



the Oxford Scientist

Oxford University's independent, student-produced science magazine

HT 26

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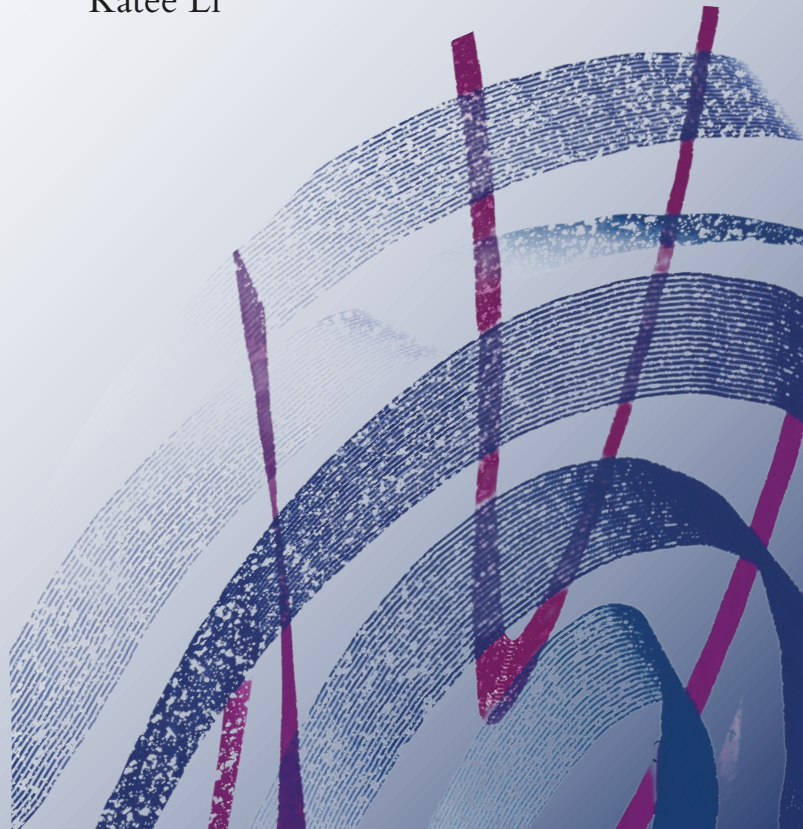
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Editors' Letter

Dearest readers,

It feels like only yesterday that I submitted the application form to be a subeditor and kickstarted my time at the Oxford Scientist. In fact, that was my first term as a fresher all the way back in MT23 – and what a journey it's been since. Three years later, as Editor-in-Chief I could not be more honoured and excited to present our HT26 edition, *Anomalies*!

Science prides itself on its capacity for empiricism. Yet in reality, science rarely progresses through certainty; instead it moves forward in the moments when data disrupts our expectations and models falter under closer scrutiny. Instead of dismissing anomalies, we may consider them as signals that something deeper demands attention.

We are living through an era that increasingly flattens nuance and tension in favour of convenient certainties. In doing so, we risk forgetting that anomalies are precisely what drive progress by compelling us to delve into our assumptions and re-examine what we hold to be irrevocably true. They expose the flaws embedded in our disciplines, our institutions, and our public narratives about science.

The role of science journalism is, then, not simply to report these findings, but to interrogate the frameworks through which those findings are understood. By examining where our expectations fail and why, we make space for more rigorous and honest inquiry. In this edition, we explore topics from climate to sports science, cosmic rays to cancer. These anomalies remind us that no discipline is immune to revision, and only by transparently confronting what does not fit can we uncover the truths which ultimately endure.

The edition would have been impossible without an incredible team behind the production of the magazine. Hilary is not an easy term to get through, and I am indebted to my senior editorial team, who never fail to approach their roles with consistent enthusiasm and dedication on top of their existing workloads (particularly as STEM students). I would like to thank the writers and artists who contributed to this edition by bringing their fascinating ideas to life; and the Print Editorial and Creative teams who have pulled together their creative sparks and sharp eyes to assemble the beautiful magazine you now hold in your hands. A special shout out to my Deputy Editor-in-Chief Iulia for her indispensable support; in her capable hands, the magazine is undoubtedly set up for a fantastic Trinity.

So without further ado, happy reading, and never stop discovering! You may not always know what you will find, but it is often in the unexpected where understanding begins.

Big OxSci love,
Ellie Yau
Editor-in-Chief, HT26

Life in the mirror

Adele Batchelor

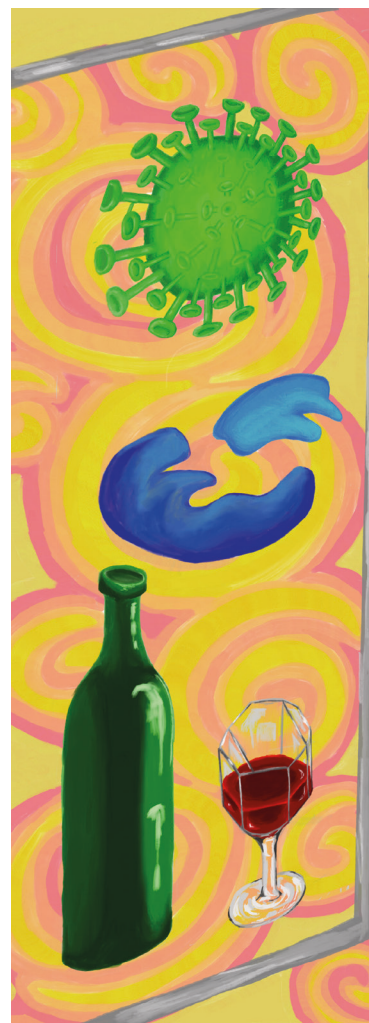
A study published in 2024 by a group of 38 scientists, including two Nobel Prize laureates, warns us of one of the biggest risks to our survival—mirror image life. Beyond the threat of nuclear war, climate change, or uncontrolled artificial intelligence, an unprecedented risk may arise from a source few anticipated: a single cell composed of reflected structures.

We must begin with the idea of chirality, a concept discovered by Louis Pasteur in 1860 when observing crystals of tartrates, a molecule found in wine. Under the microscope, he noticed a facet on the crystals which was oriented to the left in some and the right in others. The molecules were composed of two mirror images, a property defined as chirality. Put the palms of your hands against each other. They are mirror images, right? But now put one on top of the other. You cannot make them overlap; they will never occupy the same 3D space. These, like the wine molecules, are chiral.

Why do mirror image molecules matter? The case of thalidomide provides an example. This molecule was used as an anti-nausea medication for pregnant women in the 1950s. The right-handed (R) molecule was effective at pain relief; however, the

left-handed (S) one caused birth defects in their babies. The reason behind this: our bodies are chiral too. Our RNA and DNA are all “right-handed”, whilst proteins are made from “left-handed” amino acids. Our biological systems therefore react differently to mirror images.

This brings us back to the “unprecedented” risk brought to our attention in 2024. Let’s imagine we could synthesise a single mirrored bacteria cell, made of the same building blocks but arranged in opposite c o n f o r m a t i o n s . Our immunity is largely dependent on the recognition of molecular shapes on pathogens. In this scenario, since the chiral bacteria cells would have proteins made of the opposite amino acids, identification would be hindered. Similarly, the binding of antibodies and immune cells to bacteria for destruction would no longer be possible if their 3D configuration was reversed. It would



be like placing a glove on the wrong hand.

Our immune “memory”, which can produce a stronger response once a pathogen returns, would be equally compromised. Our DNA, RNA and aminoacids are conformationally locked, so we can only synthesise one-sided proteins. These would be the incorrect match. Similarly, pharmaceutically produced antibiotics would not help us fight infection, allowing new resistance pathways. Without immunity or treatment, these fast-spreading bacteria would likely result in lethal infection.



