Kinesis
magazine

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THE SILENT WITNESS

Can DNA evidence actually lead us to the truth?

INSIDE:

The Mojito Muse
Doctor-Induced Addiction
Eating to Change our Brains
Organoids: Small in Size, Big in Possibilities

LETTER FROM THE COMMITTEE

We're so excited that the fourth issue of Kinesis Magazine is finally ready and out on campus! We hope that you enjoy learning more about the variety of subjects included in this issue. There's something for everyone, with articles investigating free will, non-coding DNA and whether alcohol actually does make you more creative.

A lot of work has gone into the production of this issue, with invaluable contributions from writers, editors, artists, and members of our committee. We are so grateful for these students, from all years and many different departments, who have helped to create the magazine. Without these individuals, the quality of the articles and the accompanying artwork would not have been achievable.

If you're interested in getting involved with the magazine, we're always open to new members whether you have experience in science journalism or not. Get in touch with us or come and meet us at one of our events throughout the year and you could be published in the next issue!

In the meantime, enjoy Issue Four and see you soon!

UCL Kinesis Magazine Committee

ABOUT KINESIS MAGAZINE

Kinesis Magazine became a UCL society in 2017, having been started by a group of students who couldn't believe that a university like UCL didn't have a science magazine. The name stems from the word for "movement or motion in response to an external stimulus", a definition we keep central to the ethos of the magazine. It reflects our endeavour to report and analyse news from the ever-changing world of science, and our drive to make science more approachable to wider audiences.

Since its inception, Kinesis Magazine has only published *original articles accompanied by original artwork*, both in print copies and online. The scientific writing and unique illustrations are aspects of the magazine that we pride ourselves on, and will continue to do so for years to come.

Find out more about us on our website and social media pages.

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KINESIS MAGAZINE

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The Biology of Ageing

Written by **Bethany Evans**

Why are we programmed to grow old and die?

"Eternal Youth" is an idea that has captured our imaginations for centuries, and is evidently deeply embedded in legends and mythologies. In Norse mythology, the character Iðunn provides Gods with apples that grant them eternal youth. In the story of Tithonus, a character asks the Gods for immortality, but makes the foolish mistake of forgetting to ask for eternal youth too. While our life spans have certainly increased since the time these mythologies were composed, we still grow old. And we still die. But why?

Contrary to the idea that we humans were "designed" in a way that makes death inevitable, and that it is "programmed" in our genetics to grow old and die, it is actually widely accepted by scientists today that ageing is not a genetically-programmed process; it is not like the developmental processes that cause growth and maturation of organisms. Instead, ageing is the result of failure - failure of the molecular mechanisms that maintain cell and organ homeostasis, until eventually, we grow "old" and we die.

So, what are the mechanics of ageing? That, I'm afraid, scientists have not yet reached a consensus on. However, there are several leading theories.

One such theory argues that following the reach of sexual maturity, we gradually accumulate mutations that result in our own progressive decline. In other words, we gradually accumulate mutations that result in ourselves "ageing".

Antagonistic Pleiotropy Theory, conceived by George Williams in 1957, takes this concept even further. According to Williams, mutations favoured early on in life that promote positive effects (like reproductive success) and are selected for have harmful effects later in life. In other words, some genes "enhance" our fitness when we are young, but then reduce it as we get older. Ageing, then, becomes the inevitable side effect of a successful youth. In 1984, scientist Michael Rose confirmed this theory as observed in the vinegar fly, *Drosophila melanogaster*. He demonstrated a close link between early fertility and lifespan, where a selection for genes that translated to early fertility resulted in shorter lifespans, and a selection for genes that translated to late fertility resulted in longer lifespans.

Alternative theories argue that ageing is more of a failure in mechanisms, rather than an evolutionary failure. Oxidative Damage Theory is one such theory. This theory was the first of its kind to suggest that the molecular mechanism of ageing involves the oxidative damage of vital biological macromolecules (such as DNA) by reactive oxygen species. Mitochondrial Free Radical Theory furthered this notion, identifying the reactive oxygen species produced via metabolic processes in the mitochondria as the main species that causes this oxidising damage. The more "damaged" our biological molecules get, the more we progressively decline in terms of fitness, until we eventually die.

Telomeres (repetitive DNA sequences at the end of chromosomes) are of scientific interest too, in the pursuit of underpinning the mechanisms of ageing. Telomeres at the end of your chromosomes protect your DNA from accumulating damage. However, each time a cell divides, pieces of DNA are lost at the end of the chromosomes, in a condition known as "telomere attrition". Hence, chromosomes are no longer protected by these telomeres, generating genomic instability. DNA can now accumulate damage, leading to cellular ageing and eventual cell death.

> "Ageing is not a genetically-programmed process."

But all hope is not yet lost! Telomerase is an enzyme that synthesises telomeric sequences at chromosome ends, using RNA as a template. Extending telomere length in this way has been shown to expand lifespan. Studies in gene-targeted mice have shown that reactivation of telomerase decelerates ageing and improves health span. Maybe this is the beginning of bottled "Eternal Youth"?

Whatever theory prevails, it is clear that this is a "hot topic" in science, with our understanding of the mechanisms of ageing growing every passing day. Perhaps even one day, we might be able to grasp this "Eternal Youth" as described by those ancient mythologies. If biologically immortal creatures such as starfish can do it, why can't we?

THE FANTASTIC CASE OF Neuroscience and Free Will

Written by **Marta Caldeira**
Art by **Wenanlan Jin**

When neuroscience meets philosophy, it's at the local cafe...

It's a typical Monday morning and you are standing in line at your local cafe waiting to get your favourite breakfast pastry. When your turn finally arrives, the barista tells you they have run out. You are suddenly faced with the task of choosing between one of two alternatives: a bagel or a cake. What will you choose? I bet you have already thought of an answer. After all, this task is quite an easy one to tackle. Or so it seems…

Have you ever wondered what goes on inside your brain while you are trying to choose between two options? How do the billions of neurons in your brain behave? Is the choice truly *yours*, or is it a product of neuronal activity beyond your control? Thus, choosing between a bagel and a cake is more complex than it seems.

The progress in neuroscience research over the last decade has produced numerous explanations on how neurons generate the processes we refer to as thought, consciousness, and will. The renowned neurologist Oliver Sacks, amongst others, puts forward their views on the implications of these processes to the problem of "free will". This questions how much we control our actions.

The controversial experiments of Benjamin Libet in the 1980s have shaped the investigation into free will. Libet sought to determine the moment we first become consciously aware of our decision to perform an action. He built a special clock consisting of a dot moving around an oscilloscope screen. He then asked volunteers to spontaneously move their fingers and remember the position of the dot when they first became aware of their decision to move. The brain activity of volunteers was simultaneously monitored. Results showed that volunteers became aware of their decision to act prior to moving their finger, but unconscious activity in the brain preceded awareness.

Many philosophers have invoked Libet's experiments as definitive proof that free will does not exist. Jerry Coyne argues that these experiments show that our decisions are made by our brains even before we are aware of them, and thus free will is an illusion. How can it be real if our decision to choose a bagel over a cake is made even before we become aware of it? Sam Harris says these experiments demonstrate that the intention to act is not a product of consciousness - it is a part of consciousness. If our intention to choose one option over another merely pops up in our brain, then our choices are never truly free.

"Is the choice truly *yours*, or is it a product of neuronal activity beyond your control?"

"Results showed that volunteers became aware of their decision to act prior to moving their finger, but unconscious activity in the brain preceded awareness."

In contrast, several scholars argue that this conclusion does not justify the evidence. Daniel Dennett states that Libet's measurements were highly subjective, and that the timing of decisions could not be accurately measured. In fact, Libet himself has questioned the implications of his experiments - he concludes that free will exists. According to him, free will rests not on the ability to initiate a voluntary act, but on the ability to veto an unconscious impulse to act. Indeed, volunteers in his experiments often reported consciously suppressing the urge to move.

So, does this make us free?

