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GENDER INEQUALITY IN SCIENCE TODAY

Auckland University Women in Science explores the challenges women face in science, and how we can come together to confront these issues.

Editors' Note

Kia ora koutou katoa, and welcome to UoA *Scientific*'s Summer School Edition.

After a very busy 2022, we hope everyone enjoyed a restful holiday and a well-deserved break. We wish you a happy and healthy start to the new year, whether you are still on vacation or returning to work. Hopefully, you were able to find some sun despite all the rain!

UoA Scientific is thrilled to begin 2023 with an expanded executive team. A hearty welcome to the new members, and congratulations on a well-received inaugural issue of 2023. This edition is packed with thoughtful articles from Scientific executives and guest writers.

In this issue, Riya Balia delves into the hot topic of antibiotic resistance and how one recent study has demonstrated the possibility of using antimicrobial peptides from the human proteome. Sarah Moir delves into the research of Organoids, a rapidly expanding branch of stem cell biology, and Milly Darragh investigates a study that reveals differences in how "neutral faces," frequently used in psychological assessments, are perceived by different patients.

Finally, we are delighted to have collaborated with Auckland University Women in Science (AUWS), who has written a thought-provoking article on contemporary gender inequalities in science. This is a timely and important read that discusses the huge gaps in female representation in STEM fields today.

Thank you to all of our writers, as well as new and experienced executive members, for putting together this fantastic Summer School issue and we hope you, the readers, enjoy all of the fantastic articles it contains.

Nga mihi maioha,
Shyla Mani, Social Media Officer for UoA Scientific 2023

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Academic

The Battle Against Bacteria

Riya Balia

Microbiology

Since bacteria met antibiotics, we have been facing antibiotic resistance. How is resistance passed on between bacteria? How has it been accelerated? And most importantly, how can we fight the danger it poses to our society? One way is to find new antibiotics. We investigate an exciting study that focuses on antimicrobial peptides found in the human proteome.

"When I woke up just after dawn on September 28, 1928, I certainly didn't plan to revolutionise all medicine by discovering the world's first antibiotic, or bacteria killer."

\- Alexander Fleming [1]

Fleming's discovery of penicillin did, in fact, transform the entire field of medicine— he had introduced a completely new area of study in medicinal research. In World War II, antibiotics proved crucial in saving the lives of soldiers from bacterial infections caused by wounds or the consumption of contaminated food and water. Today, they are used to protect us from previously more dangerous and common diseases, including [2]:

- Tuberculosis
- Strep throat
- Whooping cough
- Urinary tract infection
- Sepsis
- Pneumonia

There are many specific means by which antibiotics inhibit bacteria. Penicillin, for example, impedes cell wall synthesis by interfering with the production of peptidoglycan, a molecule that surrounds the cytoplasmic membrane, protecting some bacterial cells from environmental stresses and providing cell stability. Other antibiotics can disrupt cell membranes or hinder folate, DNA, RNA, or protein synthesis. Each method of attack violates a significant function without which the targeted bacteria cannot survive.

Although antibiotics have inhibited many species of bacteria, thereby saving many millions of lives, Fleming foretold their demise. He had warned that it would not be difficult to make microbes resistant to penicillin in the laboratory. Indeed, in 1940, it was reported that an E. coli strain inactivated penicillin by producing penicillinase [3]. Resistance has since been seen to nearly all antibiotics that have been developed. The timeline for a few antibiotics is shown in Figure 1.

Resistance to antibiotics appears due to genetic mutations and is passed on by vertical and horizontal gene transfer. Mutations occur following errors in DNA replication while a bacterium rapidly reproduces. With a bit of luck, one mutation may prove beneficial against an antibiotic the bacterium is facing. This beneficial mutation can be passed down vertically through the bacterium's following generations or to any species of bacteria in the local proximity by methods of horizontal gene transfer: transduction, transformation, and conjugation, as shown in Figure 2.

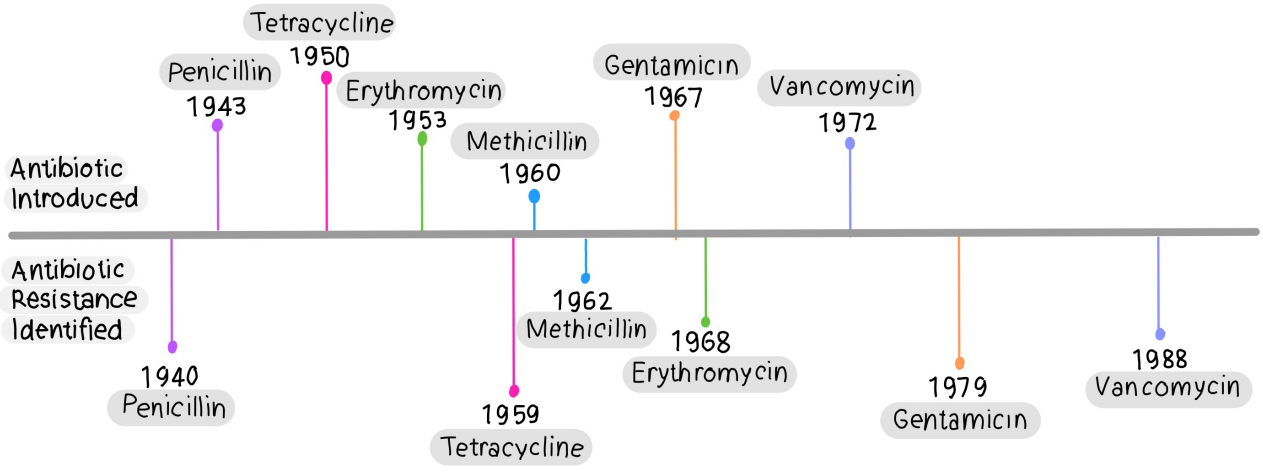

Figure 1: A timeline showing the rate at which resistance to some antibiotics has developed [4].
Note: Penicillin was discovered in 1928 but was not widely used by the general population until 1943.

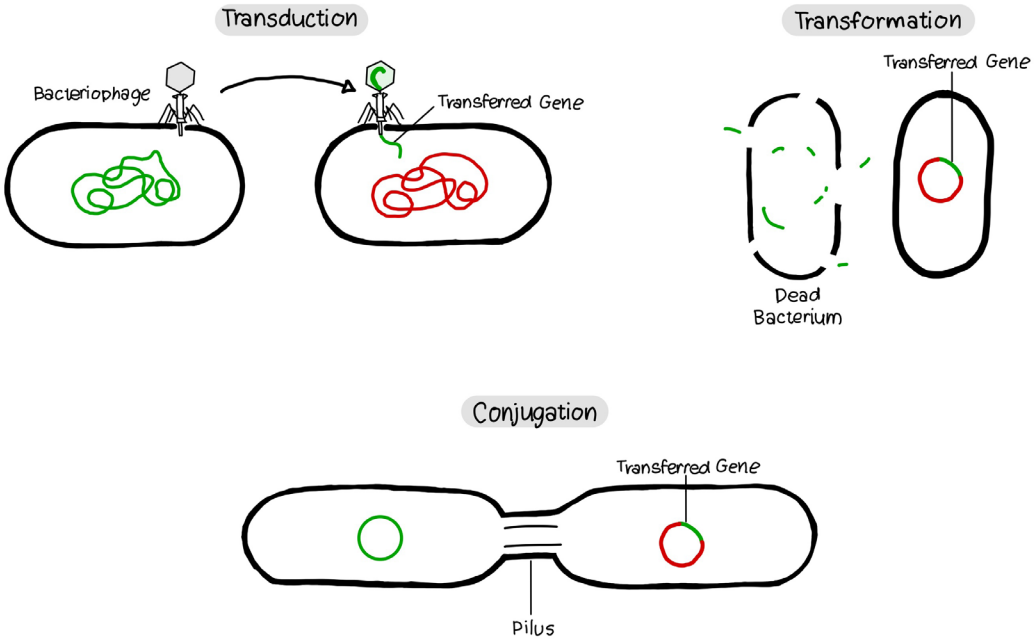

Figure 2: A diagram showing the methods of horizontal gene transfer [5].

Rapid resistance rates have been accelerated by our misuse and overuse of antibiotics. According to the New Zealand Ministry of Health, about half of the people who visited their GP in 2017 were dispensed with at least one antibiotic [6]. Antibiotics are prescribed more often than necessary because doctors feel pressured to provide a solution to common problems [7]. Examples of misuse include flu sufferers who take antibiotics without realising that the drugs are ineffective against viruses. In the agriculture industry, antibiotics are used excessively in livestock to promote growth and prevent the spread of disease [8]. Using antibiotics when not needed may lead to health consequences for individuals and increase the burden of antimicrobial resistance in the long term. Even now, the rise of antibiotic-resistant bugs has led to severe infections, killing about 700,000 people worldwide each year, with the number expected to rise to ten million by 2050 if no action is taken [9]. In short, increased mortality, prolonged hospital stays, and higher medical costs are the consequences of drug-resistant infections [10].

