BlueSci

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EXTREMES

Cambridge University Science Magazine

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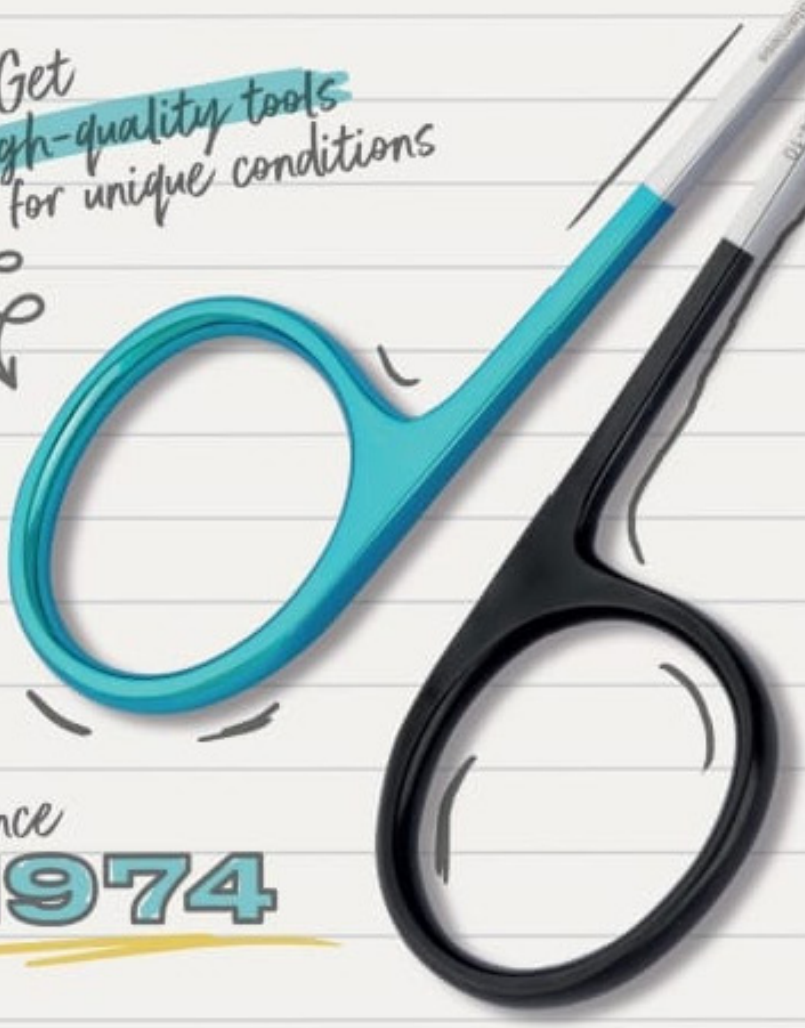

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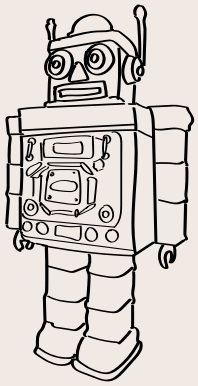
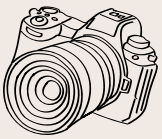




ISSUE
THEME

EXTREMES

A Message From the Issue Editor:

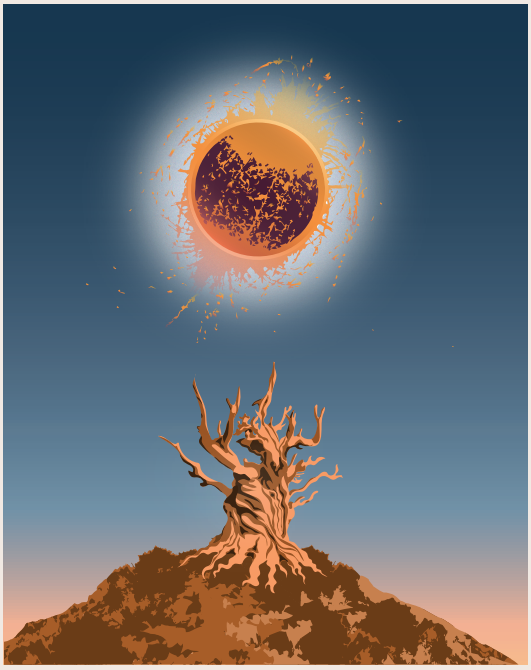
Science, more often than not, is a pursuit of the average. Experiments are repeated painstakingly, outliers excluded, all in service of a generalisable answer. Yet every now and then, a discovery emerges that shatters our understanding of what is possible, a finding so far beyond the expected that it redraws the map entirely. These outliers are worth celebrating, and the 63rd edition of BlueSci is dedicated to their stories. Welcome to **Extremes.**

We kick this issue off with a fascinating interview with Dr. Alex Cagan, touching on everything from doodling to DNA. Sara Hamaguchi then takes us through the kaleidoscopic world of synaesthesia before Courtney Kremler unpacks the cocktail of factors that provoke the human immune system to turn on itself in multiple sclerosis. Alex Barrington ponders if eons of evolution have destined males and females to clash horns, while Nicolas Branche details how those same eons have forged organisms capable of defying time itself. Moving from old to young, Timothy Lambden looks into the rise of artificial intelligence in biology - a field that, despite being in its infancy, is already revolutionising science. Sophia Belkhir and Nina Valenbreder inspect the unfortunate species plagued by transmissible tumours, which stands in stark contrast to Joshua Kocjancic Nelson's description of the few animals that seemingly avoid cancer altogether. We are then whisked from the ocean to outer space and back again: Leonid Digel details the speediest microbial swimmers; Julian Heuer asks if the building blocks of life came from beyond our planet; and Ella von Moeller shines a light on how sea creatures glow. Staying submerged, Dhruv Shenai dives into hydrothermal vents, a timely reminder that life often finds a way, as we are then plunged into darkness by Dily Duan Yi Ong, who describes the loneliest places in space - cosmic supervoids. Thankfully, Yoann Launay rescues us from this vast emptiness by taking us to one of the densest places in the early universe: primordial black holes. Whilst at the beginning of time, we find Jacob Tutt and Harry Bevins searching for a signal that might reveal when the earliest galaxies and stars formed. All this celestial travel builds an appetite, so it's fortunate that Abdullah Al Zaif then provides a recipe for cooking nuclear pasta, and having refuelled in the cosmic kitchen, we shrink down to the subatomic level with Dhruv Radhakrishnan and Aishwarya Venkatramani, who explore how quantum phenomena can be exploited by science and life, respectively. This journey takes us from blue whales and black holes to colours and cancers, demonstrating how wonder can be found everywhere we look. It's a good job, then, that Varun Rawat brings this issue to a close by guiding us through the rules that govern how complexity can arise from chaos.

The stories that make up this edition of BlueSci are those of extremes - of life that refuses to die, of forces that defy imagination, and of science that dares to ask what lies at the very edge of the possible. So, in a world that often seeks comfort in the ordinary, we hope this issue offers something a little different: an invitation to look to the edges, where the rules bend, the unexpected thrives, and science is at its most alive.

-Freddie McNay

ON THE COVER



The cover art draws on two elements that feature in the articles of this issue. In the foreground is a hill, atop which stands the world's oldest living tree, an ancient bristlecone pine named 'Methuselah', estimated to be 4,850 years old. In the background, the sky features a supernova: the explosive death of a star so massive that it collapses under its own weight, leaving behind a neutron star of incomprehensible size and density.

-Alex Neaverson

ARTISTS OF THE ISSUE

Alex Neaverson is a Postdoctorate Research Associate, who studied early neural development in the chick embryo during her PhD in Genetics at Pembroke College.

Grace Heslin is a 5th year medical student at Corpus Christi College.

Flo Studdert-Kennedy is a Part III Chemist at Trinity Hall, specialising in organic chemistry and chemical biology.

Jordan Inglis is a 3rd year materials science student at St John's.

Alexandra Kim is a first-year PhD student at TU Munich studying the biomechanics of airway cilia—the microscopic oars that row together to keep our lungs clear.

Loris Marcel is a 3rd year PhD student studying coral reefs.

Sophia Jaffer is a first-year PhD student in History, studying Ugandan Asian collective memory and place-making in Britain from the 1970s.

Nina Valenbreder is rounding off her first year as a PhD student in the Transmissible Cancer Group.
Her project focuses on investigating tumor-host immune interactions in mammalian transmissible cancers.

Anita McAuley is a medical student, currently studying for a Master's degree in Genomic Medicine.

Barbara Neto-Bradley is a postdoctoral researcher in the Department of Plant Sciences

BlueSci

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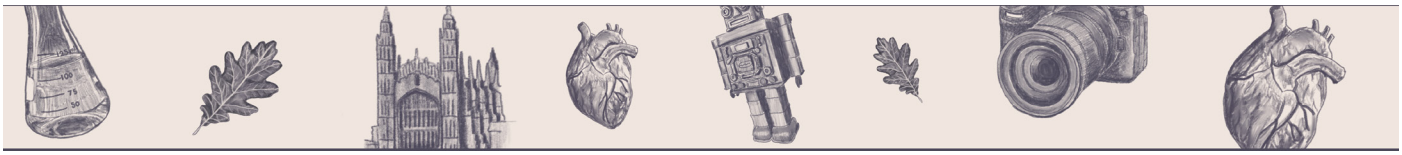

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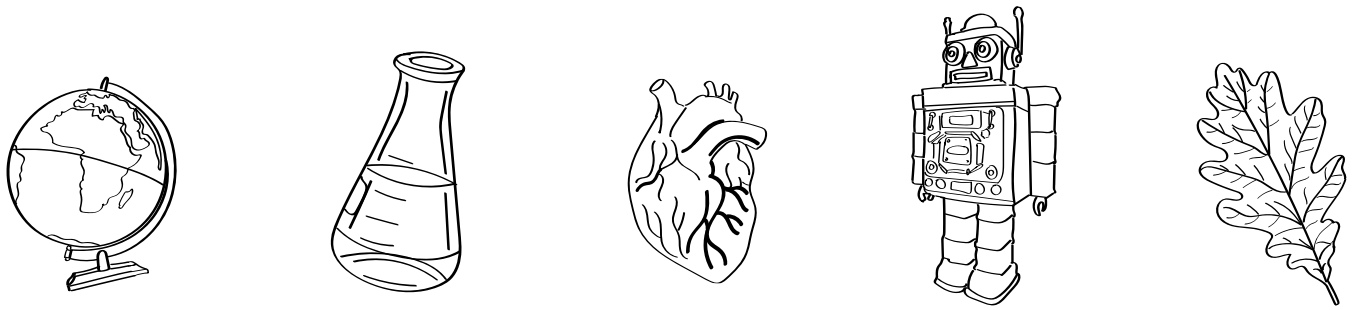
BlueSci was established in 2004 to provide a student forum for science communication. As the longest running science magazine in Cambridge, BlueSci publishes the best science writing from across the University each term. We combine high quality writing with stunning images to provide fascinating yet accessible science to everyone. But BlueSci does not stop there: at **www.bluesci.co.uk**, we have extra articles, regular news stories, podcasts and science films to inform and entertain between print issues. Produced entirely by members of the University, the diversity of expertise and talent combine to produce a unique science experience.



Thank you to everyone involved in the production of this issue.

Issue Editor: Freddie McNay

Managing Editors: Daisy Gordon, Emma Powers, Jessica Tang

Subject Editors: Andrea Rogolino (Physical Sciences), Gabriel Trujillo (Biological Sciences)

Editors: Emily Naden, Maya Juman, Maggie Szymanska, Mayha Fazel-Zarandi, Kristen Burgess

Art Editors: Alex Neaverson, Grace Heslin

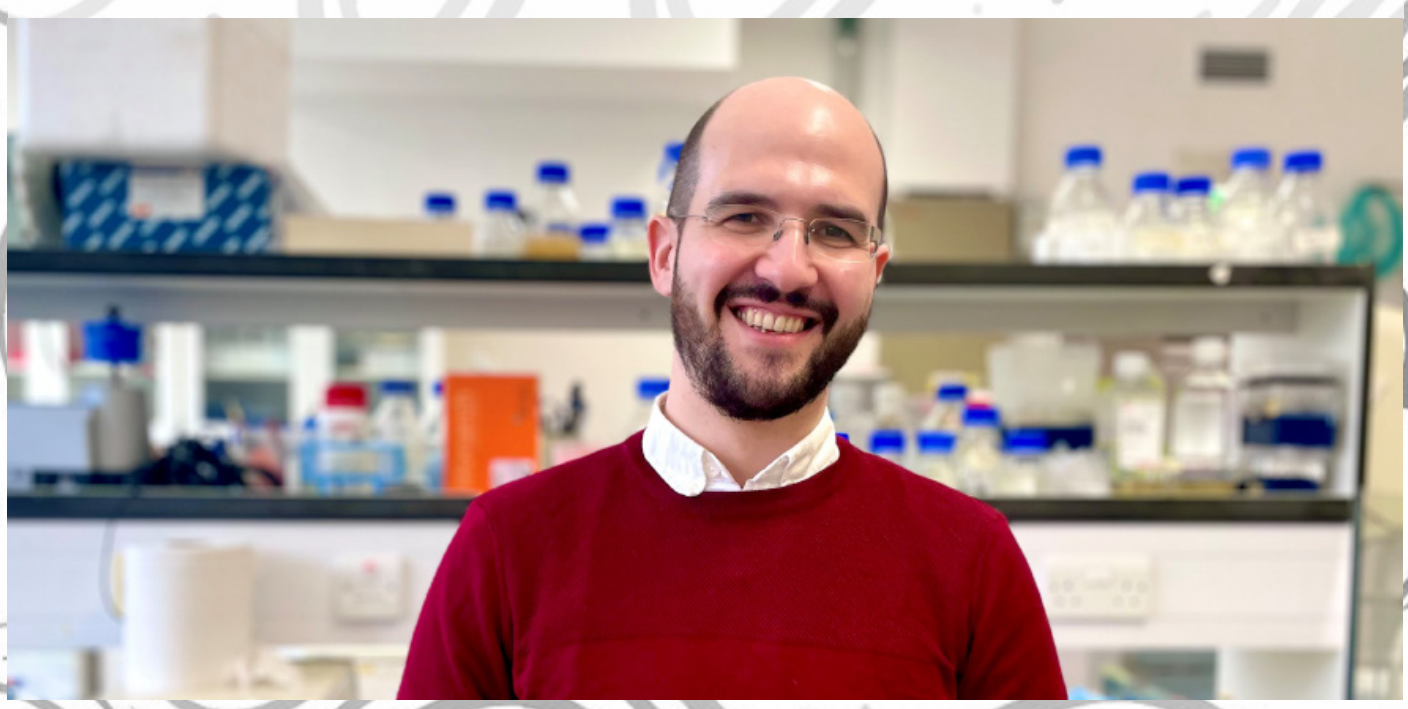
Authors: Sara Hamaguchi, Courtney Kremler, Alex Barrington, Nicolas Branche, Timothy Lambden, Sophia Belkhir, Nina Valenbreder, Joshua Kocjancic Nelson, Leonid Digel, Julian Heuer, Ella von Moeller, Dhruv Shenai, Dily Duan Yi Ong, Yoann Launay, Jacob Tutt, Harry Bevins, Abdullah Al Zaif, Dhruv Radhakrishnan, Aishwarya Venkatramani, Varun Rawat

Artists: Alex Neaverson, Grace Heslin, Flo Studdert-Kennedy, Jordan Inglis, Alexandra Kim, Loris Marcel, Sophia Jaffer, Nina Valenbreder, Anita McAuley, Barbara Neto-Bradley, Ashlyn Chew

A special thanks to Dr. Alex Cagan

THE INTERVIEW

DR. ALEX CAGAN



What do live illustration and the genomes of bowhead whales have in common? For Dr. Alex Cagan, they are all part of the same pursuit. Recently appointed as a group leader in the Department of Genetics at Cambridge, Alex researches evolutionary processes in somatic tissue, seeking to understand how these contribute to cancer and ageing — work that often leads him to some of the most extreme organisms on the planet. But Alex is far more than just a scientist: he is internationally recognised for his live drawings of science talks, and has illustrated covers for journals such as *Nature* and *Cell*.

For this edition of BlueSci, the team sat down with Alex to explore his artwork, his science, and how he brings these two seemingly separate worlds together.

Q: How did you get into art and how did you get involved with art in a science context?

My parents were always artistic. My dad was an actor who grew up exposed to humanities and art and eventually became a lawyer. My mum is a set and costume designer, so the house was always full of her sketches of costumes and sets. She'd make little models of sets for TV shows, like The Fimbles. It was kind of like osmosis–I would get taken around to the sets she had worked on and art would be left on a polystyrene block. My mum always encouraged me to draw.

Growing up, I always felt like there was a separation between art and science. I did enjoy biology when I was little, but once I got older - around when I did my A-levels - ironically, what really put me off was genetics. I loved animal behaviour, but then suddenly we were learning about Punnett squares. I thought "what's this got to do with all the interesting stuff, like the David Attenborough documentaries?!". I had the thought of going to a foundation art school, but I decided to do anthropology because I was fascinated by history, cultures, and how humans differ. I studied that here in Cambridge, where we had to take biological anthropology and archaeology. That led me to rediscover all the interesting biology stuff, like primate behaviour and human behaviour, and everything clicked.

I remember I was taking notes during lectures, and then I'd be asked to write an essay. I didn't want to write the essay, so instead of doing that, I would make an A3 illustration summary of the topic. In response to my illustration, my supervisor was like, "This is different - it's fun". So, I kept doing it.

Q: Your illustrations on Twitter/X are quite popular! Your account has more than 12.5k followers (as of March 2026). How did you get started with that?

After undergraduate, I eventually went off to do a PhD in

Germany. For the first few years, art and science were completely separate. I would be drawing and doodling, but not linking it to the sciences.

Then I met a journalist in Svante Pääbo's Lab. The lab constantly had people from the press coming to interview him about Neanderthals. I don't know how I got chatting to them about something — either they saw some drawing I'd done, or I was chatting about it — but they were like, "Oh, you should do drawings and post them on Twitter" (now known as X). So that was where the idea came from.

That was all new to me. So I went to a science conference with a mini iPad and I started drawing some of the talks. There are people who live tweet talks. I was thinking, you can kind of cheat the word limit if you do a drawing, because it's just one image that kind of summarizes the whole thing at once.

Q: What were your initial thoughts on mixing art and science in your career?

Not that long ago, I thought maybe you shouldn't mix the two, because people expect a certain type of science, and if you bring art into it, you might get a credibility issue and people think, oh, it's not really science.

Now, I'm completely sold the other way from all my experiences. It's really useful and they both support each other.

But that was definitely a concern that I had, especially when I was young, trying to get trained up as a scientist. You don't want to go outside the box, but actually, that niche is a strength, not a weakness.

Q: Did the drawings immediately take off? Did people love them right away?

Even now, I still feel like I'm still learning and trying to improve them, but they're definitely so much better than when I started. The early ones were terrible, and they're all still out there on Twitter (now X).

But the feedback I got was so good. I think it's because, at the time, I was the only person who was doing these illustrations, and people love drawings of themselves or about them.

Q: Do you ever feel nervous or anxious about people watching you while you do the live illustrations?

Yeah, every time, still. I normally try to sit somewhere out of the way, but I know people sitting behind me love watching and find it fascinating. It's one of those things where you think it's terrible, but other people really enjoy it. But it definitely plays on my mind.

Q: Do you feel like your artistic style has changed a lot over the years?

Yeah. I find it weird to say the words "artistic style", because I still feel like I'm just doodling, but the way my art looks has definitely changed. It wasn't a conscious thing, but in doing lots of drawing I guess you see which things work and which don't, and from there it evolves.

Q: Have you ever got negative reactions from people you're drawing?

I've had someone tell me that I didn't make my supervisor muscly enough, and someone else told me that I made their partner's neck too thick. I try to be flattering, but when you're drawing people it's quite risky!

Q: Do you have any experience of your art reaching people from a non-scientific background?

I do end up mostly hearing back from scientists, but I often hear from scientists they really liked the drawings as a way to show their non-science friends and family what they research, in a way that doesn't put them to sleep.

I've also done events that are science adjacent and involve patient partnership, such as Cancer Research UK. Organisations like that are always looking for ways to communicate with the public, and I think art is one of the best ways to do that.

Even then, what really matters is having a good story or narrative to communicate to people.

Q: Do you feel like your science communication work has directly helped your research skills and career?

Definitely, in lots of different ways.

It's been great for networking, which I don't really like, but now when I'm drawing at conferences people will come up and talk to me, instead of me having to awkwardly introduce myself.

It's also been great for my mental health. Science can be grueling — you work for years just for people to tell you what you did wrong — but with my art I've always got immediate positive feedback, which is really nice.

I was a bit worried about getting pigeon-holed as "the drawing person", and people forgetting I do science too, but now I'm really happy with the balance and how it's worked out.

> "Science careers offer a lot of options, and if you can bring some additional skill or creative dimension to the table, that combination puts you in a really unique position."

Q: Do you have any advice for someone looking to get into science art or communication? Do you think it's possible to make a career from it?

I'd really recommend two books on this: 'If I Understood You, Would I Have This Look on My Face?' by Alan Alda and 'Don't Be Such a Scientist' by Randy Olson. Both have great advice on connecting with a non-scientific audience.

As for making a career from illustration, I think people absolutely can, but I just use illustration to complement my research. During my PhD and postdoc I did consider going into science illustration full-time, but ultimately I realised I enjoyed the research too much to give it up.

One piece of advice that really stuck with me is: in your career, you can either try to be the very best at one thing, but then

you're competing with an enormous number of people in that space, or you can be quite good at two very different things, and suddenly you become one of the only people who combines those two skills.

That's really how it's felt with illustration and science for me. Having the ability to do both suddenly makes you far rarer than being either a great illustrator or a great scientist on their own. Science careers offer a lot of options, and if you can bring some additional skill or creative dimension to the table, that combination puts you in a really unique position.

Q: Could you tell us about what your group researches?

We study somatic evolution — the evolutionary processes that happen within our own bodies as we age. We're interested in how our cells deal with DNA damage, and when they fail to do that, they accumulate mutations. But we're not just interested in the mutations themselves; it's the fate of those mutations that matters. Which groups of mutant cells expand, and which ones don't. It's like these competitions between cells playing out across evolutionary landscapes inside the body.

My lab's particular niche is applying these methods across different species to get a comparative biology perspective. Evolution has essentially been running cancer prevention trials for millions of years: species like elephants and naked mole rats have really low cancer rates, but we don't really know how they do it. By mapping these mutational landscapes in those species, we hope to get clues, and maybe one day apply those to humans.

We have three main areas of focus:

- Ageing, by looking at species with very different lifespans
- Cancer resistance in those outlier species
- Using mutations as biosensors for environmental monitoring, a bit like a canary in a coal mine — different pollutants produce particular mutational patterns, so by looking at wild animals we can identify regions of concern and understand how those pollutants are impacting health.

"Evolution matters at so many different scales in biology, and it's mindblowing to think one of those is within your body."

Q: The theme of this edition of BlueSci is 'extremes'. Do you have a favourite extreme organism relating to your work?

My research is all about looking at extremes, whether that's species with extreme cancer rates or extreme lifespans. Allegedly immortal organisms like hydras are obviously pretty extreme, but I find the extreme lifespans within species closer to us, like mammals, really amazing.

Bowhead whales can live for over 200 years, and the way scientists have worked out their age is extreme too: by finding harpoons still embedded in their bodies, that can be dated to a particular period in the whaling industry, and therefore used to estimate the age of the whale. These animals survived a harpoon strike, swum around for over a century, and were still alive when they were caught.

Q: You study some pretty extreme and unique organisms. Do you ever face scepticism about whether it's worthwhile, and how do you respond?

When we're looking at things like ageing or cancer, it makes sense to research humans first. I wouldn't say all cancer research should be looking at naked mole rats, and if I was only funding topics on one thing, then we should focus on humans. But that's not the case, and I think the amount we can gain from looking at these naturally evolved solutions is enormous.

There's a quote from E.O. Wilson — "Biodiversity is our most valuable but least appreciated resource" — that really sums up how I feel about it. There's so much incredible diversity out there that we could learn from.

Q: What would you say have been the standout moments of your career, good and bad?

My mind immediately jumps to getting a paper published, but that always feels like more of a relief than a highlight by the time it actually happens! I think the real highlights come more from the day-to-day research, when you're busy but not overwhelmed.

One moment that stands out is when we first tried to detect somatic mutations across species. It was an ambitious idea and we weren't sure it would work: the methods were hard enough in humans, let alone other animals. We set up a collaboration with London Zoo, got our first tissue sample from a tiger, then laser-captured and sequenced it. The mutational pattern came back looking almost identical to a human colon, which is unlikely to be caused by error, and that moment of thinking "oh, this might actually work" was pretty special.

As for low points, during my PhD I wrote a small paper that got absolutely torn apart by the reviewers. I think I've mostly screened it out, but I remember one saying something like "this makes no useful contribution to the field and the methods are terrible". And now, running a group, you get used to the reality that so few grants get funded. You just have to build up a thick skin. It's not fun, but it's part of it.

Q: If you could communicate one concept from your research across to a general audience, what would it be?

Somatic evolution: the idea that evolution doesn't only happen at the level of species or populations, the way we normally think of it with Darwin's finches, but also happens within our bodies. That's what drew me to this research in the first place; it blew my mind when I realised that the same rules of evolution that we apply to species are playing out inside our own bodies, with our cells competing against each other throughout our lifetimes. This concept changes the way you think about yourself: you're not just an individual, you're a population of evolving cells.

And it's not just fun to study: these processes can determine your health as you age, and even the health of your children, because somatic evolution can happen in the cells that give rise to sperm and eggs too. Evolution matters at so many different scales in biology, and it's mindblowing to think one of those is within your body.

FOCUS

EXploring synaesthesia and cross-modality:

insights from non-human primates

Artwork by Jordan Inglis

Sara Hamaguchi explores the world of synaesthesia, starting with her own experience before venturing into evolutionary biology, animal cognition, and the intriguing possibility that we may not be alone in blending our senses.

'A' is red, but 'red' is brown, green and orange. Number '4' is a pink, easy-going young girl, while '8' is a green, mature, gentle man. The number sequence '3434' in a maths problem evokes a clear image of the pink petals from cherry blossom flowers and yellow-green leaves blown away by the wind.

This sounds literary, but I did not create it. At the age of 13, when memorising long paragraphs from Hamlet by Shakespeare in preparation for an English recitation contest, I realised I had been memorising quickly by feeling a colour from each letter and remembering the colour layout on the script. I talked about this perception to my friends, and soon noticed it was happening only to me. That was the moment when I first identified myself as a synaesthete, despite having such experiences throughout my whole life. The colours I introduced earlier were just a few examples - I have a colour for almost every single alphabet, Japanese letter (i.e. for each kanji, hiragana and katakana), and number. Rather than physically seeing colours, I strongly feel that I know what colour each letter or number should be. Number sequences sometimes show movies or images, like the '3434' example.

Chapter one: What is synaesthesia?

Definition, variation, and benefits of studying synaesthesia

My experience provided above is just one example of what synaesthetes may come across. Because just the term 'synaesthesia' encompasses a variety of different experiences, a straightforward definition is difficult. Julia Simner, one of the leading researchers in this field, defines synaesthesia as when one sensory experience is accompanied by an automatic secondary sensory experience. Regardless of the type of synaesthetic experience, what each synaesthete perceives commonly remains consistent over time - for example, if the letter 'A' is red, it is red for that person any time.