It is possible for the same experiments to support contrasting views of free will. Despite having uncovered much about neuronal function and organisation in the brain, neuroscience has yet to conclude how these allow for processes such as thought, consciousness, and will. Most of the discussion on the freedom of our actions is a reflection of metaphysical biases of authors as opposed to any concrete experimental results. Indeed, whilst Coyne and Harris are sceptical of free will, Dennett and Libet defend it. Thus, we must await more conclusive evidence before neuroscience can tell whether we exert control over our actions. Until then, we are "free" to interpret neuroscientific findings as we wish.

An insight into PSYCHEDELIC PSYCHOTHERAPY

Written by **Jenny Seok**
Art by **Winnie Lei**

Psychedelics: between abuse and cure.

What are "psychedelics"? This term was coined in the early 1950s by Humphrey Osmond, an English psychiatrist who attempted to use LSD for the first time as a psychotherapeutic agent for alcoholism. The word has a Greek origin, with psyche meaning the human mind or spirit, and dēlos, to manifest: psychedelics were thought of as a "mind-manifesting" class of drugs that could facilitate a heightened sense of consciousness. Psychedelics now comprise a wide range of substances with different pharmacological profiles; the most popular include psilocybin, LSD, and DMT.

The therapeutic potential of psychedelics for psychotherapy was implemented by Swiss chemist Albert Hoffman who first synthesised LSD in 1938 before mediating its use in clinics and universities across the world. Between 1950 and 1965, over 40,000 patients were prescribed LSD therapy for the treatment of neurosis, schizophrenia, psychopathy, and even child autism, with the publication of over 1000 scientific papers. Despite the lack of robust experimental and clinical techniques, people began to acknowledge the potential pharmacological benefits of psychedelics.

Nevertheless, the therapeutic aspect of psychedelics soon became neglected as they were gradually abused under unsupervised and non-medical contexts, especially during the period of hippie counterculture in the 1960s where LSD was popularised in the form of soaked sugar cubes, spreading from America to the UK and the rest of Europe.

Consequently, the US federal government outlawed LSD in 1968, and similarly the UK's Misuse of Drugs Act 1971 classified many psychedelics as Class A drugs, leading to the most serious punishments and fines. As a result, psychedelic research abruptly came to an end.

However, the paradigm of psychedelic psychotherapy has recently re-emerged as the government slowly began to change its attitude towards human-based psychedelic research over the past decade. Studies have now uncovered that the basic cerebral mechanisms underlying the effects of psychedelics strongly correlate with the characteristic psychological effects.

Controlled clinical studies have been carried out to evaluate the pharmacological effects of these drugs, using advanced techniques such as blood oxygen level dependent (BOLC) measures and magnetoencephalography (MEG) to better follow the consequences of psychedelic administration. As opposed to the rather unstructured psychotherapies conducted in the 1950s and 1960s, all studies now acknowledge three critical elements for enhancing positive and healing experiences: the set, which corresponds to the psychological expectations of the patient; the setting, referring to the physical environment; and the clinician-patient relationship.

> "Music in psychedelic psychotherapy sessions increased their therapeutic efficiency."

Three main cerebral consequences of psychotherapy have been identified. First of all, increased cerebral blood flow was observed with a decrease of visual cortex alpha power (alpha waves are often signs of a relaxed mental state). Furthermore, the functional connectivity profile of the primary visual cortex was found to expand after administration. These are all elements strongly correlated with visual hallucinations, meaning that the psychedelic state enhances the visual processing of the brain.

Moreover, the connectivity between the parahippocampus (region of personal memory) and the retrosplenial cortex decreased, suggesting that the hierarchy of neural networks was temporarily dissolved, allowing information to travel more freely between central junctions. This malleable state of the brain meant that it was more susceptible to external stimuli, leading to a heightened sensitivity for the environment greatly associated with feelings of "ego-dissolution" and "altered meaning".

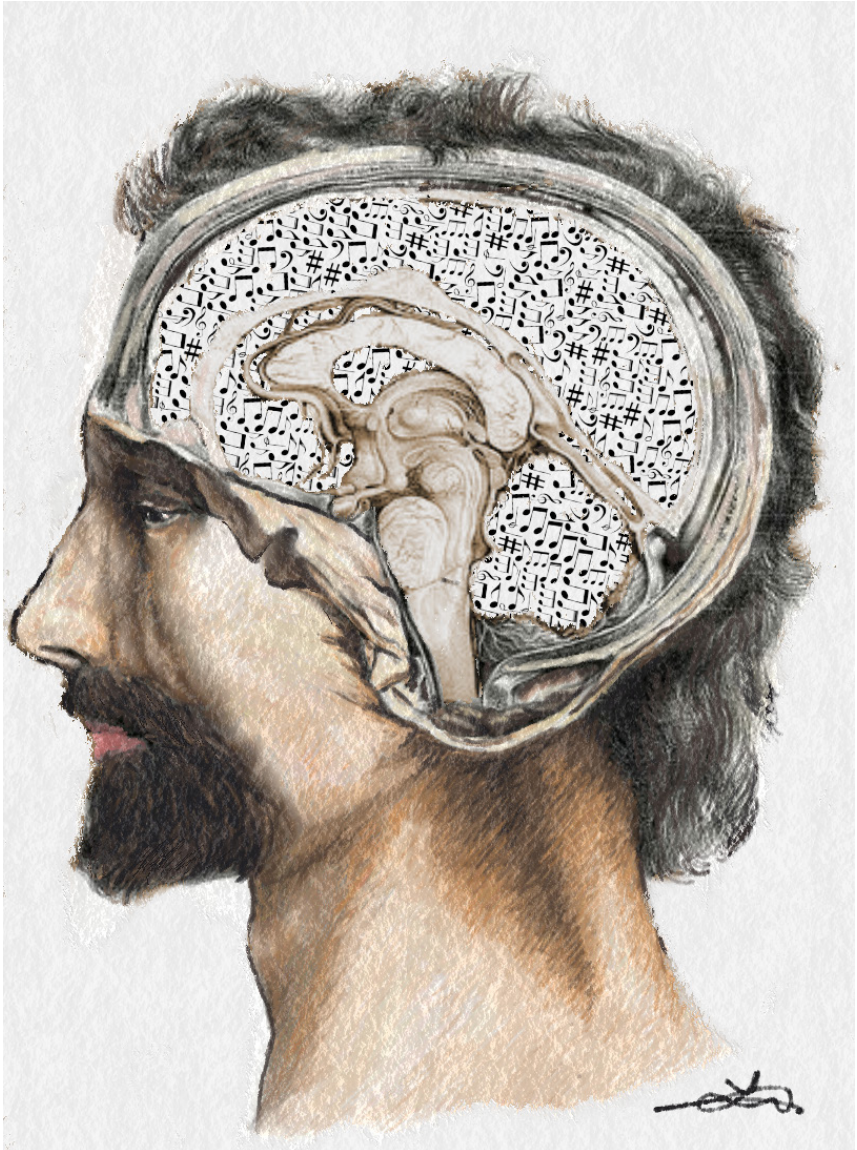
Researchers also noticed in the 1960s that introducing music in psychedelic psychotherapy sessions increased their therapeutic efficiency. Patients reported that music rendered more personal and autobiographical mental images, making their emotions more therapeutically meaningful. Different phases in psychedelic therapy sessions were identified with a distinct set of psychological outcomes music could bring: "pre-onset", "onset", "building towards peak", "peak", "entry", and "return". However, the nature of this effect remains unknown.

In a recent study, it has been found that the interaction between music and LSD increased the exchange of information between the parahippocampus and the visual cortex, and these greater interactions were shown to reinforce the autobiographical and inclusive nature of mental images.

An important feature of the music played was timbre, which causes instruments and voices to sound different from each other when producing the same note. The magnitude of timbre appeared to increase the activity of the regions of the brain dedicated to process emotion and attribute meaning to sound, such as the Broca area, causing music-evoked feelings of "wonder" and "transcendence".

With both sensations being characteristic of "peak" experiences, these findings seem to support the idea that music brings the more vivid and meaningful effects into psychedelics in therapy.

