
UCL
NEUROSOC
MAGAZINE
Where the Human Brain Meets AI: DeepMind and Neuralink
The Gut Microbiome that Influences Your Brain
Can Psychedelic Therapies be Used to Treat Mental Health Conditions?
ISSUE 1

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Note from the Chief Editor

Since its birth in 2013, the UCL Neuroscience Society has aspired to increase its outreach and impact every single year. It's achieved this by consolidating feedback from previous years, and working consistently with renewed enthusiasm and fresh ideas with every change of committee. And every year, unfailingly, our Neuroscience Society has introduced new projects to connect with and support the neuroscientific student body at University College London.

This year, we bring to you our brand new magazine!

I am honoured to have been tasked with the role of bringing the Neuroscience Society's collective vision of this new venture to fruition. I am lucky to have had the privilege of working with a particularly talented, collaborative, and motivated set of individuals. We all hope that you enjoy the collection of articles we have put together for you.

I want to offer many thanks to everyone who contributed - whether as writers, illustrators, editors, or supporters. Thank you for helping finalise our very special project; it would not have been possible with you all.

We welcome feedback, thoughts, and further enthusiasm that you can bring to the table for future ventures the UCL Neuroscience Society pursues, and especially for our upcoming magazine editions.

That said, I am excited to present the first ever edition of the magazine, and looking forward to many more to come!

- **Ilina Moitra**

From Descartes to Galvani to Mind-Controlled Prosthetics: An Introduction to the Evolution of Neuroscience

Writer: Chrysi Anastasaki
Editor: Alice Wright

Today, we know the brain holds, in just 1400 grams, one of the most complex systems in the known universe. However, thousands of years ago, lacking the technology necessary to study the nervous system, early scientists and philosophers deliberated whether it is the heart or the brain that controls the body. It wasn't until second-century physician and philosopher Claudius Galenus (Galen) used vivisection (experimental surgery on living organisms for research) to begin to understand the anatomy of the circulatory system and spinal cord, that our knowledge of the nervous system began to develop. Galen is recognised for his vast contributions to anatomy and physiology - notably his identification of seven pairs of cranial nerves (we now know there are twelve) and the discovery that the brain influences the voice via the recurrent laryngeal nerve.

In the 17th century there was a scientific revolution. This involved a dramatic change in scientific attitudes, whereby the field began to separate from church and philosophy, and new findings became more evidence-based and accessible. During this time, Rene Descartes proposed his theory of mind-body duality. This hypothesised that the mind and body are two different entities, and they communicate through the pineal gland. Building on this, he also proposed the idea of "animal spirits" that flow through nerves as a fluid, and cause contractions by filling muscles. Descartes' philosophical epistemology was one of the initial investigations of the connection between the brain, mind, and behaviour. Whilst Descartes' notion of "animal spirits" has since been disproved, it could be argued that he was still revolutionary in rivalling vitalism (the theory that living organisms possess a non-physical force that distinguishes them from non-living things) by attempting to explain nerves using physical properties.

It wasn't until Luigi and Lucia Galvani brought electrophysiology to the forefront of neuroscience research, in 1780, that understanding of the electrochemical properties of neurons began to take off. In their research, they showed that the legs of a frog would contract if exposed to electricity. These findings led Luigi Galvani to propose that electricity is stored in the neuromuscular system and is released to initiate movement. Famously, this work influenced the writing of Mary Shelley's Frankenstein. At the time there was dispute over the credibility of his experiments - Alessandro Volta doubted the contractions were caused by intrinsic electricity, but he did use information from Galvani's work to

invent the first battery. Today, it's clear some of the conclusions Galvani drew were incorrect, but he is still rightly celebrated as the father of electrophysiology.

Approximately a century later, Phineas Gage suffered a catastrophic workplace injury that destroyed much of his left frontal lobe. This is a particularly famous case study of how lesions in the brain have provided invaluable information regarding the physiology of the nervous system. After his incident, it's said he exhibited profound changes in personality and behaviour. From this came the suggestion that the brain may regulate personality, and damage to different parts of the brain may cause differing symptoms. Since then, the localisation of function theory has been defined – stating that different brain regions are responsible for different functions. Of course, we now know the brain is much more complex, interconnected, and plastic than this theory suggests. However, the importance of lesion studies in determining regional function can't be overlooked. For example, in the mid to late 1800s, Broca and Wernicke studied aphasia in two distinct areas of the brain to gain insight into forming and understanding language processing and its anatomical correlations. This same strategy was applied in the study of patient HM by Brenda Milner. HM had undergone a bilateral medial temporal lobectomy in an attempt to alleviate the symptoms of his epilepsy. Although this was partially successful, he lost the ability to form new memories – suggesting the temporal lobe may be implicated in memory consolidation.

At the turn of the 20th century, Golgi and Ramón y Cajal won the Nobel Prize for discovering and utilising a new form of tissue staining (using silver nitrate) to examine neurons. Up until this point, conventional light microscopy had been insufficient for studying neuronal structure and diversity. This was a significant turning point in the early field of neuroscience as the Golgi stain allowed Ramón y Cajal to identify the free axon endings, and subsequently publish evidence for the neuron doctrine. The neuron doctrine states that the nervous system is composed of discrete, individual cells. This was important in casting doubt over the reticular theory. To this day we understand that the neuron doctrine governs the nervous system.

Looking more locally, University College London (UCL) has been home to a long line of incredible neuroscientists from as early as 1904 when Henry Dale and Otto Loewi won the Nobel Prize in Physiology or Medicine for their study of acetylcholine as a neurotransmitter. Sir Bernard Katz is another notable neuroscientist who spent time at our university and has a campus building in his namesake. Katz built on the work of Dale and Loewi as he looked at acetylcholine as a neurotransmitter at the neuromuscular junction. He was jointly awarded the Nobel Prize in Physiology or Medicine for discovering the quantal quality of neurotransmitter release. However, it wasn't until 2008 that the neuroscience domain was independently established at UCL. Today, multidisciplinary scientists from medicine, biochemistry, psychology, philosophy, and computer science attempt to answer versatile questions under the domain of neuroscience. Recently, John O'Keefe won the Nobel Prize for his research into place cells in the hippocampus of rats, and how

they work to form an internal map of the environment. There are so many brilliant academics that have made fascinating contributions to neuroscience whilst at UCL over the years, and it's exciting to think about what other discoveries will be made at our university in the future.

One of the most recent advances in neuroscience has enabled people with amputated hands to move from conventional to mind-controlled prosthetics. These are implant devices that can be surgically attached to the skeleton, muscles, and nerves of the remaining arm. Researchers have embedded in these prosthetics a control system that uses artificial intelligence algorithms to process nerve signals. This achievement is currently on trial, and it enables people to intuitively grip objects and also feel a sense of touch - achieving a powerful imitation of the biological hand. It's remarkable to see how much neuroscience has evolved over so many years, and how our advancing understanding and technology are being used to improve the quality of life for so many people across the globe.

Despite dramatic progress over recent decades, neuroscience research methods still have their limitations. For example, fMRI data indicates that specific areas are activated during particular processes. Whilst this suggests a degree of correlation between certain brain regions and functions, it does not prove causation.

The nervous system is a network of complex interconnectivity working at tissue, cellular, and molecular levels to perceive the world and construct behaviour in response. As much as substantial progress has been made in terms of our knowledge about the nervous system, with every question we seem to answer, more arise. One thing is clear, the 86 billion neurons of the brain welcome more questions than answers.

Loneliness in Lockdown

Writer: Lauren Pereira-Greene
Editor: Alice Wright

As masks are shoved into pockets, and social distancing rules evanesce into the past, the pernicious presence of loneliness lingers. Familiar tales of isolation are deeply embedded in the COVID-19 narrative. However, for many, solitude is not new. Loneliness impacts millions of people in the UK, with those aged between 16 and 24 at heightened risk. But what actually happens to the brain of a lonely individual? This article will focus on the default mode network (DMN) and its potential association with loneliness. The default mode network in the brain is typically associated with goal-oriented tasks, but it may also hold the key to understanding how loneliness has neurological consequences.

The default mode network consists of the medial prefrontal cortex, posterior cingulate cortex, precuneus and angular gyrus that work together to form a large network of interconnectivity. It is known for its activity in states of wakeful rest (such as daydreaming and mind-wandering), during which the mind is distracted from the outside world. In addition to this, fMRI studies suggest the DMN is implicated in mental representations of the self and others. This higher associative network is considered to be the centre of the neural expression of loneliness.