Some of the different synaesthetic experiences include grapheme-colour (seeing colours of letters and/or numbers), lexical-gustatory (feeling the tastes of words), sound-colour (seeing colours when hearing sounds), flavour-colour (seeing the colours of tastes), and visual-auditory (hearing sounds when seeing silently moving objects). These examples are non-exhaustive: one study shows that there are about 130 types of synaesthetic experiences. The frequency of such traits is estimated to be found in 4.4% of the population, though it is difficult to measure the true number of synaesthetes due to its subjective nature.

Studying synaesthesia is expected to provide insights into various areas, such as how perception of the world differs between individuals, how different parts of the brain are connected, and how memory can be enhanced through cross-modality (the interaction between information of different senses). Although synaesthesia affects only around 4% of the population, people without it also sometimes connect different senses, such as perceiving a colour as 'loud'. This raises the question: does the rarity of synaesthesia simply mean synaesthetes are an 'extreme' population, or are they experiencing the 'extreme' end of a spectrum that everyone sits on? Furthermore, are humans the only species to experience such connected senses? If we share synaesthesia, or cross-modality, with non-human species, what does this tell us about its evolution?

Is what synaesthetes say real?

Since synaesthesia is such a subjective experience that is different between individuals, some might wonder whether what synaesthetes say is real, or if it is one of 'I can see a ghost' types of claims. There is much experimental evidence, however, in support of synaesthetic experience. For example, Palmeri et al. (2002) compared the consistency of the colour association with 100 monosyllabic words between a grapheme-colour synaesthete and 22 undergraduate volunteers. Over the 2-week period of experiment, the synaesthete showed 97% consistency, while volunteers were consistent to 43% on average. Moreover, the visual search efficiency was shown to have increased due to a synaesthetic effect: the experiment involved measuring the speed at which the grapheme-colour synaesthete found a number from a set of similar-fashioned numbers. This participant sees 2 as orange, 5 as green, while both 6 and 8 are blue. The result was that they managed to find a 2 from 5s faster than finding an 8 from 6s, implying that these different synaesthetic colours helped the number stand out. These results imply that what synaesthetes say is not a mere imagination.

The discussion from here on focuses on developmental synaesthesia, where synaesthetes have had synaesthetic experiences from as far back as they can remember, and cannot explain how they might have learnt such perception. Nevertheless, some people argue that synaesthetic perception is learnt. Some experiments show that synaesthesia can be induced, and participants became able to temporarily perform at a similar level to synaesthetes on relevant tests. In one such study, Bor et al. (2014) trained participants to associate a colour with each of 13 letters, 30 minutes a day over 9 weeks. By the end, participants could recall a colour from a letter or vice versa, and also exhibited Skin Conductance Reactions (SCR, a measure of sweating on the skin in response to stimuli). After repeatedly pairing a loud sound with a blue square, participants showed SCR just by seeing the letter 'b', which they had learned to associate with blue. However, this cannot explain why children who grow up in the same environment, such as siblings, develop different types of synaesthetic experience.

If synaesthesia is developmental, what causes the difference between synaesthetes and non-synaesthetes? Early brain studies of synaesthetes in the 1990s revealed that the mechanism of such a trait can be understood through investigating the brain activities. The application of fMRI (a non-invasive imaging technique that measures activity of different parts of the brain during a particular activity) to a lexical-gustatory synaesthete showed the activation of the gustatory cortex (the brain part that processes taste perception) when listening to words. A similar observation was made by Julia Nunn et al. (2002), where region v4, which is involved in colour vision processing, was actively working in the brains of grapheme-colour synaesthetes when hearing words, rather than when hearing beeps. This result was not obtained from control individuals without synaesthesia. Therefore, it can be considered that differences in brain activity cause synaesthesia.

There are many theories trying to explain why the co-experience of multiple senses occurs in the brains of synaesthetes. For example, there is a debate about whether synaesthetes have more connections between two areas of the brain, such as gustatory and lexical regions (structural difference), or the number of physical connections is the same but information flows more freely (functional difference). Briefly, a structural difference is supported by the method of DTI brain imaging, while a functional

difference implies the links between two regions are disinhibited in synaesthetic brains. The disinhibition hypothesis is supported by the fact that different sensory regions in everyone's brain seem to be connected. One study involved blindfolding participants for 5 days as they undertook different tasks, such as touching objects, while their brain was scanned by fMRI. While the visual region of the brain was not activated at the beginning of the study, it became activated by the end during touching-object tasks, suggesting somatic and visual regions of the brain have connections in the first place. This poses the possibility that synaesthetic brains have these connections disinhibited while non-synaesthetic brains do not. Since there is evidence for both sides (and limitations to each) no single answer has been reached to fully understand the mechanisms behind synaesthesia.

Chapter two: Heritability and evolution

Is synaesthesia heritable?

Early studies on the heritability of synaesthesia showed that synaesthetes tended to cluster in a family. Initially, studies at the end of the 20th century suggested that synaesthesia was linked to the X chromosome due to the absence of male-to-male inheritance and larger number of female synaesthetes than male. However, meta-analyses in the early 21st century showed that the female:male ratio of synaesthetes is much closer to 1:1, and the study by Asher et al. (2009) suggested linkage to chromosomes 2, 5, 6, 12 and 16, rather than X-chromosome linkage.

On the other hand, the twin study by Bosley and Eagleman (2015) showed 73.9% of monozygotic twins and 36.4% of dizygotic twins had both individuals with coloured-sequence synaesthesia. Although this still supports heritability of synaesthesia, the fact that not 100% of monozygotic twins shared the same condition means the genetic element cannot fully explain why synaesthesia arises. They argue that other factors, such as epigenetic modifications (reversible changes in gene expression by switching on/off certain genes without changing the base sequence) and environment could also play a significant role. It can probably be said that while synaesthesia is likely heritable with a major genetic predisposition, other external factors also determine the condition.

Can evolution explain synaesthesia?

If synaesthesia is heritable, it is possible to consider that it might have had evolutionary advantages. Very briefly, natural selection refers to the long-term process of adaptation by living organisms. Random mutations give rise to genetic variation in the population, and individuals with advantageous alleles survive long enough to reproduce to produce offspring, passing their alleles on. Over generations, the frequency of advantageous alleles increases while disadvantageous alleles are negatively selected. Does natural selection explain why synaesthesia exists?

Some scholars have attempted to explain synaesthesia from evolutionary perspectives. For instance, Ward (2019, p. 8-10) argues that synaesthesia is generally advantageous in evolution, not because its perception bears information, but because the brain type of synaesthetes has different functions, and synaesthetic condition itself is functionless but it is caused by other advantageous functions. The advantageous functions of synaesthetic brains include creativity deriving from connecting seemingly unrelated senses or concepts, increased memory efficiency, and better perception. In terms of survival chances, hunter-gatherers, for instance, would benefit from these abilities for food acquisition, which is associated with increasing chances of survival. Therefore, synaesthesia could have evolved as a by-product of the brain type with beneficial functions for survival.

Chapter three: Insights from non-human species

In further exploring the origin of synaesthesia, it is useful to look at non-human primates and whether they also have synaesthesia or not. However, as introduced earlier, synaesthesia is not caused entirely by genetics, and some variants of synaesthesia involve human-specific concepts like letters, so it is difficult to conclusively determine whether non-human primates (and other species) have synaesthesia or not. Therefore, an important and closely-associated concept with synaesthesia called 'cross-modal correspondences' is studied in non-human species. I will first introduce why it is beneficial to study non-human primates when discussing the origin of such conditions in humans, and discuss what cross-modal correspondences are, before moving on to case studies from non-human species.

Why should we study non-human primates?

When looking for the origin of a characteristic, studying the closely related species to find whether the characteristic in question is present or not is useful. If it is present, it means the characteristic is not specific to humans, suggesting it evolved before the divergence from a common ancestor. For example, Chimpanzees (Pan troglodytes) are the most closely related species to humans, which diverged from our common ancestor about 6 million years ago. If both humans and chimpanzees have the same trait, then it is likely that the trait emerged before the Last Common Ancestor diverged.

Synaesthesia and cross-modal correspondences

Cross-modal correspondences refer to the association of different sensory modalities in response to a stimulus in a way that people intuitively prefer. For example, an investigation of the 'bouba/kiki effect' involved showing two visual shapes as Figure 1 to participants, and asking which they think is 'kiki' and which is 'bouba' in Martian language. Interestingly, 95% of people answered the left shape as 'kiki' and right as 'bouba', reflecting the interaction between visual and auditory modality. It is considered that the figure on the left resembles the sharp inflection of the tongue and phones when pronouncing 'kiki', which is why most people associate these together.

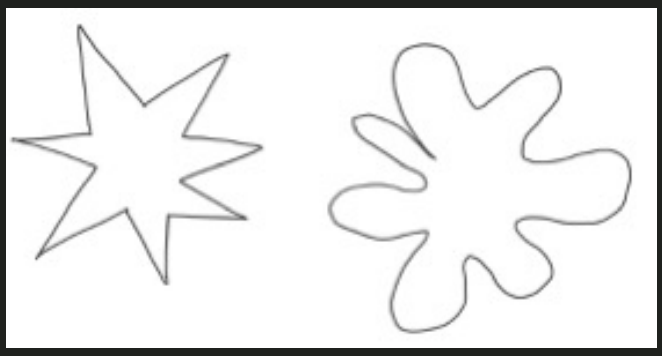


Figure 1: 'Bouba' and 'kiki'.

This example of association between visual and auditory sensations suggests that cross-modality is close to synaesthesia in definition. Such cross-modal perceptions are defined as 'weak synaesthesia' by some scholars, so it may be said that almost everyone has synaesthesia to an extent. However, the distinction between 'weak synaesthesia' and 'strong synaesthesia', or whether these two can be clearly distinguished, are matters of debate.

This might even imply that synaesthesia is simply an 'extreme' of the cross-sensory spectrum, rather than synaesthetes having completely different ways of perceiving the world. Hence, for the purpose of this discussion, 'weak synaesthesia' primarily refers to cross-modal perception (or association of different sensations) in response to a stimulus that is often common across populations. On the other hand, 'strong synaesthesia' is also cross-modal, but it is developmental, the secondary sensation automatically comes as a clear experience, perceptions are usually specific to each individual, and the experience is consistent for the individual synaesthete's life.

Synaesthesia and cross-modality in non-human primates

The question of whether non-human primates also have synaesthesia or cross-modality is first addressed by Ludwig, Adachi and Matsuzawa (2011). They investigated pitch-luminance mapping, which means associating high pitch of sound with high luminance (the use of white, high intensity of light in this case), in chimpanzees. They used 6 female chimpanzees and 33 human participants (with 20 of them being female). The subjects were asked to classify either a black and white square (stimuli) as quickly as possible, while a background noise, either high or low-pitched, is played. They found that both chimpanzees and humans performed worse (slowly) when the pitch of the noise and the luminance of a square did not match (i.e. either high-pitched sound and black square, or low-pitched sound and white square). This demonstrates that chimpanzees associate pitch and luminance in the same way as humans do - low-pitched sounds with darkness, and high-pitched sounds with luminance. The fact that chimpanzees also associate different sensations spontaneously, at least in the case of pitch-luminance mapping, suggests that cross-modality evolved before the divergence of humans and chimpanzees. This result does not automatically mean that chimpanzees, or non-human primates, also have synaesthesia; yet, the ability to relate two seemingly unrelated sensations is shared across species.

Cross-modality in associating auditory and visual signals is illustrated by Ghazanfar and Logothetis (2003). They showed 11 rhesus monkeys (Macaca mulatta) two facial gestures on a screen while playing two calls - one is a 'coo' call, used in a social context, while the other is a threat call used in agonistic encounters. As a result, all of them spent much longer time looking at the screen when the facial gesture and voice matched, rather than when they played a non-matching call to the facial gesture. This demonstrates that rhesus monkeys also relate visual and auditory sensations, suggesting that they potentially have similar cross-modality as humans.

Using a similar methodology, Sliwa et al. (2011) played voices of familiar individuals of conspecifics and humans to 6 rhesus monkeys, while showing either matching or not matching pictures of faces. The result was that they spent longer time looking at faces when they are accompanied by matching voices, further corroborating the idea of the cross-modal association of visual and auditory stimuli. The researchers consider that this cross-modal recognition illustrates their adaptability to identify socioecologically related individuals.

Such visual-auditory associations are best illustrated in humans by speech-reading, where humans interpret facial gestures while listening to the voice to support comprehension. Interestingly, humans observe the eye regions rather than the mouth while speech-reading. Ghazanfar, Nielsen and Logothetis (2006) tested the eye movement of four rhesus macaques while they were watching videos of calls and facial expressions produced by individuals of the same species in the same colony. Similar to humans, the monkeys spent more time observing the eye region rather than the mouth. This implies that rhesus monkeys share a similar strategy with humans when interpreting vocalisations by associating them with visual clues, further supporting the idea that humans and non-human primates share similar brain mechanisms for cross-modality.

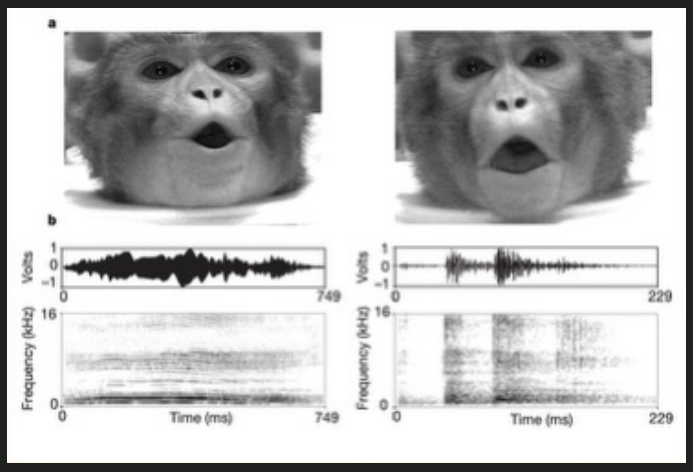


Figure 2: The facial gestures and calls used in Ghazanfar and Logothetis (2003).

Evidence from non-primate species

Even more surprisingly, studies of non-primate species, such as horses and dogs, also provide evidence for cross-modality. Proops, McComb and Reby (2008) used 24 domestic horses (Equus caballus) in an experiment which made horses watch other horses pass in front of them while playing a call from either the matching or a non-matching horse. The horses overall reacted faster and looked at the horse passing in front of them when the call did not match, probably due to the mismatch with their expectation.

A very similar result was obtained from domestic dogs. Adachi, Kuwahata, and Fujita (2006) showed 28 domestic dogs (Canis familiaris) the picture of the face of either their owner or a stranger immediately after playing one of two voices. The congruent condition involved showing and playing the picture and the voice of the same individual, while the incongruent condition mismatched the combination. The result indicated that dogs looked at the monitor of pictures longer in the incongruent condition, which is likely because the faces they saw after listening to the call were unexpected. The researchers suggest this demonstrates not only the cross-modal association of visual and auditory stimuli, but also the fact that they generate a visual image based on a voice.

Both of these results illustrate the use of cross-modal recognition in non-primate species. This suggests that cross-modality, the basis of synaesthesia, has a much older origin in the evolutionary history of animals.

Chapter four: Insights from non-human species into synaesthesia, cross-modality and language evolution

So far, we have explored what synaesthesia is, whether it can be explained by genetics and adaptation, and the evidence of cross-modality in non-human primates and non-primate species. For clarification, here is the list of what we have learnt from the three chapters:

- Synaesthesia is a trait in which one sensory experience is

accompanied by an automatic secondary sensory experience

- Synaesthetes potentially have different brain structures to non-synaesthetes
- Evolution by adaptation may be able to explain synaesthesia; rather than itself being functional, it is likely to have been a byproduct of useful characteristics, such as enhanced memory and creativity
- Although they are not the same, synaesthesia and cross-modality share similar definitions and mechanisms
- Cross-modality has been observed in chimpanzees and rhesus macaques
- Non-primate species like horses and dogs also show cross-modality

Let us return to the initial questions.

- Does the rarity of synaesthesia simply mean synaesthetes are an 'extreme' population, or are they just experiencing the 'extreme' end of the spectrum of connectivity between senses?
- Are humans the only species that experiences such connected senses?
- If we share synaesthesia, or cross-modality, with non-human species, what does it tell us about the evolution of synaesthesia?

Are synaesthetes 'extremes' of the human population?

In light of the points above, synaesthetes are both extremes and non-extremes of the human population. The presence of cross-modality of perception across population implies that almost everyone has 'weak synaesthesia'. This means that rather than the synaesthete populations being 'extremes', synaesthetes may be on the 'extreme' end of the spectrum of how strong, specific, and consistent cross-modality is.

Are humans the only species with synaesthesia?

The insights from non-human species, including chimpanzees, rhesus monkeys, domestic horses and dogs, show that cross-modality was not limited to humans. Whether they also have synaesthesia is not clear, since it is a subjective experience which is hard to study in species that lack language, and it is usually associated with human-specific cultural elements like seeing letters and numbers. However, as those species share cross-modality, it could probably be said that they also have weak synaesthesia like ordinary humans do. Hence, humans are not an 'extreme' species in having synaesthesia.

Origin of synaesthesia: insights from non-human species

Firstly, in terms of non-human primate species, as discussed earlier with reference to Ludwig, Adachi and Matsuzawa (2011), cross-modality in chimpanzees potentially indicates that the trait was present in the Last Common Ancestor of humans and chimpanzees. The cases of rhesus macaques might also mean they share similar brain mechanisms, or 'building blocks for cognition', with humans. Hence, the basis of synaesthesia evolved before humans became humans, but since we are uncertain if 'strong synaesthesia' is present in non-human primates, it cannot be conclusively determined whether developmental synaesthesia itself originated before or after the divergence.

Then, how about cross-modality in dogs and horses? Humans and non-human primates are more closely related, meaning similar traits likely evolved from a single, shared origin. However, given the great distance between dogs/horses and humans in the phylogeny, it would be more realistic to consider that convergent evolution caused this similar trait. Convergent evolution means unrelated species develop a similar or the same trait independently as a result of similar selection pressures. Since the horses and dogs used in the studies were domesticated, it is possible that individuals with more developed cognitive skills, such as identification of owners, might have been chosen by humans, for example. Hence, it is unclear whether cross-modality in dogs and horses informs us how synaesthesia evolved. Nevertheless, studying the reason why cross-modality is also found in various species of mammals should provide insights into how and why humans developed more complex cognition.

Implications for the evolution of language

The common trait of cross-modality between humans and non-human primates could also inform the origin of language in humans. The first language, or proto-language was primarily sound-symbolic, and Ramachandran and Hubbard (2018, p. 19) state that cross-modality illustrated by the 'bouba/kiki effect' could explain the natural tendency for the way in which sounds are visualised into forms. Therefore, visual/auditory cross-modality found in non-linguistic primates suggests our non-linguistic ancestors also had such a trait, which should allow us to have a more vivid picture of how the first language might have emerged.

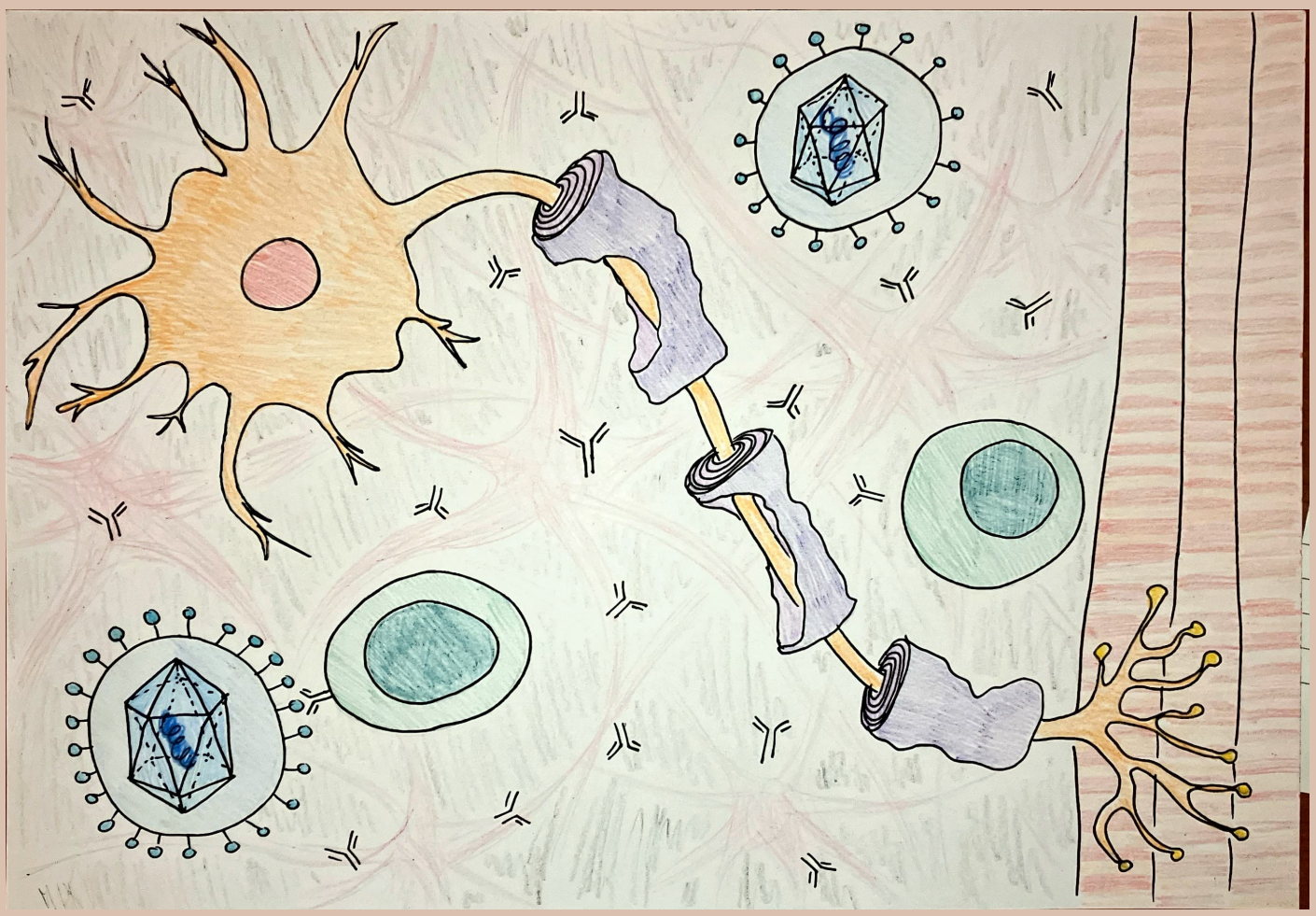
Concluding remarks

This article discussed what synaesthesia is, how it might have evolved, and what we can learn from cross-modality in non-human species. Crucially, cross-modality, which forms the basis of synaesthesia, is present in non-human primates, and thus, the trait likely originated before evolutionary divergence. It would be interesting to see further investigations into the presence and nature of cross-modality, or even synaesthesia, in non-human primates and other species.

Sara Hamaguchi is a 1st year undergraduate in Archaeology, who is passionate about biological anthropology, evolutionary medicine and primatology.

MANY SCARS, MANY CAUSES: SOLVING THE MULTIPLE SCLEROSIS PUZZLE

Courtney Kremler investigates multiple sclerosis, unpacking how a common virus, a vitamin deficiency, and a quirk of genetics might conspire to turn the immune system against itself.



Artwork by Flo Studdert-Kennedy

Multiple sclerosis (MS) is a disease whose name directly translates to "many scars". This is an extreme in the sense that it only affects about 1 in 1000 people, and it results from the body's immune system switching sides and mistakenly attacking the brain and spinal cord. Immune cells from the body cross the blood-brain barrier and attack myelin, a fatty coating on nerve cells, leading to impaired nerve signaling and, ultimately, nerve cell death. These attacks lead to "many scars" - areas without myelin - on the brain and spinal cord, which result in disability. The variety of symptoms one may have reflects the variety of functions the brain and spinal cord perform, from walking, to thinking, to feeling! But what causes the switch? Why does the immune system suddenly decide to attack myelin?

Despite a French neurologist first recognising a case of MS almost 150 years ago, its cause remains elusive. The hopes for a singular cause are dwindling, with more and more evidence to suggest there's multiple factors at play. However, a study from the Harvard School of Public Health in 2022 seems to have identified one of the most crucial risk factors for MS (Bjornevik et al., 2022). This study showed that Epstein-Barr Virus (EBV) infection was strongly associated with developing MS, and EBV infection appeared to precede nearly all MS cases in the cohort. All but one of the 801 individuals were infected with EBV before developing MS symptoms - a 32-fold increase in risk compared to infection with any other virus. And that one individual without EBV had their last blood sample taken a year before their MS onset - plenty of time to be infected. Well, that seems to solve the mystery, doesn't it?

Except there is one issue. Over 95% of adults worldwide have been infected with EBV. And if you remember the statistic from earlier – about 0.1% of the population develops MS. It seems that despite EBV being necessary, it's not fully sufficient. What else then causes such an extreme reaction to EBV infection? Why is the research community so interested in this virus?

The interest in the relationship between EBV and MS started in the late 1970s, when researchers showed that blood cells from people with MS and prior EBV infection grow and multiply more

rapidly than blood cells from people without MS (Fraser et al., 1979). From there, researchers in the 1980s found that people with MS have more antibodies (proteins that help the immune system identify foreign substances) for EBV (Sumaya et al., 1985). The research gets a bit juicier in the early 00s, when studies found an increased risk of MS following infectious mononucleosis, the symptomatic reaction to EBV (Thacker et al., 2006).

Since then, other factors have appeared on the scene as important risk factors: genetics and vitamin D deficiency (The International MS Genetic Consortium & The Wellcome Trust Case Control Consortium 2001, Ramagopalan et al., 2009, Sintzel et al., 2017).

Genetic variations in something called the major histocompatibility complex (MHC) - molecules that present fragments of proteins to immune cells to distinguish 'self' from 'foreign' - have been shown to play a key role in MS susceptibility. People with certain MHC variants seem to have immune systems that struggle to recognize myelin as part of the body's own tissue. We'll come back to why this matters in a moment.

Vitamin D deficiency is also a key risk factor for MS susceptibility and severity. This may seem strange at first glance - of course vitamins are important, but are they that important? Well, Vitamin D receptors (think of these as detectors of Vitamin D) are found on most immune cells and play a key role in modulating their activity. Without sufficient vitamin D, the immune system becomes unbalanced: macrophages (the body's clean-up crew) become less active, whilst B cells and T cells (specialised white blood cells that form part of the immune system's trained assassins) become hyperactive. This imbalance creates conditions where autoimmune attacks are more likely and the cleanup of damage is impaired.

