In addition to its large size relative to the body, the human brain is unique in its consumption of energy, as approximately 500 of every 2,000 calories consumed by the body are used to fuel just the brain.\(^2\)

These disproportions and the corresponding conclusion that the human brain is simply “special” frustrated Herculano-Houzel, who consequently began entertaining previously untouched questions regarding the precise neuronal architecture of the human brain. The neuroscientist used her neuron-counting technique on the brains of dozens of species and was able to confirm that all brains are not, in fact, built the same way. She discovered that, as the rodent brain increases in size, the average size of the neuron increases as well; that is, a positive correlation between brain size and neuron size was observed. In contrast, larger primate brains possess a greater number of neurons, rather than a constant number of neurons that simply grow in size; in other words, a positive correlation exists between brain size and neuron count.\(^3\) According to this distinguishing brain size-to-neuron count relationship, a primate brain will, by nature, contain more neurons than a rodent brain of the same size.

What is more, Herculano-Houzel determined that the average human brain actually contains 86 billion neurons, 16 billion of which reside in the cerebral cortex. As this region of the brain can be considered the epicenter for sophisticated functions such as attention, producing and understanding language, awareness, and abstract reasoning, Herculano-Houzel hypothesized that great apes, who are as much larger than we are, do not possess larger brains with greater numbers of neurons. As the energetic cost of the brain is directly proportional to its neuron count, Herculano-Houzel hypothesized that great apes simply could not afford the energy required to power both a large body and a large brain with a large number of neurons. The neuroscientist determined that, because neurons are so metabolically expensive, such a tradeoff must exist. She calculated that a primate spending an average of eight hours per day feeding could afford a maximum of 53 billion neurons, with a body weighing no more than 25 kilograms (55.1 pounds).\(^3\) Hence, to weigh any more than this, it must relinquish a fraction of its neuron density. In theory, a large primate could not only afford to possess a body weighing 86 billion neurons (2.6 pounds) which, as previously mentioned, falls within expected bounds; thus, the human brain is simply a relatively large primate brain. Its overall number of neurons is not what makes it remarkable.

The essential question left to be investigated, then, is why apes, who are as much larger than we are, do not possess larger brains with greater numbers of neurons. As the energetic cost of the brain is directly proportional to its neuron count, Herculano-Houzel hypothesized that great apes simply could not afford the energy required to power both a large body and a large brain with a large number of neurons. The neuroscientist determined that, because neurons are so metabolically expensive, such a tradeoff must exist. She calculated that a primate spending an average of eight hours per day feeding could afford a maximum of 53 billion neurons, with a body weighing no more than 25 kilograms (55.1 pounds).\(^3\) Hence, to weigh any more than this, it must relinquish a fraction of its neuron density. In theory, a large primate could free itself from these metabolic constraints by spending more time ingesting food each day; at a certain point, however, such behavior would surpass a practical limit—nine hours of feeding per day seems to be a feasible upper bound for the average primate.\(^3\)

Supposedly, then, humans would need to spend over nine hours per day feeding to account for a brain comprised of 86 billion neurons in a body weighing 60 to 70 kilograms—which, needless to say, they do not. With our disproportionately expensive brains, the only explanation for this newfound “exception” is that we are somehow able to acquire more calories in less time. Such a notion introduces another important point: while humans are primates, they do not eat like other primates. Approximately 1.5 million years ago, our ancestors invented cooking. The use of fire to pre-digest foods outside the body makes the foods easier to chew, while raw food requires more work (i.e., calories) for muscles and organs to chew and digest. This results in a net decrease in the number of calories available; hence, cooked foods provide more energy in less time.\(^3\) In turn, the human brain is given more time to engage in other activities—to conceive culture and civilization—all the while remaining a characteristically primate brain.

In summary, our exceptional cognitive abilities can be attributed to advancements made by early humans. Besson imagines a world in which modern humans have furthered this process of cognitive enhancement by accessing supposedly inactive brain regions while, in reality, we have already done so simply by exercising our ability to innovate. For this reason, dreams of expanding our cognitive potential need not be extinguished simply because the 10-percent myth has been scientifically debunked—the human brain has always been extraordinary.
A frail, elderly woman sits alone in her room. Holding a wooden pencil in her nimble, wrinkled hands, she scribbles away on some papers. She occasionally stands to fix her curtains, fold the sheets, and pour herself a glass of water. The blaring sound of the television fills the air for everyone but her, for her weak hearing only allows the equivalent of a mere vibration. People visit throughout the day, assisting her with daily tasks. “Thanks for stopping by my home,” she says to each of her medical team members, including myself, after rambling on about life stories and random events. I smile and make humor of the situation. Does she really think that we are in her own house?

“You are in the hospital,” we repeat aloud, only to receive a denial in return. Despite our numerous attempts to remind her of this fact, she remains oblivious, so we tend to play along. She repeatedly mistakes us for people from her past: the elderly woman who lives next door, the milkman who stops by on Wednesdays, or the dog walker at the end of the street.

She usually mistakes me for the teenage boy from the adjacent neighborhood, but I am actually part of her nursing team, working in the medical-surgical unit of my local hospital. We see everything from minor surgical injuries to general medical conditions, with a large population of mental health patients. Most medical staff dislike working on the mental health floors, as the patients are usually hard to connect with on a deeper level. Medical professionals have to work much harder to form genuine connections and often have to deal with deep-wired psychological breakdowns. I enjoy the challenge of trying to “crack the code” of each patient, learning more about them and their lives.

Each passing hour is an entirely new experience for our patient. As her memories fade, interacting with the same nurse is like meeting dozens of new people. Prepared to be mistaken for someone our patient knew, the nurses enter the room numerous times during their twelve hour shifts, inspiring both happiness and sometimes fear. An entire conversation goes by, twenty minutes of in-depth interaction, just to be forgotten the second they leave the room. Are her daily interactions really significant? It feels good to keep her company, but that is overshadowed by the fact that she will forget that any of us were in her room, made her coffee, or helped her eat dinner. Throughout the rest of the day, more people enter the room only to exit confused and disappointed—a sure sign of failure to convince her of who they really are. During the process, her face turns gloomy and an oblivious smile turns to a frown. “What do you mean?”
she often repeats, completely unaware of her dementia. I can see the frustration and obvious distress on her face when she cannot find a way to express herself.

We see our patient’s children and grandchildren walk somberly down the hall. I often form connections with the family, as we see each other almost every day. I talk with them about their son’s job promotion, grandson’s baseball games, and sister’s new shepherd puppy, but I do not know much about my own patient. However, when the family enters her room, she has no recollection of them. Her husband is just a blurred memory and her grandchildren are complete strangers. I watch as they sit with her for hours trying to explain who they are, but eventually accept that she will not understand. Their mother, wife, and grandmother has no recollection of them or the lifetime of memories that they made throughout the years. Even though this is very typical for dementia patients, each patient is unique and experiences can differ dramatically from case to case.

The next time I enter the room, I see her slip a piece of wrinkled notebook paper into her bedside drawer. I curiously ask what she has been working on for hours, but she is hesitant to share. “Oh, nothing” she replies, causing my curiosity about the scribbled papers to grow. Eventually she gives in and translates her scribbles into vivid stories about everyone she has met throughout her stay, mistaken as people from her past. The papers shake in her hands while the tone of her scratchy voice fluctuates with each word. Her failing mind has not yet stopped her from using one form of communication: poetry.

As I realize that the seemingly illegible handwritten notes tucked deep inside her bedside drawer do have profound meanings, I discover that she is much more observant than I originally thought. Although she cannot recall recent events, recognize us as her care team, or hold verbal conversations for more than twenty minutes, she is able to write descriptive stories about her past. She writes poems about everything; nature, her childhood, and eventful life stories.

She pulls a stack of papers out of her drawer and proudly reads one poem after another. As word spreads about our patient, more care team members pile into the room to listen. This audience grants her the newfound confidence to narrate her stories aloud. Although she reads the same stories numerous times, we still clap for her as each reading ends, while she grins and asks, “well, how did you like that one?” We each leave the room with a smile, and I find myself amazed at the fact that my dementia patient writes better poetry than I ever will.

Later that night, while listening to her enthusiastically read back her poems to us, her nurse and I realize that she actually does remember her family. We listen attentively as she reads poem after poem with subtle cues about her hardworking father in the steel mills, grandchildren playing along a stream, and beloved caregiving sister. Her capacity to write is far less damaged than her ability to convey spoken memories. The dementia left her written language virtually untouched! It became our mission as her care team not only to heal her but to heal her family as well. As she continues to read her stories aloud, we transcribe them onto hospital printer paper. The medical team realizes that she does want to convey her thoughts and memories and just needs a little help.

The next day, as the family members walk in, ready to take on another emotionally draining visit, we greet them with the notes. She never shares her poetry with them, so they are not able to unlock the memories in her vault. Her husband says that she has always loved poetry, but he had assumed that the poems diminished along with her memory, though she had just been hiding them for reasons unbeknownst to us. She usually tucks them into nearby crevices and allows them to disappear along with her memories.

Later that day while sitting down and organizing her poems, I make out the word “nurse” and hand over the paper, asking if she can read it aloud. Although most of her stories are about her family and childhood events, this poem is about one of her nurses, Tracy. Tracy spends three consecutive days in the patient’s room, listening to stories and caring for her. She describes her long blonde hair, warm hands, and sweet, motherly voice. From this we can deduce that our patient does understand parts of her hospital stay after all and even sees parts of herself in Tracy. Our patient seems to connect with Tracy and the other nurses more than anyone else.

