

# *KINESIS*

Issue 16



 **UCL 200**



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# LETTER FROM THE EDITOR

We extend the warmest of introductions to our latest special issue, celebrating UCL's bicentenary! A scientific tribute and retrospect on the former students, lecturers, discoveries and controversies through the storied history of our (sometimes) beloved institute. Our ever-devoted team of authors, editors, and artists have worked tirelessly over the past few weeks to deliver what we hope is a thought-provoking collection of art, culture, and articles.

When we decided how to go about producing this issue, we realised we needed to support our pitch writers a bit more. We delved through the recent and near-ancient history of scientific discovery with colleagues across campus, as well as our own alumni and professors, to decide what would help form our prompt list. A decision was made to seek out both the accomplishments and the history that our university aims to distance itself from, in order to present what we believe to be the most accurate tribute to UCL among all the special editions made on campus this year.

Our issue aims to embrace you, the reader, with a plethora of different topics. Whether you're drawn to the latest discoveries, such as the groundbreaking research into Huntington's disease, or you're interested in learning more about the remarkable women who continue to follow in the footsteps of pioneers, we have something to offer. From the controversies of UCL's past to the history of brains and the need for a different kind of classroom, we hope our latest issue provides something for you.

Our work for this issue serves as a time capsule for our current members. The words and design choices are carefully crafted to serve as reminders that we have not forgotten the lessons of yesterday and the need for a brighter tomorrow remains ever present. Previously we have been asked, "Why write?" for student-led magazines, to which we hope this issue serves as an answer, designed to challenge a notion that student voices hold little weight. You, our members, represent why we not only write but also why we do all of this, ensuring the next century is full of scientific curiosity and discovery.

For the final team I will ever share the honour of serving alongside, it has been an amazing journey. Whether you are reading this as a member of that first-ever committee or the one that is about to be elected, this is just the start of the tributes to scientists we intend to give as we approach our 10th birthday.

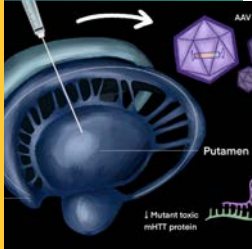
Forever grateful,

Managing Editor



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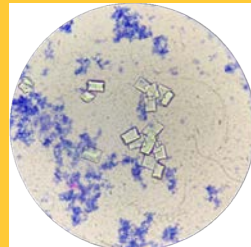
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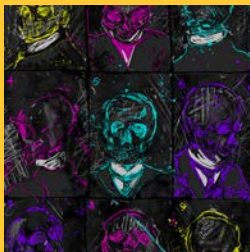
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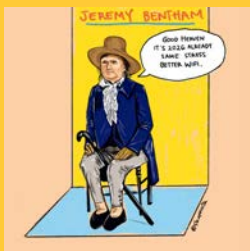


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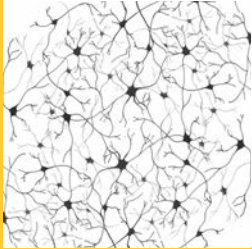
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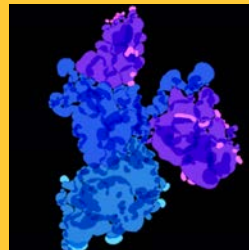


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# A GENE THERAPY BREAKTHROUGH FOR HUNTINGTON'S DISEASE

*How UCL Researchers Are  
Transforming the Future of  
Treatment*

**Naomi Choi**  
Artist: Yasmin Yong

Most people have heard of Huntington's disease, but some might not realise how severely it could affect the brain. In fact, Huntington's disease is one of the most difficult-to-treat inherited brain disorders and is characterised by its progressive symptoms, gradually impacting everything from memory to behaviour. Current treatments for Huntington's focus on managing and minimising symptoms, rather than targeting its root cause. For decades, patients and families have hoped for a therapy that could do more than simply manage the condition, and a research team at UCL has taken the first major step towards changing the future of Huntington's treatment. To understand why this research breakthrough matters, let's take a step back to look into the disease itself and what makes it so challenging to treat in the first place.

## Understanding Huntington's Disease

Huntington's disease (HD) is a fatal, inherited neurodegenerative disorder caused by a single mutation in the huntingtin (HTT) gene, leading to the progressive loss of neurones and symptoms including cognitive decline, motor impairment and behavioural changes. The HTT gene in Huntington's disease is mutated to contain an expanded CAG trinucleotide repeat, leading to the production of faulty huntingtin mutants. While the normal huntingtin protein is harmless and in fact plays important roles in cell survival, gene regulation and early

development of the brain, its mutant version is toxic since its misfolding and accumulation result in the formation of insoluble protein aggregates known as inclusion bodies within neurones. Over time, these harmful clumps of huntingtin mutants disrupt essential cellular processes, such as transcription, mitochondrial function, autophagy and synaptic signalling, ultimately triggering neuronal dysfunction and cell death. It is important to know that neurones do not replicate; thus, once a neuronal cell dies, it will not be replaced.

The region in the brain that is most affected is the basal ganglia, which plays key roles in controlling movement, cognition and behaviour. As neurones in the basal ganglia degenerate progressively, affected individuals begin to develop the characteristic symptoms of HD, including involuntary movements, impaired memory and coordination, as well as mood disturbances and personality changes. These symptoms could eventually lead to physical and psychological complications such as respiratory infections, heart failure, impaired swallowing (dysphagia) and movement (chorea), fatigue, depression, anxiety, agitation and more, with the most common cause of death being aspiration pneumonia that is often caused by dysphagia.

Huntington's disease follows an autosomal dominant inheritance pattern, meaning that people with an affected parent have a 50% probability of inheriting the disease, with

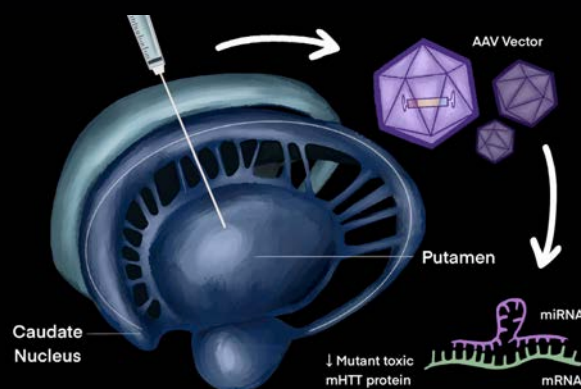
first symptoms typically arising between the ages of 30-50, and worsening over 10-25 years after symptoms first develop. Eventually, affected individuals are confined to bed and require total care, and the disease is normally fatal within two decades. Around 75,000 people across the UK, US and Europe are currently living with HD, with many more at risk of developing it.

## UCL-Led Gene Therapy for Huntington's Disease

Early diagnosis and treatment is key to treating HD, yet no treatment has been able to slow, prevent, or halt disease progression – not until now. A pioneering clinical trial led by researchers from the UCL Huntington's Disease Research Centre has revealed that a new gene therapy has shown promising results in slowing disease progression in HD patients.

Prof. Sarah Tabrizi, director of the research centre and lead scientific advisor on the clinical trial, and Prof. Edward Wild, principal investigator of the UCL Huntington's Disease Centre trial site, led the development of the gene therapy named AMT-130. The gene therapy is delivered through a one-off neurosurgery by injecting an adeno-associated viral (AAV) vector into the brain using real-time MRI scanning to guide a micro-catheter to the caudate nucleus and the putamen, which are the two regions of the basal ganglia most vulnerable to HD. The viral vector contains a DNA sequence that codes for a small fragment of microRNA (miRNA), which performs gene silencing by binding to the section of messenger RNA (mRNA) encoding the huntingtin mutant (mHTT) protein in neurones. This marks it for degradation, thus switching off its production and reducing mutant protein level.

AMT-130's clinical trial was developed by uniQure, a gene therapy company based in the Netherlands, and involved 29 patients who had tested positive for the HD gene and were diagnosed with early-stage HD. These



patient volunteers have completed up to 36 months of Phase I/II clinical trials, with 12 receiving high-dose therapy. Results showed a 75% slowing of clinical progression compared to a matched external cohort of HD patients, as measured by the composite Unified Huntington's Disease Rating Scale, as well as a decreased level of neurofilament light protein (NfL), a biomarker protein released into the spinal fluid when neurones are damaged. The treatment was also considered generally safe and well tolerated, and a single administration should last for life as neurones do not regenerate and get replaced.

## The Future of Huntington's Treatment

This breakthrough with the AMT-130 gene therapy undoubtedly marks a significant and exciting shift in how researchers approach Huntington's disease, offering the first real possibility of slowing down the condition at its genetic roots while opening the doors to treating other neurodegenerative conditions with gene therapy – a milestone that began right here at UCL. Although full clinical trial results are yet to be published and larger-scale studies might be required to confirm long-term safety and effectiveness, uniQure is already working toward securing FDA approval, reflecting strong confidence in AMT-130's therapeutic potential. As research progresses, the ultimate hope is that Huntington's disease, once considered untreatable and inevitably fatal, could one day be met with therapies that fundamentally change its course and profoundly improve the lives of hundreds of thousands of people affected worldwide.

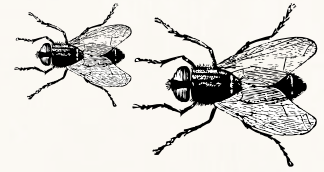
# 195 years on the *Beagle*, 199 years at Grant's

## Relooking at the History of the UCL Grant Museum of Zoology and Comparative Anatomy and Its Founder, Robert Edmond Grant

1826 was an important year for UCL, but for many others related to the newly founded university, 1826 was also life-changing. Such was the case for two figures, Dr. Robert Edmond Grant, and Mr. Charles Darwin.

