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Lobotomy

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Abstract

Lobotomy, once considered a groundbreaking treatment for mental illness, ended up being one of the darkest periods in medical history. This paper covers how the procedure began, why it became so widely used, and the serious ethical concerns surrounding it, such as the lack of consent or the long-term damage it caused to patients. We will explain what parts of the brain were affected, particularly the prefrontal cortex, and how this led to major changes in personality, emotions, and cognitive function. The paper will also compare lobotomy to modern neurosurgical techniques like Deep Brain Stimulation, showing how far we have come in treating mental illnesses with safer, precise, and careful techniques that focus on the patient's well-being.

Literature Review

Lobotomy, a once prominent psychiatric procedure, was introduced in 1935 by Portuguese neurologist Antonio Egas Moniz. He developed leucotomy, the surgical cutting of white nerve fibers within the brain, aiming to treat severe mental illness like schizophrenia and depression by severing connections in the brain's frontal lobes. His work earned him the Nobel Prize in Physiology or Medicine in 1949 (Wired, 2010).

In the United States, Dr. Walter Freeman, a neurologist, collaborated with Dr. Hames Watts, a neurosurgeon, to perform the first lobotomy in the country in 1936. Freeman later created the transorbital lobotomy, also known as the "ice-pick lobotomy," which involved inserting a sharp instrument through the eye socket to reach the frontal lobes. This method was quicker and could be performed outside of "traditional" surgical settings, which made it accessible to many under-resourced psychiatric hospitals. It allowed Freeman to perform thousands of lobotomies, often without the presence of a surgeon (Wikipedia, 2024).

Lobotomy became widely used throughout the 1940s and early 1950s, especially for mental conditions like schizophrenia, severe depression, and OCD (obsessive-compulsive disorder). However, it was also very overused, sometimes as a tool to control complex behavior in the institutionalized patients (including women and children) instead of being a medically justified treatment (Verywell Mind, 2023).

There were many ethical concerns surrounding lobotomy that held importance. Many patients did not give informed consent, especially in the psychiatric institutions where they did not have much agency (Verywell Health, 2022). The procedure was performed in a non-sterile and

uncontrolled way, often on vulnerable people. Freeman himself was known to perform lobotomies in public for demonstrations. He even used a traveling van called the “lobotomobile” to bring his services to overcrowded hospitals (Wired, 2011).

As evidence of the serious side effects of this “treatment,” such as cognitive disability, emotional blunting, personality loss, and even death, criticism of the dangerous procedure grew. The decline of lobotomy began with the invention of antipsychotic medications, especially chlorpromazine (Thorazine) in the 1950s, which provided a safer, non-surgical treatment alternative to lobotomy (Health.com, 2023).

By the 1970s, lobotomy had become a symbol of overtreatment and ethical failure. Its dark history serves as a cautionary tale in neuroscience and medical ethics, highlighting the dangers of invasive procedures performed without proper consent, oversight, and scientific evidence.

It was initially grounded in neuroscientific theory. The rationale behind the procedure stemmed from the idea that modifying the prefrontal cortex (PFC) could alleviate severe psychiatric symptoms. This notion was not entirely unfounded; the PFC plays a central role in complex cognitive and behavioral functions, including decision-making, emotional regulation, personality, and impulse control (Fuster, 2015). These functions are precisely what distinguish humans from other species in the animal kingdom.

Modern neuroimaging studies show that the structure and activity of the prefrontal cortex are often altered in individuals with mental illnesses. For example, reduced PFC volume and hypoactivity have been observed in patients with depression, schizophrenia, and bipolar disorder, conditions frequently treated with lobotomy in the past (Zhao et al., 2020; Selemon & Zecevic, 2015). These abnormalities contribute to symptoms such as impaired judgment, emotional instability, and difficulty in goal-directed behavior, all of which were once believed to be controllable through surgical intervention.

The prefrontal cortex can exert such broad influence due to its dense interconnectivity with subcortical structures, particularly those within the limbic system and the thalamus. It forms direct projections to the amygdala, hypothalamus, and septal nuclei, key regions involved in emotion and behavioral regulation. Indirectly, it communicates with the thalamus, which serves as a relay station, transmitting and modulating signals between the cortex and deeper brain structures (Zhao et al., 2020).

During a lobotomy, however, white matter tracts such as the fronto-thalamic and fronto-limbic pathways were severed, critically impairing these circuits. This disconnection often led to blunted affect, apathy, loss of motivation, and personality deterioration, outcomes that ultimately contradicted the procedure’s initial therapeutic goals (Zhao et al., 2020; Valenstein, 1986).

Methods and Materials

“A lobotomy is the sorrowful unthreading of a mind once reaching for meaning.” Lobotomy was believed to sever connections in the frontal lobe of the brain, which could reduce disruptive thoughts and behaviors associated with these disorders. However, it resulted in widespread horror and the erosion of free will and autonomy for many individuals.

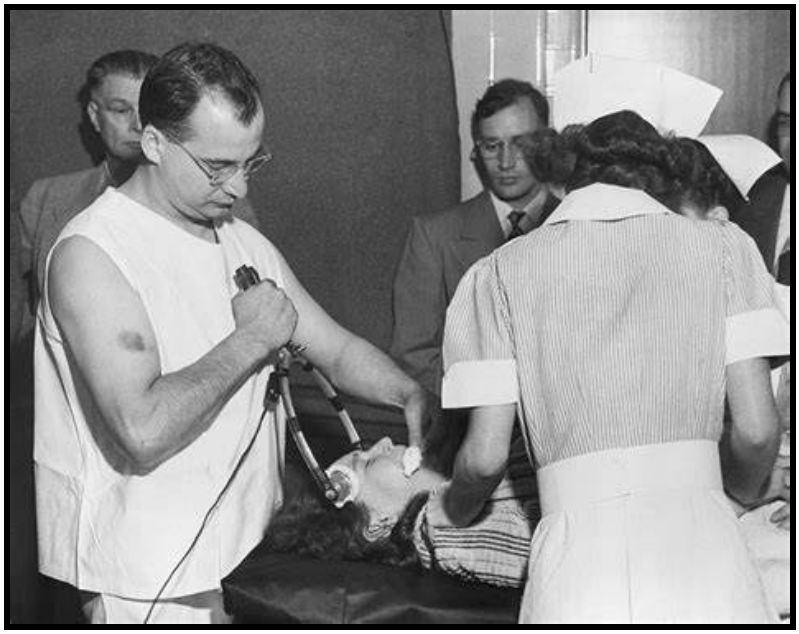

Figure 1. Image Source: Loki Rupaul Blogspot (2021) (https://lokirupaul.blogspot.com/2021/02/lobotomy-brain-op-described-as-easier.html)

In the early 20th century, patients faced a grim trinity of evils, each masquerading as a cure yet harbinger of horror. The prefrontal lobotomy involved drilling into the skull and inserting a leucotome, severing the delicate threads of the prefrontal cortex and dismantling the pathways between thought and emotion. More chilling was the transorbital lobotomy, the focus of this discussion, where an icepick-like instrument was thrust through the eye socket above the tear duct and hammered into the brain. There was no anesthesia, no sterile theater, just a chair, a mallet, and terrible silence. Then came the bilateral and unilateral lobotomies, a roulette of neurological fate, determined by which hemisphere would be “sacrificed”, a corrupt and heartless process. Lastly, the frontal leucotomy was precise, using a loop of wire to carve through the white matter, much like an escape from memory.

The procedure begins by focusing on the anatomy of the skull, particularly the frontal bone, which forms the forehead and the upper part of the eye socket, known as the orbit. This section of bone is very thin, making it a vulnerable entry point for the procedure. During a transorbital lobotomy, a tool like an ice pick is inserted just above the eye, going through the thin orbital bone (the orbital plate) to reach the inside of the skull or the neurocranium. Once the device has

penetrated the cranial vault, it enters the brain, focusing on the area between the prefrontal cortex and the thalamus.

The operation's primary goal is to sever neuronal connections between the prefrontal cortex, which regulates logical cognition, decision-making, and personality, and the thalamus, the brain's primary relay hub. To do this, the doctor inserts the instrument and then performs a quick 40-degree spin toward the midline of the brain to sever these fibers. Disabling or "deactivating" these critical networks is the end outcome. The implications of such a procedure are severe: patients often become empty shells, lacking individuality, functionality, and emotional control. Some experienced perplexity or wrath, while others died in a condition of absolute nothingness. The fact that this procedure was performed around 20,000 times in the United States as late as the 1950s is a sobering reminder of the darkest moments in medical history.

Figure 2. Eva Perón in 1947. In 2011, it was discovered that a lobotomy had been administered to her in her last days. Wikimedia

Eva Peron, a beloved figure who rose from humble beginnings to become a powerful political force in Argentina, deeply inspired the working class through her charity work and passionate advocacy. Despite her growing influence, her serious illness and radical political stance were met with control measures instead of support, where her views were viewed as a mental illness by society. The lobotomy she was unfairly subjected to, without consent, was the transorbital lobotomy. It was seen as a solution to her ongoing illness and kept her from her immoral, radical views at the time. Instead, it stripped away her vitality and hastened the end of her beautiful life, silencing a voice that held so much hope for people.

Figure 3. Alys Robi, an established Canadian singer and performer, recovered from her lobotomy but still experienced the societal stigma connected to mental illness. Wikimedia

Similarly, Rosemary Kennedy and Alys Robi were women whose struggles with mental health; they were met with neglect and secrecy rather than compassion and understanding. Rosemary, a memory of America's most famous family, was lobotomized at her father's insistence to mask her behavioral changes, leaving her incapacitated for life and hidden from the public eye. Alys Robi, a talented Canadian performer, faced a similar fate when her lobotomy was chosen over proper health care, also imposed by her own family. In all three cases, lobotomy was a cruel “quick fix” that failed both the individuals and society, destroying lives under the guise of treatment.

The lobotomy has long-term consequences for brain function. Patients frequently have emotional and psychological disorders, memory and learning problems, motor function abnormalities, and impaired decision-making and problem-solving skills. Psychiatric problems may temporarily

subside for some people, but the long-term effects are frequently dire. Patients frequently lose their capacity for critical thought, memory formation, and sophisticated social interaction. In certain instances, lobotomy may result in a condition that resembles infantile regression, rendering the patient permanently care-dependent.

Beyond the disease itself, the stigma associated with lobotomies also pervaded the care patients got. Frequently viewed as "broken" or "less human," patients' conditions were perceived by society as irreversibly changed or lacking. People were isolated from their families and communities as a result of this stigma, which exacerbated discrimination and feelings of shame.

The side effects of lobotomies, such as emotional dullness, impaired cognition, and altered behavior, made everyday social interaction difficult. The social rejection and prejudice they faced further compounded their isolation. Many lobotomy patients were pushed to the margins, viewed through the lens of their diagnosis and treatment rather than as whole people. This profound social alienation made adjustment nearly impossible, further entrenching their exclusion from meaningful social roles and relationships.

Transorbital lobotomy has a disastrous effect on persons with autism and schizophrenia, effectively silencing previously misunderstood brains. For people with autism, whose actions were frequently mistaken as insane, the process swept away their final remnants of uniqueness. The scalpel became a terrible metaphor: a blade used to slice off identity rather than a tool for healing. Many autistic children and adults, who were already suffering from speech and sensory processing, were left in a state of muteness, emotionlessness, and unreachability. Lobotomy did not alleviate their suffering; instead, it removed their ability to communicate it, transforming solid but differently wired minds into empty echoes of their former selves.

Those who have schizophrenia fared no better. Lobotomy promised relief from delusions and hallucinations, but it instead flattened affect and eliminated depth. In some cases, their hallucinations became worse, even more frequent, and crueler, enduring a constant life of horror. Instead of clarity, lobotomy produced confusion, and instead of serenity, it gave a vacuum. These patients were frequently imprisoned, not because they were dangerous, but because society feared what it could not comprehend, and used the lobotomy as a method to silence what it could not change.

"A cure worse than the disease" captures the terrible legacy of the lobotomy. Behind each surgical cut was a person with a voice, a thought, and a life worthy of appreciation. Instead, lobotomy eliminated such voices in the name of medical advancement. It was not healing, but cruel. The procedure's legacy is found not in medical textbooks, but in the broken lives of patients such as Eva Perón, Rosemary Kennedy, and countless more who remain unrevealed. These people were not damaged; they were mistreated, misunderstood, and exposed to a harsh

type of erasure. The lobotomy failed to repair the human mind, leaving not only neurological but also moral scars. Moreover, it serves as a reminder of what occurs when society chooses stigma over understanding and silence over support.

Results

There are many prevalent side effects associated with lobotomy procedures, resulting from the disruption of white matter tracts between the prefrontal cortex and subcortical structures, such as the thalamus and limbic system (Mahsour et al., 2005).

While the procedure was intended to reduce mental health symptoms and improve cognitive functioning, results did not always produce such effects. It was seen that some people did improve from past symptoms and were able to live relatively independent lives. However, others were noted to experience effects such as sudden behavioral changes, problems with mood, and lack of impulse control (Toler, 2022).

Neuropathological studies on patients who have undergone lobotomies showed the wasting and shrinking of the frontal lobe (Uchino, 2001). Further known immediate and long-term side effects are brain infection and abscess, dementia, intellectual impairment, epilepsy, and more. Following the procedure, patients frequently exhibited acute cognitive deficits, including impaired reasoning and executive function as a result of disrupted frontal lobe activity (Mahsour et al., 2005).

An element most notable towards post-lobotomy state was the emotional blunting it would cause, leaving patients to feel indifferent and fail to engage meaningfully with their surrounding environment. This was attributed to the disrupted connectivity between the prefrontal cortex and the amygdala, core structures that play critical roles in emotion regulation (Faria, 2013).

In many cases, confusion and disorientation followed immediately postoperatively, likely due to the neurological trauma and incapability of compensation for the sudden loss of frontal modulation.
Lobotomies also introduced sensory and motor disturbances, including motor weakness, sensory aphasia, and epilepsy (Lewis, 2021).

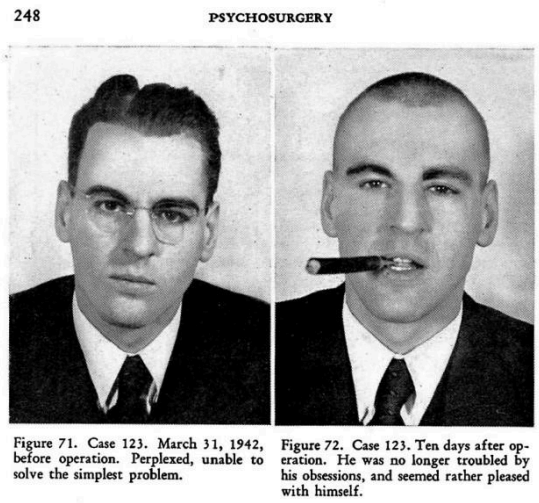
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Figure 71. Case 123. March 31, 1942, before operation. Perplexed, unable to solve the simplest problem.

Figure 72. Case 123. Ten days after operation. He was no longer troubled by his obsessions, and seemed rather pleased with himself.

Figure 4. Typical case notes in the book 'Psychosurgery' by Freeman and Watts, 1950 (2). Facsimile (Torkildsen, 2022)

Through the works of lobotomic surgeon Freeman, many of the patients he operated on stopped showing feelings of anxiety and appeared to act in a more child-like state. Although it was later reported that many also became indifferent and passive, losing all initiative and their ability to concentrate or produce an emotional response due to the severance of connections in the brain, particularly in the prefrontal cortex, which is responsible for higher-order thinking (Torkildsen, 2022).

Long before psychosurgery became a formal medical practice, an accidental case of prefrontal damage offered critical insight into brain function. Phineas Gage, a railroad worker, survived a severe accident in which a tamping iron was driven through his skull, piercing his prefrontal cortex. Following the injury, Gage exhibited drastic changes in personality, decision-making, and emotional regulation—symptoms that closely resemble those later observed in lobotomized patients. Although his case predates the development of lobotomy, it provided early anecdotal evidence that the prefrontal cortex plays a crucial role in personality and social behavior. Gage's condition, while tragic, sparked early neuroscientific interest in the relationship between brain structures and behavior. Unfortunately, it also contributed to the misguided belief that mental illness could be "treated" by damaging or disconnecting the prefrontal region, inadvertently laying the ideological groundwork for lobotomy.

Impact

Lobotomies were often performed without informed consent, particularly on institutionalized women and children, which showed a blatant lack of ethical oversight (Tidsskriftet.no, 2022). The rise of antipsychotic medications like chlorpromazine in the mid-1950s hurried the decline of lobotomy due to their non-invasive character and superior outcomes (Nursing & Health Professions).
Modern techniques such as Deep Brain Stimulation (DBS) show a complete shift in the treatment of neurological and psychiatric disorders. DBS involves implanting electrodes in targeted brain areas, like the subthalamic nucleus or globus pallidus, to regulate abnormal electrical activity. It is primarily used to treat Parkinson's disease, essential tremor, dystonia, obsessive-compulsive disorder (OCD), and treatment-resistant depression (Mayo Clinic).

In contrast to lobotomy, DBS is reversible, customizable, and guided by high-resolution imaging such as CT and MRI scans, which significantly lowers the chance of irreversible harm. Following ethical norms, the surgery is performed with informed consent after the patient has been closely observed.

Another modern alternative is stereotactic cingulotomy, used for patients with severe, treatment-resistant OCD and depression. This procedure involves lesioning specific, small portions of the anterior cingulate cortex using MRI guidance and stereotactic techniques (JNS

Neurosurg Focus; Neurosurgery Clinics of North America). It is more precise than lobotomy, and the outcomes show a reduced amount of psychiatric symptoms without the catastrophic side effects associated with older practices. Patients who go through cingulotomy are carefully screened. The intervention is typically used as a last resort, as many pharmacological and behavioral therapies precede it.

The contrast between lobotomy and modern techniques lies in the combination of science and ethics. Today's neurosurgical procedures rely on advanced neuroimaging techniques, such as MRI and CT, to precisely target parts (PubMed 35667796). Informed consent is now mandatory, and interventions are rigorously overseen and screened prior to implementation. Clinical decisions now prioritize the patient's quality of life, long-term safety, and evidence-based practice. These principles were notably absent during the lobotomy era (Tidsskriftet.no, 2022).

Modern psychosurgery emphasizes ideas such as patient dignity, autonomy, and scientific integrity. This change not only demonstrates technological advancement but also a broader shift in ethical standards within medicine.

Conclusion

Lobotomy's history reveals the harm caused when medicine overlooks ethics and consent. Although intended to treat mental illness, the procedure often led to severe cognitive and emotional damage, especially among vulnerable patients. Its misuse and lasting consequences prompted a shift toward safer, more ethical approaches. Today's treatments, such as Deep Brain Stimulation, demonstrate progress in both neuroscience and respect for patient rights.

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The Short-Term and Long-Term Effectiveness of Current Alzheimer's Disease and Parkinson's Disease Treatments

Dorisa Sun, Leah Abayomi

Abstract

Neurodegenerative diseases are brain disorders caused by the progressive loss of neurons. These diseases involve a decline in cognitive and motor function. Alzheimer's Disease (AD) and Parkinson's Disease (PD) are two of the most common neurodegenerative diseases. Current treatments for AD and PD patients focus on treating the diseases' symptoms without halting neuronal death. This meta-analysis evaluates the effectiveness of current AD and PD treatments in terms of short-term and long-term outcomes. Specifically, this meta-analysis looks at the effectiveness of AD treatments donepezil, rivastigmine, and galantamine, and PD treatments levodopa and monoamine oxidase B (MAO-B) inhibitors. This meta-analysis also evaluates AD and PD treatments currently in the developmental phases and their potential for halting neuron degeneration for each disease.

Literature Review

Alzheimer's Disease

Alzheimer's Disease (AD) is a progressive neurodegenerative disorder that impairs cognitive functions such as memory, comprehension, language, attention, reasoning, and judgment (Kumar et al.). AD is caused by amyloid beta (Aβ) accumulation and neurofibrillary tangles (NFTs) (National Institute on Aging). Aβ peptides originate from the amyloid precursor protein (APP). APP is processed through the amyloidogenic pathway to produce Aβ peptides. As neuronal protein synthesis increases, phosphate accumulates, starting a chain reaction where APP is phosphorylated more frequently, Aβ production is increased, and Aβ accumulates in parts of the brain, especially regions crucial for memory and learning (Ma et al.). This includes regions such as the amygdala and hippocampus. The tau protein is responsible for the assembly and maintenance of microtubules. During this, the tau protein becomes abnormally hyperphosphorylated (Medeiros et al.). Tau protein microtubules disassemble, allowing free tau molecules to aggregate into filaments that become NFTs (Medeiros et al.). As Aβ and NFTs

accumulate in the brain, cholinergic neurons degenerate. Cholinergic neurons are responsible for producing the neurotransmitter acetylcholine (ACh), which influences learning, memory, attention, and focus ("What Is Acetylcholine? | Mental Health America"). This leads to a decrease in ACh production. Current treatments for AD include cholinesterase inhibitors (ChEIs), partial N-methyl D-aspartate (NMDA) memantine, and anti-amyloid antibodies (Kumar et al.). Anti-tau antibodies, gene therapy, and stem cell therapy treatments are still undergoing clinical trials and development.