The poem describes our patient’s own life and former career: a nurse. She began to share more detailed poems about her experiences as an Army Nurse in World War II and the heroic actions she took part in. Dodging bullets, flying in helicopters, and hiding in trenches, our patient eagerly conveys the horrors of her youth. Her husband explained that she commanded a medical brigade on the war’s front lines, caring directly for injured soldiers, some of who might even be in the hospital room right next to hers. He assures us that even though the repeated blast injuries and horrors of war probably lead to her debilitating dementia, she would never trade her nursing experience for the world. As a young woman, she sacrificed her own life and future memory for her country and fellow Americans. I realize that my patient is much more than the confused elderly woman I met when I initially entered the room. She is an amazing poet and truly inspirational healthcare provider.

“Perhaps our lack of understanding is simply a lack of observation.”

This experience gave me a deeper understanding for my dementia patients. Many healthcare workers, including myself at times, fail to connect with them, as we assume that they will not remember us anyway. We make superficial conversations and talk as if they are incompetent, but that is far from the truth. Perhaps our lack of understanding is simply a lack of observation.

Just as our patients have trouble fathoming our explanations about the “real world,” we do not fully understand what is going on in their heads. As their minds fade, they convey their thoughts, emotions, and memories through different, less obvious forms of communication. My patient had found her memories, her voice, and a way to reconnect with her family with her silent, forgetful poetry.
A year after his arrest, he was found not criminally responsible due to mental illness. Again, he was diagnosed with schizophrenia.

These are two of the many examples that demonstrate the link between neuroscience and law. The first example is based on the M’Naghten rule. This insanity test checks if the criminal is able to understand the nature or quality of the act that he or she commits. If the perpetrator is not able to understand, the test suggests that this situation is due to a mental illness. As a result, the government decides that said person should go to a center of mental health. Even though this seems like a simple system, it brings up many questions, some of which will be discussed in this article.

One of the famous debates is the conflict between nature and nurture and its application to law. As a society, we try to generalize the practices of law and put it into a strict pattern as if we can identify the root of human behavior. On the contrary, the interactions between genes and the environment are so complex that it is difficult for us to successfully understand them. I pointed out this term here because law is a social contract between individuals in public that arises from human interactions. We are our brains; we are our biology. We change as our biology changes.

We try to generalize the practices of law and put it into a strict pattern as if we can identify the root of human behavior.

If the mind is only a manifestation of neural signaling, how much control do we have over our thoughts and actions? In other words, is free will an illusion? When we make a mistake based on our actions, is it our fault or our biology’s fault? Based on research, neuroscientists can come up with symptoms of mental illnesses. For example, Diagnostic and Statistical Manual of Mental Disorders is a product of psychiatric and neuroscientific research and it is updated with new data. How accurate are these representations? These are just labels, stereotypical models of illnesses. Each mental illness has its own checklist which shoves individuals into boxes while categorizing them. A four-year-old kid is diagnosed with bipolar disorder because he has temper tantrums. This action is nothing but a simple check on that checklist. The fact that temper tantrums are common in toddlers should...
not be ignored in this case. One physiological reaction may have more than one psychological result. For example, anxiety and anticipatory excitement create the same physiological outcome in an individual. It is not right to jump to conclusions while paying attention to only one symptom or the chemical reaction. Instead of jumping to conclusions, we should expand our minds and our checklists and consider the social aspects of a symptom. We have to understand how people actually are and how they actually act.

One other debate is about the usage of drugs used in symptomatic treatment. Even though they are possible cures for neurodegenerative diseases, these drugs affect the nervous system while changing the quantity of the neurotransmitters which are molecules essential for communication between neurons. Therefore, they change the behavior of the patient. For example, Parkinson’s disease is caused by low levels of dopamine, the neurotransmitter present in the reward system of the body. The patients of Parkinson’s disease are treated with “levodopa (L-dopa)”, the precursor to dopamine. During treatment, it is hard to maintain the sufficient level of dopamine in the body. That’s why the graph (see figure at the end) representing the dopamine levels of a patient on L-dopa shows a sinusoidal pattern. When the dopamine quantity in the brain reaches the peak value, and starts to decrease again, the body wants more. As a result, the patient starts to lean towards actions from which he derives pleasure such as gambling, alcohol, drugs, sex and so on due to the non-specific effects of adding dopamine to the brain. With these scientific facts in hand, is it right to use these drugs to treat patients? Is it right to judge similar patients as we judge a regular individual?

One other debate is about the usage of drugs used in symptomatic treatment. Even though they are possible cures for neurodegenerative diseases, these drugs affect the nervous system while changing the quantity of the neurotransmitters which are molecules essential for communication between neurons. Therefore, they change the behavior of the patient. For example, Parkinson’s disease is caused by low levels of dopamine, the neurotransmitter present in the reward system of the body. The patients of Parkinson’s disease are treated with “levodopa (L-dopa)”, the precursor to dopamine. During treatment, it is hard to maintain the sufficient level of dopamine in the body. That’s why the graph (see figure at the end) representing the dopamine levels of a patient on L-dopa shows a sinusoidal pattern. When the dopamine quantity in the brain reaches the peak value, and starts to decrease again, the body wants more. As a result, the patient starts to lean towards actions from which he derives pleasure such as gambling, alcohol, drugs, sex and so on due to the non-specific effects of adding dopamine to the brain. With these scientific facts in hand, is it right to use these drugs to treat patients? Is it right to judge similar patients as we judge a regular individual?

According to David Eagleman, we should bring the practice of customized rehabilitation and rational sentencing to the legal world. We should treat suspects individually, with neural images in hand and we should not choose incarceration as a blunt solution. Based on the results of fMRI and MRI scans, we should determine a reasonable sentence for this criminal. With similar technologies, we can stimulate the decision-making areas of the brain and while using the past results of other criminals who committed the same crime; we can compare and decide what to do with the current criminal. As we solve legal problems with rehabilitation, we are preventing human misery and expenses that worth trillions of dollars. Our goal has to be biological understanding.

Sources
I walk into the hospital at 5:00am, before the sun rises and people flood the halls. I swipe my badge and push the large wooden doors that open to the Neuroscience Critical Care Unit (NCCU). The lights are dimmed and an eerie silence sits over the unit. At first glance, the halls are idle with patients who are sound asleep in their beds, an occasional buzzer ringing in the distance. However, the inside of the unit’s conference room brings an entirely different environment. Neurosurgeons and neurocritical care physicians are debating difficult cases, residents are barely holding their heads up while gripping their steaming coffee cups, and medical students are aimlessly scribbling notes with anxious looks on their faces. Side conversations start dissipating as the lead neurological resident recounts each case to the team. He mostly uses medical acronyms and abbreviations, which sound like an alien language to any critical care outsider. The lights go dark as the resident flips through countless X-ray and CT scans while deciding how to diagnose each patient. However, just as fast as we all sit down, we all jump to our feet and, like a giant mob, make our way down the winding hallways. "Keep up or they’ll leave you behind," a resident whispers to a struggling medical student. We round in teams of ten to twelve practitioners before breaking into smaller groups to investigate each individual patient’s situation. Each room has a different story to tell and a plethora of different emotions contained within its large, glass doors.

We decide to work backwards from room four to one. Our team enters room four to speak with a patient’s family about end-of-life care and decisions. This patient has suffered from a massive ischemic stroke, thus most of her anoxic brain tissue has died. I see her chest mechanically inflate and fall as a ventilator pushes air into her lungs. I hear the dripping of the IV and see fluid rushing into her veins. The EEG scan is absent of meaningful brain activity. As the family sits around her and talks about how to proceed, their...
teardrops fall to the ground. They are torn between letting go of their beloved family member and holding onto the hope of an improbable turnaround.

We move onto room three. It appears calm and collected at first glance, but the NCCU always has an unsuspecting surprise waiting to be revealed. A critically ill patient begins to rapidly decompensate. Nurses and physicians rush into the room as alarms sound while a code blares over the loudspeaker. A resident launches the CRASH cart into the room with determination in her eyes. Chairs and bedside tables are thrown out into the hall, allowing NCCU staff to flood the room. They begin chest compressions, assist the patient’s ventilations, and apply the AED pads to prepare for a shock. To an outsider, this would seem like a blur of disarray. In reality, everyone takes action in their given role within an organized chaos.

“It’s the brain bleed,” a neurology resident shouts to the team.

Hypertonic saline solution and a cocktail of medications begin to run into his veins. Shocks are applied to his chest. A heavy silence follows as the staff search for any sign of life. Miraculously, a pulse appears and every held breath is released in a sigh of relief. The operating room (OR) team rushes him off to emergency neurosurgery and a sense of calm spreads throughout the unit.

The situation dissipates just as it arose. We all take a moment to pause and look around the room. Medication bottles, gloves, gowns, and equipment are strewn across the floor, as if a tornado passed through the room.

Some alarms continue to sound as the staff glance at each other in shock. They must be prepared for these unpredictable circumstances: a patient can crash in a matter of seconds.

We continue our rounds and move onto room two. The team enters the room to see the patient and his family sitting nervously before his surgery. A large meningioma is battling for space within his brain tissue, causing severe paralysis and partial loss of speech and sensation in his body. He suffers from recurrent seizures due to the brain tumor and needs surgery to restore function and save his life. The patient stretches his thin, blue surgical cap over his head, hugs and kisses his family, and is wheeled down the hall through the double doors in the hands of a complete stranger.

The OR is dimly lit, with plain tile walls and shiny metal equipment. The entire team wears the same baby-blue scrubs, masks, and surgical caps, but the surgeon stands out from the group. She stands confidently at the stainless-steel sink, scrubbing her hands with a soapy betadine solution, and peering through the small window into her OR room, watching her patient enter his artificial, yet sound sleep. I watch as a human life is placed in the hands of another.