That year, a young Darwin spent his time as a university student at the University of Edinburgh; just like many second-year undergraduates at UCL now, Darwin joined societies and even found an internship opportunity – collecting and documenting animal samples with a talented fellow, zoologist and marine biologist, Robert Edmond Grant.

A researcher with acute perception and the most developed methodologies, Dr Grant had brought back to Britain the most pioneering theories of evolution from Étienne Geoffroy Saint-Hilaire, disciple of French evolutionist Jean-Baptiste Lamarck. It was an unforgettable experience for Darwin to venture all the way with Dr. Grant across the Scottish coastlines, searching for small enclaves of life in shallow tidal pools.



While learning some lifelong effective research skills in the process, Darwin was also able to make his own discoveries in those fieldworks, an impressive feat for a young and inexperienced student like himself. Under Grant's directions, Darwin was brought to the cutting edges of biology,

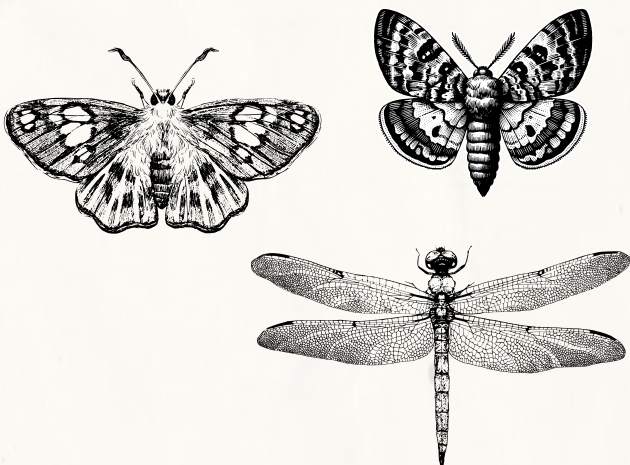
learning how to systematically document his observations of the natural world. Grant made

breakthroughs on the trip as well, surveying organisms like sponges, sea mats, and many other invertebrates.

Much to the surprise of his peers, those marine lifeforms were animals rather than plants.



In late 1827, the two talented scholars parted ways as Darwin found studying medicine in Edinburgh unfitting for him. Meanwhile, Grant accepted a job offer from a newly founded higher education facility, University (College) of London, leaving the tidal pools for a settled, urban life. Darwin stepped on board the HMS *Beagle* three years later for its second voyage (the first voyage of the *Beagle* was in 1826, exactly the year UCL was founded!). The rest of Darwin's legendary journey is now a well-known tale documented in *The Origin of Species*, a story not of Mister, but Sir Charles Darwin, the founding father of modern biology.



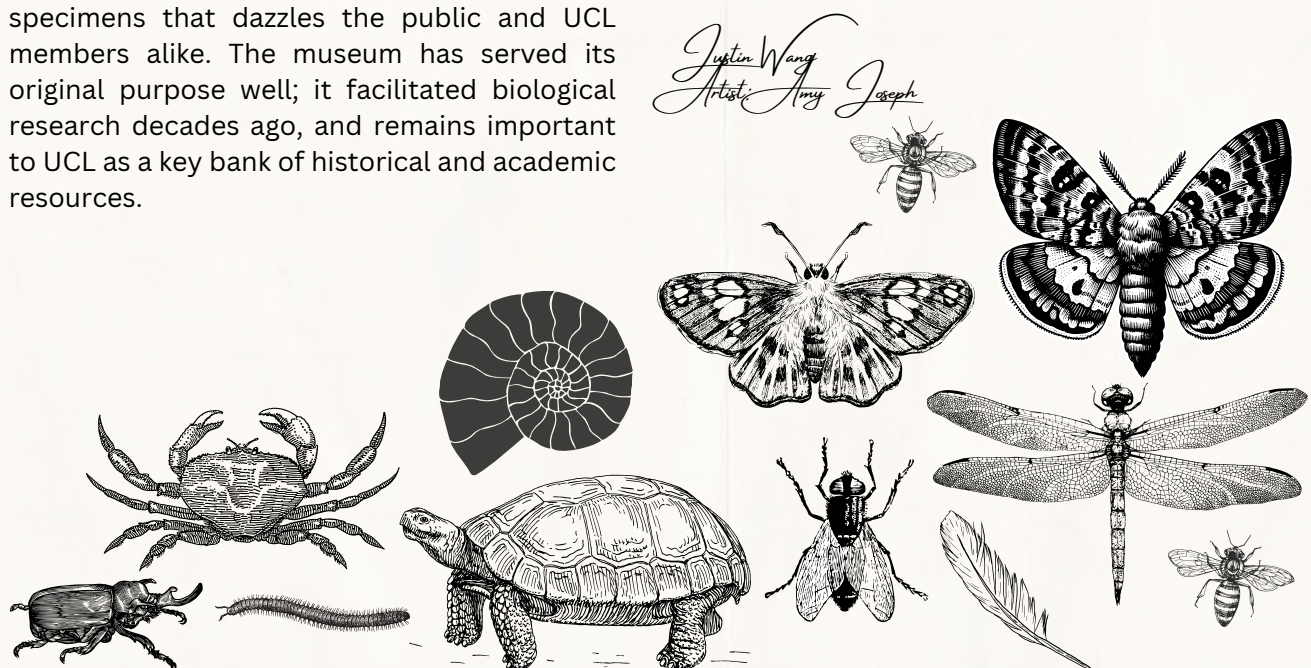
Despite the sweeping changes in scientific paradigms, the life sciences explored similar themes over the years of development. In Grant's time, comparative anatomy asked a series of questions framed very differently from traditional explorations of the mechanical details of tissues and organs. Traditionally, people ask vague and reductionist questions, such as "Why do elephants have long noses?", but biologists ask a holistic and process-oriented question, like "What kind of complex mechanism contributed to the long noses of the elephants?" Comparative anatomy tackles similar problems of evolution with insights into animal morphology. By the 19th century, a majority of natural philosophers were cross-examining different species from a more holistic perspective. Instead of treating lifeforms as isolated cases, comparative, or "transcendental", anatomy searched for trends among all living things. Such a new academic tradition is now cherished as the key spirit of the biological and life sciences.

Nevertheless, conducting studies of comparative anatomy required large quantities of well-documented specimen samples. In 1827, Grant started an initiative to tackle this problem. The newly arrived professor aimed to build a natural history museum similar to the one he likely visited in Edinburgh. This museum would later rapidly absorb animal samples and models over its nearly two-century history. Now known to us as the Grant Museum of Zoology and Comparative Anatomy, Grant's vision coalesces into a systematic showcase of specimens that dazzles the public and UCL members alike. The museum has served its original purpose well; it facilitated biological research decades ago, and remains important to UCL as a key bank of historical and academic resources.

Cheers to Dr Grant! Unfortunately, a brilliant idea lingering to this day did not bring Grant any fame and prestige while he was in London. The competitive nature of academia severely hindered Robert Edmond Grant's career in London. He was outcompeted by his political and academic rival, while a shortage of students attending his outdated lectures worsened his financial conditions. Due to Grant's habit of burning personal works, we have only a vague portrait of his late personal life. In Charles Darwin's autobiography, he described a younger Grant in Scotland as a passionate and talented scholar, well ahead of his time, but that passion ended up in a stubborn character, refusing to move on when his past trainee, Darwin, sparked a revolution in the life sciences.

Over the years, the Grant Museum has changed locations multiple times, but it has always been used for studies of animal morphology, taxonomy, and embryology. The value of its specimens persisted as biological research entered a new era: Watson and Crick's discovery of DNA and the creation of molecular biology and phylogenetics. Nowadays, the Grant Museum has become not only a treasury of animal species, but also a genome bank, and a place of historical memories, with some parts of its collection dating back to Grant himself.

The modern study of all living beings has set sail with the Beagle since 1831, with the Grant Museum since 1827, and with UCL, for a total of 200 years dedicated to scientific endeavours.



# Redefining DEATH:

## **Palliative care and the assisted dying debate**

Author: Samuel Durston

All of us know someone who has died. A relative, perhaps a grandparent or uncle, a neighbour, a former teacher. In response to death, survivors grieve, attend funerals and seek solace with family or friends. Grieving is healthy. Yet, we are also expected to move on – to grieve quietly, and then to look away. Our stance towards death reflects a deeper human unease with our own mortality. Imagine, then, being diagnosed with a terminal illness, knowing not only that you will die, but that suffering may precede it.

Palliative care is a branch of medicine focused on making this process as honourable and painless as possible. It is one of the few forms of medicine that prioritises relief over cure. It is also one of the most underfunded medical fields. In 2022, palliative care research received £10.9 million, representing just 0.26% of the total £4.2 billion in UK health research funding, according to BMJ Supportive & Palliative Care. It is estimated that up to 90% of people dying in England would benefit from palliative care, yet less than half receive it.

Dr Libby Sallnow, Associate Professor of Palliative and End-of-Life Care at UCL and Director of the Marie Curie Palliative Care Research Institute, conducts research into the various challenges and inequalities associated with providing palliative care. For instance, she highlights that access is particularly limited among disadvantaged groups, including older individuals, individuals with non-malignant conditions such as heart failure, women and those living in rural areas. As the population ages, demand for palliative care continues to rise. This article draws on her insights to explore these issues further.

In 2024, new legislation was introduced into Parliament that could fundamentally reshape end-of-life care. The bill, titled the ‘Terminally Ill Adults (End of Life) Bill’, allows terminally ill

adults to self-administer life-ending medication, provided strict safeguards are met. These safeguards include a prognosis of six months or less, full mental capacity, assessments by two independent doctors that the patient meets the eligibility criteria and oversight by a specially constituted panel. Notably, the bill does not legalise euthanasia, but limits assisted dying to patient self-administration. Introduced by Labour MP Kim Leadbeater, the bill narrowly passed the House of Commons in November 2024, but now faces significant opposition in the House of Lords.