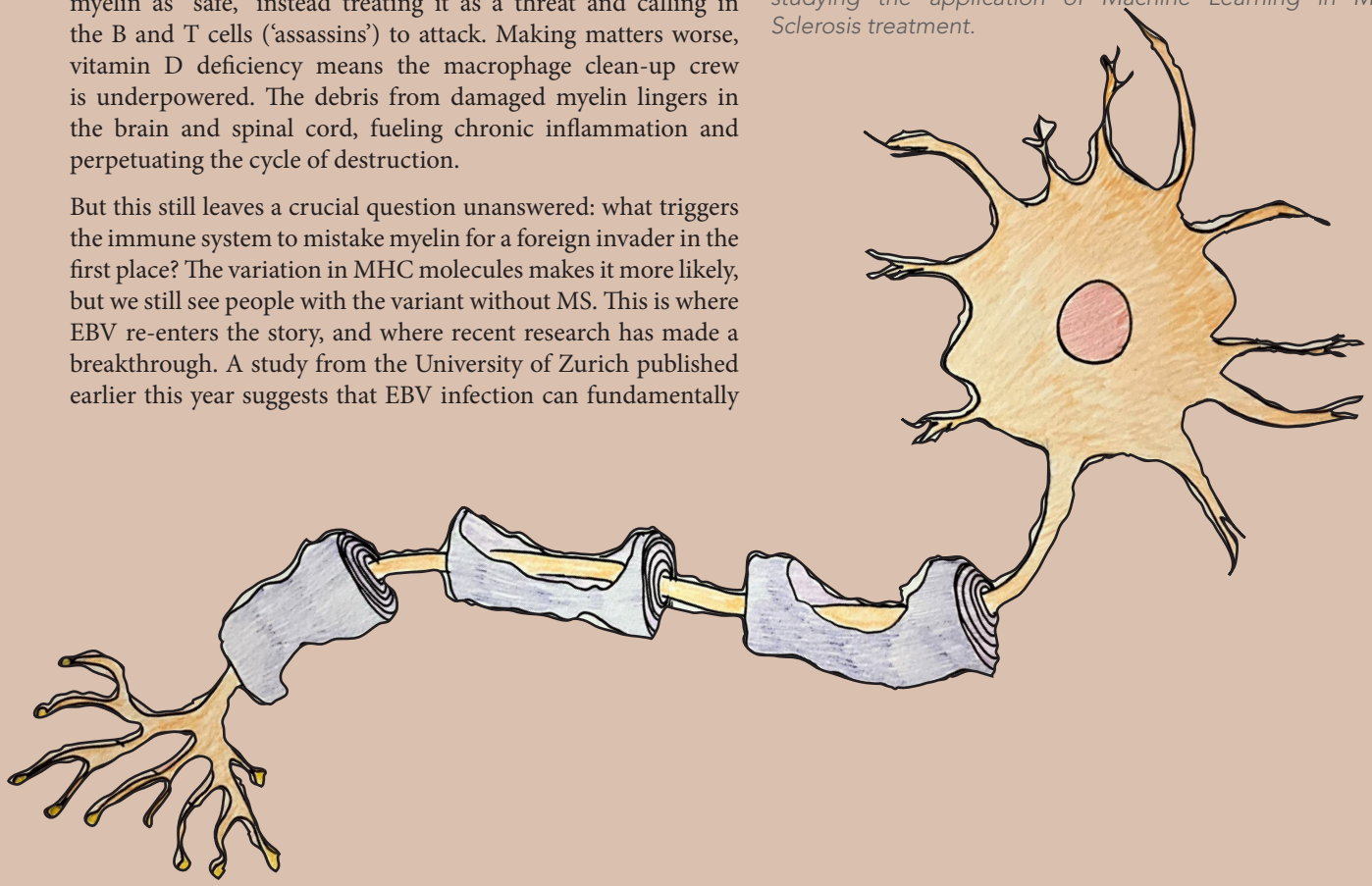
So how do these pieces fit together? One theory is that MS arises when the immune system mistakenly identifies myelin as a foreign invader rather than as part of the self. In people with certain MHC variants, these recognition molecules fail to flag myelin as "safe," instead treating it as a threat and calling in the B and T cells ('assassins') to attack. Making matters worse, vitamin D deficiency means the macrophage clean-up crew is underpowered. The debris from damaged myelin lingers in the brain and spinal cord, fueling chronic inflammation and perpetuating the cycle of destruction.

But this still leaves a crucial question unanswered: what triggers the immune system to mistake myelin for a foreign invader in the first place? The variation in MHC molecules makes it more likely, but we still see people with the variant without MS. This is where EBV re-enters the story, and where recent research has made a breakthrough. A study from the University of Zurich published earlier this year suggests that EBV infection can fundamentally alter B cells, leading to myelin being detected as foreign (Wang et al., 2026).

When EBV infects B cells (those assassin immune cells we mentioned earlier) it can change their genetics and protein composition. Think of your keys on a key chain; you can add charms to your key chain, and this might make it easier or harder, depending on the charm, to find your keys. Similarly, EBV adds molecules to the B cells' DNA that can turn certain genes on or off. These altered B cells have more proteins on their cell surface to display foreign invaders to the rest of the immune system, and a complete switch in which proteins they're choosing to display. The altered B cells now choose to present myelin basic protein (MBP), a key component of myelin's protective coating, on their surface using MHC molecules. This inappropriate presentation essentially trains other immune cells to recognise myelin as a threat. Once trained to view MBP as foreign, the immune cells attack myelin throughout the brain and spinal cord.

So, what have we learned? MS isn't caused by a single factor, but rather a perfect storm of circumstances. EBV infection can often provide the initial trigger, altering immune cells in ways that can lead them to mistake the body's own myelin for a foreign invader. But this only causes disease when combined with other risk factors: genetic variations that impair self-recognition, vitamin D deficiency that throws the immune system off balance, and other factors that are still being uncovered. This understanding transforms MS from an unpredictable autoimmune disease into a condition with identifiable risk factors and, crucially, potential intervention points. From EBV vaccines under development to investigating vitamin D supplementation strategies, the field is already working to intervene and help prevent the disease, or at least its severity, before those many scars even form.

Courtney Kremler is a 3rd year PhD in Clinical Neuroscience studying the application of Machine Learning in Multiple Sclerosis treatment.



Are the sexes evolutionarily programmed to be extremes?

Alex Barrington explores how sexual conflict, rooted in the asymmetry between sperm and egg, shapes human biology, behaviour, and culture from the genome up, and asks whether the sexes are truly two warring extremes.

Artwork by Grace Heslin

"Man is more courageous, pugnacious, and energetic than woman, and has a more inventive genius. His brain is absolutely larger." (Darwin 1871a, vol. 2, 316–317).

When Darwin wrote about sexual selection in his second book: 'Descent of Man', his Victorian prudery and misogyny saw him miss some rather key points in explaining the "taste for the beautiful" observed in animal mate choice. Luckily, we have advanced in our understanding of the coevolution of traits like the brilliant tail of the male peacock, or the energetic dance-and-drumming combination of the male peacock spider, and the corresponding female preferences. For mating to occur successfully, the two sexes must, of course, cooperate. However, the evolutionary interests of males and females are asymmetrical, leading to conflict between mates. Thankfully, unlike spiders, humans have not evolved sexual cannibalism or grasping organs. That does not mean we have not been shaped by years of sexually antagonistic coevolution. In fact, unconscious sexual conflict in humans pervades human physiology, behaviour and culture. Are the two sexes inescapably two ends of an extreme?

How does sexual conflict arise in the first place?

Imagine a population with two mating types, A and B. A individuals can only successfully mate with B individuals. At first, all gametes (the cells that fuse to form the early embryo) are the same size, meaning A and B individuals invest equally in their offspring by way of cellular contents. Say mating type B randomly acquires a mutation that makes their gametes slightly smaller than the other mating type. This is greatly advantageous: they could keep more of their hard-earned nutrients for themselves, or maybe produce more gametes as a result of investing less into each individual one. The mutation is under high positive selection due to this advantage, and will spread through population B. Now, though, each offspring is getting fewer nutrients. A strong selection pressure arises for mating type A to make up the difference, giving the offspring a better chance of survival. Gametes of A individuals should become larger. The difference will become more pronounced over time,

with mating type A having a few, high-investment gametes and B having many, low-investment gametes.

This is exactly the evolutionary process that we believe happened with sperm and egg in humans. Their size asymmetry underpins the differences in male versus female optimal mating strategies to maximise reproductive success. Trivers' pioneering theory posited that high-investing females ought to be more choosy, while males ought to be more competitive with members of their own sex for mating access to the higher investing sex[1]. This difference in optimal strategy may underpin human sexual conflict, ranging from the genomic scale right up to our cultural norms.

Sexual conflict in our genome

Human females, quite uniquely, live far longer than the time during which they can successfully reproduce, perhaps so they can help their offspring to reproduce and avoid costly within-family reproductive competition. Women go through the menopause, which is well-known to be a rather difficult and uncomfortable process spanning up to two years. But why does stopping reproduction need to come with a host of physiological symptoms?

Obviously, 50% of our genes come from our mother and 50% from our father. Some of these genes may be expressed differently depending on whether they originated from mother or father. Ancestral human lineages were patrilocal, meaning that a woman moved to her husband's kin group upon marriage. Because the individuals in her new group had low relatedness to her and her offspring, it was most beneficial for this wife to retain her own reproductive ability longer, rather than lose it earlier in favour of helping others in her group. Therefore, selection resulted in genes of the maternal lineage (those inherited from mother, who had moved groups upon marriage) to favour a later menopause. This would maximise the chance of passing on her own genes.

In contrast, the husband is related to individuals in the group besides his own offspring. Genes inherited down the paternal line favour an earlier menopause for two reasons: a woman helping her husband's kin in the group is beneficial, and the potential for a woman to participate in extra-pair matings that produce unrelated offspring is decreased.

This intragenomic conflict, shaped by thousands of years of evolution, may manifest physiologically with frequent oscillations in body hormone levels that result in the unpleasant symptoms characteristic of the menopause. A similar genomic conflict occurs in offspring themselves: paternally-inherited genes promote suckling, while maternally-derived genes promote earlier weaning (males want females to invest more; females want to hold back some resources). In pregnancy, intragenomic conflict may cause complications and disease. Women with a higher average diversity of mating partners have more disordered pregnancies: mothers are ten times more likely to suffer pre-eclampsia when they have produced offspring with multiple men. Current research on the mechanisms underpinning this is perhaps an article in itself, with suggestions that signalling foetal cells enter the maternal system and measure genetic diversity of any cells left from previous foetuses! Conflict gives the ultimate explanation, though, as less reliable monogamous mating of the mother means it pays for the offspring to extract more resources from the mother as siblings will not be as highly related as if she were monogamous. So, antagonistic coevolution between the sexes occurs at the genomic level, perhaps underlying many of our greatest medical challenges.

Sexual conflict affecting human behaviour and physiology

Not only can our physiology be affected by ongoing internal sexual conflict, so can our behavioural strategies.

As briefly discussed, parental investment shapes one side of this sexual conflict. Women's minimum obligatory investment to produce offspring is far greater than men's (nine months of pregnancy, at least!). Fast, indiscriminate mating strategies in females cost time and energy, while in males, reproduction is far less costly, and men benefit from this short-term, low-investment strategy. This asymmetric minimum obligatory investment can account, in part, for certain cognitive biases in men and women. Men consistently overperceive sexual interest from women (e.g. mistaking a friendly smile for sexual interest) as this error was far less costly for our male ancestors than underperceiving sexual interest and missing an opportunity! The opposite is true for women. Sadly, this can also partially explain why men are more likely to be the perpetrators of sexual coercion and rape. However, we must be extremely cautious with evolutionary explanations for such crimes; it is just one small facet underpinning these events, and we cannot play down the consequences of cultural and societal influence.

Paternity uncertainty is the other major driver of human evolutionary sexual conflict. Due to internal fertilisation and gestation, ancestral men could have little certainty that children were genetically their own. Female infidelity is a recurrent feature of our evolutionary history, with many evolutionary consequences: male competition adaptations range from anatomical (e.g. the anatomy of the penile frenulum suggests it is possible to displace any leftover semen from a previous partner during sex), to physiological (evidence suggests men who have spent more time with their partner ejaculate lower sperm counts at the next copulation), to psychological (men who spent a greater proportion of time apart from partners since last copulation perceive their partners to be more sexually attractive).

Our evolutionary history of infidelity also shapes why newborns all look relatively similar. Paternity cannot be assumed from the image of the baby, meaning that ancestral females' partners could not have immediate proof of infidelity and so were more likely to care for mother and baby. In the modern day, this fact unconsciously plays out. MacLain et al. (2000) photographed 160 newborns up to 3 days old and their parents, under the proviso that they were studying facial structure. When leaving the room, researchers casually asked: "which parent do you think the baby looks most similar to?". When both parents were present in the room, if mothers answered first, 88% said the father. If the father answered first, just 51% said the father looked most similar. When mothers were alone, 60% said the father. When unrelated judges were presented with photos of newborns and their parents, and asked to match them, they were more likely to match mothers to the newborns, contradicting the earlier results where fathers were perceived to look most similar. Therefore, the earlier bias in mothers' remarked resemblance does not reflect the actual resemblance, evaluated by unbiased judges. In no way do the authors suggest this is conscious, but perhaps it represents an evolved response to assure domestic fathers of their paternity.

[1] There are role-reversals! In the pipefish seahorse, males do the investing, and females are more aggressively competitive, as we would

Sexual conflict affecting society and culture

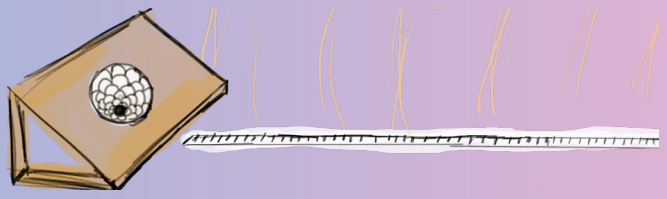
Going back to Darwin's quote at the beginning, it is clear that misogyny can pervade science and bias our understanding of sexual selection. In a reciprocal way, some argue that gender norms are a product of sexual conflict. Institutional rules, including marriage systems, monogamy, inheritance laws and even sexual reputation may have evolved to control female sexual access (ensuring paternity certainty as far as possible) in return for investment in offspring. These norms stabilise cooperation, 'solving' sexual conflict.

Gender norms can bias population sex ratios through inheritance preferences, sex-selective abortions, differential parental investment in sons vs daughters, marriage age, and migration patterns. For example, in the UAE, 70% of the population is male due to migrant workers, while in Ukraine, the population is becoming increasingly female-biased due to conflict disproportionately affecting young male mortality. Theoretically, when men are abundant, women can afford to be choosy, leading to greater female autonomy. Monogamy tends to become a behaviour norm. When women are abundant, men can afford to invest less and pursue multiple partners, and society tends towards polygamy and stricter control of female sexuality. The sex ratio changes mating strategy, which changes gender norms, which change parental behaviour, which changes population sex ratios: a cyclical relationship.

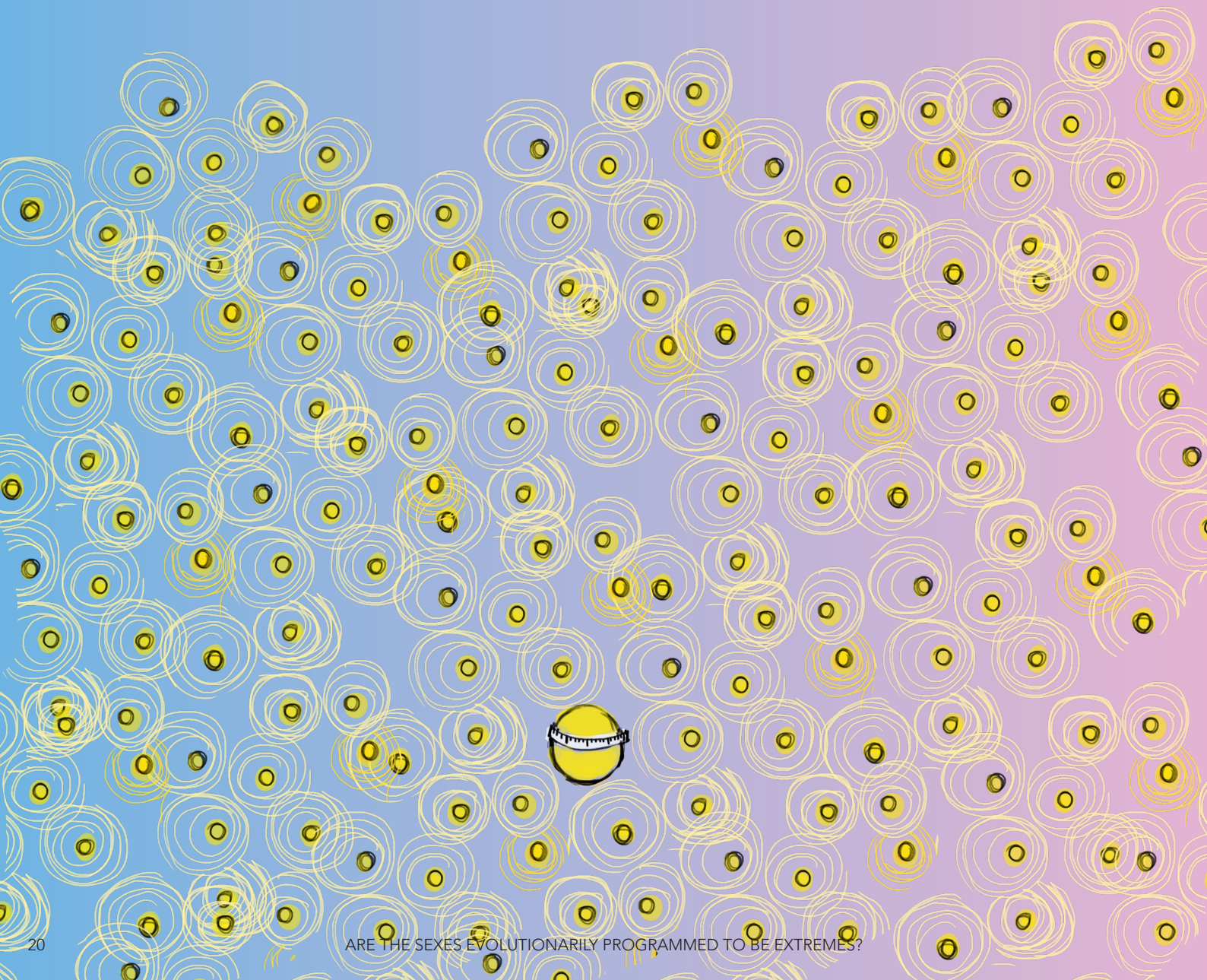
Sexual conflict plays out at all levels of human biology: but has also shaped cooperation!

At all levels, it is dangerous to take sexual conflict at face-value: evolutionary patterns are one explanatory factor in how traits spread, and their influence is interesting, but in no way does evolution fully explain or justify any cultural biases, or predict individual or societal behaviour. Cultural and gender bias can also influence how we study sexual selection and conflict, just as it did in Darwin's time.

The different optima in male and female mating strategies invite conflict, from molecular to societal scales, but fundamentally, reproduction is a cooperative affair. Conflict has also resulted in compromise, which enables the persistence of cooperation in reproduction. The two sexes are by no means two warring extremes, but our evolutionary history has shaped sexual tension from the genetic level right up to our cultural norms.



Alex is a 3rd year Zoology student, with particular interest in how animal behaviour evolves and its implications for species persistence in a changing world.



EXTREME LIFESPANS: CREATURES THAT DEFY TIME

Artwork by Annie McAuley

From Pando the tree to hydrae, Nicolas explains the creative strategies in which some species achieve longevity.

When we think of ancient living beings, giant Galapagos tortoises or long-lived bowhead whales immediately come to mind. These animals routinely surpass a century of life. Yet, it is the invertebrates that truly hold the record: the marine bivalve *Arctica islandica*, born 65 years before Shakespeare and surviving until 2006, lived an astonishing 507 years. Among plants, Methuselah - an ancient bristlecone pine - is more than 4,000 years old. Thousands of meters below the sea, black corals and the deep-sea sponge *Monorhaphis chuni* have silently witnessed the rise and fall of the ancient Egyptian empire. Nature is full of organisms that can achieve spectacular lifespans, but how do we measure it, how do they do it, and what can it tell us about ageing?

Dating the oldest: biological time capsules

How do we measure the age of these ancient beings? For trees, dendrochronology (counting growth rings) works well. An analogue approach, sclerochronology, serves clams by examining shell growth lines under a microscope. For marine vertebrates, the task is trickier. Serendipitous archaeological evidence exists, such as a 2007 Inuit discovery of a harpoon tip (patented 1885–1895) embedded in a bowhead whale's neck.

More systematically, researchers analyse the eye lens, a fully post-mitotic tissue - differentiated and non-renewable - which essentially serves as a "time capsule", preserving a record of the animal's date of birth. Biochemically, the ratio of D- and L- aspartate enantiomers (right- and left-handed variants) in the lens increases predictably with age, allowing scientists to estimate lifespan. The Greenland shark offers a spectacular case: using radiocarbon-14 from 1950s nuclear tests incorporated into lens proteins, scientists estimated that these sharks reach maturity around 150 years and the oldest individuals live up to 392 ± 120 years.

A promising approach, especially for clonal or extremely long-lived organisms, is the "molecular clock". By tracking the accumulation of somatic mutations occurring at a roughly predictable pace, we can reconstruct an organism's phylogeny and trace back to the founder genome - a historical record written in DNA.

Arctica islandica, showing distinctive annual banding and internal growth patterns on the shell. Photographs provided by The Conchological Society of Great Britain and Ireland, with kind permission from Paul Butler.

Protection against genome aging

Not even the healthiest morning routine of a L.A. millionaire can protect against the inevitable accumulation of random mutations caused by DNA replication and environmental stress. These mutations can impair genome function, contribute to ageing, and lead to cancer. However, some long-lived animals, such as elephants and bowhead whales, possess enhanced DNA repair mechanisms or extra copies of tumour suppressor genes which help to maintain genome integrity. This likely underlies their remarkable longevity and explains why these large animals, despite their huge number of cells, do not show proportionally higher cancer rates—a phenomenon known as Peto's paradox.

Telomeres, the protective caps at chromosome ends, shorten with each cell division, eventually triggering self-destruction (apoptosis) once a critical threshold is reached. In most organisms, this limits the number of divisions a newborn cell can undergo. Yet some species, like lobsters, express telomerase, an enzyme that repairs telomeres and allows cells to surpass this biological limit.

Similarly, the naked mole rat exhibits striking longevity for a rodent, with virtually no cancer incidence. This appears to be linked to high-molecular-mass hyaluronan, which may have evolved to provide skin elasticity for life in underground tunnels, while simultaneously protecting against DNA damage and tumours.

Immortality by replication: the clonal strategy

Clonality appears to be a very effective strategy to ensure one grows old. A clonalorganism has the potential to self-replicate without sexual reproduction, producing genetically identical copies of itself. This is the case for Pando (Latin for "I spread"), a giant male quaking aspen living in northern Utah (U.S.), formed from 47,000 stems, all connected by a massive root system. Pando is estimated to have been born 12,000 to 37,000 years ago, and now spans 42.8 ha, making it likely the heaviest living organism on Earth. In Tasmania, the clonal shrub King's Holly is estimated to be at least 43,000 years old. Unfortunately, it is also doomed to clonality, reproducing solely through vegetative growth via rooting branches, as it is sterile and the only known representative of its species. Similarly, the Mediterranean clonal colony of seagrass *Posidonia oceanica* may reach an incredible 100,000 years.

Clonality also exists in animals, of which the Canine Transmissible Venereal Tumour offers a striking example. Approximately 6,000 years ago in North or Central Asia, a wolf-looking dog developed a genital cancer. Remarkably, cells from this tumour managed to transmit themselves to another dog during mating, and grow as a cancer in this new "host". Long after the death of the founder dog, these cancer cells survive, still "infecting" thousands of dogs worldwide today as a 6,000-year-old asexual parasite.

Dormancy across geological time

Life can be demanding, especially in harsh environments, and survival by slowing down to near-stasis can sometimes be a good choice. Microorganisms frozen in deep layers of Siberian permafrost, especially anaerobic and spore-forming bacteria, have been revived after up to a million years of dormancy. Similarly, "zombie viruses", like *Pandoravirus* and *Pithovirus*, are still infectious and able to replicate after thawing.

This principle extends to multicellular life. Bdelloid rotifers and several species of nematode worms have persisted dormant in the permafrost since the last Pleistocene (~ 40,000 years BP). This state of suspended animation and metabolism, between life and death, is called cryptobiosis and allows survival through extreme environmental stress and maintains a reservoir of microbial diversity.

However, for larger organisms, tissue complexity and the need for coordinated hydration, oxygenation, and molecular protection limit the feasibility of such metabolic arrest.

Cnidarians and the illusion of immortality

The Cnidaria phylum contains 10,000 species of solitary, carnivorous marine invertebrates including sea anemones, jellyfishes and hydrae. They usually present as one of two basic body forms: medusa and polyp, and some of them, it turns out, spectacularly bend the rules of aging.

Hydra display negligible senescence: their probability of death does not increase with age, and their reproductive output remains constant, suggesting potential immortality. Furthermore, the hydra's continuous self-renewal allows for regeneration of the entire individual, from a batch of only a few hundred epithelial cells.

Even more strikingly, the tiny jellyfish *Turritopsis dohrnii*, found worldwide, seems to bypass death. When stressed, adults can revert to the polyp stage - an early phase of their life-cycle - through a rare process called transdifferentiation. They then mature once more into a new, genetically identical, and biologically "younger" version of themselves. This loop can, in principle, continue indefinitely, providing a form of eternal youth.

The recipe for longevity

Across life, longevity seems to rely on a few recurring strategies:

1. Modular (lacking specialized non-renewables tissues whose loss is fatal), permanent self-renewing organization (clonal plants, hydra, jellyfish)

2. Genome protection via telomerase and efficient DNA repair (lobsters, elephants, whales).

3. Ability to revert to juvenile stages or clone oneself (jellyfish, clonal trees)

4. Metabolic economization: slow growth, limited cell divisions, often in cold environments (deep-sea corals and sponge).

5. Avoidance of extrinsic mortality (predators, pathogens, accidents)

Moreover, these examples challenge our notion of individuality. What defines "an individual"? The physical body of an animal, a super-organism of social insects, a transmissible cancer, or a symbiotic coral colony? The boundaries are blurred. Cnidarian self-renewal also raises a profound question: if all the cells of an organism are progressively replaced through cellular turnover and rejuvenation, is it the same individual? Does its identity remain intact? This tension between biological and philosophical identity is known as the "Ship of Theseus" paradox.

Subjective time and the elasticity of experience

Longevity is relative. For mayflies, humans might seem immortal; for shrews, seconds feel like minutes. The "biological time hypothesis" suggests that temporal perception depends on metabolic pace: animals with extremely fast or slow metabolisms experience time differently. Shrews, with heart rates above 1,000 bpm, probably perceive the world in slow motion, whereas blue whales, with heart rates near 1 bpm while diving, may experience time more leisurely. Black corals, growing mere micrometres per year, witness centuries unfold almost imperceptibly.

Neuroethology confirms that small animals have higher critical flicker fusion frequency, meaning their "sense of time" runs faster. In other words, the total "physiological budget" of heartbeats or biological events may remain broadly similar, despite lifespan variation across species. Humans also construct time cognitively, influenced by attention, emotion, cross-modal sensory integration, age, and environment. Therefore, our subjective lifespan may be expanded not by living longer, but by living more richly - through novel experiences, emotional intensity, and active learning.

Nicolas Branche is a medical doctor and visiting researcher in the Transmissible Cancer Group at the Department of Veterinary Medicine.