Our last room is number one, but unlike all previous rooms, I hear cheers and laughter as I make my way through the doorway. A lone wheelchair waits outside of the door, abandoned by its intended passenger. “You’re going home!” the neurocritical care intensivist beams as the family celebrates. “Get-well-soon” balloons float in the air and rainbow colored flowers sit on the bedside table. The young patient had been shot in the head three weeks prior, and both family and physicians were unsure if she would survive. “When this first happened, they told us to get ready for the end...we said our goodbyes,” the patient’s mother says. The bullet had grazed the top-left side of the patient’s brain, leaving her with diminished motor function and weakness in her right arm and leg. Despite this, the patient always vowed that she would walk herself out of the hospital...and so she does. She would never let a bullet halt her life, nor a wheelchair hinder her dreams.

In just a single day, the NCCU witnesses an uncertain future, an inevitable death, the heroics of lifesaving action, and the miracles of neurological medicine at work. “We are here for the most critically sick and injured neuroscience patients. It is what we are trained to do and what we love,” says the neurocritical care attending physician. She smiles and adds, “We see it all, everyday, on the front lines of neuroscience.”
Santiago Ramon y Cajal is honored as the father of neuroscience. Cajal is known for his study and documentation of the brain in great detail and his use of art to capture the brain's complexity. The level at which he was able to study the brain is awe-inspiring, especially since he didn't have the assistance of the neuroimaging technology that is often used today. For his work with the brain and the rest of the central nervous system, Cajal received the Nobel Prize in Physiology in 1906 along with Camillo Golgi (3).

Cajal’s Artwork

Eighty of Cajal’s pieces are currently on tour across the United States under the name The Beautiful Mind. Originally organized by the University of Minnesota and the Weisman Art Museum, Cajal’s artwork is currently at the MIT Museum as of May 2018 until December. The current selection was chosen from among thousands of Cajal’s drawings from the Cajal Institute in Madrid, Spain. The Cajal Institute records and stores all of Cajal’s artwork, and continues his studies in neurobiology (2).

In conjunction with Cajal’s artwork, the MIT museum has other visual imagery using modern technology to compare to Cajal’s work. Many of Cajal’s drawings have been closely matched with images obtained from modern technology, including MRIs.

Cajal's training as an artist is evident in the detail of his work and his use of art techniques, like cross-hatching and shading, to bring a real-life depth to his work. He perfectly blended science and art in order to take a major step forward in our understanding of brain anatomy and function as we know it today.

Methods

How was Cajal able to depict individual neurons in the brain with so much detail without the neuroimaging techniques that we rely on so heavily today? Cajal was able to study a variety of microscopic features with the use of histological staining and a high-powered - for that time - microscope (1). Histology is a method used widely throughout the biological sciences where organisms or parts of organisms are stained or marked with certain chemicals so that they are easier to observe under a microscope. One of the histological staining methods Cajal used most frequently was the silver nitrate reduction method, where tissue cultures are exposed to silver nitrate and then a strong light, causing the silver nitrate to reduce. Cajal invented this method by building on the Golgi Method, which allowed...
him to study the interior of cells in the nervous system, especially when placed on a yellow background (1). The majority of his histological work used variations of the silver nitrate method or Golgi method, since he was heavily focused on neurons and the nervous system.

**Major Contributions to Neuroscience**

Cajal studied many aspects of the brain. He toured neural pathways, visual and auditory pathways, as well as the inner workings of neurons and neurodegeneration. He famously proposed the Neuron Doctrine, which stated that neurons were individual units that together composed the brain. This idea was in conflict with the Reticular Theory, the accepted theory at the time, that described the brain as a singular network of physically connected cells, ie a net.

Cajal's Neuron Doctrine forms the basis of the modern study of neuroscience, but his contributions to the field did not stop there. As seen in the “The Beautiful Mind” exhibit, Cajal explored everything from our sensory systems to brain pathology, including an exploration of different diseases and how the brain degenerates after death. Driven by the Neuron Doctrine, Cajal studied certain brain areas more deeply, beginning with the cerebellum, a brain area that is crucial for the coordination of movement and balance.

Using histology, he began to unravel how neurons function. Cajal even postulated that neurons grew in length in correspondence with chemical signals (chemotropism), something that would not be proven until much later (5). He also described the Law of Dynamic Polarization, which explained how individual neurons might communicate through the polarization of the cell and what direction this flow traveled in. He used his knowledge of the visual system to establish which direction polarization might occur.

Cajal made progress in the fields of pathology and in understanding how the brain functions after injury, something applicable to neurodegenerative diseases like dementia. He studied how the brain recovers after traumatic brain injuries and observed the degeneration of cells in animals with traumatic brain injuries. He found that after a few days new fibers began to grow, suggesting that the nervous system has some regenerative capacities. This was observed in several parts of the nervous system including the cortex (the outermost covering of our brain responsible for logic, reasoning and combining stimuli) and the optic nerve (responsible for transmitting visual information from eyes to different parts of the brain), indicating an ability to heal after sustaining injury; this has implications for many neurological diseases and foreshadowed the idea of neuronal plasticity (7). Cajal predicted that the prevention of the natural decay of neurons would be the impetus of future science. In many ways, he was correct in assuming so, as we are in an era where more and more people are being diagnosed with neurodegenerative disorders and scientists are looking for a way to halt or reverse this progression.

Despite the time of his discoveries and the limitations of his technology, Cajal was far ahead of everyone else in his understanding of the basic mechanisms of neuronal function, which have been confirmed in the modern era of neuroscience. Although he is mostly known for the Neuron Doctrine, he proposed many theories that have outlived him and withstood the test of time. He truly deserves his title as the Father of Neuroscience.

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The Snatcher: A Poem about Alzheimer's

WRITTEN BY: PAYTON CABRERA

Gawky thoughts turned into frustrations, you sat patient, awaiting a truth that would turn an already difficult world upside-down. Despite protests, he came to your door.

He was like a long lost relative, annoying, yet manageable. He took the space better left alone and made it inconvenient, he knew everything about you and made sure you slowly lost it all. They called him The Snatcher.

At first, he was simply an unwelcome resident, splaying his things across a mind that could hardly keep up as it was. Your thoughts turned clunky while he stayed, a clear mind blocked by his clutter.

But still, your life was so beautiful. You knew that. You remembered your highschool sweetheart, a life forged with love and dedication. You remembered every panic attack and laugh attack and the struggles that molded a life worth looking back at.

Words spilling at the tip of your tongue held back by teeth clenched in frustration. You knew his name, your stubbornness refusing to give up the fight against his settlement. He was good at stealing your words. But soon, he grew, an evolving beast latching tighter to the memories once held so sweet. The Snatcher took over your mental real estate, evicting the history of love and joy and replacing it with his own mess. Muddled messages lost in translation, you deserved all of the clarity that was taken from you. The panic of your passing overwhelmed by the Snatcher's terrors.

Kept in a home, you held on to a life no longer your own in a house you could no longer remember. But you deserved a lifetime of knowing your daughter's face, of growing old with your sweetheart, and of remembering every person worth never forgetting.

There must be another side to this, you just aren't there yet.

Intertwined: A Poem about Synesthesia

WRITTEN BY: PAYTON CABRERA

ART BY: AMANDA FORTIN

6 was afraid of 7 because 6 is a little boy with trust issues. 7 may have 8 9, but 9 was a crotchety old man who had it coming. Mondays felt silver, but more of the dull shine to catch your eye while walking down the street than anything worth paying attention to. Everyone thought my crooked perspective was some poorly-thought-out joke, but you thought it was special. Your name tasted like oranges. You reached for my hand and suddenly I was intertwined in more than just my mind.
“It is known that music strengthens emotional and cognitive functioning in both healthy individuals and clinical patients.”
Germany has one of the most efficient and successful sewage systems in the world. However, even Germany’s waste management department cannot build anything nearly as intricate and successful as the sewage system that exists in the human body. The body’s so-called sewage department is known as its lymphatic system. The lymphatic system is composed of vessels that are parallel to the blood vessels and run throughout the body. The collection of vessels, along with a clear fluid called lymph, the fluid found in the lymphatic system, performs many essential immune functions, such as transporting and deploying white blood cells to fight infection. Most importantly, it filters fluids through lymph nodes—such as the tonsils or the spleen—and disposes of bodily toxins by draining fluid around the cells, thus preventing infections or imbalances.

Certainly, scientists have known about the lymphatic system and its functions since the 17th century. However, until now, any practicing doctor would vehemently deny that this system existed inside the brain. It was previously thought that the brain had no connection to the lymphatic system. Instead, scientists believed that all of the brain’s waste was drained into the cerebrospinal fluid. The existence of structures such as the dura, pia, and blood-brain barrier seemed to support the belief of the complete separation of the brain’s immune system. Yet, in a recent study conducted by the U.S. National Institute of Neurological Disorders and Stroke in 2017, researchers discovered that the brain does indeed contain lymphatic vessels. These vessels had previously gone undetected in the brain, since they were hidden inside the nervous system’s outermost, thickest protective covering—the dura mater.
After injecting dye into the brain’s blood vessels in humans and nonhuman primates, scientists used MRI to view these vessels in the dura. The dye leaked out of the blood vessels as waste and was collected in the lymphatic vessels, which was then displayed as white areas in the MRI images. These images, along with extremely complicated physical calculations, allowed the research team to find the precise location of these key vessels in the brain.