Several measures can be taken to address the issue of antibiotic resistance. Campaigning for awareness, creating guidelines for prescriptions, and placing restrictions on drug use in agriculture are a few ideas. Another approach would be to hunt for new antibiotics. Scientists are doing exactly that. Not only have we developed new scaffolds in labs, we have also found more in nature. Potential antibiotics have been discovered in bacteria itself, fungi, and now in a previously untapped source — our very own bodies.

"The human body is a treasure trove of information, a biological dataset. By using the right tools, we can mine for answers to some of the most challenging questions." says Dr Cesar de la Fuente, the leader of an exciting study at the University of Pennsylvania [11]. De la Fuente's team identified encrypted* peptides with antimicrobial characteristics by using artificial intelligence (AI) to screen the human proteome [12].

AI technology used a specific algorithm to find encrypted peptides based on their ability to fit into the parameters of the search. The parameters focused on physiochemical characteristics all antimicrobial peptides have in common: 8 to 50 amino acids in length, positively charged and possessing both hydrophobic and hydrophilic parts [13]. An initial scan of the proteome identified 2,603 peptide antibiotics. Only 55 of these were synthesised in the lab to verify the antimicrobial characteristics of the algorithm-derived peptides. When assessed against eight common pathogens (*E. coli ATCC11775, Pseudomonas aeruginosa PAO1, P. aeruginosa PA14, Staphylococcus aureus ATCC12600, E. coli*

Note

*The term 'encrypted' is used to describe the antimicrobial peptides because they are hidden within larger proteins that were traditionally thought to have one biological function [14].

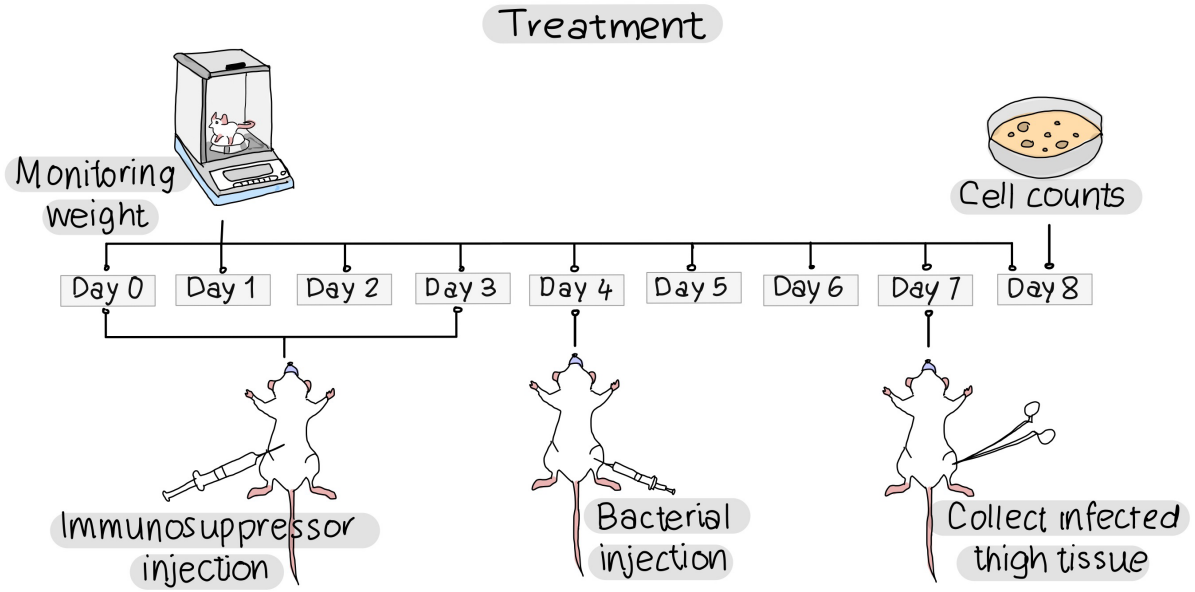

Figure 3: A schematic showing the timeline of the leg infection experiment [12].

AIG221, E. coli AIG222, Klebsiella pneumoniae ATCC133883, Acinetobacter baumannii ATTC19606), 63.6% of the selected peptides displayed antibacterial capacity. Additionally, the encrypted peptides exhibited only minor effects on gut and skin microbes. This evidence suggests they may contribute to the equilibrium between the human body and its inhabitant microorganisms.

The study identified encrypted peptides as agents that do not readily select for bacterial resistance. The peptides damage the outer membrane of a chosen bacterium, a crucial permeability barrier. According to de la Fuente, damaging membrane permeation would require more energy and multiple generations to confer resistance in bacteria [13].

For antibiotic discovery, like many other branches of pharmaceutical science, animal testing for reactions is a crucial component. This study used mouse models to assess the activity of selected encrypted peptides. In one mouse experiment involving two of the most potent peptides, the number of bacteria present in skin infections was reduced by magnitudes of five to six. Another mouse experiment involved a combination of peptides for an infection in the leg. The result showed a reduction of bacteria by a magnitude of three. This experiment is outlined in Figure 3.

The method of attack and the results of the mouse experiments** are among the many pieces of evidence that indicate the newly discovered peptides are promising candidates for sustainable antibiotics. However, more work will be required before antimicrobial peptides may be used by patients. The task of finding, developing, testing, and validating antibiotics is a lengthy process. Nonetheless, it is necessary if we wish to hold higher ground in this war. While our battle against bacteria will never cease, we can continue outwitting it.

Note

**To add to the large reduction of bacteria in infections, the mice did not lose weight under experimentation so we may believe that the treatments are safe.

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Riya is a second-year Biomedical Science student specialising in infection and immunity. Aside from her interest in all things immunology, she is passionate about space, art, and fresh fruit ice cream.

Review

Do Psychiatric Conditions Change the Way We See Faces and Emotions?

Milly Darragh

Neuroimaging

This article reviews a meta-analysis regarding how neutral faces may not be neutral for axis-I psychiatric disorders. We explore the mechanisms of why each disorder may not see a neutral face the same way a neurotypical mind might.

A review of:
Rethinking the use of 'Neutral' faces as a baseline in fMRI neuroimaging studies with Axis-I Psychiatric Disorders
(Filkowski & Haas, 2016)

When you look at the following face, which emotion do you see?

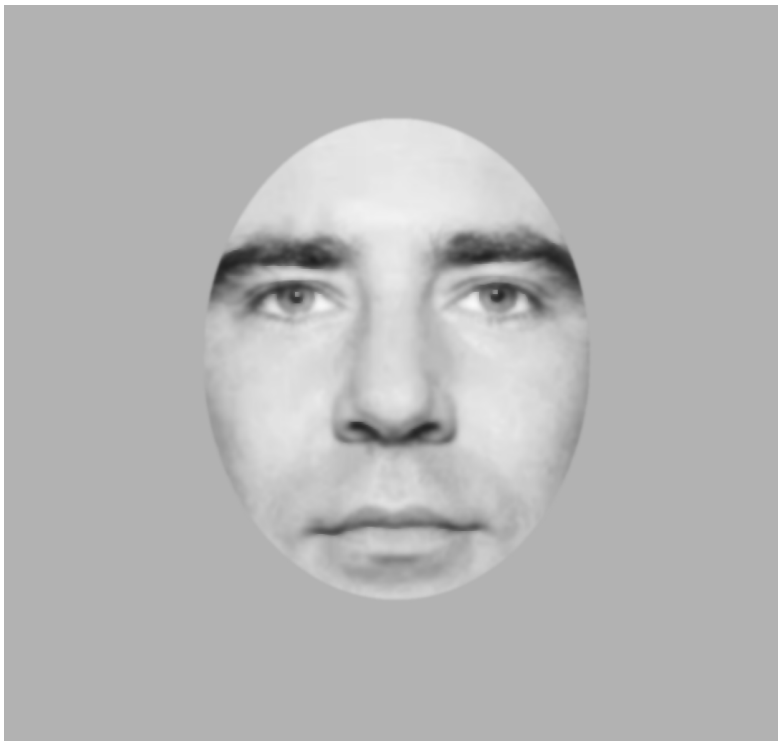


Figure 1: An example of neutral face stimuli. Adapted from an image by STYLEPICS from depositphotos. https://depositphotos.com/13653328/stock-photo-handsome-caucasian-man-blue-eyes.htm.