All the Pando trees are reproductions ("modules") derived from the same original individual ("genet"). Credits: Wikimedia Commons; Friends of PANDO.

From AlphaFold to AlphaGenome: Google Deepmind sets its eyes on biology.

Timothy Lambden investigates how Google DeepMind's AlphaGenome is transforming biology from a natural science into a data science — and the uncomfortable questions its success raises about profit and transparency in science.

Waiting for Google to drop their new AI4Science Nature paper is the scientific equivalent of waiting for your favourite artist to drop their new album. This time they have their sights set on genomics with AlphaGenome, which "decodes the genetic 'operating system' of human life" – Quote from Gemini 3, Google's AI LLM model.

The landscape of modern biology, like most of society, is undergoing a Silicon-led shift increasingly shaped by big tech. For decades, the field was defined by the scent of agar plates, the painstakingly slow process of pipetting and cell culture and long weekends in the lab. But as Google DeepMind unveils its latest project, AlphaGenome, it is becoming clear that biology is no longer just a natural science, it is a data science. We discuss here its history, what this means for scientists, and future ethical implications.

History:

The structure of DNA was discovered in 1953, long hypothesized by Erwin Schrödinger's book "What is life?" many years prior. In 2003, the International Human Genome Sequencing Consortium announced the completion of the first human genome sequence, costing $2.7 billion. Today, thanks to next generation sequencing it is now possible to sequence your entire genome for approximately $100. Despite all this progress in the recent decades, understanding how your genome (all 3 billion base pairs) contains enough information to create a human being or what happens when a few mutations cause genetic diseases (e.g. cancer) remain complex challenges.

In 2020, Google DeepMind released AlphaFold. It marked a major breakthrough in the protein folding problem, predicting how a sequence of amino acids would fold into the functional machines that power biological life. It was a triumph for structural biology, and for Google DeepMind, but it only addressed the 1-2% of our DNA that codes for proteins, the coding genome. Biological information generally flows from DNA to RNA to proteins, forming the central dogma of molecular biology.

AlphaGenome looks at the remaining 98%: the once-dismissed "junk DNA" that may not really be junk at all. This vast, non-coding expanse is the regulatory "dark matter" of life. It functions as the cell's internal operating system, containing the complex

switchboard that tells protein machines when to turn on, and when to stop, alongside remnants of millions of years of natural selection. It's a bit like a software engineer trying to decipher leftover code written by other software engineers.

AlphaGenome is aimed at understanding sequence-to-function. It can process up to one million base pairs, and observe "long-range" interactions with single letter accuracy. It was able to simulate known DNA mutations responsible for leukaemia, predicting the same results as those observed in the lab, located hundreds of thousands of letters away. 3000 scientists in 160 different countries have used AlphaGenome and are submitting around one million requests each day. However, predictions for underrepresented tissues or rare cell types with limited training data remain poor. While AlphaFold predicts the structures of the tools of life, AlphaGenome is deciphering the logic that governs them.

What this means for scientists:

This transition from predicting shapes to interpreting logic is driving a fundamental rethinking of biology. In the corridors of top research institutes, you'll hear whispers saying: "If you want a career in biology, you're better off studying physics or computer science." This is not a slight against biology but an acknowledgement of its new big data identity. Similar breakthroughs in structural biology occurred with new tools from physicists like X-ray diffraction and CryoEM, both pioneered in Cambridge. Now with breakthroughs in computational power and AI models, biology has entered its *in silico* era, drugs are designed and tested on computers well before any pipetting has begun, and Google leads the way with a new spinout in Isomorphic Labs.

However, this rapid advancement brings a sharp tension between academic transparency and commercial gain. Google DeepMind faced backlash from the scientific community for keeping AlphaFold closed-source. This frustration led to an emergence of "reverse-engineered" models like Boltz-1, designed by a few former Cambridge mathmos which achieved similar accuracy, but was designed with open-source at its heart. Making these models open-source may limit Google's ability to profit from them in the future, but there is a deeper ethical question at play regarding the training data that makes these models possible. AlphaGenome was trained on massive, open-source datasets like ENCODE (Encyclopedia of DNA Elements) and GTEx (Genotype-Tissue Expression)—the fruit of decades of taxpayer funding from NIH, alongside labour by thousands of independent researchers. Its performance directly correlates to the data it was trained on, and improvements to scaling laws of these models require more experimental data to be generated, similar to AlphaFold being trained on data from the Protein Data Bank. Yet, researchers who generated and maintained the data "for public good" see none of the financial windfall.

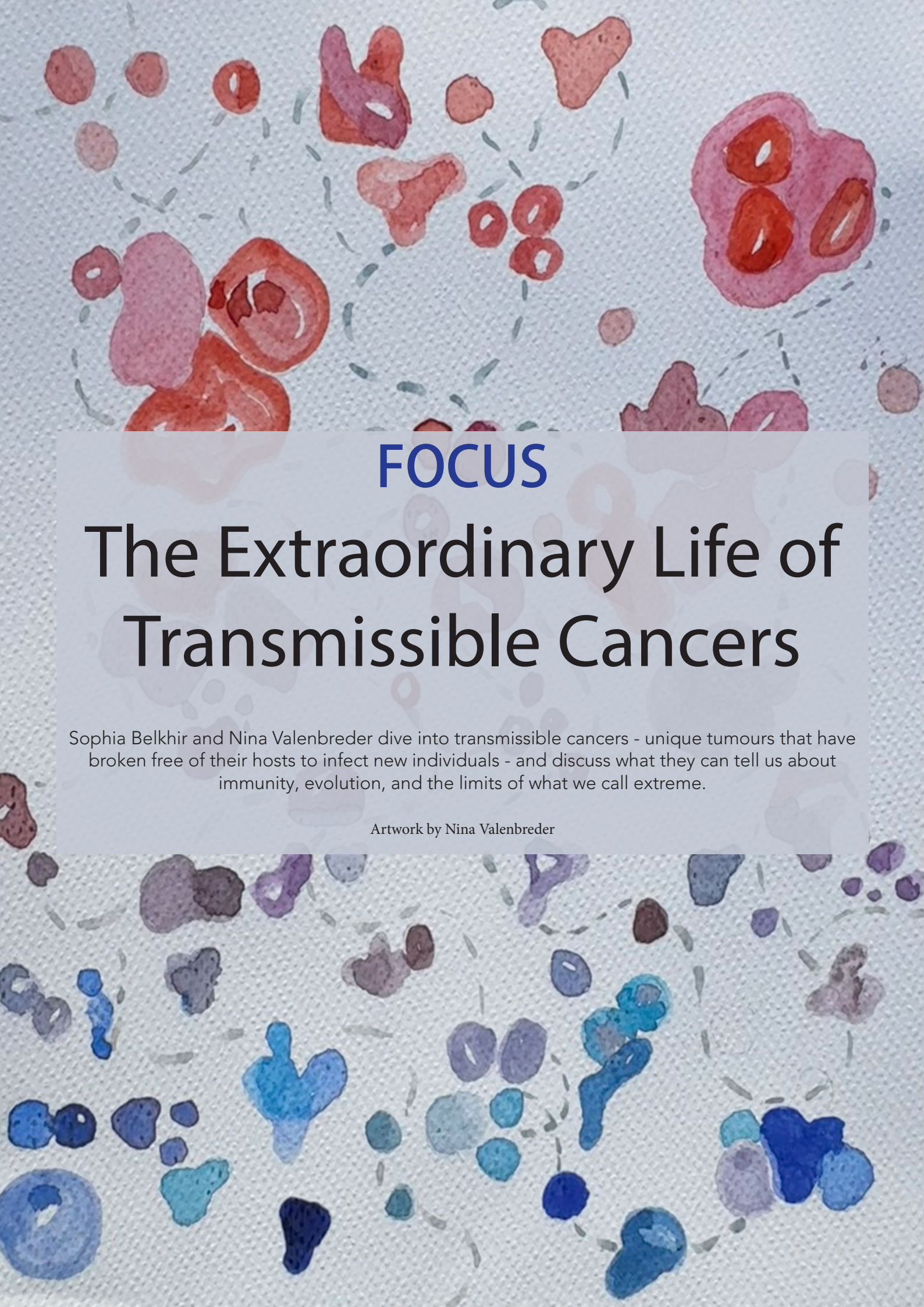
The ethics dilemma for science:

As Google DeepMind enters the biology playground distributing tools to scientists, what does this mean for scientists and how science is done? Are we moving too fast to ensure the benefits remain open to everyone, just like the data that built the models? Will companies only be pressured to open sourcing their work only after copycat labs are able to reproduce it? While scientists begin to adopt these models into their work, hoping to create new groundbreaking discoveries, they must confront these new challenges head on.

What remains at the core of Google DeepMind's mission is to use "AI for good", and AI4Science is a huge part of that mission. However, that will require a symbiotic relationship with scientists: while science is built on open access, big tech companies are not. With Google setting its eyes on biology, other fields such as weather prediction, material science and robotics are also on the horizon, with clear signs of commercial potential. The question is no longer whether AI will shape the future of biology, but rather who controls the trajectory and who ultimately benefits?

Timothy Lambden is a final year PhD student on the NanoDTC program, specialising in electron microscopy of radiation sensitive materials.

Timothy thanks Anna, Jeff, Kou and Max for proof-reading the article.



FOCUS

The Extraordinary Life of Transmissible Cancers

Sophia Belkhir and Nina Valenbreder dive into transmissible cancers - unique tumours that have broken free of their hosts to infect new individuals - and discuss what they can tell us about immunity, evolution, and the limits of what we call extreme.

Artwork by Nina Valenbreder

The accumulation of genetic mutations can give rise to abnormal or malignant cells in the body. Luckily for us, these aberrant cells usually ring the body's alarm bells, triggering their swift detection and removal. Some cells, however, will escape detection, proliferate uncontrollably, and exploit local resources in the body, shaping what we call a cancer. Conventionally, cancers are constrained to the individual in which they have arisen. In most cases, tumours die out either by means of treatment or when their affected host succumbs to the disease. But what if cancers could break their host-imposed handcuffs? While that sounds rather unusual, in rare cases, cancers can do exactly that.

Welcome to the world of transmissible cancers. These cancers act like infectious agents, with cells jumping from one individual to another as viruses or bacteria do. With their Houdini-esque properties, these extreme cancer lineages can survive in populations for years, even centuries, achieving a form of biological "immortality".

The Unlucky Species that Developed Transmissible Cancers

Despite the strong barriers to the emergence of infectious cancers, a few unlucky species have developed this one-of-a-kind disease. The series of unfortunate events leading to the emergence, spread, and establishment of a continued lineage of transmissible cancers has occurred across different phyla within the Animalia kingdom. As you will realise, these animals do not have much in common and demonstrate independent examples of these rare cases where cancer becomes contagious. The most emblematic example of animals suffering from such cancers is undoubtedly the Tasmanian Devil (*Sarcophilus harrisii*), a species of marsupial endemic to the Australian island of Tasmania. Tasmanian Devils have been unlucky not just once, but twice, as a second type of their Devil Facial Tumour (DFT) transmissible cancer, named DFT2, emerged less than twenty years after the first one (now known as DFT1). The tragic population decline provoked by DFTs caused this species to be classified as endangered by the International Union for the Conservation of Nature (IUCN). The other mammalian species affected by transmissible cancers is more well-known, although its transmissible cancer is surprisingly less notorious. Canine Transmissible Venereal Tumour (CTVT), as its name implies, affects dogs (*Canis familiaris*) and is endemic in at least 90 countries across the globe.

Stepping away from man's best friends and the furry but mighty devils, we turn to what happens underwater, such as in the muddy depths of lakes, where a few surprises await. Indeed, there is ongoing work to confirm whether the black skin tumours that are highly prevalent in brown bullhead catfish (Ameiurus nebulosus) from North American lakes are transmissible melanomas.

Invertebrates get transmissible cancers too. In fact, seven species of marine bivalves, a class of aquatic molluscs that includes clams, cockles, or mussels, are affected by distinct lineages of bivalve transmissible neoplasia (BTN), a leukaemia-like cancer.

To successfully establish as a transmissible cancer, there are a few steps that tumour cells need to go through (see Fig. 1):
1) Escape their original host and physically transplant into a new host.
2) Manage to survive in the new host by evading its immune system.
3) Grow into substantial solid tumours (in the cases of facial tumours of Tasmanian Devils, CTVTs of dogs, and melanomas of catfish) or further proliferate and circulate in the haemolymph (equivalent to the blood for marine bivalves).

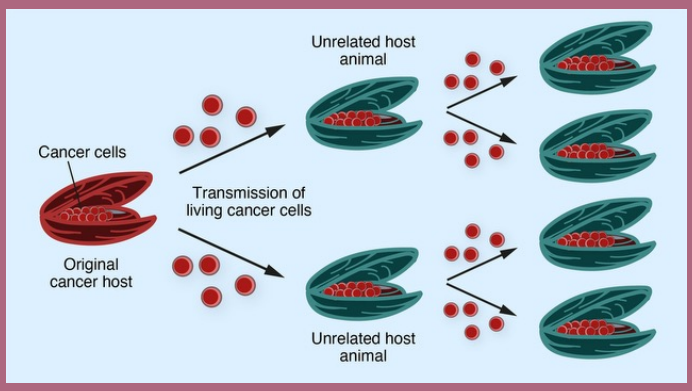


Figure 1: Transmission diagram of a cancer spreading to new hosts (bivalves) via the physical transfer of cancer cells. Image credit: Adrian Baez-Ortega.

Each species' specific behaviours provide an optimal route of transmission for its transmissible cancer(s). Tasmanian Devils have a terribly strong jaw, packing the most powerful bite relative to body size of any living mammal. Their survival relies on being able to scavenge carcasses, and they take part in aggressive behaviours for securing mates and reproducing. This includes both males and females biting each other around the face, often inflicting many injuries. These bites spread DFTs both from biter to bitten and from bitten to biter. CTVT, on the other hand, is a sexually transmitted disease that takes advantage of dogs' particular mating mechanism to transmit genital tumours affecting both males and females. Oro-nasal forms of the disease can also develop from social behaviours like licking or sniffing. Finally, marine bivalves are filter-feeders, meaning they sift nutrients suspended in the seawater that passes over them, and might end up filtering cancer cells.

I think we can all agree that transmissible cancers are an intrinsically exceptional biological phenomenon that gets increasingly fascinating when we look at the specifics of each affected species and existing lineages.

Cancers Surviving in Sea Water, Travelling the World, and Jumping to Different Species

Let's get back to the bivalves' transmissible neoplasia (BTN), whose cancer cells disseminate in water and are picked up by filter-feeding. This highlights the resilience of BTN cells, which survive in the extreme environment of seawater before being taken up by a new host. Notably, mussels affected with BTN lineages MtrBTN1 or MtrBTN2 can be found in both the Northern and the Southern Hemisphere, in the Atlantic and Pacific Oceans, in the Baltic Sea, the Adriatic Sea, the Sea of Japan, and even in the subarctic Sea of Okhotsk. These cancers are therefore robust enough to live in very distinct environments. This geographical distribution is likely due to diseased animals attaching to boats and hitchhiking from port to port, exposing the contribution of human activities to the spread of this disease worldwide.

Like mussels, the common cockle and the basket cockle suffer from two distinct forms of their respective BTN cancers (CnuBTN1 and CnuBTN2 in the basket cockle, CedBTN1 and CedBTN2 in the common one). This suggests that some species are particularly prone to the emergence of these unusual, parasite-behaving forms of cancer. Perhaps even more astonishing, a single lineage

of BTN in mussels (MtrBTN2) is capable of infecting different species from its founder host (*Mytilus trossulus*). For the average person, *Mytilus trossulus*, *Mytilus edulis*, *Mytilus galloprovincialis* and *Mytilus chilensis* might look very similar- they all are "blue mussels" that certainly taste good with a side of French fries. In reality, they are distinct species that diverged from each other a few million years ago. Although not much is known about the recognition of "self" versus "non-self" in bivalves, a paucity of immune rejection barriers might make it more likely for this kind of cancer to emerge and cross species barriers.

Cancers Flying Under the Immune Radar

At first glance, few readers would group sea-dwelling bivalves with large, land-bound carnivorous marsupials (though you may be swayed by a quick search for "Tasmanian devils swimming"). Despite differences in ecology and behaviours, both species are vulnerable to transmissible cancer lineages. In marine bivalves, hitchhiking BTNs may spread in part because immune barriers between individuals are relatively lax. In contrast, the two devil facial tumour lineages (DFT1 and DFT2) represent an intriguing case of solid cancers that slip undetected under their host's complex immune radars. While a conventional cancer must "only" navigate the immune landscape of its hosts, transmissible cancers must also dodge inter-individual immune barriers.

Along with other, albeit less powerfully, jawed vertebrates, Tasmanian devils have advanced mechanisms to distinguish "self" from "non-self" material, a phenomenon also known as "allorecognition". One of these critical defences is the major histocompatibility complex (MHC), a diverse set of genes that alert the immune system to the presence of viruses, bacteria, and other invaders. MHC molecules are grouped into three classes depending on their structure and function. MHC class I molecules, expressed on the surface of all nucleated cells, display endogenous material to patrolling white blood cells. After encountering "warning signs" on the surface of foreign or aberrant cells, these white blood cells trigger a rapid immune killing cascade. Class II molecules form another arm of MHC that are only expressed by professional immune cells (also called antigen-presenting cells). As the VIPs of the immune system, they can directly engulf, process, and present material from invader cells, initiating diverse immune responses. In human medicine, MHC-mediated allorecognition is a critical consideration. To ensure successful organ and graft transplants, for instance, MHC alleles between donor and host are matched to reduce the chances that the "non-self" donor material is rejected by the host immune system. In most cases, this requires lifelong treatment with immunosuppressive medicines. Together, MHC class I and class II form a formidable immune barrier for mammalian transmissible cancers to overcome.

Studying DFTs revealed some, but not all, of the ways by which these transmissible cancers escape immune detection. One of these mechanisms is the downregulation of MHC class I molecules on DFT1 cell surfaces. This "missing self" adaptation is observed across several human cancer types because it allows cancer cells to fly undetected under the immune radar. Luckily, mammalian immune systems have learned to deal with such stealthy cells. Natural killer (NK) cells, for example, are immune first responders that are recruited to sites of injury and inflammation. They also activate specifically in response to any "missing self" cells. Thus, an outstanding question in the realm of DFT research is why devils' NK cells seem to not detect and attack DFT1 cells. This is especially intriguing as tumour seeding happens at bite wounds, where inflammation and secondary infections are common. On the other hand, DFT2 cells do express MHC class I molecules, raising the question of how this second lineage evolved to evade allorecognition. Of note, as DFT2 arose in a male host, females may be better equipped to reject the cancer cells presenting Y-chromosome-derived antigens.

Another aspect of DFT immune escape relates to the similarity of tumour and host MHC alleles. When these are very dissimilar, it is harder for DFT cells to impersonate a normal host cell. This hypothesis was put forward by observations of increased anti-DFT1 antibody responses in devils with MHC alleles most different from the cancer, although antibody responses alone were not found to be fully protective against cancer progression.

Finally, solid tumours often co-opt their local micro-environments to escape immune detection. The reciprocal interactions between tumour and host cells in the tumour microenvironment (TME) can have a supportive or inhibitory role in tumour initiation, progression, metastasis, and response to therapy. In recent years, TME diversity in DFTs has gained more attention, but several questions remain as to how diverse the TME is across devils, between primary tumours and metastases, and spatially within single tumours.

Not only can studying DFTs help us understand how these extreme transmissible cancers evolve to repeatedly escape allogeneic and anti-tumour defences in mammals, but it can also have strong implications for conservation. For example, around 80% of the population of Tasmanian Devils has been wiped out by this disease. However, there are exciting vaccine trials underway, spearheaded by the Save the Tasmanian Devil Programme and researchers at the Menzies Institute for Medical Research at the University of Tasmania. Newer research also suggests that the host population may be adapting to the devastating effects of these cancers.

DNA Thieving Cancers

An inherent characteristic of transmissible cancers is that they can reach very long lifespans. However, they multiply clonally, through classic cell division (mitosis), and so never undergo any sexual reproduction that would provide the chance to mix their DNA with that of another cell. A typical hallmark of cancer is genome instability, so this could pose an issue after centuries of mutations accumulating. Some of these mutations are potentially deleterious and would never be purged. But, once again, transmissible cancers prove to be remarkable as they can "renew" some parts of their DNA by acquiring it from their hosts along the chain of transmission. They mostly do so with mitochondrial DNA, where this gets acquired and replaces the original mitochondria of the cancer cells. This has been observed in many of the bivalves BTN, in CTVT, and in DFT1. What's more is that CTVT has even acquired pieces of chromosomes from a normal dog that was infected with it a few thousand years ago. That DNA is now incorporated into the cancer's DNA and so gets passed along with the tumour. If, tomorrow, a dog living in India was to get infected by this lineage of CTVT, it could then carry the DNA of no less than four different dogs! That is: 1) its own DNA in all the normal cells of its body; 2) the DNA of the CTVT cancer cells, which is in fact a mutated version of the DNA of the first ever dog to get CTVT, probably somewhere in Siberia or North America; 3) the few extra pieces of nuclear DNA that got incorporated from a dog that lived in the Middle East a few thousand years ago, and 4) the mitochondrial DNA of another dog that transferred its mitochondria to the tumour

somewhere along the chain of transmission.

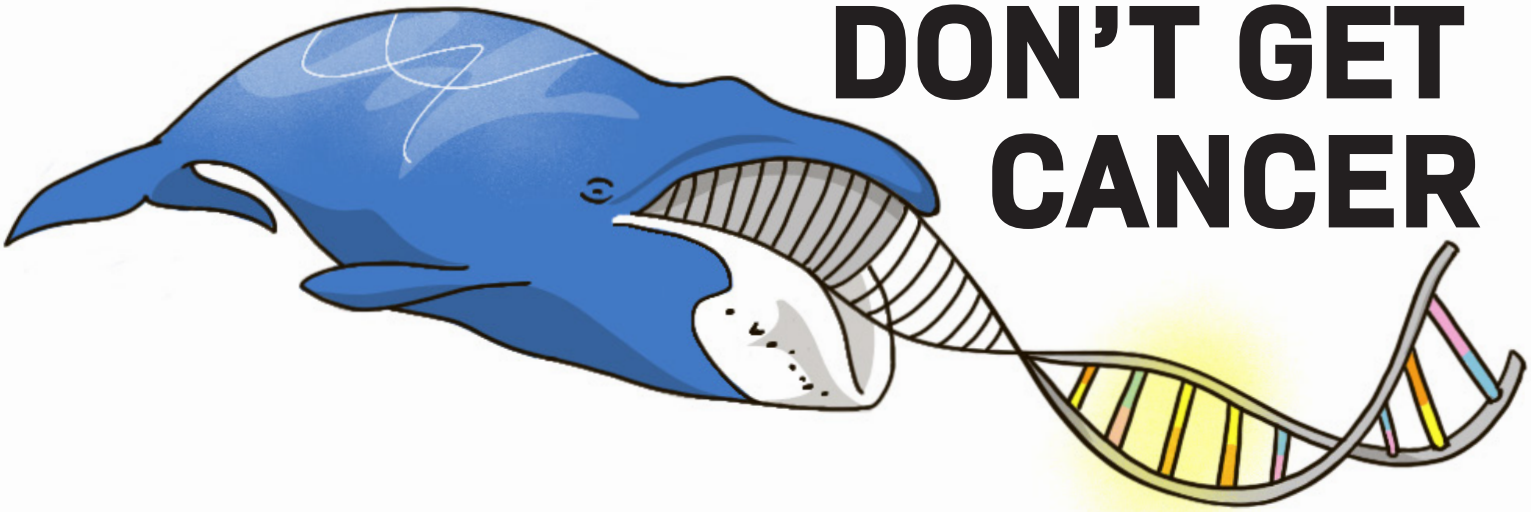
The Perfect Storm

The theme "Extremes" reminds me of the book, *Bitch: What Does It Mean to Be Female*, by the zoologist Lucy Cooke. Drawing inspiration from across the animal kingdom, Cooke argues that, when studying the evolution of sexual selection, characteristics that were long viewed as extreme were heavily framed by the prevailing social norms of the Victorian era. This raises the question of how our human-tinted lab goggles cloud the definition of an extreme. At the same time, extremes introduce creativity in scientific research. Naturally, if an "extreme" exists, it implores us to ask whether a "more extreme" is also possible. Transmissible cancers, to our knowledge, affect a limited number of species. In a sense, it's a game of odds to achieve the "perfect storm" of a transmission route and overcoming physical and immune barriers on repeated occasions over long stretches of time. Undetected transmissible cancers may have arisen in other species across the tree of life, with lineages dying out prematurely if they fail to meet these conditions. To illustrate, if a cancer is too aggressive, frequently causing host death, this poses problems for persisting in populations over long periods of time. All in all, transmissible cancers have perhaps evolved to become an "ultimate" cancer, exposing vulnerabilities in their affected host populations. The question of whether this is a true "extreme", or more common than we think, remains unknown. Nonetheless, it's an extremely fascinating phenomenon to investigate and explore.

Sophia Belkhir is a 3rd year PhD studying the evolution, genomics, and transmission of Tasmanian Devils transmissible cancers. She works in the Transmissible Cancer Group, at the department of Veterinary Medicine.

Nina Valenbreder is rounding off her first year as a PhD student in the Transmissible Cancer Group. Her project focuses on investigating tumour-host immune interactions in mammalian transmissible cancers.

THE SECRET TO WHY WHALES DON'T GET CANCER



Joshua Kocjancic Nelson discusses the mystery of why larger, longer-living animals aren't more prone to cancer, and reveals the diverse biological strategies species use to protect themselves from the disease.

Humans have approximately 1000 times more cells than mice. Similarly, some whale species have around 1000 times more cells than humans. Since cancers arise due to the accumulation of randomly occurring mutations in genes within cells throughout the body, it would be expected that cancer incidence in humans is 1000 times that of mice, with a similar fold increase in whales. Yet this is not the case. In fact, regardless of size, cancer prevalence is likely seen within 1-10% of any species population. This contradiction to what biological logic would suggest – that larger, longer-living organisms have more cells susceptible to harmful oncogenic (cancerous) mutations, and more time for these to accumulate – is known as Peto's paradox after the British epidemiologist Richard Peto. It is important to note that this phenomenon is not observed within species: research has revealed that being taller, though it may have some benefits, does come with a greater risk of developing tumours.

This puzzling observation has been studied across a variety of organisms. Suggested hypotheses for how large animals avoid cancer onset include lower mutation rates, increased numbers of tumour suppression genes, or perhaps alternative cancer-combatting mechanisms.

Potential methods of defence

Mutations are randomly occurring alterations to the DNA sequence. They can increase the risk of developing cancer if they affect the functionality of genes coding for proteins involved in processes such as cell division, metabolism, or cell survival. For example, the genes *p53, c-Myc,* and *KRAS* have all been studied extensively for their role across many forms of cancer. Reducing mutation rates in these genes would ensure the continued tight regulation of the aforementioned cellular processes and prevent aberrant cellular growth that would give rise to tumours.

Conversely, increasing the number of certain genes could also aid tumour suppression. Although this would increase the number of cancer-causing mutation targets, it would also increase the regulation occurring in each cell, helping to combat the effect of potential oncogenic mutations.