A team of psychologists in the United States have shown that within the medial prefrontal cortex (a core node of the DMN), lonely individuals display a larger distinction between the neural representations of themselves versus the representations of others. In most of us, the brain activity exhibited when reflecting on ourselves is very similar to the activity shown when we're thinking of other people. The lonelier we are, the less similar these patterns of brain activity become. Social isolation results in a 'lonelier' neural self-representation, and growing evidence is beginning to shine a light on how this physically manifests.

In 2020, a team of researchers from McGill University carried out a multimodal analysis study using MRI data from the UK Biobank initiative. The acquired data was of grey matter morphology from 38,701 people, to study the neural model of loneliness. They found that when the DMN was compared to other cortical networks, loneliness was most strongly linked to increases in grey matter volume in the DMN. Furthermore, lonely participants showed stronger functional communication in the DMN. The DMN is normally active when reminiscing, daydreaming, and thinking about the self or others. A deprivation of social activities in the real world can encourage us to mentally simulate social events instead. Therefore, the link between loneliness and greater functional connectivity in the DMN may suggest that we adopt those mental behaviours in order to fill a "social void".

As well as this, lonely individuals had greater microstructural integrity of the DMN fornix pathway. Microstructural integrity is the quality of subcellular components, such as cell membranes and myelin - the properties of which affect the generation and propagation speed of action potentials. The fornix pathway connects the DMN to the hippocampus - an area responsible for memory. Since this link is strengthened in the lonely, they may be more likely to ruminate on previous social experiences. The hippocampus is also connected to the amygdala - an area that processes strong emotions. This implies that reminiscing about former events is likely to evoke a significant emotional response. Loneliness in individuals correlates with dwelling over past social interactions, or imagining new ones, and therefore the enhanced fornix pathway could evoke stronger or more frequent emotional responses in these people.

There is no doubt that loneliness is a hugely complex phenomenon, and research into the neurobiological characteristics of loneliness is currently scarce. What we do know is that strong social connections are integral components of a healthy and happy life. But as COVID-19 regulations ease around the world, loneliness still lingers throughout much of society. If you're ever feeling lonely, be sure to communicate those feelings with a loved one or health professional, and try to regularly check in with family and friends!

Inception – The Neuroscience Behind False Memories

Writer: Alessia Qiu
Editor: Ellie Roberts
Artist: Junhui Hu

In the famous 2010 science fiction film directed by UCL graduate Christopher Nolan, Cobb (a professional thief portrayed by Leonardo DiCaprio) steals information by infiltrating the subconscious of his targets. Cobb is then offered a chance to erase his criminal history as payment for a challenging mission involving the implantation of a false memory into a target's subconscious - this is Inception.

This leads us to consider, from a neuroscientific point of view, what are false memories and is inception possible?

In psychology, a false memory is a phenomenon where someone recalls something that did not happen or recalls it differently from the way it really happened. Sometimes, accumulation of false memories can result in a condition (the false memory syndrome) in which a person's identity and relationships are affected by these strongly believed recollections that are factually incorrect.

Interestingly, false memories are not only an individual phenomenon and can be shared by a large number of people. A study in 2010, coincidentally the same year that Inception premiered, examined people who were familiar with the clock at Bologna Centrale railway station, which was damaged in the Bologna massacre bombing in August 1980. In this study, 92% of the participants falsely remembered that the clock had remained stopped since the

bombing when, in fact, the clock was repaired shortly after the attack. This shared false memory phenomenon was named "the Mandela effect" – a term which has since claimed global recognition.

According to William Hirst, Professor of Psychology at The New School (NY), it is relatively easy to implant memories, and, in certain situations, you can implant them about 30 to 40% of the time. Once a memory has been implanted, the process of memory formation continues and is followed by a studied stage named 'updating'. To illustrate this, let's consider the case of false news about the Iraq War early on. It was observed in one study that if you were in Germany or Australia, you were likely to update the information, but if you were in America, you weren't as likely to do so. Thus, even though some people were told a fact was wrong, they would still remember the previous incorrect fact. This suggests that memory is schema consistent, so if something fits into the way you think things should be, you don't update the memory as easily once it's been formed.

In terms of groups talking together, studies suggest that false memories are more likely to arise in a group discussion than individually because there is a higher chance of somebody offering a false memory which can then be implanted. However, if somebody in the group disagrees with the recalled memory, that will mitigate the influence. It is worth noting that for groups with a strong shared interest, for instance Donald Trump supporters, they are less likely to dispute one another and therefore it is less likely for their memories to be updated.

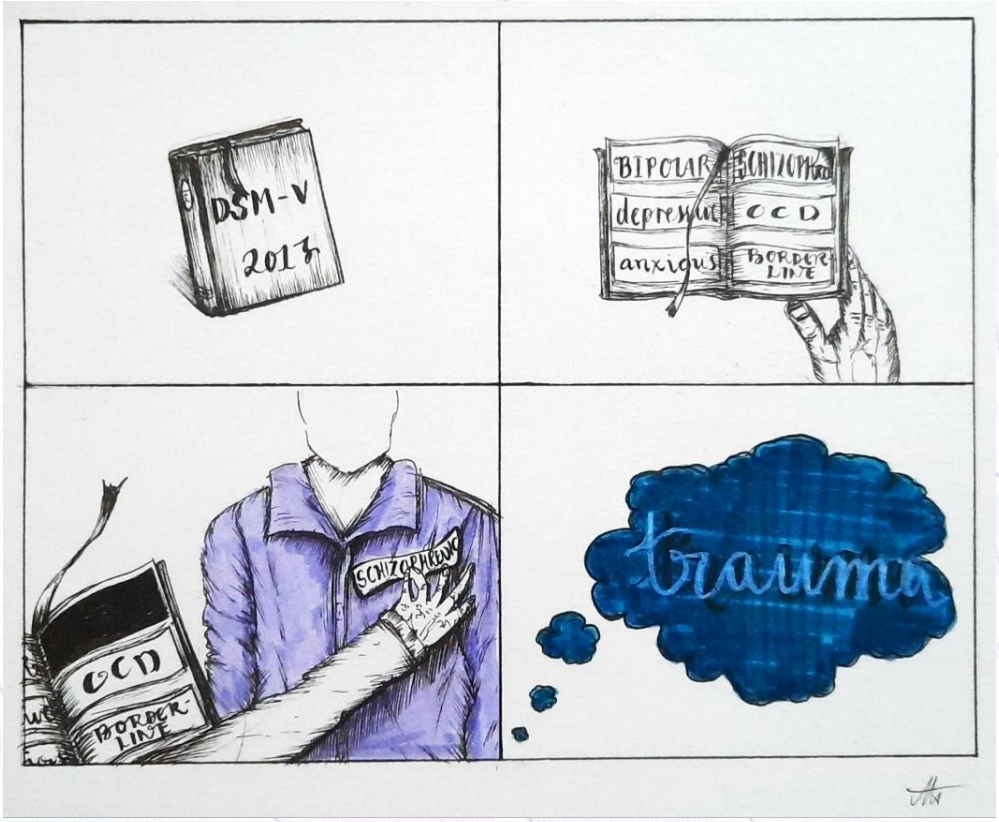
Our memories fit into a way of viewing and interpreting the world. There's not really a "truth" to memory. As Sir Frederic Bartlett, the first experimental psychologist at the University of Cambridge, had called it, memory is a continuous reconstruction. This reconstruction is guided by an individual's view of the world. This means that your current view of the world, your current attitudes, allow you to reconstruct your own past to be consistent with your present self. Thus, we are constantly reshaping our memory to essentially reinforce our present attitudes. Our memory is not only our own, but also that of all we interact with.

Delving into the basic mechanism of memory formation, memory is the reactivation of a specific network or circuit of neurons, formed from persistent changes in the strength of connections between neurons. Memory formation and encoding relies on a mechanism called synaptic plasticity. This describes the neuronal synapses' dynamic activity whereby they can change the strength or efficacy of an existing connection, or even form new ones.

In 2013, neuroscientists from MIT Steve Ramirez and colleagues, targeted the neural circuit involved in the encoding of a particular memory and successfully manipulated it to encode a false memory in mice. "If mice had Hollywood, this would be 'Inception' for them", commented Dr Steve Ramirez at the time. The authors of the study revealed that this type of research could one day help treat emotional problems, such as post-traumatic stress disorder, which involves the intrusion of unwanted memories. However, ethical questions in this field are vast and open to discussion.

The DSM-V Debate: On Diagnosing Psychiatric Conditions

Writer: Aya Tarabien
Editor: Ellie Roberts
Artist: Tahira Siddiqi



Occasionally, we engage in a conversation that shakes our beliefs to the core. A while ago, I chatted with a friend who does not believe in diagnosing mental illnesses. At first, I was puzzled by such a seemingly bizarre notion. But eventually, they drew me to think beyond diagnoses and question everything I learned about the DSM-V during my academic journey. For example, whether receiving a diagnosis could stigmatise someone or provide a framework for treatment. I immersed myself in current research and was astounded to find leading experts contributing to the debate of whether mental illnesses exist.