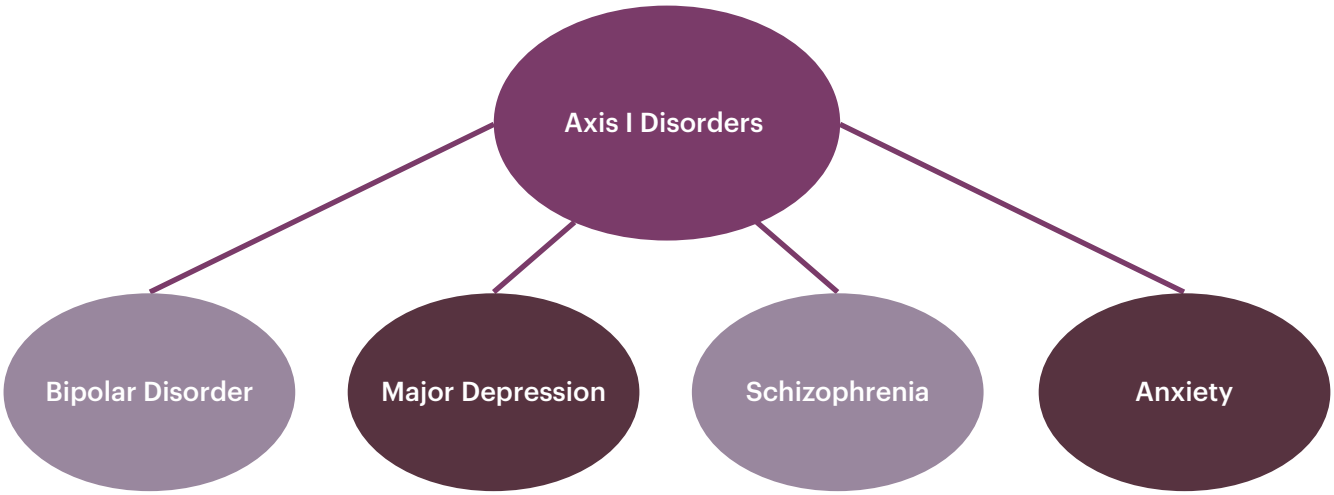
Happy? Sad? Angry? Scared?

Statistically speaking, you probably said 'neutral' or 'normal'. That is because this stimuli has been designed and used as a 'neutral stimuli' in psychology and neuroscience in many experiments and studies [1]. This face is supposed to represent no emotions, or act as a palette cleanser in experiments where multiple faces or emotions are being presented. However, does this stimulus actually work as planned? Questions regarding the subjectivity of a neutral face in psychological studies are growing, yet research diving into the effectiveness of this method has only just begun [1]. Specifically, how do different cognitive conditions affect the processing of a neutral face? With mental health conditions being one of the most common conditions worldwide, Filkowski & Haas (2016) explored why it may be time to rethink the use of neutral face stimuli in neuroscientific research [1].

Many psychological and neurological studies have been published using neutral face stimuli as an experimental technique. Facial stimuli in scientific experiments can be used to test a multitude of effects, with well-documented activity in the amygdala, the fusiform gyrus, and the prefrontal cortex of the brain [1]. Of course, we know that the brain cannot be separated into specific functions, yet these areas have continually shown activation when participants are exposed to facial stimuli. The use of neutral face stimuli assumes that each patient will respond and process these stimuli the same way in order to create a baseline of activity; however, this may not be the case [1].

The validity of the 'neutral face' across participants have recently been called into question – how can we ensure that each participant is processing these stimuli as intended? A large aspect of this issue lies in how neurodiverse brains process stimuli differently from neurotypical brains [1]. Whilst some research has investigated the effect autism, ADHD, or Tourettes have on facial processing, little work has explored how mental illness influences this processing [1]. Axis I Disorders refer to a class of disorders that are considered mental health issues, and refer to the most common mental illnesses. These include anxiety disorders, mood disorders, substance-abuse disorders, and behaviour disorders [1]. Interestingly, the abnormal processing of emotional faces in patients with Axis-I disorders has been recorded across many neuroscience and psychological studies. Axis-I Disorders are fairly prevalent in the general population, estimated as affecting 1.6% - 12.2% of people. Therefore, cognitive differences attributed to Axis-I Disorders could have significant impacts on previous, and current studies.

Major Depressive Disorder (MDD) is one of the most common mental illness diagnoses,



affecting 5% - 18% of the population. This disorder involves low mood, loss of interest in daily activities, severe fatigue, and other physiological symptoms [1]. Neurologically, MDD patients have shown changes in the frontal lobe, amygdala, hippocampus, temporal lobe, and thalamus. Furthermore, decreased levels of activity in serotonin circuits have been shown in MDD patients. These changes have been shown to significantly impact behaviour, mood, and cognition, so the application of baseline measures to neutral face stimuli is certainly important [1].

Bipolar Disorder (BD) is an Axis-I disorder that is characterised by periods of depressive and elated mood. This disorder can have significant impacts on a patient's daily life and affects approximately 4.4% of the wider population. Prefrontal cortex abnormalities, as well as a clear link between the thinning of the cortex and episodes of mania, have been observed in BD using structural MRI [1]. Decreased volume in the frontal lobe of BD patients has also been detected, a section of the brain involved with future thinking and problem-solving.

Anxiety Disorders are a collection of mental illnesses that involve overthinking, panic, avoidance behaviours, and exaggerated worry. The most common types of anxiety disorders include GAD (Generalised Anxiety Disorder), OCD (Obsessive-Compulsive Disorder), Panic Disorder, SAD (Social Anxiety Disorder), and PTSD (Post-Traumatic Stress Disorder) [1]. These disorders can vary in their pathology, severity, and symptomatology, but impaired hippocampal and prefrontal cortex functioning have been observed across the varying types of anxiety.

Schizophrenia is a complicated disorder where patients struggle to differentiate and interact with reality and fiction. Schizophrenia has hereditary influences and affects approximately 0.3% of the population [1]. The main symptoms of this disorder include deluded thinking, hallucinations, and beliefs that interfere with a patient's ability to function. One of the main differences of a schizophrenic brain is the decreased volume of temporal lobe matter [1]. This difference supposedly contributes to the auditory and visual hallucinations that patients with schizophrenia experience.

Knowing what we now know about mental illnesses, what impacts have these conditions had on neutral face stimuli?

Depression & Neutral Faces

Filkowski & Haas (2016) observed a negative bias associated with MDD and neutral face stimuli. This would suggest that decreased neural activity has been found in MDD patients, specifically in areas such as the amygdala, medial prefrontal cortex, and the dorso-lateral prefrontal cortex. Each of these regions of the brain has been associated with certain cognitive processes and roles in neuroscience. For example, the amygdala has shown significant activity in facial recognition studies, therefore, decreased activity in this area was almost expected [1]. Abnormal (decreased) activity leading to abnormal cognitive processing is a concept that thoroughly agrees with the current research regarding cognitive mechanisms. Since the amygdala is heavily associated with facial processing and emotional regulation, this would account for the changes seen in MDD patient data [1].

The medial Prefrontal Cortex (mPFC) is involved in memory recollection, specifically episodic memory. This type of memory uses the experiences of an individual to understand the context of events, memories, or information [1]. Neurotypical individuals in experiments have reported activity in the mPRC in response to both neutral faces and emotional faces, and a major difference in MDD patients was reported within this activity as well. Less activity in the mPFC of MDD patients is supposedly due to the decreased activity of serotonergic circuits in the brain that MDD patients experience [1].

Finally, the dorso-lateral prefrontal cortex (DLPFC) has also had decreased activity recorded in MDD patients upon neutral stimulation. This area of the brain is associated with executive control, meaning cognitive tasks such as attention, working memory, and planning [1]. The role of working memory is colloquially known as short-term memory, and therefore can be believed to play a role in facial recognition in addition to other cognitive tasks. Furthermore, studies have shown that successful antidepressant treatments of MDD patients increased the use of the DLPFC across a multitude of cognitive tasks.

One consistency found across a multitude of studies is the abnormal frontal lobe activity observed in MDD patients presented with neutral face stimuli. The frontal lobe is involved in many areas of cognition and cannot be separated into areas of specific function. Instead, we can infer that abnormal frontal lobe activity can explain the observed behaviours of MDD patients [1]. However, this data also showed variance of amygdala activity in MDD patients, with some studies showing increased activity but others reporting decreased activity. Amygdala activity has been altered to neurotypical levels when MDD patients have successful antidepressant treatments, which would certainly suggest the impacts of MDD are a main factor of the abnormal activity observed in the amygdala [1]. Whilst the literature may be conflicting regarding how MDD processing interacts with amygdala activity, abnormal processing is certainly present in this condition.

Bipolar & Neutral Faces

A clear negativity bias has been reported in patients with Bipolar Disorder (BD); specifically, BD patients identify neutral faces as sad or upset [1]. Previous literature discusses the abnormal activity of the amygdala, hippocampus, and prefrontal cortex (DLPFC and inferior frontal gyrus) BD patients [1]. The presence of abnormal amygdala cognition in BD patients is inconsistent regarding the mechanisms of processing. Some studies have reported decreased firing rates, but others have shown increased firing rates. However, decreased DLPFC activity is well documented in BD patients. This circuit involves executive function (such as attention, coordination, and inhibition), and a decrease in activity could account for cognitive disturbances that are experienced with BD [1]. Specifically, decreased DLPFC activity may contribute towards the planning and motivation inconsistencies that are observed in BD patients. Filkowski & Haas (2016) discuss the emotional attachment that BD patients show when presented with neutral face stimuli, even suggesting that the strong correlation of decreased DLPFC activity may potentially be used as a biomarker for individuals at risk or suspected BD [1].