Back in 2012, a team at the University of Chicago headed by Dr Vincent Lynch, looked to understand the elephant's role in this paradox. Lynch wanted to determine if elephants – who have a notoriously low cancer rate –had more cancer defences than

other organisms. They decided to focus on the *p53* gene for this study. This gene is involved in the response to DNA damage, either stopping the cell from dividing until this damage can be repaired or inducing apoptosis, a form of cell death, if the damage is deemed too severe. They discovered elephants have 20 copies of *p53* compared to only one in humans. Lynch and his team then looked to the even more extreme end of the animal size scale, the whale, to see if this mechanism is conserved. Despite their large size and remarkably low cancer rate, whales only have one copy of *p53* like humans. Though *p53* likely plays a role in elephant defence against tumour onset, there appears to be no correlation between body size and numbers of these genes throughout the animal kingdom, suggesting an alternative mechanism.

Many other cancer-combatting strategies have been proposed, often in a species-specific manner. Though they may not be the biggest looker of the animal kingdom, the naked mole rat has a remarkable ability to withstand cancer and many other diseases too. These creatures, whose Latin name, Heterocephalus glaber, directly translates into 'different headed bald thing' have a lifespan of around 30 years, compared to only 2 years for their hairy relation – the rat. Many investigations have sought to understand this ability, with some suggesting a novel method of reducing cancer risk. Hyaluronan is a molecule found in naked mole rats, humans, and all other animals (as well as many upmarket skin care products). It exists between cells and, in these rats, it is five times as it is in humans and is not broken down actively by its enzyme, prompting scientists to theorise that this may factor into its tumour-combatting ability. They suggested that the increased size of hyaluronan acts as a barrier against cells congealing into a tumour; inducing cancer-associated mutations saw an upregulation of cell division but not of tumour formation, fuelling this idea.

These findings are cropping up across different species and inspiring scientists to further reveal the secrets that explain Peto's paradox.

Extreme sizes require their own solutions

Following on from the findings which suggested whales do not use extra *p53* gene copies to reduce cancer risk, a team of researchers at the University of Rochester, New York, recently addressed the question of what alternative method might be being utilised here. Reaching over 80,000 kg, the whale is the second largest organism on the planet. Paired with a lifespan of over 200 years, the bowhead whale is the ideal candidate to explore Peto's paradox.

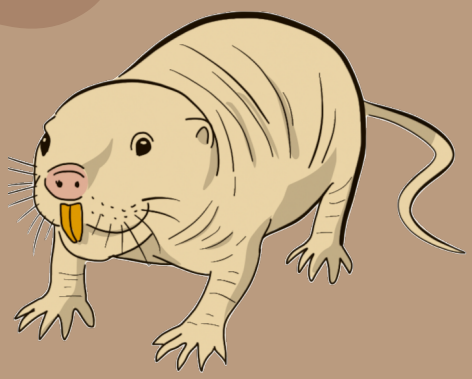
Their investigation got off to a slow start. Following the findings of Lynch's team, they initially proposed that an increase in apoptosis and *p53* activity could explain the reduced tumour risk and accommodate for the low copy number of the *p53* gene. However, they found no difference in either of these factors within whale cells relative to humans. Next, they questioned whether perhaps more mutation hits per cell in these cetaceans were needed to induce cancer transformation compared to humans. Again, no luck. In fact, whale cells transformed into tumour cells after 2 oncogene mutations relative to 4 on average in human cells.

Maybe whales have lower mutation rates than humans? Bingo. They found lower frequencies of less significant insertion-deletion mutations and of more structural variant mutations including severe deletions, insertions, and duplications. But how do whales avoid mutations despite their longer lifespan and large number of cells? Published in *Nature*, Vera Gorbunova and her team hypothesised the idea of improved DNA repair mechanisms. Species longevity has previously been associated with the relative ability to carry out double-strand break repair, making this a promising avenue to explore. They found that bowhead whales exhibit enhanced repair mechanisms when compared to mice, cows, and humans, which led the team at the University of Rochester to infer this may play a key role in whale cancer resistance.

Peto's paradox suggests there is no correlation between lifespan and size with cancer prevalence. Whether it is improved DNA repair mechanisms demonstrated in whales, higher numbers of gene defences against cancers in elephants, or the proposed idea of specialised molecules protecting naked mole rats against cancer, there also appears to be no pattern for how organisms explain this conundrum. With a number of unique cancer-prevention methods already found, who knows what other methods have evolved across the animal kingdom?

Joshua Kocjancic Nelson is a Developmental Biology MPhil student at Hughes Hall

Artwork by Loris Marcel

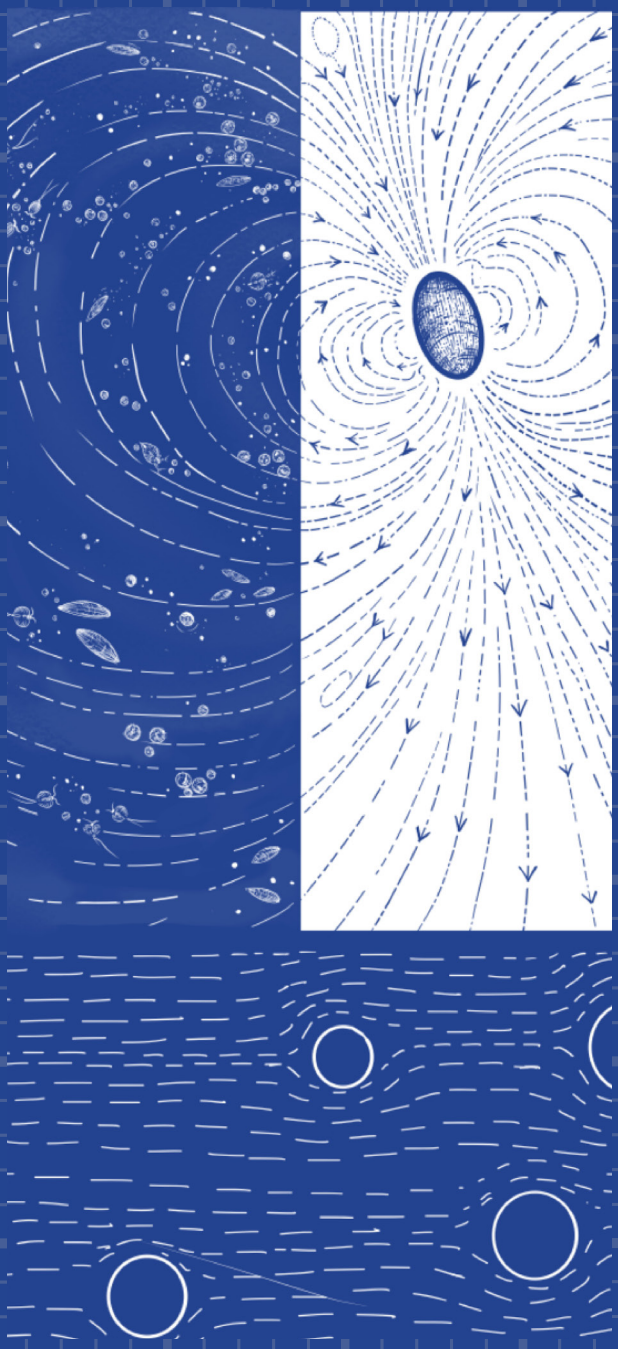


Speed Records in the Microbial World

Leonid Digel explores how microscopic life goes about its buisness at remarkable speeds.

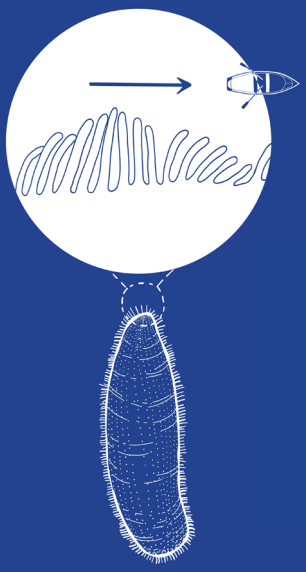
Big challenges at small scales

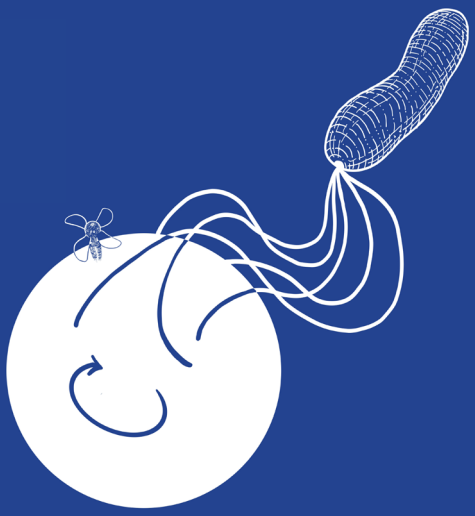
The Global Ocean is a vast reserve of biodiversity from the sunlit surface to the seafloor. Its very first microscopic inhabitants have been facing the same challenge since the dawn of time – finding food while making sure that waste does not accumulate around them. And the universal evolutionary solution to that has been to navigate and control the flow of water. At microscopic scales, liquid water behaves very differently to the way we are used to. For example, its viscosity starts to dominate over inertial forces, which scale proportionally with the size of the moving object. Queens' College Cambridge alumnus, Osborne Reynolds, described this relationship in the 19th century, which is now termed 'the Reynolds number'. Living at low Reynolds numbers, microbes experience nearly absolute absence of inertia, and without active propulsion come to a complete stop within a distance smaller than a hydrogen atom's diameter. Diffusion too starts to play a major role, but in stagnant waters nutrients become quickly depleted and diffusion alone is too slow to keep up with the speed of microbial consumption. Intuitively, two survival strategies come to mind; start moving and search for nutrients, or create water currents that can actively transport nutrients towards you. Both strategies have persisted through the course of evolution, and modern theories suggest that either strategy can provide sufficient nutrient flow. Here, I will focus on the microbes that chose to swim and have gone to great lengths in developing cellular machineries suited for the task.



How fast is extremely fast?

Awarding Olympic gold in swimming to a specific microbe is not trivial, because the differences in cell size play a big role here. Some of the fastest microbes cover less than a millimetre per second – a modest distance, until you realise the cells themselves are thousands of times shorter than that. Others swim even slower, but themselves are smaller too. To enable a more fair comparison across different sizes a helpful unit is used, which describes speed in body lengths per second (BL/s), i.e. if you are 1 mm in size and can swim at 50 mm/s, then you swim 50 BL/s. Using this measurement, predatory *Bdellovibrio bacteriovorus,* swimming at 160 BL/s, would leave the average human, at a mere 1–5 BL/s, far behind. In absolute terms, of course, they rarely move faster than a quarter of a millimetre per second. Turn the analogy around, though, and a human swimming at 160 BL/s would cross the Atlantic faster than an airplane, nearly boiling the water around them while they're at it. So how do microbes swim so fast?





Molecular propellers and oars

When it comes to motility, both eukaryotic and prokaryotic microbes have converged on using extracellular appendages that beat, rotate, or contract to push the cell in different directions. Well-known eukaryotic microbes, like *Paramecium* or *Volvox*, use numerous synchronously beating cilia. Prokaryotes, instead, mainly use rotating corkscrew-like flagella. The illustration above is a general comparison between the two mechanisms. To imagine how cilia work, one could picture a boat with people rowing, oars being the cilia surrounding a microbial cell. Bacterial flagella work more like motorboat propellers, but do remember that at the microscale there is almost no inertia. The fastest bacterium by absolute speed, *Candidatus Ovobacter* propellens, has as many as 400 flagella forming a tuft on one side of the cell allowing it to quickly navigate oxygen gradients at 1 mm/s. Although not as fast as *Ovobacter*, *Thiovulum majus* deserves an honorary mention here because they form a rather conspicuous whiteish veil in stagnant waters visible to the naked eye. They are fast, too, of course, with numerous flagella pushing them at 0.6 mm/s. *Magnetococcus marinus*, in contrast, has less than 20 flagella, but on each pole and, therefore, can push and pull at the same time reaching the speed of about 0.5 mm/s. And it has an internal compass, a chain of iron mineral crystals that allow it to sense magnetic fields (hence its name is *Magnetococcus*). Surprisingly, the aforementioned *Bdellovibrio* has only one flagellum.

While continuous swimming is one strategy, sudden thrusts that help with avoiding predators or catching prey are also very common among ciliated microbes. *Halteria grandinella* plankton, for instance, can perform swift jumps at ten times faster than their normal speed by doubling the beating frequency of their cilia, when disturbed. Others, like *Gymnodinium* or *Mesodinium* plankton, are ambush predators that usually sit and wait until another microbe swims by. The same strategy has also been proven useful in searching for environments with more abundant nutrients.

From the ocean into the lab

Despite gaining some insights into their physiology, we still know very little about these fascinating microbes. Even the absolute champion among bacteria, *Candidatus Ovobacter* propellens, keeps the prefix "Candidatus" next to its provisional name because it is yet to be characterised under controlled laboratory conditions in a pure culture. Learning from them, however, can help us design novel micro-robots for performing various tasks in viscous fluids or establish better architectures for microfluidic devices.

Aside from their use in microbial locomotion, cilia are also found in many human tissues where they perform protective or sensory functions. Discoordination in ciliary movement, or ciliopathy, is a frequent symptom of respiratory diseases, such as chronic obstructive pulmonary disease. The mechanisms controlling fluid flow via cilia are highly evolutionarily conserved from microbes to humans, so understanding ciliary beating in these diverse microorganisms can inform new treatments of clinically relevant ciliopathies.

The ocean's fastest microbes have spent billions of years solving problems we are only now starting to explore. I wonder what secrets lie beyond the microbial swimming championships?

Leonid Digel is a Carlsberg Foundation Postdoctoral Fellow at the Yusuf Hamied Department of Chemistry researching microbial electricity.

Artwork by Alexandra Kim

Ovobacter
~400 flagella
200 BL/s
1 mm/s

Thiovulum
~100 flagella
40 BL/s
0.6 mm/s

Magnetococcus
~20 flagella
500 BL/s
0.5 mm/s

Bdellovibrio
~1 flagellum
160 BL/s
0.16 mm/s

Extraterrestrial Prebiotics: How the Extremes of Space Forged Life

Julian Heuer explores how life began through chemical reactions on early Earth, and asks whether the ingredients for life came from our planet or the wider universe.

Every single aspect of life is governed by chemistry, more specifically chemical reactions. When we consider these reactions we tend to focus on the modern triumphs of humankind: from industrial catalysis and the synthesis of plastics, to the recent development of proteinogenic drugs. Centuries of scientific research has culminated in achievements that have transformed our lives simply by decoding the nature of chemical processes. However, chemistry didn't start with humanity.

Over billions of years, a sequence of complex reactions occurred, forging the diverse life forms we see today. With Earth forming around 4.5 billion years ago and the oldest evidence of terrestrial life dating back to approximately 3.7 billion years ago, life appears to have emerged with remarkable speed.

These findings refer, of course, to single-celled microorganisms. A prime example of early evidence comes from the Isua Greenstone Belt in Greenland, where ancient sedimentary structures resemble stromatolites — microbial reefs formed by sediment-trapping and directional growth. To date such ancient fossils, scientists rely on uranium-lead geochronology, which tracks the radioactive decay of U-238 to Pb-206 within zircon crystals. The more famous radiocarbon (C-14) dating cannot be applied here; with a half-life of only 5,730 years, it is limited to samples younger than 50,000 years. The age of these ancient geological structures exceeds that limit by a factor of 80,000.

The emergence of microbial life was only possible due to the existence of complex prebiotic compounds. Amino acids, lipids, sugars, and nucleotides represent the essential building blocks for life and are composed of carbon, hydrogen, nitrogen, oxygen, sulfur, and phosphorus. The actual chemical formation of these prebiotics is still a topic of active research and under heavy debate. The popular 1952 Miller-Urey experiment was an early attempt at simulating the conditions of prebiotic Earth to investigate the origin of life. It was proposed that the transition from Earth's first atmosphere, consisting of gases, to the second prebiotic atmosphere, was initiated by the cooling of Earth's surface from up to 8000K to the condensation point of water, forming a stable atmosphere and liquid oceans. Miller and Urey imitated this environment in a flask, half-filled with water and its headspace filled with methane, ammonia and hydrogen. Electrodes within the headspace discharged a continuous electrical spark of 60-70,000 volts, mimicking high-energy lightning. After only a single day, the solution was found to exhibit the amino acids alanine, aminobutyric acid and glycine. Follow-up experiments by Joan Oro expanded the Miller-Urey concept to the formation of adenine, a nucleobase. Carl Sagan introduced UV-photolysis into the system, tackling the "faint young sun paradox", which states that the young sun was shining 30% fainter (which would mean Earth would be completely frozen), but probably exhibited higher levels of UV-radiation. These findings represent an initial proof for the potential formation of prebiotic compounds on Earth and gave an explanation on the origin of life.

For a chemical reaction to occur, the right parameters must be satisfied. Temperature, pressure, and concentration must align with the reactivity of the reagents. The major criticism of the Miller-Urey model stems from the applied highly reducing atmosphere of methane and ammonia, while contemporary geochemical findings suggest a less reducing environment, mostly consisting of carbon dioxide and nitrogen gas. Given likely surface conditions of around 50–90°C and 10–100 bar, combined with the dilution of chemicals in vast oceans, the formation of life solely via this pathway is questionable. Consequently, scientists have looked to the stars for answers.

The hypothesis of Panspermia, the exogenous delivery of prebiotic compounds to complement the terrestrial emergence of life, has recently gained significant traction. For this, the field of astrochemistry investigates the formation of chemical species under extraterrestrial and interstellar conditions. From the viewpoint of an earth-bound organic chemist, the investigated reaction conditions are daunting. Synthetic chemistry on Earth for such prebiotic compounds typically is carried out between -40 to +200°C at atmospheric pressure and takes hours to days to complete. Extraterrestrial conditions are far more extreme. Temperatures in space can range between -270°C, the cosmic microwave background and the baseline temperature of space, to billions and trillions of degrees in supernova cores, which are responsible for creating ~10% of known mass and most elements in the periodic table.

While for most purposes laboratory vacuum is considered "clean" at 10^{-9} bar, intergalactic voids exhibit pressures as low as 10^{-22} bar. Molecular clouds typically show pressures of 10^{-15} bar, while at the upper end, neutron stars exhibit a staggering 10^{29} bar. In these neutron stars, the pressure is so high that even atoms collapse into their subatomic particles. However, the average interstellar pressure, like in molecular clouds, is so low that atoms statistically almost never collide. So, how does chemistry happen? Recent findings reveal that nature exploits specific phenomena to overcome these barriers. In the vastness of interstellar space, chemistry occurs on the nanoscopic surfaces of cold dust grains. In recent investigations, dust particles (composed of carbon, ammonia, and carbon monoxide) were placed in a vacuum chamber with 10^{-10} bar at -263°C, the condensation of carbon atoms on grain surfaces facilitates reactions such as the polymerization of aminoketenes into polypeptides[1]. This atomic carbon accretion is facilitated by the collision of carbon atoms and bonding in the cooling stellar outflow, forming amorphous carbon or polycyclic aromatic hydrocarbons, ultimately solidifying around the dust grain. These cosmic dust grains act as solid-state catalysts. Covered in a mantle of ices, they allow hydrogen atoms to "tunnel" through energy barriers, hydrogenating carbon into methane or nitrogen into ammonia. Simultaneously, bombardment by high-intensity UV light creates reactive radicals, which recombine to form complex organics like methanol or ethanol.

Another important aspect of life is chirality. Chiral molecules are identical in composition, but differ exclusively in atom arrangement, representing the mirror-image of each other. Terrestrial life utilizes exclusively left-handed amino acids, the L-enantiomer. The Miller-Urey experiments struggle to explain how a racemic mixture (50:50, left/right) could evolve into a L-enriched system. However, astrochemistry offers a solution. The Panspermia theory suggests that as meteorites drift near massive stars, they are exposed to circularly polarized light. Experiments confirm that this light interacts differently with chiral molecules, potentially degrading right-handed compounds faster and leaving an excess of the L-type precursors that seeded life on Earth.

Modern radio telescopes (such as ALMA) and the James Webb Space Telescope have identified key prebiotic precursors in deep space. Sugars like glycolaldehyde, nucleobases like uracil, and amino acids like tryptophan have all been detected in extraterrestrial environments. These findings suggest that the major building blocks of life are not unique to Earth but exist in extraterrestrial environments as well.

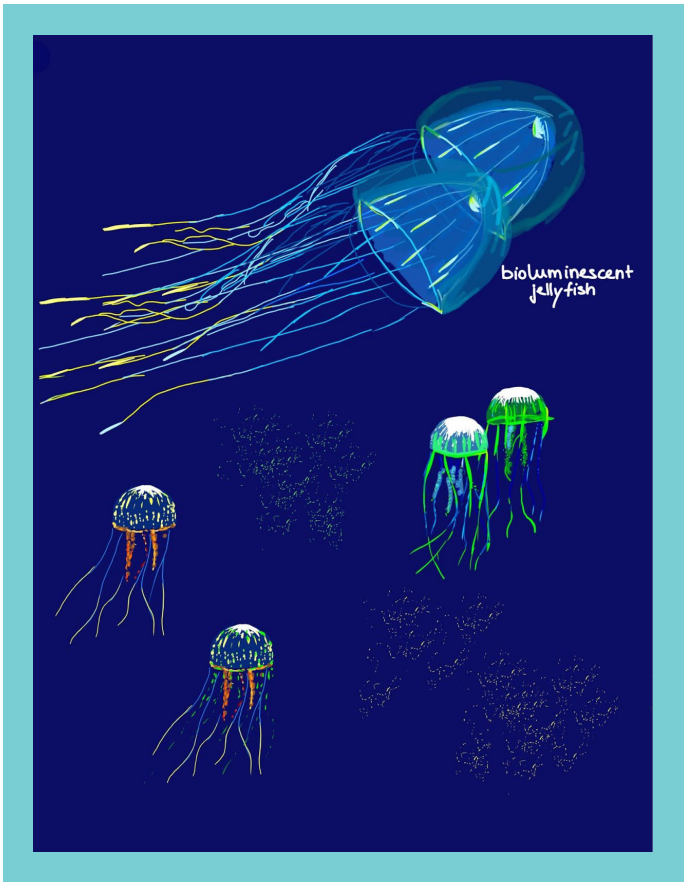
Our understanding of the extraterrestrial formation of prebiotic compounds and its connection with the terrestrial origin of life stands just at the beginning. As advanced technologies push the boundaries of what we can observe, many of our current hypotheses will likely be rewritten. The extremes of astrochemical reactions, from timescale to temperature and pressure, are far out of the comfort zone of our chemical understanding, that the discrete replication of astrochemical processes with our current tools is just impossible.

[1] Krasnokutski, S.A., Chuang, KJ., Jäger, C. et al. A pathway to peptides in space through the condensation of atomic carbon. Nat Astron 6, 381–386 (2022)

Julian is a postdoctoral researcher in the Department of Chemistry, specialising in redox flow batteries.

Hidden Glow – The Extreme Wonders of Bioluminescence

Article and artwork by Ella von Moeller



Swimming - the magic of bioluminescence

I remember that moment to this day. I was thirteen years old, and went for a night swim with my sister at a beach in Ibiza, Spain. It was dark, and we could see the moon reflected on the water surface. At some point, we noticed something strange – when we swam faster, the water around our hands started to glow with thousands of small bluish-green particles. We splashed our hands around in the water – and again – there were glowing drops all around us. As if we were swimming through liquefied glowing stars! Movement made the water glow; otherwise, the small particles stayed hidden and dark. I later learned that we were swimming through bioluminescent dinoflagellates, rare ecosystems of tiny glowing plankton. Bioluminescence is light produced by organisms through chemical reactions. Organisms emit light when two different chemicals react: luciferin and luciferase. In this reaction, luciferin reacts with oxygen and emits blue light. This reaction is triggered when the water around the dinoflagellates is disturbed.

Glowing living organisms in the deep sea

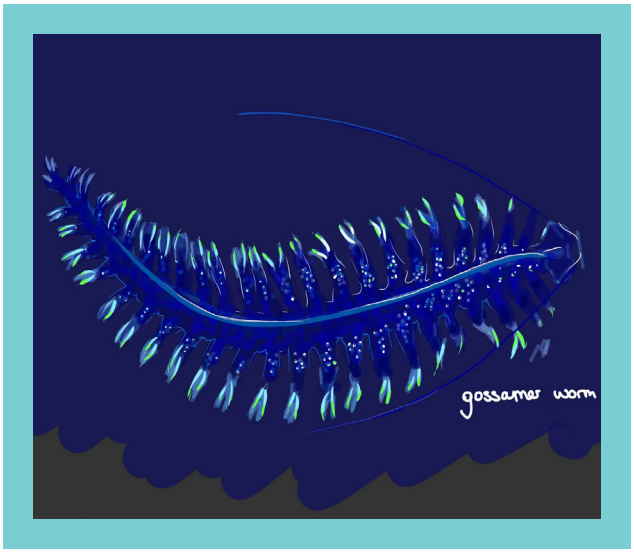
I couldn't fathom that I was only scratching the surface – quite literally – because the real glow was kilometres below me. Bioluminescence is used not only by small plankton, but a diverse range of marine species – bacteria, jellyfish and, in the deep-sea, even fish, which use it to find their way around! Marine biologist Steve Haddock, who works at the Monterey Bay Aquarium Research Institute (MBARI), says that "the deep sea is actually full of light". According to him, 90% of species living there might be bioluminescent! Fish use it to hunt and defend themselves. For example, the anglerfish uses it to attract prey, while other organisms, like the gossamer worm, can release clouds of bioluminescence to distract predators. Extremely dark and extremely glowing!

How the glow came into the vial

The glow of dinoflagellates is not only beautiful. A group of researchers at the Oceanographic Institute in São Paulo, Brazil, has proposed using them as indicators of chemical toxicity in water. With high concentrations of certain metals in the water, dinoflagellates cannot glow as strongly because their cellular metabolism is impaired – making them living indicators of toxicity.

Scientists' fascination with bioluminescence extends far beyond the deep sea. Already in the 1960s, the "glowing gene" was extracted from the jellyfish *Aequorea Victoria* for the production of the green fluorescent protein (GFP). This discovery was awarded the Nobel Prize in Chemistry 2008 and has revolutionized molecular biology ever since. The glowing molecule helps to trace molecular processes in real time using fluorescence imaging. There are now thousands of fluorescent proteins used as precise biological markers to trace molecules in the cell.

Whether we use it to detect toxins or study molecular processes in cells, we are still groping in the dark when it comes to understanding how these organisms really use bioluminescence to communicate and survive. Countless shimmering secrets are still waiting to be unveiled.



Ella von Moeller is a master student in Biomolecular Sciences working at the Cambridge Stem Cell Institute. Ella is currently developing a virtual human twin for drug safety assessments and has a special interest in Philosophy of Biology.