Although the importance of these findings has not yet been fully realized, they have critical implications for the future of neuroscience. Since the lymphatic system is responsible for immune system activity, it plays a crucial role in the inflammatory process, one of the body’s main response mechanisms towards infection. This inflammatory process is thought to affect, or even cause, many neurological diseases. For example, Multiple Sclerosis is a disease that affects sight, memory, vision, and motor skills, and is caused by “rogue” immune cells attacking and inflaming the myelin sheath surrounding neurons. The electric signals are now disrupted as they move down the neuron. Alzheimer’s, Parkinson’s, and aging are all thought to have ties to chronic neuroinflammation (brain swelling). Scientists, such as the ones at the U.S. National Institute of Neurological Diseases, have found many connections between the lymphatic system’s functions and common symptoms of these illnesses. For example, sleep is usually disrupted in patients with these conditions, and seems to be connected to an increased flow into the brain’s lymphatic vessels, which indicates that more toxins are drained from the brain while the body is at rest.

Before the discovery of the brain’s lymphatic system, concrete evidence indicating the cause of brain inflammation did not exist. Due to this research, scientists may now be on their way to a breakthrough. Of course, with the new discovery comes plenty of new questions: How does the lymph fluid enter these vessels? How do these vessels interact with the rest of the body? What other functions may these vessels serve? The road towards discovering cures to many of the diseases that plague society may not necessarily be nearing an end anytime soon. Nevertheless, discovering that the brain has a waste management system will undoubtedly aid scientists on the journey.

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One thing is for sure: I was born a runner. The urge to run, to move - it flows through my blood. As a child, I never walked around with bare knees. I cannot remember a time when I was not covered in bruises acquired from clumsily tripping and falling during a run. I found an odd euphoria in the sport, one so rare it was only accomplished after my exercise. I especially favored running in the rain. Feeling the heavy drops splatter on my face while I flew effortlessly through the air made me feel invincible.

Once my sneakers are on, I come to life. Every concern I have melts away as my feet pound against the pavement. I feel liberated. The faster I run, the more powerful I feel. I am captivated by this feeling of independence and exhilaration. Over the years, my affection for exercise has grown into an obsession. I joined my high school's track team, eager to develop and flaunt my skill. My exercise has turned from a casual hobby into an obsession.

Over the years, my affection for exercise has grown into an obsession. I joined my high school's track team, eager to develop and flaunt my skill. My most memorable race occurred in my junior year.

As I aligned my feet at the tip of the starting line, I glanced at my competition. Her muscular legs throbbed, and her eyes were glued to her front. There were six others, but in that moment, it was just the two of us. My heart quivered in my chest as I prepared myself for the gunshot. It was my last track meet of the season. My hands were clenched into powerful fists. An excited audience watched, waited.

Without warning, the gun was fired and I was swiftly thrust into the air. My feet pounded on the blue stretch. With each long stride, my name floated around the stadium. I ran as fast as my legs could carry me despite the building pressure in my knees.

With twenty yards to go, I locked gaze with my opponent. Her fierce eyes reciprocated my stare. In the last two yards, I extended my right leg as far as it could reach. Then, everything went dark. Silent. I won. In the blink of an eye, it was all over as quickly as it began.

Running taught me that every single attempt counts, and that the effort of one single stride can account for the difference between defeat and victory. All my years of running amounted to that one race. I started off as one of the weakest links of my team, and ended up becoming captain for two years. Most associate a learning curve with being embarrassingly incompetent at a skill, then slowly learning that skill until it becomes second nature. The major barrier to learning something new is not intellectual, it is emotional - in the beginning, you might feel stupid, but after 20 hours you improve a lot compared to when you started. Some studies cite that it takes ten thousand hours to fully master a skill, which is the equivalent of a full time job for five years. Is this true?

It turns out that the ten thousand hour rule applies to expert level performance. The rule originated from Florida State University professor K Anders Ericsson (1947), who studied professional athletes, world class musicians, and chess grand masters. All of his subjects were ultra competitive people in high performing fields.

Malcolm Gladwell's Outliers, which climbed to the top of New York's best seller list for three months, encapsulates the rule in three parts. Practice often, practice well, and you will do extremely well in whatever field you pursue. Gladwell's book, albeit highly regarded and widely read, caused confusion. His inclusion of Ericsson's rule fell victim to a massive game of telephone, where people turned “it takes 10,000 hours to master a skill” to “it takes 10,000 hours to get good at something.” In fact, with only 20 hours of focused and deliverable practice, you can go from knowing nothing about a topic to being relatively skilled.

A large amount of evidence has demonstrated the power of exercise to support cognitive function, the effects of which can last for considerable time. An emerging line of scientific evidence indicates that the effects of exercise are longer lasting than previously believed, to the point where it could possibly affect future generations. The action of exercise on epigenetic regulation of gene expression seems central to building an “epigenetic memory” to influence long-term brain function and behavior. There have been new developments in the epigenetic field connecting exercise with changes in cognitive function, including DNA methylation, histone modifications, and microRNAs (miRNAs). The understanding of how exercise promotes long-term cognitive effects is crucial for directing the power of exercise to reduce the burden of neurological and psychiatric disorders.

The positive actions of exercise on learning and memory in humans and animals have received abundant support. In older adults, exercise has been shown to improve cognitive performance and counteract the mental decline associated with aging. These effects have been associated with modifications in hippocampal size. In one study, 21 women between the ages of 67 and 81 participated in exercise for 80 minutes per day. After 24 weeks, their hippocampal volume increased. In school children, exercise has been found to be associated with cognitive performance: children who engaged in greater amounts of aerobic exercise generally performed better on verbal, perceptual, and mathematical tests. Recently, a meta-analysis study reported that a single bout of moderate aerobic exercise...
improves inhibitory control, cognitive flexibility, and working memory in preadolescent children and older adults. The results indicate that beyond the well known effects of long-term exercise on the brain, acute exercise also can be used as a tool for situations demanding high executive control. Interestingly, we and others have found that a single bout of both aerobic and resistance exercise is able to enhance memory consolidation in rats.

The epigenetic research has been centered on the analysis of changes on top of the genome that do not involve alterations in the nucleotide sequence. The two most studied epigenetic mechanisms are covalent modifications of DNA (methylation) or of histone proteins (i.e. acetylation and methylation), and their resulting effects on altering gene expression. The phosphorylation and methylation of histones are also tightly associated with regulation of learning and memory.

In agreement with its role in cognition, physical exercise can coordinate the action of genes involved in synaptic plasticity with resulting effects on memory preservation. For example, while exercise enhances the expression of genes (i.e. Bdnf, igf-1 and creb) that positively regulate memory consolidation, it down regulates genes (i.e. PP1 and calcineurin) with a repressive role in these events. Evidence shows that DNA methylation is an important mechanism by which exercise affects gene expression. It is known that exercise differentially modulates the methylation pattern of specific CpG islands located at Bdnf gene, decreases hippocampal expression of DNMTs, attenuates the global methylation changes induced by stress, and increases Bdnf transcription through demethylation of its promoter IV.

It has been shown that the acetylation of histone proteins is a requisite for long term memory. For example, intrahippocampal injection of global HDAC inhibitors (HDACis) such as sodium butyrate (NaB) and trichostatin A (TSA) enhances long term potentiation (LTP) at Schaffer collaterals in CA1 area of the hippocampus, and results in improved consolidation of CFM. The pro-cognitive function of HDACis is partially attributed to their ability to increase histone acetylation and consequently the establishment of an open chromatin state. Interestingly, a previous study has shown that, like HDACis, physical exercise has the ability to transform a learning event that does not normally lead to a stable memory trace into a long-lasting form of memory (Interfolker et al., 2013). Additionally, it was found that physical exercise increases histone acetylation and reduces HDAC expression and neural activity in the hippocampus.

Global HAT activity has been found to be increased in the cortex and hippocampus of rodents subjected to treadmill exercise. Moreover, in a recent study, Zhong et al. (2016) observed that exercise-induced memory improvement was associated with enhanced expression of cAMP response element-binding protein (CREB)-binding protein (CBP) in the hippocampus. Acting more than just a molecular scaffold for recruiting components of the transcription machinery, CBP serves this capacity by inducing chromatin remodeling via its HAT activity, which is likely necessary for activity-dependent gene expression in LTP and long-term memory formation. Mechanistically, the recruitment of CBP triggers histone acetylation and the formation of a transcriptional complex at the promoters of many CREB-target genes to activate transcription. Mutations in the CBP gene are responsible for the mental retardation syndrome Rubinstein–Taybi (Petrij et al., 1995) and CBP mutant mice exhibit profound deficits in synaptic plasticity and LTM. Altogether, the aforementioned findings raise the idea that physical exercise promotes synaptic plasticity and memory improvements by altering the balance of HAT/HDAC enzymatic activity to favor a permissive state of chromatin, leading to the transcriptional activation of a myriad of genes with preponderant roles in cognition.

I was not cognizant of how greatly running impacted so many aspects of my life. Perhaps the youthful memories of running through the rain and feeling invincible conditioned me to believe that I love feeling of the lactic acid buildup in my knees, and the feeling of almost passing out from exhaustion. Or perhaps, I have put in too much time to turn back now. Either way, neuroscientists are beginning to uncover the magic of brain-body connection, and cannot run away from it.
Getting lunch with your favorite celebrity, going to the moon, flying - it is possible to experience all of these things, and some people do every night. How, you might ask? It’s done by lucid dreaming.

Lucid dreaming is a phenomenon where the dreamer is aware that he or she is dreaming. This ability is rare, only experienced regularly in about 20% of people. However, a half of sleepers have done it at least once in their life. People are typically unaware of the state that they are in while sleeping. Perceptions and emotions are felt, but there is no conscious awareness of the dream itself. Nevertheless, those who lucid dream are aware of the state that they are in.