Other areas of abnormal processing include the insula and anterior cingulate cortex (ACC). The ACC links the limbic system (emotional cognition) and the prefrontal cortex (practical cognition), meaning decreased ACC activity may be affecting both of these elements of healthy cognition [1]. Furthermore, the DLPFC is also involved in working memory and executive function. Again, decreased DLPFC activity may explain some of the abnormalities observed in the behaviour of BD patients and the changes that occur with processing neutral face stimuli.

A unique concept in BD is how the differing mental states (manic vs. depressed) may affect the results of all studies regarding neutral face stimuli. Currently, research is unclear on how these two varying states impact the emotional and cognitive processing of stimuli, such as neutral vs. emotional faces.

Anxiety & Neutral faces

Anxiety is a broad spectrum, with each subsection of anxiety affecting cognition and neural activity in different ways. One study has focussed on how anxiety disorders are overall affected in their processing – with the key finding that amygdala activity was increased when compared to healthy controls [1]. The amygdala is not only involved in facial processing, but is a hub for fearful and threatening stimuli processing and output. The increased activity would then suggest that patients with anxiety disorders are experiencing hyperactivity of fear centres in their brains [1]. Furthermore, studies using different stimuli found activation of the amygdala produces pharmacological anxiogenic (anxiety-inducing) effects, whilst inactivation of the amygdala produces pharmacological anxiolytic (anxiety-reducing) effects [1]. This concept can be applied to our understanding of how patients with anxiety process neutral face stimuli to understand the data we have on amygdala hyperactivity.

Image by AbsolutVision from Pixabay

Social anxiety disorder (SAD) is a very common form of anxiety and manifests in the worries and fearfulness of social situations. SAD patients had observed increased amygdala activity, believed to be the result of patients unsuccessfully searching for social cues within the neutral stimulus, resulting in increased fear and anxiety [1]. SAD patients also reported higher insula and superior temporal sulcus (STS) activity compared to healthy controls. The insula has a well-established connection with anxiety disorders and behaviours, and anxiolytic effects (benzodiazepines, antidepressants, meditation etc.) have decreased firing rates in the insula. Aside from the link between the insula and anxiety, strong correlations between emotional processing and increased neural activity have been observed in this region [1]. This would support the idea that SAD increases the emotional processing of patients in an attempt to focus on non-existent social clues in neutral faces, which are regulated by anxiogenic regions of the brain such as the amygdala and insula. The STS is a region of the brain associated with theory of mind, or the ability to understand other's emotional and mental processing [1]. An increased firing rate in this region would again suggest that SAD patients are searching for emotions and social cues in a neutral face, which is not a process observed with healthy controls.

Panic Disorder (PD) is a form of anxiety which presents as frequent, unexpected panic attacks when no threatening stimuli are present to elicit this response. PD patients have shown clear increases in amygdala activities, suggested to be due to the increased cortisol levels, fear signals, and perception of threatening stimuli. Aside from the amygdala, the anterior cingulate cortex (ACC) has reported abnormal processing in PD patients [1]. This area of the brain is involved in processes such as emotional regulation, attention, and mood regulation. Increased firing and activity in the ACC may be occurring in PD due to the hypervigilance PD patients show towards their environment.

Interestingly, another study found that healthy controls who displayed anxiety tendencies had longer periods of amygdala processing when compared to healthy controls who did not display anxiety tendencies. This would again suggest that the amygdala plays a large role in anxiety disorders and behaviours.

Overall, this data supports the idea that abnormal processing of neutral facial stimuli occurs within patients with anxiety disorders – and anxiety tendencies. The main regions of interest for abnormal processing in anxiety disorders is the limbic system which includes the amygdala, ACC, and insula.

Schizophrenia & Neutral faces

There is quite a consistent difference between healthy controls and Schizophrenic patients' processing of neutral faces, with schizophrenic patients attaching emotions to the neutral faces presented to them [1]. The association that schizophrenic patients make with emotions and neutral faces varies between studies and between patients, whether it is happy, sad, angry, or scared emotions observed — but neutral faces are usually not seen as neutral. Schizophrenics experience delusions, hallucinations, and struggle to define reality, so it is much easier for clear emotions to be seen in a face that is designed to be emotionless [1]. This is often a large issue with schizophrenia patients, as the inability to correctly identify emotions can lead to miscommunications, social issues, and amplified delusions.

Filkowski & Haas (2016) found consistent evidence that hypoactivity of the ACC may be a contributing factor to the abnormal emotional processing that schizophrenic patients experience. Decreased grey matter in the ACC has been a consistent finding in MRI studies of schizophrenic patients, with this atrophy preceding schizophrenia onset, suggesting a potential biomarker for at-risk schizophrenic patients [1].

Similarly, the DLPFC is associated with dysfunction in schizophrenic patients, accounting for some of the behaviours observed in schizophrenic patients, such as abstract concept regulation, using appropriate responses, working memory, and attention [1]. Reduced engagement with the DLPFC in these patients may explain why reduced ability of these executive functions is observed in schizophrenic patients.

There is a significant increase in amygdala activity that is observed in schizophrenic patients, as well as increased amygdala activity in emotional face stimuli when compared to healthy controls. Since the amygdala has a strong relationship with emotional processing and output, increased activity with schizophrenics may contribute strongly to the observed behaviour of hyper emotional attachment and an inability to accurately process emotionless stimuli [1]. As well as the limbic system experiencing abnormal processing, the hippocampus — a region of the brain largely dedicated to memory — has shown increased activity in schizophrenics. However, some studies have reported a reduced rate of firing in the hippocampus of schizophrenics, but Filkowski & Haas (2016) chalk this up to differences in study design and sample size. Varying schools of thought have attempted to explain why increased or decreased hippocampal activity is observed, yet neuroscientists will agree that abnormal activity in the hippocampus is not unexpected for schizophrenia patients.

24 out of 26 studies analysed by Filkowski & Haas (2016) showed significant differences between Axis-I disorders and healthy controls when shown neutral face stimuli. Specifically, these studies showed that frontolimbic and prefrontal areas (amygdala, ACC, DLPFC). Clearly, neutral faces are not perceived as such by patients with Axis-I disorders, showing that this stimulus may not be a robust baseline measure in neuroimaging studies [1]. Furthermore, some studies have shown that the choice of neutral face stimuli can alter results in healthy controls,

suggesting that this choice of a baseline measurement may not be as accurate as neuroscientists think.

One of the largest things to take into consideration with this article is the practicality of this knowledge. Many studies involved here do not disclose or discuss the medications or treatments that patients are undergoing for their diagnosed conditions. For example, a patient with MDD who is undergoing successful treatment via antidepressants and psychotherapy may have shown very different results if they did not have these interventions [1]. The effectiveness of mental health treatments is still being discovered, with many inconsistencies found within treatment options. These demographics are important to consider but are certainly easier said than done. Clearly, this paper presents evidence that suggests directional abnormal activities across axis-I disorders, as well as the observation that certain regions of the brain are affected by these illnesses in relation to neutral face stimuli.

A specific issue with this topic is the nature of mental illnesses and the unpredictability that follows. For example, a BD patient in mania could strongly differ from the same patient in a depressive episode. This idea supports how such common and affective mental disorders are overlooked in academia, even within the subjects of psychology and neuroscience [1]. This article suggests that previous, current, and future research regarding any of the topics covered may be brushing past a large demographic factor — how do axis-I disorders impact processes that are considered neutral or normal for neurotypical participants? Approximately 1/5 people live with a mental illness worldwide, and this paper suggests that academia may have been using an inaccurate baseline measurement tool across a multitude of cognition and behavioural studies.

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Milly is entering her 4th year of the Bachelor of Advanced Sciences (Honours) programme in cognitive neuroscience. As an honours student she is fascinated by translational neurology and neuroscience, specifically neurodegenerative diseases. She is the current vice-president of *Scientific*, and the president of UoA Campus Neuroscience Society.

Explained

Gender Inequality in Science Today

Auckland University Women in Science

Auckland University Women in Science explores the challenges women face in science, including representation in academia, the struggles of the tech industry, the ramifications of male-focused research, and how we can come together to confront these issues.

Women in Academia

Many universities around the world value the ideals of inclusivity and diversity in their environment. However, women remain disproportionately represented in Science, Technology, Engineering and Mathematics (STEM) fields, especially in executive and senior leadership positions [1]. This is mostly due to gender discrimination and personal obligations such as childcare and taking care of their family. This calls for a greater need for more female representation in science [1], associated diversity training and initiatives [3], and, overall, an attitudinal change [2] to overcome barriers that women face when reaching senior academic positions [3]. Historically, university institutions were systematically designed to discriminate against people of colour and women from engaging [4], whereas those of privileged nature, including being white or being male, were catered to.

Even when minority groups were allowed to conduct research and join these academic institutions, the working environment/culture had been formed to benefit men, who experience life very differently from women [3]. As described by Laureate Professor Marilyn Fleer, "a male with a wife at home default' [5].

Today, much of the conversation around achieving gender equity in academia has been dominated by the idea of raising women to the same standard as men in terms of salary, leadership positions, career progression, and abolishing workplace harassment [6]. These are all important conversations to be had, however, we need to consider other aspects such as childcare, family responsibilities, and how empowering more women in academia would impact their personal and social values (for women who choose to have these responsibilities, as not all women want to have kids and raise a family), which often goes hand in hand with personal satisfaction and wellbeing [3, 6].