The threat doesn't stop there. Bacteriophages, the viral predator to bacteria, would be unsuccessful in binding to the surface proteins, and their replication machinery would be unable to work in the chiral cell. Other bacterial predators, including amoebas, would face weakened recognition, similar to our immune system. Without predation, transmission would be facilitated, making it likely that

surface proteins, and their replication machinery would be unable to work in the chiral cell. Other bacterial predators, including amoebas, would face weakened recognition, similar to our immune system. Without predation, transmission would be facilitated, making it likely that

bacteria would become invasive species across ecosystems, exposing humans, animals, and plants to lethal infection.

Despite this apocalyptic description of chiral life, their biodegradation avoidance has potential in therapeutics, and so research in the area continues. An achiral drug could last longer with a lower dose. Many mirrored peptides and RNA molecules are currently in development to treat cancer, metabolic, and inflammatory disorders. Researchers have recently synthesised an oppositely configured enzyme, T7 RNA polymerase, which is a key step in creating left-handed RNA. When combining this RNA with other enzymes, you could create a mirror image ribosome—a cellular structure which would be able to link chains of right-handed amino acids in protein synthesis. This development could drastically increase the production of chiral pharmaceuticals with high output of peptides, but it is also a potential structural component of mirror image cells.

The creation of mirror image organisms would be a feat of biological engineering, more complex than has ever been achieved. The current developments are far from it, but to some, it is an ominous stepping stone. Debate in this area is rife—is it worth exploring life in the looking glass?

How Sports Science Fails the Individual

Jasmine Price

In the late 1940s, the US Air Force had a problem: pilots were losing control of their planes at an alarming rate, but they had great pilots and better planes than ever before. The issue was traced to the cockpit. The rational solution was to design it for the “average” pilot, but this was the fatal mistake that cost them 17 pilots a day at its peak.

A study by Gilbert S. Daniels (1952) tested over 4,000 pilots on ten key physical metrics. The assumption was that most pilots would fit the “average” parameters, defined as the central 30% of the results range, but surprisingly, not a single pilot did. By designing for the average, they’d designed for no one. The study led to a complete redesign of the cockpit and mortality rates plummeted—a huge success.

Just as the Air Force nearly destroyed its fleet by building for an “average” pilot that didn’t exist, modern sports science risks failing their athletes by prescribing “average” training loads and recovery protocols for jagged biological profiles that don’t fit the mean. Average kills talent.

Consider the example of the glycaemic index: the gold-standard metric used to describe blood glucose spikes after

consuming carbohydrate-containing foods. A study by the Weizmann Institute of Science (2018) demonstrated enormous variability in participants’ glycaemic response to a food. Some experienced wild blood sugar spikes after consuming a banana and almost no spike after a cookie, whilst others experienced the exact opposite, though both foods provide the same amount of available carbohydrates. These findings suggested that the glycaemic index is not an inherent property of food. Instead, it is a property closely related to an individual’s unique biology and microbiome. Thus, dietary recommendations and guidelines (based on the “average”) are flawed—health is personal.

Consider another example: one in five people are cardio “non-responders”. The HERITAGE Family Study (1999) uncovered that this group of people could train for months and see no improvement in their aerobic capacity. To them, the UK guideline of 150 minutes of aerobic exercise per week is irrelevant.

Furthermore, the majority of endurance athletes use caffeine to aid their physical performance. This benefit is governed by the CYP1A2 gene, which controls

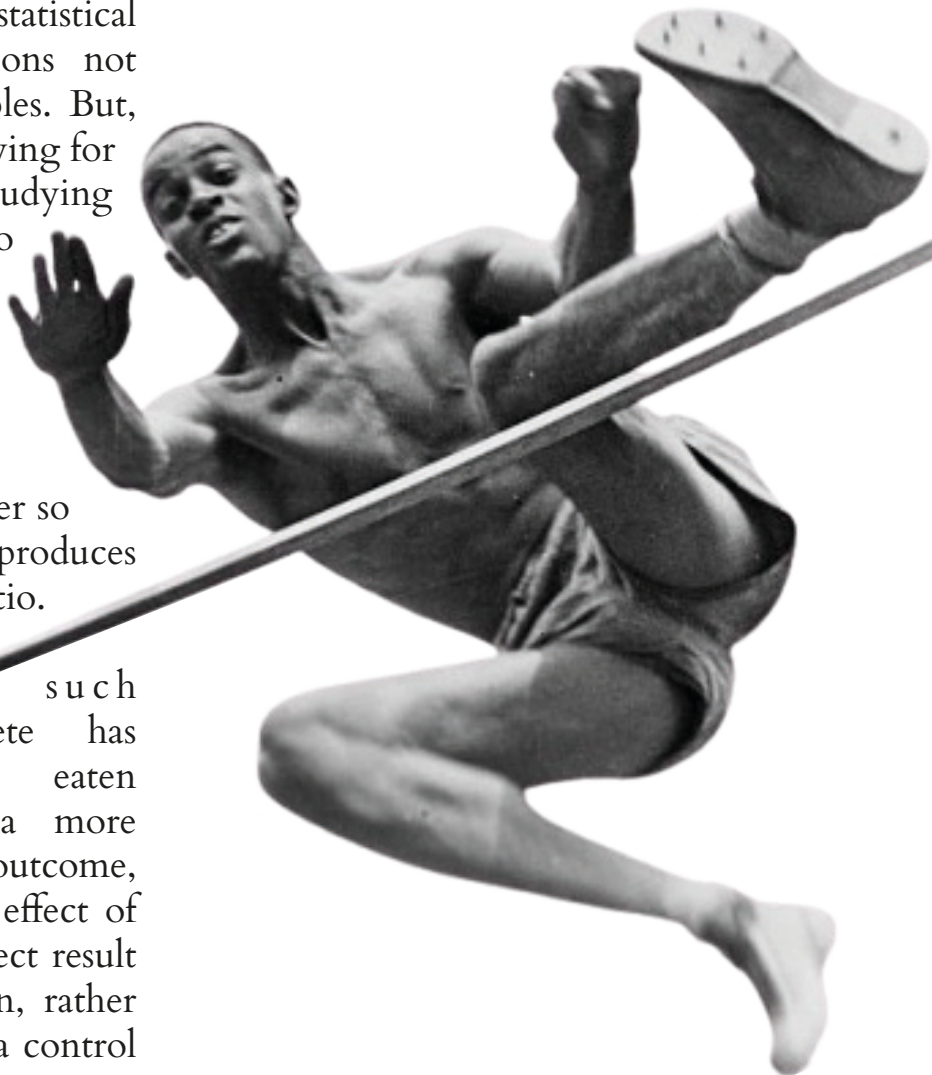
the rate of caffeine metabolism. In spite of that, a study by Guest, N., et al. (2018) showed that for the genetic “slow metabolisers” (consisting of 40–45% of the population), caffeine negatively influenced their time in a ten-kilometre cycling time trial. There are many other cases of the “adverse responder”: individuals who adhere strictly to the guidelines yet face negative performance outcomes or even injury.

Good science seeks to explain such outliers, but sports science research often chooses to disregard outliers as noise. Research has continued to rely on group-style statistical analyses that hold assumptions not achievable with smaller samples. But, in an industry continually striving for advancements, we should be studying the outliers. Only by doing so might we find guidelines for the “adverse responder”, and discover what makes the “super-responder” elite.