However, more research needs to be carried out in order to fully validate this hypothesis. The study earlier reported that although some patients manifested these "peak" experiences, musical styles were not appreciated by 58% of the participants to various extents. This shows one of the biggest challenges in psychedelic research: despite having the scientific groundwork explaining the positive effects of psychedelics in psychotherapy, the subjective nature of these experiments means that more investigation needs to be done before a real implementation of psychedelic usage in clinics comes into play.



ORGANOIDS: SMALL IN SIZE, BIG IN POSSIBILITIES

Written by **Thomas Drobkiewicz**
Art by **Charlotte Capitanchik**

How to test drugs and treat people better, faster, and for less money.

What if I told you that we can put drugs through clinical trials faster and for less money, that we can screen drug-to-drug interactions and its threats, and that we can actually prepare personalised cancer therapies? Sounds like science fiction, right?

Well, there is a new technology that can be used in drug discovery, drug toxicity, and personalised medicine – organoids. This idea was proposed for the first time in 2008 by Dutch professor Hans Clevers. Organoids are 3D tissue cultures that can be as small as the head of a needle. You can think of them as small organs because they consist of all cell types and mimic the function of their organ equivalent. They are produced from human-induced pluripotent stem cells (HiPSC) and since their discovery, scientists have successfully created organoids of all major organs.

Okay, but you may still be wondering, what is all this fuss about? To begin with, the use of organoids can decrease the amount of time needed to develop new drugs. As an example, organoids are being used at Harvard University in the laboratory of Dr. Lee Rubin to develop medications for neuronal disorders. In his research, Dr. Rubin needs billions and billions of neurons. Using organoid technology, he can grow them in one flask in approximately 50 days, something he can never do by using 2D cell culture on 100 plates. Moreover, with saved time comes saved money, and because of that, this technique draws a lot of attention.

Secondly, let's talk about drug toxicity. You may think every drug in the pharmacy is safe. Unfortunately, some of them, in spite of passing all tests during clinical trials, come out to be toxic to humans. Some examples include Valdecoxib (a non-steroidal anti-inflammatory drug), Pemoline (used to treat ADHD), and Rapacuronium (used in anaesthesia). Organoids enable better toxicity testing on human tissues and can help to detect unexpected and long-term complications that are hard to detect in human clinical trials.

> "Patient-derived organoids would give doctors the best information on what works best."

Moreover, in his 2015 TEDmed talk, Dr. Russ Altman was talking about how difficult it is to analyse drug-to-drug interactions. He and his student were looking for adverse effects of patients who took both Pravachol (an anti-cholesterol drug) and Paxil (an antidepressant) that wouldn't occur while taking these drugs separately. What was surprising is that patients on both medications had higher blood glucose levels of about 20mg/mL. This difference is significant and can lead to diabetes. Thus, the interactions between drugs are very important, but remain unpopular because they would cost too much, and it does not affect many people. The point of Dr. Altman's talk wasn't to introduce the idea of organoids. He has simply shown a problem. Now think about it – we could use organoids to test drug interactions to resolve this problem, and patients would be given better treatment that doesn't endanger them.

Organoids can also be used while deciding on the best therapy for the patient. I am not talking about mild diseases like flu here. I am talking about probably the most controversial of them - cancer.

Every tumor is different and yes, we can grow its organoids too. These can be used to test what therapeutics would work best, and depending on the place, stage, and genetic mutation of cancer, different approaches should be considered by oncologists. Yes, we could use mice models to do this. However, they lack a human's immune response and don't reflect the real disease in the best way. Patient-derived organoids would give doctors the best information on what works best, possibly lowering the cost and shortening the time of therapy.

Let's go back to the beginning. What if I told you, that we can put drugs through clinical trials faster and for less money, that we can screen drug-to-drug interactions and its threats, and that we can actually prepare personalised cancer therapies? Sounds like science fiction? Not anymore, not with organoid technology.

Interested in being part of the next issue of Kinesis Magazine?

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THE LETHAL NEW FACE OF NON-CODING DNA

Written by **Zohar Mendzelevski-Steinberg**
Art by **Bella Peng**

What explains a patient's genetic disease when no genetic cause is known?

Only 20% of families with inherited early-onset breast cancer have a mutated version of BRCA1/2, the tumour suppressor genes best known for their involvement in breast cancer when silenced. Researchers have not been able to identify the cause of the other 80% of families' heritable cancer until now – because they were looking in the wrong place.

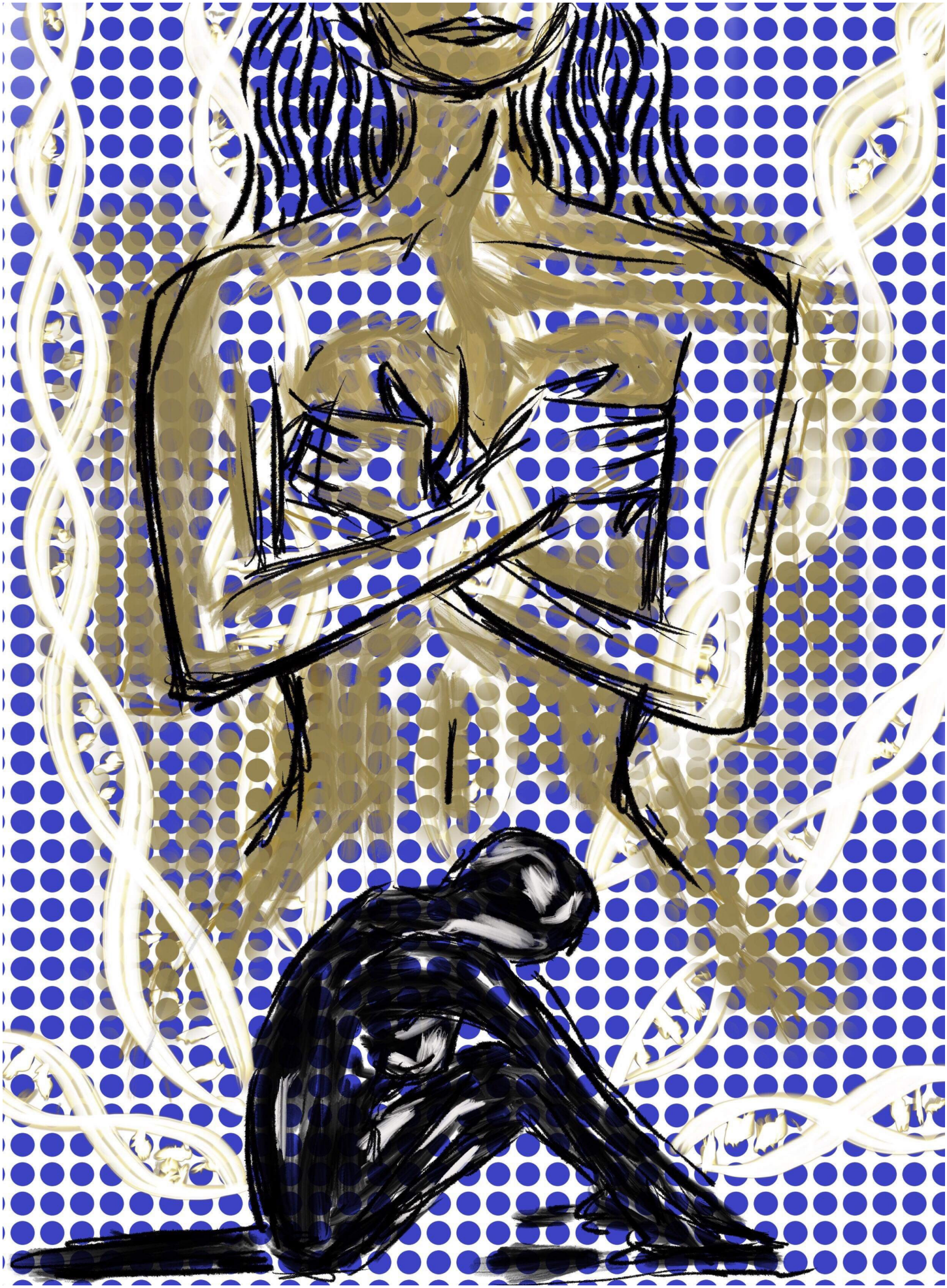
Recent work by the Newman group at the University of Manchester has shown that BRCA1 promoters in the non-coding region of DNA are epigenetically silenced, leading to the effective silencing of BRCA1. They aren't the only ones: across the globe, labs are discovering that in a range of diseases for which no genetic basis was previously known, epigenetic and genetic mutations to regulatory regions in the non-coding DNA are the cause. This ranges from cancer, to neurodevelopmental disorders, and to rare congenital diseases. In patients who present with only one risk allele for a recessive disorder, researchers are discovering non-coding DNA mutations which seriously reduce the expression of the remaining healthy allele. So, what do we know so far?