ChEIs are split into two categories: acetylcholinesterase (AChE) and butyrylcholinesterase (BuChE) inhibitors. AChE and BuChE are enzymes that hydrolyze ACh. ChEIs increase ACh levels by inhibiting the enzymes that break the neurotransmitter down ("Drug Treatments for Alzheimer's Disease"). AChE inhibitors such as donepezil, rivastigmine, and galantamine are inhibitors that prevent the enzyme acetylcholinesterase from breaking down acetylcholine in the brain. This allows for an increase in acetylcholine, promoting nerve cell communication and improving learning, memory, and cognitive functions (Kumar et al.). Donepezil inhibits acetylcholine hydrolysis by reversibly binding to acetylcholinesterase, leading to an increase in the neurotransmitter's availability in the synaptic cleft and assisting in cholinergic transmission (Kumar, Sharma, et al.). The drug also upregulates nicotinic receptors in cortical neurons, which can promote cortical neurons to act neuroprotectively. Donepezil has the ability to reverse inhibition on voltage-activated sodium currents, which delays potassium currents. It has also been discovered that the drug downregulates neuroinflammatory responses, including microglial and astrocytic activation (Kumar, Sharma, et al.). While donepezil has not been proven to directly alter the progression of AD. However, it can alleviate specific symptoms in patients by improving cognition and behaviours (Kumar, Sharma, et al.). The FDA also approved a fixed-dose combination of donepezil and memantine for patients with dementia associated with AD. Donepezil can be administered to patients via a "transdermal system" that does not require patients to swallow the medication (Kumar, Sharma, et al.). Rivastigmine is also a BuChE inhibitor. Rivastigmine, compared to galantamine and donepezil, works by inhibiting both acetylcholinesterase and butyrylcholinesterase. It is a pseudo-irreversible, carbamate-type, brain-selective, dual inhibitor (Kandiah et al.). Rivastigmine can also be taken in various forms, through methods such as oral consumption and transdermal patching. Its use is effective, as PET imaging has proved that neurodegenerative diseases such as AD and PD cause a deficiency in cortical acetylcholinesterase. Rivastigmine increases acetylcholine levels in the brain, making it more readily available for synaptic transmission. Rivastigmine has a bioavailability of ~36% for a 3 mg dose (Kandiah et al.). Dosages are most effective when at 1.5, 3, 4.5, or 6 mg (capsules); 2 mg/mL (oral solution); 4.6, 9.5, or 13.3 mg/24hr (transdermal patches) (Kandiah et al.). It inhibits acetylcholinesterase for 8-10 hours when taken orally, and ~9 hours when transdermal patches are applied (Kandiah et al.). Around 40-170% of patients experience temporary symptom relief for 6-12 months ("Drug Treatments for Alzheimer's Disease"). Benefits include reduced anxiety, improved cognition (motivation, memory, and concentration), and enhanced physical movement, with side effects being loss of appetite, nausea, vomiting, diarrhoea, muscle cramps,

headaches, dizziness, fatigue, and insomnia (Raina et al.). However, after this period, symptoms gradually worsen. Galantamine elevates acetylcholine levels in the brain to improve cognitive function and memory in patients with neurodegenerative disease. Galantamine's exact therapeutic mechanism is unknown. However, scientists hypothesize that it enhances cholinergic function. Galantamine may increase the availability of acetylcholine in the synaptic cleft the selectively inhibiting cholinesterase, preventing acetylcholine from degradation (Kalola and Nguyen). Galantamine is also a unique drug in that it can release acetylcholine from presynaptic neurons, providing clinical significance to its mode of action. This classifies galantamine as an allosteric potentiator of α4β2 and presynaptic α-7 nicotinic acetylcholine receptors (Kalola and Nguyen). Studies have found that when the activity of nicotinic acetylcholine receptors is reduced in AD, galantamine can intervene to promote activity (Kalola and Nguyen). As a cholinomimetic agent, galantamine binds to nicotinic acetylcholine receptors at the allosteric site, releasing acetylcholine and enhancing the activity of serotonergic and glutaminergic neurons. In detailing the pharmacokinetics of galantamine, absorption is most effective at 8 to 32 mg/day. Its absolute bioavailability when ingested is 90%, and peak concentration occurs around 1 hour after ingestion (Kalola and Nguyen). The drug has a mean volume of 175 L at distribution (Kalola and Nguyen). In whole blood, 52.7% of galantamine is distributed to red blood cells. Galantamine is also permeable across the blood-brain barrier (Kalola and Nguyen).
Short-term-wise, ChEIs delay nursing home placement, improve quality of life, and improve cognition. Determining the long-term benefits of these inhibitors proves to be difficult due to the loss of follow-ups on patients in some studies. Long-term use of these inhibitors may slow cognitive decline, reduce the risk of myocardial infarction, and reduce the risk of strokes.

Partial NMDA memantine protects brain cells by blocking the effects of excess glutamate. When glutamate accumulates in the brain, neurotoxicity can be induced, leading to neuronal damage, synapse loss, and the buildup of Aβ plaques (Bukke et al.). Partial NMDA memantine is often recommended to individuals with moderate to severe AD who cannot take a ChEIs due to the inhibitor's side effects ("Drug Treatments for Alzheimer's Disease"). In the short term, partial NMDA memantine reduces clinical worsening and behavioral issues, and has a positive effect on language, memory, praxis, communication, and physical activity (Wilkinson). In the long term, memantine delays nursing home placement and enhances quality of life, especially when used in combination with ChEIs.

Anti-amyloid antibodies include Lecanemab and Donanemab. Lecanemab, which aims to lower levels of Aβ in the brain, is for patients with mild cognitive impairment or mild dementia due to AD (Rosen). Lecanemab can sometimes cause swelling or bleeding in the brain due to amyloid-related abnormalities (Rosen). Short-term effects include reduced cognitive decline through reducing amyloid and other AD biomarkers. The long-term effects of Lecanemab are still unknown and under study. Donanemab slows amyloid and tau accumulation. In the short term, Donanemab reduced the decline in physical activities in patients by 35% (Reardon). Additionally, it slowed the decline in memory and thinking abilities and induced microglial-mediated clearance of Aβ plaques ("Week 2, July 2024: An Update on Alzheimer's

Antibody Drugs"). In patients with early AD, Donanemab was able to slow amyloid and tau progression down at 76 weeks (Sims et al.).

Some AD treatments that are currently in progress include anti-tau antibodies, gene therapy, and stem cell therapy. Semorinemab and Tilavonemab are anti-tau antibodies that are currently being researched. Semorinemab showcases potential microglial activation, but does not have a significant impact on cognitive decline; research on Tilavonemab was discontinued due to its lack of efficacy(Cai et al.). Gene therapy targets nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF), apolipoprotein E2 (APOE2), and human telomerase reverse transcriptase (hTERT). In animal trials, these proteins can alleviate AD neuropathology (Ortega et al.). Neurons in the brain are able to respond to these growth factors. NGF seems especially promising: it stimulates cholinergic neurons, lasts anywhere from 7-10 years after gene editing, and increases cortical 18-fluorodeoxyglucose after treatment (Tuszynski et al., Castle et al., Thal, et al.). Stem cell therapy for AD is largely cell replacement therapy. Animal trials for stem therapy transplant human neural stem cells into animals. AD mice with stem cells had earning curves restored to resemble the control mice with normal learning and memory, and expressed 1000+ genes differently in memory centers (Fromson).

Parkinson's Disease

Parkinson's Disease (PD) is caused by progressive degeneration of midbrain dopaminergic neurons in the substantia nigra, a region involved in motor control (Damier et al.). The motor symptoms exhibited by PD patients—such as tremors, slowness, lack of coordination, and rigidity—are due to the loss of these neurons and a decrease in dopamine production (Ramesh and Perera). The PD treatment levodopa is used in combination with MAO-B inhibitors and/or COMT inhibitors.

Levodopa, a dopamine precursor, is the most common dopaminergic treatment for PD (Salat and Tolosa). Once levodopa enters the blood-brain barrier, the enzyme aromatic amino acid decarboxylase (AADC) begins dopamine synthesis to convert levodopa to dopamine (Nishijima and Tomiyama). In the short term, levodopa is able to manage PD motor symptoms such as slowness and tremors. However, some patients experience a return of motor symptoms during an "off" time, which can last between 15-60 minutes (Kantor). Many long-term effects of levodopa might also impact the quality of life of PD patients. For instance, some patients who have used levodopa for a long time develop dyskinesia (Kantor). Additionally, dopamine levels in that patient become increasingly dependent on more doses of levodopa ("Maximizing 'On' Time: Longer-Lasting Levodopa Therapy Options for Parkinson's Disease").

MAO-B inhibitors inhibit the monoamine oxidase B, an enzyme that breaks down dopamine in the brain. MAO-B inhibitors are able to delay the need for levodopa (Turnbull et al.). In the long term, MAO-B inhibitors can improve motor symptoms during an "off" time that PD patients experience with levodopa and potentially delay disease progression (Tan et al.).

COMT inhibitors inhibit catechol-O-methyltransferase, a body enzyme that deactivates levodopa before it enters the central nervous system ("COMT Inhibitors | Parkinson's Foundation"). COMT inhibitors prevent levodopa from breaking down, allowing for more levodopa to be converted to dopamine. This is helpful for managing the "off" period. In the long term, using COMT inhibitors with levodopa contributes to levodopa tolerance, meaning that lower doses of levodopa can be used less often ("COMT Inhibitors | Parkinson's Foundation").
Ambroxol, stem cell therapy, and gene therapy are current treatments for PD that are still under investigation. Ambroxol (ABX) is a cough medicine that has been shown to increase glucocerebrosidase (GCase) activity (Kopytova et al.). Gcase is an enzyme that is used to break down proteins. In PD, the accumulation of the protein alpha-synuclein disrupts dopaminergic neuronal function, leading to a decrease in dopamine production (Barkley). Additionally, Gcase activity is decreased. Currently, clinical trials for ABX are in phase 3 ("Phase 3 Trial of Ambroxol Is Underway"). Dopaminergic neuron progenitor cell product (bemdaneprocel) derived from human embryonic stem (hES) cells is currently being studied. These cell products were grafted into the putamen and were then able to produce dopamine, even 18 months after the grafting occurred (Tabar et al.). Gene therapy with the glial cell line-derived neurotrophic factor (GDNF) is currently being studied. The viral vector AAV2-GDNF is used to deliver the GDNF to the putamen, hopefully stimulating the long-term production of GDNF and restoring neurons affected by PD (Gilbert).

Methods and Materials

Literature Searching

A literature review was conducted using Google Scholar and PubMed databases to search for articles about clinical trials, reviews, and cohort studies. This search was applied to studies published between January 1998 and May 2025.

Effectiveness of Donepezil

Multiple clinical studies were conducted to determine the short-term and long-term effectiveness of donepezil. Participants in these studies experienced mild to moderate AD and were 60 years or older. These clinical studies were double-blind, placebo-controlled trials that lasted 12 weeks, 15 weeks, 24 weeks, or 30 weeks. The participants in these studies received 5 mg of donepezil a day for the first week. Then, their dosage of donepezil was increased to 10 mg/day. To gauge the effectiveness of donepezil, participants took the Alzheimer's Disease Assessment Scale-Cognitive Subscale (ADAS-cog) assessment to measure cognitive

impairment, the Clinician's Interview-Based Impression of Change Plus caregiver input (CIBIC-plus) to rate how quality of life changed, and several dementia rating assessments.

Effectiveness of Rivastigmine

Preclinical studies on the effectiveness of rivastigmine were conducted in vitro on male AChE knockout mice and male Wistar rats. Participants in clinical studies on the short-term effectiveness of rivastigmine engaged in a double-blind, placebo-controlled study for 6 months. These participants experienced moderate AD symptoms. During these clinical studies, these participants were given either 3, 6, or 9 mg of Rivastigmine a day, or they were given a placebo. Then, PET imaging and functional magnetic resonance imaging were used to determine how brain activity and metabolism in the study's participants were affected.

Effectiveness of Galantamine

Participants in the studies on the short-term and long-term effectiveness of galantamine had either vascular dementia or AD. These studies lasted 24 weeks and spanned across countries in Europe and North America. Participants in these studies either received 24 mg of galantamine per day or a placebo. These participants were initially given 4 mg of galatamine per day, and then the dosage of galatamine gradually increased to 24 mg/day in week 6. The ADAS-cog assessment with 11 items and 13 items and the CIBIC-plus assessment were given to participants. Additionally, participants were regularly monitored to track adverse events.

Effectiveness of Levodopa

Participants in the studies detailing the short-term and long-term effectiveness of levodopa were in the early stages of PD. They were initially given doses of 300-600 mg of levodopa per day. This dose of levodopa was increased to 1000 mg/day if the patient exhibited tolerance to the original dosage. Less than 12 months of treatment was considered the short-term period. In addition to the oral dose of levodopa, some participants were also given the COMT inhibitor entacapone. The participants assessed their motor symptoms using the Unified Parkinson's Disease Rating Scale (UPDRS). Additionally, the researchers followed up with the participants during semi-annual or annual visits. During these visits, the participants' levodopa dosage, motor symptoms, and quality of life were recorded.

Effectiveness of MAO-B Inhibitors

Participants in the studies detailing the long-term effectiveness of MAO-B inhibitors were patients with early PD who had received no treatment for at least 12 months. Additionally,

these patients had no impairment of balance. Trials that included patients with extreme motor fluctuations were excluded. The participants either received an MAO-B inhibitor—either selegiline or lazabemide—or were given a placebo. Selegiline was administered at a dose of 10 mg/day while lazabemide was administered at a dose of up to 100 mg/day. These trials lasted over a year. To evaluate the effectiveness of MAO-B inhibitors, the severity of impairment, disability, quality of life, the number of patients with motor fluctuations, and the number of patients with dykinesias were reported. To evaluate the safety of MAO-B inhibitors, the number of patients with adverse symptoms, the number of patients with withdrawals, and the total number of withdrawals were recorded. Additionally, levodopa was administered when a participant's symptoms worsened dramatically.

Results

The results obtained from this meta-analysis have many crucial implications for society and the future of neurodegeneration and the methods used to heal such diseases. As a result of the research conducted, further ideas on neuronal health in terms of neurodegeneration can be generated.

Alzheimer's Disease

Finding biomarkers for AD, particularly amyloid, tau, and phospho-tau levels, has allowed researchers to determine the main features of AD and identify if one has the disease. AD is now known to be caused by an accumulation of amyloid beta protein in the brain, leading to neural toxicity and the altered biochemistry of these beta proteins. Tau is another protein that is considered when researching AD. Tau is regularly used in assembling and maintaining microtubules; however, when abnormally hyperphosphorylated, it can disrupt cellular signaling and cause an imbalance of proteins within the nervous system. This understanding has led to the development of various medicines and treatments for AD.

Current treatments for AD are effective in managing and regulating the disease. These treatments include acetylcholinesterase (AChE) inhibitors, which target neurons and synapses to break down acetylcholine (ACh). ACh and AChE inhibitors are cholinergic and form most of the autonomic nervous system. Cholinergic medicines used in treating AD include donepezil, rivastigmine, and galantamine. Each of these medicines has been found to prevent acetylcholinesterase from breaking down acetylcholine in the brain, further improving neuron communication. It is reported that 40-70% of people with AD benefit from the use of these medications, emphasizing the potential of chlorogenic medications to develop in the future. Partial N-Methyl D-Aspartate (NMDA) Memantine is another drug used to treat AD, which protects brain cells by blocking symptoms of excess glutamate, a neurotransmitter released in

excess when the brain is damaged by AD. NMDA is beneficial in that it can substitute for those with moderate to severe AD who cannot take cholinesterase inhibitors. It can slow the progression of symptoms, and its side effects are less acute. Anti-amyloid antibodies can also be used to treat patients with mild cognitive impairment, although their effects are less well-known. Donanemab is also used to treat patients with early AD, slowing down amyloid and tau progression. Its symptoms are less apparent, but more clinical trials are needed to determine its effectiveness and symptom development. Treatments currently being trialed are anti-tau antibodies, for their slight reduction in tau levels; gene therapy, which can factor into nerve growth, brain-derived neurotrophics, and human telomerase reverse transcriptase to relieve AD neuropathology; and stem cell therapy for its potential to replace damaged neurons.

Parkinson's Disease

Similar to AD, PD is another neurodegenerative disease that results in the loss of brain substance. Midbrain dopaminergic neuron loss, in collaboration with decreased dopamine production and substantia nigra dysfunction are the causes of PD-related motor symptoms. This has led to the development of treatments that increase dopamine, where levodopa, MAO-B inhibitors, and COMT inhibitors are used. Further research and development are being conducted on ambroxol, stem cells, and gene therapy in treating PD. Stem cells are being used to increase dopamine in the substantia nigra, through the transplant of bendaeneprocel into the putamen to the neurotransmitter. Gene therapy, alternatively, was found to stimulate long-term production of GDNF, restoring neurons impacted by PD.

Summary

None of these developments in treatment could have been found without preceding knowledge on which neurotransmitters and proteins are impacted in neurodegeneration. Through consistent research, study, and discovery, the best treatments for neurodegeneration are being found and applied to save the lives of those living with AD and PD. With the hopes that treatments like stem cell therapy, anti-tau antibodies, and gene therapy present in restoring damaged neurons or reducing the development of tau in the brain, these therapies can be primary or alternative medications to use in substitution of those that cannot be used by patients; similarly to how NMDA is used for those who cannot take cholinesterase inhibitors.

Impact

Beyond just looking at the short-term and long-term effectiveness of treatments for AD and PD, this meta-analysis also draws attention to the need for treatments that help prevent neuron degeneration. Generally, existing treatments for AD and PD help improve the quality of life of the patient by delaying nursing home placement and improving cognition. However, the symptoms of AD and PD quickly resurface during an "off" period. One of the most popular solutions to this issue is increasing the dosage of medicine given to the patient. In the long term, the current treatments for AD and PD fail to address neuronal death that causes cognitive and motor decline. This shows that there is a need for more long-term studies on these treatments and a need for more research on potential treatments that focus on doing more than just addressing the symptoms of AD and PD. With more research being done on neurodegenerative diseases, the global health community is gaining a broader understanding of the neurodegenerative mechanisms surrounding AD and PD. This has led to research and development into treatments that aim to stop neuron degeneration as a whole. Stem cell therapy and gene therapy are currently being researched for AD and PD treatment. Currently, these new treatments remain experimental due to issues with delivery, immune responses, and cost.

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Hyperconnectivity Patterns in Psychological Disorders: A Literature Review of Connectivity Patterns within Depression and Anxiety Compared to Other Psychiatric Disorders

Madelaine Nguyen, Eva Strádalová, Akshda Sharma, Emily Castillo

Abstract

Hyperconnectivity is the increase in the strength of connections within brain regions and networks, regularly observed in the brains of patients with neurological or psychological conditions. Comparisons between connectivity in anxiety and depression and other psychiatric disorders have found their hyperconnectivity patterns to be vastly different. In mood-related disorders, hyperconnectivity frequently occurs in the brain's default mode network (DMN), the amygdala, and other brain regions involved in emotional regulation and cognitive processing. These patterns suggest heightened emotional regulation and cognitive dysfunction, highlighting the main symptoms of these mental conditions. Patterns are often observed through methods like imaging, which monitors the increased activity of neurons in various brain hubs, usually used for the diagnosis of brain disorders. By exploring trends of hyperconnectivity in psychiatric conditions, particularly the most common, anxiety and depression, offers the potential for earlier and more efficient diagnosis and treatment of psychological disorders. Hence, an understanding of the patterns and functions within these brain regions allows for an explanation of how hyperconnectivity is unique to each disorder. This paper aims to analyze how hyperconnectivity patterns in the brain of individuals with anxiety and depression differ from those with other psychological disorders, such as post-traumatic stress disorder (PTSD) and eating disorders (ED), and how different functions and reactions are impacted in the brain.