In a study examining parenting engagement and academic performance, they also looked at parenting labour by gender and found that women were more likely to be the primary caregiver for their children (30.6% vs. 3.9%), which is an important baseline to establish that women in academia are disproportionately taking the lead in parenting, and hence suffering higher penalties at work in regard to feeling inadequate in fulfilling their responsibilities. This can lead to stress, imposter syndrome, and not wanting to continue progressing through their field.

In Aotearoa, there is a lack of representation of females in a variety of areas of academia, especially associate professor and HOD positions, where 64-69% were men, and professor and dean positions, where 74-81% were men over the period from 2012-2017 [7]. Additionally, this was lower for women in senior positions at crown research institutes [7]. In 2020, at the University of Auckland, women made up 31% of professors and 39.1% of associate professors [8]. Across New Zealand, women made up 38.6% of associate professors and 27.4% of professors [8]; however, there were more women in research fellow, senior tutor, professional teaching fellow and graduate teaching assistant/teaching assistant roles as a whole at the University of Auckland.

A Harvard study [9] proposed that 30% of an institution needs to be filled with minority groups across all levels of superiority to feel represented. When you look at representation, you can see that we clearly match the minimum to see representation. However, when you take a closer look at individual fields, we do not hit critical mass for some of these fields.

The University of Auckland has pledged to close this equity gap in the Taumata Teitei — Vision 2030 and Strategic Plan 2025, which outlines a goal to 'determine and craft changes to recruitment, career entry pathways and mentoring initiatives aligned to the needs of specific cohorts.' Part of this plan involves consistent monitoring of the University's equity statistics via the Equity Profile, last conducted in 2020. The Equity Profile states that in the UoA Science Faculty, women are underrepresented (under 30%) in computer science, environmental sciences, mathematics, and physics [8]. Women are also under-represented in Engineering (below 10% in senior positions and below 30% in other positions) [8] and in Medical and Health Sciences (especially Optometry and Vision Science) [8].

However, in the last ten years, we have seen dramatic positive shifts. The proportion of associate professors who are women has increased substantially from 27.7% in 2010 to 39.1% in 2020, and the proportion of professors who are women increased from 20% in 2010 to 31% in 2020 [8]. We can only hope that the University continues this trend, and keeps on making these huge improvements in gender disparity.

The Leaky Pipeline: Women in STEM Workforces

These inequities seen in academia prevail in private industries as well. Women are consistently underrepresented among industries that hire large numbers of science graduates, including technology, data science, finance, and engineering. Martin et al. [10] describe the 'leaky pipeline' that occurs when

fewer and fewer women are represented in STEM at each stage, from training to employment to promotion. As a result, there is a scarcity of women in high-level leadership positions in STEM industry fields.

Indeed, while 48% of the New Zealand STEM workplace are women, only 21% of New Zealand's tech executives are women, and only a quarter of the small to medium-sized enterprises in the tech sector report having a gender-balanced leadership team [11]. The shortage of women in these higher-paid positions contributes to the significant wage gaps in our STEM-affiliated industries: 17.3% in Information, Media and Telecommunications, 3.4% in Construction, and 31.0% in Financial and Assurance services [12]. Attention given to the representation of women should also be given to retainment and career development.

This leaky pipeline phenomenon can, in part, be attributed to the significant bias against women in STEM industries. Women are regularly perceived as less capable and less suitable for STEM careers, and this bias is not always explicit; Martin et al. cite multiple studies that suggest significant subconscious bias may be held even by those who reject gender stereotypes [10]. A study by Moss-Racusin et al. produced conclusive results that indicated women who are exposed to the reality of gender bias in STEM are less likely to identify, engage and positively associate with a career in STEM [13].

This evidence highlights the need for these biases to be addressed, as they pose a significant barrier to female success in STEM pursuits. In particular, it is significantly harder for women to succeed in start-ups in comparison to their male entrepreneur counterparts. In 2019, ventures led by women received less than 3% of the global venture capital investments [14]. Furthermore, many women who have a male co-founder report how during introductory meetings with investors, investors would assume that they were 'not founders, nor key decision-makers' [15].

These challenges are compounded by the addition of the 'maternal wall': additional workplace discrimination due to the need to take time off of work for pregnancies and motherhood. For this reason, many women in STEM feel time pressure to build a stable career before they have children. Crosby et al. go as far as to say that 'motherhood is the worst economic decision a woman can make' [16].

Bias, barriers to entrepreneurship, and the maternal wall all exacerbate the leaky pipeline phenomenon in the STEM industry. Our female science graduates who choose to forgo academia and pursue corporate careers face significant challenges all the same.

From Causes to Consequences

To tackle an issue this systemic, we must look to where it all starts — as a pervasive culture. From a very young age, girls are taught, whether implicitly or explicitly, that science and mathematics is a field for men. Brain scans have shown that males and females have equal processing ability for mathematics, and additionally, mathematical achievement seems to be similar for young boys and girls until partway through primary school, when children start becoming more aware of social gender factors [17]. There is no biological reason for any gender differences in mathematics achievement, and so the disproportionate number of men in numerical fields compared to women is almost certainly an environmental factor.

By high school, interest in these subjects among female students drops significantly. For example, in the UK in 2017, only 3% of female high school students reported a career in technology or computer science as their first choice, compared to 15% of their male counterparts [18]. Over the years, from when girls first start school until they choose what career path they would like to pursue post high school, they are being diverted away from these STEM subjects, leading to a lower number studying them at university, which drops even lower as they continue into their permanent career paths.

There is currently a significant gender imbalance in some science subjects, seen most significantly in computer science, mathematics and physics. As of 2020, at the University of Auckland, only 22.2% of computer science students were female, and 0.43% were gender diverse [19]. This was closely followed by 36.6% female and 0.56% gender diverse in physics, with 33.3% female and 0.37% gender diverse in mathematics [19].

Our own club, AUWS, was created as a response to the isolation we felt as women in STEM in our degrees. Many of the founding members were in mathematics or physics degrees, where the proportion of men was notoriously high. An executive officer recounts, 'once in first-year Physics, a male classmate told me to shut up since the men were talking.' Another executive member remembers the day she sat down in a maths lecture and realised that every single person in the room was a man.

This gender imbalance reinforces the way in which science is being taught and learnt, by which it is projected through a male lens. Thus, studies, experiments, and research are often focused on issues that are primarily relevant to men and lack the inclusiveness that could be extended to minority groups in the classroom. Seatbelts in cars are tested on crash dummies based on the average male body, leading to women having a higher risk of injury in a collision [20]. One in ten women suffer from endometriosis, but we know shockingly little about it — indeed, this applies to the entire female reproductive system. Historic ADHD and ASD research were primarily done on young white boys, and the diagnostic criteria reflect that, making it harder for people who don't fall into this category to get help [21]. Sexism in science literally makes it harder to live as a woman in this world!

It would be remiss to discuss these issues without taking an intersectional lens. Similar barriers exist racially for Māori and Pasifika students in New Zealand. Most scientific research takes a Western lens and often focuses on Caucasian people, and little to no scientific research acknowledges people who sit outside the gender binary. In New Zealand, the life expectancy of Māori women is 77, and for non-Māori women, it is 85 [22]. Scientific and medical inequity stretches far beyond the boundaries of gender, and the horrifying statistics show there is a need for all types of diversity in science.

How do we fix this?

We cannot expect female and gender non-conforming scientists to learn, grow and thrive in environments such as these. Something needs to change. It will be a long and arduous road to scientific gender equality, but by reading this far, you are already helping us spread the message. However, it is fixable! We are already starting to see gradual improvements across STEM gender equity. For example, there was a 3% increase in women studying Mathematics and Physics at UoA from 2016 to 2020 [19].

AUWS hopes to help support this change, as every bit counts, however small. It is extremely important to have places within these STEM fields where women and gender minorities can come together to combat this feeling of isolation. This is where groups such as AUWS, as well as the WEN (Women in Engineering Network) and WIHN (Women in Health Network) come into play. It has been shown that having a sense of belonging increases the retention rates of women in STEM fields [23]. We hope that one day, not only scientists, but all people will be treated equally regardless of gender or race. However, we must consistently do the mahi and actively make change. Only then will equality be possible.

Auckland University Women in Science:
Josie Greenwood, Alisha Keshaw, Angeline Xiao & Emily Caldelari-Hume

AUWS is a community with a goal to connect, uplift, and empower women & gender non-conforming individuals within the UoA science community. They were founded in 2021 and run both social and academic events throughout the year.

Explained

Clever Stem Cells Self-Assemble Into Mini-Organoids

Sarah Moir

Biology

A decade ago, Lancaster discovered the first 'organoid' upon inspection of her neural stem cells. Now, cell scientists are exploring the diverse possibilities and ethics of this technology, from models of disease and development to their creation and use.