In the case of BRCA1, it appears that the promoters upstream of the BRCA1 gene are being hypermethylated, a heritable epigenetic modification which silences the promoter. This prevents transcription of the functional BRCA1 protein, a DNA repair protein which suppresses tumour formation, leaving only the dysfunctional allele to act in all cells of a patient's body. In the case of muscle disorders, researchers found that mutations in introns (the non-coding DNA interspersed within genes, spliced out of the mature mRNA that codes for proteins) can disrupt normal splicing, producing malformed proteins. In severe collagen VI-related dystrophy, these introns promote their own inclusion in the final protein, altering the structure of specific collagen chains.

The rare syndrome thrombocytopenia with absent radii (TAR) reduces platelet numbers and causes severe bleeding. However, it is a recessive condition, and in many patients only one null allele has been inherited (i.e. an allele which is largely a deletion). The lack of activity from the remaining functional allele has been linked to mutations in both the regulatory region upstream of the key gene and in its first intron. In 2015, a similar story was unearthed in the case of congenital scoliosis – one risk allele and one functional allele with a mutated regulatory region resulted in recessive disease.

> "Researchers have not been able to identify the cause of the other 80% of families' heritable cancer until now."

While many of the disorders for which non-coding mutations are relevant are rare, there are ground-breaking applications for treatment. In families with high cancer incidence, members can be tested for BRCA1 promoter methylation to inform their preventative treatment programmes. Large cohort studies can be employed to catalogue benign or damaging variants in non-coding genomic and epigenomic data so that we can catch genetic conditions early, when treatment is most effective. Personal genetic risk profiles can be compiled for patients which take into account the interactions between sections of DNA, allowing a future of exquisitely personalised treatments. Databases of this kind can also begin to teach us about the pathological mechanisms of diseases of which we still have a poor understanding of and even provide new drug targets. We have only begun to scratch the surface of what can go wrong with our non-coding DNA, but now that the causes are being unravelled, the hope for genetic treatment returns.



DOCTOR-INDUCED ADDICTION

Written by **Rachel Rubinsohn**
Art by **Chika Nwaka**

How corporate greed and duped doctors unleashed an epidemic of opioid addiction across America.

It kills 130 Americans every day. Like a virus, it has swept ruthlessly across the country, leaving burgeoning orphanages and mass memorials in its wake. Its pervasive reach transcends class, age and ethnic divisions, uniting communities in agony. This is America's opioid epidemic – a tragedy whose seeds were sown and diligently cultivated by a medical community in the pocket of big pharma. Twenty-first century medicine has become entangled in a web of corruption. It, and its patients, need rescuing.

Opioids are a class of drugs originally derived from the opium poppy, including morphine and heroin. Humans have been exploiting them for pain relief and euphoric highs since 3400 BC Mesopotamia, thoroughly aware of their addictive nature. In the early 1900s, the West was so acutely afraid of addiction that opioids were virtually eliminated from medical practice and were reserved solely for palliative care.

Now, just a century later, America is ravaged by a humanitarian crisis in prescription opioid abuse. How did the pendulum swing from one extreme to the other, so quickly?

"Like many tragedies, this one began with good intentions."

Stringent restrictions on opioids throughout the twentieth century left doctors ill-equipped to manage chronic pain. The worn-out bones and inflamed joints that plagued everyday life for millions went untreated. Pain specialists in 1980s America began to question the establishment's dogmatic dismissal of opioids: why deprive patients of the drugs that relieve end-of-life pain so wondrously?

What started as a perfectly honest question quickly descended into a ruthless crusade.

Saturating news media with the image of a nation crippled by pain, dissident campaigners extolled the virtues of opioids. The risk of addiction, they promised the masses, was negligible, because chronic pain would "neutralise" the feelings of euphoria; that this theory was based on the experience of a single patient was hidden from the cameras.

Their campaign gained the support of Purdue Pharma, a pharmaceutical company that had recently patented a morphine-based pain-killer called OxyContin. The powerful coalition demanded recognition of pain as a "fifth vital sign", for which treatment with opioids was a fundamental human right.

In 2001, their efforts paid off: the Joint Healthcare Commission declared that every single patient, irrespective of their reason for seeking medical help, must rank their pain level on a scale of 0 to 10, and be referred to a pain consultant should they exceed a 5. It put pain at the forefront of doctors' and patients' minds, medicalising the discomfort of everyday life and creating an unachievable expectation that pain could be totally eradicated. Months later, legal restrictions on medical opioids were relaxed and the floodgates were opened for mass prescription.

No stranger to aggressive advertising, Purdue Pharma besieged doctors with promotional material and sponsored illustrious conferences at holiday resorts. Video after video, doctors were promised that OxyContin's "delayed release" formulation had made addiction a relic of the past.

It was a lie; delayed release or not, OxyContin was heroin in a pill. As prescriptions were doled out freely, the opioid-binding receptors in the brains of unassuming patients began to undergo biochemical changes that would pave the way to addiction. Within a few months, they would feel their pain returning as they developed tolerance to the drug, and would plead for higher doses. Invariably, doctors would comply – showing reluctance to prescribing opioids landed many doctors before hospital ethics committees. When prescriptions ended, patients would be racked by withdrawal symptoms, driving many to seek relief through illegal substitutes.

It took the FDA (US Food and Drugs Administration) seven years to mandate that the risk of addiction must be openly stated on OxyContin's packaging. When Purdue Pharma was finally taken to federal court in 2007, company officials admitted to having known for years that OxyContin was being stolen, snorted, injected - all the while espousing its safety - and were charged $600 million, a mere fraction of the drug's $3 billion revenue.

The tide may finally be turning. As of June this year, 45 states have filed independent lawsuits against Purdue. But for the hopeless addicts, broken families and destitute towns, it's too late. Close to 200 million opioid prescriptions are still written each year, and opioids now kill more people than traffic accidents and firearms combined.

One might think that the FDA would have learnt a thing or two as they've watched this harrowing tragedy unfold. However, their decision in 2012 to approve a new opioid, Zorohydro ER, ten times as powerful as OxyContin, indicates otherwise. The FDA, once a beacon of scientific integrity, has been corrupted from within. An investigation by Science magazine in 2018 found that many "independent advisors" who vote on drug approval panels receive personal payments from pharmaceutical companies, with these conflicts of interest going unnoticed because the perks were being rewarded after a drug's approval.

This, if nothing else, is a potent warning to healthcare systems around the world. Murky relationships, creeping privatisation, and unchecked corruption can sabotage any organisation, even those whose very purpose is to keep us well. It is high time that we take back control of our health.

Why You Shouldn't Give Your Genetic Data to 23andMe

Written by **Becca Muir**
Art by **Mazeda Khanam**

Genetic-testing companies are transforming our lives. But why are they are so appealing?

Have you analysed your DNA? Although this might have been a weird question to ask a few years ago, in 2019 it would be unusual if you didn't know at least one person who has bought a direct-to-consumer genetic testing kit.

What purpose do these DNA kits serve? Health is an aspect of the service, where you can find out your risk for certain diseases based on whether you carry a rare variant or not. However, as the health tests included are not particularly insightful, there is a reason to suspect that this isn't why the tests are so popular.

Genetic testing has become another way of finding out the truth about who you really are. The test delves into your DNA to help you discover your ancestors, connect with long lost relatives, and could even be adapted by third parties to help you make daily decisions such as what type of sushi you should eat.