The Diagnostic and Statistical Manual of Mental Disorders (5th edition), also known as the DSM-V, is an essential source of information for mental health professionals. However, there is a high degree of inconsistency and contradiction within and across the diagnostic categories of the DMS-V. For instance, a prominent study from the University of Liverpool analysed five key chapters of the DSM-V on schizophrenia, bipolar, and depressive, anxiety and trauma-related conditions. One of their key findings was that, while no two psychiatric diagnoses use the same decision-making rules in diagnosis and treatment, they seem to share numerous symptoms. Allsopp and Kinderman, the researchers leading this study, expressed how

unhelpful it is to diagnose various mental health conditions. They postulated that diagnoses are not capable of accurately portraying the role of trauma or adverse events, and are therefore insufficient to help us assess the state of someone's mental health. Furthermore, even though the researchers overemphasised their support for this notion, they still provided a corrective view to our over-medicalized approach. Their work was further bolstered by the DSM-IV Task Force Chairman Allen Francis, who stated that the DSM-V was only used in certain countries, such as the United States, because labelling a condition would guarantee that insurance companies paid for the treatment.

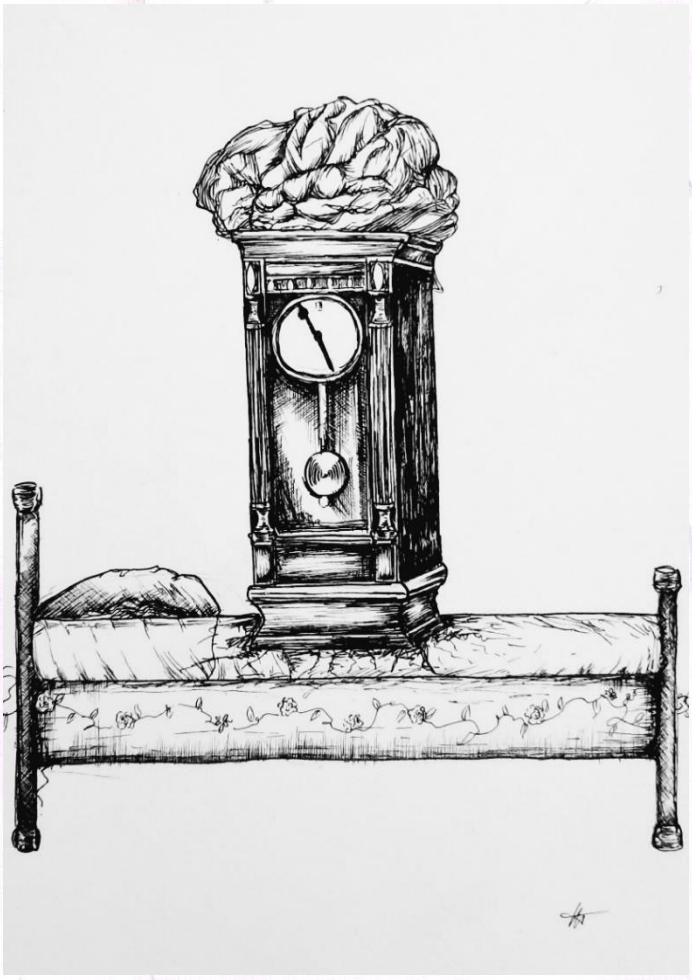
Kinderman's notion that a diagnosis is a one-word summary, and not the whole story, is incontestable. Similarly, most doctors, patients and families can completely lose sight of what's important and get distracted by the labels. Take having a headache as an example - of course, you can tell what a headache feels like, but labelling it as a headache only gives you a summary of the symptoms. It does not inform you anything about the underlying cause of pain in your head. Everyone knows that an individual is never just manic depressive, bipolar, or schizophrenic, however, unfortunately, this is often forgotten.

On that matter, one should note that the debate does not seek to reject the whole notion of diagnosis and instead aims to point out the inconsistencies and limitations of the DSM-V as a handbook. Despite all these issues, this handbook does serve as an essential tool through facilitating communication among clinicians and patients by providing an idea of what a patient may be experiencing. Indeed, Professor Wessely expressed how the diagnosis is essential to medical practice since anorexia is not the same as schizophrenia.

Overall, after examining the heterogeneous nature of categories in the DSM-V, it is clear that a 'one size fits all' solution is not suitable for such complex conditions. However, there is hope in constructing a pragmatic approach to psychiatric assessment, which will allow for the acknowledgement of individual experiences. Hopefully, we hold the chance of finding an effective way to comprehend the distress caused by abiding by this categorical system. Lastly, I will leave you with the words of the mental health activist Dr Eleanor Longden, whose TEDTalk video about her personal experiences with auditory hallucinations has been viewed more than five million times. She was told by her psychiatrist that she would be better off with cancer than schizophrenia because "cancer is easier to cure". I highly resonated with her statement that "the most important question in psychiatry should not be what is wrong with you, but what is it that happened to you?"

The Relationship Between Sleep and Psychiatric Conditions

Writer: Angela Lee
Editor: Alice Wright
Guest Editor: Ashvni Narayanan
Artist: Tahira Siddiqi



Sleep is a naturally recurring state marked by reduced or absent consciousness, as well as inactivity of most voluntary muscles. There are two main stages of sleep, known as rapid eye movement (REM) and non-rapid eye movement sleep (NREM). During REM sleep, brain activity is similar to an awake person, whereas NREM is highlighted by periods of slow wave and synchronised neuron activity. Generally, sleep is regulated by a circadian cycle, which is part of the internal body clock, controlled by the suprachiasmatic nucleus (SCN) in the hypothalamus.

There are several physiological processes that occur in the central nervous system (CNS) during sleep that are important for bodily functions, though its exact role in these processes is still being uncovered. It is probable that the purpose of sleep in the CNS is much deeper than we are currently aware of.

Schizophrenia is a major psychiatric condition that has significant, long-lasting impacts on patients. While positive symptoms (for example, delusions and hallucinations), negative symptoms (i.e., anhedonia, and social withdrawal), and cognitive impairments are traditionally considered the most prominent features of this condition, the role of sleep has gained increasing prominence in clinical practice. Indeed, the vast majority of patients with schizophrenia report sleep abnormalities, which tend to precede illness-onset and can predict acute exacerbation of psychotic symptoms. Furthermore, patients with schizophrenia often have a comorbid sleep disorder. Despite accumulating data, the links between sleep disorders

and schizophrenia have not been thoroughly examined. This is partly due to difficulty disentangling the factors that contribute to the comorbidity - including medication.

Additionally, sleep disorders are often not the primary focus of clinicians treating this population, despite studies suggesting that comorbid sleep disorders carry their own risks, including worsening of psychotic symptoms and poorer quality of life. There is also limited information about effective management strategies for schizophrenia patients affected by significant sleep disturbances.

In a current study monitoring circadian rhythms of people with schizophrenia, researchers found more sleep disturbances in patients with schizophrenia versus control patients who were deemed mentally healthy. This supports other emerging studies suggesting sleep and schizophrenia are more closely intertwined than previously thought. Both human and animal studies propose that circadian rhythms, dopamine regulation, and psychosis are closely linked. This suggests treating circadian disturbances may be a useful tactic in improving the lives of patients with schizophrenia.

Bipolar is a mental illness characterised by alternating periods of elevated and depressed moods. Sleep disturbances in patients with bipolar are present during all stages of the condition, and exert a negative impact on overall quality of life and treatment outcomes. Biomarkers of depressive episodes include heightened fragmentation of REM sleep, reduced REM latency, increased REM density, and a greater percentage of awakenings. Biomarkers of manic episodes include reduced REM latency, greater percentage of stage I sleep, increased REM density, discontinuous sleep patterns, shortened total sleep time, and greater time awake in bed. These findings highlight the importance of targeting novel treatments for sleep disturbance in bipolar.

Chronotherapy involves intentionally delaying sleep by two or three hours on successive days until you are able to fall asleep at the desired time. This can be difficult to do at home and is sometimes done in a clinical setting. After this, you must strictly enforce this schedule. Chronotherapy is helpful for those who have difficulty initiating sleep, such as those that suffer from insomnia. Insomnia interrupts sleep patterns, and may have many causes - for example, stress or mental illness. Sleep patterns follow a circadian rhythm, and problems with this can lead to one of the circadian rhythm sleep disorders, most commonly delayed or advanced sleep phase syndrome.