But why is studying the outlier so uncommon? Using an N-of-1 produces a far smaller signal-to-noise ratio. Measurement errors and interactive effects, such as how the athlete has recently eaten or slept, have a more significant impact on the outcome, potentially masking the true effect of training. Identifying the correct result requires repeated intervention, rather than a simple comparison to a control

group. Moreover, due to gaps in literature, many practitioners currently rely on arbitrary thresholds, such as a “5% change in x”, to dictate results. Such practical difficulties mean that the study of outliers requires pragmatic alternatives to current research.

No pilot was average on all ten metrics, nor are we. By ignoring the tails of the Gaussian curve, practitioners and researchers will continue to face challenges in advancing health and athletic performance. We must stop sending our pilots to die. Average kills talent.



The glass scalpel

Kate Burke

An unremarkable exchange:

WOMAN: My immune system is attacking my body; I have reams of examples.

DOCTOR: Must be anxiety, depression, your period, or you're fat and need a healthier lifestyle. Make some lifestyle changes and come back if you see no change.

REPEAT UNTIL DEATH.

Women's health has not come a long way. That may sound like a harsh statement, even ungrateful, but when modern medicine can 3D print human tissue, edit genes, and utilise animal organs for transplants, it is abundantly clear that progress is not evenly distributed. Women are an afterthought, an abnormality, a blip; something to circle back to rather than begin with. Men are the neutral, standard body while women exist as a footnote—an anomaly to be adjusted for once the norm has been established.

The prioritisation of male bodies in medicine has deep historical

roots. Western medicine has long conceptualised women's physiology as unstable or pathological, from Plato's theory of the "wandering womb" to the 20th century lobotomy, which primarily targeted female "hysteria"—hysteria coming from the Greek word *hystera* meaning womb or uterus. As a result, many

physiological processes unique to women like menstruation, pregnancy, and menopause have been devastatingly under-researched. Issues that don't appear gendered are nevertheless entrenched with division. For example, only recently has research begun to explore how immune function is shaped by sex-specific factors such as hormones, X-chromosome gene expression, and pregnancy-related

immune adaptation. Emerging evidence suggests women's stronger immune response may increase susceptibility to autoimmune disease, whilst also complicating diagnosis and treatment. These differences have been routinely ignored, and a countless number of women have had their physical symptoms attributed to psychosomatic causes, prolonging



patient suffering.

Diagnostic delays are not just uncomfortable and inconvenient but clinically significant. In diseases like lupus and multiple sclerosis, early treatment can slow progression. Yet women frequently report years-long gaps between the arrival of symptoms and being diagnosed. During this gap, their experiences are minimised or reframed as poor mental health and caused by lifestyle-related issues. Research practices have played a crucial role in reinforcing these disparities, with women being routinely excluded from clinical trials, particularly those of reproductive age, due to concerns about hormonal variability. This exclusion has produced a medical evidence base built largely on male subjects. Many drugs enter the market without adequate testing for sex-specific effects. Sodium valproate, which was prescribed for decades to treat epilepsy and bipolar disorder, provides a stark example. Despite long-standing evidence of its severe teratogenic effects, including a high risk of developmental disorders, valproate continued to be routinely prescribed to women of reproductive age with limited safeguards. It was not until 2018 that the Medicines and Healthcare products Regulatory Agency introduced strict prescribing restrictions following a substantial history of documented harm.

Inequities in healthcare are further intensified by race. Women of colour

face higher rates of misdiagnosis and significantly elevated maternal mortality. Historical myths about biological differences and pain tolerance continue to infect medical judgement. The speculum was developed through 19th century gynaecological experimentation on enslaved black women conducted without consent or pain relief. The fact it has persisted until now in near-identical form serves as a striking example of how women's pain—particularly the pain of black women—has been built into medical design and remains woven tightly into its foundations.

The cumulative effect of these failures is that women are almost expected to compensate for institutional shortcomings by exhaustively advocating for attention to women's health. This need for constant self-advocacy is often framed as empowerment rather than a garish, flashing sign that the system needs reworking.

The problem, then, is not a lack of medical capability but the claw marks on the ancient idea that only male bodies are central to medical knowledge. Until women are no longer regarded as deviations, they will continue to suffer. Progress in women's health requires not empty, vapid declarations of inclusion but a fundamental shift in how medicine defines the human body.

Are they alone? A celestial approach to contagious cancers

Sebastian Evans

Despite affecting around 3.5 million people in the UK, cancer is a very unusual disease. It happens when the body loses control of a cell, culminating in the collapse of the entire system. The body puts a huge number of measures in place to prevent this—from killing cells that behave abnormally, to having cells self-destruct if they are damaged beyond repair. So much needs to go wrong for a cancer to develop. But at least they aren't contagious...

Until they are. So far, 14 types of “transmissible cancers”, cancer cells that can infect new hosts, have been identified. Thankfully, none of these are in humans: 1 strain of transmissible tumour in dogs, 11 strains of clam “leukaemia”, and 2 strains of facial tumours in Tasmanian devils (which are driving the species towards extinction).

So much needs to go wrong for a cancer to threaten an individual. So much more needs to go wrong for a cancer to threaten an entire population. Not only does a cancer need to develop in the first place, but it must also get out of its original host before killing it, and then must survive within a new organism's body. Transmissible cancer develops from a chain of unlikely events, one after the other.

This idea might seem remarkably familiar. The development of intelligent life itself is characterised by a series of unlikely circumstances and events. So much needs to go right—from ideal conditions within a star system, to organisms figuring out how to use tools—for an advanced civilisation to develop. This string of events is so unlikely that we currently only have one example.

Us.

But that hasn't stopped people from wondering: are we alone?

In 1961, as part of the intrigue surrounding this question, Frank Drake developed a formula to estimate the number of intelligent civilisations in the galaxy. The Drake Equation defined a series of then-unknown numbers that could be used for this estimate.

$$N_c = R^* \times f_p \times n_e \times f_l \times f_i \times f_c \times L$$

Some of these are easy to find through simple astronomical observation, such as R^* , the rate of star formation. Our current technology stops us from finding others, such as f_l , the proportion of habitable planets that develop life. The issue is, we only have

a single datapoint to base our estimates on. Until we can get concrete results (positive or negative) whether other habitable planets also harbour life, we're missing fl.

Whilst unsolved, the Drake Equation still provides a useful framework to estimate how often such anomalies occur. As such, in 2020 Dujon et al. modified the equation to predict the number of transmissible cancers that have arisen on Earth.

$$N_c = N_s \times f_c \times f_e \times f_s \times f_i \times D$$

N_c = Number of detectable transmissible cancers
 N_s = Number of species
 f_c = Proportion of species where cancer is possible
 f_e = Proportion of f_c where cancer actually develops
 f_s = Proportion of f_e where cancer escapes original host
 f_i = Proportion of f_s where the cancer infects a new host
 D = Sensitivity of current detection methods

Unlike with intelligent civilisations, we have multiple datapoints. As a result, researchers could calibrate the equation. N_s is a fairly simple number to find estimates for; and as we have found cancer in many animals, the researchers assumed that f_c and f_e were both close to 1. That just left f_s and f_i , which were estimated using probability modelling under multiple scenarios.

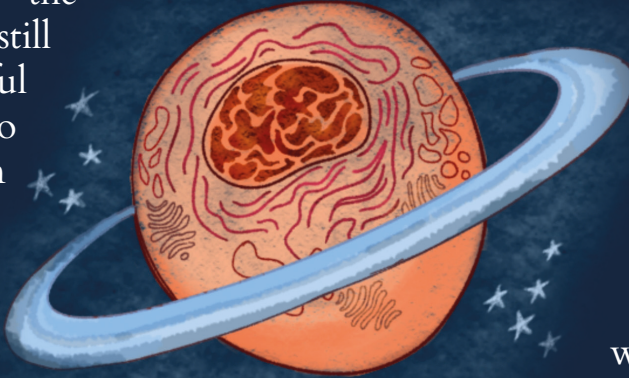
The results are concerning. Under this model, f_s and f_i could be fairly small

and still provide a number of species with transmissible cancers that is larger than the number we currently know of. Under the conditions defined by the researchers, it was extremely likely

that at least 8 species (the number of strain emergences that were known at the time) had developed transmissible cancers. Which meant that there were likely more, with estimates ranging from the tens to the thousands.