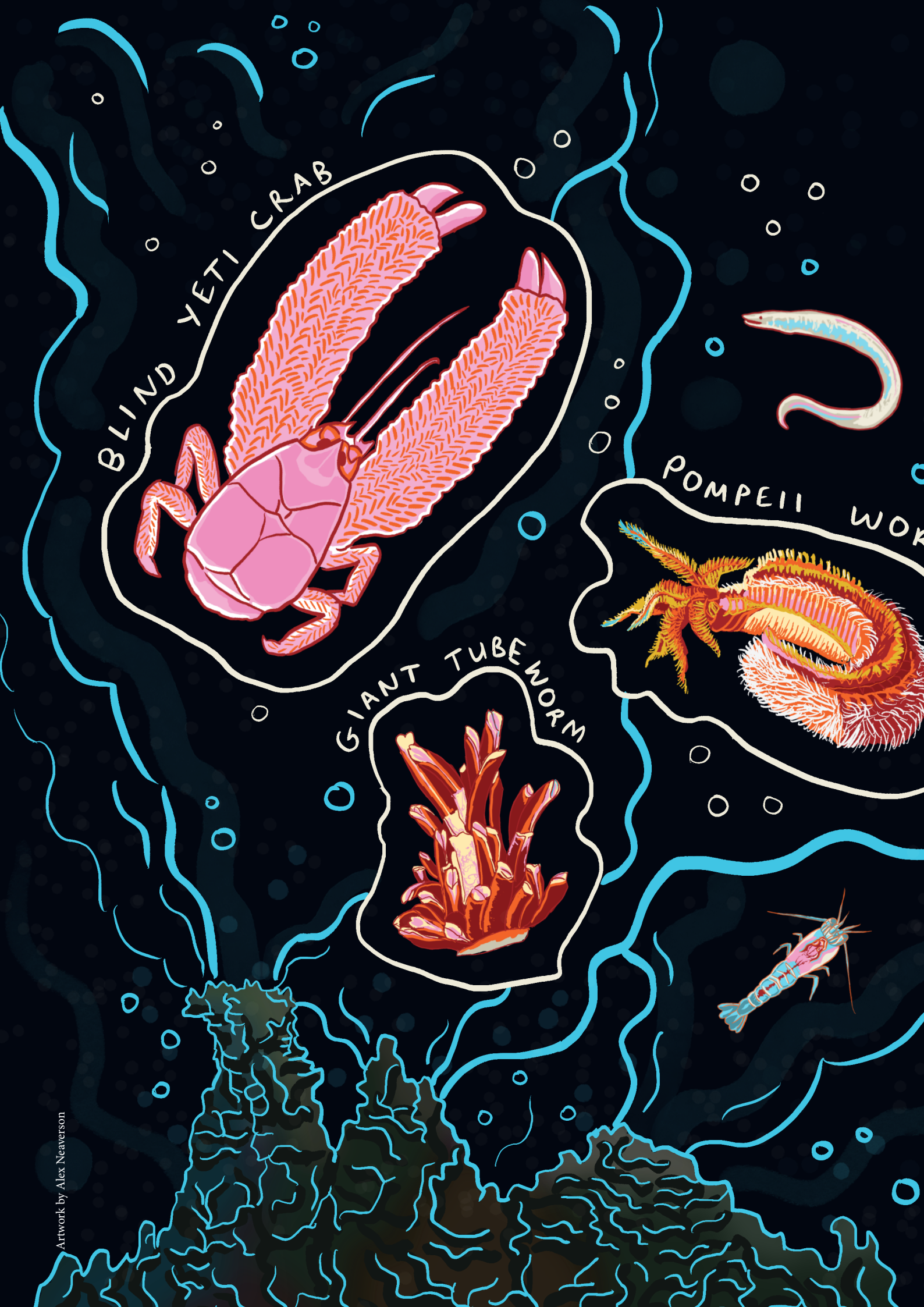
To learn more about these sparkling organisms - some recommended reads and videos:

Article: Chemistry World (February 2026) "Unravelling the chemistry behind the sea's bioluminescent sparkle" by Rachel Brazil

Video: MBARI (Monterey Bay Aquarium Research Institute). 2022. "Bioluminescence in the Deep Sea: How and Why Do Animals Create Their Own Light?"

Video: MBARI 2024. "MBARI Researchers Discover Remarkable New Swimming Sea Slug in the Deep Sea."

Video: PBS Terra "Why Does This Sea Glow in the Dark?"



Artwork by Alex Neaverson

Down where it's wetter

the extreme ecosystem in hydrothermal vents

Dhruv Shenai takes us thousands of meters beneath the ocean where extraordinary organisms thrive in scorching extremes by partnering with chemosynthetic bacteria that can turn inorganic material into food.

It's difficult to imagine a silent Earth—one where the fauna and flora are muted by a drastic reduction in biodiversity. Yet increasingly, with climate change and other ecological pressures, the news of another species dying out is becoming all too common. Extinction, once associated with large cataclysmic events and long timescales, is now being reported in real time.

Even the deep seafloor, seemingly removed from human influence, is affected by pollution and ecological damage [1]. Furthermore, as demand rises for the rare metals used in modern technology, the deep sea is becoming a potential site for industrial exploitation [2]. Without preaching to the choir, it's imperative that we protect not just the ecosystems we see, but also those less obvious to our daily lives.

The deep sea is home to a diverse set of animals and microbes, many of which remain undiscovered. One of the most remarkable examples of deep-sea ecosystems are hydrothermal vents, and analysis of these structures reveals the true beauty of evolution and biochemistry.

Hydrothermal vents are often formed at boundaries of tectonic plates, which are moving apart. Water can seep through these fractures in the Earth's crust and be expelled as a hot fluid through the vent structures. The vent fluid can reach temperatures as high as 400°C and pressures hundreds of times greater than atmospheric [3]. With such extreme conditions, it is astonishing to observe the adaptations that life has evolved.

Extremes

Though the superheated water can reach up to 400°C, this quickly mixes with the surrounding water, creating a temperature gradient. Therefore, many members of the vent's ecosystem inhabit patches of water with a more hospitable temperature of about 20°C.

Many hydrothermal vents are found hundreds to thousands of metres below sea level and thus experience high pressures. Researchers therefore use remotely operated vehicles (ROVs) to image and collect samples of the environment.

No plants?

Compared to our usual English landscapes, the lack of our green chloroplast friends may worry us. How is life supported with no light and no photosynthesis? The answer is chemosynthetic bacteria. In chemosynthesis, inorganic chemicals from the vents, such as sulphur compounds, are oxidised, turning carbon dioxide into sugars. This process is the fundamental basis for life around hydrothermal vents.

These chemosynthetic bacteria are also found in some giant tubeworms that live around hydrothermal vents. They completely lack any ingestion or digestion organs, instead relying solely on these bacteria to produce food. This is probably one of the most extreme examples of a symbiotic relationship in nature. Tubeworms have also evolved special haemoglobin that binds to hydrogen sulphide, allowing the transfer of sulphur to the chemosynthetic bacteria whilst circulating oxygen for respiration.

Blind yeti crab

Like the giant tubeworm, yeti crabs also have evolved under the extreme conditions of no sunlight. They have strongly reduced eyesight, a result of natural selection and the lack of light at that depth. Also known as yeti lobsters, they crawl along the seafloor and wave their hairy arms over the vents, collecting sulphur and other toxins. Their pincers contain hair-like structures known as setae, which house chemosynthetic bacteria and create food from the toxins.

Using isotope analyses, researchers have identified that the yeti crab likely intermittently consumes these bacteria for food, hence it is another example of a symbiotic relationship. There is plenty of ongoing research on the effects of these animals and microbes on the sulphur cycle, and their role in the ecosystem is fascinating and undeniable.

Pompeii worms

Pompeii worms make their homes directly near the superheated streams of hydrothermal vents [4]. Their superior resistance to heat makes them extremophiles. They live happily in temperatures above 50°C due to a layer of bacteria which provides protection. Named after the Roman city Pompeii, these animals build their own tube-like homes by secreting their epidermis. It forms plywood-like layers that also strangely incorporate chemosynthetic bacteria, which probably was trapped in the epidermis of the worm before being shed. Researchers can identify this by tracing the sulphur in the tube structures.

The Pompeii worm lives by keeping its head exposed to cool water (around 20°C) whilst its tail gathers sulphur and other materials for food (at higher temperatures of the superheated fluid). It's hypothesised

that cool water is circulated around the worm to keep it cool. However, a full analysis of this is difficult, since samples of the worms can't be collected easily as changes in pressure would damage the worm's internal structure.

Types of vents

There are several different types of vents, but the most common are black smokers and white smokers. When superheated water, with dissolved metallic compounds, reaches the near-freezing deep ocean, the metal particles precipitate into clouds of tiny particles. This gives the smokers their colours: black because of metal sulphides and white because of barium and calcium. Therefore, white smokers tend to be alkaline whereas the black smokers are often more acidic. The other difference is their temperature, black smokers reach temperatures up to 400°C whereas white smokers only release fluid closer to 100-300°C. Due to the high pressures, the water is superheated and doesn't boil even at these temperatures.

Pompeii worms, yeti crabs and tubeworms are commonly associated with black smokers, though many similar organisms inhabit white smokers. Vent type and chemical makeup dictate which chemosynthetic bacteria can thrive within it.

Hydrothermal vents and the origin of life

In the 1920s, scientists Alexander Oparin (1924) and J.B.S. Haldane (1929) hypothesised the idea of a primordial soup, suggesting that the basic building blocks of all life, like amino acids, were created from inorganic compounds under ultraviolet light [5]. Following this initial conjecture, further studies have proposed hydrothermal vents as a possible alternative source of these conditions, since vents provide large chemical gradients and stable energy sources. Unfortunately, the limited evidence we have of early life in fossil records limits any certainty we can attribute to this origin of life theory.

Sustainability and conservation

Though they exist globally, hydrothermal vents are rare habitats, as they're small and scattered geographically. Therefore, many species are only found in one specific location. The fragility of the hydrothermal vents makes them vulnerable to destruction: they rely on a continuous source of hydrothermal fluid and therefore on tectonic activity [6]. Vents can sustain themselves for a long time or become inactive after short time periods and so life for the ecosystem is conditional. Therefore, any human intervention could disrupt these sites.

Deep sea mining aims to obtain valuable mineral deposits from the surface of the ocean floor. Hydrothermal vents are a source of polymetallic sulphides which can be harvested for their concentrated source of metals. However, it is suggested that mining with the equipment currently developed would damage the ecosystem. Firstly, it would directly kill many sessile species that exist near the sulphide deposits and secondly, the plumes of sediment from the mining waste can smother and suffocate nearby life. There is also the problem of light and noise pollution, which could disrupt organisms in unpredictable ways [7]. Even if mining is proven to have a low impact, the cumulative effect of resource extraction, deep-sea tourism, and trawl fisheries are unknown.

Hydrothermal vents are a reminder that life can find a way to survive in the harshest of conditions, and possibly even start there. Yet, despite its resilience, it is not indestructible and the ecosystems remain fragile. Awareness is growing, and many scientists and environmental organisations argue that conservation is paramount, stating that deep-sea mining should not proceed without far stronger evidence that these rare ecosystems can recover.

If the deep ocean does become a site for mineral extraction, we may lose species before we even discover them, possibly destroying the secrets to our own early origins.

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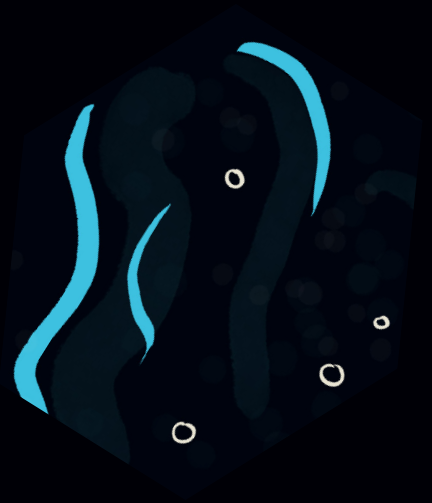

Dhruv Shenai is a 4th year materials science undergraduate.

The Loneliest Places in the Universe – Cosmic Supervoids

Written by Dily Duan Yi Ong

To the public imagination, the study of the universe is tied to its most luminous and massive observables, such as the searing plasma of stars, the spiral arms of galaxies and the violent accretion disks of black holes. However, to understand the universe's true structure, we must look at the extreme opposite: the vast, cold expanses where matter is extraordinarily sparse. These are cosmic voids-the loneliest places in the universe.

On the largest scales, the universe resembles a three-dimensional spiderweb. Gravity draws matter into dense filaments and leaves behind bubble-like regions of emptiness. Our standard model of cosmology predicts statistically how these voids should grow. We expect them to follow a pattern where most are of average size and very few are extremely small or extremely large. However, observational data has revealed the existence of supervoids so vast that they are rare in the standard cosmological model [1].

These are one of the universe's true extremes: while a typical void might span 50 to 100 million light-years, supervoids can stretch for over a billion light-years or more [2,3]. One such candidate is the Eridanus Supervoid. This region is not only an abyss of emptiness but is also linked to the Cold Spot in the Cosmic Microwave Background - a mysterious chill in the afterglow of the Big Bang that is colder than typical regions in standard cosmological predictions.

Why do these extremes matter? Because they let us stress-test our models where they are most delicate. If the universe began with tiny, random quantum fluctuations, structures as extreme as supervoids are rare [1]. This creates a tension worth examining. It forces us to ask uncomfortable questions. Is our understanding of gravity on the largest scales correct? Is dark energy behaving differently in these extreme environments?

These rare giants act as a useful lens on dark energy. As photons pass through a supervoid, they traverse a region where matter is sparse and cosmic expansion dominates. This can leave a subtle imprint on the light known as the Integrated Sachs-Wolfe (ISW) effect, which can be used to test how cosmic acceleration influences the growth of structures [4].

By hunting for the largest, emptiest and most unusual patches of the sky, we are stress-testing the laws of physics. By staring into the extremely lonely places, we might just find one of the clearest windows into how our Universe works.

Dily Duan Yi Ong is a final year PhD student in Physics, specialising in machine-learning-enhanced Bayesian inference in cosmology.

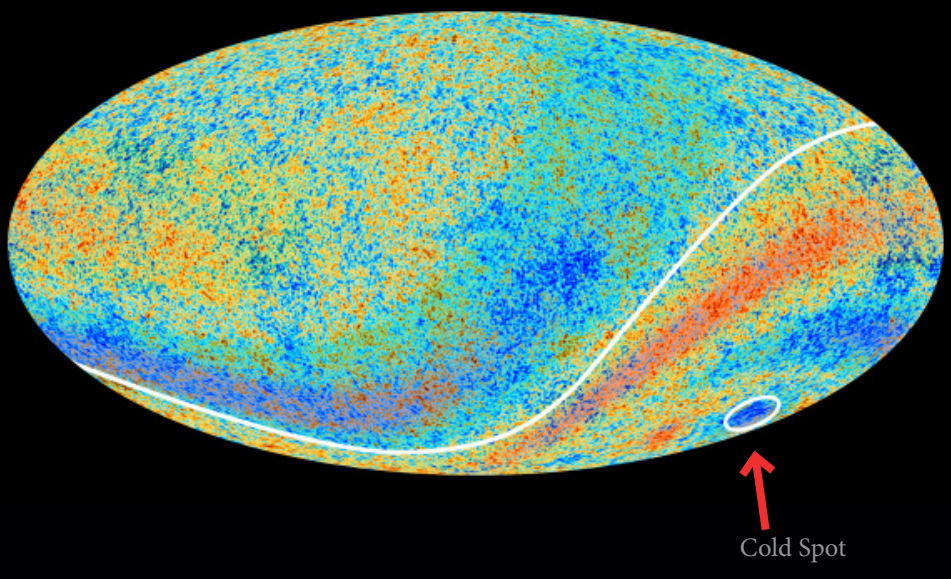

Figure: Planck's map of the cosmic microwave background, the faint afterglow of the Big Bang. The highlighted region marks the Cold Spot, an unusually large cooler patch in the sky [5].

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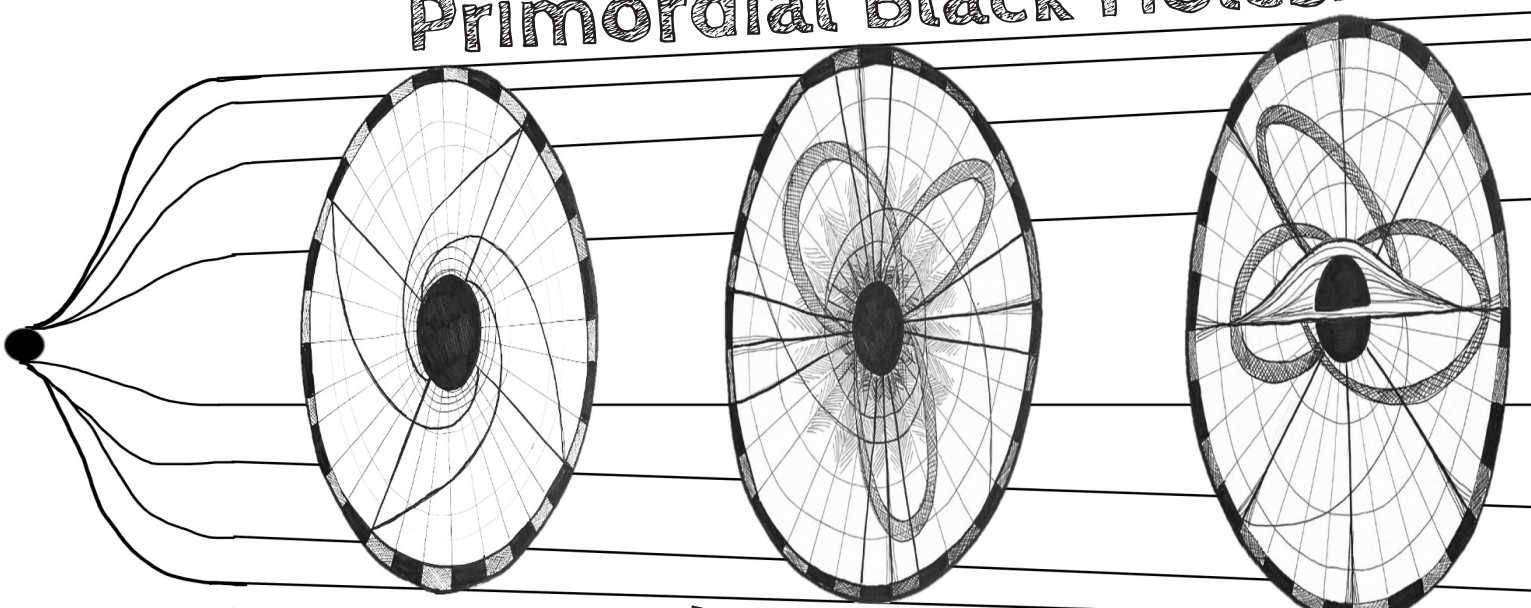
Primordial Black Holes: a tale of extremes



Artwork by Sophia Jaffer

Merging the studies of black holes and the origin of the universe, Yoann Launay explores what we know about a theoretical entity—one that weighs as much as a mountain yet fits within the size of a proton—and what it may (or may not) tell us about dark matter and the origin of time.

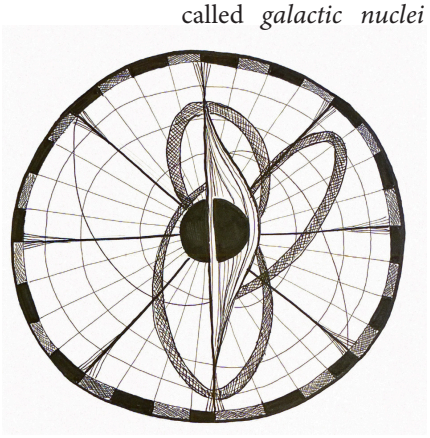
There is a reason why Stephen Hawking spent his life torn between the study of the origin of the universe and of black holes, a region in space with gravity so intensely strong that supposedly nothing, including light, can escape. As one might infer from his most famous book, *A Brief History of Time*, both phenomena challenge our understanding of time, and their study is inextricably intertwined.

It is true that time loses its standard meaning in both contexts; while one infinitely stretches the time difference between the observers it absorbs and external ones, the other prescribes the birth of time itself. One of Hawking's great ideas was the so-called no-boundary proposal, effectively removing the notion of time zero and merging it with the notion of space, inspired by what is—in theory—seen inside a black hole. Black holes also have what we call a *horizon*, similarly to the fast-expanding newborn universe, defined as a limit beyond which communication with the outside world is impossible. In the former case, it is impossible to escape because the gravitational field is too strong even for light, while in the latter case, light is too slow to keep up with the universe's expansion. However, the origin of the universe and black holes have more in common than an extreme notion of time or causality. In fact, their greatest common trait is their high energy density.

The high energy density of a black hole is due to its essence: in most cases it is the natural outcome of a massive star whose internal pressure cannot overcome gravity. When a star exceeds a critical mass compared to its size, there is too much mass per unit volume, and the sheet of spacetime 'breaks' to form a black hole, a singular region of spacetime compacted into a few kilometers. We see a similar case with the origin of the universe when going back in time, or equivalently, reversing the expansion. If we start from today and assume that the total mass and energy in the universe is unchanged, we see that the universe becomes denser or more compact as we trace back to the origin, which also means it was hotter. In both scenarios, this represents the modern theorists' playground: the limit where there is so much mass that gravity dominates, but also where the typical scale of phenomena approaches that of fundamental particles. At such small scales, physics becomes deeply strange: objects no longer have fixed positions or speeds, but exist as clouds of probability, and can interfere with one another in counter-intuitive ways. When we try to bring gravity into this picture, we arrive at one of the deepest open questions in all of science: *quantum gravity*.

But before reaching the need for those theories that many theoretical physicists dream of finding and testing, keeping quantum effects at a reasonable level for both scenarios was something that proved very fruitful in Hawking's breakthroughs. Among the most famous formulae of physics never directly measured is that of the so-called Hawking temperature or Hawking effect. It states that tiny quantum effects near the horizon of a black hole allow some radiation to escape its gravitational pull. As a natural consequence, this can lead to the black hole evaporating completely, contrary to common beliefs of an endless and Gargantuesque growth, as suggested in Christopher Nolan's *Interstellar*. Those supermassive black holes are indeed the most common type, formed from star collapse or mergers in matter-rich zones of the universe. The laws of clustering from gravity cause dense patches of the universe to become even denser over time, which, coupled with cosmic expansion, give rise to a cosmic web of

galaxy clusters: ideal environments for immense black hole growth. So-called *Supermassive black holes* usually sit at the core of a galaxy's engine, absorbing surrounding material while also being central to what is called *galactic nuclei*



feedback, which influences the energetic balance of the entire galaxy. One might imagine that bigger black holes would produce more Hawking radiation, but it is in fact the opposite, as Hawking radiation is a quantum phenomenon inversely proportional to mass. In fact, for a black hole of about one solar mass (a medium sized black hole formed from stellar collapse), it would take approximately [1 followed by 67 zeros] years of radiation to completely evaporate. That is roughly [1 followed by 57 zeros] times longer than the current age of the universe (approximately 13.8 billion years)! Such a black hole would have a surface temperature [1 followed by 11 zeros] times colder than that of the Sun's–deserving the name 'black' twice: once for absorption, once for its feeble glow. For a supermassive black hole, the evaporation time scales with the cube of the mass, reaching [1 followed by 100 zeros, or a googol] years or more for the largest known ones.

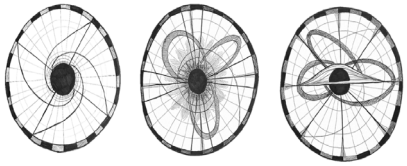
One can see why Hawking's law has had very little chance of direct measurement yet, although its formulation is among the few which unify Newton's gravitational constant (G) and Planck's quantum constant ($\hbar$).

Historically, the way out of this frustrating limit was to assume the existence of black holes that would have already evaporated today, or alternatively, black holes light enough that they could constitute an important component of our universe. In both cases, such light-mass black holes are unlikely to have anything to do with modern stellar collapse and must have had a primordial origin: these are called Primordial Black Holes (PBHs). Today it is believed that their most likely source is a *primordial form of dark energy* named *inflaton*. *Primordial* here refers to the era predating our universe with its current constituents (known particles such as electrons, protons, quarks, photons etc). *Dark energy* designates an unknown repulsive force of the vacuum that accelerates the expansion of the universe (and it does so faster than today's dark energy). Some properties of this primordial dark force could have triggered the creation of ultra-dense regions of energy, despite the expansion pulling things apart. After the transition to modern matter, light, and other constituents, these regions are too dense, thus creating black holes. This transition proceeds such that compact patches freeze first and then collapse when unfreezing, the lightest primordial black holes being the first to appear. For these objects to be the lightest, they also need to be the smallest. For some of them, we are talking about a proton-size, with the weight of a mountain. It is difficult to go more extreme.

The challenge with the transition hypothesis is that practically any mass range for PBHs is theoretically acceptable and should all be investigated whether we expect them to produce detectable Hawking radiation or not. In fact, one way to detect a black hole is via the surrounding spacetime distortion it creates (the phenomenon of gravitational lensing, producing for instance so-called *Einstein rings*), as was seen in recent experiments. Alternatively, when black holes merge or absorb a star, gravitational waves (ripples or waves of spacetime deformation) are emitted during their spiral cosmic dance and can nowadays be measured.

What can PBH astronomers and cosmologists claim today? Some are still pursuing direct measurements of Hawking radiation, thinking that (now-dead) PBHs could be radiating in the spectrum of *gamma-ray bursts*, puzzling radiation measurements that have baffled researchers for decades. This remains highly hypothetical and contested. In recent years, PBH science peaked when they were considered a potential candidate for dark matter, which makes approximately 27% of our universe's content, compared to ordinary matter's 5% (the rest being a modern form of dark energy). Dark matter is the mysterious substance whose gravitational effects we can measure but which we cannot directly see, and which is needed to explain—among other things—the expansion of the universe as we measure it. PBHs fit the dark matter profile nicely because they do not collide like ordinary matter (behaving like ghosts) and they absorb light. However, for most of the mass spectrum, observations have constrained PBH abundance by not detecting any PBH gravitational lensing with our current telescope resolution. With these results, PBHs can only explain a tiny fraction of dark matter, except for two very narrow mass ranges: those around the mass of a large asteroid (which are extremely difficult to detect) or those with about 20 to 100 times the mass of the Sun. Such narrow ranges are generally not favored by physicists, especially for such a continuous phenomenon. This makes it clear that a lot of researchers now believe PBHs are ruled out as the main component of (dark) matter, although they remain tied to it as one minor possibility in the mix.

On the theoretical side, mostly among those studying the Big Bang, skepticism about PBH discoveries has persisted for a long time. Some would indeed claim that PBH predictions are not mature yet and are based on questionable assumptions or just because predictions from one year to the other vary by multiple orders of magnitude. However, rigorous work in recent years has given hope that these rare events could be fully understood one day. Most importantly, not observing a phenomenon still helps placing constraints on our theories, and that is why such theoretical and experimental investigations are vital to science, with the benefit of doubt. What is at stake is not so much Hawking's work on black holes or Hawking radiation itself, but rather what they say about their birth: the origin of time itself, even if PBHs might have never existed.



Yoann Launay is a final year PhD student in Theoretical Cosmology. His research focuses on the early universe.

COSMOLOGY'S NEEDLE IN A HAYSTACK: TUNING INTO THE FAINTEST WHISPER OF THE EARLY UNIVERSE

Jacob Tutt and Harry Bevins explore how the 21-cm signal from neutral hydrogen helps uncover the history of our universe.