We live in the age of Buzzfeed quizzes and Myers Briggs personality types, so it is unsurprising that these kits are popular. In an unpredictable world where the key demographic for quizzes and tests are facing economic and political uncertainty, genetic testing has honed into the very human urge to create order out of chaos. These companies have convinced us that these tests are just another way of becoming informed about our identity and that this is a pretty harmless activity, akin to finding out which Disney princess you are based on your favourite snacks.

But there is more to these tests than what meets the public gaze. It turns out that we're sharing much more than previously thought, and that the potential for our data to be abused is something which is only beginning to be questioned.

The dramatic increase in data has transformed the power of genetic testing. This is because when you share that saliva, you're adding to a big collection of saliva samples owned by only four companies, containing the genetic information of 26 million people across the world. It is really hard to overstate how much information that is. It is the biggest biobank in the whole world, and of all human history. For comparison, the UK's highly commended biobank only has genetic information on 500,000 people at best. It's so huge, that if this was an academic study, it undoubtedly would be one of the most expensive, time-consuming, and laborious studies ever accomplished in biomedicine.

"When you share that saliva, you're adding to a big collection of saliva samples owned by only four companies, containing the genetic information of 26 million people across the world."

In contrast though, this whole enterprise has been rapid, and very cheap. In fact, these companies are making a profit. Not only are people giving up their data readily, but they are also paying for the privilege. However, the profit coming from the consumer was never the point. The main profit-making part comes after: when our biobank data is sold.

Of course, theoretically, there could be positives to sharing the data in these databases. We can't just automatically assume that capitalism-fueled progress is the slippery slope leading to the plot of GATTACA. No one can argue that medical progress is a bad thing. The question is, can we trust that these companies have pure intentions?

We can look back at the history of genomics to understand why we really should not trust them at all. In the early 2000s, the race to sequence the human genome saw Craig Venter try to patent over 6500 gene sequences. This move would have made it so anyone wanting access to the genetic code would have to pay-for-gene through his company, essentially holding the entirety of progress in the academic biosciences ransom to the whims and questionable ethics of a capitalist tycoon. The only reason that Venter did not succeed is because President Clinton announced that genes could not be patented. So, straight off the bat, the biotech industry has tried to commodify all of the available code to human life itself.

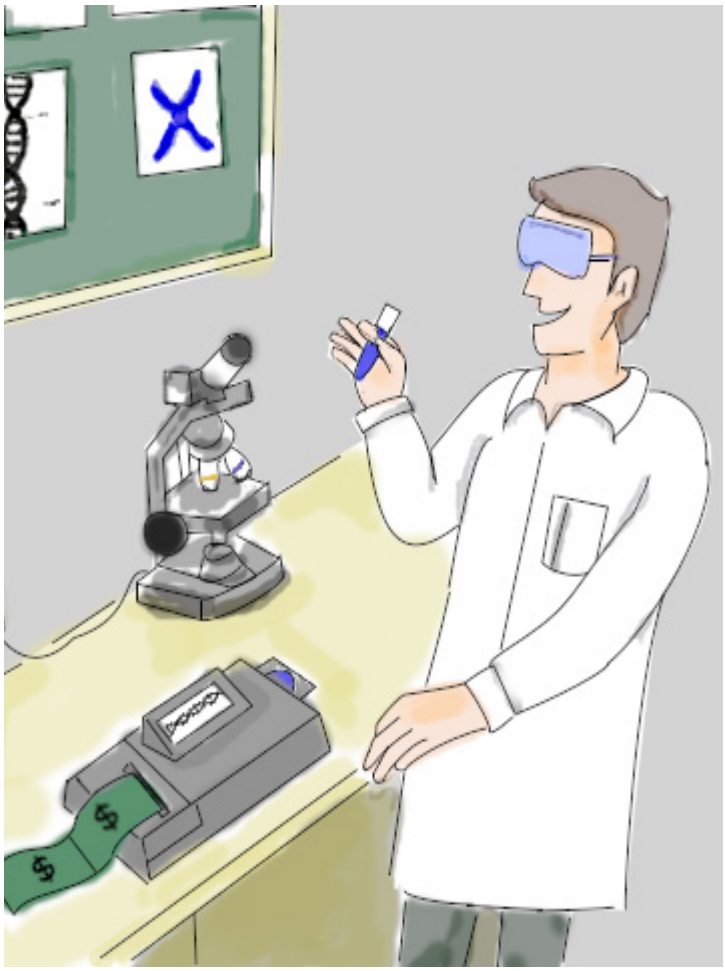
Today, not only are our world leaders too dangerously apathetic about science to regulate properly, but they also just don't grasp the magnitude of technological change. This is not just because their genetic literacy is, at best, at the level of a 19th century white supremacist. There is a deeper issue reflecting our lack of understanding about the omnipotent power of Big Tech. As James Bridle, author of The New Dark Age warns, we have a "simple-minded acceptance of technology as a value-neutral tool, one to be freely employed for our own betterment".

But the problem is that these tests place medicine into the hands of companies who ultimately care about profits, not the enhancement of human health. So, even when companies promise miracle cures for cancer, this is not the main motivation behind gathering your data. It never was. The purpose is to make money, at any expense.

The monetisation of your genetic data reflects the wider trend of surveillance capitalism, where your personal life and intimate details are seen as extractable capital which can be sold to whoever pays the price. As we have already seen, countries such as Canada are using ancestry companies to deport people, and the US government will happily give your SNP s to the FBI to "help catch bad guys".

If this seems bad, try to not think about how terrifying it is that the big pharma company GSK, who can receive $3 billion worth of fines for bribing doctors and still remain a FTSE 100 giant, has now been granted at least four years of access to 23andMe data.

It's dark. We need to realise that capitalism has left a monstrous mark on genomics since its inception, and will continue to do so until we stop letting companies sell us as products. Until then, sticking to the Disney Princess Buzzfeed quizzes might be for the best.



The Mojito Muse

Written by **Sunny Liu**
Art by **Lisa Burna-Asefi**

Is there any scientific evidence behind the Creative Alcoholic stereotype?

The morning sun shone through my window. Wearily, I forced my eyes open, only to see an unfamiliar… thing. A traffic cone. In my bed. Under the covers. This raised many questions. Where had I gotten it from? Who did it belong to?

After a mild rest, I retraced the remnants of my memory. Last night, after finishing some vodka at a pre-event, I had realised three things on the way to the club:

1. I'm slurring a little.
2. I (accidentally?) texted my ex :O
3. I knew the solution to a question in my calculus problem set.

A monumental discovery of that night was the phenomenon of alcohol-improved problem-solving skills. The next day, I sat down with a beer (and a few more), my favourite traffic cone, and went about my problem set.

Tragically, but unsurprisingly, I only got one question correct. It wasn't even the vodka-inspired one. Perhaps that's why I'm not studying maths.

To examine the effects of alcohol on creativity, we must first define creativity. In psychology, creativity is thought to rely on both controlled and spontaneous cognition. On one hand, mental resources are required to focus our attention on a problem and think of various approaches. On the other, one also needs to balance that focus by "loosening up" and thinking outside-of-the-box, especially when one is fixated on a dead end – an impasse of thought, such as the infamous writer's block. Creativity can thus be measured by a combination of divergent thinking tasks and the Remote Associates Test (RAT).

An example of a divergent thinking task is one in which participants are asked to find creative uses for common objects which are scored by six independent judges. The RAT presents three unrelated words to the participant, who is tasked to find the connection. For instance, "cheese" connects the words "blue, cottage, cake".

A brain region that is heavily implicated in creativity is the prefrontal cortex. This area is known to be responsible for inhibitory control, executive function, and logical thinking. As expected and controlled cognition is highly associated with prefrontal cortex activation, the lack thereof is associated with spontaneous cognition, the other half in the dichotomy of creativity.