Researchers from the Max Planck Institutes of Psychiatry (Munich) and Human Cognitive Brain Sciences (Leipzig) conducted a study where they compared the brain patterns of lucid dreamers and normal dreamers (5). In this study, participants were asked to complete questionnaires where they were asked about their lucid dreaming frequency, intensity, and degree of control. They were also asked questions about their ability to self-reflect and their degree of self-consciousness (6). The volunteers then underwent brain imaging while reporting every thought caused by some external factor (for example, the humming of a machine). For example, this means the things that they see around them or the noises they hear in their environment, as opposed to internally oriented thoughts like remembering what they had for dinner last night or what they will eat later that day.

From this study, several key differences were found between the brains of those who lucid dream and those who do not. For lucid dreamers, these differences included: increased awareness of their own thoughts, larger brain regions, and increased brain activity while dreaming.

More specifically, lucid dreamers were observed to have a larger anterior prefrontal cortex (4). This area of the brain is responsible for higher cognitive processes and plays an important role in our ability to self-reflect. The brains of lucid dreamers also have more gray matter in their frontopolar cortex. This is a small area right in the front of the brain, known for higher-order processes like critical thinking and problem solving.

Along with an increased size of certain brain regions, those who lucid dream have more brain activity while they dream, specifically in frontal lobe areas, whereas during REM sleep people typically have less brain activity. Specifically, right dorsolateral prefrontal
cortex tends to be active during lucid dreams. This brain area is mainly responsible for higher level cognitive capacities including memory, decision-making, and self-assessment (2). The parietal lobes, responsible for reception and sensory information, are also highly activate during lucid dreams. Lastly, the precuneus, which is responsible for self-perception, also is more active during lucid dreaming. The increased activity in all of these areas combined likely underlies the ability to experience a lucid dream.

These findings offer a breakthrough in the neuroscientific understanding of human consciousness. Researchers are currently trying to determine whether self-awareness can be taught (2). They plan on conducting a new study where they will teach subjects to lucid dream and see if their self-reflection improves. Furthermore, researchers are looking to see if lucid dream training can be used to treat hypnagogic hallucinations (hallucinations seen as one falls asleep), recurrent nightmares, and pathological dream vividification (6).

Now you might be wondering: how does one even go about lucid dreaming? Here are a few steps to increase your chances of having a lucid dream, according to AsapScience (1):

- Keep a dream journal: right when you wake up, remember to write down your dreams. Even if you can't remember anything, log it. This helps you recall your dreams better and boosts lucidity.
- Perform reality checks: reality checks are simply evaluating and making mental confirmations of your surroundings. For example, checking the time, looking away, and checking again to confirm. This is because, during a dream something as simple as reading the time can be bizarre: you may check the time one moment and the next see something completely different. Therefore, if you get in the habit of checking your reality in your day-to-day life, you may be more likely to do so in your dreams, and thus be able to determine when you are in one.

Wakeful Induced Lucid Dreaming (WILD):

This is the process of remaining aware as your body is falling asleep. This skill is rather difficult to master and requires practice and experience with meditation. Though the idea of being able to control one's dreams sounds fun, there are some precautions to take. Lucid dreaming increases one's likelihood of experiencing sleep paralysis. Sleep paralysis is a phenomenon where an individual is unable to move as they fall asleep or upon waking up, due to the lingering natural paralysis the body experiences during REM sleep. Sleep paralysis can be frightening, as most people describe seeing dark figures approaching them while in this immobile state. Like the sleep paralysis itself, this symptom is an artifact of the brain's visual cortex activity from dreaming.

Summary:

In studies of lucid dreaming, lucid dreamers have increased self-awareness, different brain structure, and higher levels of neuronal activity. These findings can perhaps be used to treat several negative sleep symptoms. Learning how to lucid dream isn't difficult, but requires a great deal of patience. And when deciding whether you want to lucid dream, don't forget the potential downsides.

Bibliography

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“Its ghastly signs and tokens are not so palpable to the eye and sense of touch as scars upon the flesh; because its wounds are not upon the surface, and it extorts few cries that human ears can hear; therefore I the more denounce it, as a secret punishment which slumbering humanity is not roused up to stay.”

In the early 1900s, Charles Dickens described his experience at Eastern Penitentiary in Philadelphia, one of the first prisons to accept and practice solitary confinement. Though he was only allowed to observe the conditions of their in-house prisoners, his perception of the circumstances with which these individuals dealt with on a daily basis proved more grotesque and inhumane to him than to the guards and politicians who ran the prison.

But what exactly bothered him? The prisoners were not observably being tortured. In fact, several looked content with their situations. One German man, as Dickens describes, had even “painted every inch of the walls and ceiling quite beautifully” during the two years of his confinement. Though there was no glaring, discernable root of Dickens’ suspicion, he nevertheless believed that this method was “worse than any torture of the body.”

In reality, Charles Dickens was a Victorian writer who had little background in the
The current version of solitary confinement is not what you might have seen in Frank Darabont’s The Shawshank Redemption (1994). The prisoner is kept in an actual cell, as opposed to an obscure hole. They are provided a bed, sink, toilet, and food through a door slot. They might even be given an hour to exercise or roam about the yard under intense supervision, but they are otherwise confined to their cells for 23 hours a day.

The issue arises with time. Craig Haney, a social psychologist at the University of California, Santa Cruz, asserts that the most “insidious” aspect of solitary confinement is how it “requires people to learn to live in a world without people” (Skibba). He adds how the prisoner must structure their world and psyche in a way that is dismissive of social interactions. He then speculates that this could explain why confined prisoners’ “social skills begin to atrophy [or worsen].” ‘The degree of degradation in sociability could range from feeling a general discomfort whenever a prisoner is around other people to an inability to adhere to societal norms. In the latter case, a prisoner might experience visible instances of “panic… paranoia, aggression, or depression.” The worst of these effects often appear in social environments, where, for instance, a feeling of paranoia can be magnified in the presence of a large amount of people. One would have difficulty eating at restaurants, going bowling, walking through a park, or even going to a library with the feeling of how someone could be ‘out to get you.’ With little rehabilitative help available outside of psychiatric hospitals, these individuals are typically confined to their own homes.

He also mentions how the experience is unique and depends on the individual. Some prisoners experience an initial “period of vulnerability” where they heavily doubt their potential to survive in such an environment. Craig Haney notes that this is especially true in the case of inexperienced prisoners. This period is typically marked by anxiety attacks, suicidal thoughts, and instances of catatonia. Only afterwards does the negligence of meaningful social relationships occur.

One specific effect that can be attributed to solitary confinement is how prisoners tend to experience severe hallucinations. Stuart Grassian, a psychiatrist and former faculty member at Harvard Medical School, told how an inmate he interviewed “developed some obsession with his inability to feel like his bladder was fully empty” (Breslow). He discussed how the prisoner “spent hours” in front of the toilet while attempting to relieve himself of this “sensation.”

While the duration of a prisoner’s stay in a solitary confinement cell may seem correlated with their declining state of mind, it must also be considered how these people have pre-existing psychological problems that are actually promoted under such extreme conditions. Kelli Klebe, a psychiatrist at the University of Colorado, conducted a longitudinal study of the psychological effects of prisoners who were being held in administrative segregation. After observing 64 male inmates and 24 control prisoners, she observed their growing mental instability and was led to believe that a causative relationship

**Works Cited**


Introduction

Imagine waking up, only to find yourself in a real-life nightmare. You can't move anything from head to toe. When you try to call for help, you can't open your mouth or even mumble. Essentially, it is like being trapped in your own body.

For those who have never heard of sleep paralysis, waking up and not being able to move or understand why this is happening can be terrifying. This terror can be augmented when it is accompanied by visual or auditory hallucinations and being unable to breathe. Sleep paralysis can be accompanied by other sleep disorders as well, like narcolepsy. Sleep paralysis can produce all of these symptoms, but what in the brain is triggering these terrifying responses to a non-present threat?
Historically

Almost as many as 4 out of 10 people will experience sleep paralysis during their lifetime; it is a lot more common than thought previously by scientists (8). We have records of sleep paralysis existing for centuries and such evidence is embedded in folklore, myth, and superstition. Sleep paralysis’ prevalence across multiple cultures is a testament to the universality of this experience; therefore, it’s important to understand sleep paralysis through the perspective of different cultures. Different cultures have different names for the “demons” that they see at night as a result of sleep paralysis. In Egypt, the attacker is known as a jinn, in Italy it’s known as a giant cat Pandafeche. Across the world in Japan, the dark figure is known as the Kanashibari.

The most prevalent symptoms of sleep paralysis are auditory and visual hallucinations, often causing people to believe that someone else, or something else, is in the room with them. In the past, this has often been interpreted as the presence of the “devil” or “demons” (2). Many people also report a choking feeling during the night, increased heart rate, and the inability to move or speak. These sensations can be combined with the hallucination to create a narrative to explain the symptoms. This supports the description of many saying that the demons or devils that they saw in their rooms attacked them or tried to hurt them.

What is Sleep Paralysis?

Sleep paralysis occurs when someone is falling asleep or waking up; it is a disruption of REM sleep, a literal cross-over of our waking and dreaming states. One of sleep paralysis’ most poignant symptoms is hypnopompic and hypnagogic hallucinations that are reported in cases all over the world. Hypnopompic hallucinations occur specifically during the transition from sleeping to wakefulness, while hypnagogic hallucinations occur during the transition from wakefulness to sleep. The experience of these hallucinations is like having a dream unfold before your eyes, but perceiving it as reality. Think of the worst nightmare you’ve ever had coming to life and not being able to seek the consolation that it was a dream.

Research has found that sleep paralysis also has a tie to anxiety disorders and fear. In cultures that have an extensive narrative and lore surrounding sleep paralysis, it is more common to experience these symptoms. The cultural influence of learning to fear sleep paralysis is correlated with higher incidences of sleep paralysis and experiencing longer episodes (5). Having experienced trauma or other anxiety disorders, might predispose people to showing more signs of sleep paralysis as well.