In 2011, postdoc Madeline Lancaster discovered she accidentally cultivated spherical clumps of neuronal cells on petri dishes. These clumps were resemblant of an embryonic brain, featuring with it the cells of developing retina [1]. Predictably, over the past decade stem cell scientists have been excited by the development of these lab grown 'mini-organs', or organoids. Almost anything from intestine to kidneys, gut, liver, lungs, and even a brain, are able to (rudimentarily) be generated in miniature from stem cells, informed upon her intended research to generate neural rosettes (a signature of neural progenitors). Evidently, we have learned our cells are pretty talented at directing their own assembly into three-dimensional diverse and hierarchical cellular networks, and surprisingly at the minimal manipulation of human hands [1].

This is not so surprising, however. We all know that our sophisticated body plan develops from a single cell, the fertilised egg. Through this process, cells self-direct their organisation and development using chemical signals to 'communicate' with surrounding cells. Remarkable, considering we have a guesstimate of a few trillion cells with around 200 different cell types. However, observation and testing beyond the 14-day stage of embryo development leaves much of organ development understudied. What's useful about organoids is that scientists can study the development of organs *in vitro* to avoid the ethical and accessibility caveats associated with embryonic developmental research. I.e. We might study the full or near development of the 'embryonic brain' in a lab, although these methods present their own logistical and ethical complications to be discussed later.

Mini Brains as a Model for Neurodevelopment and Disease

Nonetheless, Trujillo (2019) and their lab partners have already established brain organoids that are in resemblance to premature baby brains. This work measured the EEG patterns of organoid brains and showed patterns of synchronised activity in short bursts similar to that of 25-39-week-old infants' post-conception. While exciting, there is emphasis on the leaps required before these organoids could be considered a 'real human brain' [2]. Missing cell types, key brain structures, and connectivity within the structure remain to be developed. Additionally, neurophysiologist Sampsa Vanhalto (who developed an infant EEG database) highlights that EEG resemblance does not necessarily equate to functional resemblance [3]. Therefore, the suitability for neural organoids as a model for neural dynamics is yet to be clearly defined; although, this group suggest the robustness of their small-scale cortical organoid model could be used to study not only neurodevelopment but also neuropsychiatric pathologies like epilepsy [2].

Mini Brains as a Model for SARS-CoV-2 Infection

Organoid technology is not limited to organogenesis and developmental research. In fact, creativity within the stem cell field yields many possibilities including personalised patient drug testing, transplant therapies, a model for disease, and biotechnologies. One relevant example is how we are using organoids to learn about the SARS-CoV-2 virus. You may have heard of the neurological effects associated with SARS-CoV-2: dizziness, confusion, or stroke. However, limited accessibility to the brains of living patients limits what we can learn about the virus and how — or if — the virus affects neuronal cells. In a pre-print, Muotri et al. (2020) used human brain organoids to determine that SARS-CoV-2 can infect and kill neural cells and cortical neurons, while also impairing synapse function [4]. Development of an infected brain model also meant the group could test drugs on the model instead of infected patients. They found an FDA approved anti-hep C drug (Sofosbuvir) RNA polymerase blocker — that could also treat coronaviruses — yielded improved neural cell survival in the model [4]. Another group led by Akiko Iwasaki also used brain organoids to determine evidence for infection but by observing their metabolic changes using single-cell RNA sequencing [5]. Using monoclonal antibodies to block the cell surface ACE2 receptor, they detected low viral concentrations in cells [5]. Imagine SARS-CoV-2 is the key that unlocks the door (ACE2), for cell entry. Now imagine the antibody as a large barricade blocking access between the key and the door. By blocking this mechanism and observing reduced infection, we can validate the hypothesis that ACE2 is important for viral entry. Ultimately, mini-brain organoids are a promising and developing model for investigating viral infection in living cells, which was previously largely impossible and only accessible post-mortem.

A Recipe for Organoids

To create an organoid with a variety of cell types organised into a 3D structure, we need to begin with stem cells; cells capable of a variety of fates that become differentiated into their respective roles and location within the organ [6]. But not all stem cells are equal. The 'steminess' of a stem cell dictates its capacity to differentiate into a variety of cell types. I.e. blood progenitor cells are limited to producing cell types associated with the blood cell lineage. Embryonic Stem

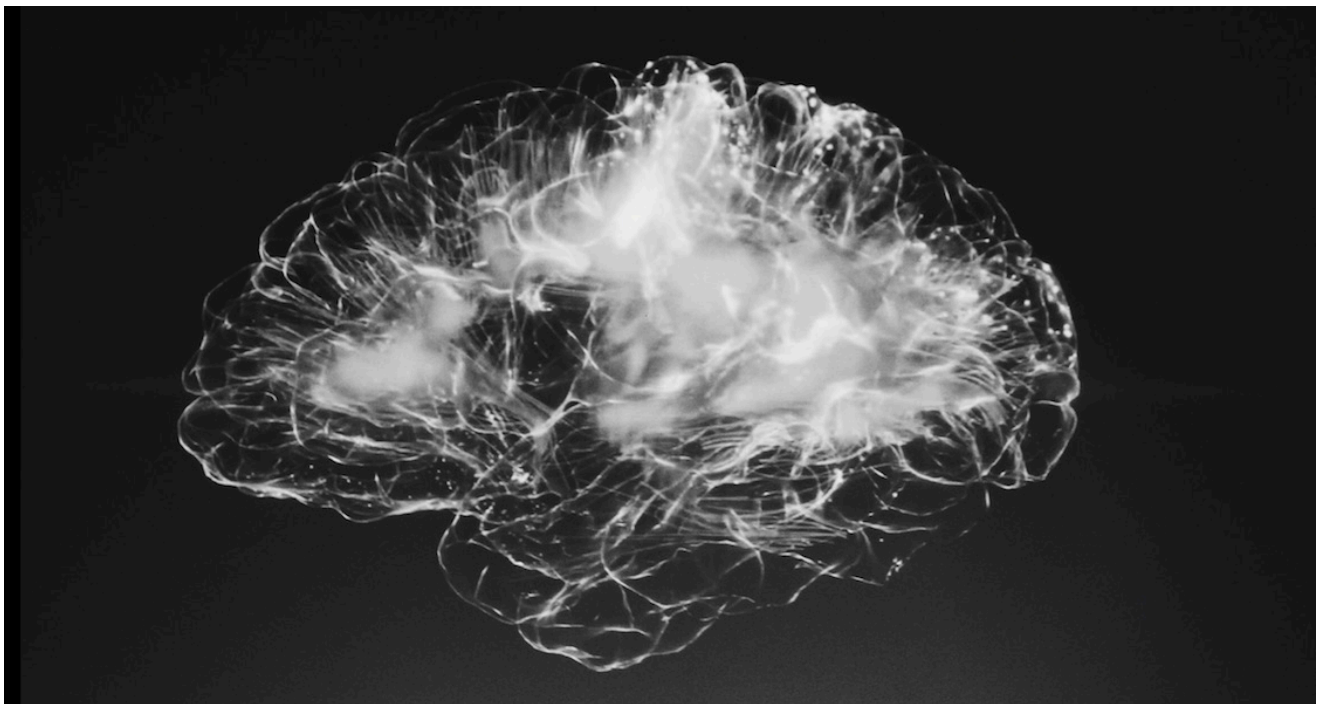

Photo by Alina Grubhyak from Unsplash

Cells (ESC) are capable of differentiating into any cell-type, thus, deemed 'pluripotent' [6]. We need pluripotent cells to produce organoids, but these can be sourced from more ethical sources than ESC. Induced pluripotent stem cells (iPSC) are a popular option, used in aforementioned research by Muotri and Iwasaki [4-5]. This method takes human somatic cells and reprogrammes them back to their undifferentiated state using Yamanaka or OKSM transcription factors [7]. Note that if cells can be sampled from a patient to generate organoids, we can create personalised cell therapies using models identical to the patient's genetic profile. iPSCs are placed within the appropriate cell medium and scaffold to guide desired differentiation pathways as the cells self-organise. Currently, cell scientists are overcoming a few major hurdles that limit organoid potential. These include the lack of homogeneity between organoid models which is inherent of their self-assembly [8]. Additionally, lack of vessel networks prevents nutrient supply and waste removal that reduces organoid lifespan and subsequent accessibility [8]. Thus, organoids lack the complete functional repertoire of their respective organ. They lack in the morphological and cell-type complexity that is a feature of multi-organ systems, rather than in isolation [8].

Just Because We Can... Should We?

Talking about growing mini brains in a dish inevitably stirs interesting and slightly unsettling debate around the ethics of such pursuits. What if this 'brain' has intelligence? Is it conscious? Does it feel emotion? We need to consider if ethics should differ between brain, heart, or liver organoids, and what it all means within the context of 'personhood', rights, and ownership of the organ [9]. Bioethicist Sarah Chan highlights brain organoids are far behind this level of cognition, emphasising this isn't something we need to worry about but rather issues of consent, ownership, and cell sources are more pressing [10]. Chan suggests the multitude of organoid research benefits far outweigh the risks, at least in terms of where the technology currently resides, but as technology develops risks should be re-assessed.