Throughout these debates, palliative care has been repeatedly invoked. Yet the implications for palliative care research are rarely examined. If passed, the bill would not only alter clinical practice, but also reshape the priorities of those working in palliative and end-of-life care.

Dr Sallnow explains that a main concern among palliative care clinicians is the ethical burden that comes with classifying someone as eligible for assisted dying. Specifically, a major issue is prognostication: a ‘six-month’ prognosis is unreliable. Doctors are, in Dr. Sallnow’s words, “wrong more often than right”. Such uncertainty raises questions regarding eligibility and fairness.

Imagine, for instance, being diagnosed with a terminal illness and given two years to live, only to suffer a complication related to the condition and pass away six months later. In this scenario, you would have been ineligible for assisted dying, despite having a terminal illness, and therefore unable to die on your own terms.

Beyond this prognosis, statements like “unbearable suffering” and “mental capacity”, which appear throughout the bill and are used to determine eligibility, are difficult to assess. These challenges reflect the complexities of measuring suffering, which can be physical, emotional, social or spiritual in nature and can be difficult to quantify using traditional biomedical research methods. Palliative care research, as Dr. Sallnow notes, can help clarify, but cannot provide conclusive answers to individual cases.

Dr. Sallnow notes there is precedent for similar policies across other western countries, such as Canada, the Netherlands, Belgium and some US states. Assisted dying and euthanasia policies in these countries offer valuable insight into how assisted dying would work practically, who typically receives access, what safeguards should be present and what unintended consequences may occur. We can learn how a palliative care clinician deals with the difficult decision to classify someone as eligible for assisted dying, and adapt other countries’ legislative pitfalls to improve UK implementation. However, we cannot fully predict, without local implementation, how this translates to the UK’s NHS, legal and social context. Dr. Sallnow stresses the importance of thorough transitional and implementational research if the bill passes.

Regardless of whether the Assisted Dying Bill is passed, the debate surrounding it has brought death and dying back into public conversation. Death is universal, but in this context, has been marginalised and medicalised. As Dr. Sallnow states, the debate surrounding assisted dying has become poorly curated and highly polarised, framed through fear and misleading narratives. She advocates for widespread participatory research and citizens’ assemblies while introducing assisted dying to the UK. This would encourage informed deliberation rather than headline-driven debate, smoothing the implementation of the bill, were it to be passed.

The Assisted Dying Bill represents a significant step towards ensuring all British citizens have greater autonomy at the end of life. Dr. Sallnow’s research focuses on ensuring palliative care is available to all, and advocates for re-humanising healthcare. The Assisted Dying Bill could serve as a cornerstone of this broader goal. While polarising, the Bill has brought death and dying to the forefront of public conversation, and encourages broader thought on how to give each person an “honourable death” - one that aligns with their wishes, values and dignity.

*A special thanks to Dr. Libby Sallnow for her participation and collaboration in this article.*

**Artist: Doğa Aslan**



# *Tiny Leaders, Big Impact* How Bacterial uORFs Orchestrate Protein Synthesis

Author: Jihyeong Chang

Bacteria may be tiny, but their efficiency is staggering. They can sense environmental stress and adjust protein production within minutes – far faster than human cells. By producing only the proteins required in response to environmental cues, bacteria conserve energy while responding quickly to fluctuating environmental conditions. This remarkable adaptability, however, has consequences for humans: rapid bacterial responses contribute to the emergence of antibiotic resistance and enable pathogens to evade immune defences, driving an ongoing evolutionary arms race that makes the development of new antibiotics a constant necessity.

So, what allows bacteria to respond so quickly? The secret lies in a combination of factors, including fast reproduction and mutation rates, two-component regulatory systems, and the close coupling of

transcription and translation enabled by the absence of membrane-bound organelles. Together, these features allow bacteria to sense environmental changes and respond almost instantly.

More recently, advances in ribosome profiling and mass spectrometry have revealed the importance of small upstream open reading frames (uORFs) in bacterial adaptation. For decades, research focused on long, canonical ORFs, often filtering out short uORFs as background noise or non-functional elements due to their size or codon usage. However, emerging evidence shows that uORFs serve as key regulators, providing an additional layer of control over bacterial gene expression beyond classical promoter-based mechanisms. These tiny regulators influence which proteins are made, when, and in what quantities, thereby enhancing bacterial adaptability. Importantly, they also provide insight into host-pathogen interactions – knowledge crucial for developing strategies to combat antibiotic resistance in pathogenic bacteria.

Upstream open reading frames act as metabolic and stress sensors, modulating downstream gene expression in a condition-dependent manner. A classic example is the transcriptional attenuation of the tryptophan operon. Ribosomes translating a leader peptide sense intracellular tryptophan levels through the availability of charged tRNA-Trp. When tryptophan is scarce, limited tRNA-Trp causes the ribosome to stall. Since transcription and translation occur simultaneously in bacteria, this stalling directly influences the folding of nascent mRNA, favouring the formation of an anti-terminator hairpin that prevents premature transcription termination. The stalled ribosome along with the secondary mRNA structure, sterically blocks Rho-independent transcription termination and allows uninterrupted transcription of downstream tryptophan biosynthesis genes. This mechanism ensures rapid adaptation to tryptophan scarcity but also maintains stoichiometric protein synthesis in polycistronic operons. Polycistronic operons coordinate multiple proteins for a single function, and disruption of this balance can lead to misassembled multi-subunit complexes or accumulation of toxic intermediates.

Furthermore, uORFs are central to translational attenuation, adjusting protein synthesis in response to specific environmental cues, such as the presence of antibiotics. In macrolide-dependent regulation of the *ermC* operon, the antibiotic binds within the ribosomal exit tunnel, stalling the ribosome at defined codons in the leader peptide. This stalling alters mRNA secondary structure, exposing the ribosome-binding site (RBS) of the downstream *ermC* genes. The RBS is critical for initiating translation in

in prokaryotes, as it interacts with the 16S rRNA of the 30S ribosomal subunit. Exposure of the RBS enables translation of a methyltransferase, which modifies ribosomes to prevent macrolide binding, thereby conferring antibiotic resistance. Because methyltransferase production is energetically expensive, this conditional regulation ensures that the enzyme is only synthesised when needed, optimising energy use and allowing bacteria to respond rapidly without relying solely on transcription factor-mediated networks. However, such condition-dependent regulation can be detrimental if applied to housekeeping genes that require stable and constitutive expression to maintain cellular homeostasis.

Beyond their well-established cis-regulatory roles, preliminary evidence suggests that leader peptides may also function in trans, influencing multiple RNA targets throughout the cell. This raises the possibility that a single uORF could integrate into a broader, complex network controlling bacterial protein synthesis. While these transregulatory functions remain under active investigation, this rapidly emerging field is uncovering previously unrecognised layers of gene regulation. Together, these discoveries are not only reshaping our fundamental understanding of bacterial gene expression but also have potential implications for human health, including the development of novel antibiotics and strategies to combat antibiotic resistance. The continued exploration of uORF-mediated regulation promises to reveal new principles of cellular control and adaptive response, demonstrating that sometimes, the smallest elements can have the biggest impact.

# EUGENICS, BIOMETRICS, AND SURVEILLANCE

HAVE WE LEARNT OUR LESSON?

Author & Artist:  
Naomi Chung

In 2018, an inquiry led by Professor Iyiola Solanke from the University of Leeds to examine UCL's complicity in the development of eugenics led to the renaming of the Galton and Pearson Lecture Theatre, a public apology and a three-year Eugenics Legacy Education Project (ELEP) in 2021. UCL initiated the project with the aim to address and educate the public on the university's history with eugenics. The university has successfully emphasised its past in eugenics and the negative impacts of scientific discrimination through the project, which engages different departments in teaching the topic and displays related materials that most students and members of the public who have walked through the student centre would have noticed.

But what of the dangers in biased biometric data collection? With the university being scrutinised for its investments in the development of AI surveillance technology by genocide-complicit companies like Nice Ltd., which sells to Israeli military companies and is therefore accused by both the media and the student body of being complicit in the genocide committed against Palestinians, this begs the question: have we really learnt our lesson?



## THE INSEPARABILITY OF SOCIETY AND SCIENCE

There are plenty of resources on eugenics history available both in UCL and in academic literature, so I will not be reiterating that in this article, but I encourage you to give them a read if this article sparks your interest. One of the less commonly known but no less alarming facts is

that both Galton and Pearson started off as mathematicians and statisticians, and Galton's Laboratory for National Eugenics at UCL started as a founding ground for statistical studies. Galton, passionate about data collection and interpretation, invented the Galton board, which

visualises the central limit theorem (the normal distribution curve of a standard distribution). He also developed the criminal identification system with Parisian policeman Alphonse Bertillon, which records an individual's anthropometric data (like fingerprinting), and founded the new discipline of eugenics, the study of selectively breeding desirable traits in humans.

Although seemingly objective and innocent, contemporary social attitudes towards race, gender, and intelligence were incorporated into Galton's scientific research. Moreover, eugenics attributes characteristics such as criminality,

intelligence, mental illness (or 'feeble-mindedness'), and poverty to genetics rather than social conditions. In turn, these pseudo-scientific theories shaped society to fit into these theories by influencing governing policies to achieve population control. The most notorious example was the Holocaust under the Nazi regime, where ordinary people with 'undesirable' traits of Jewish or Roma heritage, disability, and/or homosexuality were eliminated in the name of eugenics. Dehumanisation becomes easy when human differences are interpreted as data points, and individuals are reduced into a generalisation to justify scientific racism.