In the treatment of psychiatric conditions including bipolar depression, a form of chronotherapy combining intermittent sleep deprivation and morning bright light has shown efficacy and relative tolerability in a number of controlled studies.

Can Psychedelic Therapies be Used to Treat Mental Health Conditions?

Writer: Angela Lee
Editor: Alice Wright
Guest Editor: Ece Gokbayrak
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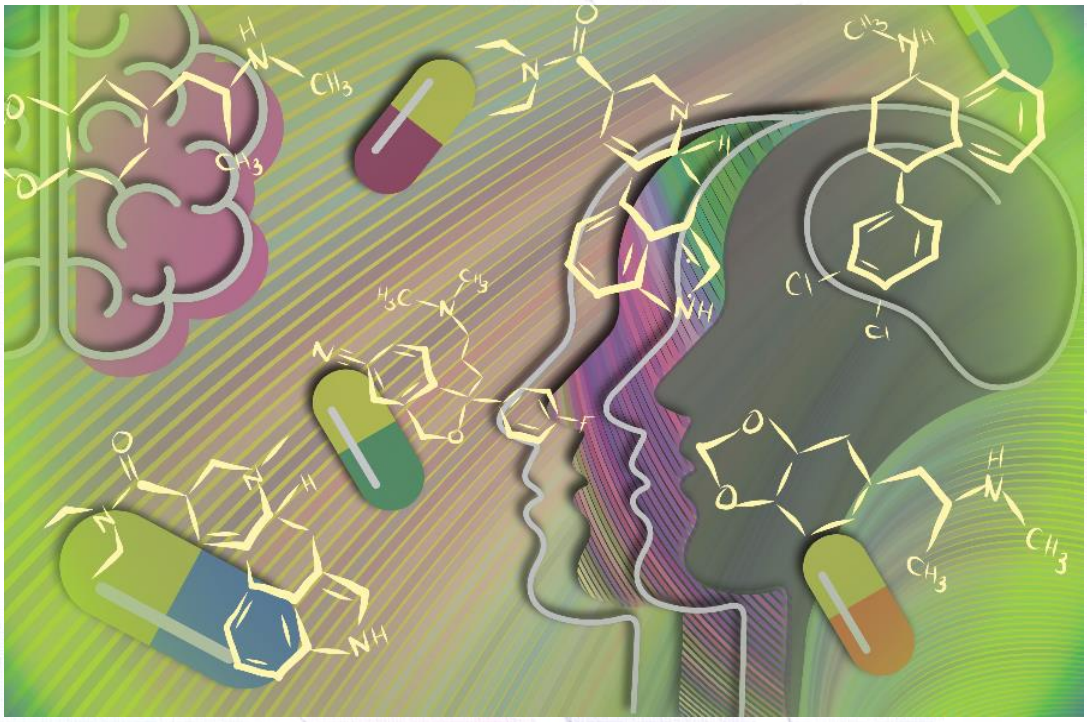


Psychedelic drugs (commonly termed hallucinogens) are substances that alter and, in some cases, enhance sensory perceptions and thought processes. They include chemicals such as LSD, MDMA, and Psilocybin. For over a decade, studies have increasingly shown that psychedelic drugs once thought to be dangerous may actually have therapeutic benefits— psychedelic therapy may even be helpful for people dealing with types of depression, anxiety, addiction, and PTSD.

Lysergic acid diethylamide (LSD) was studied from the 1950s to evaluate behavioural and personality changes, as well as the remission of psychiatric symptoms in various conditions such as schizophrenia. LSD was used in the treatment of anxiety, depression, psychosomatic conditions, and substance dependence. However, most studies were not performed under contemporary standards, and it has taken several decades to revive interest in LSD research and its therapeutic potential for psychiatry.

Colloquially referred to as ecstasy or molly, MDMA became popular in the rave and electronic dance music culture of the 1980s and 1990s. It is a psychoactive drug sourced from safrole oil - an essential oil

derived from the sassafras tree endemic to North America. MDMA has been reported to elicit feelings of euphoria, inner peace, and increased self-confidence and empathy.

Psilocybin, the active chemical compound derived from certain fresh or dried hallucinogenic (magic) mushrooms, is at the centre of the scientific community's psychedelic renaissance. In recent research, it's been found that psilocybin-assisted therapy can be helpful for people with treatment-resistant depression as well as end-of-life depression and anxiety.

In the last decade a number of research groups in Europe and the Americas have conducted studies into the safety and effectiveness of psychedelics for conditions such as depression and PTSD. The new Imperial College London centre is the first to gain a major level of stature within this field. Imperial's Psychedelic Research Group was the first to investigate the effects of LSD on the brain using modern brain imaging, and the first to study psilocybin for treating severe depression. These studies have laid the groundwork for larger trials now taking place around the world.

Most drugs that treat depression and anxiety can be picked up at a local pharmacy. These new psychedelic approaches, by contrast, use powerful substances in therapeutic settings under the supervision of a psychotherapist. Regulators and treatment providers will need to grapple with how to implement this safely. There are also risks; in extremely rare instances, some psychedelics can evoke lasting psychosis - more often in people with a family history of psychosis. For example, those with schizophrenia are excluded from trials involving psychedelics. MDMA, moreover, is an amphetamine derivative, and could come with risks of dependence.

However, many researchers are excited. Several trials show dramatically promising results: in a study from November 2020, 71% of people who took psilocybin for major depression showed a greater than 50% reduction in symptoms after four weeks, and half of the participants entered remission. Some follow-up studies after therapy, although small, have shown lasting benefits.

Serotonin is a target of the predominant class of psychiatric drugs known as selective serotonin reuptake inhibitors, or SSRIs (most commonly known as Fluoxetine or Prozac). It is thought that these antidepressants work not by flooding the brain with serotonin, as initially assumed, but by stimulating neuroplasticity — the brain's ability to forge new neuronal connections. There is some evidence that psychedelic drugs, such as psilocybin, enhance neuroplasticity. This is just a small glimpse into growing evidence that psychedelics may possess antidepressant properties.

Although it may seem like psychedelic therapy is a new invention, the psychedelic renaissance is really a return to decades-old research - and even older traditional spiritual practices. With continuing research and even a few Phase 3 clinical trials in the works, this type of treatment might soon be available to more people and get the scientific recognition it deserves.

The 2021 Celebration of Women in Neuroscience: A Review

Writer: Alice Wright
Editor: Ellie Roberts

The Society for Neuroscience (SfN), which aims to advance understanding of the brain and nervous system, was founded in America in 1969. In 1980, the Women in Neuroscience initiative was created to promote professional advancement and communication.

Women in Neuroscience is an annual event that provides advice and encouragement for many female scientists. The 2019 Women in Neuroscience event celebrated the 50th anniversary of SfN. Dr. Bernice Grafstein, the first female president of SfN (1985-1986), was presented with the special achievement award. As a postdoctoral researcher, Grafstein studied cortical connections alongside JZ Young at UCL. It was inspiring to see a neuroscientist with such a distinguished career (and links to our university) receiving an award in recognition of her contributions to neuroscience. At the 2021 event, she also received an award for mentoring and advancing women in neuroscience.

The 2019 Women in Neuroscience event also featured three panellists: Huda Akil, Carol Mason, and Carla Shatz.

Dr. Huda Akil spearheaded research in the neurobiology of emotions and substance use. She is perhaps best known for providing evidence of endorphins, activated by stress, causing pain inhibition.

Dr. Carol Mason studies how to restore vision to people who are blind, and is currently researching the transformation of stem cells into retinal ganglion cells.

Dr. Carla Shatz was the first woman awarded a PhD in neurobiology from Harvard University. She also popularised the phrase "cells that fire together, wire together" and is credited for discovering that spontaneous neuronal activity in utero is critical for developing neural circuits in the central nervous system.

Carla Shatz shared a touching story about her endometriosis diagnosis after years of failing to conceive. She and her husband at the time disagreed on adopting and, unfortunately, she never got the family she'd hoped for. Remarkably, Dr Shatz is working to make this positive as she supports younger people in her lab who hope to start their own families without hindering their careers. Huda Akil and Carol Mason, both of whom have children, shared their struggles with balancing work and family, and often feeling they were failing at both. This openness from

the panellists highlighted the importance of discussing how adversity can impact not only people's personal lives, but also their professional ones. This is a challenge that definitely needs to be spoken about more, with the aim of not just raising awareness, but also improving support in the workplace for those that need it.