The Future of Organoids

Lancaster's surprising observations a decade ago sparked a frenzy in the stem cell world to explore the possibilities of organoid technology [1]. From the study of neurodevelopment and neurodegenerative disease to testing the consequences of viral infection or therapeutics, organoids grant access to the previously difficult-to-study organs of the human body. Exciting yet somewhat unsettling, we are all captivated to see where this technology leads us.

Sarah Moir - BSc, Biological Sciences
Sarah has just finished her third year of study at UoA as a Biology major. She is staying with *Scientific* as the head editor this year before she returns for post-grad, and is currently underway on her summer research project investigating umbilical stem cells.

Research

High-speed Videography of Water Droplets Impacting Polydimethylsiloxane Microarrays Reveals Interesting Behaviour at Small Scales

Aimee Lew

Physics

The wetting of microstructured flat surfaces is affected both by the properties of water and the properties of the surface, namely the arrangement, shape, height and surface area of the micropillars. Microstructured surfaces as models are studied extensively to understand how topographical or chemical heterogeneities influence wetting phenomena. Such phenomena include super-hydrophobicity, 'fakir' droplets, electrowetting, and more. The same apparatus in the Dynamic Microfluidics laboratory can also be used to study the wetting of surfaces other than polydimethylsiloxane, such as metals, leaves, crystals and other polymers.

Introduction

Water, in scales large and small, in speeds high and low, remains an active focus of physical inquiries. Dynamic microfluidics, the study of water at small scales and high speeds, sheds important insights on the interaction between fluids and surfaces at the microscopic level. The University of Auckland's Dynamic Microfluidics laboratory conducts various research projects on water behaving with plastic, crystalline, metallic and organic surfaces, as well as milk-drying experiments in air. In 2022 I experimented with water droplets on samples of polydimethylsiloxane (PDMS). Exactly how water wets different surfaces, and thus how a surface becomes hydrophobic or hydrophilic, has important implications for materials sciences, chemical engineering, and food sciences.

Background

It is entirely possible to put a drop of water on both a hydrophobic (water-repelling) and hydrophilic (water-attracting) surface and have it stay there. How then, is hydrophobicity and its counterpart quantified? In fluid dynamics, wetting and wetness is defined through the contact angle that water makes with a surface. The contact angle is measured from the flat surface along the tangent of a water droplet at the point of contact. A surface with a contact angle less than 90° is hydrophilic. A surface with a contact angle greater than 90° is hydrophobic.

There are two ways a surface's hydrophobicity can be altered: chemical or structural. Frequently these two techniques are used in tandem with each other to design surfaces with manifold wetting properties, fit for manifold purposes. Chemical hydrophobicity comes from using substances like oils, waxes, acrylics, and other polymers that repel water by the very nature of their chemical makeup. Structural hydrophobicity comes from patterning and texturing a substance so that the equilibrium contact angle (the contact angle when water sits on a flat surface with homogeneous surface chemistry), and thus the wetting behaviour, changes. Whether water droplets spread or not on rough surfaces can be reduced to energy considerations, namely from surface tension (surface area of the droplet) and gravity (mass of the droplet). Roughening a surface, all other things equal, increases

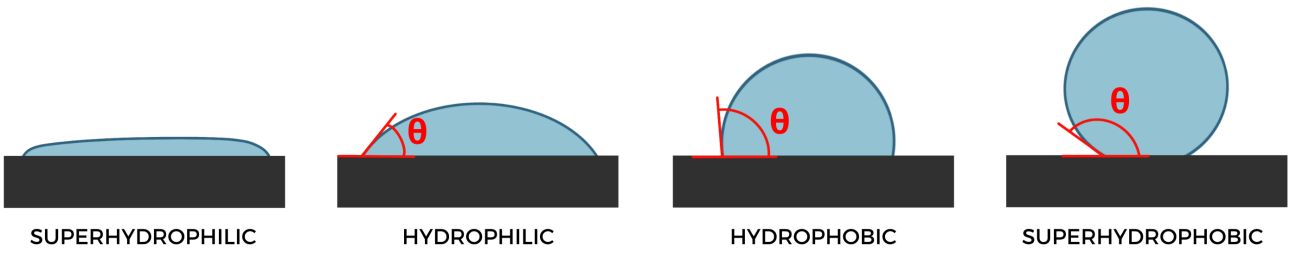

Figure 1: Diagram of water droplets on four different surfaces, with their contact angles marked. Proceeding from left to right, the surfaces are superhydrophilic, hydrophilic, hydrophobic, and superhydrophobic. Artwork by the author.

the surface area of the material by introducing hills and valleys, divots and folds. If a flat surface was originally hydrophilic, then as the surface area increases, water spreads even more. If a flat surface was originally hydrophobic, then an increased surface area makes spreading/wetting less energetically favourable. Subsequently, roughness intensifies the original behaviour of a material. Hydrophobic surfaces become more hydrophobic, and vice versa.

Methods

In my experiments, water droplets are dropped onto slides of polydimethylsiloxane (PDMS). The independent variables are the microstructure of the array and the height of droplet release. The dependent variable is the wetting behaviour, with droplet volumes and release heights controlled electronically. The water droplets are dispensed onto the PDMS sample by an automatic syringe pump controlled by a computer programme. The volume of each droplet was kept constant at five microlitres. The syringe mount height is adjustable by raising or lowering, by computer, its vertical stage. High-speed videography records the behaviour of the water immediately after impact.

A lateral camera (foreground) records a side view of a falling droplet. A second camera (right) focuses on a mirror which directs overhead light, yielding a bottom view of a falling droplet. A

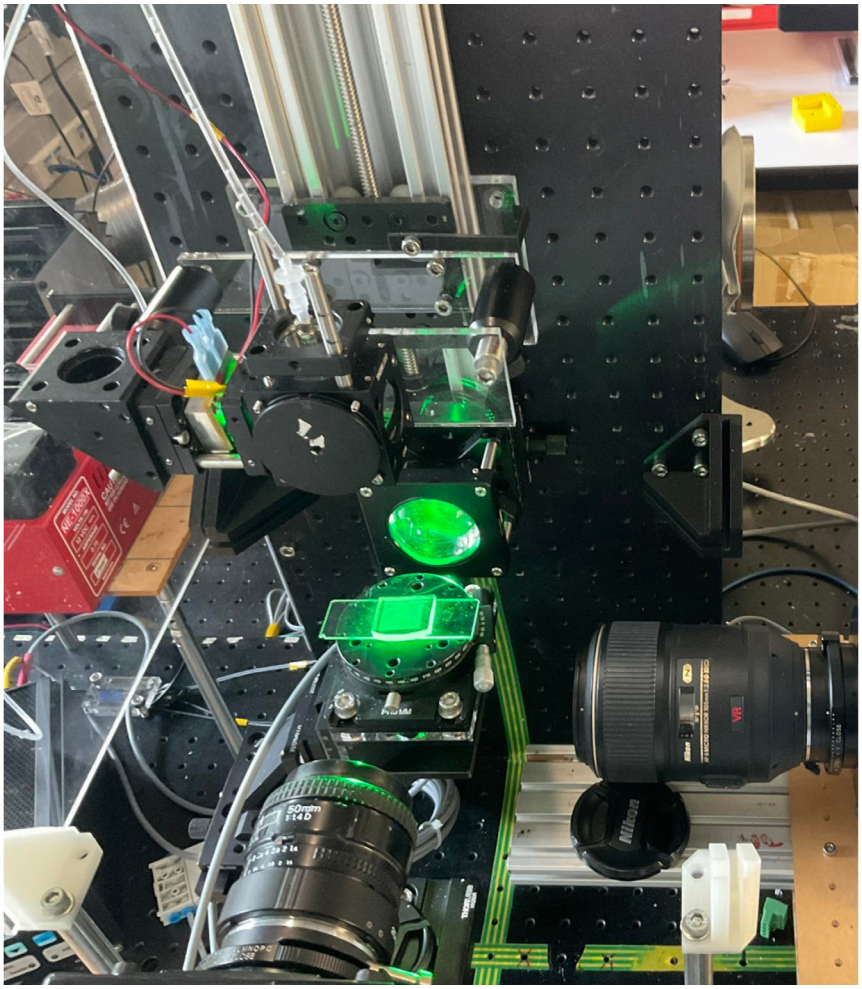

Figure 2: Experimental apparatus. Photography by the author.

green laser light backlights both cameras to increase the contrast between a light background and a dark water droplet. Photron FastCam Viewer is the software used to capture and process the high-speed footage. Trials were conducted at low droplet heights (less than 20 cm above the PDMS sample) to limit their acceleration under gravity. At slower droplet speeds, the effects of splashing and motion blur are mitigated.