## BIOMETRIC IDENTIFICATION AND SURVEILLANCE

The photographic portraits, phrenology head casts, and biometric measurements of Galton's archive represents a form of surveillance and social deviant regulation through the collection of biometric data that still persists in present times. Nowadays, the average person would be familiar with the normalised daily usage of fingerprint and iris scanning, facial and voice recognition, or keystroke recognition. Heat sensors and surveillance cameras are often used in public spaces to monitor population flow, just like the Passive Infrared (PIR) sensors used to monitor occupancy patterns for more efficient energy management on the UCL campus back in 2018-19. As Pramod K. Nayar states, surveillance has less to do with an individual's identity than identification; it authorises who we claim to be. It turns the individual into a set of data that becomes inseparable from the body. Whilst surveillance technologies benefit us in deterring crimes and verifying identities, concerns about breaches of privacy and data security are reflected by public sentiment towards the non-consensual use of facial recognition software at

the King's Cross Estate in 2019 and the recently proposed mandatory digital ID in December 2025, which was quickly withdrawn a month later.

The undeniable fact is biometric data and identification have become intrinsic to social systems and infrastructure, whether it is at the local scale of the UCL campus or the global scale of international border control. Some fear that the progressive increase in control and invasion of privacy is an alarm bell for an Orwellian surveillance state. However, whilst the individual might feel a lack of control over one's biometrics, it is our moral duty as the future generation of policy makers and society stakeholders to regulate and safeguard the ethics of data usage. UCL's past complicity cannot be changed, but it does not have to repeat its own history.




# The squid & THE SCIENTIST

Maxime Chautemps  
Artist: Amy Joseph



Imagine this: you are a spiny dogfish - a slender, smooth and spined shark travelling in a pack of 100s. You swim through the dim light of the benthic depths, darting aggressively after whatever prey you spy that's smaller than you. Then, you see them. A soft ethereal form at the edge of your vision, drifting gently, almost passively, through the murky water. You know that movement, that shape - sense their electric field - and so you thrust yourself towards them using the sideways undulation of your caudal fin. You extend your jaw and launch at it, only to shake your head in frustration milliseconds later as you get a mouthful of inky black mucus that scrambles your senses instead. In the distance you can just make out your missed meal already darting tens of metres away.



Jet propulsion is a mode of transport available to most cephalopods - the class of molluscs that includes octopuses, nautiluses, and cuttlefishes - but is perhaps most famous in the squids of the group. It serves a range of functions: from quick escapes, powerful predatory strikes, and even temporary "rocket propulsion" flight in several species.

Most of the time squids move by undulating their fins and gently expelling water out of their mantle, the muscular organ surrounding the core body like a hat. Water is taken in and expelled through a tubular extension of the mantle called the siphon that can orientate itself in multiple directions. When they need to generate a powerful but energy-taxing thrust, they can relax their mantle to draw in a larger amount of water then contract rapidly to force it out. This rapid movement is stimulated through an instantaneous electrical impulse

that reaches all the muscles of the mantle simultaneously, only made possible thanks to specialised giant axons. These massive nerve fibers can be up to 1mm in diameter, 1000s of times wider than those in most vertebrates.

John Zacchary Young (1907-1997), a decade before he took up a post as Professor of Anatomy at UCL, redescribed these giant nerve fibres and brought them into the limelight of neurophysiology. Seemingly forgotten since Leonard Worcester Williams first described it in the longfin inshore squid (*Loligo pealii*) in 1909, Young rediscovered the giant squid axon completely by accident.

He had been working at the Stazione Zoologica in Naples where his collaborator Enrico Sereni introduced him to cephalopods as model organisms. Without an express end-goal, Young began investigating different structures in cephalopods, leading him to compare a strange ganglion structure he found in *Eledone* octopuses with *L. pealii* squids. Whilst he didn't find the same structure, he did find something even stranger. A large ganglion in the mantle with star-like radiations of tubular structures big enough to see with the naked eye.

It wouldn't be until 1938, after years of work split between the Plymouth Marine Laboratory and the Marine Biological Laboratory at Woods Hole, that Young would officially redescribe the stellate ganglion and prove its fibres to be massive motor neurons.

From the moment of its discovery, the squid giant axon revolutionised neuroscience. Its unique size allowed electrodes to be placed on both sides of the neuron, which teams at





Plymouth and Woods Hole used in 1939 to measure the action potential across the axon for the first time. Alan Hodgkin and Andrew Huxley would later win a Nobel prize for their use of the giant squid axon to describe action potentials at the molecular level.

J. Z. Young went on to contribute to many areas of research in zoology and physiology. During WWII he worked with Peter Medawar at Oxford, where they once again used squids and octopuses as their models: this time to find treatment for wartime nerve injuries. Afterwards, he moved to UCL, where he worked for nearly three decades.

Reflecting on the impact cephalopods have had on neuroscience, J. Z. Young mused in a 1984 article that, “It is curious to think how different neuroscience would have been had I not made sections of a yellow spot out of simple curiosity.” He wondered then if, had he been an early-career scientist instead, whether the climate of science at the time would have allowed him to make such a discovery.

But forget 1984, what would Young think of the state of science and academia in 2026? How can that “simple curiosity” that research and innovation thrives off survive today when papers are trapped in the purgatory of a broken peer-review system, when wealth inequality prevents so many aspiring scientists from advancing, and when governments continue to gut funding?

We often characterise the investigative behaviour of octopuses as curiosity. Serious change is needed at UCL and throughout academia if scientific research is to continue being accessible, inclusive, and by extension curious. We shouldn’t be afraid to be more like an octopus, allowing students and researchers to stretch out their limbs towards wherever their curiosity is calling, sometimes in multiple directions at once.



# 200 YEARS OF BRAINS AT UCL AND ONE THAT DID NOT AGE WELL

*AUTHOR: AMY JOSEPH*

Jeremy Bentham is a name familiar to most philosophy enthusiasts; to many UCL students, he is a more literal presence. Bentham's 194-year-old 'Auto-Icon' (a self-coined term to describe his preserved body) sits in the Student Centre, watching over the university shaped by his utilitarian ideals. While it is widely known that Bentham's preserved head was once stolen by KCL students (for a fine ransom of £10), far fewer are aware that Bentham requested his head be preserved using the Mokomokai method of the Māori people—and that his friend (who also served as his embalmer) attempted to carry this out with disastrous results.

Central to Mokomokai preservation was Ta moko, intricate tattoos carved into the face using bird bones. When a person with facial moko passed away—often a great leader or a cherished individual—their head would be preserved in a process developed over generations. It was only in the 19th century that this tradition declined, as British colonialism and the commodification of Mokomokai as 'curios' fuelled their trade in exchange for weapons. After the Musket Wars subsided, this ancient practice diminished.

Mokomokai preservation was relatively simple but scientifically grounded. Firstly, biological control was established: all soft tissues, most notably the brain, were removed to prevent putrefaction. The head cavities and nasal orifices were then filled with clay and vegetal fibres, respectively. These steps reduced moisture, limiting

bacterial metabolism and consequent gas build-up. Mechanical interventions then followed to preserve facial topology. Using vegetal fibres, the eyelids and nostrils were sewn shut, which also prevented the entry of microbes and insects. The cervical skin was fixed to the skull base and a wooden pin was inserted under the nose to prevent lateral deviation of the skin when dried in subsequent steps. These measures ensured even shrinking, allowing both the detailed Ta moko and the facial features of the deceased to remain recognisable.

Thermal fixation was the next ingenuity: the head was steamed or boiled, then smoked, and finally sun-dried. The use of wet heat ensured controlled and sustained temperatures that destroyed both decomposing microorganisms and tissue-digesting hydrolytic enzymes from the body's own cells. Furthermore, when exposed to high enough temperatures, proteins first denature (uncoil their chains) and then permanently coagulate (clump together).

Think of the way an egg white firms up as it cooks. Once coagulation occurs, proteins in the skin form a dense, three-dimensional network that resists the mobility of small molecules, such as water, perhaps contributing to the long-term stabilisation of the tissue. In addition, phenols and acids present in the smoke contribute to its antimicrobial properties, further preventing decay. Sun-drying as the penultimate step ensured slow, even dehydration of the skin.

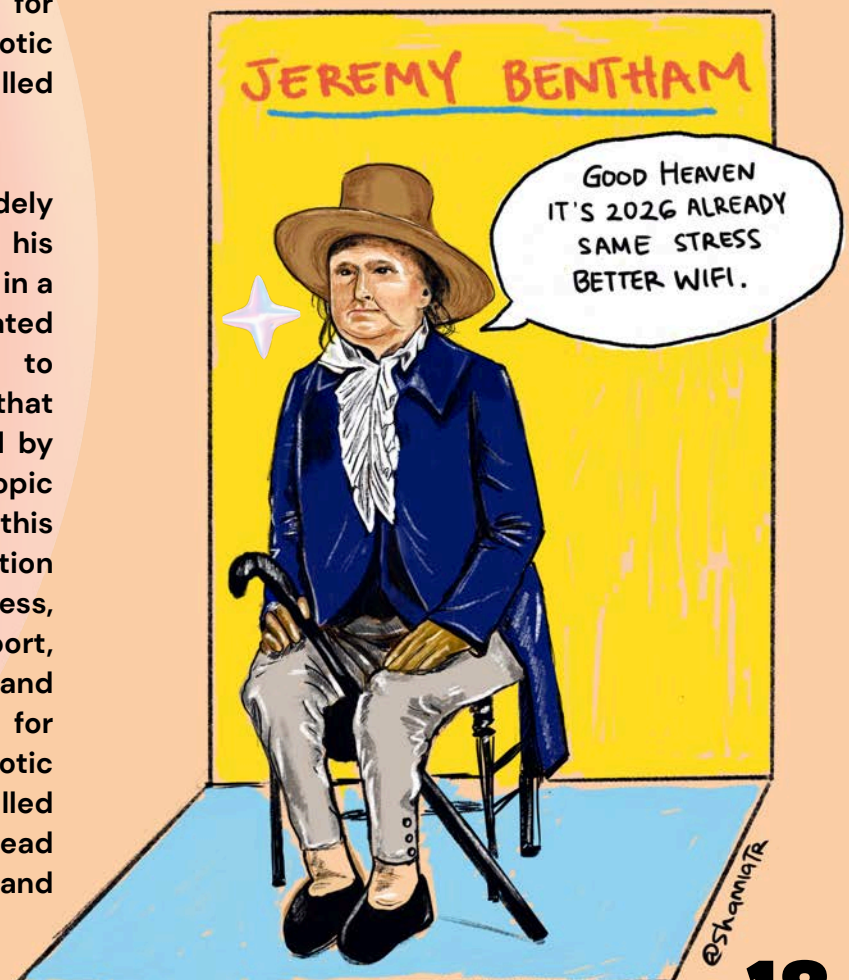
The finishing touch—the application of shark oil on the Mokomokai—both moisturised the skin and reduced its consumption by insects. Shark oil is hydrophobic: insects repelled by its stench would struggle to lay eggs on its slippery surface. These factors, coupled with the possibility of the oil clogging the spiracles of the hungry invertebrate, would make the embalmed head a very unprofitable meal.