That being said, the model is primitive. For example, it doesn't consider the possibility that some of these cancers have died out. Or maybe f_s and f_i are even lower than originally estimated, making the emergence of transmissible cancers a truly abnormal phenomenon.

The Drake Equation is an interesting lens to look at anomalous events through. Even if the predictions turn out to be wrong, our attempts to understand the unknown numbers can lead to fascinating discoveries. As for transmissible cancers—let's just hope the mammalian ones don't make the unlikely jump across the species barrier.



Sleeping while awake: the anomaly of the fragmented brain

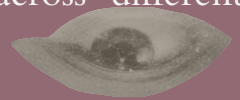
Menagaa Sarvananthalingam

There are moments in everyday life when you do not feel fully present: driving home without remembering much of the journey, or realising midway through a conversation you didn't listen to what was being said. We often dismiss these incidents as being tired or distracted, but they point to a much more fascinating concept—the brain is not always in a uniform state. Instead, wakefulness and sleep can coexist within different brain regions simultaneously.

Sleep has traditionally been conceptualised as a discrete process: you are either awake or asleep. Clinically, it can be divided into states of **wakefulness, rapid eye movement (REM), and non-rapid eye movement (NREM)**, which are defined by their characteristic patterns of brain activity. This can be measured using **electroencephalography (EEG)**, which detects changes in the frequency and amplitude of electrical signals across the cortex. However, standard EEG recordings use the average activity over large regions of the cortex (they



assume that sleep is imposed uniformly across the brain). High-density EEG recordings use many closely spaced electrodes across the scalp to increase the spatial resolution in comparison to the standard EEG. They have demonstrated that activity **varies significantly** across different cortical regions.



At the cellular level, sleep-like activity is thought to be an intrinsic property of neural networks. This is illustrated in experiments with cultures of dispersed neurons that display oscillatory rhythms, which resembles **slow-wave activity (SWA)**. SWA is characterised by high amplitude, low frequency oscillations and is the hallmark for NREM sleep. Although sleep is also regulated by interactions between the thalamus, hypothalamus and brainstem, the cortical neurons themselves can generate these oscillations independently, without an external pacemaker.

An extreme, organism-level case of this can be seen in dolphins who rely on **unihemispheric sleep**. This means one cerebral hemisphere displays slow-wave sleep, while the other half stays in a wakeful state. Dolphins cannot lose all consciousness, as they need to surface to breathe, so this enables one half of the brain to rest at a time. The key here is that global sleep is not always biologically necessary. Depending on the context, it can be more functional for the brain to work in a combination of states.

Humans do not use this model, but they use a related concept known as **local sleep**. This is supported by the uneven distribution of SWA observed across the cortex. Instead, regions that are heavily activated whilst being awake have higher SWA during subsequent sleep. For example, learning a demanding motor task increases SWA in the motor cortex compared to learning a simpler version of the same task. Similarly, tactile stimulation of one hand results in increased SWA, during sleep, in the opposite somatosensory cortex, which is the region of the brain responsible for processing sensory

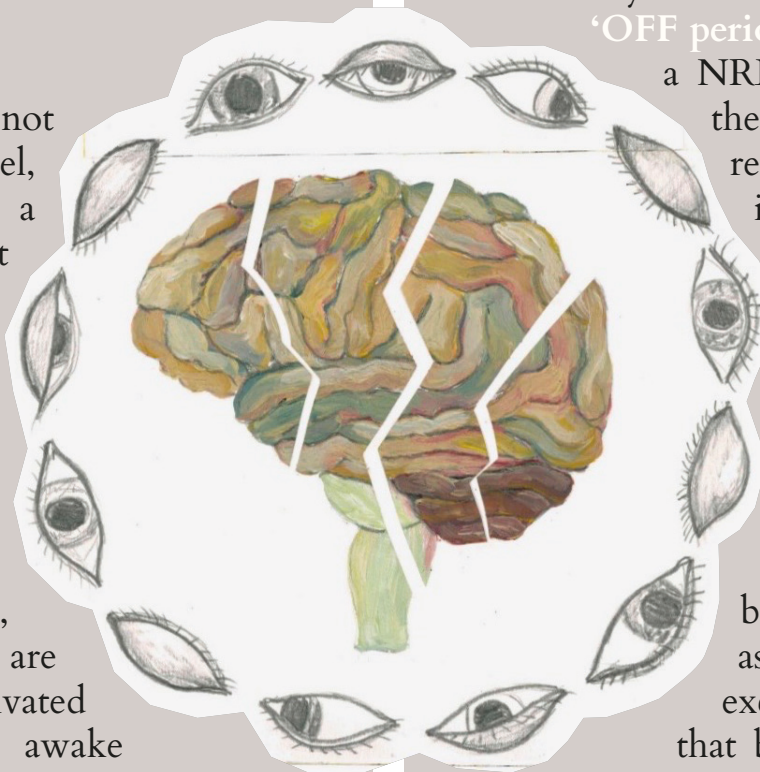
information. This again aligns with the idea that the brain is not in a uniform state, but a **combination of locally regulated states**.

Local sleep is also present while we are awake, explaining brief lapses in everyday life. During long periods of being awake, the neurons are continuously active and use increasing amounts of energy. Over time, this creates a build-up of **'sleep pressure'** within the cortical circuits. The neurons may transiently enter brief

'OFF periods', resembling a NREM state, while the individual remains awake, indicating that only certain **cortical** regions enter this state.

Wakefulness and sleep should not be viewed as mutually exclusive states that brain undergoes

globally. It instead works as a combination of local networks, each capable of transitioning between states based on their demand. In this context, **'sleeping while awake'** is not a contradiction, but a real physiological anomaly that enables the brain to recover locally when needed.



Why do **negative results** go unpublished?



Belinda Kusuma



When the results of a study do not support the initial hypothesis, where do they go? Does the initial hypothesis get revised to fit the results (known as HARKing)? Do they get published as is? Or do they get forgotten inside researchers' file drawers?

Unfortunately, it is the last fate that awaits many of these **negative, or null, results** in almost every research field. When null results are not made public, either in a formal publication or in some other form, experiments are unnecessarily repeated, wasting resources, time, and money. Moreover, the lack of published null results can cause **bias** within the field. This could ultimately lead to slower progress in research and might even cause incorrect conclusions.

There is a widely-held belief among researchers that journals are biased against negative results. Results that do not prove a new hypothesis are generally considered **less newsworthy**, except for the very few that displace long-held beliefs. Academic publishers

are businesses who, as described in Ben Goldacre's *Bad Pharma*, 'compete for readers'; therefore journals might hesitate to publish negative results. However, it is **inconclusive** whether journals are really biased against negative results, with several studies showing that there are no appreciable differences on the rate of acceptance between positive and negative results.

So maybe there is a different cause.