The 'rough' surfaces used are PDMS micropillar arrays. Micropillar arrays are surfaces with posts in a regular grid, made using lithography. The dimension of the grid is determined by properties such as the arrangement (the length and width between each post; the array can be square, rectangular, even triangular); the shape of the posts (the top of the posts can be circular, square, hexagonal); the height or 'pitch' or the posts; and the surface area Φ of the posts. Microstructured surfaces such as these are used to model different combinations of

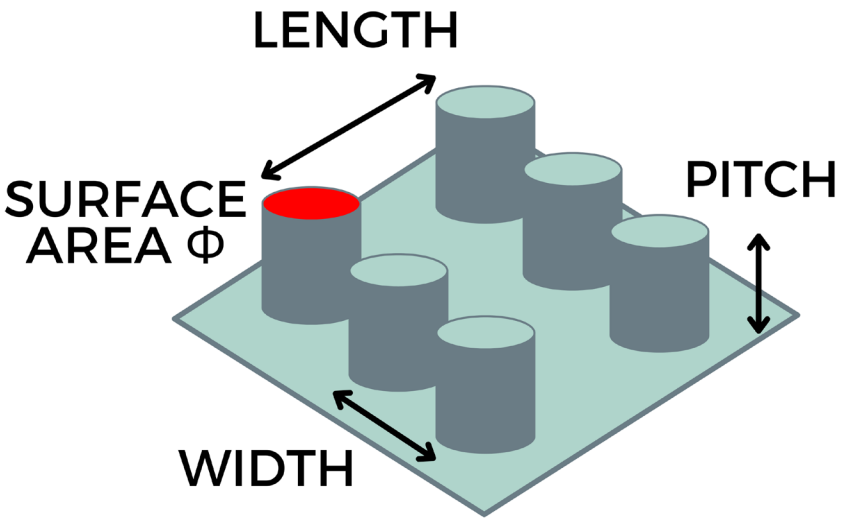

Figure 3: Schematic of rectangular array with circular micropillars. Artwork by the author.

surface chemistry and topography. In experiments they reveal numerous wetting phenomena on heterogeneous surfaces, like super-hydrophobicity [1], 'fakir' droplets [2], and electrowetting [3].

Two samples of PDMS were used. The shape of the pillar cross-section in this experiment is square. Both samples have 21.2μm pitch (pillar height) and surface area Φ = 20μm. They are both microarrays with pillars arranged in a rectangular grid. The M2A sample is a rectangular 40μm x 60μm array. The M2B sample is a rectangular 60μm x 80μm array. Samples were cleaned with water, dried with nitrogen gas, and stored in sealed containers.

Results

The images displayed were obtained with the M2B sample, which best demonstrated the wetting phenomena. Increasing the height from which the water droplets are released increases the droplet's inertia to surface tension ratio (also called the Weber number). The spread factor, a ratio of the droplet's original diameter to its diameter at the maximum spread, increases with increasing height. At the origin of the impact, a microbubble of air forms as incoming water compresses and traps the ambient gas. Fringing this origin point is an 'impact region' — appearing as darker, dense spots — which also traps air between the pillars of the microarray, forming a partially-wetting area that often takes on geometric shapes [4].

Another feature observed is the formation of fingers from droplets released at larger heights, and thus travelling faster upon impact, along one axis of the rectangular array. See Figure 3(m). This follows from the fact that in a rectangular array, pillars are clustered more closely in one direction than the other. Water travelling perpendicular to the axis with more tightly-spaced pillars faces more resistance, and while water travelling along this axis can extend into lobes and fingers at the edge of the droplet. Another interesting observation were triangular wetted regions at the border of the droplets at intermediate release heights. See Figure 3(i), where triangular shapes appear at the top and bottom of the droplet's profile. These regions also only appear along the axis that the fingers form, which indicates they could be a result of the same fluid velocity and fluid pressure differences.

Conclusion

In the microscopic world of these arrays, numerous combinations and designs are possible, each presenting a new line of inquiry for researchers. Adjusting the properties of the grid, pillars, or material can bring about new surfaces with new wetting behaviour. The wetting of microarrays and its applications are widespread and ever-developing. It builds better understanding and control over how water interacts with different materials at the microscopic and nanoscopic level. Applications are interdisciplinary, spanning biomedical engineering, materials science, nanotechnology, and more. For example, self-cleaning surfaces utilise hydrophobic materials, while probes are deposited on DNA chips through miniscule and precisely-placed hydrophilic spots [5]. While wetting in different regimes continues to be explored, turn a new eye to raindrops on the car windshield, or shower spray on the wall. There's a lot of potential in things so tiny. Thanks to Geoff Willmott for his supervision and guidance.

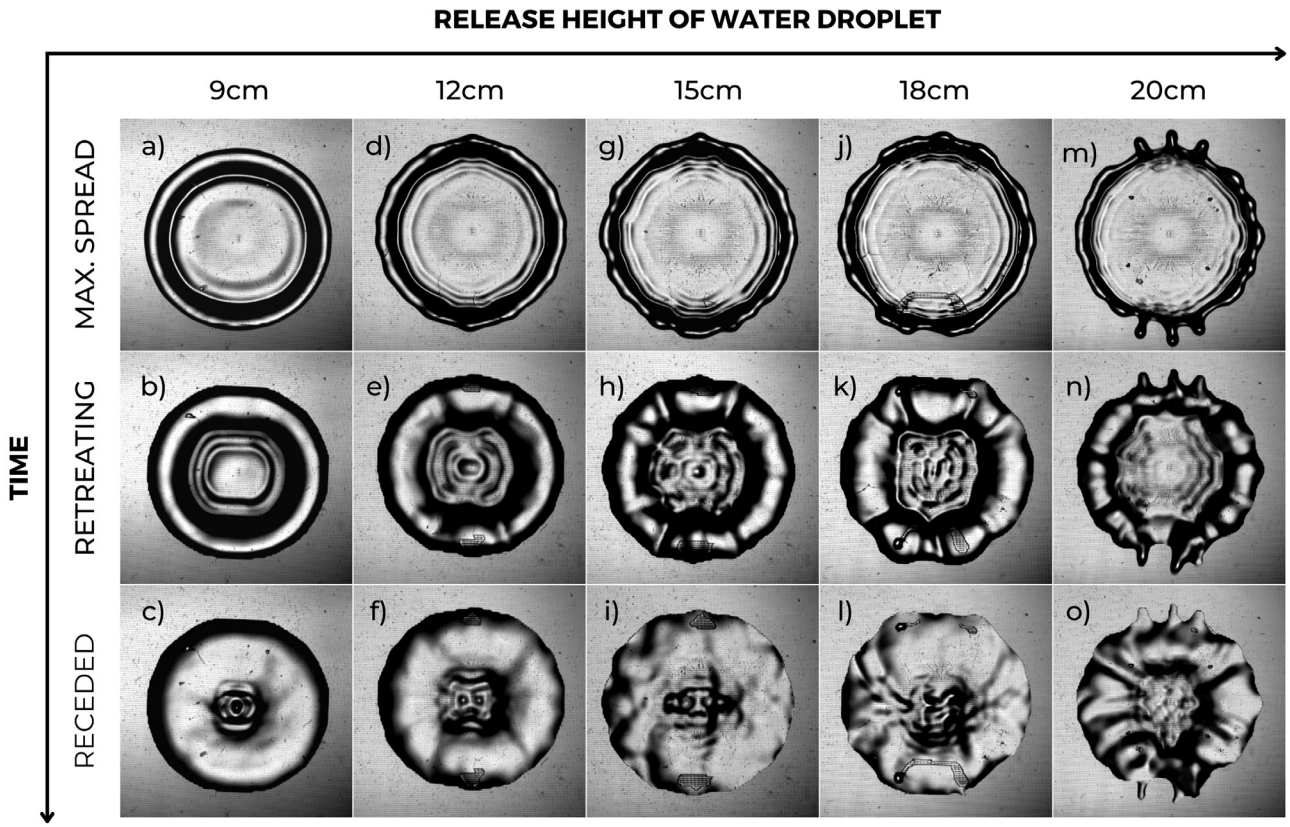

Figure 4: Radial images of water droplet impacts. Down the vertical axis, images advance through time. Across the horizontal axis, the release height of the droplet increases. Image by the author.

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Clever Stem Cells Self-Assemble Into Mini-Organoids

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High-speed Videography of Water Droplets impacting Polydimethylsiloxane Microarrays Reveals Interesting Behaviour at Small Scales

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Fun Fact

Bananas are (lightly) radioactive as potassium atoms undergo decay.

Closing Comments

That's a wrap for our Summer School edition 2023. Thank you so much for supporting UoA Scientific, we are excited to showcase the excellent scientific writing that comes out of our University.

We are appreciative of our new members for starting the year off strong and getting stuck into working on this issue. We look forward to seeing the amazing ways that you contribute to our publication as the year continues.

A big thank you to our writers for their hard work! Special thanks to Auckland University Women in Science, it was a pleasure to collaborate with your team. We are looking forward to working with all of you again soon.

We have a lot in store for 2023, with a fresh new website thanks to our Marketing & Creative Design team, as well as some awesome publication cycles throughout the year. We hope to engage more with the wider science community at the University, so make sure to connect with us on our socials for updates.

We hope you've enjoyed our Summer School 2023 edition, and we can't wait to see you again in Semester 1 for Volume 3...maybe as one of our guest writers?

Until then, Ka kite anō au i a koutou

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I'm a magazine, but I have feelings too. Don't throw me away!