In contrast, Bentham's head was crudely preserved. After removing the brain, his friend Dr Thomas Smith placed the head in a sealed chamber with concentrated sulphuric acid, using an air pump to circulate acidic vapours. Moisture that evaporated from the head was trapped by the acid, which is strongly hygroscopic (extremely absorbent of water). While this technically fulfilled the dehydration requirements of the embalming process, due to the lack of mechanical support, Bentham's face became shrunken and unrecognisable. Furthermore, disregard for thermal fixation caused chaotic denaturation of proteins and uncontrolled lipid oxidation.

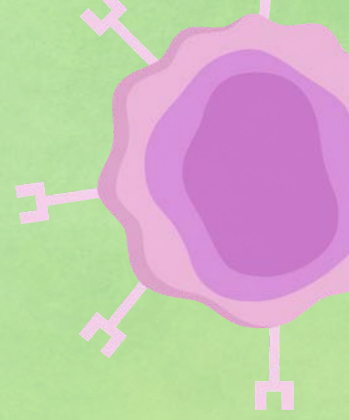
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Today, Mokomokai heads are globally dispersed in museums, including the British Museum, as a result of their theft and trade. Remarkably, Te Papa Tongarewa's ongoing repatriation efforts have led to the return of several Mokomokai heads to their rightful communities. Bentham's head, however, is kept in a secret location in UCL - presumably to hinder further thefts by unscrupulous students or prevent nightmares among those unfortunate enough to glimpse his macabre expression.

## ARTIST: SHANNIA TRITAMA



# CAR-T CELLS



## A NOVEL CANCER TREATMENT BY GENE EDITING

Patrick Toh & Celine Tedja

Artist: Tiana Lee

Cancer is well known to be highly complex and diverse, which makes it difficult to combat with conventional methods such as surgery, chemotherapy, and radiation. Thanks to advancements in gene-editing technology and ongoing efforts to explore new treatment modalities, scientists have successfully developed a 'living drug' in which a person's immune cells are genetically engineered to fight cancer cells. This is known as CAR T-cell therapy. Recently, a team led by Professor Waseem Qasim at UCL and Great Ormond Street Hospital (GOSH) made headlines for reversing an aggressive blood cancer with CAR T-cell therapy, with the first treated patient having been cancer-free for 3 years now. This story has sparked even greater interest in this novel treatment, which is considered a major leap in cancer research. UCL, in fact, has been at the forefront of CAR T-cell therapy research, having established its own CAR T-cell program.

### THE SCIENCE BEHIND CAR-T CELLS

CAR T-cell therapy is a type of adoptive transfer therapy, a form of immunotherapy that collects immune cells from a patient, grows them in a laboratory, and returns them to the patient. CAR T-cell therapy involves T cells, which are immune cells that recognise foreign pathogens or particles not normally found in our bodies. T cells have been shown to kill cancer cells; however, they struggle to recognise them. This is because cancer cells can 'hide' from them in various ways – for

example, by losing key antigens that allow immune cells to recognise them or by releasing signals that prevent anti-tumour activity. This is where chimeric antigen receptors (CARs) come into the picture. Engineering T cells to express these receptors on their surface enables them to recognise specific antigens on cancer cells, consequently activating T cells to kill cancer cells.

To engineer T cells to express CARs, blood is first collected from the patient of interest. T cells are isolated from the blood, and the remaining blood is returned to the patient in a process known as leukapheresis. Next, the collected T-cells are genetically altered by inserting the gene encoding the CARs using a viral vector. The edited T cells are then grown in the lab until there are millions of them – enough to target cancer cells effectively. Finally, the products are returned to the patient's bloodstream as an infusion. At present, this entire process takes about 3–5 weeks.

### ADVANTAGES AND LIMITATIONS

What has made CAR T-cell therapy a spotlight in cancer research? The answer lies in its ability to overcome the limitations of conventional treatments. CAR T-cells are 'living drugs', as they are cells. These cells persist long-term in the body, so even a single infusion can result in remission that lasts for years, as shown in clinical trials.

Moreover, CAR T-cells are designed for precision targeting – their chimeric antigen receptors (CARs) make them highly selective for cancer cells, unlike chemotherapy or radiotherapy, which also damage healthy cells.

Nonetheless, CAR T-cell therapy poses several drawbacks. In some cases, it led to toxicities, namely cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS). These are inflammatory responses caused by the surge of cytokines, a group of signalling molecules that activate other cells along the immune response pathway. To manage this side effect, drugs that inhibit IL-6, a major cytokine, can be administered. Another limitation is that CAR T-cell therapy currently appears to have limited efficacy in solid tumours, such as lung cancer, as the microenvironments associated with solid tumours prevent CAR T cells from accessing them.

## BRIEF HISTORY

Despite the relatively recent success of CAR T-cell therapy, the development of T-cell editing dates back to the 1980s. The concept of adoptive transfer therapy was first described by Dr Steven Rosenberg, but it wasn't until 1989 that the first generation of CAR T cells was developed by immunologist Dr Zelig Eshhar. His team developed chimeric T cells known as 'T-bodies', which are essentially simple T cells fused with antibodies. In 1993, Dr Eshhar collaborated with Dr Rosenberg and Dr Patrick Hwu to develop receptors to be engineered onto tumour-infiltrating lymphocytes, and Dr Hwu later showed that these cells could kill ovarian cancer cells in mouse models in 1995. Ever since, new generations of CAR T cells have been developed, each time introducing new features to enable them to recognise and fight against cancer cells more effectively. As of now, six CAR T-cell therapies have been approved by the FDA for treating various forms of blood cancer, such as acute lymphoblastic leukaemia (ALL), diffuse large B-cell lymphoma, and multiple myeloma.

Scientists are now engineering 'off-the-shelf'



allogeneic CAR T cells, in which T cells are collected from healthy donors rather than patients. This method offers the advantages of reduced cost, improved efficacy, and enhanced safety. One such off-the-shelf CAR T-cell is base-edited CAR T-cell therapy developed by Professor Waseem Qasim and his team at UCL and Great Ormond Street Hospital (GOSH) to treat T-cell ALL, which commonly affects children. They used gene-editing technology to remove a CD7 marker that causes CAR T-cells to recognise and kill one another. In its phase 1 clinical trial involving eight children and two adults, 82% of patients achieved remission, and 64% remained disease-free three years after administration.

## FUTURE DIRECTION

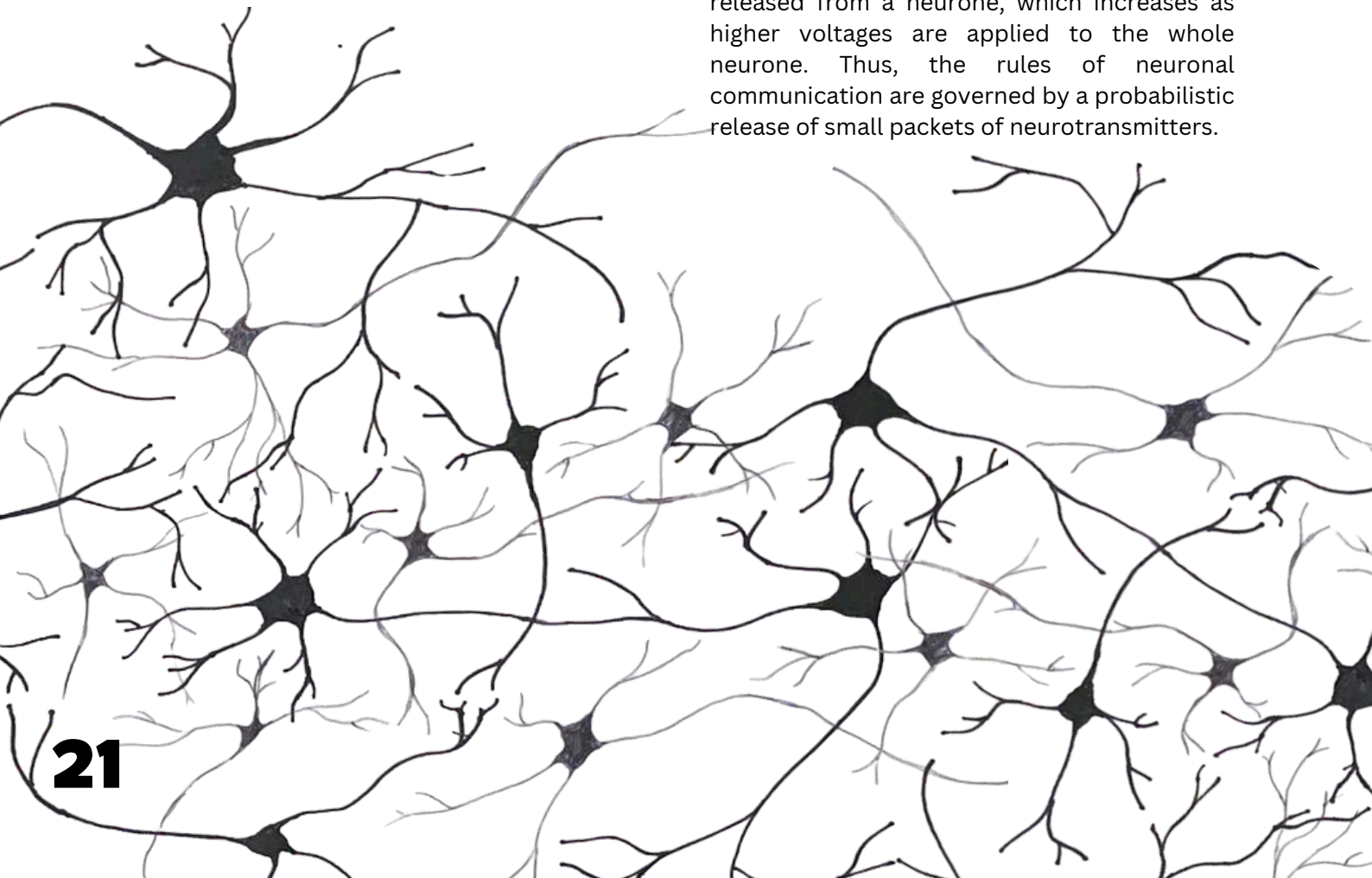
Given the optimistic results of CAR T-cell therapy trials, this innovative treatment has significant potential for widespread implementation, subject to regulatory approval. One particular challenge in translating CAR T-cell therapy from bench to bedside is the high manufacturing cost; however, this has been addressed by developing off-the-shelf allogeneic CAR T cells, which can be manufactured in large quantities and stored until needed. Another challenge is that progress in CAR-T cell development against solid tumours has stalled, so research on CAR T cells has been primarily focused on blood cancers. This highlights an unmet need and raises an important question: how can CAR T-cell therapy be adapted to treat a wider range of cancers?