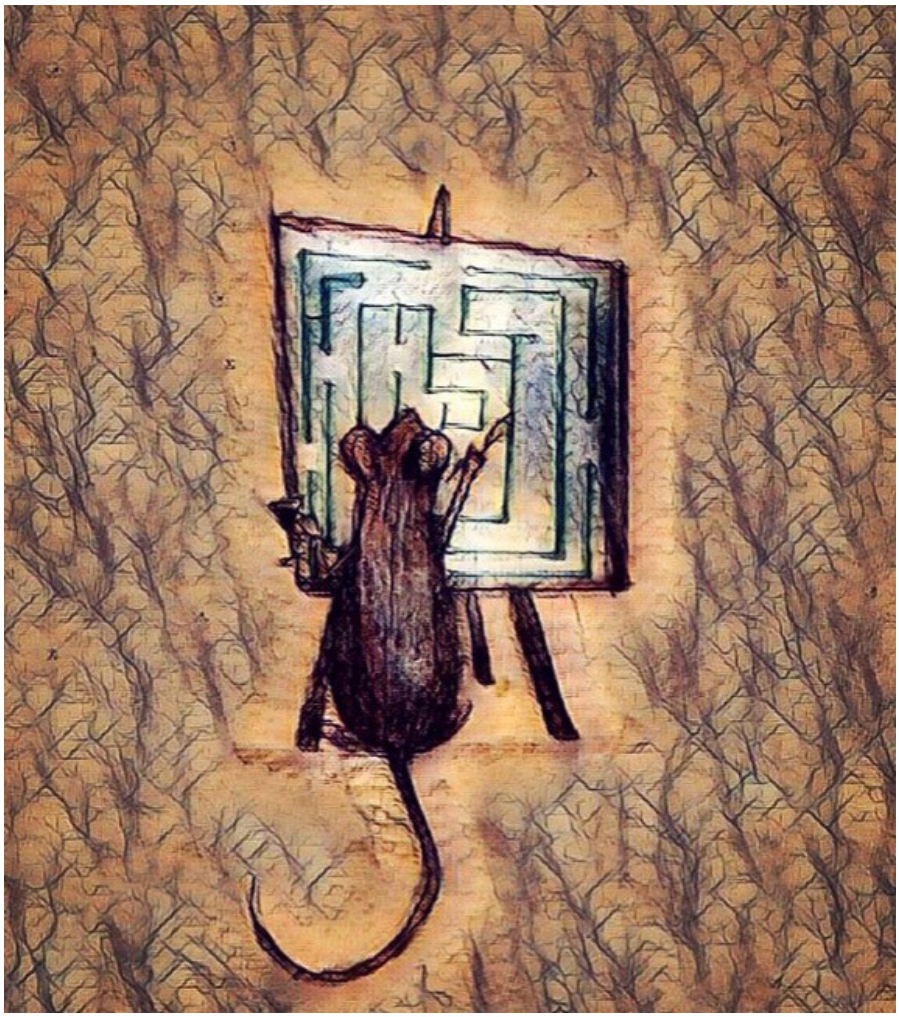
This is where it gets interesting.

Alcoholic beverages, specifically the psychoactive component, ethanol, is known to have a multi-faceted effect on our brain. Aside from its addictive and euphoria-inducing properties, ethanol is known to inhibit prefrontal cortex function. As anyone who's had one too many drinks and subsequently made questionable (if not regrettable) decisions can attest to, ethanol's ability to "loosen" one's inhibitions cannot be doubted. Personally, I think some of these decisions can be rather creative as well, as evident in the number of drunk students I've witnessed being stuck in shrubberies.

"In psychology, creativity is thought to rely on both controlled and spontaneous cognition."

This may be one of the roots of the urban myth that one becomes much more creative when inebriated. Tying back to neuropsychology, it appears that alcohol may have a mixed effect on creativity – while it is expected to hinder controlled cognition which is vital when approaching a problem, it is hypothesised to enhance spontaneous cognition. This leads us to a paradox - will alcohol therefore enhance or hinder creativity?

A professor in Austria sought answers in what is, undoubtedly, a rather exciting research experiment to participate in. Mathias Benedek and his team from the University of Graz found that alcohol did, in fact, aid creative thinking.



Participants who drank moderate amounts of beer had a statistically significant higher creativity score than those who were sober. Despite inhibiting executive control (even at very low amounts), which likely reduced the participants' ability in controlled cognition, the creativity score was more than compensated by the improvement in measurements of spontaneous thinking.

As the authors noted, alcohol may possibly mitigate fixation effects, which is the phenomenon when previous conceptions block the generation of novel ideas, and this is perhaps something to keep in mind the next time you have an essay to write but you're suffering from a mental block (please note: I am not responsible for your grades). Unfortunately, likely due to ethical reasons, research was conducted only on lower amounts of alcohol – we do not know if there is a dose-dependent effect. I'd like to think that there is. There probably isn't.

Of course, this may not be the whole picture. Other researches have shown that ethanol makes one perceive his own ideas to be a lot more creative than they are – undoubtedly, one of the factors contributing to the urban legend of the Mojito Muse.

Ultimately, you should understand the effects of alcohol on your own cognition. If you want to drink, do so in moderation – as in everything else. As Mark Twain said, "I never smoke to excess – that is, I smoke in moderation, only one cigar at a time."

the silent witness

Written by **Rupali Dabas**
Art by **Mazeda Khanam**

DNA on trial: can DNA evidence actually lead us to the truth?

Accused of at least 13 murders, more than 50 rapes, and over 100 burglaries, the infamous "Golden State Killer" was finally apprehended after 45 years, based on a DNA profile recovered using a rape kit.

Detective Paul Holes, who oversaw the case, uploaded the murderer's DNA profile into a personal genomics database called "GEDmatch" under a fake account. A large family tree was subsequently discovered from which two suspects were identified. To affirm their suspicions, a team of officers surreptitiously collected a DNA sample from the door handle of the suspect's car. The sample was an exact match. On April 24th 2018, Joseph James DeAngelo was arrested, and initially accused of 13 accounts of first-degree murder.

The power of forensic DNA analysis is unquestionable – it is seen as the "gold standard" of crime scene evidence. Since Sir Alec Jeffreys' innovation of genetic fingerprinting in 1984, DNA analysis has become a major focus of study for forensic scientists. It has played an irreplaceable role in the criminal justice community by facilitating the conviction of the guilty and exoneration of the innocent.

Since the 1980s, forensic DNA protocols have become faster, stronger, and increasingly accurate through the application of more sensitive technologies that enable greater access to the investigative potential of DNA testing. Forensic scientists now use single-nucleotide polymorphisms (SNPs) and short tandem repeat (STR) sequences in DNA profiling.

These sequences represent highly variable regions that differ between individuals. And these differences, called polymorphisms, are inherited from our parents and are used to generate a DNA profile. As these sequences are only a few bases long, they are highly sensitive and allow the recovery of information from degraded DNA samples.

However, despite our greater understanding of the benefits of forensic DNA analysis, there are some problems associated with its use. Forensic scientists are becoming increasingly frustrated with the time taken for existing technologies to generate reliable DNA profiles. Sylvain Hubac, from the Forensic Science Laboratory of the French Gendarmerie (IRCGN), revealed that "the main goal today is obtaining a DNA result as quickly as possible…a mobile-DNA lab with fast and easy workflow can be very useful in obtaining DNA results in real time after collection".

So, how close are we to achieving this goal?

Currently, it takes up to 72 hours for the police to receive a DNA profile from a crime scene, by which time the suspects have often been released. Keeping this in mind, a team of scientists at the University of Arizona developed "MiDAS", a Miniaturised Integrated DNA Analysis System. This is a chip (no bigger than your hand!) that can extract, amplify, and analyse DNA. It only takes two hours to generate a complete DNA profile based on expert STR analysis! It can also process samples in parallel, which can be used to investigate multiple suspects.

Despite the numerous benefits of this technique, it is far from being integrated into current protocols that forensic scientists use in crime scene investigations (CSIs). This is because using such rapid-DNA techniques entails the risk of losing potential evidence, which is significant in cases where there is not much available. Hence, there might be no DNA left to be analysed by more trusted and existing techniques in forensic labs – this may let a criminal walk free! Using such rapid-DNA technologies may also generate less accurate results than the conventional techniques used in forensic laboratories. Therefore, despite the greater speed of DNA analysis offered by MiDAS, there is a need for more accuracy before it can be routinely used in CSIs.