Key Words

Amygdala: either of the two areas in the brain that are linked to memory, the emotions, and the sense of smell
Anxiety disorder: Any of various mental disorders characterized by extreme anxiety and including panic disorder, post-traumatic stress disorder, and generalized anxiety disorder
Clinical depressive disorder: a type of affective disorder characterized by major episodes of depression without intervening manic episodes
Hyperconnectivity: functional overconnectivity, a phenomenon in the brain where there's an increase in the strength or number of functional connections between different regions and

networks—detected through brain imaging techniques such as fMRI (functional magnetic resonance imaging and EEG (electroencephalography).
Default mode network (DMN): a system of connected brain areas that show increased activity when a person is not focused on what is happening around them

Introduction

The human brain consists of complex networks that interact with each other to govern thoughts, feelings, and behavior. In normal subjects, the networks possess a delicate balance that results in emotional and cognitive potential. Recent research in neuroscience has, however, shown that in depressed and anxious individuals, there can be hyperconnectivity in which specific regions of the brain are excessively connected and hence process information incorrectly. This hyperconnectivity is most apparent in areas like the default mode network, linked to self-referential thinking and rumination, and the limbic system, linked to emotional regulation. This essay explains how the hyperconnectivity between these brain networks results in symptoms like shattered concentration, overthinking, and emotional fragility. Through examining the cognitive and biological effects of hyperconnectivity, we are able to construct a deeper understanding of the relationship between brain functioning and mental health, thus developing more effective interventions.

Literature Review

Hyperconnectivity between key brain regions has been increasingly associated with various psychiatric and neurological disorders and among these, anxiety disorders have been closely linked to abnormal connectivity between the amygdala and specific areas of the prefrontal cortex (PFC), including the inferior frontal gyrus (IFG), orbitofrontal cortex (OFC), and ventromedial prefrontal cortex (vmPFC) (Cha, et al). A study has shown that higher connectivity of the amygdala with the IFG, OFC, and vmPFC aids in maintaining performance during times of anxiety. As a result, altered connectivity with the amygdala has been linked with anxiety disorders, as these disorders may present with an inability to perform when placed under stress. Specifically, the connection of the ventromedial prefrontal cortex with the amygdala is crucial for emotional regulation during the decision-making process and the vmPFC is also involved in risk evaluation (Jaryd et al). The prefrontal regions are additionally involved in executive control, emotional regulation, and the modulation of threat responses. Furthermore, a positive correlation between amygdala and vmPFC activity has been observed during extinction recall following fear conditioning, further emphasizing its importance in updating memories produced in the time of stress. Dysfunction in these networks may be the basis of some anxiety symptoms seen in clinical populations (Gold, et al).

Depression is one of the most common mental health disorders, with over 300 million people estimated to suffer from depression (Stringaris). Characterized by persistent sadness, lack of motivation, and physical symptoms that interfere with everyday life, patterns in hyperconnectivity directly influence an individual's ability to perform tasks and emotional processing in depression disorder. Existing literature supports increased activity in the default mode network (DMN), which is linked to depressive symptoms such as negative self-perception and rumination. The DMN is a network of connected brain regions that are consistently active when the brain is not performing a task, including regions such as the medial prefrontal cortex (mPFC), posterior cingulate cortex (PCC), precuneus, and inferior parietal lobule (IPL). These regions are active during periods of rest and associated with tasks such as daydreaming and recalling memories. Moreover, the amygdala, a brain region involved in emotional processing, plays an essential role in emotional responses in depression. Observations demonstrate greater hyperconnectivity activity in these key regions, suggesting reinforcement of negative moods, (Wade-Bohlber, et al). Furthermore, the findings suggest that this increased activity exacerbates depressive symptoms of persistent negative thinking and consistent re-experience of distressing memories. Emotional regulation may be more prevalent in individuals with depression due to elevated activity in these emotion centers of the brain. Further research has investigated functional connectivity in individuals with major depressive disorder (MDD), presenting increased connectivity in the brain regions left superior frontal gyrus (SFG) and left posterior cingulate cortex (PCC), (Zhu, et al). These regions are associated with cognitive control, memory, and DMN network activity. Increased hyperconnectivity in these regions further support rumination and excessive self-focus in depression, some of its leading symptoms. Excessive connectivity in DMN networks, the PCC, contributes to repetitive thinking patterns, potentially resulting in pessimistic thoughts and promotes internal distress. Overall, these patterns of hyperconnectivity in depression are heavily linked to prominent depressive symptoms of excessive negative thinking and self-reflection.

Schizophrenia, often presenting with paranoia and psychosis exhibits distinct forms of hyperconnectivity. Increased functional connectivity between the hippocampus and amygdala in individuals with paranoia has been associated with heightened threat sensitivity and disrupted affective memory regulation. Paranoia has also been linked to enhanced connectivity between the OFC and medial PFC. Resting-state imaging studies reveal that patients with paranoia present with hyperconnectivity within frontal hubs of the default mode network (DMN), which is involved in internal self-awareness and mind-wandering. This may further impair emotion regulation (Kalin).

The ventral hippocampus, which plays a central role in affective memory and stress response, appears particularly vulnerable in paranoid states. Altered connectivity between the ventral hippocampus, the basal amygdala, and medial/orbital prefrontal cortices seems to be the cause of delusion formation and impaired stress regulation (Walther, et al).

Tourette syndrome (TS), another neurodevelopmental disorder frequently accompanied by psychiatric conditions, also reveals significant hyperconnectivity, particularly within limbic circuits. Approximately 82% of TS patients present with heightened anxiety and impulsivity. The insular cortex is involved in interoceptive awareness and emotional salience, and its hyperconnectivity with subcortical structures could contribute to the symptoms observed in Tourette syndrome (Temiz, et al).

Bipolar disorder is a psychiatric disorder associated with extreme shifts in mood swings and energy levels, resulting in manic and depressive episodes. Bodies of literature have found that DMN is a key brain network involved in the increased connectivity in bipolar disorder. Increased connectivity has been found in the particular regions, PCC, medical cortex, and other cortical regions, (Liu, et al). These findings suggest that self-processing and thoughts is heightened, with these regions being linked to inner and self-referential thinking. Furthermore, elevated DMN connectivity leads to rumination and self-focus, contributing to both depressive and manic episodes. Furthermore, the increase in activity may lead to cognitive dysfunction, followed by emotional dysregulation, disrupting emotional control and impaired executive functions such as memory in individuals with bipolar disorder. Moreover, a study published in the *Journal of Affective Disorders* by Gong Jiaying, et al, has found that hyperconnectivity in the salience network (SN) is increased in individuals with bipolar disorder. The SN is a brain network involved with detecting and responding to stimuli relevant to an individual's needs, including regions like the anterior insula (AI) and dorsal anterior cingulate cortex (dACC). Findings reveal increased connectivity within the left anterior insula, dACC, and other brain areas like the parietal cortex. This suggests heightened reactivity to emotions and stimuli, which contributes to bipolar disorder's main symptoms of manic and depressive episodes. Manic symptoms often include impulsivity while depressive symptoms present extreme emotional sensitivity, leading to frequent mood swings and instability. Moreover, over-responsiveness to stimuli may impair decision-making and executive functions within individuals, impacting their processing and overall cognitive performance. In essence, increased hyperconnectivity in the DMN and SN contributes to the severity of mood-related symptoms in bipolar disorder.

Post traumatic stress disorder, also called PTSD, is a mental health condition caused by an extremely stressful or traumatic event through witnessing or being directly a part of it, and difficulty recovering from it. Studies have explored hyperconnectivity patterns in individuals with PTSD, highlighting increased activity in the amygdala as a key brain region. Other brain regions involved include the anterior hippocampus, precuneus and posterior cingulate cortex (PCC), which are associated with memory encoding and self-referential thought. Specific patterns of greater functional connectivity between the amygdala and these brain regions are linked to a greater amount of intrusive memories, a common symptom of PTSD. Moreover, heightened connectivity between the amygdala and anterior hippocampus leads to intensified memory and trauma encoding, suggesting emotional memories are more easily triggered. Dysfunction in memory with frequent re-experiencing and difficulty distinguishing the past and present with PTSD flashbacks may be a result of connectivity with the DMN regions, PCC and

precuneus. The insula plays a critical role to various functions, including emotional regulation and cognitive functions. As a result, connectivity between the amygdala and insula may amplify fear responses through increased awareness of internal bodily processes, leading to sweating and a faster heart rate. This is commonly seen in PTSD responses where individuals experience emotional and physical distress due to intensified flashbacks that make them feel as if they are experiencing the traumatic event again. In addition, research has found that there is decreased functional connectivity in the medial prefrontal cortex (MPFC) in the dorsal DMN, suggesting impairments in emotional and cognitive processes, (Bao, et al). This results in persistent negative states and cognitive failure within PTSD patients, exacerbating the symptoms of emotional dysregulation.

Methodology

This research used a small-scale correlational and observational design to explore the association between hyperconnectivity in the brain and depression and anxiety symptoms. The research set out to explain patterns of connectivity that may be linked to emotional distress by following a literature review design. This research aims to synthesize current scientific findings on hyperconnectivity in the nervous system and its relationship to anxiety and depression. Rather than collecting new data, this study draws on existing peer-reviewed research to identify consistent patterns, neural mechanisms, and theoretical interpretations.

Over the past few years, scientists have been learning more about how our brains stay connected and how sometimes, they can become too connected. Hyperconnectivity refers to when certain parts of the brain are constantly talking to each other, even when they're supposed to rest or switch off. While that might not sound bad at first, too much brain activity in certain networks has been linked to mental health issues like anxiety and depression. With this base we started our research regarding the mentioned. To gather the information, we searched for reliable studies and reviews in trusted academic databases like Google Scholar, PubMed, ScienceDirect, and PsycINFO. We used keywords like: "brain hyperconnectivity and anxiety", "neural circuits depression", "default mode network and overthinking", "resting-state fMRI mental health"

Articles were only included that were: Published between 2010 and 2024, written in English, focused on human participants, peer-reviewed (so they went through a scientific quality check). We specifically looked for studies that used brain imaging tools like fMRI, EEG, or DTI, since those are used to actually measure connectivity in the brain. Articles were avoided that focused only on medications or treatments without any brain-related data, because the goal was to understand the "why" behind the emotions and not just treatments and procedures.

The Analysis Approach for this research was to Select studies that were reviewed and grouped based on: Type of neural network involved (e.g., Default Mode Network, Salience Network, Amygdala-Prefrontal pathways), Type of psychopathology studied (anxiety, depression, or both), Methodology used (resting-state vs. task-based imaging) Recurring patterns, converging findings, and gaps in the literature were identified to inform the discussion

of how hyperconnectivity may contribute to emotional dysregulation, rumination, and heightened stress sensitivity.

Results

Several studies have shown that hyperconnectivity between brain regions correlates with both the presence and the intensity of symptoms related to various psychiatric conditions, including anxiety disorders, depressive disorders, schizophrenia, Tourette syndrome, bipolar disorder, and post traumatic stress disorder. Anxiety disorders often observe dysfunctional connectivity of the amygdala and the prefrontal cortex, specifically the IFG, OFC, and vmPFC, while depressive disorders have been seen to show hyperconnectivity between the DMN, including the mPFC, PCC, and IPL. Notably, the amygdala has also been linked to emotional responses in depression, on account of the significant overlap and co-occurrence of the symptoms of anxiety and depressive disorders. Paranoia, a symptom of schizophrenia, similarly shows patterns of connectivity between the OFC and medial PFC, however, the connectivity is excessive, which differs from the altered and often decreased circuits of connectivity seen in anxiety. Anxiety is often present in Tourette syndrome, explaining the presence of PFC connectivity. It additionally presents with connectivity between the subthalamic nucleus and the striatal area of the subcortical structures of the brain. Bipolar disorder presents with varying symptoms in its depressive and manic states, resulting in increased hyperconnectivity in the DMN and SN. To summarize, though hyperconnectivity greatly varies with each condition, overlapping symptoms account for the resemblance in some hyperconnectivity pathways.

Discussion

The findings of this paper provide a review of existing research of hyperconnectivity patterns within anxiety and depression to other psychiatric conditions, suggesting similarities and differences that influence different symptoms and functions. Several brain regions associated with emotional regulation and cognitive processes exhibit increased connectivity within psychiatric disorders as a whole. Mental health disorders, or mental illnesses, are conditions characterized by disrupted mood, thinking, and behavior. The link between increased connectivity in regions impacting cognition and emotions and the definition of mental illness makes it clear to understand the neurological underpinnings of this relationship. The results may assist to further our understanding of hyperconnectivity patterns in a range of psychological disorders and improve clinical strategies to prevent exacerbation of symptoms of mental health patients.

The DMN is a network of interconnected brain regions that are active when the brain is at a period of rest, associated with internally focused processes such as daydreaming and mental processes. It is vital to self-referential thoughts, self-concept, social cognition, and memory. The

dysfunction of this network is highly prominent in mental health disorders, such as mood and anxiety disorders. It is a key region in a variety of psychiatric conditions, like depression, anxiety, and bipolar disorder, exhibiting similar activity patterns which lead to exacerbation of mood-related symptoms. Mood and emotional dysregulation is an overarching main symptom of psychological disorders, often leading to depressive and/or manic episodes seen in bipolar disorder and depression. Specific DMN regions, the PCC and precuneus have been found to have increased activity in PTSD, contributing to intensified emotional and fear responses within this disorder.

Areas within the prefrontal cortex, the brain region responsible for cognitive functions, are linked to abnormal connectivity with the amygdala in several disorders. Most prominently, these patterns in anxiety are involved with the IFG, OFC, and vmPFC. Increased connectivity in these regions suggest an inability to perform efficiently during periods of stress, suggesting an inability to perform executive functions within anxiety. Furthermore, a multitude of brain regions associated with cognitive control, including PCC and SFG, have been found to have increased connectivity within MDD. This further highlights the similarities between anxiety and depression connectivity patterns, which may lead to their similar symptoms of inability to carry out tasks properly and dysfunction with memory. As well as schizophrenia, a condition linked with paranoia, is associated with enhanced connectivity between OFC and the medial PFC. The hippocampus, a region critical for memory, is additionally involved in connectivity patterns in schizophrenia, indicating impaired memory and motor coordination which leads to difficulties within memory and reality perceptions.

The insula, also called the insular cortex, plays an essential role in emotion and memory processing, and regulation of the autonomic nervous. system The autonomic nervous system regulates involuntary processes including heart rate and blood pressure. Likewise, the insular cortex has exhibited increased activity within TS and PTSD, contributing to physical symptoms of rapid heart rate and excessive sweating during fear responses. The increase in connectivity with the insula to the amygdala is more prevalent in these disorders compared to other psychological disorders, highlighting their recurring physical symptoms.

A range of limitations should be considered when interpreting the findings of this study. Several studies and literature were analyzed to interpret similarities and differences in hyperconnectivity patterns within psychological disorders, which may have included their own study limitations. The sample sizes were not limited to age or gender, broadening results but the lack of specificity may make it difficult to categorize the findings to a conclusive statement. Specific methods should be considered when selecting studies for future reference when analyzing hyperconnectivity patterns, as well as a selection of disorders. Psychological disorders include up to 200, and cannot be all included in comparison to anxiety and depression, with anxiety and depression being differential.

The findings can be used to support further research in hyperconnectivity activity and patterns within psychiatric disorders, potentially assisting in the development of improved treatment strategies and methods. Mental health conditions is one of the top causes for disability

in individuals, with an estimation of 26% of Americans ages 18+ suffering from a diagnosable mental illness in a year, (John Hopkins Medicine). Many people suffer from more than one disorder at once, making further treatment methods increasingly relevant. Understanding the neurobiological aspects of psychiatric disorders through a variety of clinical methodology and data analysis allows for policy and medical practice implications to reach healthier outcomes.

Conclusion

Hyperconnectivity of the nervous system offers a framework to account for much cognitive and emotional symptomatology in depression and anxiety. Where brain networks become overactive or fail to communicate, this can lead to repetitive negative modes of thought, increased reactivity to stress, and difficulties with emotion regulation. This research indicates the importance of understanding mental illness, not just as chemical imbalance, but also as disruption of brain network dynamics. With improving neuroscience, the study of such patterns will better diagnose and treat mental illness. More research can bring new avenues for the recovery of loved ones suffering from anxiety and depression with the restoration of healthy brain function.

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Neuroplasticity: How the Brain Reprograms after Injury

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Abstract

Neuroplasticity enables the brain to reconstruct functions, structure, or connections based on environmental influences or brain damage. This process is evident throughout several brain injuries, particularly stroke. When the brain is deprived of proper blood flow, a clot may form, and result in a cerebrovascular accident (CVA). Several animal studies show a significant role in action potentials and repetitive stimuli. The body responds to cerebral malfunction with a natural approach to repair damages, neuroplasticity. The understanding in stroke recovery requires an understanding of the immediate effects from stroke, the neural pathways that play a significant role during brain development, the long lasting effects of stimuli, and the relation between motor rehabilitation and neuroplasticity.

Literature Review

Neuroplasticity, the brain's phenomenal aptitude to rebuild itself, is the ability for the nervous system to respond to stimuli with modifications in the brain's structure, function, or neural connections. The brain is an intricate system that consists of billions of linked neural pathways, neurons, and subsystems within it. Neuroplasticity's complex process involves neuron regeneration, potentiation, cortical connections, synaptogenesis, and several other underlying categories that serve a contributing role in it. Synaptic plasticity plays a significant role in strengthening the bonds and connections between neurons through experience-dependent and long term adjustments in the brain (Puderbaugh). Whether driven by a brain injury or the challenge of learning a new language, the impact of these adaptations allow the body to systematically adjust based on its needs. When it comes to traumatic brain injuries (TBI), neuroplasticity takes on a crucial position in determining the effectiveness of rehabilitation (Zotey). Disturbances caused by injury lead to disruption in basic neural connections, but unveil unique processes such as equipotentiality (Puderbaugh). Both researchers Zotey and Puderbaugh conclude that the brain reorganizes and redirects neurons in adjustment to an impaired function in neuroplasticity, while also analyzing the effects of stroke in brain rewiring. The cortical layer surrounding the cerebrum is an exemplary example pertaining to the mechanisms of neuroplasticity. As a result of a cortical infarct, tissue death, an instantaneous remodeling occurs in distant areas of the brain (Dancause). Vicariation enables the cerebral cortex to reorganize other parts of the brain to compensate for functions that were not originally assigned to that region (Puderbaugh). Therefore, cortical infarct demonstrates the traits of vicariation with the brain's preparation in distant, unaffected areas. Although Puderbaugh briefly discusses vicariation, Dancause finds that PMv axons after M1 hand injuries respond to injuries with

adjustments and axonal sprouting. The in-depth research article on Extensive Cortical Rewiring conducted an experiment of eight squirrel monkeys categorized into groups to discover the effects of an ischemic injury. By discovering the corticocortical connections of PMv with various other areas after Primary Motor Cortex (M1) injury, it illustrates the efficiency of the Cerebral Cortex to reorganize. Cortical injuries allow for improved environments that are suited for anatomical reorganization and axonal sprouting (Dancause). The Primary Motor Cortex is necessary for motor function, thus the interplay between M1 and PMv axons provide researchers with a reference point for motor rehabilitation in patients with Traumatic Brain Injuries such as Stroke.

Erin M. Schuman describes Long-term potentiation (LTP) as an activity-reliant plasticity that is thoroughly studied in the hippocampus, and is triggered by postsynaptic neuron depolarization. Potentiation, in the context of neuroplasticity and Traumatic Brain Injury, relates to synaptic transmission and excitability. With a cohort of rabbits, research concludes that longer-lasting potentiation results from an increase in synaptic transmission productivity and neuron excitability (Bliss and Lomo 331). When the studied neurons experience repetitive stimulation, a stronger neural connection and action potential is observed. The brain indicates long-term effects from reacting to stimuli, which shows how the brain responds with spikes of potentiation depending on surrounding activity. Puderbaugh references Bliss and Lomo's trial using a rabbit hippocampus under recurring stimuli–the examination revealed that synapse plasticity has the ability to improve based on several factors of stimuli (exercise, environment, repeated stimuli, motivation, drugs, and others). When prolonged potentiation develops, the risk for neurodegenerative diseases decreases and neuron regeneration is encouraged (Puderbaugh). In correlation to poststroke physiology, skill specific training induces potentiation, increasing neurological activity and can indicate signs of improvement in a specific impaired skill (Dimyan 76). Researchers Puderbaugh and Dimyan reinforce the positive impacts of extensive potentiation when comparing the effects to neurological diseases. These discoveries allow for apt room for future utilizations of potentiation in rehabilitation.

A stroke, otherwise known as a "brain attack", is an umbrella term. Generally, the category of stroke indicates brain damage as a result of an occlusion or haemorrhage of blood vessels that supply the brain (Lo, Eng, Dalkara, Moskowitz 399-414). Most typically, a stroke is a result of damaged blood flow from the formation of a blood clot (1). Although the OHSU Brain Institute and research article Mechanisms, challenges, and opportunities in stroke disagree in stroke's severity ranking–both recognize the daunting toll that stroke presents to patients. The neurologically damaging disorder causes one-third of Cerebrovascular disorder patients to suffer with permanent repercussions of motor disability (Dimyan 76). Directly after a stroke injury, the first forty-eight hours typically entails cell death and loss of cortical pathways. During this time-frame, the brain attempts to restore the affected region with secondary neuronal networks. Within weeks poststroke, support cells form, and cortical pathways shift from being inhibitory to excitatory–new connections develop, and plasticity adaptations begin. Months after the Traumatic Brain Injury, the cerebrum utilizes axonal sprouting to reconstruct and neaten the

damaged area (Puderbaugh).Once ischemia occurs in the brain, neuronal death in neural tissues occurs. When a tissue dies in the cortical vicinity, a remodeling of other distant areas across the brain occurs (Dancause). This indicates a potential connection between the impaired region and distant regions, which may lead research to the theoretical idea of vicariation and equipotentiality. These processes may be apparent in stroke patients, specifically the weeks post-stroke, when the brain initiates primary plasticity. Current research offers thorough insights on the stages of poststroke patients, the relationship between damaged and unaffected neural regions, and neuroplasticity in post-stroke patients.