The last few decades have seen the study of our universe transform from a largely theoretical frontier into a data-rich reality, marking a definitive shift into the era of 'precision cosmology'. With access to a plethora of datasets across the electromagnetic spectrum, astronomers are now able to examine various periods of cosmic history with unprecedented scrutiny ... yet a crucial chapter remains largely uncharted.

Our earliest observational record exists as a snapshot of relic radiation left over from the Big Bang, known as the Cosmic Microwave Background (CMB). This primordial blueprint from when the universe was around 380,000 years old reveals an epoch of simplicity, in which matter and radiation exhibited density fluctuations of just one part in 100,000, reflecting an extraordinary degree of uniformity. In striking contrast, modern surveys mapping the nearby cosmos paint a very different picture: a richly structured cosmic web of galaxies, filaments, and vast underdense voids.

Despite these two well-characterised bookends enabling stringent constraints to be placed on the fundamental parameters governing the Universe's evolution, much of the vast interval of cosmic history separating them remains unexplored. As a result, many fundamental questions are left unanswered. Theoretical models suggest that this period, spanning from the formation of the CMB to roughly one billion years after the Big Bang, can be divided into three distinct epochs. The earliest of these, the 'Dark Ages', saw initial gravitational instabilities slowly collapse, giving rise to the first structures. Within the hearts of these newborn haloes, the first stars ignited, marking the onset of the 'Cosmic Dawn'. Their radiation then gradually transformed the surrounding intergalactic medium, culminating in the `Epoch of Reionisation', when the Universe transitioned from a largely neutral state to the ionised, transparent cosmos we inhabit today. However, the timing of the major milestones in this transformation, and the pace at which they unfolded, remain shrouded in mystery.

While the unprecedented sensitivity and angular resolution of the James Webb Space Telescope is currently revolutionising our understanding of the closing chapters of this period, even it, and future observatories, face fundamental physical limitations. The first flickers of light from the 'Cosmic Dawn' are simply too faint — and too deeply veiled by primordial gas — to remain within reach of direct detection.

However, hope is not lost. Where traditional telescopes see an impenetrable fog, radio observatories can hear an articulate broadcast.

Astrophysicists have shown it is possible to uncover the properties of the earliest populations of stars, galaxies, and black holes not through direct observation, but by studying the imprint they left on the neutral hydrogen that once permeated space. At the most fundamental level, hydrogen's constituent proton and electron both possess a quantum property known as `spin', and their relative alignment places the atom in distinct energy states. The exceptionally subtle transition between these two states with aligned and anti-aligned electron and proton spins allows hydrogen to absorb or emit a photon at a characteristic radio frequency of 1.4 gigahertz, corresponding to a wavelength of 21 centimetres. Although vanishingly rare for any single atom, this transition, integrated over the immense volume of neutral hydrogen in the infant Universe, produces a measurable statistical signal that appears in absorption or emission against the background radiation from the Big Bang.

The strength and evolution of this signal promise a unique way to address some of the most fundamental questions remaining in astrophysics and cosmology. When did the first galaxies form? How big and bright were they? How did the light that they emitted affect the surrounding gas and formation of subsequent generations of galaxies? How quickly did exotic objects such as X-ray binary and black holes form and how abundant were they? Each of these possibilities would leave a distinct imprint on the depth, timing, and overall shape of the 21cm signal, offering a powerful way to test competing hypotheses about the origin of the first structures.

Crucially, it is cosmic expansion that makes the 21cm signal from neutral

probe. As the Universe expands, the signal is stretched to progressively longer radio wavelengths, allowing astronomers to trace its evolution and reconstruct a timeline of early structure formation. However, this very stretching also renders the signal notoriously difficult to observe, shifting it into a frequency range dominated by foreground emission (from our own galaxy and beyond) that is four to five orders of magnitude brighter.

What remains is only a faint murmur, nearly lost beneath far louder foreground emissions.

Researchers at the Cavendish Laboratory, the Institute of Astronomy, and the Kavli Institute for Cosmology are pursuing this elusive signal through two complementary approaches. The Radio Experiment for the Analysis of Cosmic Hydrogen (REACH), a collaboration between the University of Cambridge and Stellenbosch University, aims to use a highly stable radio spectrometer to detect the so-called global signal. Deployed deep within the quiet, sparsely populated plains of South Africa's Karoo Desert — where human-made radio interference is kept to an absolute minimum — REACH measures the evolution of the sky-averaged emission across cosmic time. Isolating this faint imprint from the inescapable radio foreground and interference like FM radio is, however, an extraordinary challenge. Success demands an unprecedented understanding of both the sky itself and the instrument used to observe it, driving the development of sophisticated data-analysis and machine learning techniques, alongside state-of-the-art precision radio engineering.

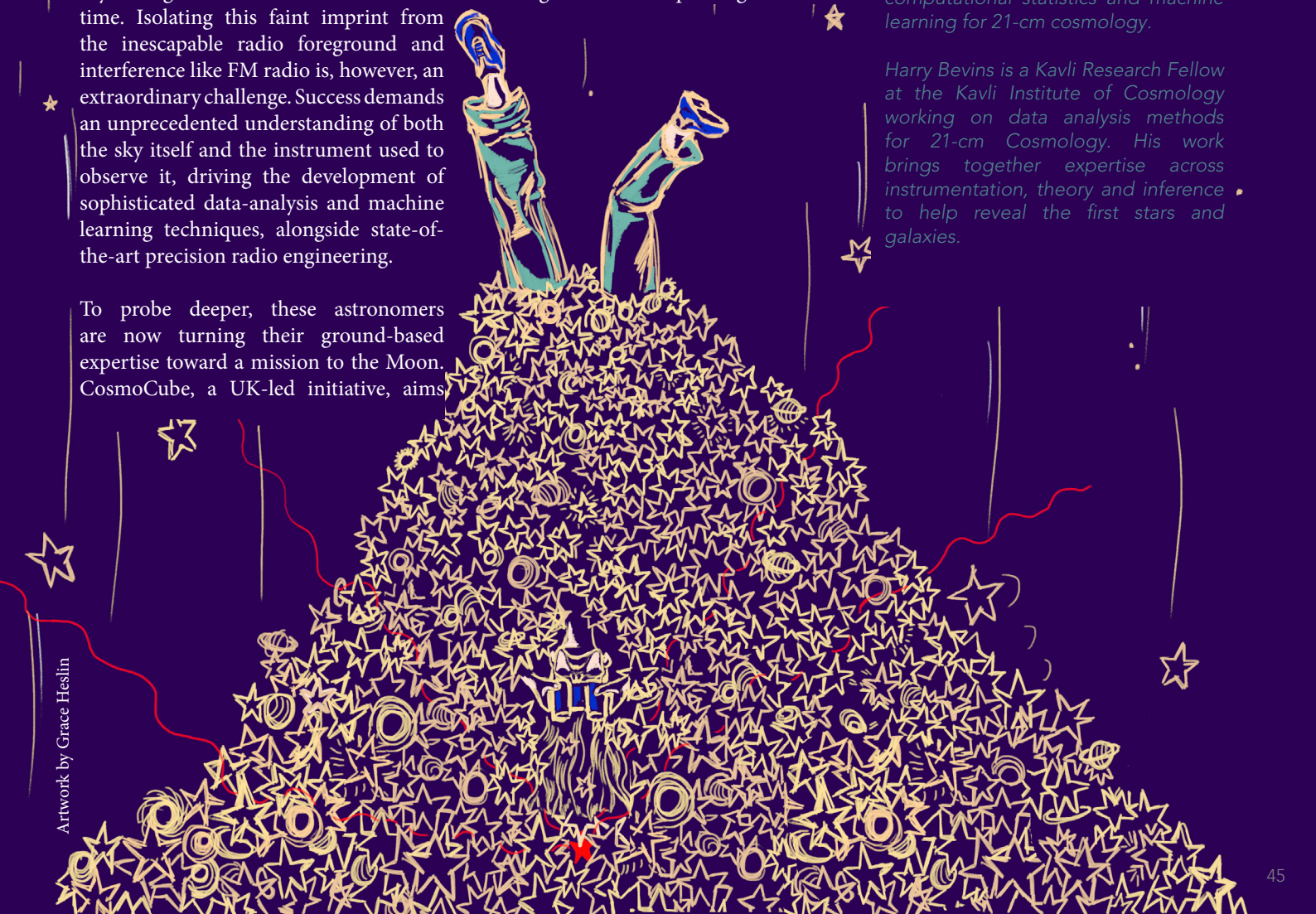
To probe deeper, these astronomers are now turning their ground-based expertise toward a mission to the Moon. CosmoCube, a UK-led initiative, aims to exploit one of the quietest radio environments accessible: the lunar far side, permanently shielded from Earth's incessant radio chatter and free from the shimmering distortions of Earth's ionosphere. From a CubeSat stationed in this uniquely silent setting, CosmoCube will listen for the faintest whispers from the Universe's earliest and most elusive chapter before even the first stars formed, revealing the nature of dark matter.

While instruments like REACH and CosmoCube look for the sky-averaged signal over time, radio cosmologists are also looking for the spatial fluctuations in the 21cm signal. Using vast networks of radio antennas, they aim to assemble a three-dimensional map of the Universe's earliest structures through tiny variations in the emission from hydrogen across the sky and through cosmic epochs. Achieving the required sensitivity has motivated the construction of the Square Kilometre Array (SKA), the largest radio telescope ever built. Spread across the remote landscapes of Western Australia, the SKA's low-frequency component alone will consist of more than 131,000 antennas operating in unison as a single observatory to detect the 21cm signal. Often dubbed the ultimate 'Big Data' challenge, the SKA is pushing the limits of modern computing, with data rates expected to exceed 150 terabytes per second – enough data to fill around 2.5 million laptops every day. At this scale, the challenge becomes as much computational as it is astronomical. Extracting a faint cosmological signal from such overwhelming data volumes is catalysing advances in high-performance computing, statistical inference, and artificial intelligence to recognise subtle patterns buried deep within noise.

While the challenges of detecting the 21cm signal are immense, success from REACH, CosmoCube, the SKA, or one of the many other instruments targeting this signal would reveal a formative chapter of cosmic history that has so far remained beyond observational reach. Such measurements promise to transform our understanding of the Universe, shedding light on its expansion, the role of dark matter, and the emergence of the first stars and galaxies.

Jacob Tutt is a PhD student at the Cavendish Laboratory and the Kavli Institute for Cosmology, researching computational statistics and machine learning for 21-cm cosmology.

Harry Bevins is a Kavli Research Fellow at the Kavli Institute of Cosmology working on data analysis methods for 21-cm Cosmology. His work brings together expertise across instrumentation, theory and inference to help reveal the first stars and galaxies.



Artwork by Grace Heslin

Cooking Nuclear Pasta in the Cosmic Kitchen

Abdullah Al Zaif delivers a deliciously digestible take on the remarkable material science of nuclear matter.

If you're looking for a fine-dining experience, the inner crust of a neutron star is likely the last place you'd check. It is arguably the most hostile environment in the known universe, where the laws of physics are pushed to their limits. Yet tucked away in this cosmic basement is an ingredient that you might easily find in a Cambridge student's cupboard: pasta.

The process of making this pasta begins with a supernova: an explosion that occurs when a very massive star exhausts its nuclear fuel and its core collapses catastrophically under its own gravity. If the remnant mass falls between 1.4 and 2.5 times that of our Sun, the result is a neutron star: a city-sized sphere so dense that a single teaspoon would weigh 100 million tonnes. At this extremity, electrons are forced into protons to create a ball of almost pure neutrons packed into a space thousands of times smaller than the original star. At the surface of a neutron star, ordinary nuclei like iron can still exist, but venture deeper, and the pressure creates something altogether stranger.

The pasta phases emerge within a 100-metre-thick mantle inside the inner crust of a neutron star, with a density of around half that of the atomic nucleus. This is a battleground between two fundamental forces: the strong nuclear force, which tries to bind protons and neutrons into compact spherical nuclei, and the electromagnetic Coulomb force, which makes protons repel each other. At these extreme densities, matter becomes "frustrated" and the system cannot satisfy all its competing interactions simultaneously. The result? Nuclear matter abandons conventional spherical nuclei and reorganizes into bizarre, pasta-like geometries to minimize its total energy.

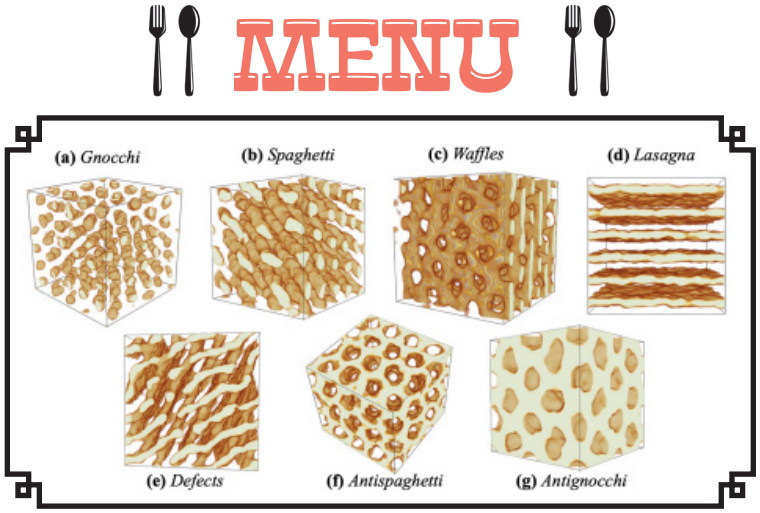
Artwork by Nina Valenbreder

Physicists cannot yet probe neutron star interiors directly, but quantum molecular dynamics simulations modelling tens of thousands of nucleons on supercomputers reveal a remarkable progression of phases as you descend through the inner crust. At the shallowest depths of the pasta mantle, the gnocchi phase appears first. Conventional nuclei (which reside in the core of the atoms that make up everything we see around us) usually consist of no more than a couple of hundred nucleons (protons and neutrons) and account for nearly all the mass of the atom. They are about 10,000 times smaller than the atom itself, which means more than 99% of an atom is just empty space! Inside a neutron star, however, the density and pressure are so high that the nuclei merge into massive, semi-spherical clumps containing many hundreds of nucleons each, resembling small gnocchi dumplings suspended in a sea of free neutrons. Increase the pressure, and these dumplings elongate and fuse into the spaghetti phase: long, thin nuclear rods stretching through the neutron sea, each potentially thousands of nucleons long. Compress further, and the rods themselves merge sideways, forming the lasagna phase: flat, parallel sheets of nuclear matter separated by layers of neutron-rich material, like geological strata of protons and neutrons. Then something remarkable happens: the geometry inverts. Rather than clumps of nuclear matter suspended in a neutron sea, you get a very dense neutron sea with holes of nuclear matter. The bucatini phase (or "anti-spaghetti") features cylindrical holes running through an otherwise uniform nuclear background, pasta tubes formed by the absence of nuclear material. Finally, at the deepest layers of the crust, the Swiss cheese phase emerges: spherical bubbles of neutron-rich matter scattered through a uniform nuclear background, like air pockets

in cheese. Descend any deeper, and even these bubbles dissolve into the uniform liquid neutron matter of the core.

These exotic phases are believed to play an important role in governing the neutron star's behaviour. Nuclear pasta is predicted to be the strongest material in the universe, with a shear modulus billions of times greater than steel. This incredible stiffness allows neutron stars to support "mountains" – small lumpy deformations that are mere millimetres high but of incredibly large mass on the surface of otherwise perfectly spherical neutron stars, supported by gravity and the crystalline structure of the outer crust. As the star spins at hundreds of rotations per second, these deformations can generate gravitational waves rippling across spacetime. When stress builds up in these rigid pasta layers, they eventually snap in catastrophic "starquakes", releasing enormous bursts of X-rays and gamma rays. These have been observed from highly magnetized neutron stars called magnetars, whose outbursts can briefly outshine the entire galaxy in high-energy radiation.

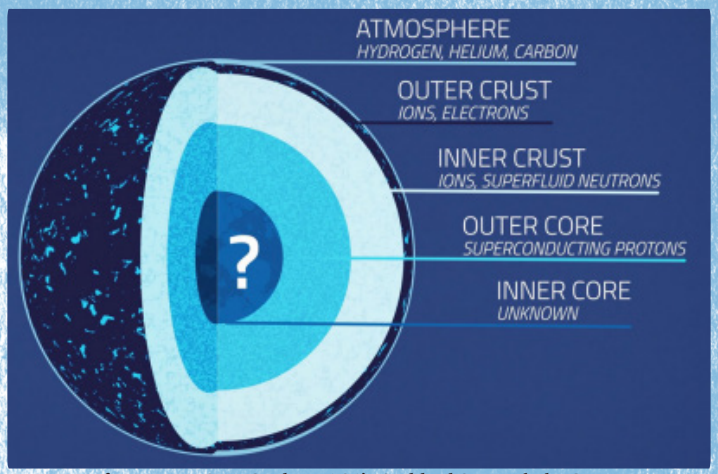
Nuclear pasta represents the ultimate frontier of material science. While it may sound like comfort food, it's a dish that remains fundamentally indigestible to human understanding, at least until the next generation of gravitational wave detectors can finally taste what's cooking in the cosmic kitchen.



Nuclear Pasta configurations. Taken from "Astromaterial Science and Nuclear Pasta" by Caplan and Horowitz (2017)

The article has been inspired by "Nuclear Pasta Formation" by Schneider, Horowitz, Hughto & Berry; Published in Phys.Rev.C 88 (2013) 6, 065807

Abdullah Al Zaif is currently a Teaching Assistant at the Institute of Astronomy, having graduated in summer 2025, with an interest in Gravitational Waves and researching in Strong Gravitational Lensing.



Anatomy of a Neutron Star. Credit: NASA's Goddard Space Flight Center (Conceptual Image Lab)

Watercolor brush stroke courtesy of University of California, Davis

THE FASTEST CAMERA IN THE WORLD

Dhruv Radhakrishnan brings us up to speed on the way scientists "capture" the motion of electrons and how this technology could improve how we fuel our world

Everyone wants a better camera. One that never blurs. We can watch a hummingbird beating its wings in crystal-clear slo-mo at 120 fps on an iPhone (please sponsor me Apple), but can we catch the most minute quantum-scale electron motion with that same clarity?

Most students have heard of the uncertainty principle. The whole idea that it's impossible to be able to tell where exactly an electron is in space. But what if I were to tell you that we now have a camera that we can use to 'see' electrons move with remarkable accuracy? Let's begin by discussing how a regular camera works.

How cameras work:

The principle behind everyday cameras is that images are formed by light reflected off an object being focused by a lens. These rays are focused onto an image sensor composed of a lattice of light-sensitive pixels. The light intensities at each pixel are then converted into digital signals to produce pictures.

However, if the lattice is exposed to light for too long, all pixels will register a high brightness, reducing the contrast and clarity within the picture. This is why the camera needs a device to regulate the exposure time – this is the shutter, which works by opening and closing to allow light in for a certain period. For a bright scene, a small exposure time is needed, as the required intensity contrast will be reached quickly, and vice versa for a dark scene. But, if the scene changes during this exposure time, the image becomes blurred – which becomes an issue when investigating processes on the subatomic scale.

Why an electron camera has to work differently, and what it actually measures:

At the super-small level, if we want to determine the motions of particles like electrons, we cannot simply take photos, as the events happen at attosecond scale, meaning that it's impossible to open and close a shutter in time for the image not to be blurred. For context, an attosecond is 10^-18 (0.000000000000000001) seconds. There are more attoseconds in one second than seconds that have passed since the start of the universe 13.6 billion years ago.

So, a different angle of attack on the problem must be taken.

Image generated by author with ChatGPT and DALL-E.
Outlines made with Vecteezy.com

The process:

An intense laser pulse is directed at an inert gas, which in turn makes the gas emit pulses of higher-order harmonics (higher frequency pulses) of the initial pulse. These emitted pulses are very short: they have a lifetime of about 100 attoseconds. A pulse will hit a sample (typically a gas), and then ionise it, releasing an electron, which initiates the measurement process. Now to take the photograph. After a determined delay, an infrared laser pulse is released, creating an IR field. An electron interacts with the field, and its momentum will change depending on the delay of the IR pulse. An electron's momentum can be detected, and from this, the instant of ionisation can be deduced, allowing us to track the motion of the electron. By this method, we essentially have a camera which records the instants of ionisation of electrons.

Why it's useful:

"After all this faff and millions of pounds of investment, what is the point of having an electron camera?", I hear you say. Well, I'm glad you asked, as there's plenty of reasons why this is, in fact, useful.

Atomic modelling & Quantum Tunnelling

When we observe electrons after being ionized, we can piece together details like how long it takes electrons to absorb the energy or how the specific orbital an electron is in can change its ionization time. By collecting these data for different atoms and complexes, a more complete picture of the atomic model can be built, furthering our understanding of small-scale physics.

Let's say you go outside and kick a football against a wall. You'd expect the ball to just bounce back at you right? I wouldn't know because I've never done it before. But, imagine if the ball just appeared on the other side of the wall, yet there was no visible hole you could see. Your jaw would drop, you'd think there was some black magic going on right in front of you. To a quantum physicist though, it would just be a macro-scale quantum tunnelling event occurring. Quantum tunnelling is when particles behave like waves, allowing them to "tunnel" through firm boundaries without having "enough energy". Previously, tunnelling was shrouded in mystery, with many physicists believing it to be instant, with no clue as to how it occurs. Now, with our attosecond cameras, we can track the exact process, essentially catching the electrons "in the act", demystifying these curious events.

More practical applications: chemical reactions and solar energy

Electrons are the driving force behind chemical reactions. In molecules, because of the overlap of electron orbitals, charge is often not centralised on specific atoms – rather, it is spread out as a distribution across all atoms. So, by using our attosecond electron camera, the charge distributions within a molecule can be studied, allowing us to work out which chemical reactions will happen in given conditions. From here, reaction pathways can be tinkered with to boost yields and cut down on waste in a plethora of fields, whether it be pharmaceuticals, plastics or petrochemistry.

These cameras even have applications in everyday objects like solar panels. Solar panels work through the photoelectric effect (when light hits them, they release electrons, causing a current), which happens at the attosecond scale (it was previously thought to be instantaneous). Now, on observing interactions on this scale, new materials and structures can be experimented on to maximise the efficiency and speed of the electron flow, which will lead to more potent and profitable energy production.

Attosecond physics has the potential (and is currently fulfilling it) to be a very successful new foray into undiscovered territory. As well as its more obvious purpose in attacking fundamental questions like quantum electron motion and better modelling the nature of the atom, further research will also lead to other, more tangible benefits, like improving efficiency on industrial scales such as energy production and the vast chemical sector. It would certainly be wise to watch this space.

Dhruv Radhakrishnan is a 1st year undergraduate in Physical Natural Sciences, especially interested in physics and mathematics.

FOCUS

The Smallest Engine in Biology

Artwork by Jordan Inglis

Aishwarya Venkatramani explores how quantum mechanical phenomena are not merely incidental to biological systems but are actively exploited by evolution to drive fundamental life processes such as photosynthesis, enzyme catalysis, and animal navigation.

Biology operates across a range of scales that defies easy intuition, stretching from a 3,000-mile migration arc down to a 0.3 nm electron orbital. To a European Robin, the travel from Scandinavia to Africa is a feat of macroscopic endurance, guided by perception of the Earth's magnetic field. What seems like a marvel of animal instinct is, at its mechanistic root, a quantum phenomenon occurring in a single protein in the bird's eye. The emerging field of quantum biology proposes that life is not merely a collection of big things made of small things: it is a macroscopic consequence of subatomic phenomena. The most fundamental biological outcomes—the turn of a wing, the synthesis of a sugar, the repair of a genetic code—are dictated by events at the extreme lower end of what physics permits.

This question is not new. In 1943, Erwin Schrödinger stepped into a lecture hall in Dublin to ask: what is life? He, along with Niels Bohr and Max Delbruck, sensed that the classical laws of physics, the predictable, averaging world of billiard-ball atoms, were insufficient to explain the staggering stability of heredity: the fact that biological traits are copied with near-perfect fidelity across thousands of generations, despite the thermal chaos. His answer was the aperiodic crystal, a molecule whose chemical bonds were stable enough to resist the disruptive jostling of thermal noise. It was a prediction that pointed directly toward the structure of DNA, which Watson and Crick would confirm a decade later. Schrödinger's deeper intuition was that life is a physical system that exploits quantum discreteness to store and preserve order with a fidelity no classical mechanism could achieve. Bohr had independently suggested that biology might require physical concepts not yet developed, that the same revolution which had overturned the picture of the atom might eventually overturn the picture of the cell. They were right, but a century too early. The tools required to watch a molecule occupy a quantum state, femtosecond lasers and two-dimensional electronic spectroscopy, did not yet exist. In their absence, the consensus hardened: the cell was too "warm, wet, and noisy" for quantum coherence to survive. That consensus is now being systematically dismantled.

Consider the photosynthetic apparatus which consists of the molecular machinery by which plants, algae, and cyanobacteria convert sunlight into the chemical energy that sustains almost all life on Earth. When a photon strikes a light-harvesting antenna protein, it promotes an electron to a higher energy state, creating a quasiparticle known as an exciton. This excitation must migrate from the periphery of the antenna complex to a reaction center, where it drives charge separation and the synthesis of stable chemical fuel. In a classical framework, this migration would proceed by incoherent Forster hopping, a stochastic process in which energy jumps between pigment molecules in a random, thermally driven search. Given the thermal noise of a living cell, such a process predicts substantial energy dissipation before the exciton reaches its destination. Measured quantum efficiency in living photosystems routinely exceeds 95%.

The resolution to this discrepancy came in 2007 when Fleming and colleagues applied two-dimensional electronic spectroscopy to the Fenna-Matthews-Olson complex, a light-harvesting protein from green sulphur bacteria. The spectra revealed long-lived oscillatory features that are the spectroscopic signature of quantum coherence: the exciton was not hopping between chromophores stochastically but propagating as a coherent superposition across multiple pigment molecules simultaneously. This quantum walk samples all available transfer pathways in parallel, identifying the most efficient route to the reaction centre with a probability that no classical random process can match. Subsequent experiments confirmed that coherence persists for several hundred femtoseconds at room temperature, long enough to influence energy routing under physiological conditions. The sub-femtosecond behaviour of a single absorbed photon therefore determines, through this mechanism, the energy flux of the entire photosynthetic apparatus, and ultimately the oxygenation of the atmosphere.

Every chemical reaction in a cell faces the same fundamental problem: to transform one molecule into another, you first have to break existing chemical bonds, and breaking bonds costs energy. Picture a ball sitting in a valley, with a hill between it and the next valley. The ball must be pushed over that hill before it can roll down the other side. In chemistry, this hill is called the activation energy barrier, and classical physics is clear about how molecules get over it: they absorb heat from their environment until they have enough energy to climb. Temperature matters because warmer molecules move faster and collide harder, crossing the barrier more frequently. This relationship between temperature and reaction rate, described by the Arrhenius equation, is one of the most reliable predictions in chemistry.

For enzymes that transfer hydrogen atoms or protons, something goes wrong with this picture. The rates are too fast, the apparent barriers too low, and in some cases the reaction speed barely changes with temperature at all, as though the molecule is not climbing the hill at all. It is not. It is passing through it.

This is quantum tunnelling. Because matter at small scales behaves as a wave rather than a particle, a proton does not have a precise location the way a tennis ball does. Its wave function, the quantum mechanical description of its state, extends through and beyond the barrier. There is therefore a finite probability of the proton appearing on the far side without ever having had enough energy to cross classically. For protons, which are light enough for this effect to be significant, tunnelling can contribute substantially to reaction rates in the confined geometry of an enzyme active site.