Though DNA analysis is a key element in forensic analysis of crime scenes, contrary to popular belief, it is but a small part of discovering the truth behind a crime. Having blind faith in DNA evidence is dangerous and has led to wrongful incriminations of innocent people in the past – a topic that is seldom explored in popular crime shows.

In 2011, a woman was brutally raped in Plant Hill Park, Manchester. DNA evidence taken from the victim led the police to Adam Scott, who was promptly arrested. Upon questioning, he said that he'd been in Plymouth on that night, 200 miles away from the scene of crime. As no one could corroborate his claims, he was incarcerated. Scott spent five months in jail before it was discovered that the swab used to collect Scott's DNA in an earlier incidence was accidentally re-used in the Manchester case. After further investigation into his phone records, he was liberated. Unfortunately, this isn't an isolated case. Even now, the FBI is attempting to amend sentences delivered based on faulty DNA evidence.

Isn't it astonishing then that we blindly trust the judgements of evidence based on something invisible to the naked eye? We must be aware of the incredible power DNA has to initiate a chain of events that could culminate in the conviction of an innocent person.

For this reason, DNA alone is almost never enough to deliver a conviction. Powerful it may be, it still remains a silent witness.

OPEN ACCESS AND PLAN S:

THE ROCKY ROAD TO ACADEMIC UTOPIA

Written by **Bruno Reynell**
Art by **Mazeda Khanam**

What does Science Europe's recently announced initiative mean for the open access movement?

Imagine a world of academia without the frustration of paywalls. A world where researchers are afforded unfettered access to the findings of others, which they can then discuss, challenge, and add to. One where taxpayers are able to see the results of the research they have helped to fund, and teachers and students in institutions around the globe can read the latest findings in any field.

This is the idealistic vision of open access, and one posited by the Budapest Open Access Initiative in 2002 - a seminal moment in forming our contemporary understanding of this term. Its statement identified a hitherto unavailable opportunity found at the intersection between the desire and willingness of academics to disseminate their research for the public good, and the possibilities offered by the new technology of the internet.

In the years since, however, a range of developments have made clear the complexities involved in defining openness. For example, in terms of publishing methods, two broad models of open access have established themselves – "gold" and "green". Gold open access means that an article is made freely available from the publisher upon publication. Meanwhile, green open access sees the self-archiving of a specific version of a manuscript into an open repository. An example of such a repository is our university's very own UCL Discovery, which provides free access to UCL research outputs from all disciplines.

However, even within these two terms, there are greater levels of nuances to be considered. For example, one can look at the megajournal PLOS's "HowOpenIsIt?" guide, which was created in conjunction with the Scholarly Public and Academic Sources Coalition and the Open Access Scholarly Publishers Association. This evaluates six different factors from reader rights to machine readability, demonstrating the extent to which openness can be seen as a spectrum. This multi-dimensional nature of openness means that it often becomes an area of great complexity and debate.

It is against this backdrop that Plan S, an initiative supported by an international consortium of research funders (cOAlition S), was announced last autumn. Plan S mandates that, by 2020, scientific research funded by these participating bodies must be published in compliant open access journals or platforms. For many, the transition to open access has been lethargic, and thus Plan S has been celebrated as reinvigorating the movement.

The initiative's principles are certainly bold, and the kind of language it employs leaves little doubt about its ambitions or expectations. For example, on the subject of academic responsibility, the cOAlition S website states that "researchers must realise that they are doing a gross disservice to the institution of science if they continue to report their outcomes in publications that will be locked behind paywalls".

"If an academic publisher is no longer gaining revenue from subscription fees, who is paying for the value that they are adding to research?"

For open access advocates, Plan S is a clear leap forward to a future that could see the gradual abolishment of the traditional (and incredibly lucrative) subscription-based model, whereby librarians have to pay publishers substantial sums of money to have access to research in journals.
The argument goes that this will allow for greater dissemination of knowledge by helping researchers and driving innovation, ultimately benefitting society as a whole.

This all sounds wonderful, so where's the catch? Well, unfortunately, at the moment, there are many catches, and these concern money. If an academic publisher is no longer gaining revenue from subscription fees, who is paying for the value that they are adding to research? Publishers in this situation require the payment of an article processing charge (APC) to cover the cost. However, this spells problems for authors who haven't budgeted for APC costs in research grants.

Furthermore, this becomes even more of an issue for researchers in low-income countries who might lack the funding to pay the APCs to publish in prestigious, high-impact journals.

For example, the 2018 STM Report commented that "a single APC of $2,000 is equivalent to many months of a researcher's salary". This gives rise to concerns about the creation of "new professional hierarchies", especially considering the fact that APCs at top ranking OA journals can range all the way up to $5,000.

Then there is, of course, the small matter of academic freedom. While this isn't something enshrined in law, there is a longstanding tradition and understanding in academia that researchers should be able to publish without institutional restriction. Many commentators have highlighted the fact that Plan S would impose great constraints on an author's choice of publishing venues.
Lynn Kamerlin, a biochemist at Sweden's Uppsala University, coordinated an open letter criticising Plan S last November with over 950 signatories and claimed that cOAlition S-funded authors would be forbidden from publishing in more than 80% of existing journals. She also argued that not being able to publish in certain journals would stall the career progression of researchers.

Finally, it is difficult to predict the extent to which Plan S will trigger a shift to openness in academic publishing. While it is backed by some powerful bodies like the Wellcome Trust and the Bill and Melinda Gates Foundation, the grantees of these funders still account for only a small fraction of the audience of prestigious journals in many fields. In other words, unless the initiative gains greater traction, it will remain difficult to convince publishers to "flip" to open access models.

Wider transmission of knowledge leads to enriched discussion of science and an efficient uptake of research by society. Few question the worthiness of the idea of open science, and all the potential benefits that it entails. However, as concerns and criticisms around the open access movement and Plan S (of which the above are but a small selection) demonstrate, there remain many questions to answer and a long way to go before it becomes a widespread reality.

Eating to change our brains:

THE GUT-BRAIN AXIS

Written by **Charlotte Li**
Art by **Bella Peng**

Is having a healthy gut the key to your mental health?

You're waiting for your date to arrive and your stomach is fluttering. Your "gut" feeling tells you something is wrong, you're anxious and miserable, but what you don't realise is that this stems from the trillions of bacteria that make up your gut microbiome, and these outnumber even your human cells.

How you feel now is affected by the bacteria in your gut, with the gut and brain being interlinked by the Gut-Brain Axis. This is influenced by the vagus nerve that runs from the brain all the way to the colon, and innervates a major depression system, the Hypothalamic Pituitary Axis.

From the moment we are born, our microbiome continually changes due to our diet and feeding patterns, determining whether we are more or less prone to disorders such as obesity and even autism.

Gastrointestinal (GI) dysfunction is very common in neurodevelopmental conditions such as Autism Spectrum Disorder (ASD) and schizophrenia. In fact, when researchers treated an ASD mouse model suffering from GI dysfunction with the bacteria *Bacteroides fragilis*, this not only restored intestinal permeability and metabolite levels. It also alleviated ASD-related behavioural symptoms such as anxious marble-burying, and even produced a better response to social encounters, thus demonstrating a clear relationship between the gut and brain.

Autistic patients have been found to have more species of Clostridia bacteria and higher levels of propionic acid (a product of Clostridia fermentation) in faeces. When autistic children were put by researchers on a specific antibiotic (Vancomycin) that depletes Clostridia bacteria, their autistic behaviour drastically improved, demonstrating the critical role microbes play in ASD.

"Studies show us that our behaviour and even our memory is dependent on the microbes living in our gut."