Treating Sleep Paralysis and Future Research

Despite the level of fear that people might experience due to repeated exposure to sleep paralysis, there are several options to help people combat sleep paralysis. Using relaxation, meditation techniques, and better sleep hygiene have been found to help decrease incidence of sleep paralysis among long-term sufferers. Sleep paralysis has been prevalent in the general world population for a long time, but hardly receives the recognition that it deserves when it comes to understanding other parts of the brain relevant to our perceptual experiences.

Sleep paralysis shows a lot of promise in providing a gateway to understand the pathology of other psychiatric disorders. Sleep paralysis has many overlapping symptoms with common psychiatric disorders in sleep disorders (narcolepsy) and schizophrenia. There are also ties between sleep paralysis and anxiety disorders, where one increases the incidence of the other (7). In certain countries with fear-inducing lore surrounding sleep paralysis, heightened anxiety of sleep paralysis increases the likelihood that someone will experience it. Sleep paralysis’ role in the brain in disrupting our own self-perceptions and manipulating our experiences into dreams, also poses explanations to symptoms in other disorders like hallucinations, paranoia, and persecutory (8).

References

Procedural Memory: “Just Do It” WRITTEN BY: FARAZ ZAIDI

It is a saying we have been hearing since we were 5, “practice makes perfect.” The idea that the repetition of a certain action or behavior over time allows for it to become second nature is not a mere coincidence, rather it is the direct result of the laws of neuroscience. Procedural memory is a part of the long-term memory system that is responsible for remembering certain motor skills. Examples of motor skills that we become adept at through our procedural memory include tying our shoes, riding a bike, and playing an instrument. The regions of the brain associated with building and storing these memories are the basal ganglia, the motor cortex, and the cerebellum, as their interaction is responsible for encoding and storing memories related to the coordination of timed-movements relative to body position.

Procedural memory was discovered through a series of experiments performed on the patient Henry Molaison or “H.M.” H.M. grew up suffering from epileptic shocks, and when all known remedies proved to be ineffective, he resorted to surgery. After undergoing a surgical removal of part of his hippocampus which was believed to be the cause of his seizures, H.M.’s epileptic shocks were gone, but he was unable to encode new short-term memories, a condition known as anterograde amnesia. He was also unable to recall certain memories before his surgery, a condition known as retrograde amnesia.

As we perform a task repeatedly, the connections between the motor neurons that facilitate certain body movements become stronger and quicker. Due to the strength of these connections, we are often able to perform tasks. In some instances, the nuclei within the cell bodies of some neurons transcribe more receptors at the post-synaptic terminals of the cell, allowing for more rapid firing and transmission of electrochemical signals. This occurs when glutamate released from the presynaptic terminal binds with AMPA ionotropic receptors, initiating a series of excitatory postsynaptic potentials. The overwhelming positive charge within the neuron then causes NMDA ionotropic receptors to open as well, allowing calcium ions to enter the inside of the cell. Calcium ions serve as secondary messengers, prompting a phosphorylation cascade which ultimately leads to the transcription of more receptors by the nucleus. In essence, this entire process is a cellular process that supports learning and is soon encoded as procedural memory. Unlike other forms of memory, procedural memory resides below the conscious level of awareness, and therefore actions executed through this form of memory take place almost automatically.

Procedural Memory in Amnesia

We are all familiar with the dreadful diseases which cause people to lose their declarative memory, including horrifying cases of individuals who fail to recall their own relatives. One of the more rare amnesiatic disorders is known as Korsakoff’s Syndrome, which is generally due to “a severe deficiency of thiamine” (or Vitamin B-1). Symptoms faced by patients with Korsakoff’s syndrome include an inability to remember new information, inability to remember recent events, and even long-term memory gaps. Oftentimes, these patients fabricate memories they have never experienced. Despite the seeming hopelessness of this morbid disease, scientists have discovered that victims of Korsakoff’s Syndrome remember a lot more than originally believed. In a study conducted by the neurology department at Albert Einstein College of Medicine in New York, a patient suffering from Korsakoff’s Syndrome was examined after a motor vehicle accident. He was a musicologist with 12 years of formal musical training and had worked for over 40 years as a music editor. Throughout his adult life he had set aside two hours daily to practice piano. With a desire to cultivate his passion for music within his children, he made them listen to classical music during dinner and match them to the name of the composer for any of those pieces of classical music, he played. Following his accident he was unable to remember the name of the composer for any of those pieces of classical music, not even when presented with a set of multiple choice options. However, when the experimenter played the first few bars of 15 different classical songs, the patient was able to continue to play 13 of them without interruption. Thus, it was discovered that when it came to playing the piano his procedural memory was still largely intact. This led the researchers to conclude that both anterograde and retrograde procedural memory was relatively functional in patients with Alzheimer’s disease.

LOOKING AHEAD

The question now remains, what is it about procedural memory that immunizes it from the deleterious effects of Alzheimer’s disease? Since Alzheimer’s disease develops in patients in the later years of their life, perhaps there exists a pre-emptive treatment yet to be discovered that can protect us, especially those with a biological history of amnesiatic disease, from developing Korsakoff’s Syndrome or Alzheimer’s Disease. With so many mysteries yet to be uncovered in the realms of neuroscience, there is a deficit of studies dedicated to procedural memory. However, research like the one conducted at Albert Einstein College of Medicine continues to give us hope that the solutions to these mysteries lie waiting within arm’s reach.
It was Rudyard Kipling who once wrote that “Smells are surer than sounds and sights to make the heartstrings crack.” He was neither a neuroscientist nor an aromachologist. He was simply a poet who observed the simple yet powerful sense of smell. Take for instance, your mother’s perfume wafting through the air. Jasmine, carnations, or the soft scent of tobacco mix exotically to form what you instantly recognize as your mother’s favorite perfume. Maybe she only wears this perfume for special occasions, and the odor is instantly comforting. Or maybe you notice rich, burnt coffee roast in the air, and you think of your best friend in high school - the one who would always chug espresso shots before your first class each morning. A slight pinch of anxiety or excitement puts a bounce in your step as you think of your early mornings studying for Organic Chemistry in the musty library. And maybe now you smell an old foe - sweet cinnamon seeping from the beer that bested you last Halloween. None of this seems like neuroscience - but it is. Whether we realize it or not, smell has an intrinsically emotional component, which, even at an anecdotal level, is important for memory and emotion. So if that is the case, what does research have to say about this?

For decades, olfaction, or smell, has been put on the sensory-research back burner. Maybe this is because smell is an underappreciated sense. While animals like dogs can detect molecules that are as dilute as one part per trillion, for humans, smell is essentially useless (1). In this regard, humans are inferior. Then, there are pheromones - animals release and detect...
these odors, and it can significantly impact their behavior and mating. While some people spritz on perfume or cologne to stimulate these effects, many people simply use deodorant and carry on with their lives. So why is smell so underappreciated amongst homosapiens? Should it be given more importance? Is it important? The answer is: absolutely.

Smell may not be the sexiest line of research, but its implications for psychiatric and neurodegenerative disorders are undeniable. For example, studies conducted as early as 1988 show that patients with Alzheimer’s disease have hindered senses of smell (3). While this reduction is currently attributed as a side effect of disease progression, it may also lead to diagnosing and treating Alzheimer’s earlier with “sniff-tests.” Patients with Schizophrenia also experience smell deficits; yet this finding is harder to explain (4). Current literature explains this phenomenon by examining the anatomy of the brain. The brain regions implicated in memory and mood are also heavily implicated in processing odor. In fact, the human olfactory bulb, which is responsible for processing smell information, rests just in front of the amygdala, a region implicated in emotional processing, and hippocampus, a region implicated in memory consolidation. The amygdala and hippocampus became famous with patient H.M., who had most of these structures removed. As a result, olfaction is still poorly understood. However these studies can be done. The neuroscience community’s difficulty with smell can even be seen in the textbooks used to teach Neuroscience. The textbooks for Boston University’s NE101, NE102, and NE203 gloss over olfaction, and fail to discuss the subject in the same depths as the other senses. This attests to how sparse the research is, if even textbooks deem the subject worthy of only a few paragraphs. Certainly, for decades, scientists have teased out the functions of our eyes, ears, and ability to sense. While these senses all have their mysteries that still require more research, olfaction has seemingly been avoided, or forgotten.

These findings seem to suggest that there is a link between odor and memory.

These findings may be contributed to the anatomy of the brain. The brain regions implicated in memory and mood are also heavily implicated in processing odor. In fact, the human olfactory bulb, which is responsible for processing smell information, rests just in front of the amygdala, a region implicated in emotional processing, and hippocampus, a region implicated in memory consolidation. The amygdala and hippocampus became famous with patient H.M., who had most of these structures removed. As a result, he could not form new episodic memories - or memories of facts and events that occurred after his surgery. With further testing, it was found that besides his issues with memory, H.M. also had an issue with recalling and identifying some odors (2). Despite that patient H.M. was the subject of a considerable number of studies, this finding was not discovered until 1983 - almost 20 years after patient H.M’s initial surgery.

If that is the case, then psychiatric and memory researchers have their work cut out for them.

Sadly, odor research seems to oscillate, piquing the interest of researchers, before teetering to a stop. The receptors are difficult to study, and it is frustrating to control and present smells at specific times in experiments. Even worse, smell is subjective – what one person might find pleasant, another person might find abhorrent. As a result, olfaction is still poorly understood. However these studies can be done. The neuroscience community’s difficulty with smell can even be seen in the textbooks used to teach Neuroscience. The textbooks for Boston University’s NE101, NE102, and NE203 gloss over olfaction, and fail to discuss the subject in the same depths as the other senses. This attests to how sparse the research is, if even textbooks deem the subject worthy of only a few paragraphs. Certainly, for decades, scientists have teased out the functions of our eyes, ears, and ability to sense. While these senses all have their mysteries that still require more research, olfaction has seemingly been avoided, or forgotten.