Issues of balancing career progression and family will face many in the future – regardless of which field we may go on to work in. Despite unanimous agreement there's not enough support for those starting families whilst working in academia, it's a little heart-warming to know there are some individuals trying hard to change this. It is apparent that communication and mentoring is such a huge part of trying to support women in all areas of research.

Gender inequalities still exist in academia - evidenced not just in the still-present gender pay gap but also the under-representation of women in STEM (see stemwomen.com). Whilst that can be disheartening to female science students, events such as Women in Neuroscience highlight the importance of representation. The complexities of gender discrimination in the scientific world can't be sufficiently addressed in this article, not least because sexism is not just an academic issue, but a societal one.

Hopefully events such as Women in Neuroscience can inspire people to recognise the importance of challenging gender inequality, and this can spark a new wave of awareness that there remains much progress to be made.

Recordings of the Women in Neuroscience events from 2018, 2017, and 2016 are also available on the SfN website.

Busting the Myth of a Gendered Brain

Writer: Aya Tarabien
Editor: Ellie Roberts

It is not all black and white when it comes to our brains. The history of sex-difference research is rife with innumeracy and inadequate controls. Luckily, a new pop field has come to the rescue. The idea that you can "sex" a brain and classify it as being male or female has been heavily argued against in the rising field of neurosexism, established under the premise that variations in expressed behaviour between males and females do not stem from differences in brain development, but rather from socialisation.

From the old-age work to modern brain imaging studies, neuroscientists like Gina Rippon have conducted robust research to debunk the "pernicious" sex differences myth. Rippon's main aim was to raise awareness of how "a gendered world will produce a gendered brain". As Rippon shows, beyond the "missing five ounces" of the female brain, when brain size is scaled in relation to overall body size, there is no link between brain size and functional capability. Rippon also suggested that, once any differences in brain size were accounted for, all the observed structural differences between the two sexes tend to vanish. In addition, Dean Burnett supported this in his comprehensive study demonstrating how most of the sex brain difference research has failed to look at the neural underpinnings of these claimed distinctions, therefore debunking the "pernicious" myth of brain sex differences.

From this lens, it would be foolish to further explore to the age-old belief of differences between male and female brains being present since birth. The notion of neuroplasticity, which has been around for over 30 years now, simply proves our brains are shaped and moulded by various factors from birth - all the way through the 'cognitive cliff' and into old age. It cannot be denied that releasing our grip on age-old facts can be quite daunting. However, fortunately enough, our brains are great learners, and we just need to have the courage to teach them the right lessons.

It is evident that once we acknowledge the malleable and ever-changing nature of our brain, we can notice how it is capable of interpreting and adapting to novel experiences. This includes all the signposts it crosses beginning from birth, which lead to various paths of development trajectories. Moreover, we should be wary of the consequence of gendered rules, as these sex-role stereotypes can function as self-fulfilling prophecies. For example, male babies are more often dressed in blue, and females in pink - this is binary coding which lacks scientific justification. Just look at what we are plunging into our children's brains, from the neatly labelled toys to the various social and cultural norms. "Blue-fiction and Pinkification", as Rippon said, must go. Don't you think it is about time that these binary distinctive labels get challenged?

Consider a game of Tetris or a construction toy like Lego, which happens to be more encouraged by parents for boys to play with. As the child plays these games, they

will be creating neural networks related to spatial and visual processing. Then, they enrol in school where they might improve even more at these tasks, and eventually, they may even find a profession, based on these skills, that asks them to spend all day, every day, strengthening those abilities further. From this perspective, it can be deduced that if these toys and training opportunities are gendered, then a clear gender divide based on the biological sex of an individual will be recognised - not the training an individual had.

Thus, currently, we should begin to take initiative in shifting our focus from simply contemplating the old answers spoon-fed to us, to also challenging the questions themselves. Re-framing our understanding of the idea of sex differences in the brain is crucial, especially in the context of justifying our beliefs and stereotypes. These tend to provide the binary labels of "boy" and "girl" and contend us into limiting ourselves to supposedly scientifically proven information. We need to let go of judging people based on their idiosyncrasies and adopt a mind rich in thoughts that are as flexible as our ever-changing brains.

With input from eye-opening breakthroughs in the avenues of neuroscience, it has come to my belief that nature and nurture are inextricably interconnected. Nevertheless, we should be aware that this debate is not limited to nature vs nurture but is also relying on the two-way constant flow of information between our brains and what surrounds them. Neurosexism ostensibly argues that researchers possess a bias to "hunt" for and overemphasise differences. I hope this article has sparked a little 'Aha!' moment in you, causing you to realise that the brain should not be more gendered than our beating heart or gurgling stomach.

Neural Portrait of the Human Mind: A Review

Writer: Ece Gokbayrak
Editor: Alice Wright

The following article is a review of the TEDTalk A neural portrait of the human mind, by Nancy Kanwisher. The opinions expressed belong to the author.

Brain imaging pioneer and Massachusetts Institute of Technology professor Nancy Kanwisher defined the human mind in her wide-spread Ted Talk video as: "A collection of highly specialised components, each solving a different specific problem, collectively making up who we are as human beings and thinkers."

Nancy Kanwisher began her talk by explaining prosopagnosia as the result of a damage to the part of the brain that results in only facial recognition impairment mental deficit. Kanwisher cleverly illustrated prosopagnosia by providing the scenario where a parent is going to pick their child up from school. She said "You realise that the children's faces don't look distinct, and none of them look familiar. You are only able to identify your daughter by the orange ribbon in her hair."

As prosopagnosia is associated with damage to the fusiform gyrus the current example supports the idea that the human brain is divided into distinct structures; each structure is responsible for different physiological and psychological functions. Luckily the effort to outline and understand these structures occurred rapidly, and somewhat easily, due to the advancement of imaging technology - especially Magnetic Resonance Imaging (MRI). Scientists figured out that MRI enables us to see internal anatomy at very high resolutions. When our neural circuits are activated, they need increased blood flow in order to supply the nutrients necessary to sustain that heightened activity. The functional MRI (fMRI) correlates the increased blood flow with higher intensity light on the brain scans, and this allows us to correlate activity in specific brain regions with certain cognitive functions.

In another experiment, Kanwisher described how scientists worked to identify the reason for seizures in a man with epilepsy. By total chance, two of the electrodes used to pinpoint the source of his seizures happened to be on top of his fusiform face area. Doctors asked him what happened when they electrically stimulated that part of his brain. This experiment proved that the fusiform face area is not only selectively responsive to faces but also causally involved in face perception. Therefore, this experiment is important in underlining the fact that brain imaging can never tell you which brain region is necessary for a specific mental function. Overall, there are mainly two takeaways from Kanwisher's speech: the brain is made up of structures that can simultaneously

have specific functions and wide-reaching general actions.

Kanwisher reminded her audience that the effort to understand the human mind and brain is worth investigating - even if it doesn't lead to the treatment of disease.

Personally, I found the video incredibly successful in breaking down complex science to be understandable to those without a background in neuroscience. It also sparked an interest to investigate how research in this field has progressed since the video was posted in 2014. There's been vast amounts of neuro-anatomy and -physiology published, plenty of which is accessible online. One experiment from 2021 developed an artificial neural network that could predict responses to stimuli in specific regions of the brain (one of them being the fusiform face area). This is just one project providing evidence for domain specificity in the brain, and there's still work to be done. Future research needs to make progress to understand neural computations that enable us to complete everyday functions.

A Focus on Yasmin Hurd

Writer: Priya Ord
Editor: Alice Wright

Dr. Yasmin Hurd is the Ward-Coleman Chair in Translational Neuroscience as well as Professor of Psychiatry, Neuroscience and Pharmacological Sciences at the Icahn School of Medicine in New York. She is also Director of the Addiction Institute at Mount Sinai, New York. Dr. Hurd's multidisciplinary research is globally recognised as she investigates the neurobiology underlying substance use and related psychiatric illnesses. Her laboratory uses a translational approach to research - the team studies addiction from a variety of perspectives: animal behaviour, molecular and cell biology, pharmacology, psychology, neuroimaging, bioinformatics, and biotechnology.

A major focus of the research is directed to risk factors of addiction, including genetics and developmental exposure to drug use, such as to cannabis. The group also conducts human clinical trials for developing novel therapies for opioid dependence.

Her Early Life:

Hurd grew up in Jamaica and later attended Binghamton University in New York. She described her time there by saying: "I was a nerd and loved school… I didn't fit in." It was clear that Hurd had an insatiable scientific curiosity regarding the workings of the brain. To help pay college expenses, she worked as a technician in a vivarium (where research animals are housed) - an experience that undoubtedly set her on a path to a career in neuroscience research. She completed her PhD at the Karolinska Institutet in Sweden and worked on micro-dialysis. Hurd was also a Pharmacology Research Associate Fellow at the National Institute of Health, and later a Staff Fellow there too.