The clearest way to test this is to swap the proton for a heavier version. Deuterium is the isotope of hydrogen with an extra neutron, making it twice as heavy. Heavier particles tunnel less readily, so if tunnelling is occurring, replacing hydrogen with deuterium should slow the reaction by a factor that classical chemistry cannot explain but quantum mechanical models predict precisely. This measurement is called a kinetic isotope effect, and when Judith Klinman's group at Berkeley and Nigel Scrutton's group at Manchester applied it to enzymes including alcohol dehydrogenase and aromatic amine dehydrogenase, the slowdown they observed was an order of magnitude larger than any classical model could account for. Tunnelling was not a minor correction. It was the dominant mechanism.

What emerged from structural studies was more striking still. The active sites of these enzymes are shaped specifically to promote tunnelling. The atoms between which the proton transfers are held at distances fine-tuned to sub-angstrom precision, and the protein flexes at the critical moment to compress the barrier

further. This is not passive quantum physics happening to occur in a biological context. It is a protein architecture that natural selection has refined, over billions of years, to maximise a quantum mechanical probability.

Some enzymes go further still, incorporating metal ions directly into their catalytic machinery. Purple acid phosphatase, found across plants, animals, and bacteria, contains two metal ions at its core, one iron and one zinc, held in precise proximity by the surrounding protein. These metals do more than simply position the substrate. Their electrons occupy quantum mechanical orbitals that overlap directly with the electrons of the reacting molecule, creating an electronic pathway through which the chemistry flows. The reaction rate, and the metabolic function that depends on it, is a direct consequence of this quantum electronic architecture at the scale of individual atoms. The metal centre is not a passive scaffold. It is a quantum mechanical device, and the protein around it has been built to make it work.

The same quantum architecture underlies something far more immediate: the fact that you are reading these words at all. When a photon enters your eye and strikes the rhodopsin protein in a retinal cell, it triggers a geometric rearrangement of the retinal chromophore, a light absorbing molecule that can switch from one shape to another in approximately 200 femtoseconds. This is the fastest known photochemical reaction in any living system. It works because the protein environment quantum mechanically funnels the excited electronic state toward a single specific outcome, rather than allowing it to dissipate as heat across multiple competing pathways. Colour vision is, in this sense, a set of three quantum detectors. Each of the three cone photoreceptors contains the same retinal chromophore, but surrounded by a slightly different protein environment. Precisely positioned amino acid residues shift the quantum energy levels of the chromophore, tuning its absorption to a different wavelength. The colour you perceive is a quantum energy gap, shaped by a protein. Every colour you have ever seen is a consequence of quantum electronic structure engineered by evolution inside a molecular cavity a few nanometres across.

This principle of protein-tuned quantum optical control extends well beyond the eye. Fluorescent proteins, derived from jellyfish and coral and now among the most widely used tools in cell biology, work by exactly the same mechanism as rhodopsin: a chromophore buried inside a protein barrel whose quantum electronic state is precisely controlled by the surrounding amino acid environment. The story begins in the 1960s, when Osamu Shimomura painstakingly isolated a glowing protein from the jellyfish *Aequorea victoria*, catching and processing hundreds of thousands of jellyfish off the coast of North America to do so. That protein, GFP, fluoresces bright green because its chromophore is shielded and quantum mechanically tuned by the surrounding protein barrel. Martin Chalfie then showed that the gene encoding GFP could be inserted into living organisms, causing specific cells to light up under ultraviolet illumination and making the invisible visible for the first time. Roger Tsien went further still, engineering single amino acid changes near the chromophore that shifted its quantum energy levels and produced an entirely new palette of colours: cyan, yellow, red, each a different quantum energy gap, each generated by minimal structural changes to the same protein scaffold. The variants that now underpin modern fluorescence microscopy are, in physical terms, colour-tuneable quantum optical devices operating at room temperature in water. Shimomura, Chalfie, and Tsien were awarded the Nobel Prize in Chemistry in 2008 for this work. The quantum architecture was already there, built by evolution for bioluminescence. They learned to read it, and then to rewrite it.

These considerations return us to the robin. The radical pair mechanism, proposed by Schulten in 1978 and substantiated experimentally over the following two decades, identifies cryptochrome, a flavoprotein expressed in retinal cells of migratory birds, as the magnetic sensor. When a photon is absorbed by cryptochrome, it drives an electron transfer that generates a radical pair: two molecular radicals each containing a single unpaired electron, created in a spin-correlated singlet state. From this initial entangled state, the radical pair evolves under two competing interactions: hyperfine coupling between electron and nuclear spins within the molecule, and the Zeeman interaction between the electron spins and the external magnetic field. The balance between these interactions governs the rate of singlet-to-triplet interconversion. The two distinct quantum spin states of the electron pair differ in their total angular momentum and in the chemical reactions they are permitted to undergo. Because the Earth's field, approximately 50 microtesla, is commensurate in energy with the hyperfine coupling constants, it shifts this product ratio in an orientation-dependent manner. Classical chemistry is entirely insensitive to a field of this strength — its Zeeman energy is orders of magnitude below the thermal energy available in a single molecular collision. The radical pair mechanism is not, because it operates on spin state dynamics rather than energy thresholds.

The most direct experimental evidence came from Peter Hore's group at Oxford. European Robins exposed to a weak radiofrequency magnetic field oscillating at frequencies resonant with the electron spin transitions of the cryptochrome radical pair lost their magnetic orientation entirely. The disorientation was frequency-specific and amplitude-specific, ruling out any classical explanation and matching precisely what the radical pair mechanism predicts. What this requires is that the spin-correlated state of the radical pair survives for microseconds inside a living retinal cell—long enough for the Earth's field to influence the interconversion rate before the quantum state collapses. How the protein environment achieves this, rather than destroying it, is one of the deepest open questions in quantum biology.

The three mechanisms described—quantum coherence in photosynthetic energy transfer, proton tunnelling in enzymatic catalysis, and radical pair spin dynamics in magnetoreception—share a common structure. In each case, a quantum mechanical property is not merely present in the biological system but is the operative mechanism by which a physically constrained problem is solved. The inference is that these properties have been maintained, and in some cases actively optimised, by natural selection over billions of years. If that is correct, then biological macromolecules are not simply the substrates on which quantum physics operates. They are structures whose geometry and dynamics have been tuned, by evolution, to exploit it. Peter Maurer's group at the University of Chicago is now investigating whether cells function as quantum information processors in a technically precise sense — not as a metaphor, but as a physical mechanism. The question Schrödinger asked in 1943 is beginning, at last, to have an answer.

It involves quantum mechanics not as background physics but as an active mechanism. The cell is a quantum machine. It has been one since long before there was oxygen in the atmosphere.

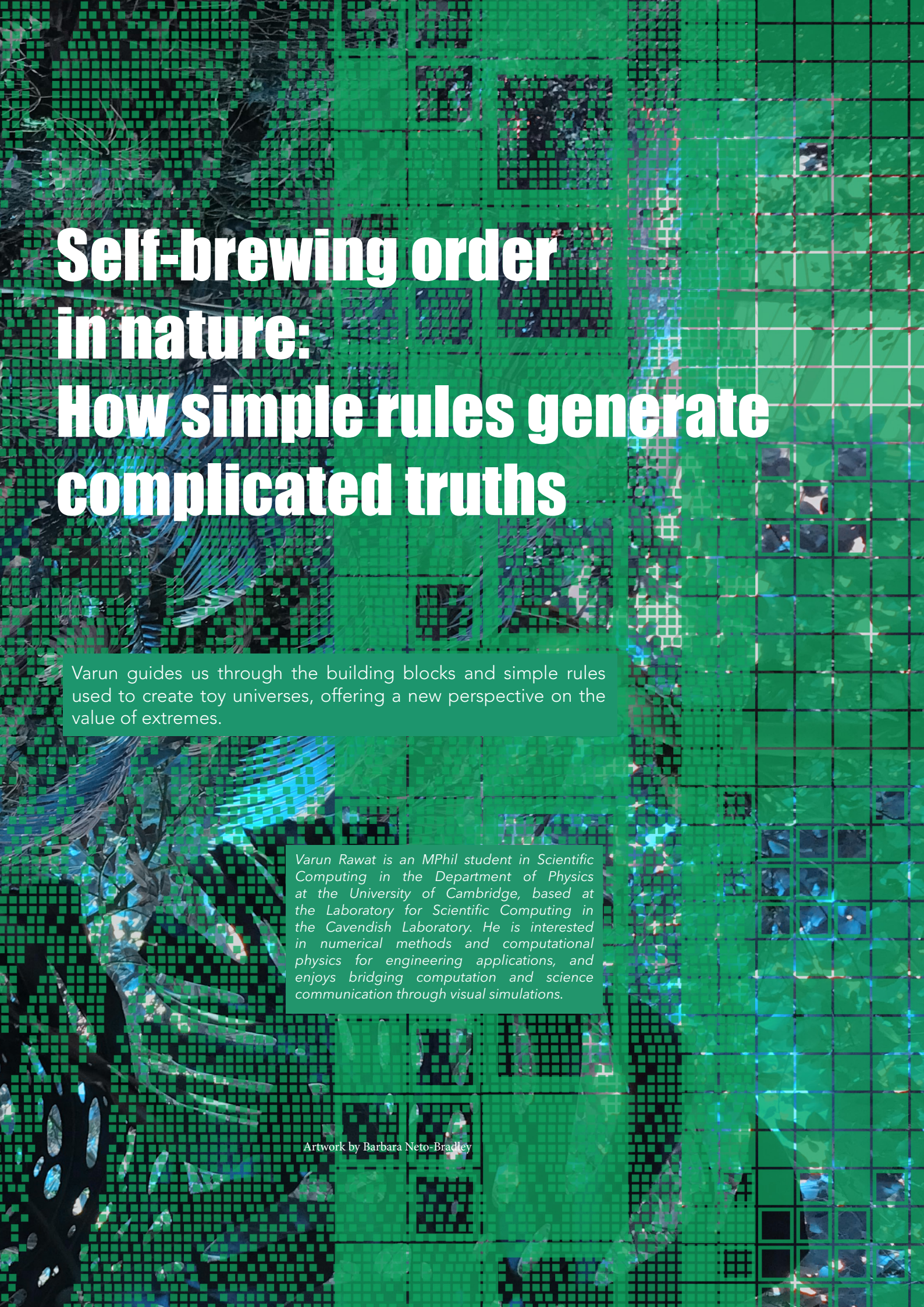
A robin does not know this. Every autumn it turns its head, reads a field that human instruments can barely detect, and flies. Three thousand miles, guided by the spin of two electrons in a protein in its eye—electrons whose quantum states persist in the "warm, wet, chaos" of a living cell because evolution, over billions of years, found a way to make them last. The smallest things in physics are doing the largest work in biology. They always were. We are only now learning to see it.

Aishwarya Venkatramani is a postdoc in Biotechnology, interested in colour, proteins, and molecular properties that lead to colour in life.

Self-brewing order in nature: How simple rules generate complicated truths

Varun guides us through the building blocks and simple rules used to create toy universes, offering a new perspective on the value of extremes.

Varun Rawat is an MPhil student in Scientific Computing in the Department of Physics at the University of Cambridge, based at the Laboratory for Scientific Computing in the Cavendish Laboratory. He is interested in numerical methods and computational physics for engineering applications, and enjoys bridging computation and science communication through visual simulations.



Artwork by Barbara Neto-Bradley

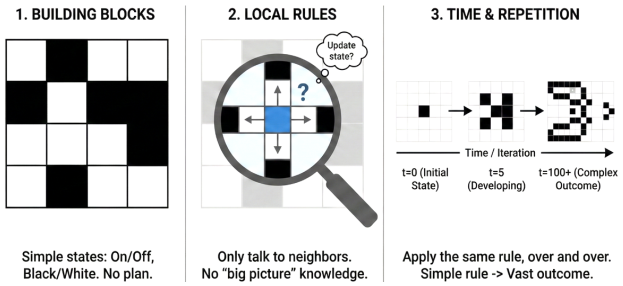
A small mystery about big structures

The first time I saw diffusion, which normally blurs things out, turn faint noise into spots and stripes, I was confused. Diffusion is supposed to smear and spread things out, smoothing the world into blandness.

And yet on my screen, a field that began as faint noise slowly tightened into spots and stripes, as if the simulation had quietly decided on a style. It did not look like a blurred image but like nature sketching.

That moment is why I keep coming back to these toy universes. They are tiny, built from almost childish ingredients, and still they produce structure that feels too rich for the recipe that made it. For this article, I want to focus on a different kind of extreme. Not an extreme of temperature, but an extreme mismatch between a tiny rule and the rich world it can generate. By toy universes I mean small computer simulations where I specify a few building blocks, a local update rule with world-specific parameters, and then let the system evolve step by step.

The question I am chasing is simple and slightly annoying. How can simple rules generate complicated truth?



The whole recipe fits in one breath

The recipe to build these worlds has three ingredients. You pick building blocks. They can be bits on a line, cells on a grid, or concentrations on a lattice. You pick a local rule. Each block looks at a small neighbourhood and updates its state. Then you do the only thing that really matters. You repeat.

Time is the amplifier. Repetition is the engine. A single update is a shrug. Ten thousand updates is a world.

If you have never played with these systems, the most natural mistake is to expect the rule to resemble the outcome. I used to think that if something looks complex, its rule must be complex too. But these systems keep teaching the opposite lesson.

A rule is not a description. It is a generator. A generator does not tell you what the world will look like. It tells you how the world will be made, one step at a time, as it unfolds. That shift matters because it explains why prediction is often stubborn. Even when the rule is tiny, the space of possible outcomes is enormous. And for many rules, there is no clever shortcut that replaces running the process. If you want to know what the system makes, you let it run and watch.

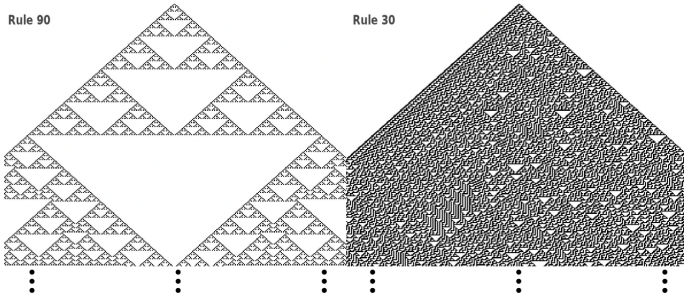
Most generators do not give you what you secretly hope for. Many give you nothing. Many give you noise. A few give you something that feels alive.

Extremes are landmarks

When I run a new rule, I want a miracle. Sometimes I get it. Often, I do not. The system freezes into dull repetition, or it dissolves into restless fuzz. At first, that felt like failure. Then it started to feel useful.

Much like a real landscape, deserts and oceans are not the point, but they are the landmarks. In this zoo of self-brewing order, deserts are runs that quickly settle into trivial order, and oceans are runs that stay noisy and restless without stable motifs. In these toy universes, trivial order and featureless noise are the poles that make everything else legible. The interesting places are often the coastlines in between. With that in mind, I want to take you through three small worlds.

World one: A line of bits that grows geometry



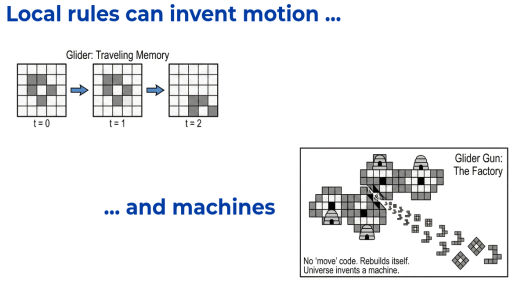
The first world is a one-dimensional cellular automaton. Each cell is "on" or "off". To compute the next row, a cell looks at itself and its nearest neighbours and decides its next state. That is basically all the information in the universe.

Start from a single "on" cell and one rule grows a crisp fractal triangle, repeating its logic at smaller scales. This is the kind of pattern people often show for one particular rule choice, often labelled Rule 90 in Wolfram's numbering. Change the rule slightly, for example to Rule 30, and the same setup produces a very different world that looks chaotic and woven.

This is where I learned to separate two kinds of surprise. There is local surprise. Zoom in, and the next step can feel unpredictable. Then there is global coherence. Zoom out, and the world still has a stable personality. You can recognise it at a glance.

A rule can be locally wild and globally coherent. That is why randomness is not the same thing as the kind of complexity we find interesting. Noise can be endlessly surprising, but it does not tend to build anything interesting you can point at and name. The patterns that hold our attention are constrained enough to be recognisable and loose enough to keep unfolding.

World two: A grid that invents motion and machines



The second world is Conway's Game of Life. Each cell is alive or dead. A cell counts its living neighbours and decides whether

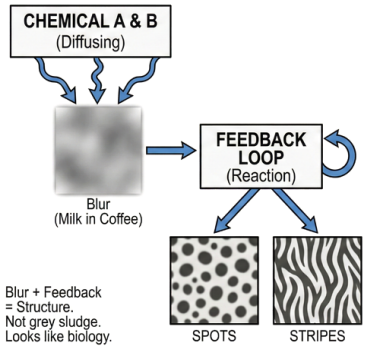
it lives, dies, or is born. The rule is tiny, but the results can feel like magic.

You can see a glider, a small shape that travels diagonally across the grid. Nothing pushes it. It moves because the update keeps rebuilding it one cell over, step after step. Then you see a pattern that behaves like a factory. A glider gun keeps producing gliders again and again.

At this point, it is hard not to use the language of purpose. The pattern looks like it is doing something. But Life is not aiming for anything. There is no target. So why do we keep finding motifs that feel functional?

Local rules plus repetition can act like a search through possibility space, even when nothing is searching on purpose. The world explores many configurations because it must keep updating. Fragile structures vanish, and compatible structures persist. They travel and keep coming back.

World three: Blur that becomes a paintbrush



The third world is that of Gray-Scott's reaction-diffusion system, and it is where my initial confusion began.

Here, the building blocks are not bits. They are concentrations, fields on a grid. The rule is still local, but now it combines two forces. The first is diffusion, which spreads things out and thus should erase structure. The second is reaction, which can amplify or suppress interactions locally and thus can create structure.

When the two compete, something strange can happen. The system chooses a spacing. It settles on a preferred length scale where noise turns into spots and spots can turn into stripes. Locally, the reaction is nonlinear and sensitive. Globally, diffusion plus feedback can settle into a stable texture, like a fabric with a consistent weave.

It also illustrates the choreography idea in a very physical way. The pattern is constrained and has a characteristic length scale. But it is not rigid. It can evolve, merge, split, and drift. It sits between order and randomness in a way that feels endlessly watchable.

And once again, if you run many parameter choices, you see the landmarks. Some settings wash out into uniformity. Some become messy and noisy. Some lock into a clean structure. The interesting behaviour often lives near the boundary between regimes, where a slight change flips the world into a new family.

Why I needed a map

If you only ever show one beautiful simulation, it is easy to treat it like a miracle. But once you start collecting them, you run into a different problem. You run out of adjectives.

Fractal, chaotic, lifelike, textured—they start to blur. So I built a small atlas of thirty-six worlds. I turned each simulation into a simple fingerprint image, then measured a handful of traits with names you can almost guess, like compressibility, spectral spread, dominant length scale, coherence length, anisotropy, texture, and how much the pattern changes over time. Then I used Principal Component Analysis to squash each world down to two coordinates so that similar fingerprints land near each other.

The result is a map you can point at showing landmarks, dead zones, noisy regions, and the borderlands where patterns keep their shape while still doing something interesting. It also sharpens arguments. If a pattern looks complex, we can ask in what way. Is it locally unpredictable, globally coherent, active in time, or structured across scales?

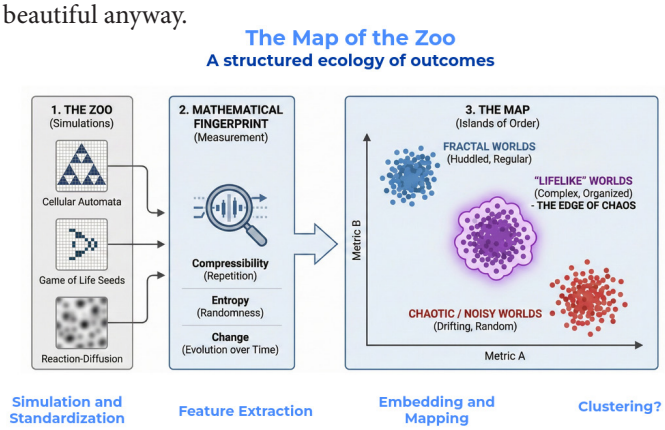
The atlas also changes how we talk about these worlds. Instead of arguing about whether something is emergent, we can point to a neighbourhood and say this is the kind of world it is under this lens. The map does not answer every question, but it makes the conversation and comparisons clearer because claims about worlds become locations on an evolving map.

A small shift in how to see

I do not think these toy universes prove a law that complexity must always rise. They do something quieter by dismantling the assumption that complicated outcomes necessitate complicated ingredients. They make it hard to equate complexity with randomness. And they suggest a practical attitude to prediction. In some systems, understanding is less like reading the rule and more like running the simulation.

For an issue themed Extremes, that is the perspective I want to leave you with. Extremes are not just outliers. They are the landmarks that teach you the shape of a world. And in the borderlands between frozen order and featureless noise, simple rules can brew structure that feels almost unreasonable.

The universe may not be obliged to be beautiful. But sometimes, when you give a small rule enough time, it becomes beautiful anyway.



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EXTREMES

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Artwork by Ashlyn Chew

Cambridge University Science Magazine

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The community sites hosted by The Company of Biologists – the Node, preLights and FocalPlane - together offer an easy way for early-career researchers to get involved in the discussions that highlight, question and help shape scientific progress. Contributing to the community sites helps researchers build practical communication skills - science writing, presenting, and interviewing - while publishing short pieces, reviews, interviews and image features. Outputs that are shared with peers, editors and potential collaborators expanding the professional networks of our contributors.

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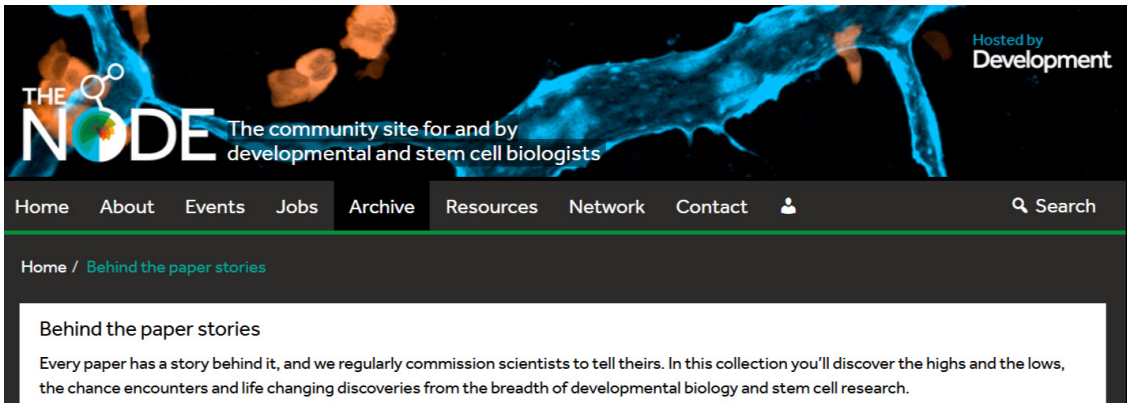

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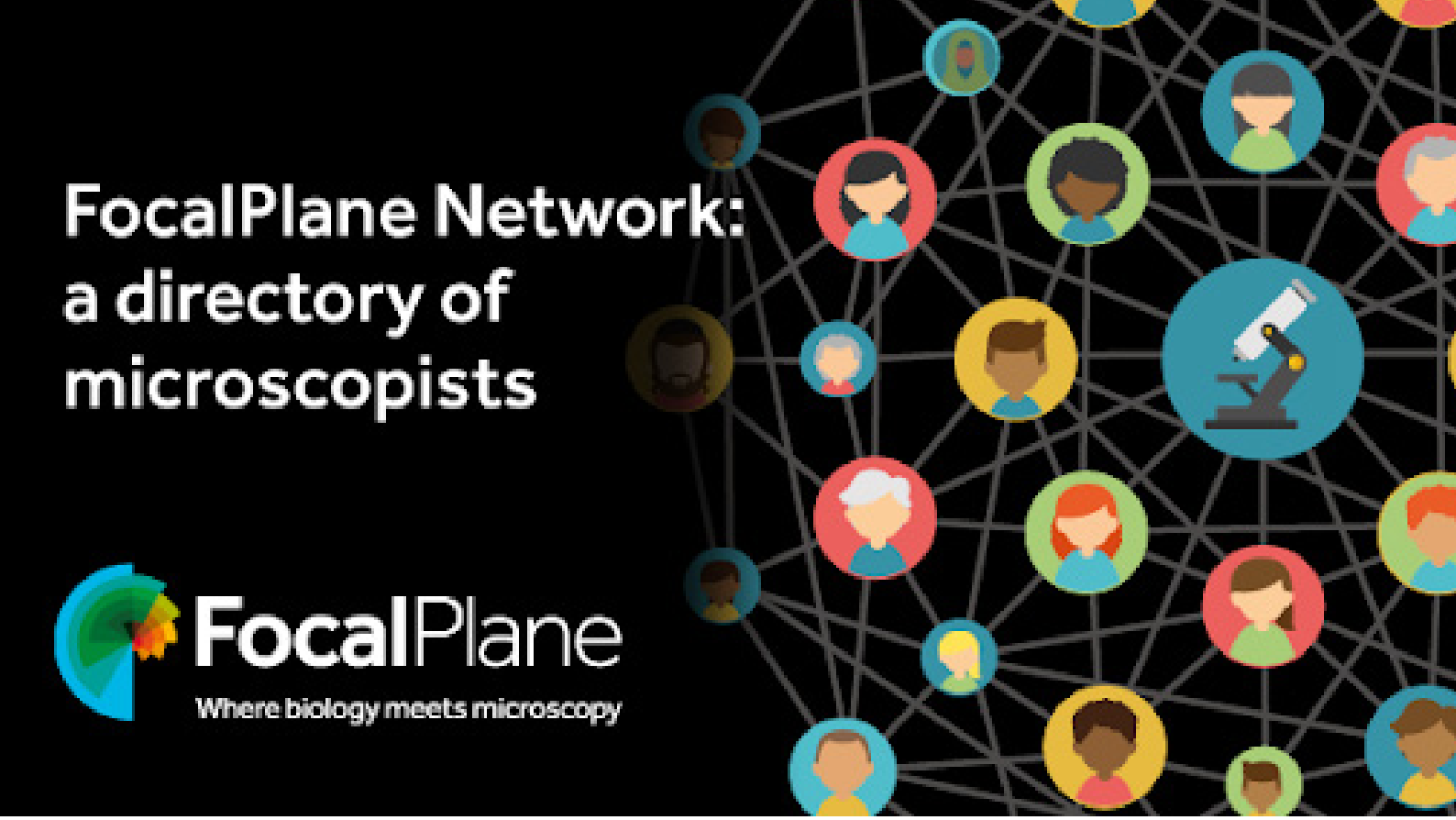

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