With current spotlights into neuroplasticity, the dynamic brain's power to adapt has been a focus in recent studies pertaining to stroke patients. Factors such as sex, age, brain disease, and network function hone into the variability of neuroplasticity (Voss). The brain disease categorizes general disorders and injuries that may permanently affect the nervous system, such as a stroke. Approximately one-third (fifteen to thirty percent) of stroke patients suffer continuous disabilities and hardships after their first "brain attack" (Dimyan 76). In poststroke physiology, studies have shown that patients with specific activity and potentials at certain areas that experience neuroplasticity as a result of stroke, tend to more likely recover completely (Dimyan 76). An original idea established by Konorski, cortical plasticity occurs after an injury and changes the brain. Neuroplasticity occurs in the affected region of stroke, therefore the recovery process is dependent on the quality of specific activity and potentials. Immediately following a stroke, the brain carries out neuroplastic alterations such as axonal branching, collateral creation, and shifting brain activity to healthier areas (Zotey). Dimyan identifies that MrI and PeT scans are not able to identify whether abnormal brain activity after a stroke is inhibitory or excitatory, which restricts the efficiency in examining a Traumatic Brain Injury. With setbacks and gaps throughout the current post-stroke care, harnessing plasticity remains a pivotal intervention in post-TBI care. Current techniques like pharmacological interventions, nerve growth factors, nervous system stimulation, stem cells, and neuroprosthetics continue to expand as neuroscience grows rapidly (Dimyan 76).

Methods and Materials

This review draws upon peer-reviewed articles across public databases such as Google Scholar, PubMed, and National Library of Medicine (NIH).While Adaptive Neuroplasticity in Brain Injury Recovery: Strategies and Insights focuses on reputable research paper databases as the primary study, the vast majority of studies rely on past animal models. For instance, Puderbaugh mentions a discovery found in four adult patients' MRI scans, indicating increased activity in the left motor cortex after an ischemic stroke of the right main motor cortex. In scientist Zotey's research article, 120 initial articles were found (n=120), but was ultimately narrowed down to sixty-seven full text articles for qualitative synthesis (n=67). The selected articles were published between January 2000 to September 2022 to ensure modern data. The selective sequence to choose specific articles allows our research paper to cite various other qualifying papers. Contrary to the methodology from Zotey, Puderbaugh references the

discovery of Bliss and Lomo while studying a rabbit hippocampus. Although the research paper does not conduct an individual trial, the described rabbit brain simulation in Long-lasting potentiation of synaptic transmission in the dentate area of the anaesthetized rabbit following stimulation of the perforant path utilizes a cohort of eighteen adult rabbits (n=18) in both sexes. The equipment consisted of NaCl-filled glass micro-electrodes, stimulating electrodes built from electrolytically grinded tungsten, and an oscilloscope to display the rabbits' repetitive stimuli response. The recognition of long-term potentiation and significance of stimuli in potentiation is prevalent in stroke recovery and neuroplasticity as potentiation involves the strengthening of neurons and connections. Research utilizing Bliss and Lomo's research has allowed the neuroscience community to associate potentiation methods to motor rehabilitation.

Results

In consequence of the recent heavily-concentrated research across neuroplasticity, the trials included significant discoveries among the mechanisms and utilizations of cerebral plasticity. In the study Neuroplasticity in the Context of Motor Rehabilitation after stroke, the review refers to innate physiological and anatomical plasticity directing motor function gain after a stroke. The recognition in primary areas that undergo neuroplasticity enables the promotion of rehabilitating mechanisms. Pharmacological, neuroprosthetics, and brain computer technology indicate techniques for plasticity promotion, further harnessing neuroplasticity for future stroke treatments (Dimyan 76). In correlation to promoting plasticity, rodent models of stroke illustrate axonal reorganization, the role of synaptogenesis, task-specific training, and amphetamine treatment (Dimyan 76). With our current knowledge of the regions of the brain that experience reconstruction in poststroke patients, the possibility of predicting poststroke recovery success is probable. Specific activity and potentials throughout impacted areas from neuroplasticity can be measured to demonstrate the effectiveness of natural reconditioning. Electrophysiological and neuroanatomical tensor imaging display the corticospinal integrity significance in the acute stage of stroke recovery, with the local motor cortex circuitry being notably influential during the healing process (Dimyan 76). The paper Extensive Cortical Rewiring experiments on adult squirrel monkeys to prove that PMv axons surrounding lesions undertake axonal sprouting as a result of M1 injury. This trial demonstrates the cortical connections and neuroplasticity in the ventral premotor cortex when a connected area is damaged.

In Zotey's research team database, the cohort discovers the cellular mechanisms of neuroplasticity, reaction to traumatic brain injury, and rehabilitation methods using neuroplasticity. As a result of the condensed exploration, the article mentions restoring strategies with brain plasticity such as motor integration, auditory rehabilitation, memory training, cognitive remediation, and memory training (Zotey). Although concepts involving equipotentiality and vicaration are theoretical, MRI scannings after an ischemic stroke in the right main motor cortex measured shifted increased activity to the other motor cortex (Jaillard et. al.). The imaging of four adult patients proves the idea of brain reorganization to substitute for a missing function. Most significantly, an experiment including rabbits under continuous brain

stimulation concluded a timeframe of potentiation after the stimuli was induced (Bliss and Lomo 331). The activation of neurons from repetitive stimuli in perforant path fibres of the hippocampal formation region revealed the strengthening of neural connections and action potentials (Bliss and Lomo 331). A spike of positive potential indicates the activation in the perforant path synapse, which leads to a positive potential in the cell body following depolarization (Bliss and Lomo 331). Presynaptic triggers the postsynaptic neuron, which ultimately decreases the requirement of energy necessary to command reactions to stimuli (Puderbaugh). Therefore, the synapse will theoretically improve, and the synapse plasticity is able to improve based on external factors. Overall, the examination on the rabbit hippocampus uncovers the effectiveness of stimuli to action potentials, and the significance in long-term potentiation.

Impact

The advancements of neuroplasticity promotion plays a pivotal role in neurological rehabilitation and biomedical engineering. The intention of initiating modern rehabilitation methods is for increased patient satisfaction, stimulating neuroplasticity for medicinal use, and inducing improved activity and participation (Dimyan 76). With the goals of neurological treatments for rehabilitation in mind, techniques such as auditory rehabilitation, memory training, cognitive remediation, and sensory motor integration enable patients to explore various treatments as opposed to being strictly restricted (Zotey). Even though the utilization of neuroplasticity brings ethical concerns, leading discoveries concentrated in the mechanisms of stroke, neuroplasticity, synaptic plasticity, diaschisis, and hundreds of varying topics may correlate to an increase in developed target therapies for motor function regain and recovery (Puderbaugh). Currently, the mirror therapy approach applies a physical reflection that covers an amputated limb to aid phantom limb pain (Puderbaugh). Specific therapies such as mirror therapy provide relief for patients and stimulate recovery due to the explicit research conducted on phantom limb treatments.

Trials such as the Long-lasting potentiation of synaptic transmission in the senate area of the anesthetized rabbit following stimulation of the perforant path open up additional exposure on the long-term effects of stimuli. The data provides an outlook on long-term potentiation after repetitive stimuli, which revealed the ability for synaptic improvement (Puderbaugh). The indicated range of long-term potentiation displayed thirty minutes to ten hours of effect after the initial conditioning (Bliss and Lomo 331). This experiment branches into differing areas like post stroke neuroplasticity as it may uncover the speed plasticity occurs following a brain injury. By understanding Traumatic brain injury, the publicity for prevention and potential symptoms spread across the internet to increase exposure for common injuries. A recorded 795,000 citizens in the United States of America experience a stroke, so improving treatment methods has the capability to care for millions of individuals worldwide (1).

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The Neuroscience of Dance: How Movement Enhances Brain Function

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Abstract:

As a structured form of movement, dance offers a unique lens into how motor activity can shape and support brain function. This review synthesizes recent interdisciplinary findings at the crossroads of neuroscience and dance, underscoring its role in promoting neural health across motor, cognitive, and affective domains. Despite growing interest, the precise mechanisms by which dance influences neuroplasticity and emotional well-being remain unexplored. This paper consolidates current evidence on how learning and practicing dance routines enhance brain plasticity, particularly by reinforcing neural circuits involved in coordination, attention, and memory. Through the analysis of neuroimaging and behavioral studies, the review examines dance's engagement with rhythm, spatial-temporal processing, and complex motor sequencing, and how these contribute to improved executive function and creative cognition. Findings show that dance practice stimulates the release of neuromodulators such as dopamine, oxytocin, serotonin, and endorphins, activates the limbic system, and improves emotional regulation and resilience. Furthermore, the paper highlights therapeutic applications of dance in neurological rehabilitation, including Parkinson's disease, post-stroke recovery, and Alzheimer's disease, positioning it as a non-pharmacological intervention with wide-reaching benefits. Overall, dance emerges not only as an art form but also as a potent, evidence-based modality for enhancing brain function and fostering cognitive and emotional vitality throughout the lifespan.

Literature Review:

Brain Plasticity & Coordination

Dance routines are a combination of complex steps put together in a set sequence for each movement. The timing and coordination of these dance steps, while maintaining the necessary technique of the specific movements, initiate routines that require memory. Through constant practice, neural connections associated with memory strengthen, particularly in regions of the brain responsible for executing such movements. Practicing also engages various parts of

the brain simultaneously, promoting neuroplasticity and creating and reorganizing synaptic connections.

When you learn new dance routines, your brain activates the motor cortex and cerebellum, which strengthens pathways coordinating movements. Practicing the dance routines multiple times activates the hippocampus and prefrontal cortex, which strengthens your ability of procedural memory. The complexity of the rhythm and music, which syncs with the movements, enhances interhemispheric communication within the corpus callosum. Using sensory integration to understand visual cues and proprioception strengthens the neural circuits that manage balance. Each person's brain is activated and utilized uniquely based on the skills they possess when working with movement. The strengths and abilities associated with producing dance routines in a unique pattern sustained for each individual body, skill set, and experience level affect the parts of the brain that are in effect when performing.

Lastly, the emotional flow of a dance using facial expressions and body language activates the limbic system, which promotes emotional regulation and social cognition. Overall, every part of the brain is activated differently when a dancer dances with passion in their movements, and each step promotes the increased health of a dancer's brain in a unique pattern.

Emotion & Mental Health

Dance can release a stream of neurotransmitters. In addition to Endorphins, the body’s natural painkiller, dopamine, serotonin, and oxytocin, the neurotransmitters in charge of sensations of pleasure and happiness, are increased by physical activity.

Neurotransmitters are brain molecules that aid in message transmission throughout the body, and they alter our mood based on the activities we are doing. A special combination of Dopamine, Oxytocin, Serotonin and Endorphin (DOSE) is released during dancing.

Dopamine

Dopamine is linked to enjoyment, pleasure and reward. This hormone stimulates our motivation and curiosity when we engage in or anticipate doing an enjoyable, spontaneous activity, such as dancing.

The release of dopamine is also associated with routine activities, as these activities regularly activate dopamine pathways. Consequently, rhythmic movement such as dancing can become a dopamine stimulus.

Oxytocin

When we connect interpersonally, oxytocin gets released. This connection can include a friendly touch, eye contact or even coordinated movement. It promotes positive social feelings such as connection and trust, and diminishes anxiety. Accordingly, group dance and movement mirroring strengthen interpersonal connections between dance partners and crew members.

Serotonin

Serotonin is the zen chemical. It promotes emotional contentment and ease, acting as a protective shield against anxious moods and depressive tendencies, in addition to improving sleep quality. It is also sometimes referred to as the "happy chemical," as it contributes to a positive mood and overall well-being. As dancing stimulates the brain to secrete serotonin, it increases its circulation around neural pathways, which slows the ageing of the brain.

Endorphins

Endorphins are secreted during exercices and movement. The euphoric feeling associated with intense activities is a result of Endorphins, as they inhibit undesired sensations, allowing for an enhanced mood. In other words, they are the pleasure of the brain, allowing us to limit pain and fatigue, resulting in a sense of lively energy which helps us push through during dancing and enjoy the activity.

The DOSE chemicals do not act individually; they combine and influence one another to create the cycle of happy hormones and positive emotions in the brain.
Accordingly, Dancing is a holistic activator of the brain's core happiness and resilience chemicals. It stimulates the release of dopamine by rewarding the brain with pleasure and motivation, enhances oxytocin through social bonding and coordinated movement, boosts serotonin to promote emotional calm and mental well-being, and triggers endorphins that elevate mood and reduce physical discomfort.

Consequently, dancing can serve as a stress reliever. Dance's physical motions have also been demonstrated to lower tension, anxiety, and depressive symptoms as it stimulates the release of the 4 DOSE chemicals.

It also helps to relieve stress and enhance how the mind perceives the outside, improving sleep quality. In addition, dancing can reshape our self-image and make us feel more comfortable in our bodies, as it helps slow brain ageing by developing new neural connections, especially in regions involved in executive function, long-term memory, and spatial recognition.

By learning new moves, we can acquire self-confidence and boost our mind-body connection, which can act as a coping strategy.

Regularly, it improves self-esteem because it helps people become more conscious of their bodies and increases coordination, flexibility, and body awareness. It encourages you to be fully present, stay grounded and let go of tension and anxieties.

Cognitive Benefits

Executive functions are a collective term for cognitive functions which allow for the purposeful control of behaviour in your life and permit abstract solving of problems and adaptation in new situations.

Dancing incorporates all three of the foundational components of executive function. Dancing incorporates working memory, flexible thinking and self-control. When a person participates in dance, they must engage in cognitive processes such as learning and remembering

choreography; flexibly adapting to unexpected changes; and maintaining attention or focus as they process multisensory stimuli, such as music, movement, and changes to their environment-using these fundamental cognitive skills.

In one empirical study with 4-year-old children in street dance classes over eight weeks, there were significant improvements in tasks that measured inhibitory control compared to a control group. This demonstrates the impact of dance on the brain. While dance may slow age-related declines in cognitive processing, improvement in attention regulation, working memory, and cognitive flexibility implies that dance is a method of cognitive maintenance in older adults (Chichinina et al., 2022, vol. 12, p. 838).

Spatial awareness is an individual's understanding of their physical surroundings and their position within them. It involves understanding your position in space relative to the other objects or people in your surroundings. Dance develops awareness since it requires strength in movement, along with control of movement.

Early childhood education with dance may improve spatial awareness, reasoning, and even math skills, since children learn to "move" in their spaces. Additionally, concepts like ballroom dancing activate the motor cortex in the brain, which can build spatial awareness and hand-eye coordination, as dance requires movement, coordination of the body, and spatial awareness.

Dancing nurtures creativity by building people's capacity to express both their emotions and ideas/intentions through movement. This creative process involves different regions of the brain associated with sensory, motor, cognitive, and emotional processing. There is empirical evidence that dance-related creativity can provide benefits to executive functioning in early childhood. This demonstrates the connection between ideas of creativity and cognition.

Via timing, rhythm, and movement patterns, the brain's functioning can be enhanced. Dancing enhances executive functions by constantly challenging the brain to manage timing, coordination and decision-making in real time. When a person dances, they must remember a series of steps, shift attention quickly between the music and their body movements. They also have to quickly adjust to changes in rhythm and tempo. All of this activates working memory and cognitive flexibility. The brain's prefrontal cortex, which is responsible for planning and controlling actions, becomes highly engaged as dancers synchronize their movements with the beat. It also helps inhibit automatic responses in favor of intentional ones. This continuous mental exercise strengthens executive functions like self-control and problem-solving, making dance not just a physical workout but a powerful cognitive training tool.

Dancing also enhances the brain's spatial awareness and creativity by constantly challenging it with complex movement patterns. As dancers move through space, they must continuously monitor their body's position relative to the floor and other dancers. This strengthens the brain's parietal lobe, the area responsible for spatial reasoning. Navigating dance patterns and formations improves the sense of body position and spatial memory. This helps individuals become more aware of their surroundings. In addition, the creative aspect of dance, such as improvisation, stimulates the brain's default mode and sensorimotor networks. These are

linked to divergent thinking and novel idea generation. These combined challenges force the brain to integrate sensory input, motor coordination, and imaginative processes, making dance a unique activity that boosts both spatial and creative capacities.

In conclusion, dancing has many benefits on the human brain and causes some significant and positive effects on its executive functions, spatial awareness and creativity.

Therapeutic Uses

"Dance is my type of therapy" is a frequently heard phrase among dancers. It may seem sceptical to refer to dancing as therapy; however, dance is increasingly being implemented as a therapeutic intervention for individuals with neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD). In these sessions, dance is considered a multisensory therapy which targets both neurological and psychosocial deficits. Unlike traditional exercise, dance combines many aspects: movement, rhythm, music, memory, and interpersonal interaction. This usage of dance in therapy offers a comprehensive but also an enjoyable approach to neurorehabilitation.

- In Parkinson's disease, dance therapy addresses core impairments in an individual's motor skills. This type of therapy helps engage brain circuits involved in timing, coordination, and balance. Rhythmic auditory stimulation (RAS), frequently seen in dance itself, aids the human brain in terms of external timing cues. The overall process helps facilitate smooth and coordinated movement as it compensates for impaired internal timing mechanisms caused by basal ganglia dysfunction. Moreover, this has been shown to improve gait (movement in limbs) symmetry, stride length, and postural control. Additionally, dance routines often involve shifting one's weight in a certain way, turning, and generally changing direction. These skills directly combat the freezing of gait and postural instability common in PD. Over time, these repeated movement patterns have the potential to enhance neuroplasticity, leading to improved motor skills even outside of an individual's typical dance sessions.
- In Alzheimer's disease (AD), dance is utilized in a non-verbal therapeutic mode, which stimulates cognitive function as well as emotional expression. The combination of music and movement activates neural networks. This includes networks involved in memory, attention, and emotional regulation. In terms of attention and memory, an example is participation in choreographed dance routines. These routines need to be manually learned, so they require short-term memory and focus, which supports cognitive engagement and helps delay any functional decline. Moreover, due to music being processed in brain regions which are often preserved longer in AD, dance therapy helps stimulate memories and positive emotions. Thus, dance is a powerful tool that should be considered for not only mood enhancement but also social connection.

Dance therapy is a method that provides a safe and engaging space for both self-expression and social interaction. For instance, in a group-based format, common in dance

therapy, this method builds community and emotional support, which can significantly improve overall well-being and help reduce feelings of agitation and apathy. Furthermore, dancing in a group setting also helps reduce isolation while increasing self-esteem and promoting a sense of agency for that individual. Utilizing this form of exercise as a therapy method is often more enjoyable and motivating than traditional exercise. This promotes long-term adherence and increases the likelihood of sustained benefits. Its adaptability across physical ability levels also makes it accessible to a wide range of patients, from early-stage to more advanced cases.

To sum up, dance offers a holistic, evidence-based approach that improves physical coordination, cognitive function, emotional well-being, and social engagement in individuals with neurodegenerative diseases. Its effectiveness lies in its ability to integrate multiple therapeutic domains into a single, motivating intervention that is both functional and meaningful.

Case Study

This research is based on a case study analysis of an existing scientific investigation by Jeong et al. (2005). It explored the psychological and neurochemical effects of dance movement therapy (DMT) on forty 16-year-old middle school female students in Korea with mild depression during 12 weeks. By measuring psychological distress and neurohormones, it was found that the dance movement therapy has increased the concentrations of modulated plasma serotonin and dopamine, improving the negative psychological symptoms. This indicates possible therapeutic benefits for the decrease in depression observed in the dance movement therapy group.

Methods and Materials

Our team did not conduct the study; rather, we analyzed the design, tools, and results of the original peer-reviewed research to understand the methodologies applied in dance-related neuroscience.

The original study involved 40 adolescent participants (ages 16–18), all diagnosed with mild depression. These individuals were randomly divided into two groups:
1. a dance movement therapy group (n = 20) with no intervention
2. a control group (n = 20)(DMT) participated in 12 weekly sessions of dance therapy over three months, with each session lasting 60 minutes. The dance sessions were designed to combine structured and expressive movement exercises aimed at emotional expression and bodily coordination.

To assess changes in mental health, the researchers used two standardized self-report tools:

- The Beck Depression Inventory (BDI) is used to measure the severity of depressive symptoms.

- The State-Trait Anxiety Inventory (STAI) is used to evaluate both short-term (state) and long-term (trait) anxiety levels.