Her Career:

Hurd returned to the Karolinska Institute as a faculty member and professor for 13 years, before beginning her career at Mount Sinai.

She is the former director of the medical school's combined MD/PhD Medical Scientist Training Program and has served on numerous advisory boards and societies, including the National

Institute of Drug Abuse (NIDA) Board of Scientific Counsellors, and the National Academy of Medicine.

According to Scopus, Hurd's work has been cited approximately 13,000 times, by 9550 documents. She has an incredible H-Index (scientific research impact) score of 67. As of 2020, Hurd was receiving six ongoing research grants from the National Institute on Drug Abuse (NIDA).

Hurd's work on the neurobiology of addiction has been shared by a variety of popular media sources, such as podcasts and documentaries. Some of Hurd's findings have gained public attention. For example, her investigations into the 'gateway drug theory', and revealing the potential for cannabidiol (a chemical found in marijuana - often referred to as CBD) to be a treatment for patients experiencing opioid dependence, gained a great deal of recognition.

Dr. Hurd has made momentous findings, but her journey to success has been far from easy. Hurd is the only black tenure-track basic science professor at Mount Sinai and, as one of few black female neuroscientists in high level academia, she often experiences racial biases. Hurd wrote 'Addressing racism and disparities in the biomedical sciences', published in Nature Human Behaviour in July 2020, to shine a light on and further detail her experiences with these issues.

Dr. Yasmin Hurd has undoubtedly demonstrated how to break down scientific barriers and carry out patient-first research, while encouraging and inspiring the next generation of neuroscientists.

Learn more about Dr Hurd's work:

Podcasts:

Mind & Matter by Nick Jikomes– Yasmin Hurd: CBD (Cannabidiol), Opioids & the Neurobiology of Addiction (#19)

People Behind the Science by Dr. Marie McNeely: Episode 594 – Paving Pathways to Success Studying Substance Abuse and the Brain – Dr. Yasmin Hurd

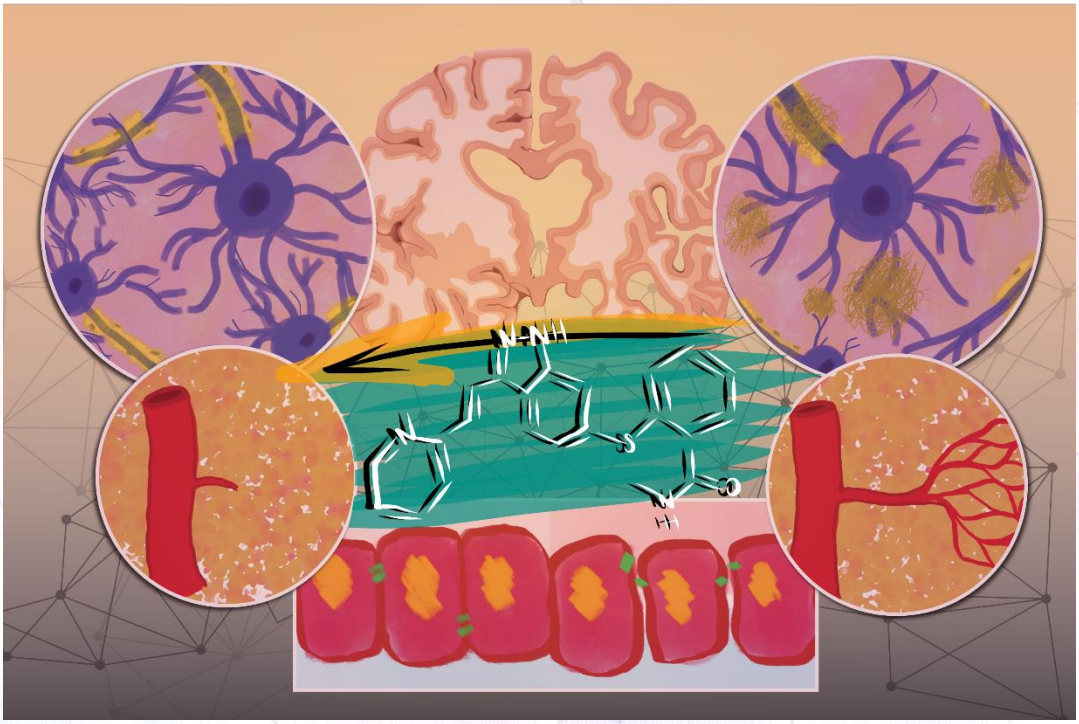
Netflix:

TEDTalk: Could CBD help opioid users overcome addiction?

Lecture: The Vulnerable Brain: Pathways to and from Addiction with Yasmin Hurd, Ph.D.

The Chemotherapy Drug That Reverses Alzheimer's Symptoms in Mice

Writer: Anaiya Kaka
Editor: Alice Wright
Artist: Jorvani Cruz Villarreal



Alzheimer's Disease is a form of dementia that causes loss of memory, orientation, reasoning, and other cognitive functions. The Alzheimer's Society published a statistic stating that there are nearly 40 million people living with dementia worldwide, and 50-75% of those cases are caused by Alzheimer's Disease.

Current theories regarding Alzheimer's suggest that mutations in the amyloid precursor protein (APP) cause increased amyloid beta production together with the appearance of neurofibrillary tangles (NFT), which are composed of hyperphosphorylated tau protein. Accumulation of these leads to widespread neuronal dysfunction. In recent years, 65% of clinical treatments have focused on amyloid beta as a therapeutic target; however, have been unsuccessful. Existing treatments focus on "symptomatic" agents that seek to improve cognitive and behavioural symptoms but do not treat the underlying course. Therefore, new approaches and therapeutic targets are needed to find a cure for Alzheimer's.

There is growing evidence to suggest that, alongside neurons, the neurovascular unit is altered in Alzheimer's, and the disease is mediated by vascular pathogenesis. Cerebrovascular amyloid beta deposition is seen in the majority of Alzheimer's cases, and this causes vessel stiffness and smooth muscle degeneration. A recent hypothesis suggests amyloid beta deposition causes cerebrovascular neoangiogenesis - the growth of blood vessels from existing

vasculature in the brain, along with disruption to the blood-brain barrier junction. This leads to defective neuro-vasculature, which alters the blood-brain barrier and coronal blood flow, which in turn compromises amyloid beta deposition clearance. This vicious cycle is what this study looks to target and treat.

If elevated neoangiogenesis underpins the progression of Alzheimer's, then anticancer drugs that inhibit this could reverse the pathology associated with Alzheimer's. In this study, the anticancer drug Axitinib was examined in a mouse model to see whether neoangiogenesis, blood-brain barrier leakiness, and amyloid beta deposition could be resolved by this new therapeutic intervention.

The Experimental Trial:

A trial was done in September 2021 by a research group from the University of British Columbia, Canada, to see whether Axitinib could restore memory and cognitive processes in mice.

Mice that expressed the mutant APP were used alongside wild type mice which did not express the mutation. Axitinib, at a dose of 10mg/kg dissolved in 100% dimethyl sulphoxide (DMSO) at 40 mg/ml and subsequently diluted in lead sulphate (PBS), was administered to both groups of mice. The control groups were given the drug delivery vehicle (PBS+DMSO) alone. The mice were treated three times a week for one month before being tested for cognitive performance.

The first test was the open field test, which assessed cognition and awareness of potential danger. A plexiglass chamber was used, where the floor was divided into central and peripheral regions. A tracking camera and computer system recorded the path travelled and time spent in the different field regions. In this test, mice with intact cognition and awareness of potential danger tended to stay in the peripheries and away from the open and brightly lit centre. The wild type mice (pre- and post-treatment) spent the majority of time in the peripheral region, while the mice with the mutant APP and treated with the control explored the field indiscriminately. However, after being treated with Axitinib, the mice behaved more like the wild type mice - exploring the peripheries more than the centre, implying their cognitive abilities and danger awareness improved after treatment.

The next test - a Y-maze test - assessed spatial and working memory. A symmetrical Y-maze with a grey floor plate and grey walls was used. The wall at the end of each arm was identified by a different colour: white, blue or red. A one day trial was done where the mice were tracked while moving freely through the arms. Animals with sustained cognition would remember the arm it entered last and would avoid re-entering. As expected, pre-treatment mice with the mutant APP performed more poorly than their wild type counterparts. However, the mice with the mutant APP who were treated with Axitinib had a performance that was indistinguishable from wild type mice, showing that this drug improved sustained cognition and spatial awareness in these mice.