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Think back on a vivid memory you have of any event or day. Most likely, you are not thinking of a boring, unimportant day. You are probably thinking of a moment in your life that was significant to you. A moment where you felt strong emotion such as surprise or fear. When humans go through a very emotionally arousing event, that memory tends to be embedded in our brains more strongly than others, like a “snapshot” of that moment in our life. These memories are called emotional memories. There is an extremely rare form of these memories, however, called flashbulb memories. Flashbulb memories are defined by Brown and Kulik as much more vivid, detailed, and long-lasting compared to a normal memory (1977). In these memories, we are able to remember specific details, such as the clothes we were wearing or exactly what we were doing at that moment. Flashbulb memories are different from first-hand or emotional memories: flashbulb memories are what we were doing at the time of learning about the event (i.e September 11th, assassination of John F. Kennedy), while emotional memories are things that happened to us first-hand (Hirst and Phelps, 2016). Emotional and flashbulb memories are more complex than normal memories, because there is a neurological process behind the encoding and storing of these memories using our bodies’ flight or flight response and our amygdala.

During a very emotionally arousing (i.e. tragic, surprising, etc.) event the sympathetic nervous system kicks in and initiates the stress response. The sympathetic nervous system is autonomic, meaning this happens subconsciously. By initiating this response, stress hormones, like adrenaline, are released. These stress hormones activate the amygdala, an almond- shaped brain mass located in both halves of our brain whose function is highly involved with experiencing different emotions. The activation of the amygdala alerts the brain that our life may be in danger. From here, the brain decides whether or not the stimulus that caused this activation is truly dangerous, and whether the stress response should be turned off or left on (Cannon, 1915).

The hippocampus is another piece of brain mass on both sides of the brain that is both physically and functionally connected to the amygdala. The hippocampus is the area of the brain that consolidates memories and, like the amygdala, is involved with emotional processing and is linked to the sympathetic nervous system. In memory formation the first step is encoding, which the hippocampus is mainly responsible for. A main factor in how well a memory is encoded is how well we are able to “perceive and attend to” the stimulus. If a stimulus is arousing we must attend to the stimulus faster and with more detail. The amygdala comes into play here, as it attends to the arousing stimulus faster than a neutral stimulus, thus “altering the encoding of the hippocampal-dependent memory” so that “emotional events receive priority” (Phelps, 2004). The second step is consolidation, which the hippocampus is also largely responsible for. This step is crucial for survival, because it ensures that an emotional response immediately follows an event so that we can ‘take flight or fight’ against this stimulus. When we are going through this emotionally arousing event, our hippocampus encodes and helps store the memory, while the activation of our amygdala makes the encoding of the memory stronger, due to the emotional attachment to the stimulus (Phelps, 2004). The result is an extremely vivid memory. When this process encodes a vivid memory of an event we didn’t experience, but rather learnt about, it is considered a flashbulb memory.

Various studies have been done to test the stress response’s effect on memory, and determine whether the emotional arousal involved really has an impact on the encoding process. One study by McGaugh and Cahill (1996) aimed to investigate the role of emotion and the amygdala on the creation of memories. Participants of the study were allocated into two different conditions, and each group told different stories. In one condition...
patients were told a mundane story, and in the other condition were told a more emotional version of the story. Two weeks later the participants were asked to complete a recall task consisting of questions about the stories. The participants in the emotional condition remembered the story significantly better. In order to better control the effect of adrenaline release in the brain they added a third condition. Participants listened to the emotional story but were given beta-blockers, which interfere with the release of adrenaline. They found that the ones who were given the beta-blockers recalled the story no better than those who listened to the mundane story. This study provides evidence to support the role of the amygdala activation in the formation of more vivid memories. Beta-blockers restrict the release of adrenaline, thereby deactivating the amygdala. Thus, the weaker encoding and consolidation of memory by participants in the third condition, suggests it was the inhibition of their emotional arousal and amygdala function, that decreased their recall compared to those in the second condition.

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Human interaction is centralized around our ability to communicate through language, and the processing of language occurs with a complexity that has many contributing factors. Languages are composed of three core building blocks: phonemes, morphemes, and syntax. Of these, phonemes are the smallest distinguishable units of language.

For example, in the English language, consonants “b”, “n”, and “t” are singular phonemes. Phonemes, of course, vary across languages. This phonetic variability is responsible for the differences in the structure of languages and allow us to distinguish Mandarin speech from English speech. Mandarin is classified as a tonal language, meaning each syllable produced has a tone which changes the semantics of a word (ASHA). English on the other hand is not a tonal language, so as you can imagine, having no familiarity with Mandarin as an English speaker renders it almost impossible to understand, while some familiarity with the language makes it easier to comprehend. This phenomenon is called the language familiarity effect.

The Communications Neuroscience Research Lab, led by Tyler Perrachione, conducted an experiment to better understand the language familiarity effect in talker identification. They tested the ability of English listeners to identify voices speaking native English, lightly-accented English, heavily-accented English, and Mandarin. These groupings allowed them to assess the effect that familiar phonology has on a listener's ability to identify a speaker's voice. During this portion, participants were not given feedback on whether or not their answers were correct.

It was found that participants were able to identify English speakers with the highest accuracy and identified Mandarin speech with the lowest level of accuracy. The experiment showed that unfamiliar sound patterns impose a cost in talker identification accuracy. This suggests talker identification depends on the relationships between familiar words and familiar sounds (McLaughlin, Cheng & Perrachione, 2017). These relationships allow us to understand what is required to distinguish voices across various phonological structures. Evaluating the comprehensibility of various degrees of accentedness raises the importance of how accents, especially those of second language speakers, can impose a social barrier to community integration. Understanding the perceptual and mnemonic processes that underlie talker identification will help us properly address this social issue.

Sources:
Isabelle Pelcher  
Major: Neuroscience  
Minor: Deaf Studies  
Position: Co-editor in Chief  

I have always loved studying the brain, it is so fascinating. It is the source of everything we do and yet there is still so much we do not know about it. Reading research papers regarding neurodegeneration in high school solidified my interest in studying the brain and learning more about how it functions and dysfunctions. Alzheimer’s Disease is an area of studying I am particularly interested in.  
Favorite Brain Region: Hippocampus

Payton Cabrera  
Major: Neuroscience  
Minor: Deaf Studies  
Positions: Writer, Editor  

The brain is simply beautiful. It controls who you are and if anything is off, it can completely change the way you see the world. I grew up researching psychological disorders and found them to be fascinating, and I fell more in love once I took psychology. As I got older, I had a mentor who spoke candidly on her father’s battle with Alzheimer’s. Hearing her personal account of the disease made me realize that I wanted to be in the world of neuroscience and learning everything I could to hopefully one day help families like Mrs. LoBue’s.  
Favorite Brain Region: Basal Ganglia

Noelle Wojciechowski  
Major: Neuroscience  
Minor: Public Health  
Position: Co-editor in Chief  

I love neuroscience because it allows you to explore questions beyond the “how”. As someone who hopes to work with addiction treatment in the future, I find the neuroscientific approach to research fascinating, well-rounded, and fun. Neuroscience touches literally every facet of life, and involves a great deal of mystery. Plus, no department beats BU Neuro!  
Favorite Brain Region: Anterior Cingulate Cortex

Martinelli Valcin  
Major: Neuroscience  
Position: Artist  

Neuroscience involves more than just simple biology — involving the holistic mind and brain in determining of the significance of myriads of multi-modal processing mechanisms makes this such a productive and intriguing science to study.

Francesca Davy-Falconi  
Majors: Neuroscience / Philosophy  
Minor: Psychology  
Position: Layout Designer  

Oliver Sacks and funky brain facts  
Favorite Brain Region: Fusiform Face Area
Enzo Plaitano  
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Minor: Chemistry  
Position: Writer  

The Brain is amazing! The wide variety and complexity of neurological conditions initially peaked my interest in neuroscience. I’ve been both intrigued and excited about what I am learning, as well as what I still do not know. My inspiration to write for the journal stemmed from the millions of people around the world who suffer from neurological deficits. I hope that my pieces provide an insight into the miraculous work of neurology and highlight the mysteries of brain science.  
Favorite Brain Region: Brainstem

Michele Assef  
Major: Human Physiology  
Minor: Medical Anthropology  
Positions: Writer, Editor, Artist  

I’ve never liked questions that don’t have answers. When you’re young, people tend to expect your acceptance that some things simply cannot be explained, although I’ve never been one to settle for ambiguity. My tendency to question everything didn’t fade as I grew older, which is why my introduction to neuroscience was, for lack of a more fitting description, earth-shattering. Neuroscience taught me that many of these allegedly unanswerable questions can be explained by the one thing that ultimately distinguishes each of us from everyone else—our brains. The brain is the only organ that studies itself, constantly proposing questions that only itself can discover answers to. This continuous process of asking for and seeking out new knowledge is a game I don’t anticipate ever wanting to sit out.