A survey by Springer shows that although 85% of surveyed scientists see the importance in sharing null results, 'only **68%** of those who have generated null results have shared them in

some form, with less than a third (30%) having submitted them to a journal'. This might be due to the previously stated belief that journals will **not accept** negative results. However there are other factors. Articles with negative results are cited half as often as articles with positive results. In academic environments, **high citation counts** are required for future positions and funding, so it could be more valuable and time efficient for researchers to devise

a new hypothesis and experimental design, instead of writing up a negative result. Moreover, there is also the concern that by publishing negative results, they might be viewed as lacking in either rigor or competence in their **experiment design and execution**.

Results certainly do not have to be published as a journal article to be made public. **Personal communication** between groups is one of the most popular ways of sharing negative results. Nevertheless, as research is now a worldwide activity, this method is no longer sufficient to reach every person who would benefit from the information.

There are efforts in the publication industry to include more null results, with some journals specifically geared towards negative results, or special issues in more mainstream journals. We as scientists must then contribute to this effort by **submitting our manuscripts** with null results. We can also submit our negative results to available databases and cite the articles with negative results that are useful in our studies, particularly those that inform our methodologies. Finally, **educational and awareness campaigns** on the importance of null results might also reduce bias against them.

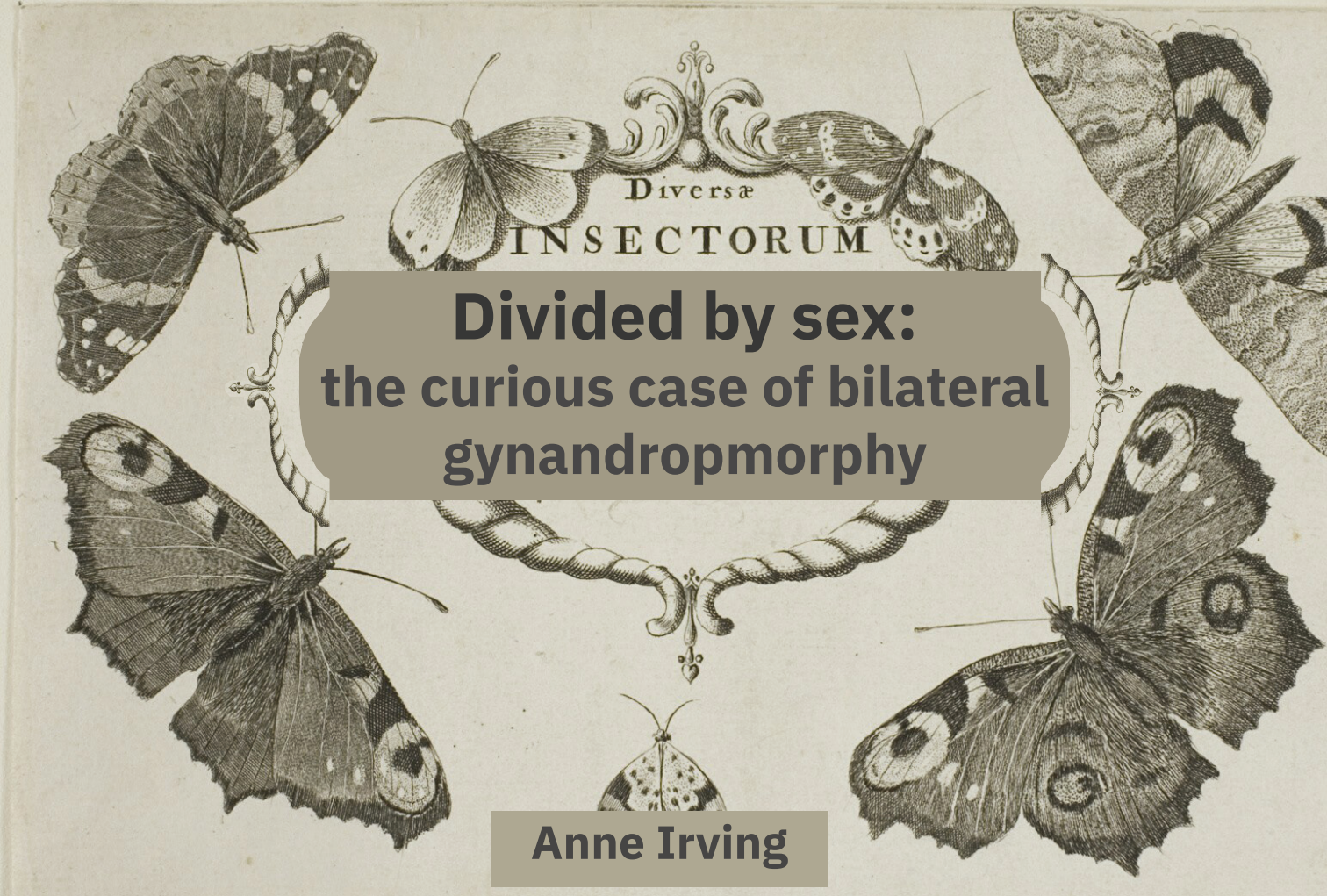
On the other hand, there is also concern that by publishing every single negative study, any useful data would be lost in the already oversaturated environment of research articles. Searchable, public databases of experiments with null results, such as the **Negatome database**, might be the answer for this, with the additional benefit of reducing the time commitment and effort of formally writing up null experiments.

There is a saying that **‘science is the process of becoming less wrong over time’**.

An important part of it is to share the null hypotheses that fail to be rejected, and the experiments that led to that conclusion.



negative results



Diversæ
INSECTORUM

Divided by sex: the curious case of bilateral gynandromorphy

Anne Irving

A rare developmental anomaly, **bilateral gynandromorphy** is estimated to occur in roughly 1 in 10,000 to 1 in 100,000 individuals of affected species. These are organisms that develop as **both male and female** simultaneously, with the two sexes divided sharply down the middle of the body. Unlike **intersex** or **hermaphroditic** conditions, where individuals have sex characteristics that are not clearly male or female, gynandromorphy can affect many tissues throughout the body—**influencing external appearance as well as internal organs**. Although cases have been recorded across a wide range of animals, the condition is most obvious in **sexually dichromatic** species, where males and females differ clearly

in colour/pattern. Therefore, the phenomenon has been most extensively studied in birds and insects, particularly the **Lepidoptera** (butterflies and moths).

The exact biological mechanisms underlying bilateral gynandromorphy are not fully understood, but several explanations have been proposed. In birds, **errors during egg formation** may produce embryos fertilised by two sperm cells containing both male (ZZ) and female (ZW) cell lineages, leading to a bilateral split. The study of a gynandromorphic zebra finch (*Taeniopygia guttata*) found that despite invariable hormone exposure, the brain was still divided between the two sexes; therefore, individual sex determination

of the brain cells controlled the final state of the brain, rendering hormones secondary.

Beyond their striking appearance, gynandromorphs provide valuable insight into development. Plebejus anna butterflies' wing patterns fall within normal male or female ranges,

but reproductive organs often develop irregularly. For the zebra finch, the song-producing region of the brain was also divided into male and female halves, impacting communication and courtship. Perhaps unsurprisingly, most bilateral gynandromorphs are infertile, due to mismatched reproductive organs. Their existence highlights the independence of different tissues during development, challenging simple ideas about how sex is determined and expressed.



From progerians to supercentenarians: learnings from the outliers of ageing

Phoebe Bedford

Ageing is an inevitable part of human life, marked by a range of changes in health and wellbeing. In high income countries, average life expectancy is now around **80 years**, but many live with significant health problems from age 65. Ageing is the main risk factor for the most prevalent diseases in developed countries—**cancer, cardiovascular disease, and neurodegeneration**. With this in mind, the main goal of ageing research is not simply to extend lifespan; instead, research aims to **extend healthspan**, the period of life spent in good health.