In addition to psychological assessments, biological markers were analyzed. Blood samples were taken before and after the 12-session therapy cycle to measure the concentrations of dopamine, serotonin, and cortisol.

The increase in dopamine and serotonin signified an enhancement in mood, while cortisol levels acted as a biomarker for stress.

ANOVA was used in the statistical analysis. This test compares the means of multiple groups to determine the effects of therapy.

This scientific technique provides quantitative and molecular proof of dance's impact on teenage emotional and neurochemical health.

Results

Following the completion of the 12-week dance movement therapy (DMT) program, participants had significant emotional and physiological alterations. The measurements of Depression and anxiety levels by the Beck Depression Inventory and the State-Trait Anxiety Inventory displayed a significantly lower rate in the DMT group, but the control group exhibited no change.

Biochemical research found higher dopamine and serotonin levels, as well as lower cortisol levels, indicating a better mood and less stress. As Jeong et al. previously reported, "dance movement therapy positively influenced both emotional responses and neurohormone modulation in adolescents with mild depression".

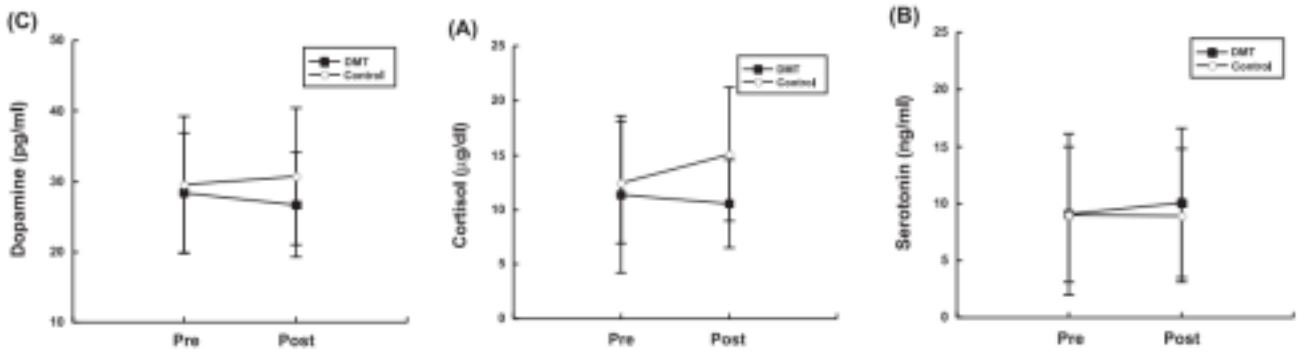

Figure 1. Effects of dance movement therapy (DMT) on plasma concentrations of (A) cortisol, (B) serotonin, and (C) dopamine. There were group × time interaction in serotonin [$F(1, 38) = 9.8$, $p < .005$] and dopamine concentrations [$F(1, 38) = 27.0$, $p < .001$].

Impact

The case study by Jeong et al. (2005) provides significant support for the broader literature on dance's neurological and emotional effects. The observed increases in dopamine and serotonin and decreases in cortisol in adolescents following 12 sessions of dance movement therapy are consistent with the "DOSE" neurochemical model presented in our review. This demonstrates that dance not only improves mood but also changes brain chemistry, reducing stress and supporting emotional regulation.

The findings also support the mechanisms discussed in sections 1.1 and 1.2 of our review, which explain how coordinated, rhythmic movement activates neuroplasticity and enhances memory and attention networks.

Furthermore, the research supports section 1.3 on cognitive benefits, which states that dancing improves executive function and spatial awareness through repeated and challenging sequences of movement. Another aspect is that the increases in mood and neurotransmitter modulation may indirectly improve focus, memory, and learning ability.

These findings also support the therapeutic uses discussed in Section 1.4. If short-term dance intervention may modify neurohormones and promote emotional health in adolescents, its implementation in therapeutic settings such as Parkinson's, Alzheimer's, or stroke rehabilitation would be transformative.

However, the Jeong et al. study had a few shortcomings. The sample size was limited, and the intervention was brief. Future research should explore long-term impacts, use neural imaging data, and expand to diverse age groups and dancing genres. Furthermore, controlled comparisons of group vs. solo dancing or choreographed vs. improvised formats would help us better understand which components are most useful.

Conclusion

Dance is a fascinating mix of art and biology, presenting as a structured yet expressive way to move. This study indicates that dancing enhances neural systems' activity, helping the brain stay flexible and improve emotional health and resilience.

According to research, including Jeong et al. (2005), dancing is closely associated with the release of neurotransmitters: dopamine, oxytocin, serotonin and endorphins. Consequently, it improves executive functioning and helps in emotional control by activating the limbic system.

Dance is also a transformative way to health betterment as it combines rhythm, spatial awareness, and creative expression. It is an evidence-based way to help people in healthcare and education. It can help teenagers' mental health and people with neurodegenerative diseases, among other things.

In essence, dance neuroscience is more than just a scientific curiosity; it is a new field with rapidly growing implications. To unlock dance’s full potential in brain health, interdisciplinary collaboration between neuroscientists, educators, and artists is essential.

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Out and In: The Environmental And Biological Impact on the Development of a Teenager's Mind, And the Future of Our World

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Lastly, congrats to the rest of the class, as we finish the first half of 2025 with new learnings and lessons.

Thank you so much!

Abstract

In this paper, the researchers will present evidence on how a teenager's environment and biological features play in their mind. Topics like Nature VS Nurture will be shown and reveal which one is the correct answer. To add, the importance of knowing a person's surroundings and its relevance today are mentioned. To see how external factors impact the behavior of an individual, the researchers conducted an online survey that questioned teenagers about their childhood, personalities and environment. Everything will be summarized at the end of the document and present how these learnings can help the future of the world.

Literature Review

People tend to say that "experience is the best teacher," implying that individuals had a lesson learned from either mistakes, activities, or both that they can use today, so that they prevent

accidents from happening. These experiences can impact many lives so greatly that they will be a constant reminder to the individuals, those memories cannot easily be forgotten, so what will happen if already in a young age, the individual will be trapped in a box of bad influences, how would a child identify if something is bad or good? How would a child know its consequences?

In this digital age, influences are brewing more than before. The easy access of education, opportunities and news is beneficial, however the accessibility of inappropriate content, scams, and fake news has increased with it. This improvement of media can be a double-edged sword, sadly a lot of the younger generation can easily acquire and much more easily consume these data as adults could. The uproaring rise of AI or Artificial Intelligence is another tool that can be overused and misused by users, like generative AIs (e.g. ChatGPT, Bard). The advancement of technology can make our future brighter than in the past, but the chance of it losing its spark is equal to it, since every pro seems to have a con added (example, artists losing their jobs over AI due to it being able to do the same work and replace them)[1]. Another reason for the research's purpose is the popular debate topic of Nature VS Nurture, both sides of the argument are understandable and logical, which leads to a lot of discourse over which side is more 'correct'. We hope in this research we will have a deeper understanding of how does the environment plays a big role in a child's mind. We must also understand why this is relevant today and how we can help to contribute in a brighter future.

Nature VS Nurture

Nature in definition by the Oxford Languages is the basic or inherent features of something, especially when seen as characteristics. On the other hand, Nurture is defined by the sum of the environmental factors influencing the behavior and traits expressed by an organism, mentioned by Merriam-Webster. The debate started when science and philosophy contradicted each other.

The larger investigation on genetics was first coined by Francis Galton, the younger half-cousin of Charles Darwin, after reading Darwin's book On the Origin of Species when he proposed his idea of eugenics. This inspiration ended up making Galton write the Hereditary Genius. Much later, 19th century scientists would study French children for their preparedness for school; they did this since they believed they could predict intelligence depending on the skills inherited by family or parents. They focused on studying the impact of genetics and biology, prioritizing it rather than focusing on the social and environmental influences. While heredity was being prioritized, philosophers Jean-Jacques Rousseau and John Locke independently concluded that humans are born as blank slates (tabula rasa) and dependently rely on experiences to improve intelligence. 20th century behavioral psychologists carried these thoughts, stating that events taken place during childhood effects greatly compared to the impact of the person's genetic makeup.

Ever since then, multiple experiments were performed to identify which factor (Nature and Nurture) is more impactful than the other. The two major experiments were studying twins who were separated from birth or early childhood and studying how the environmental influences impact the genes, this type of study is called epigenetics.

For the first experiment, Galton was the first ever scientist who studied twins and differ nature and nurture. Based on his study, he states that genetic makeup or imprint is larger than nurture in all areas (health, intelligence, appearance) Later, in 1883 he invented the word eugenics— which is a set of beliefs and practices that highlights or improves the good quality human traits rather than the bad ones. Many of Galton's claims are not debunked by newer and better experiments. Eventually, researchers would establish twin registries identifying twin births until their death to take note of their development and circumstances. Such as the Swedish Twin Registry and the Twins UK Registry. Two types of twins that were studied were: identical and fraternal twins. Identical twins both share the same set of genes, but fraternal twins share almost half of the same genes. The differences between the types are essential, for example, if identical twins are more likely to suffer from high or low cholesterol levels than fraternal twins, it could lead to an explanation that genes play an important role during a person's development.

The Minnesota Center for Twin and Adoption Research conducted a research where more than 137 pairs of twins (mayde triplets) were separated and given to different parents which resulted in most twins growing up, having the same or similar personalities and interests as each other. Another twin experiment was whether adolescent marijuana use drops IQ to decrease or are adolescents at more risk to smoke marijuana are likely to have their IQs to drop. Researchers discovered that the only twin who used marijuana IQ dropped meanwhile the other twin's IQ dropped as well, following their other half. This concluded with the answer that IQ dropping due to marijuana use is not related to the activity (smoking) itself, instead its the possibility or vulnerability of it dropping present before taking marijuana.

Epigenetics is the study of how cells control gene activity without the DNA changing and is closely related to neural plasticity since it changes the way brains think and adapt as the person grows. Epigenetic changes modify DNA whether to turn on or off genes, or activate certain genes and leave out the rest. Turning on genes is called 'gene expression' and turning them off is known as 'gene silencing'. Gene expression is created when information coded in a gene turns into a function, this transpires by the transcription of RNA molecules that code for proteins or non-coding RNA molecules that work for other functions. External factors such as smoking, exercise and diet can activate gene expression. Epigenetics constantly changes most of the time, our epigenetics back then are not the same we have now. For example, an adolescent's behavior today was different when they were a child. Some epigenetic changes do not change as genetic information passed down throughout generations, this is called epigenetic inheritance. An

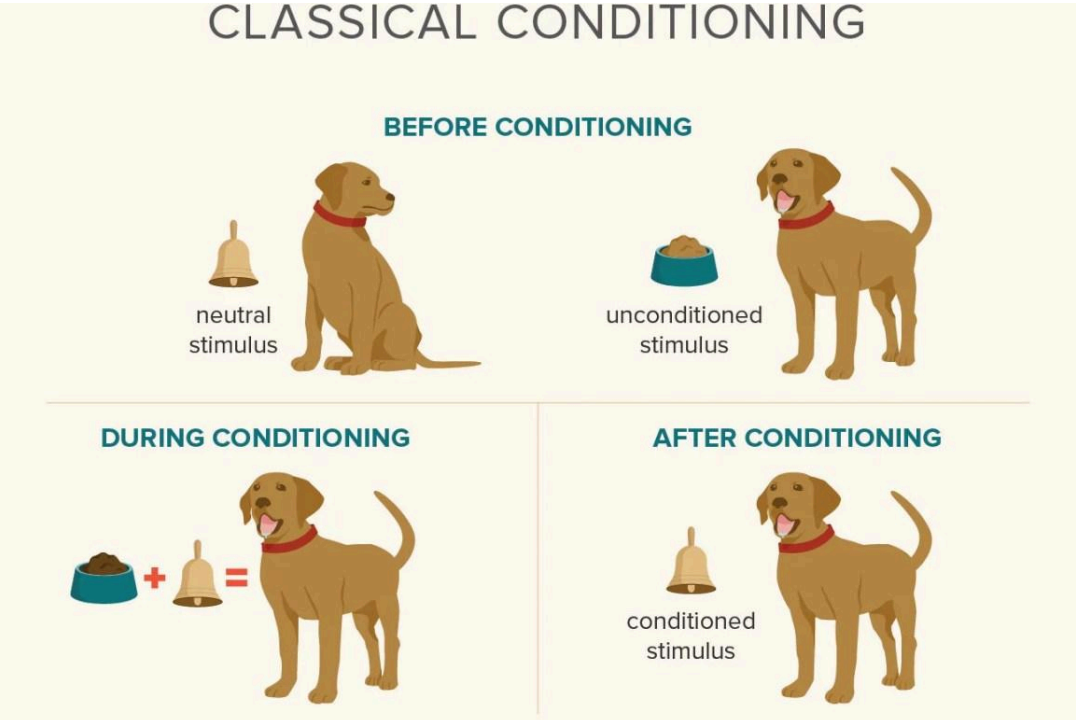
example of epigenetic inheritance is family trauma, such as PTSD or mental health issues. Many children of Holocaust survivors had PTSD and people with low cortisol are likely to have PTSD. Epigenetics will be important for psychological theory— Behaviorism.

The Importance of Knowing our Surroundings

Behaviorism is based on the concept that behaviors are obtained by interactions with the environment. John B. Waston parroted, the father of behaviorism: "Give me a dozen healthy infants, well-formed, and my own specified world to bring them up in and I'll guarantee to take any one at random and train him to become any type of specialist I might select—doctor, lawyer, artist, merchant-chief and, yes, even beggar-man and thief, regardless of his talents, penchants, tendencies, abilities, vocations, and race of his ancestors." Waston knew he was going against the facts, but so did his advocates. There are two main types of behaviorism: Methodological behaviorism and Radical behaviorism. Methodological behaviorism suggests observable or seen behaviour is studied scientifically and that mental states and cognitive processes do not add to the understanding of behavior. The first type is the same idea of Watson's beliefs. Radical behaviorism is a theory that behaviour can be understood by one's past and current surroundings for good and bad. This was created by psychologist, B.F. Skinner.

Classical conditioning is a practice used for Behaviorism where a neutral or unconditioned stimulus and an occurring or conditioned stimulus are paired, causing a reaction to the subject. Afterwards, even without the occurring stimulus, the subject will have the same reaction to the neutral stimuli. In Fig. 1, an example is shown where psychologist Ivan Pavlov used the relationship between dogs and food. With the first stimulus (food) and second stimulus (bell ring) is given to the dog, a salivation response occurs due to the food, but once the first stimulus is not in the presence of the dog, it still activated a salivation response. This type can also indicate how a phobia is formed.

(Fig. 1, An example of Classical conditioning, image from Simply Psychology)



There are step-by-step parts in this experiment. The first section is acquisition, a response is set and nourished. Multiple factors like the importance of the stimulus and timing of when it is presented plays a valuable role in how the subject can add a link to both stimuli. This creates generalization. Next step is called extinction. This happens when you repeatedly show the conditioned stimulus without the unconditioned stimulus, the subject slowly forgets their conditioning. This causes discrimination.

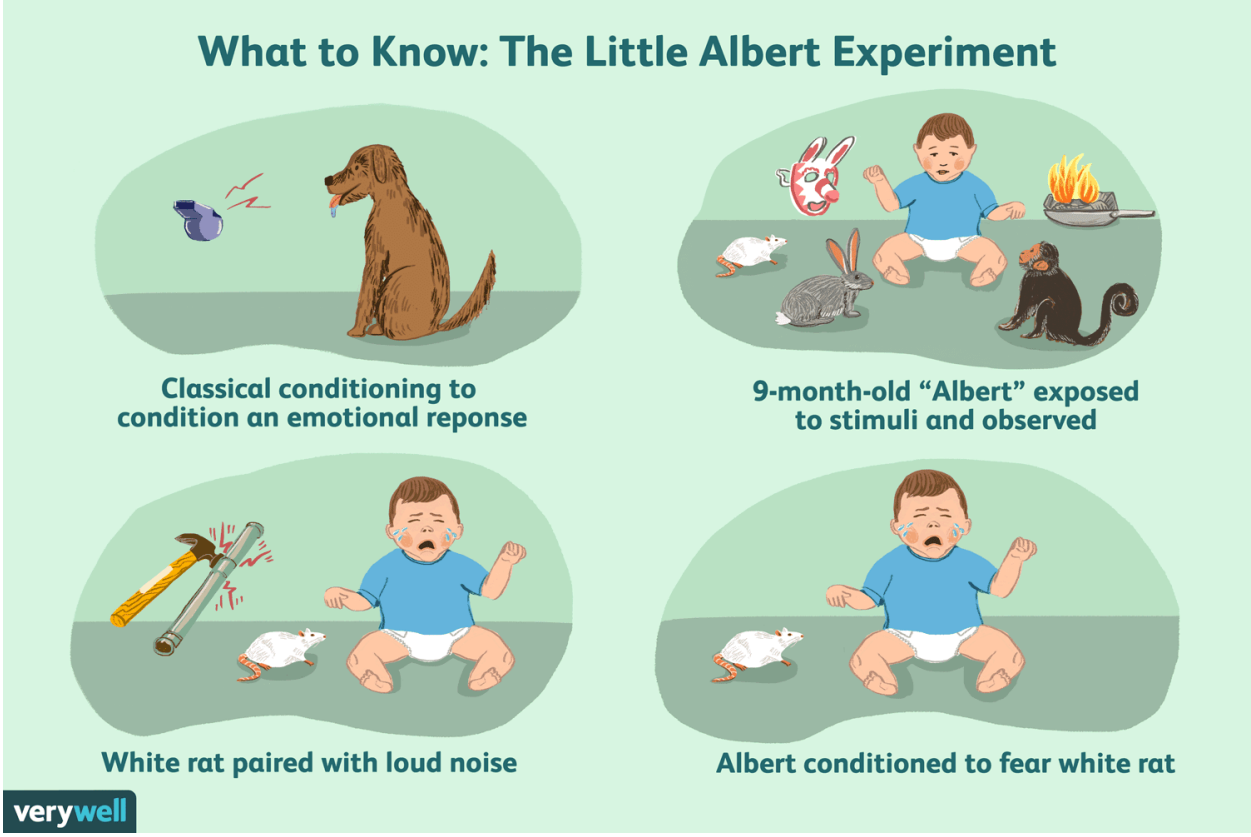
Operant conditioning, also referred to as instrumental conditioning, is a learning that happens by reinforcement and punishment. A cause and effect-type of learning. This uses the past to decide options with an idea of what will follow if that action is done. Timing also has a big impact on this type of behaviorism, reinforcement should be continuous or partial for operant conditioning to be quickly effective.

Behaviorism can be helpful in multiple aspects in daily living, but more focused on mental health. Behavioral therapy started by the ideas of behaviorism. This improved people to change problematic thoughts and behaviors, which improved mental health. Intensive behavioral intervention, behavior analysis, token economies, and discrete trial training are practices in therapy that came from behaviorism.

John B. Watson and Rosalie Rayner made a controversial and infamous experiment, The Little Albert experiment and followed Ivan Pavlov's experiments with humans, where the baby was

paired up with multiple items or stimuli. A white rat, rabbit, monkey, masks, and burning newspapers were the all stimuli, yet the baby did not show fear. Until, Watson combined the white rat with a noise of a metal pipe being hit using a hammer. The baby immediately cried, and once the loud noise stopped and the white rat was shown again, he cried and tried crawling away (Fig. 2).

(Fig. 2, The Little Albert Experiment, image from Verywell Mind)



Despite behaviorism being old and outdated, it is a good basic step to understand how our environment impacts us and our actions. People tend to act a certain way due to the fact they will know what will happen next if an action happens based on the past and due to an intuition of what will happen because of a stimulus. Initially, behaviorism goes against the claims of earlier 19th century scientists who believed everything in a person can be predetermined by their family genes. However, is it true that multiple aspects of a person (intelligence, health, appearance and many more) are inherited, but the environment shapes or polishes those aspects for better or worse.