Yasmine Sami  
Major: Neuroscience / Psychology  
Positions: Artist, Layout Designer  

I have been interested in the brain, the ways in which people act, and what causes them to act in these ways ever since I was little. When I was younger, I’d watch videos and read books and articles on psychology, including watching Crash Course Psychology videos on youtube. When watching tv shows, I’d always be drawn to shows that have a psychology aspect to them, like Criminal Minds or Lie to Me. I even wanted to be a forensic psychologist at one point due to those shows! But then, as I got further into high school and took biology and AP biology, I realized that psychology with a biological basis of why psychological events happened was truly what interested me – and that is when I decided that I wanted to pursue neuroscience. The brain and the neurons that make it up are truly the root of everything psychological, so in order to understand the things that people do, I found it extremely intriguing to also study the mechanisms that produce those behaviors; this guided me to pursue a double major in both Neuroscience and Psychology.  
Favorite Brain Region: Prefrontal Cortex

Erika Pettway  
Major: Neuroscience  
Minor: Psychology  
Positions: Writer, Editor, Layout Designer  

I believe the brain is the most important organ in the body: It controls everything!  
Favorite Brain Region: Insula
Zoe Malone 2022
Major: Neurobiology
Position: Editor
I read a book on prions and took a psychology course my sophomore year of high school. I already had a love of biology, but that’s when I became really curious about the nervous system and I wanted to explore the field more.
Favorite Brain Region: Prefrontal Cortex

Sawan Patel 2022
Major: Neuroscience
Position: Writer, Editor
Taking AP Psychology got me really interested in the brain - especially the realization that I had no idea how the brain worked. Additionally, the idea that we constantly seek to understand the world around us yet we each have a sort of immensely complex universe between our own two ears is fascinating.
Favorite Brain Region: Medial Prefrontal Cortex

Lawrence Ullman Jr. 2022
Major: Neuroscience
Positions: Writer, Layout Designer
What makes neuroscience so interesting is that the underlying mechanisms in the brain are fascinating and complex, and to understand such microscopic events is to truly understand what it means to be human. Thank you to everyone who contributed to making The Nerve so enjoyable and successful this year!
Favorite Brain Region: Hippocampus

Nicole Tacugue 2021
Major: Neuroscience
Positions: Artist, Layout Designer
I love figuring out what goes on in the mind that encompasses the human experience!
Favorite Brain Region: Amygdala

Nicki Goldfeder 2022
Major: Biology
Position: Layout Designer
I took AP Psychology in high school and found it very interesting. Although I am not a Neuroscience or Psychology major, I still am interested in researching topics regarding the brain in my free time, so The Nerve is a great way to do that.
Favorite Brain Region: Hypothalamus

Brandon Molligoda 2022
Major: Neuroscience
Minor: Chemistry
Positions: Writer, Layout Designer
I got interested in neuroscience while reading for leisure when I was younger and a desire to understand the molecular basis behind our everyday actions.
Favorite Brain Region: Hippocampus

Otto Holbrook 2022
Major: Neuroscience
Minor: Chemistry
Positions: Writer, Editor, Layout Designer
I have always enjoyed helping people with any trouble that they may have. A lot of problems that people face arise from issues within the brain so I think a deeper understanding about how the brain works is essential to helping people through their issues.
Favorite Brain Region: Amygdala

Ankur Bamezai 2022
Major: Neuroscience
Minor: Sociology
Position: Layout Designer
I am interested in neuroscience because of my grandfather’s dementia and understanding how people think and behave fascinates me.
Favorite Brain Region: Hippocampus
<table>
<thead>
<tr>
<th>Name</th>
<th>Year</th>
<th>Major</th>
<th>Minor</th>
<th>Position</th>
<th>Quote</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brian Privett</td>
<td>2021</td>
<td>Neuroscience</td>
<td>Computer Science</td>
<td>Editor</td>
<td>The brain originally fascinated me as the physical root of the philosophical. I am interested in neuroscience by the practical opportunities our increasing knowledge of the brain will unlock. Favorite Brain Region: Amygdala</td>
</tr>
<tr>
<td>Leanna Reynolds</td>
<td>2022</td>
<td>Neuroscience</td>
<td></td>
<td>Editor</td>
<td>I had early exposure to neurodegenerative diseases, and became very passionate about learning about Alzheimer’s and neurobiology! Favorite Brain Region: Hippocampus</td>
</tr>
<tr>
<td>Gabrielle Bogut</td>
<td>2022</td>
<td>Neuroscience</td>
<td></td>
<td>Layout Designer</td>
<td>I love learning about the brain and how the different aspects of the brain control different parts of who you are. The brain is such a small object but it has such a huge impact on our lives! Favorite Brain Region: Hippocampus</td>
</tr>
<tr>
<td>Ivan Kondratyev</td>
<td>2022</td>
<td>Psychology / Philosophy</td>
<td></td>
<td>Artist</td>
<td>Studying the source of all knowledge we have (the brain) is a nice shortcut to knowing everything (or at least having the possibility of knowing). And knowledge is power. Favorite Brain Region: Hypothalamus</td>
</tr>
<tr>
<td>Sarah Jehle</td>
<td>2022</td>
<td>Undecided</td>
<td></td>
<td>Writer</td>
<td>I have always been fascinated by this thing inside of us that controls everything we do, and when a few of my relatives were diagnosed with brain conditions my passion only grew. I had to know how and why some things happened and others didn’t when my Grandma got Alzheimer’s and my Uncle a brain tumor, which ultimately led to my interest in neuroscience. Favorite Brain Region: Cerebellum</td>
</tr>
<tr>
<td>Amanda Fortin</td>
<td>2020</td>
<td>Neuroscience</td>
<td></td>
<td>Writer, Editor, Artist</td>
<td>Besides the fact that it looks like a squishy walnut, I think the brain is an incredible organ. Anything that can hold meaningless song lyrics and invaluable memories is pretty cool. Also, there’s no other organ in the body (that I know of) that named itself.</td>
</tr>
<tr>
<td>Michelle Njoroge</td>
<td>2020</td>
<td>Neuroscience</td>
<td></td>
<td>Writer, Editor</td>
<td>As a freshman I knew I wanted to get involved on campus as much as possible. Being a part of The Nerve staff has given me the great opportunity to learn so much through articles I’ve edited. Most importantly, I have learned about many fields, and research, within neuroscience that I have the opportunity to explore here in my time at Boston University.</td>
</tr>
</tbody>
</table>
Can Volkan Yumuk
2020
Major: Neuroscience
Position: Writer

After my biology teacher in high school made us watch an interview about the brain, I was captivated by its mysterious nature which helped me to create new questions in my mind. I wanted to find the answers to those questions, that is why I chose neuroscience. I am looking forward to find my way through the forest of neurons with the proper knowledge that I will gain in this field.

Colin Stuart
2018
Major: Neuroscience
Position: Editor

Every single person has a brain, but there’s still so much we don’t know about it. When we solve mysteries about the brain, we learn about ourselves!

Victoria Martinez
2020
Major: Neuroscience / Psychology
Position: Writer

I find it interesting how we use our brain to learn about the brain.

Erin Ferguson
2018
Major: Neuroscience / Public Health
Position: Editor

I decided to major in neuroscience because of my family’s history with neurological disorders. At various doctor appointments, doctors would try to explain to me what was going on in the brain - but they’d always dumb it down and it frustrated me. So, I told myself, “I don’t want to have this dumbed down for me - I’m going to go study it myself.”

Colin Stuart
2018
Major: Neuroscience
Position: Editor

Every single person has a brain, but there’s still so much we don’t know about it. When we solve mysteries about the brain, we learn about ourselves!

Victoria Martinez
2020
Major: Neuroscience / Psychology
Position: Writer

I find it interesting how we use our brain to learn about the brain.

Alison Gu
2020
Major: Neuroscience
Positions: Writer, Editor

I find it interesting how the brain affects how people act and function.
Undergraduate Program in Neuroscience

The Undergraduate Program in Neuroscience is an interdisciplinary major leading to a Bachelor of Arts in Neuroscience that takes advantage of the rich neuroscience mission of multiple departments and campuses of Boston University. As a field, neuroscience has grown considerably over the last few decades through its integration of multiple disciplines; and, a current understanding of the field requires knowledge that spans traditional approaches while moving into the intersection between far-reaching technologies and new computational methods. This program combines breadth of exposure to the field as a whole with the opportunity for depth of experience in one of three central domains of neuroscience: Cellular and Systems, Cognition and Behavior, and Computational Neuroscience.

Neuroscience students will have access to the extensive resources and expertise of affiliated faculty across multiple departments and colleges throughout the university. A wide array of courses are offered through the departments of Biology, Chemistry, Computer Science, Mathematics & Statistics, Physics, Psychology, and Health Sciences in Sargent College. Together more than 50 upper level neuroscience electives are offered, including laboratory courses and seminars.

Opportunities for independent laboratory research are available through multiple departments in the Colleges of Arts and Sciences and Engineering, and at Boston University School of Medicine, including Anatomy and Neurobiology, Biochemistry, Neurology, Pathology, Pharmacology & Experimental Therapeutics, Physiology and Biophysics, and Psychiatry. Undergraduate research opportunities in neuroscience laboratories expand throughout the university across both the Charles River and Medical campuses.

Mind and Brain Society

The Mind and Brain Society (MBS; formerly known as the BU Organization for the Mind and Brain Sciences) was founded in the fall of 2008 in concert with BU's new undergraduate program in Neuroscience. The group aims to create a network for undergraduate students who wish to take an active role in current issues and research. MBS serves as a hub for not only Neuroscience majors, but all students interested in Psychology, Biology, Philosophy, Computer Science, and more. Our goal is to support an eager multidisciplinary undergraduate community with the conversations and resources fundamental to Neuroscience today.

Throughout the academic year, MBS hosts events spotlighting many different facets of Neuroscience. We hold discussion sections during which we informally discuss a topic of interest over coffee; previous topics include “The Neuroscience of Religion” and “NeuroEthics.” The group also hosts research presentations by BU professors and screenings of thought-provoking films pertaining to neuroscience.