We all age differently, but some individuals are anomalies of ageing. For example, **supercentenarians** live to over 110 years old, whereas progerians experience accelerated ageing. By studying these groups, we can improve our understanding of the molecular and cellular mechanisms underlying ageing, potentially enabling the development of treatments for ageing and age-related diseases.

The hallmarks of ageing

The external signs of ageing result from a range of changes at the cellular level, termed the “hallmarks of ageing”.

Different researchers identify between **8 and 12 hallmarks**.

Some hallmarks develop at the **genome** level, such as genomic instability



– an increased rate of mutation during the lifespan of a cell. The **epigenome**, a set of chemical markers on DNA which controls gene expression, is also altered with age. Telomeres are caps at the ends of chromosomes, which are lost with age. These changes underlie and interact with other hallmarks, such as **cellular senescence** (in which cells stop dividing). Ageing is also accompanied by changes in the **microbiome and metabolism**, as well as increased **inflammation**.

Research in both progerians and supercentenarians underscores the importance of these processes, albeit at opposite extremes.

Supercentenarians

Supercentenarians are exceedingly rare individuals, living to over 110 years old. However, they are becoming increasingly common, especially in **high-income countries**. As well as leading exceptionally long lives, they often live independently and in good health for many years, making them excellent models of ageing.

Many supercentenarians do not lead especially “healthy” lives in terms of diet, exercise, and access to medicine, suggesting a strong genetic component underlying their unusual lifespans. For example, the FOXO3 gene has a range of functions across metabolism, immunity, and cell cycle regulation, and a particular variant has been linked to longer lifespans.

The immune system is crucial for protecting against infection,

eliminating potentially cancerous cells, and controlling inflammation. Supercentenarians show an **increase in groups of immune cells** which are mostly absent in younger groups. Rather than simply resembling the immune systems of younger individuals, supercentenarian immune systems have undergone age-related change—however, in many individuals, these changes do not constitute decline. **Specific genes** have also been implicated in reducing inflammation and autoimmunity (where the immune system attacks healthy body cells), demonstrating the importance of genetics for healthy ageing.

Case study: Maria Branyas Morera

Maria Branyas Morera was the world’s oldest living person. She was an American-Catalan supercentenarian, **aged 117** when she died in 2024.

A recent study investigated the factors contributing to her exceptional lifespan. Despite her age, Branyas showed **no evidence of neurodegeneration**; this is linked to her possession of genes related to improved cognition. She also possessed genes associated with strong immune function, cardiovascular protection, and metabolism, including a variant of a metabolic gene known to be associated with longevity. Importantly, her lifespan cannot be attributed to a single gene, but instead resulted from the presence of **several rare variants**, each with a small positive effect.

Shortened telomeres are one of the

hallmarks of ageing. Unlike other aspects of her physiology, which appeared surprisingly youthful, Branyas' telomeres were extremely short. However, because telomere shortening limits uncontrolled cell division, it could act as a protective mechanism against cancer. Branyas remained in good health despite her extremely short telomeres suggesting that they may be a marker of extreme age, rather than a predictor of age-associated disease.

When studying ageing, it can be challenging to disentangle the effects of environment and genetics. Maria followed a **Mediterranean diet, had good sleep habits, and an active social life**, and all of which are known to promote health and longevity. Her gut microbiome resembled that of a much younger individual, possibly due to her consumption of three yoghurts per day. A **healthy microbiome** is linked to anti-inflammatory responses, making it an important aspect of longevity—this highlights the importance of diet, and possibly treatments like faecal transplants, to encourage healthy ageing. Branyas also possessed a **youthful epigenome**, suggesting the potential for future treatments to target epigenetic markers.

Progerians

Progerians lie at the opposite extreme to supercentenarians, suffering from rare disorders which lead to premature ageing. The best-known progerias are **Hutchinson-Gilford Progeria**

Syndrome (HGPS) and Werner Syndrome, which affect around 1 in 20 million and 1 in 5 million living individuals respectively. In some cases, progeria refers specifically to HGPS—however, for the purposes of this article, it refers to any premature ageing syndrome.

Werner syndrome is caused by **mutations in the WRN gene**, leading to reduced genome stability. In contrast, HGPS is caused by **mutations in a lamin gene**, resulting in the production of progerin. This destabilises the nuclear envelope and disrupts processes such as DNA repair.

Progerias for the study of ageing

The significant overlap of progeria symptoms with the hallmarks of ageing makes them ideal models for **ageing research**. Because a single gene causes all the observed changes, these conditions point to links between specific molecular changes and ageing-like phenotypes, highlighting potential avenues for future research.

The link between progerias and genes involved in DNA repair and genome stability highlights the importance of genome integrity in human ageing. In contrast to progerians, Branyas' genome revealed genetic variants associated with genome stability. In both HGPS and Werner syndrome, **genome instability leads to senescence, telomere shortening, increased inflammation, and epigenetic changes associated with ageing**.

Cellular and animal models of progeria are also extremely useful for investigating ageing. Because age-related defects appear faster than in healthy counterparts, research can be carried out more rapidly in progeroid models, and may predict the effect of an intervention on normal ageing. Studies in progeroid mice found that increased DNA damage caused senescence, with further downstream consequences such as inflammation. **Senolytic drugs**, which eliminate senescent cells, are therefore a possible

avenue
for
the

treatment of age-related pathologies. Mouse models also suggest that **supplementing molecules involved in metabolic function**, such as NAD⁺, could alleviate disease symptoms associated with ageing.

Importantly, progeroid models are **not perfect**. Progerias typically arise from a single mutation, so may only mimic a subset of the hallmarks of ageing. However, they provide insight into key mechanisms underlying many aspects of the ageing process, highlighting potential targets for therapeutic intervention.

Conclusion—learnings from the extremes

The biological changes associated with growing old are driven by a complex range of cellular and molecular factors. Studying individuals at the extremes of ageing can provide insight into these complex mechanisms.

Research in both progerians and supercentenarians suggests that ageing may be delayed and reshaped across a range of pathways.

Interventions which **reduce inflammation, alter metabolism, or support a healthy gut microbiome** may not grant immortality, but they could allow more people to live longer, healthier lives.

Cosmic ray in the machine

Hani Moussa

Charged particles are constantly hitting the Earth's atmosphere, causing computer errors in anything from Mario games to aircraft systems. These errors are known as bit flips (or the switching of a binary digit) and were estimated by an IBM study to happen about once per day in 8GB of RAM—a typical quantity of memory for a personal computer. With added scale, these faults become a statistical inevitability, serving as an unfortunate consequence of computers' electronic medium.

Modern computers can hold hundreds of gigabytes of data and are composed of billions of transistors—electronic gatekeepers blocking current flow based on a voltage input. Transistors like the MOSFET can be used as switches in digital logic, allowing the calculations computers are known for.

These devices are also extensively used in memory and storage. Transistors can be seen everywhere, from long-term solid-state drives to volatile (power-dependent) data like random access memory (RAM), generally storing programs and data currently in use. Stored data can, therefore, change when transistors are struck by incoming particles.

When nuclei enter the atmosphere, they collide with the air to form new particles which can affect transistors upon impact. When charged particles strike a transistor, they leave an ionisation trail. Charge collected in this region can cause a change of state in the transistor, bypassing the typical control mechanism. A similar effect plays out with high-energy neutral particles like neutrons. These can collide with silicon atoms, thereby liberating charged particles and indirectly also creating an ionisation trail. The consequence is a switching of the expected electrical signal—in practical terms, the wrong binary digit.

Bit flips were brought to the internet's attention in 2013 when a Super Mario 64 player suddenly found the titular character teleporting across the game level. The cause was suspected to be a change in one single binary digit, part of a number recording the character's location. In this case, the phenomenon allowed the player to skip a handful of obstacles, but cosmic rays aren't always a positive. In another prominent example, a candidate in the 2003 Belgian election was given 4096 extra votes by an error blamed on cosmic rays.