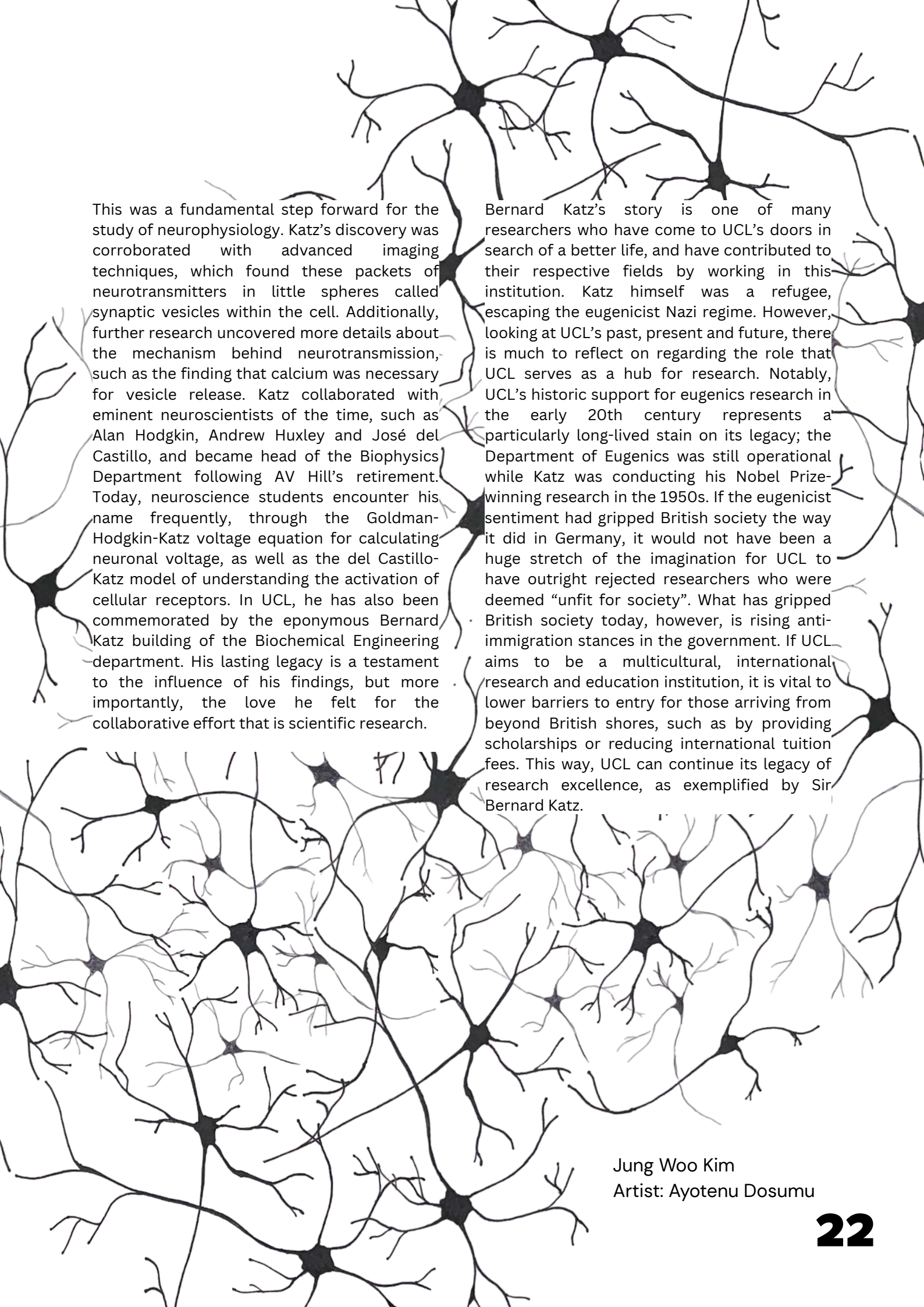
# BERNARD KATZ: UNDERSTANDING THE GRAMMAR OF NEURONES

In neuroscience, there are numerous mathematical equations and models immortalising the names of neuroscientists who have revolutionised the field. As we celebrate UCL's bicentenary, marking 200 years of being a vibrant hub for education and research, let's take a look at one such name whose work on neurotransmission at UCL earned him the Nobel Prize in Physiology or Medicine in 1970: Sir Bernard Katz.

Bernard Katz, son of a Jewish fur trader, arrived on British soil penniless in February 1935, less than a year after completing his medicine degree in Leipzig, Germany. Fleeing Nazi persecution, he was accepted by Professor AV Hill of UCL to pursue PhD work in understanding the communication of neurones. After a stint in Sydney, in which he obtained British nationality and served in the Royal Australian Air Force, he returned to UCL in 1946 to set up the Department of Biophysics with AV Hill and to continue his work on neuronal communication.

The early 20th century was an exciting time for neuroscience research. It was marked by the discovery that, though neurones conduct electrical signals, they communicate with each other chemically by releasing chemicals called neurotransmitters across the synapse, the gap between neurones. This raised the question: How? There were many possibilities, from emitting a fluctuating stream of neurotransmitters, to releasing them in an on-or-off way. To answer this question, Katz worked with the frog neuromuscular junction, a common animal model due to the large size of synapses between neurones and muscle cells. By inducing a tiny voltage on the neurones to induce spontaneous neurotransmitter release, he found a set of rules that governed the amount of chemicals released: the distribution of neurotransmitter release could only be explained if they were released in standardised packets, described as "quanta", the same word used to describe the smallest discrete units of light called photons. Indeed, he found that these packets each have a probability of being released from a neurone, which increases as higher voltages are applied to the whole neurone. Thus, the rules of neuronal communication are governed by a probabilistic release of small packets of neurotransmitters.





This was a fundamental step forward for the study of neurophysiology. Katz's discovery was corroborated with advanced imaging techniques, which found these packets of neurotransmitters in little spheres called synaptic vesicles within the cell. Additionally, further research uncovered more details about the mechanism behind neurotransmission, such as the finding that calcium was necessary for vesicle release. Katz collaborated with eminent neuroscientists of the time, such as Alan Hodgkin, Andrew Huxley and José del Castillo, and became head of the Biophysics Department following AV Hill's retirement. Today, neuroscience students encounter his name frequently, through the Goldman-Hodgkin-Katz voltage equation for calculating neuronal voltage, as well as the del Castillo-Katz model of understanding the activation of cellular receptors. In UCL, he has also been commemorated by the eponymous Bernard Katz building of the Biochemical Engineering department. His lasting legacy is a testament to the influence of his findings, but more importantly, the love he felt for the collaborative effort that is scientific research.

Bernard Katz's story is one of many researchers who have come to UCL's doors in search of a better life, and have contributed to their respective fields by working in this institution. Katz himself was a refugee, escaping the eugenicist Nazi regime. However, looking at UCL's past, present and future, there is much to reflect on regarding the role that UCL serves as a hub for research. Notably, UCL's historic support for eugenics research in the early 20th century represents a particularly long-lived stain on its legacy; the Department of Eugenics was still operational while Katz was conducting his Nobel Prize-winning research in the 1950s. If the eugenicist sentiment had gripped British society the way it did in Germany, it would not have been a huge stretch of the imagination for UCL to have outright rejected researchers who were deemed "unfit for society". What has gripped British society today, however, is rising anti-immigration stances in the government. If UCL aims to be a multicultural, international research and education institution, it is vital to lower barriers to entry for those arriving from beyond British shores, such as by providing scholarships or reducing international tuition fees. This way, UCL can continue its legacy of research excellence, as exemplified by Sir Bernard Katz.