Relevance Today and Its Impact on The Youth

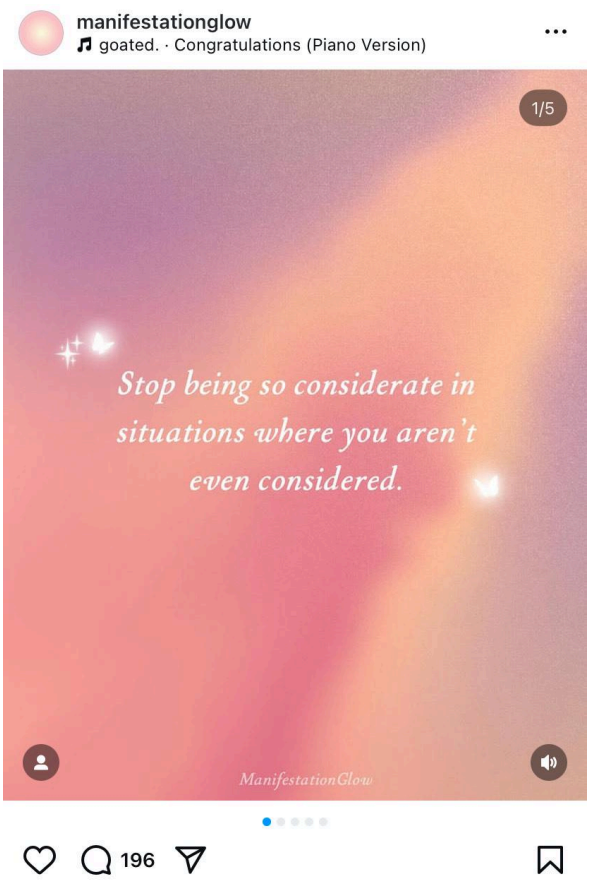
GPTZero states that nearly half or 46% of people aged 18-29 use AI on a weekly basis, while Aithor says almost 80% of Gen Z professionals, aged 18-21 use AI tools for over half of their work tasks. A survey conducted by Digital Education Council Global AI Student Survey, 86% of students already use AI in their studies. A research by Michael Gerlich called 'AI Tools in Society: Impacts on Cognitive Offloading and the Future of Critical Thinking,' this statement is found in the conclusion: "Younger participants who exhibited higher dependence on AI tools scored lower in critical thinking compared to their older counterparts. This trend underscores the need for educational interventions that promote critical engagement with AI technologies, ensuring that the convenience offered by these tools does not come at the cost of essential cognitive skills." Arun Luthra's article 'Is AI Shaping the Next Generation's IQ, Especially in Kids?' proclaims AI can help with education, providing immediate feedback and summarizing complex topics, but over-reliance is a concern as AI can lead to a decline in traditional learning methods (e.g. memorization, manual problem-solving) which some are essential for cognitive development.

AI cannot be used without a mobile device or more commonly, a smartphone. Mobile devices make receiving information easier than before, which the future generation has easier access to unlimited sources. Generation Alpha (2010-2014) is the first generation to have advanced internet access. Even a phone or any device is addictive. Cell phone addiction refers to a compulsive and excessive use on mobile devices that causes negative consequences in multiple parts of life. Most 13-year-olds have smartphones and almost half acknowledge they are addicted to it. Phone addiction can lead to negative effects in the brain, like sleep deficit, lower concentration, creativity blocks, aggravated ADD, anxiety, reduced cognition, stress, loneliness, insecurity, impaired relationships, poor grades, and psychological disorders. A study in Korea created in 2017, the average smartphone usage was 2.7 hours a day and for a week was 15.79 hours. CNA Insider made an experiment not long ago and uploaded it to YouTube. The title of the videos are called 'Hooked On Screens: Can Five Teens Survive 10 Days Without Smartphones | No Screen, No Life - Part 1' and 'Testing Five Teens After 10-Day Digital Detox: How Will They Perform? | No Screen, No Life - Part 2.' Before the detox started, the researchers did cognitive tests and gave the teenagers brick Nokia phones. After 10 days they will do the cognitive tests again to see if their brain activity is more focused and better, and then they will receive their phones back. Overall, all participants had better results in the tests and had a higher brain performance compared to their pre-detox. In 2 months, all the five contestants lowered their screen time after the experiment. All teenagers admitted they have been productive during and after the detox.

With mobile devices being addictive, a factor to that addictiveness is social media. The average use of social media platforms is 2 and ½ hours each day. Which is more than ⅓ of the total

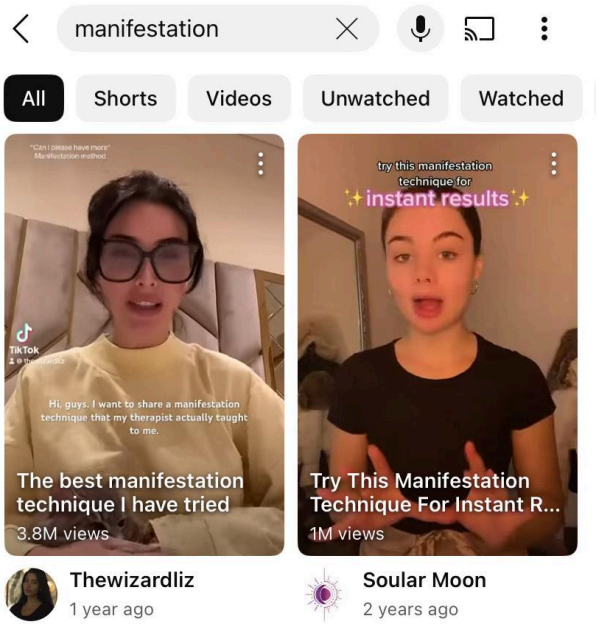
online time. A viral trend called Manifestation is in every social media platform (Tiktok, Instagram, Facebook, YouTube) and uses the law of attraction by William Walker Atkinson. It uses visualization, positive affirmations, and setting specific intentions. Manifestation is practiced by thinking and feeling of winning or getting a goal. The process of manifestation can actually cause the person to act a specific behavior to get that goal, which ends up having the person to get what they have manifested. Manifestation is linked to neuroplasticity as it rewires our brain to think, say, act, and feel differently. Some examples of the viralization of manifestation (Fig. 3, Fig. 4, Fig. 5, Fig. 6, Fig. 7, Fig. 8) are below:

(Fig. 3, A post for manifestation on Instagram, post from @manifestationglow)



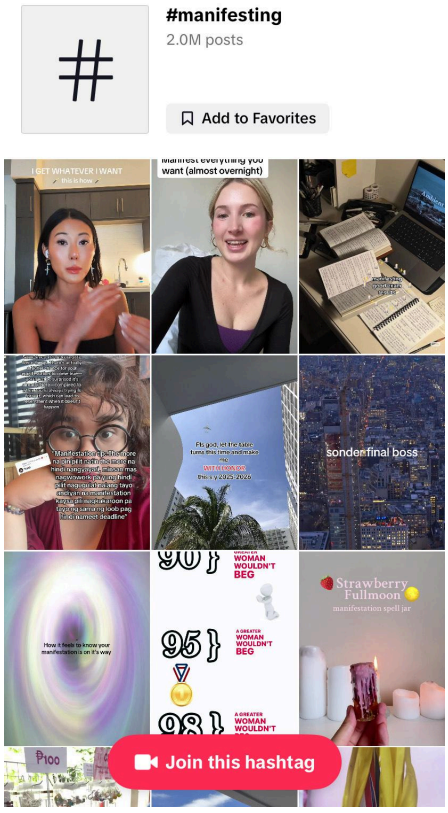
(Fig. 4, A post for manifestation on Instagram post from @manifestassembly)

(Fig. 5, the results if typed ‘manifestation’ on YouTube)

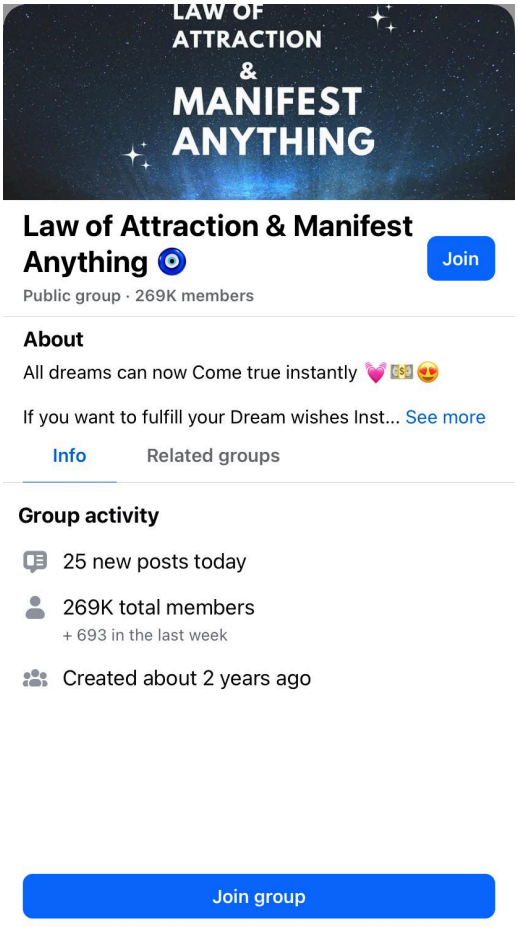


(Fig. 6, a video on YouTube with 11 million views)

(Fig. 7, #manifesting on Tiktok)



(Fig. 8, a Facebook manifestation group with 269 thousand members)



Methods

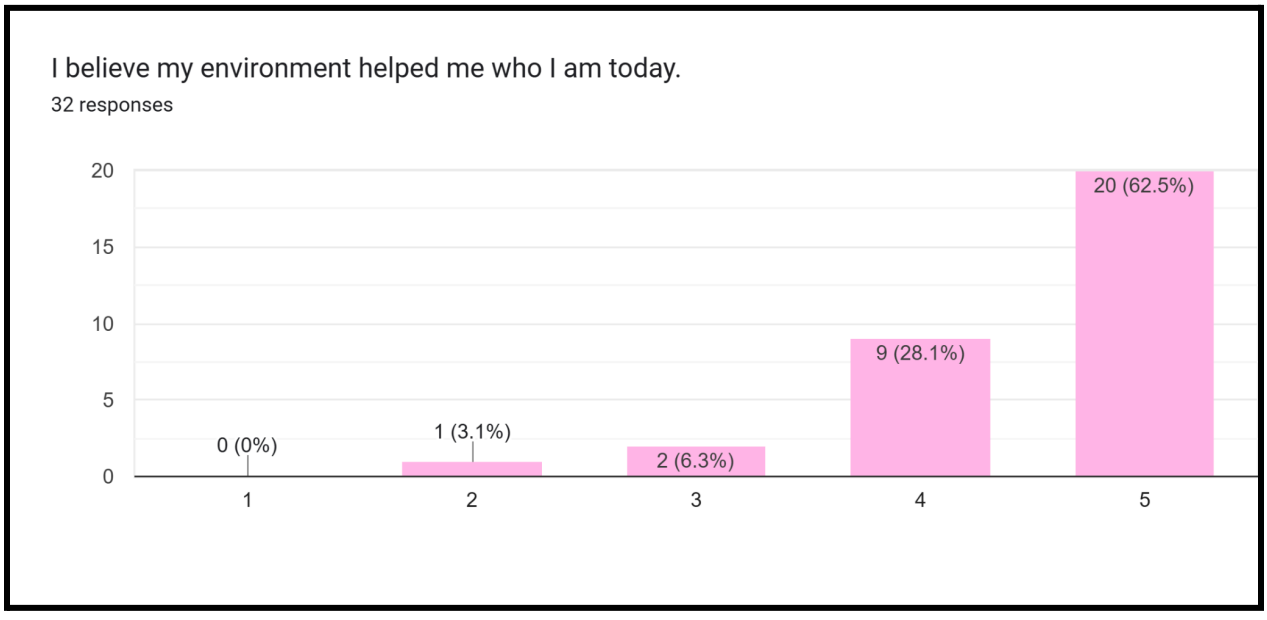
This study was conducted through the use of an online survey (Google Form) where participants (specifically teenagers) rate the question by 1-5 stars depending if they strongly agree (5 stars), agree (4 stars), neutral (3 stars), disagree (2 stars), or strongly disagree (1 star) with the question. Each question asks the individual about their childhood, if any environmental factors influence their choices and lifestyle, and what the participant thought of themselves as naturally intelligent/reliable. The survey was administered and forwarded to social media sites (Discord, Instagram, Tiktok) and amongst teenage peers of the researchers to allow for a larger generalizability factor of whether or not outside factors such as social media and environmental play impacts the development of a child's mind. Age, race, and gender of the participants were not accounted for due to the nature of the survey being anonymous.

Results

(All of the questions on the Google Form were only centered around the Nurture VS Nature topic.)

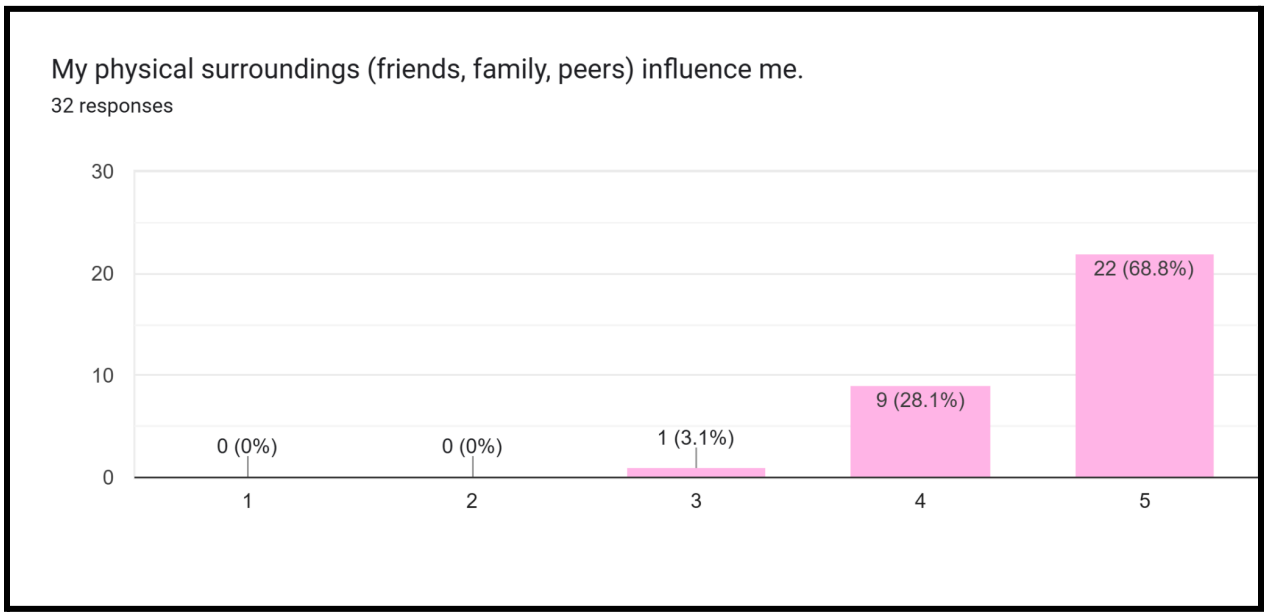
As of June 14, 2025, 32 participants answered the form.

(Fig. 1, database for question 1 in the survey sent)



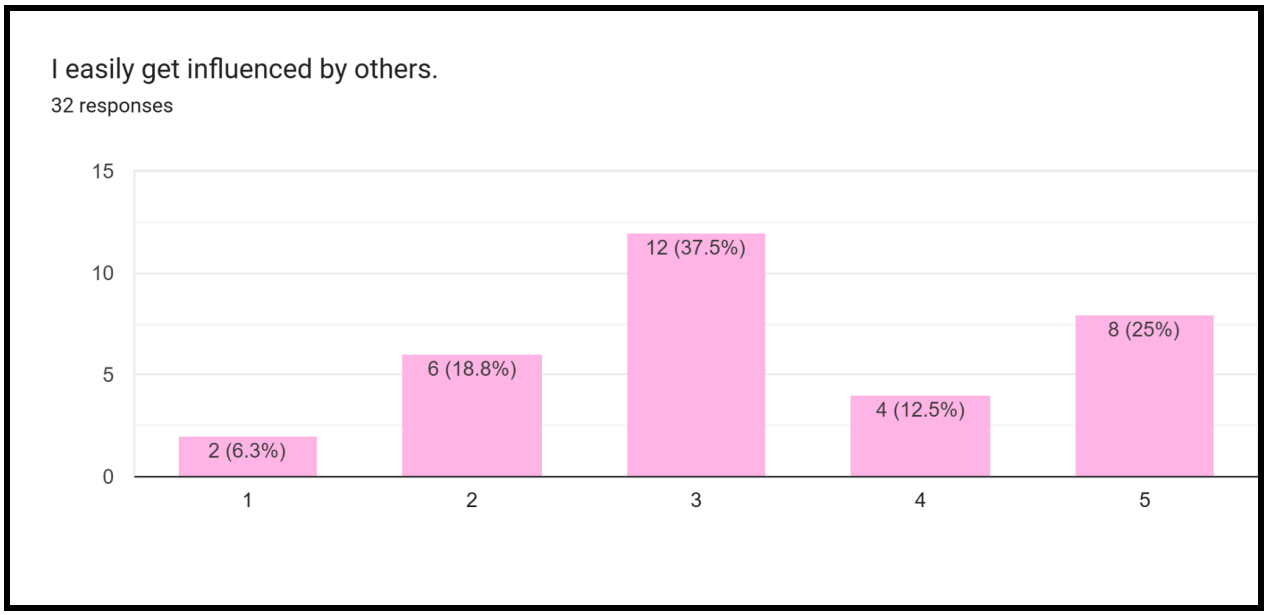
In Fig. 1, the question that participants answered was “I believe my environment helped me who I am today,” 62.5% (20 people) answered Strongly Agree (5 stars) while 28.1% (9 people) answered Agree (4 stars). 6.8% (2 people) input Neutral (3 stars) and 3.1% (1 person) said Disagree (2 stars).

(Fig. 2, database for question 2 in the survey sent)



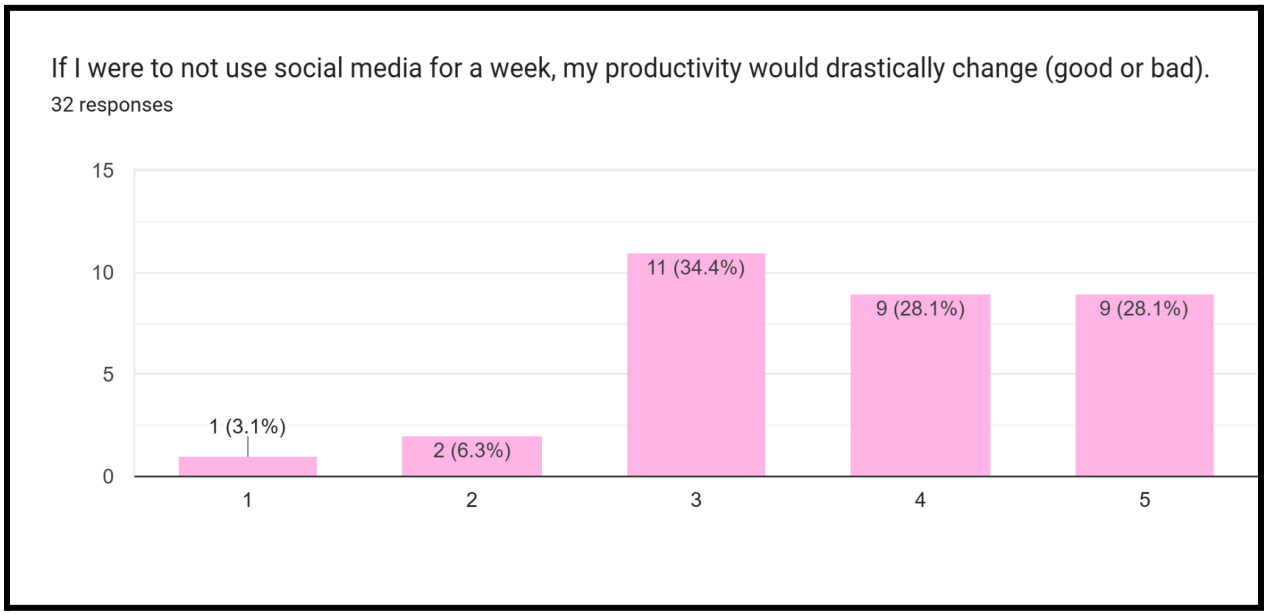
In Fig. 2, participants answered the question: “My physical surroundings (family, friends, peers) influence me.” 68.8% (22 people) said Strongly Agree (5 stars) and 28.1% (9 people) said Agree (4 stars). 3.1% (1 person) only said Neutral (3 stars).