Germ-free mice are found to have lower levels of Brain-Derived Neurotrophic Factor (BDNF), a protein important in memory and learning (low levels were linked to depression and anxiety). When compared with normal mice, germ-free mice had noticeable memory defects as well as an exaggerated anxiolytic behaviour. In another experiment by McMaster University, when the faeces of extroverted and introverted mice were exchanged, their anxious behaviours and BDNF levels also changed accordingly. These studies show us that our behaviour and even our memory is dependent on the microbes living in our gut.

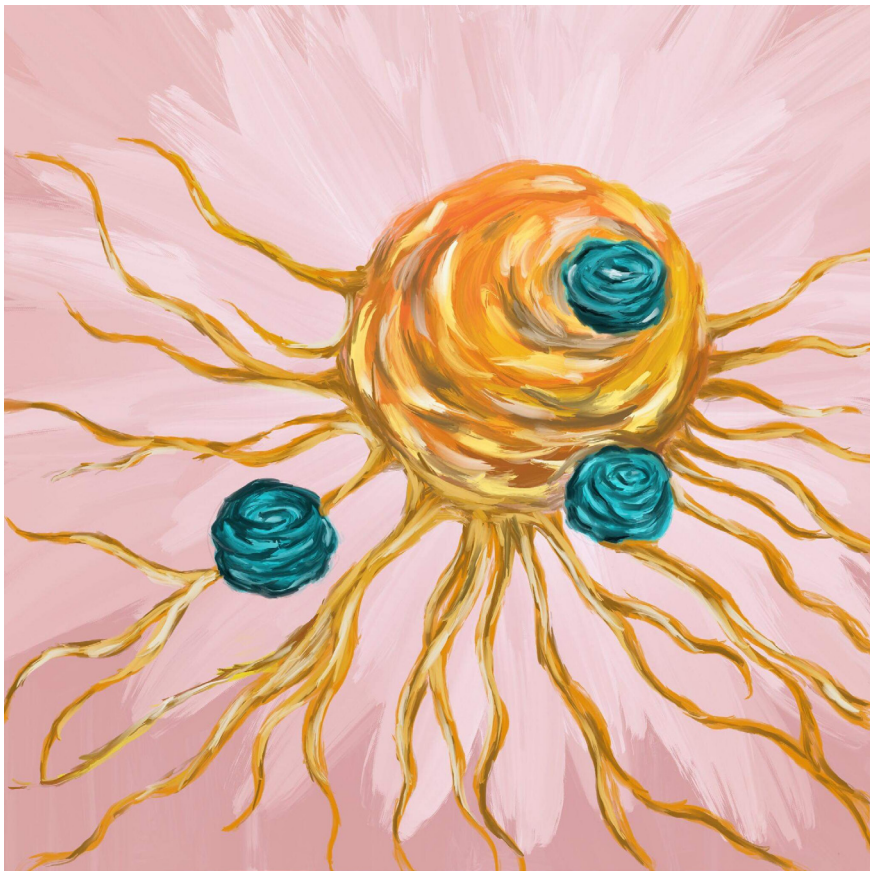
Despite what sceptical parents might say, depression is not just "all in our head". 90% of the neurotransmitter serotonin is produced in our GI tract, and it is the most common target for anti-depressants, regulating our mood and circadian rhythm. Microbes have also been shown to produce neurotransmitters which produces relaxing and anxiolytic effects. Our diets can hence affect the neurotransmitters released, affecting our emotion.

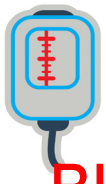
> "A 'Western' diet has been associated with a smaller hippocampus and a 25% higher risk of depression as compared to eating a 'traditional diet'."

Since now we know that the microbes in our gut affect the development and function of our brain, so what we eat is very important to our mental health. Ancient civilizations swore by drinking sour milk before going to battle, an example of probiotics - fermented food that can stimulate the growth of "good microbes". These probiotics have been shown to have anti-anxiety and anti-depressive effects that are similar to therapeutic drugs such as Citalopram.

An example of natural probiotics are the antioxidant compounds found in fruits and vegetables. These can reduce neuronal damage induced by oxidative stress, especially in the hippocampus, an area of the brain involved in memory. A "Western" diet has been associated with a smaller hippocampus and a 25% higher risk of depression as compared to eating a "traditional diet". This is because the latter includes unprocessed food with unrefined sugars, such as lentils and asparagus, which are other natural probiotics. Our "good" gut microbes ferment prebiotic fibres (e.g. garlic and artichoke), producing short-chain fatty acids that strengthen our gut lining as well as our blood-brain barrier, which has also been implicated to control our gene transcription.

Taking care of our microbiome by eating right is one of the most important things you can do for your mental and physical health. Improving cognitive function or treating brain-related disorders with bacteria is not too far-fetched. Professor John Cryan, a microbiome expert from University College Cork, commented that "microbiome-derived medicine is the future of precision medicine". This suggests that we might even be en-route to "poop-doping" with faecal transplants for greater performance and mental agility.





Does the Cure for Blood Cancer Lie Within?

Written by **Anisa Mohamed**

Through the cellular modification of your own blood, a notorious cancer can be treated.

Cancer, the world's greatest medical challenge, is one of the leading causes of human deaths worldwide, accounting for 9.6 million deaths in 2018 according to the World Health Organisation. We question whether the billions of funds going towards its research is fruitless. We build conspiracy theories on whether pharmaceutical companies have already developed cures for the hundreds of different cancer types.

We are sitting ducks waiting for news outlets to report on the latest updates, be it the successes or failures of novel chemotherapy. Nevertheless, the wait for a promising treatment for Acute Lymphoblastic Leukaemia (ALL) has ended, thanks to a leading multinational pharmaceutical company - Novartis. Novartis has made an early commitment to the emerging field of immuno-oncology with one of its facilities being the first manufacturing site approved by the Food and Drug Administration (FDA) for immunocellular therapy production in the US.

Haematological oncology anticipates the wide scale use of chimeric antigen receptor (CAR) T-cell therapy in the treatment of ALL. ALL is a rapidly progressing and aggressive blood cancer affecting the individual's white blood cells. It is commonly diagnosed in children and young adults and yet has a poor prognosis and outcome, hence the dire need for clinical development and therapy. In collaboration with the University of Pennsylvania, Novartis has successfully utilised CAR T-cell technology to outsmart cancer cells.

So, how does CAR T-cell therapy work?

In a clinical setting, the patient's blood is first drawn and white blood cells, including T-cells, are extracted. The T-cells are then genetically engineered to produce CAR proteins on the cell's surface. The CAR T-cells, once infused back into the patient's bloodstream, aim to attack and eradicate cancerous cells, thus resulting in prolonged remission. This dynamic therapy allows the modified cells to circulate the bloodstream mimicking normal blood cells.

When in contact with cancerous cells, they release cytokines which are responsible for the recruitment of other cells that now also target the malignancy. Essentially, the modified T-cells are able to recognise and target antigens on cancerous cells in the bloodstream.

> "The beginning of a new era of innovative and effective chemotherapy is amongst us."

After centuries of endless research, promising results in clinical trials in 2017 enabled the FDA to approve the use of "Tisagenlecleucel", which are humanised CD19 targeted CAR T-cells, branded as Kymriah™ for the treatment of relapsed or refractory B-cell ALL in children and adults under the age of 25. Kymriah™, being the first CAR T-cell therapy to obtain FDA approval, has revolutionised the research scene in oncology. This decision was influenced by the outcomes of three key clinical trials, one of which was deemed as the pivotal study. The JULIET trial has demonstrated that 64% of patients remain relapse-free with a 43% overall survival rate in patients with relapsed or refractory disease 18 months into the treatment. With the initial reservations that the National Health Service (NHS) had regarding the complexity of manufacturing and cost of Kymriah™ (which stands at a staggering £282,000 per patient), negotiations with Novartis and the National Institute for Health and Care Excellence has led to the availability of this therapy on the NHS to those eligible.

The beginning of a new era of innovative and effective chemotherapy is amongst us with individualised CAR T-cell therapy standing on the frontlines. Cancer survival rates in the UK are currently at the highest they have ever been, and the development and adoption of cutting-edge technology will be critical in the transformation of future cellular therapies.

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