Jung Woo Kim  
Artist: Ayotenu Dosumu

# The Public Health Reforms behind Cruciform's Fairy Tale Tile Paintings

*Victoria Mesrobyan*  
*Artist: Ayotenu Dosumu*



UCL's Cruciform Teaching Labs: a space to spend three hours pipetting and trying your hardest to remember what Step Twelve of the protocol meant. However, right out in the open, only noticed by those who have waited forty-five minutes to process a gel electrophoresis, are a set of Royal Doulton tile paintings displaying a series of fairy tales. These tiles nod to a time when the Cruciform building was a hospital: when the very same teaching labs served as a children's ward. Although they may seem like simple decoration, it was crisis that painted these fairytale tiles.

The early twentieth century was a pivotal period in the UK for advancements in understanding disease and public health. Led by social reformer Edwin Chadwick in collaboration with various scientists, the "Sanitary Awakening" linked poor sanitary conditions, overcrowding, inadequate water sanitation, and waste disposal to disease, using data to encourage parliamentary action. This awakening paved the way for successive Public Health Acts, which created and updated public health structures, laws, and administrative systems. The 1875 Public Health Act made it compulsory to provide fresh water, improve sewer systems and appoint medical officers, marking the beginnings of modern public health infrastructure and establishing a national standard.



Despite these advancements, inner cities were still subject to rapid urbanisation with overcrowding and poor sanitation persisting. These conditions were reflected in hospitals, which became an environment for both healing and the contraction of disease. Hospitals began using tiles around 1901 for their sanitary properties. They were valued for being non-porous, easily cleaned, and resistant to harbouring infection.

With children being a group of utmost concern, tiles were especially favoured in children's wards. Most children at the time were malnourished and lacked acquired immunity, making the younger population particularly susceptible to parasitic and infectious diseases. These diseases included epidemics of diarrhoea, tuberculosis, diphtheria and scarlet fever, causing children under five years of age to make up nearly a third of annual deaths during this period.

With the foundations laid and serious problems identified, action followed. The appointment of a new Liberal government in 1906 created an opportunity for legislation to focus on specific populations: children, the sick, the elderly, and the unemployed. In addressing childhood disease, attention naturally turned to schools. Schools adopted free school dinners in 1906 to improve malnutrition, and the Education (Administrative Provisions) Act in 1907 instilled a School Medical Service, providing three compulsory medical inspections for primary students per year, with the goal of identifying problems early. Although there was limited treatment for issues found in the inspections, this represented a significant and popular step in public health.

With increased attention on child health and welfare, sanitation enabled art. Hospitals began commissioning artists for murals, focusing particularly on children's wards, with the goal of adding colour and entertainment. Known for her murals with characteristic fairy motifs, Margaret E. Thomson was arguably at the forefront of this trend. She completed work for the Royal Victoria Infirmary, St Thomas's Hospital and the former University College Hospital: now the Cruciform building. Today, the Cruciform tiles, which previously brightened a children's ward, remain on UCL's walls as a testament to how public health reforms and scientific understanding can reshape cities.



# Engineering the Future

## UCL's Bicentennial AI Breakthrough in Immunology

**Pathrakorn Kuratana**

Artificial Intelligence (AI) has been rapidly rising in development and usage, with notable AI models from industry increasing from 60% in 2023 to 90%. Developments for the biological academia field include technology such as AlphaFold that uses an amino acid sequence to predict a protein's 3D structure, helping researchers identify protein structures which would've needed crystallography instead. Growth for this field continues to develop and is happening close by. Within the Faculty of Life Sciences at UCL, a team of researchers has developed an AI tool, ImmunoMatch, that predicts which antibody components naturally pair together.

### **ImmunoMatch – What is it, and how does it work?**

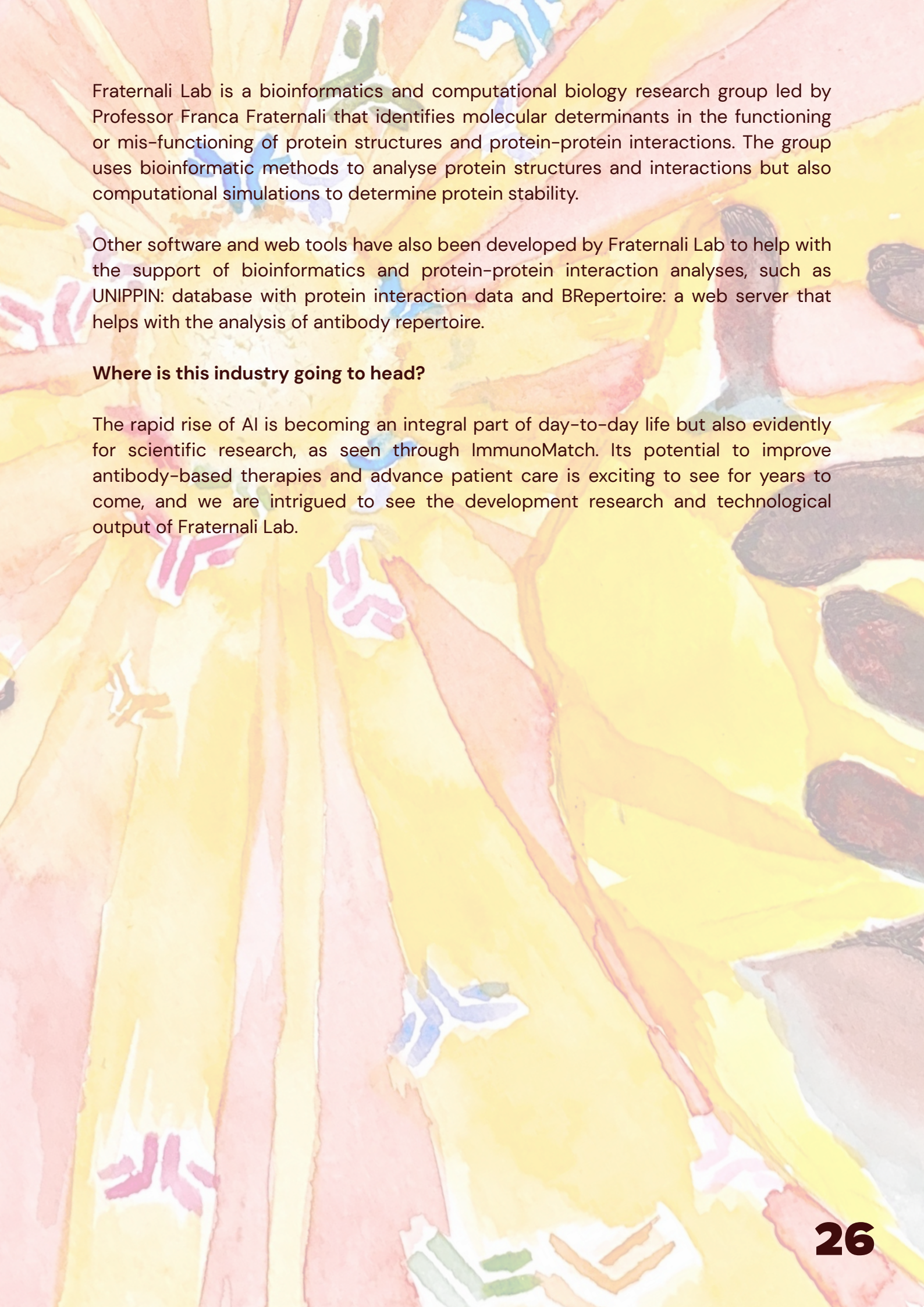
ImmunoMatch is an AI tool developed to help predict and understand the assembly of antibodies in the human body, allowing the speeding of therapeutic antibody design.

Antibodies are “a protein component of the immune system that circulates in the blood, recognises foreign substances like bacteria and viruses, and neutralises them”. They are incredibly important, as they are key players in immune defence and are used as therapeutic drugs. ImmunoMatch is able to correctly distinguish heavy-light pairs from random pairings and learns features associated with different chain types. It can then be applied to spatial sequencing data to reconstruct antibody pairing when direct pairing information is not available.

Strong advancements in immunological research can be boosted by ImmunoMatch, where underlying rules of antibody assembly in human B cells can be revealed. Improvement in the computational design of antibodies and validation of therapeutic antibodies as a result can also potentially accelerate drug development, allowing for pharmaceutical progression to take place and plausibly leading to next-generation drugs. This technology could help scientists decode immune responses, design better antibody-based therapies, and even use it for personalised medicine.

### **Faces behind the paper**

Co-authors Donjun Guo, Joseph C. F. Ng and Franca Fraternali are all part of the Fraternali Lab here at UCL, where they worked alongside Deborah K. Dunn-Walters, a Professor of Immunology and Associate Dean Research and Innovation at the University of Surrey.

The background of the page is an abstract watercolor illustration. It features a warm color palette of yellows, oranges, and pinks, with some cooler tones of blue and green. The style is soft and painterly, with visible brushstrokes and blended colors. There are several stylized, colorful shapes scattered throughout, which appear to be representations of biological structures, possibly antibodies or protein complexes, rendered in shades of blue, green, pink, and purple. The overall composition is dynamic and artistic, suggesting a focus on life sciences or biotechnology.

Fraternali Lab is a bioinformatics and computational biology research group led by Professor Franca Fraternali that identifies molecular determinants in the functioning or mis-functioning of protein structures and protein-protein interactions. The group uses bioinformatic methods to analyse protein structures and interactions but also computational simulations to determine protein stability.

Other software and web tools have also been developed by Fraternali Lab to help with the support of bioinformatics and protein-protein interaction analyses, such as UNIPPIN: database with protein interaction data and BRepertoire: a web server that helps with the analysis of antibody repertoire.

### **Where is this industry going to head?**

The rapid rise of AI is becoming an integral part of day-to-day life but also evidently for scientific research, as seen through ImmunoMatch. Its potential to improve antibody-based therapies and advance patient care is exciting to see for years to come, and we are intrigued to see the development research and technological output of Fraternali Lab.