The effect of Axitinib on associative memory was tested using contextual fear conditioning. Normal mice were expected to remain stationary when placed in an environment they had previously been electrically shocked in. Once again, the pre-treatment mice with the mutant APP had significantly lower scores compared to the wild type mice and the mice who had the

mutant APP but were treated with Axitinib had scores similar to the wild type mice. This showed that Axitinib improved associative memory in these mice.

Assessment of long-term memory and short-term memory was performed by testing mice with the 5 day Radial Arm Water Maze. Eight arms extended form a central area with an escape platform at the end of any of the four alternate arms. The mice were tasked with locating the escape platforms. Performance of memory and learning was gauged based on the average time taken to find an escape platform and the number of errors. It was found that the Axitinib-treated mice with mutant APP showed cognitive learning (reference memory and working memory) that was indistinguishable from wild type animals while the control mice had significantly worse results.

Additionally, Axitinib was found to reduce the cerebrovascular pathology seen in Alzheimer's. The brains of the mice were analysed, and researchers found decreased amyloid beta expression, and an angiogenic marker. In the brains of untreated mice there was hypervascularity, but that was greatly reduced in the group treated with Axitinib. It also reduced blood-brain barrier disruption.

Overall, the study was extremely promising and found that spatial awareness, exploration, associative memory, short and long term memory all increased in the mice with mutant amyloid precursor proteins that were treated with Axitinib. It altered the cerebral pathology of Alzheimer's and reduced neoangiogenesis. However, the treatment has only been applied to mice, so clinical trials will be needed to assess the effectiveness of this treatment in human patients.

Nevertheless, researchers are hopeful this drug can be used in humans. They also hope that a new therapeutic approach, looking at the neurovascular unit as opposed to just amyloid beta deposits, could be a new milestone in the clinical treatment of Alzheimer patients, and one that can help the millions of people in the world who are suffering from this devastating disease.

The Link between Inflammation and Mental Health

Writer: Angela Lee
Editor: Ellie Roberts

Inflammation is a defence tool used by the body in response to perceived threats, including stress, injuries, and infections. As a result, the immune system recruits inflammatory mediators - this strategy is a result of our evolution. The soreness surrounding an infected wound is an inflammatory response required to isolate bacteria and viruses, ensuring that they are destroyed before spreading further. When inflammation is triggered, small proteins called cytokines are produced - these are signalling molecules which facilitate the response. Cytokine levels can be measured to assess the level of inflammation in the body.

There is growing evidence to suggest that inflammation in the brain can affect how we feel. This influence can occur through many systems, including the immune system, metabolism, mood, stress responses, sleep, and more. There is much that remains to be discovered regarding the mechanisms underlying the effects of inflammation. The link between inflammation and these systems, each a vital component of emotional function, is now irrefutable from research findings, including higher levels of cytokines in patients with depression.

So, why do people's brains become inflamed in the first place? Exposure to stress, poor diet, infections, and environmental toxins can all cause inflammation and subsequently initiate associated mental and physical symptoms.

Stress is relayed to the central nervous system, where it stimulates the release of the hormone cortisol. Cortisol increases the level of inflammatory mediators both centrally and peripherally. Prolonged stress, or exposure to childhood adversity, has been linked to the later development of inflammatory conditions. Experiencing prolonged stress during childhood is associated with gut inflammation, which can lead to mental health problems, as well as physical conditions like Crohn's disease.

Stress hormones also appear to affect the balance of bacteria in the gut microbiome. Microbial imbalances in the gut can lead to the damage of the gut lining (known as "leaky gut"). This can result in toxins and bacteria leaking through the intestines into the bloodstream, triggering inflammatory responses. Inflammation can eventually spread to other organs, such as the brain, increasing vulnerability to a variety of conditions including autism, attention deficit hyperactivity disorder, borderline personality disorder, bipolar, depression, and anxiety. One study found that those with post-traumatic stress disorder (PTSD), obsessive compulsive disorder, or anxiety, had significantly higher levels of inflammatory markers compared to those without.

Adverse experiences during childhood can also affect the gut microbiome. One study found that "children with a history of early caregiving disruptions had distinctly different gut microbiomes from those raised with biological caregivers from

birth." Brain scans of the children included in the study showed correlations between brain activity and the presence of particular bacteria within the gut microbiome. Children raised by parents showed greater gut microbiome diversity, in addition to increased activity within the prefrontal cortex - a region of the brain associated with emotional regulation.

One study compared the gut bacteria of trauma survivors who developed PTSD with the bacteria of those who did not. Those who developed PTSD demonstrated significantly lower levels of Actinobacteria, Lentisphaerae and Verrucomicrobia. Experiencing childhood trauma was also associated with significantly lower levels of these bacteria, suggesting that those with childhood trauma are at greater risk of later developing PTSD. The authors theorised that low levels of these bacteria could result in immune system dysregulation and high levels of inflammatory markers in trauma survivors with PTSD; measuring levels of inflammatory markers in individuals following a traumatic event was shown to predict the development of PTSD later in life.

Doctors already routinely test inflammation levels in patients with, for example, cancer, Crohn's disease, and rheumatoid arthritis, and this could potentially be done for patients with depression to show who might benefit from existing anti-inflammatory drugs. These range from standard painkillers such as aspirin and ibuprofen - the so-called non-steroidal anti-inflammatory drugs (NSAIDs), to heavyweight rheumatism drugs. The fact that patients with depression and chronic inflammation are likely to have raised cortisol levels from stress means that relaxation therapies such as yoga and meditation might also be helpful to them.

The connection of inflammation with depression is still at a nascent research stage, and further clinical trials are required. Even so, it is already encouraging researchers to experiment with combining drugs that affect the various mood-changing brain chemicals or neurotransmitters, such as dopamine and endorphins, rather than just targeting serotonin with SSRIs. This is because one of the effects of inflammation is to alter the levels of all these brain chemicals. Bringing inflammation into the picture does not just hold out the hope of a new, improved toolbox, but rather, views depression in a new light — not as a mysterious mind malfunction, but something as physical as arthritis or heart disease.

More and more studies suggest that depression and bipolar are accompanied by immune system dysregulation and inflammation, and high levels of cytokines. Inflammation has been found to trigger depression, almost like an allergic reaction, and one study suggested that immunotherapy has potential in the management of depression.

About one in three people with depression don't respond well to existing drug and psychological therapies, and experiencing drug side effects is common. There is an increasing amount of evidence suggesting that inflammation contributes to the development of depression, although the results from clinical trials attempting to use anti-inflammatory agents as a potential treatment have so far proved inconclusive. However, research is still in its early-stages, and as more is discovered about the relationship between inflammation and mental health conditions, there will be greater hope for potential future treatments which target this link.

The Gut Microbiome that Influences Your Health

Writer: Alara Egeli
Editor: Ellie Roberts
Artist: Jorvani Cruz Villarreal

To some extent we can all relate to the feeling of two-way communication between our gut and brain. If we drink alcohol and feel down the following day, it's partly because it has played havoc with our gut microbiome (microorganisms living in our digestive tract). Extended alcohol consumption can increase the permeability of the gut, which may cause microorganisms to enter the circulatory system and trigger inflammatory responses. This has a knock-on effect on the production of various neurotransmitters, such as serotonin, glutamate, and GABA. Similarly, when we anticipate exams, we may feel 'sick to our stomachs'. These 'gut feelings' are simply thoughts relayed from the brain to the gut via the Gut-Brain Axis.

The Gut-Brain Axis is the bidirectional communication between our central nervous system and the neurons and cells in our gastrointestinal tract (termed the enteric nervous system). The establishment of the Gut-Brain Axis Theory isn't new, it dates back to the 1800s and has been undergoing research ever since.

Our gut is a major producer of neurotransmitters such as histamine and serotonin, which are essential for peristalsis (the series of muscle contractions that moves food through the digestive tract) and 'gut feelings'. The microbiota are capable of directly activating the vagus nerve, which transmits information regarding gut health to the brain. The afferent endings of the vagus nerve can be stimulated by metabolites that microorganisms produce in the gut. In

the brain, this information is coordinated into actions that alter inflammatory actions and gut permeability. In cases of stress, proinflammatory mediators, such as cytokines, are released.

Similarly, stressful situations activate the hypothalamic-pituitary-adrenal axis. This uses the actions of hormones and inflammatory mediators to increase gut permeability, altering the composition of the microbiota and the gastro-intestinal epithelium. This, like in cases of prolonged alcohol consumption, can lead to microorganisms from the gut entering the circulation and triggering immune responses.