(Fig. 3, database for question 3 in the survey sent)



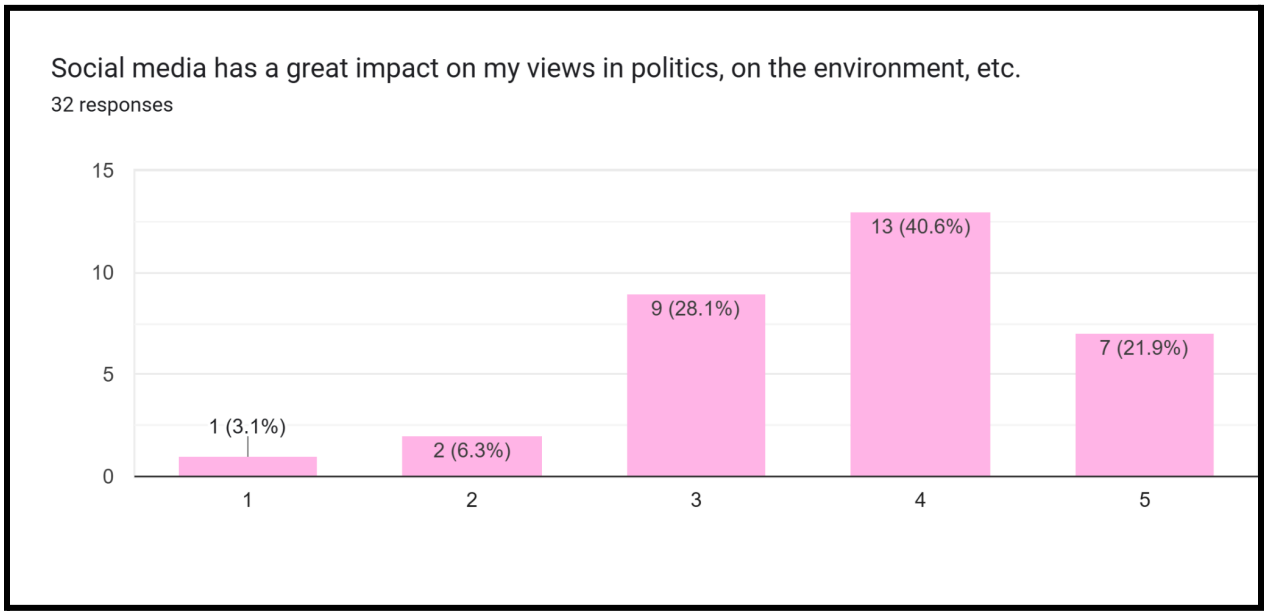
In Fig. 3, participants answered the question: "I easily get influenced by others." The majority or 37.5% (12 people) replied with Neutral (3 stars), 25% (8 people) answered Strongly Agree (5 stars). 18.8% (6 people) said Disagree (2 stars) and 12.5% (4 people) input Agree (4 stars). 6.3% (2 people) finished with Strongly Disagree (1 star).

(Fig. 4, database for question 4 in the survey sent)



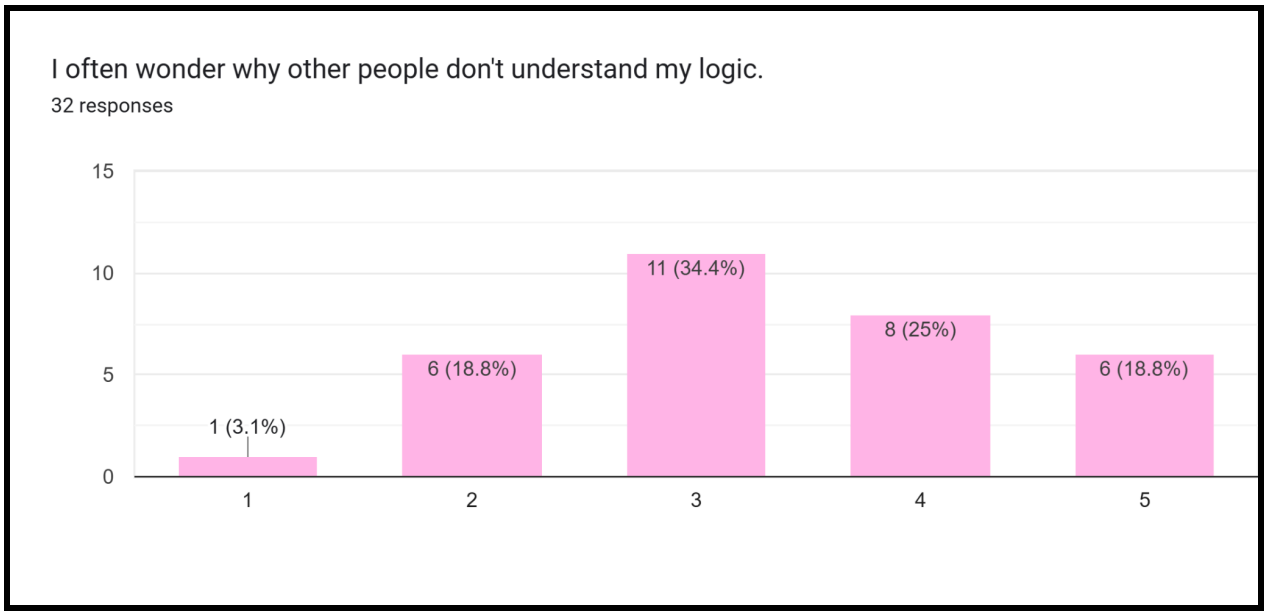
In Fig. 4, participants answered the question: “If I were to not use social media for a week, my productivity would drastically change (good or bad).” The majority or 34.4% (11 people) replied with Neutral (3 stars), Agree (4 stars) and Strongly Agree (5 stars) received the equal number of people answering them with 28.1% (9 people) on both. 6.3% (2 people) input Disagree (2 stars) and 3.1% (1 person) ended at Strongly Disagree (1 star).

(Fig. 5, database for question 5 in the survey sent)



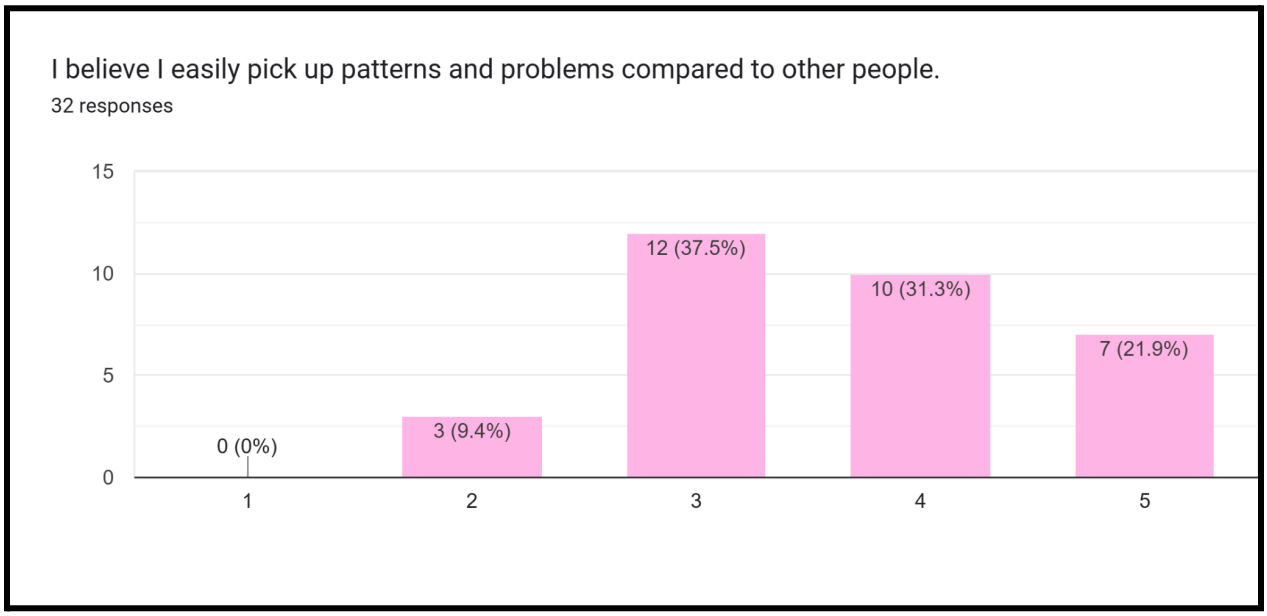
In Fig. 5, participants answered the question: “Social media has a great impact on my views in politics, on the environment, etc.” The majority of participants or 40.6% (13 people) replied with 4 stars (Agree). 28.1% (9 people) answered 3 stars (Neutral) and 21.9% (7 people) input 5 stars (Strongly Agree). 6.3% (2 people) said 2 stars (Disagree) with 3.1% (1 person) ending it at 1 star (Strongly Disagree).

(Fig. 6, database for question 6 in the survey sent)



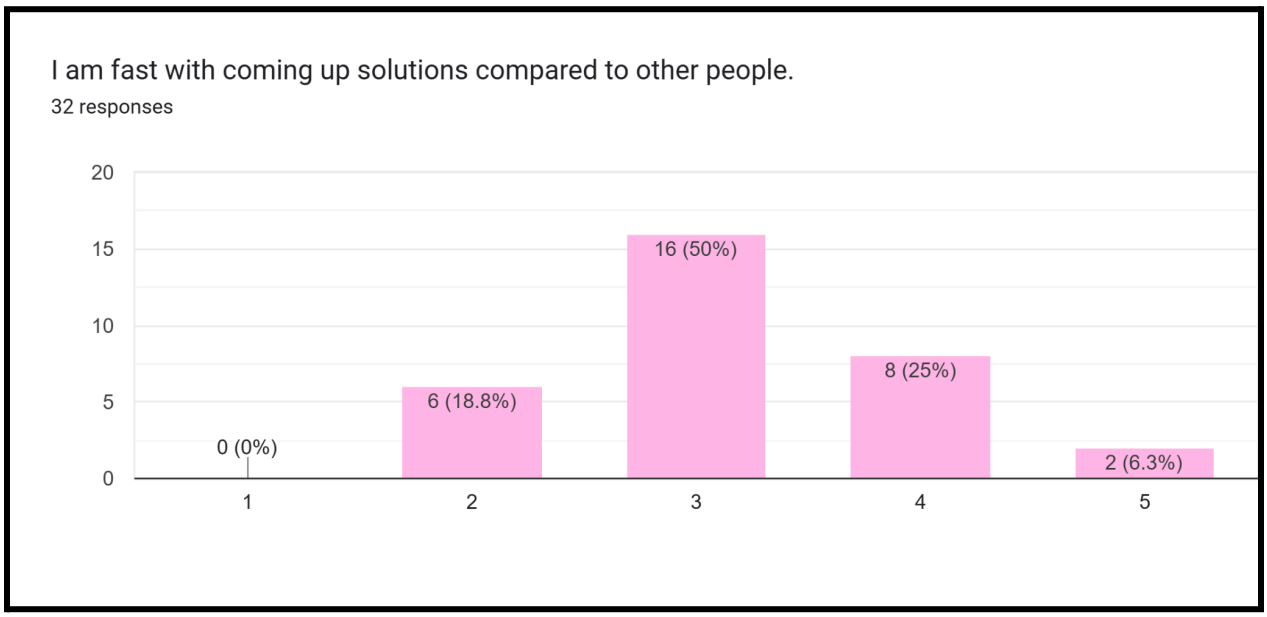
In Fig. 6, participants answered the question: “I often wonder why other people don’t understand my logic.” The majority of participants or 34.4% (11 people) replied with 3 stars (Neutral). 25% (8 people) answered 4 stars (Agree). 5 stars (Strongly Agree) and 2 stars (Disagree) had the same amount of people answer at 18.8% (6 people) individually. 3.1% (1 person) input 1 star (Strongly Disagree).

(Fig. 7, database for question 7 in the survey sent)



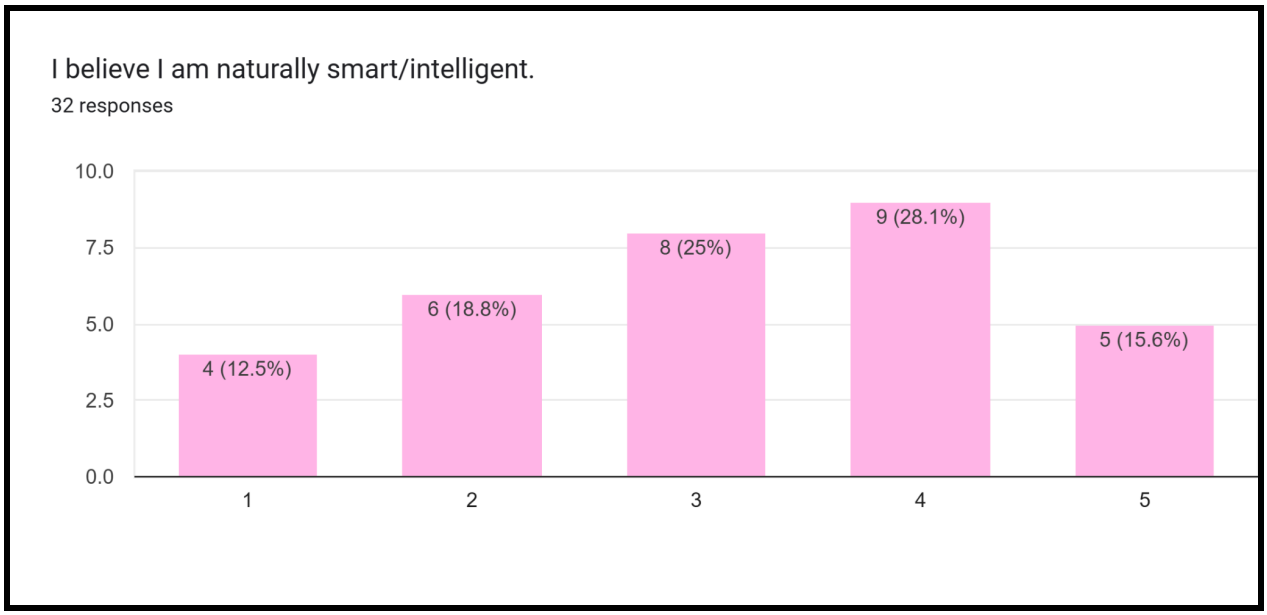
In Fig. 7, participants answered the question: “I believe I easily pick up patterns and problems compared to other people.” The majority or 37.5% (12 people) replied with Neutral (3 stars), 31.3% (10 people) said Agree (4 stars) and 21.9% (7 people) put Strongly Agree (5 stars). 9.4% (3 people) stated Disagree (2 stars) and no one said Strongly Disagree (1 star).

(Fig. 8, database for question 8 in the survey sent)



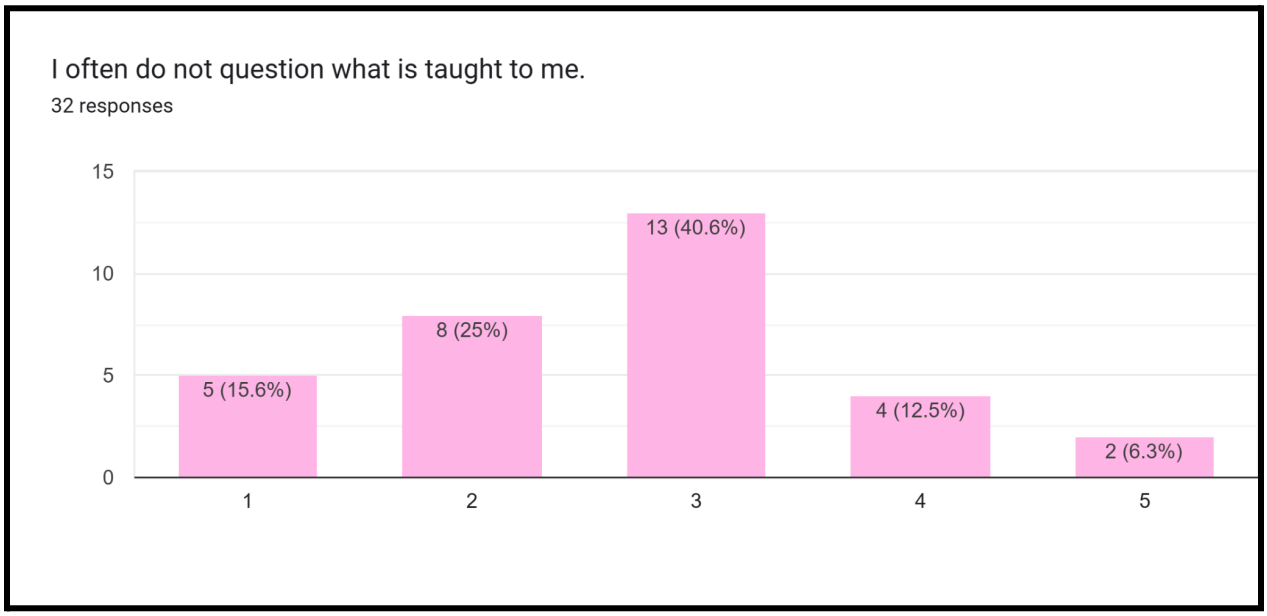
In Fig. 8, participants answered the question: “I am fast with coming up solutions compared to other people.” The majority or 50% (18 people) replied with Neutral (3 stars), 25% (8 people) said Agree (4 stars) and 18.8% (6 people) put Disagree (2 stars). 6.3% (5 people) stated Strongly Agree (5 stars) and no one said Strongly Disagree (1 star).

(Fig. 9, database for question 9 in the survey sent)



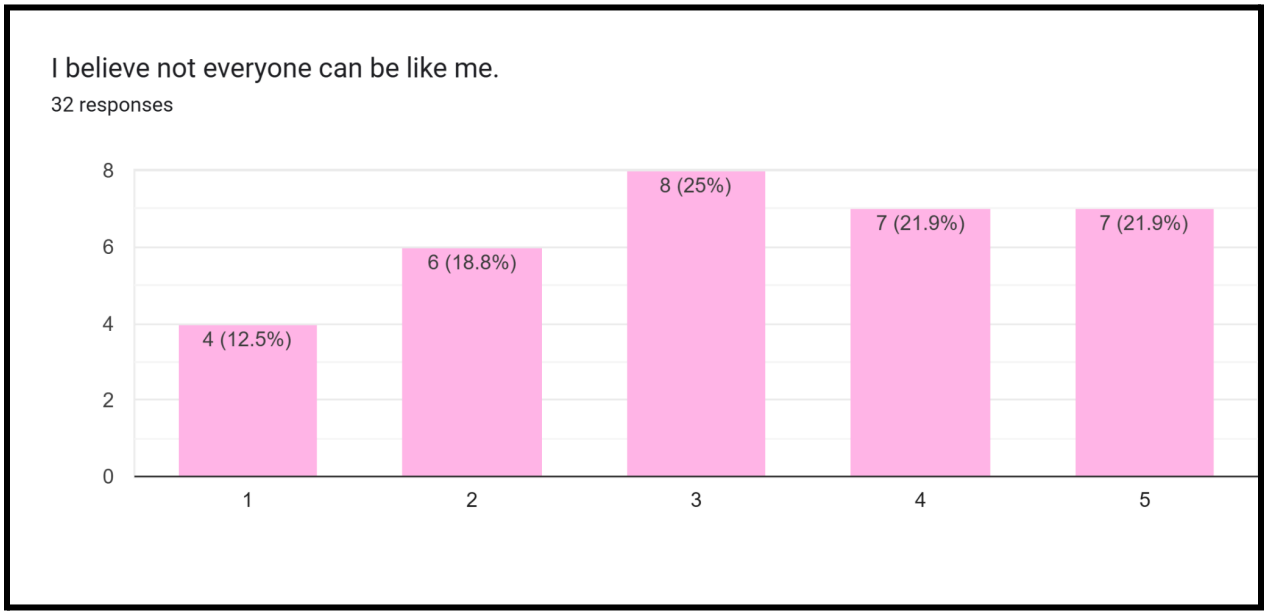
In Fig. 9, participants answered the question: “I believe I am naturally smart/intelligent.” The majority or 28.1% (9 people) replied with Agree (4 stars), 25% (8 people) said Neutral (3 stars) and 18.8% (6 people) put Disagree (2 stars). 15.6% (5 people) stated Strongly Agree (5 stars) and 12.5% (4 people) said Strongly Disagree (1 star).