A pacemaker, designed to correct heartbeat rhythm in patients, was thought to have had its data corrupted by the effects of a cosmic ray whilst on a plane. Aircraft in general are disproportionately affected by cosmic rays, for the simple reason that they are



0 1 0 0 1 0
0 0 0 1 0 0
0 0 1 0 0 1

higher up. Cosmic rays and their by-products struggle to reach the ground, and are more common tens of kilometres into the air. The consequences were clear in November 2025, when 6000 Airbus A320 planes were grounded to increase their resilience to cosmic rays. This large-scale endeavour followed multiple injuries caused by one of these aircraft unexpectedly dropping in altitude. The flight landed after being diverted, but the issue required both software and hardware updates to minimise the risk of a repeat.

The need to reduce this risk has increased over time. More performant computers typically have smaller, denser groupings of transistors, shown to more easily succumb to bit flips. Error correcting codes, often used against data transfer errors due to electrical noise, can detect the loss of data and ensure the computer

repeats its steps to retrieve it. Spacecraft electronics, which have no atmosphere to shield them from most cosmic rays, are often fitted with radiation shielding to defend against rogue particles. They may also use redundant systems to more often avoid the consequences of a bit flip—verifying the data through multiple sources. This strategy is helpful even on the ground, especially in safety-critical systems.

Even the most perfectly designed system is subject to the whims of physical phenomena. Whilst computing theory assumes ideal conditions for logical implementation, the devices that put the logic into effect are far from ideal. Events like cosmic rays can cause anomalies in the expected causal chain, creating issues in systems we depend on. Extra effort can allow engineers to mitigate (but not eliminate) these risks.

cosmic rays —

Dark matter: an incomplete explanation of the universe

Olivia Miller

The beautiful thing about anomalies in physics is this: every inconsistency reveals more than it obscures—the answers are hidden in the very problems that we tackle. The anomalous behavior of our own galaxy, for example, has revealed inconsistencies in our understanding of the universe, and ultimately pushed us to further develop our eternally-incomplete mathematical explanation of everything.

The stars at the outer edge of spiral galaxies (such as the Milky Way) move at the same speed as those close to the centre. What's the problem? Well, they should move much slower. They are much further from the galaxy's centre of mass, and therefore experience far weaker gravitational forces pulling them along as the whole galaxy rotates. In fact, those stars move so fast that they should be flung out of orbit entirely.

So, either our entire understanding of gravity—all of our formulas and theories—is wrong; or more simply, there is mass that we cannot detect, generating gravitational forces that we cannot predict. Both hypotheses, if supported by evidence, would be

groundbreaking. We're essentially proposing that either general relativity or particle physics are incomplete, if not horribly misguided. But we've been wrong before, haven't we? Mind you, we once thought that Earth was the centre of the universe.

We can't seem to find an adequate alternative to general relativity. No other model or rulebook of laws comes close. But we have found pretty convincing evidence that there is "invisible" matter contributing to these inexplicable gravitational forces (which are, of course, generated by mass; force equals mass times gravitational constant, and all that). Because it influences gravity, this matter actually bends the fabric of space, and we see evidence of this bending when light warps around places with no detectable matter while on its journey to reach us. Scientists use a technique called gravitational lensing to measure the warping effect and make maps of the invisible matter causing it. Now, we have evidence of the existence of dark matter.

And our mapping of dark matter is clearer than ever before. Just this

January, scientists at NASA's Jet propulsion Lab in Southern California published a high-resolution map created with the James Webb telescope. The map clearly depicts the proximity of dark and regular matter; the dark matter hotspots are found in all the same places as large clusters of galaxies. This visualization further confirms that dark matter pulls regular matter towards itself through gravitational force.

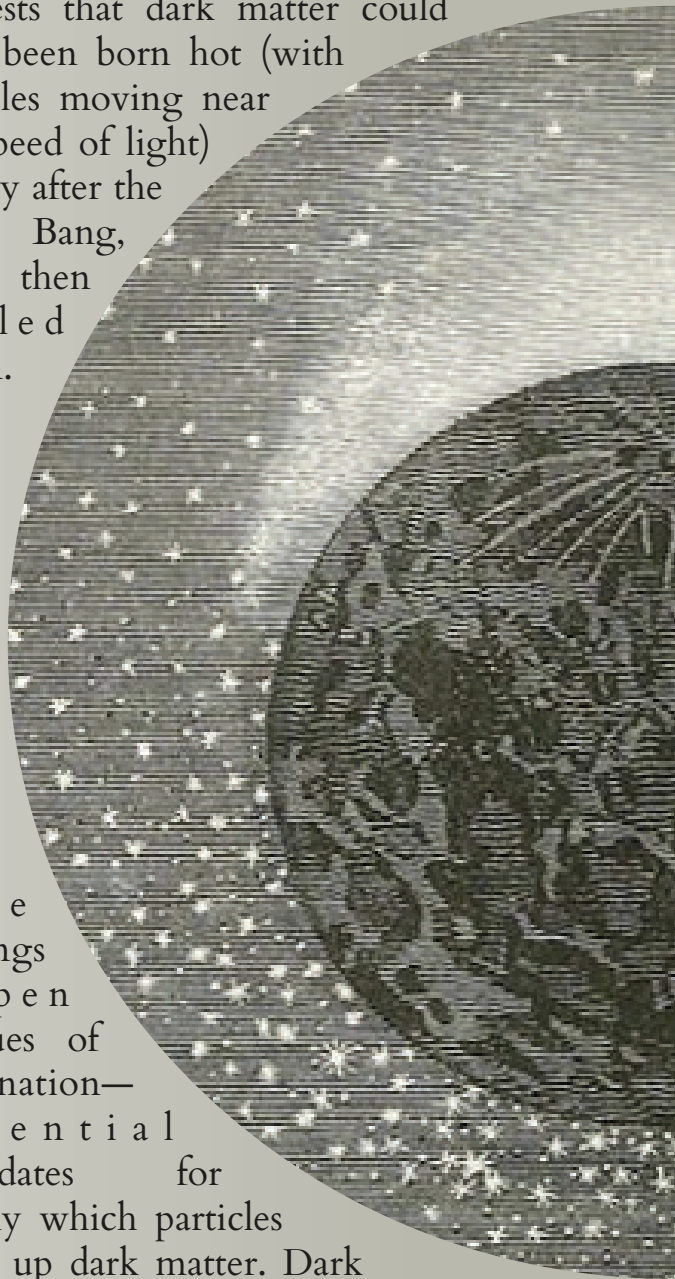
Dark matter is unique because it is not only invisible, it's also untouchable. It isn't affected by electromagnetic repulsion or attraction, which means that it cannot technically touch or be touched. It's like a ghost, and for some unknown reason, it doesn't seem to interact with any forces except those of the gravitational type.

Dark matter also informs our understanding of the creation of the universe, and its hundreds of billions of galaxies that we know today. The story goes something like this: many, many years ago, in the aftermath of the Big Bang, dark matter clumped together, and then the mass of these clumps generated gravitational forces that attracted regular matter, until there was enough material for stars and galaxies to form. Ultimately, the existence of dark matter catalysed the creation of elements, planets—the character of the entire cosmos.

Physics is never so simple, though. Its anomalies have anomalies. Until

recently, we've hedged our bets that dark matter has moved slowly throughout cosmic history; in other words, dark matter is born "cold". But new research suggests that dark matter could have been born hot (with particles moving near the speed of light) shortly after the Big Bang, and then cooled down.