With trillions of microorganisms living in our gut, what we put on the end of our forks has the greatest impact on our health, and that includes mental wellbeing. Many patients who suffer from depression have raised monoamine oxidase enzyme (MAO) levels. This MAO enzyme causes inflammation and breaks down mood regulating neurotransmitters such as dopamine, serotonin, and norepinephrine.

The American Gut Project is the largest people's science microbiome project, and includes over 150 countries. The single biggest factor affecting gut health found by this project is the diversity of plants we need to eat to support a healthy gut microbiome.

If getting the right mix of nutrition to feed the gut microbiome is vital for better health, including brain health, how can we feed the vast variety of good bacteria living there?

The short answer is fibre. Fibre comes exclusively from plants, and how we get that fibre is critical to its effectiveness. Plant-based whole foods, naturally high in fibre, aid the repair of inflammation in the brain, restoring the equilibrium of neurotransmitters. When we eat certain fibres, they are converted to Short Chain Fatty Acids (SCFAs). SCFAs restore the balance between the healthy microbes and the inflammatory microbes, repairing the blood-brain barrier which affects our memory and mood.

There are three main SCFA types: acetate, propionate, and butyrate. They are made up of two to four carbon atoms connected by acetate, propionate, or butyrate compounds. Due to study design, SCFAs are usually looked at in isolation, but in the real world these molecules don't function in isolation. Nutrition functions as a whole. Highly processed foods, with essential components removed and isolates added, (including most breakfast cereals, white flour, sugar, and oil) can't provide us with the same effects and benefits as wholefood fibre.

Regular consumption of fat and junk food decreases our dopamine sensitivity, and this has been measured in real time by PET scans. A neuroimaging study found that the regular consumption of ice-cream is linked to a reduction in senses of pleasure, parallel to observations in drug addicts. The consumption of whole foods doesn't illustrate the same effect, although it's important to note that economic or educational barriers may reduce the accessibility of whole foods for some people.

In summary, improving the state of our gut microbiome not only enhances our brain's function, but also helps to prevent disease, promotes longevity, and increases our overall happiness.

For more information from the writer of this article, see openwater.uk.com

Where the Human Brain Meets AI: DeepMind and Neuralink

Writer: Ashvni Narayanan
Editor: Alice Wright
Guest Editor: Alessia Qui

In many regards, artificial intelligence (AI) advancement is working to emulate aspects of human intelligence in computers. As early as the 1940s, our understanding of neuroscience inspired the inception of the field of AI. In 1957, John von Neumann suggested imitating simple neuron functions using telegraph relays or vacuum tubes. A year later, intrigued with the operation of fly eyes, Frank Rosenblatt began work on the Perceptron - the world's oldest neural network still in use today. The revolutionary 1957 Nobel Prize-winning work of Hubel and Wiesel on the primary visual cortex of cats inspired further investigation into neural networks. After that, AI research strayed from neuroscience, but due to renewed funding, efforts have resumed since the 1980s. AI is fast becoming a game-changer - companies like DeepMind and Neuralink are teaming together neuroscientists, computer scientists and mathematicians in a bid to win the race to perfecting AI.

DeepMind, backed by Google, researches new methods of machine learning. They aim to replicate brain functions and come up with algorithms that only the human brain has been known to do. Their claim to fame was AlphaGo - the first program to beat a human World Champion at Go (a high complexity strategy game). In 2016 they began work on AlphaFold, which is an AI that can accurately predict protein shapes. AlphaFold won the protein structure prediction challenge in the Critical Assessment of Protein Structure Prediction (CASP13) 2018 project. Recently, DeepMind developed a deep reinforcement learning algorithm (Deep Q-Networks) to understand what human neural activation was evoked by video games. The algorithm stored all of the experiences of the AI playing the game, and then replayed these experiences to develop reinforcement learning. The results shed light on the principles underlying reward-guided decisions in naturalistic domains. The AI achieved human-level performance in about 25 games, which is a remarkable development.

Neuralink, an Elon Musk product, on the other hand, focuses on neural engineering. They aim to build a brain-machine interface that uses signals from the motor cortex to control a computer. First tested on a rodent model, the interaction occurs via a neurosurgical robot implant that uses a dense array of electrode "threads". The Link was first tested by recording somatosensory signals cued from pigs exploring their environments. A video Neuralink released showed Pager, a macaque monkey, playing MindPong - a video game using brain signals sent wirelessly via an implanted device. Elon Musk claims the Link will be useful for paralysis patients, who will be able to use smartphones with their mind faster than manually. Conditional to obtaining the

required medical permissions, the company is set to begin clinical trials on patients with spinal cord injuries in late 2022.

There are several concerns regarding the ethics of these companies' practises. Reinforcement learning models (both supervised and unsupervised) require vast amounts of data. Google's acquisition of DeepMind made controversial news as more than a million confidential patient records had previously been shared by the NHS with DeepMind, in order to build an app warning doctors about patients at risk of acute kidney injury. This was done without the knowledge of the patients, thus raising privacy concerns. Neuralink, too, is rife with speculation and controversy. While some are afraid that their innermost thoughts might be recorded, others are sceptical that the Link might later be used as a medium to control the masses, or even a target for hacking.

Technological advancement has always been a double-edged sword. These ideas are ripe with the promise of a better future, sometimes at the demise of privacy and safety. Whether these advancements will come to fruition is yet to be seen. One thing is certain: where these companies source their data from, and how they use it, must be transparent with the public.

How Brain-Computer Interfaces Could Change the World

Writer: Beyza Cetin
Editor: Ellie Roberts

Before we can begin to address the current developmental status of Brain-Computer Interface (BCI) technology and its applications, it is necessary to establish what it comprises. A BCI can be defined as a system involving communication between the electrical activity of the brain and an external device such as a computer.

As of now, BCIs have predominantly been used to help people living with disabling conditions associated with impaired motor or sensory function. BCI systems have been designed to allow people to control robotic arms or wheelchairs, simply by imagining the action. This type of system comprises a wireless electronic scalp helmet which uses electro-encephalography (EEG) to translate neural activity into action. Machine learning is used to analyse and subsequently identify the neural signals which are produced when the wearer imagines motor activity.

Deep Brain Stimulation (DBS) has been used to enhance the living standards of patients with movement disorders such as Parkinson's disease. DBS is a type of BCI, whereby a device called a neurostimulator is implanted just below the collarbone and sends electrical impulses through implanted electrodes to target specific brain areas. DBS is approved in the UK, as well as other countries, to treat Parkinson's disease, tremor, and other conditions. However, it has not yet been approved for NHS funding in England to treat epilepsy, where it may be useful in the management of refractory seizures.

Sensory BCIs have also been developed with the aim of replacing the loss of sensory function, such as hearing. Cochlear implants convert sound from an external microphone into electrical stimulation of nerve fibres throughout the cochlea, providing artificial sensory inputs to the auditory system to improve hearing.

Established in 2017, Facebook Reality Labs (FRL) created a BCI project with the aim of allowing Facebook users to write at least 100 words per minute, just by imagining what they wanted to say. Facebook has used this project, and research it's been funding at the University of California San Francisco (UCSF), to help develop an implantable prosthesis for those who have lost the ability to speak. In 2019, the researchers at UCSF demonstrated that a small set of words and phrases could be decoded, in real time, from brain activity. However, in 2021, huge progress was made as the development of the UCSF BCI device had allowed someone with severe speech loss to type out what they want to say, just by attempting speech. This was achieved through decoding brain signals from the motor cortex to the muscles of the vocal tract.

By linking brains to the internet, BCIs could allow some individuals to increase their cognitive capacity. Neural nanobots could be used to connect the neocortex to a "synthetic cortex" in the cloud. These nanobots would cross the blood-brain barrier and position themselves among or within neurons before wirelessly transmitting information to and from a cloud-based supercomputer network. It is suggested that this would allow individuals to access all cumulative knowledge available in the cloud. Furthermore, the idea of a "global superbrain" has been proposed, whereby networks of individual human brains would be connected to the cloud, enabling collective thought and collaboration.

From a different perspective, BCIs could also be used for military purposes. The National Defense Strategy (NDS) highlights that the implications of such technologies on the battlefield must be anticipated in case of future conflict. According to one article, the testing of BCIs in animals will likely lead to a variety of military applications, and that targeted stimulation of various areas of the brain may also boost learning or physical capabilities which would be useful in military practice.

In conclusion, BCI technology is far from being science fiction-esque, and is indeed a useful reality with wide-spread applications that we should be ready to embrace in the near future. However, maximisation of privacy and security are key to eliminating the ethical issues associated with BCIs, for example through using de-identification methods such as Privacy-Preserving Data Mining (PPDM) and Privacy-Preserving Data Publishing (PPDP). These methods work to allow information extraction without disclosing sensitive or identifying details.

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