(Fig. 10, database for question 10 in the survey sent)



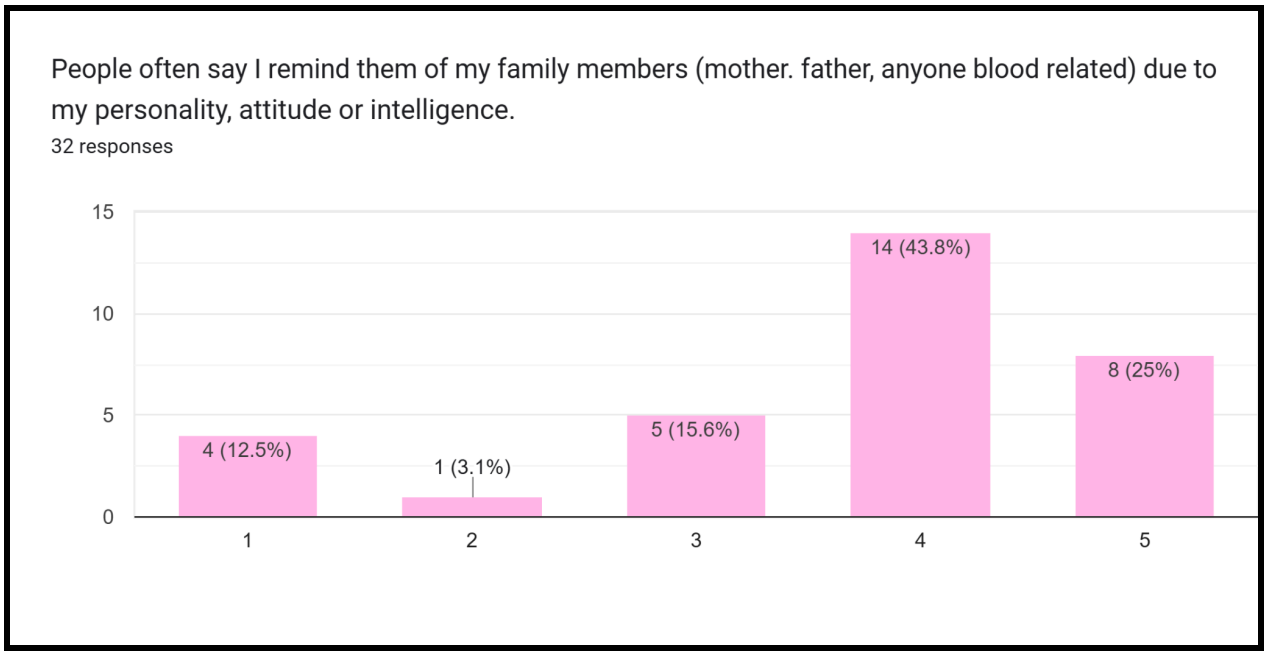
In Fig. 10, participants answered the question: “I often do not question what is taught to me.” The majority or 40.6% (13 people) replied with Neutral (3 stars), 25% (8 people) said Disagree (2 stars) and 15.6% (5 people) put Strongly Disagree (1 star). 12.5% (4 people) stated Agree (4 stars) and 6.3% (2 people) said Strongly Disagree (5 stars).

(Fig. 11, database for question 11 in the survey sent)



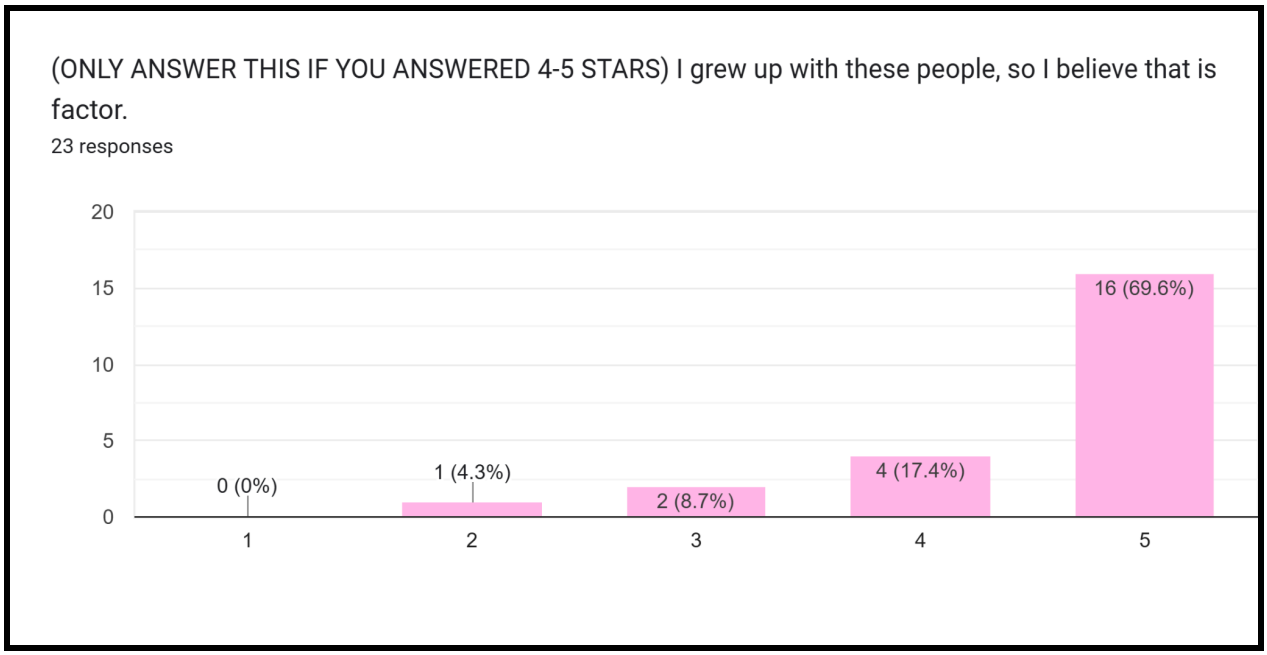
In Fig. 11, participants answered the question: “I believe not everyone can be like me.” The majority or 25% (8 people) replied with Neutral (3 stars), Both Agree (4 stars) and Strongly Agree (5 stars) both have the same amount of people picking them with 21.9% (7 people) separately. 18.8% (6 people) input Disagree (2 stars) and 12.5% (4 people) put Strongly Disagree (1 star).

(Fig. 12, database for question 12 in the survey sent)



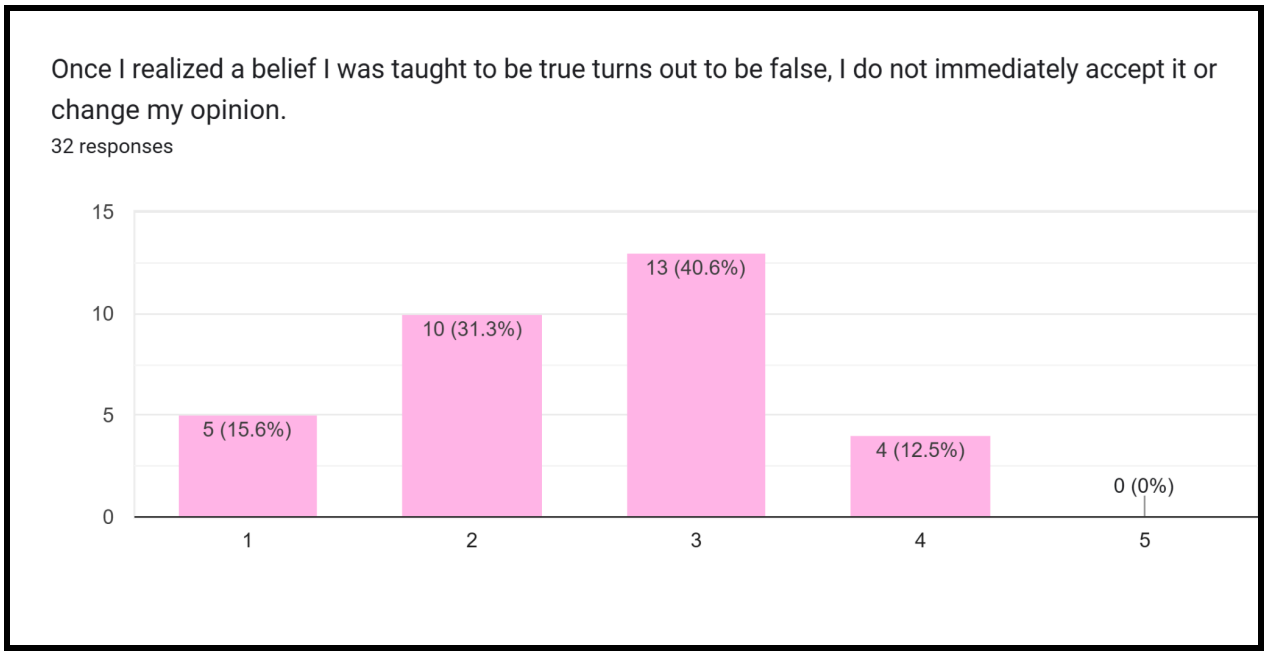
In Fig. 12, participants answered the question: “People often say I remind them of my family members (mother, father, anyone blood related) due to my personality, attitude or intelligence.” The majority or 43.8% (14 people) replied with Agree (4 stars), 25% (8 people) said Strongly Agree (5 stars). 15.6% (5 people) stated Neutral (3 stars). 12.5% (4 people) input Strongly Disagree (1 star) and 3.1% (1 person) said Disagree (2 stars).

(Fig. 13, database for question 13 in the survey sent)



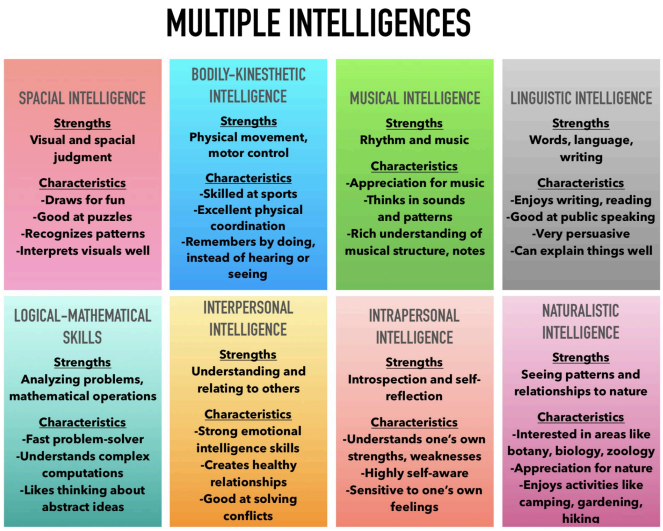
In Fig. 13, participants who answered the question below 4-5 stars, are inquired with this: “I grew up with these people, so I believe that is [a] factor.” The majority or 69.6% (16 people) replied with Strongly Agree (5 stars), 17.4% (4 people) said Agree (4 stars). 8.7% (8 people) stated Neutral (3 stars). 4.3% (1 people) input Disagree (2 stars) and no one said Strongly Disagree (1 star).

(Fig. 14, database for question 14 in the survey sent)



In Fig. 14, participants answered the question: “Once I realized a belief I was taught to be true turns out to be false, I do not immediately accept it or change my opinion.” The majority or 40.6% (13 people) replied with Neutral (3 stars), 31.3% (10 people) said Disagree (2 stars). 15.6% (5 people) stated Strongly Disagree (1 star). 12.5% (4 people) input Agree (4 stars) and no one said Strongly Agree (5 stars)

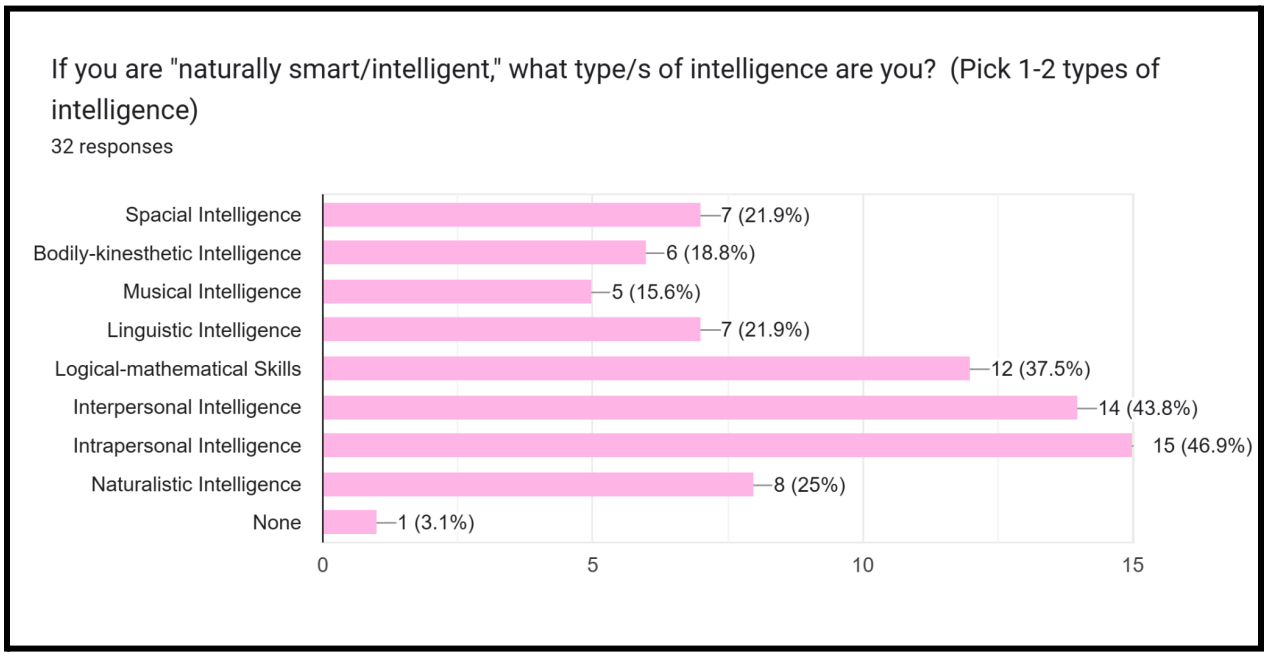
For both question 15 and question 16, please use this image as a guide:



Howard Gardner’s Theory of Multiple Intelligences

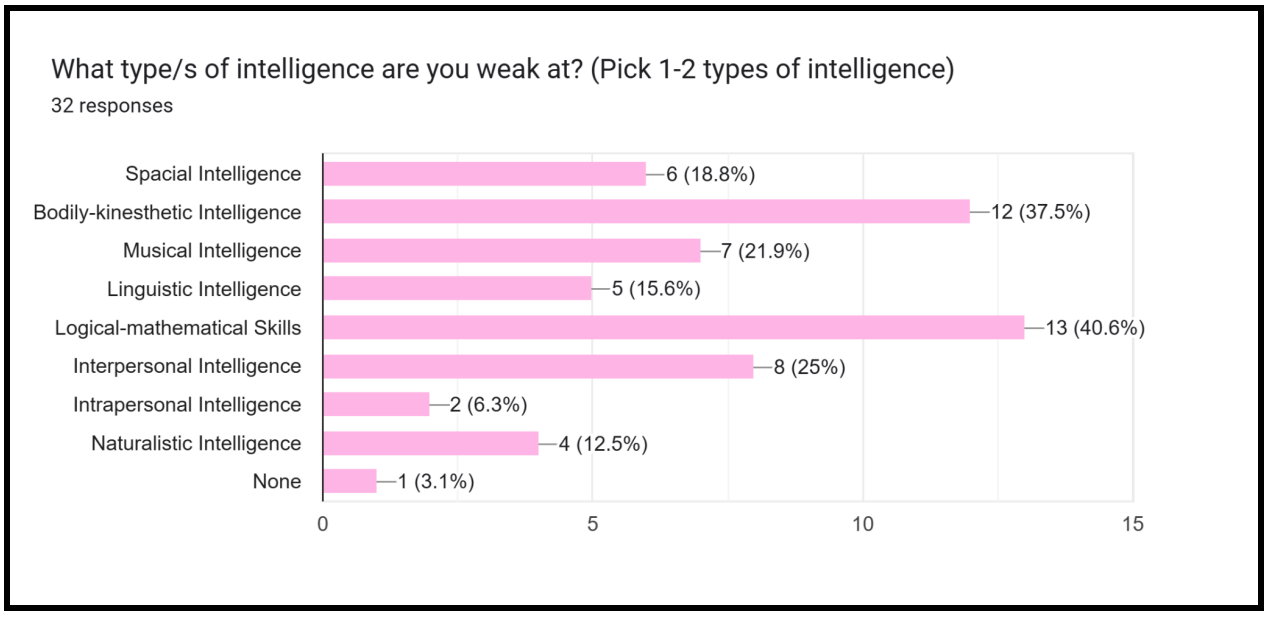
Credit: Kumar Mehta, CNBC Make It

(Fig. 15, database for question 15 in the survey sent)



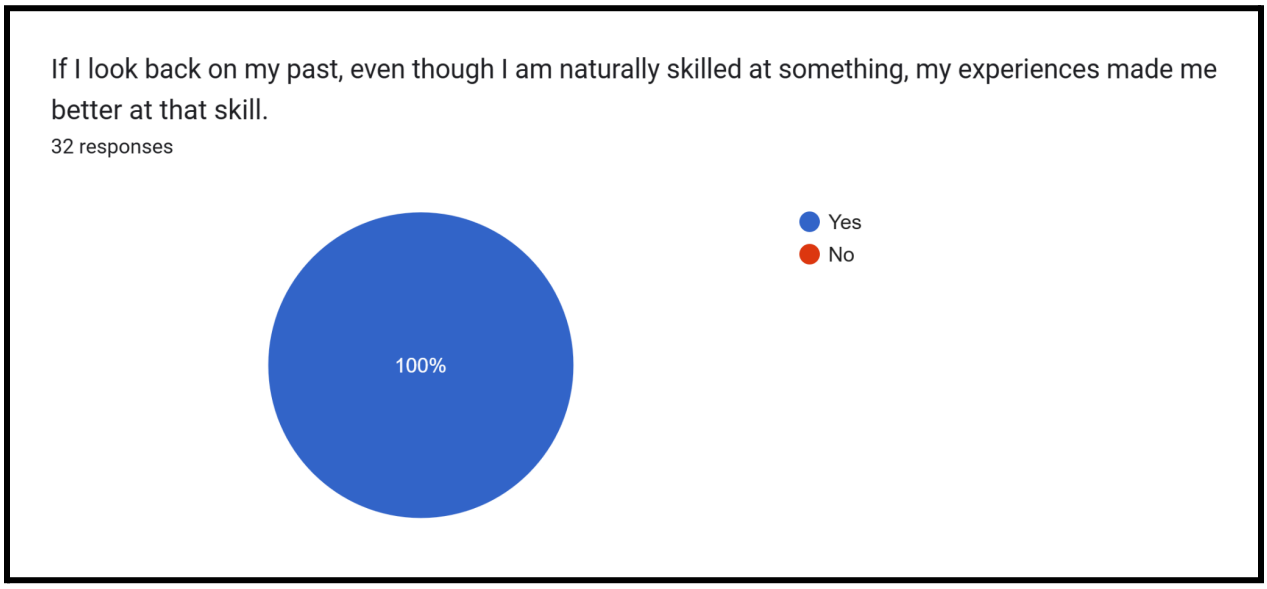
In Fig. 15, participants answered the question: “If you are naturally smart/intelligent, what type/s of intelligence are you? (Pick 1-2 types of intelligence)” The majority or 46.9% (15 people) are Intrapersonal Intelligence. 43.8% (14 people) are Interpersonal Intelligence. 37.5% (12 people) are Logical-mathematical Skills. 25% (8 people) are Naturalistic Intelligence. 21.9% (7 people) are Spacial Intelligence and Linguistic Intelligence. 18.8% (6 people) are Bodily-kinesthetic Intelligence. 15.6% (5 people) are Musical Intelligence. 3.1% (1 person) said none.

(Fig. 16, database for question 16 in the survey sent)



In Fig. 16, participants answered the question: "What type/s of intelligence are you weak at? (Pick 1-2 types of intelligence)" The majority or 40.6% (16 people) are weak at Logical-mathematical Skills. 37.5% (12 people) are weak at Bodily-kinesthetic Intelligence. 25% (8 people) are weak at Interpersonal Intelligence. 21.9% (7 people) are weak at Musical Intelligence. 18.8% (6 people) are weak at Spacial Intelligence. 15.6% (5 people) are weak at Linguistic Intelligence. 12.5% (4 people) are weak at Naturalistic Intelligence. 6.3% (2 people) are weak at Intrapersonal Intelligence. 3.1% (1 person) said none.

(Fig. 17, database for question 17 in the survey sent)



In Fig. 17, participants answered the question: “If I look back on my past, even though I am naturally skilled at something, my experiences made me better at that skill.” 100% (32 people) said yes.

Impact

The debate of Nurture VS Nature has been running for centuries, however it is understood that both biological and environmental factors play a role in a teenager’s mind. Epigenetics is concrete proof that despite having biological features that could predetermine our personalities, behavior, health, intelligence and many more, people can change or lessen those factors as they grow older due to environmental changes.

Despite behaviorism being outdated, it is a start on how to shift a mind’s thinking. Which could be linked to a trend called Manifestation, that changes the way of thinking to get a specific goal and change how people behave. AI or Artificial Intelligence has been so crucial lately with a younger audience, which is worrisome since it is also used in the workforce.

The results indicate that the majority of the participants' environmental factors, such as social media and physical surroundings (friends, family, and peers), does influence the behavior of the participant regardless of genetic factors. However, the effect of genetic influences on the participants vary with each answer.

Conclusion if it is nature or nurture is that no matter what, a person can change if they want to or not, with changing your environment and surroundings. However due to biological factors such as DNA, some might have a harder time to do such. Teenagers understand and know the bad and good impacts of our environment, but it has been hard to do so since it has been so normalized, such leads to phone addiction. Generation Alpha will be born in an age where there is unlimited access to information, which could lead to much more negative effects than the previous generation (Generation Z).

Because of this, the researchers would like to urge the readers to try limiting the usage of AI and mobile devices if not necessary, but to use these with consideration and caution. The researchers would want the readers to use these learnings to their lives, so that they could achieve their best selves. With changes, the researchers believe that this generation will influence the next one to use their surroundings to their and society’s advantage.

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