The findings reopen avenues of explanation—potential candidates for exactly which particles make up dark matter. Dark matter could be the key to completing one of science's most important (and most difficult) tasks: piecing together the cosmic timeline of the universe.





How unusual is today's climate

Lessons from past climate anomalies

Jemima Dinsley

When considering our current climate, it is undeniable that human-driven rising global temperatures are presenting immense challenges. Temperatures are rising faster than societies and ecosystems can adapt to. But this isn't the first time Earth's climate has changed rapidly. Climate anomalies have occurred throughout Earth's history, often caused by volcanism or variations in solar radiation. Studying these past anomalies helps us answer a question often raised in scientific debates: just how unusual is the climate change we are seeing today?

What makes a climate “unusual”?

An “unusual” climate is defined by its magnitude and duration of the anomaly, rate of change, geographic extent, and the type of driving mechanism. Climate variability can also be understood across different temporal scales as some changes unfold over millions of years, while others happen over decades. Variability over long timescales often isn't considered an anomaly because it dominates large portions of Earth's history and short-term changes are weather responses. Most climate anomalies fall in between, bringing rapid, large-scale disruption.

The Toba super-eruption: a volcanic disruption

The Toba super-eruption in northern Sumatra occurred 74,000 years ago, and triggered a climate anomaly. As this event occurred after the emergence of anatomically modern humans, it provides interesting insights into our human response to climate stress. Volcanic eruptions result in large ejections of aerosols and gases into the atmosphere, a process which can add to the greenhouse effect, or alternatively block out the solar radiation and cause cooling. This is thought to be the largest eruption in human history, producing an ash blanket covering at least 4 million square kilometres (roughly the size of all of the countries in the EU!). Large eruptions can have catastrophic effects on global temperatures, as seen after the Mount Tambora eruption in 1815. A VEI-7 (volcanic explosivity index, with VEI-8 being the highest) eruption caused a ‘Year Without A Summer’ and caused global temperatures to fall 0.7°C cooler than average, and widespread crop failure in the Northern hemisphere.

But Toba was a VEI-8 eruption, making it at least one order of magnitude larger than Tambora. Climate models of the eruption suggest that there was a global cooling of up to 3.5°C and a 25% reduction in precipitation due to the large ejection of sulphur

dioxide into the atmosphere. There was likely ozone depletion between the tropics, which was a result of large scale atmospheric circulation changes. This would've created an immense UV index increase (less ozone shielding the surface, hence more UV) in equatorial regions. High UV causes damage to all DNA and stresses ecosystems.

Despite its magnitude, the eruption's climatic impact was temporary. Volcanic sulphate aerosols remain in the stratosphere for only a few years to decades before being removed through rainfall and atmospheric circulation. As their concentration declined, the cooling effect weakened and global temperatures rebounded. This temporary residence time is why modern proposals for stratospheric aerosol injection would require continuous replenishment to maintain cooling. Greenhouse gases contrastingly stay in the atmosphere for decades to centuries, causing long-term radiative forcing with no clear natural mechanisms for reversal. When looking at the impact of the Toba eruption on humans, small hunter-gatherer populations would have faced shrinking habitats and unreliable resources, pressures not unlike those confronting vulnerable communities today. Similarly, modern climate change is already driving displacement and food insecurity, but unlike Toba's short-lived shock, today's warming is sustained and human-driven.

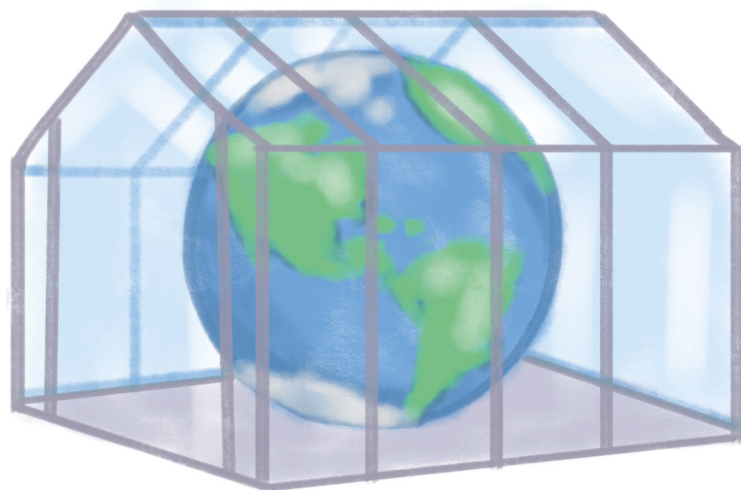
Whilst past climate anomalies such

as Toba were extreme in magnitude, they differ in terms of duration and underlying cause. Despite this, the example is worth studying as it shows that if atmospheric carbon dioxide levels are reduced or the forcing mechanism is eliminated, the climate system has the capability to stabilise over time.

Medieval climate anomaly: A misrepresented change

When looking at other periods of climate change, some turn to the Medieval Warm Period. This period, between roughly 900-1300 A.D., is often portrayed as a warming event. But recent studies suggest this was a regional event specific to the Northern hemisphere, rather than a global climate change. In addition, it appears that it was caused by natural variability in solar radiation. This makes the event less relevant in comparison to today's climate issues. Its regional nature and smaller magnitude make it incomparable to today's global warming, despite its frequent use in climate debates.

climate



A climate warning : the Palaeocene Eocene Thermal Maximum

When looking at a longer example of a climate anomaly, the Paleocene-Eocene thermal maximum (PETM) can be explored. Although this event was around 56 million years ago, it remains one of the most well researched climate events. Global temperatures rose by 5–8°C and the event was around 200,000 years long. Geological records seen in marine carbonates show how the Earth's system responded to a significant increase in temperatures. During this event, there were pronounced changes in ocean and atmospheric temperatures, ocean chemistry, and the carbon cycle. The mechanisms of these changes are attributed to elevated concentrations of greenhouse gases in the atmosphere. It is believed that the large release of carbon into the atmosphere was caused by the immense volcanism in the opening of the Atlantic along with sediments releasing methane and carbon dioxide caused by the heat from the magma. This positive feedback loop (increase in greenhouse gases due to volcanism causing an increase in methane) resulted in sustained carbon release over tens of thousands of years into the atmosphere. It took the global climate around 200,000 years to recover.

The consequences were profound. Ocean temperatures rose, oxygen levels declined, and ocean circulation patterns shifted. Some sea levels rose by over 20m, an increase which today would displace millions of people and cover significant parts of countries such as the Netherlands and many island nations. Marine ecosystems

experienced significant extinction, particularly in deep-sea environments. In contrast, mammals on land were able to expand into new habitats.

Why contemporary climate change is different to the PETM

Although rapid on a geological time scale, the PETM unfolded much slower than today's increases. Humans are currently releasing around 10 PgC/year, which is 10 times faster than the maximum PETM estimates. This means we are essentially causing changes over a couple hundred years which are usually only seen on a geological time scale. In addition, the release of carbon (albeit in the PETM was as a result of volcanism) resulted in more carbon being released due to methane-hydrate release. This means there could be associated positive feedback today that we haven't yet seen, due to the relatively short timescale of the changes.

Despite the Earth's dramatic historical perturbations, contemporary climate change is very different. This can be observed by comparing climate change with events such as the Toba super-eruption and the Paleocene-Eocene Thermal Maximum. Historical anomalies were either short-lived and rapidly reversible or occurred over timescales that allowed ecological adaptation. The speed of climate change is a central challenge facing our generation and impacting almost all species on the planet. Earth's climate has changed before, but never this fast, and never because of a single species. That is what makes today's climate truly unusual.



art by Ella-Rose Gasking