# BLAKENEY POINT

## *The need for living classrooms*

Lauren Grady

Artist: Annie Hollis-Barter

Along the Norfolk coastline and bordering the North Sea lies Blakeney Point, a seemingly unassuming field station nestled among sand dunes and grassy hills. A closer look into the point reveals a strikingly biodiverse ecosystem, a crucial piece of UCL history, and the starting point to coursework for biodiversity postgraduates.



Students leaving Blakeney Point in June 1955  
(Image provided by Tim Blackburn).

Blakeney Point has seen visits from UCL students for over a century. First established by Francis Wall Oliver, a professor of botany at UCL and field ecologist, the location of Blakeney Point was chosen due to its unique biodiversity and the dynamic nature of its geography. Today, a field trip to Blakeney Point serves as the start of two master's courses: the MRes in Biodiversity, Evolution, and Conservation and the MSc in Biodiversity and Global Change.

"I've been going to Blakeney Point with UCL since about 2013 or 2014, but I've been going as a birdwatcher since the mid-1980s,"

says Dr. Tim Blackburn, a professor of invasion biology and course lead for the MRes in Biodiversity, Evolution, and Conservation. "I just love the whole thing really."

The start of the master's courses through a field trip to Blakeney acts as an introduction to ecology and fieldwork, as well as the professors and course mates. While at the point, students are able to explore the local ecosystem through an exercise known as a 'bio blitz,' wake up early with the professors to open moth traps, and gain insight into field data collection and experimental design. This year, students were instructed to design a study to measure the prevalence of brown hares on the point. When asked why students are taken to the point at the beginning of each academic year, Blackburn emphasised the importance of experience. "It gives students a feel for what working in the field is like and exposes them to the systems we talk about [in lecture]."

Hands-on experience and the concept of a 'living classroom' are a crucial part of learning, especially in subjects like ecology. Living classrooms provide an immersive experience where students can apply the concepts and terminology they have learnt to the real world. These ideas are long-standing in environmental education. According to Kinslow et al. (2019), field-based environmental curriculums promote environmental literacy and socio-scientific reasoning in high school students, which aid critical thinking on modern environmental issues. Similar studies have been conducted on students in higher education, in which students not only benefitted academically from field-based

learning opportunities but also preferred it to more traditional classroom learning. Blackburn echoed these sentiments when discussing the importance of Blakeney to environmental education at UCL: “Most of the course is theory, so getting people out sort of into the systems we’re talking about [and] thinking about the actual organisms in these systems is key.”



The Blakeney Point bunkhouse  
(Image credit: Lauren Grady).

Aside from hands-on learning, the Blakeney Point field trip also serves as a way for students and professors to connect with nature and each other. The single bunkhouse, split over two rooms and 10 bunks, is a cozy space where up to 15 students stay at a time, and a small dining hall with a singular table is the site where all meals are shared. “We really value the opportunity to get to know [the students],” says Blackburn. At downtime, students and professors find themselves conversing, reading, or exploring the nature preserve. The sense of community at Blakeney amongst students is palpable, and post-dinnertime allows for cards, games, and

more bonding in the dining hall.

Though the time students share at Blakeney is brief, the experience is invaluable and remains a frequent topic of discussion in the taught coursework of biodiversity postgraduates. The introduction to experimental design prepares master’s students for their research projects later on in the course, and the exploration of Blakeney’s biodiversity aids students in discovering an appreciation for local wildlife. Blakeney Point has played a crucial role in environmental education at UCL for over 100 years and will continue to do so for generations of ecology students to come.



# Science: A Revolution Led By Women

**Erika Gherman**

Since its foundation in 1826, UCL has emerged as a symbol of reformism and inclusivity. In a time of great disparities, the university promoted progress by accepting students from different backgrounds and faiths, distinguishing itself from other UK educational institutions and the conservative society. 50 years later in 1878, UCL pushed this mindset further, opening its doors to a repressed but consequent part of the society. A community which despite representing half of the population was often erased from history, unrepresented, powerless and restricted from freedom: women.

UCL was one of the first UK universities to enrol female students on the same terms as their male counterparts, marking a turning point in the intellectual and social construction of society and academia. Since then, thousands of female students, researchers, and professors have roamed these hallways, each adding a stepping stone to science while facing challenges and defying conventions.

While celebrating UCL's 200th anniversary, this revolution is still ongoing, with changes actively being made by the new generations to reduce the still-existing inequalities.

Women's access to education in the UK began in 1868 in London, when the University of London's Senate authorised female candidates to sit examinations across a panel of subjects. Although this initial reform did not lead to degree completion at the time, it proved to be a necessary *démarche* that yielded its first results 10 years later, when the first women were able to enrol and earn a degree under the same conditions as men.

However, the acceptance of female students by educational institutions was only the start of a long battle for recognition. Criticised for pursuing

a career, stolen work, exclusion from leadership: those are only a few of the difficulties a woman had to overcome to complete her studies and to be acknowledged for her contribution to research.

Even in a field full of impediments, women created their own opportunities and, one step at a time, revolutionized science.

Spanning centuries and still ongoing, this transformation is one that each individual woman plays an essential part of, from the first two female students graduating with a BSc in 1881 to some of the biggest researchers of today. One of the pioneers of science and women's emancipation, was UCL's first female professor in 1949, Dame Kathleen Lonsdale for chemistry. She is particularly esteemed for her use of crystallography and her nomination in 1945 as one of the first two women fellows of the Royal Society.

Through education women forged themselves an active place in the world and brought profound changes in science; with women came new perspectives and discoveries that built the knowledge we have today.

UCL recognizes the importance of gender equality in science for its evolution and stands out through its dedication to reducing gender gaps and supporting the ambitions of female students. Key actions taken within this scope include the annual celebration of Women's Day, adherence to a gender equity programme; and compliance with the Athena SWAN Charter, a UK framework aiming to reduce gender inequalities in STEM fields.

Alongside the institution, its female scientists are first to take an active role in leading those movements.

That may be respected researchers, such as

Dame Uta Frith, Emeritus Professor of Cognitive Development for UCL's Institute of Cognitive Neuroscience, and previous member of two MRC's units affiliated to UCL. Renowned in the field for her research on developmental disorders such as autism or dyslexia, she is also a militant for women's rights in academia, represented through numerous actions such as her co-founding of UCL's "Women Network" in 2013, a social organization offering a space advocating for women's empowerment.

Or it could be students who create societies to empower their female peers. Two visionary students, committee members of UCL's Women in STEM Society, shared their own actions and the message they want to communicate. This society supports events ranging from relaxed socials for female students, to regular talks with guest speakers from different areas of STEM. Their aim is "to provide a platform for women in different STEM disciplines to network and be inspired by each other" according to the society's events officer, Sun Yu. Their social media officer, Ayotenu Dosumu, expressed the society's desire to create a strong sense of community and to make career opportunities accessible to members. Both members made a point of women's challenges and underrepresentation in STEM being their strongest drive in pursuing their roles.

Ayotenu Dosumu emphasizes the inequalities met by women of colour in science, recalling the lack of Black STEM lecturers or students on her course: "Experiences like this highlight how isolating STEM spaces can feel and how important spaces like Women in STEM are". The society makes it possible for all female students to find a role model in their career path.

Even after 200 years, UCL remains a pioneer of inclusion, following its foundation principles by encouraging positive change in the community. Through the support of students' projects and the creation of safe and inspirational spaces for women, the university actively reduces gender inequalities. These actions led to certain disciplines, such as biology, reaching over 50% of female students. However, those advancements are not equally distributed, as subjects like engineering remain predominantly dominated by men.

Outside of UCL, the battle also continues with deep inequalities persisting across the UK, as only 27.6% of the UK STEM workforce is composed of women, according to workforce data, with only 17% of science professors being women. Those numbers highlight persistent gender gaps, even more pronounced for women of colour.

The under-representation of women in science brings much deeper concern, as it is one of the origins of scientific bias. In medical research especially, male bias has been recognised and discussed for years by many medical institutions. Healthcare doesn't meet women's needs due to insufficient understanding of diseases and symptoms affecting them, stemming from the lack of research done on women's biology and health conditions.

The situation calls urgently for an increase in equity and training of female scientists. Female professors are critical contributors, preparing the new generations of students who carry the development of science.

In between the legacy of our predecessors and the future yet to be written, it is in the hands of today's students to continue this revolution.



**Artist: Megan Wilkinson**

# PROTEINS

*at life's origins*

**AUTHOR: ANOUSKA ALUNI**

If you've ever attended a biology lesson, you likely know something about the importance of proteins. They're essential for muscle and tissue growth, digestion, immune defence... and almost every process that keeps organisms alive. Proteins are fundamental for life.

In modern cells, proteins are synthesised in two tightly controlled steps: transcription and translation. During translation, an enzyme called RNA polymerase reads a specific region of DNA and produces a complementary RNA copy. This process occurs in the nucleus, where DNA is protected. Instead, the RNA copy carries the instructions needed to synthesise a protein and can leave the nucleus, acting as a molecular messenger. Hence, it is known as messenger RNA (mRNA).

The mRNA then enters the cytoplasm, where it binds to the ribosome, where translation takes place. The ribosome reads the sequence of nucleotides and converts the genetic information into a sequence of amino acids, forming a polypeptide chain. This process is highly efficient and accurate as it is driven by enzymes and carefully controlled molecular interactions.

A key role in translation is played by transfer RNA (tRNA). For tRNA to participate in protein synthesis, it must first be chemically bonded to an amino acid in an activated form, forming aminoacyl-tRNA, which delivers the specific amino acid to the corresponding sequence on the mRNA strand. As translation proceeds, the growing chain of amino acids is temporarily attached to another tRNA, peptidyl-tRNA, which holds the peptide chain together as new amino acids are added.

But this raises a deeper question: how did

protein synthesis first evolve? This process depends on the ability of amino acids to bind to RNA. Understanding how amino acids could have attached to RNA under prebiotic conditions, before enzymes existed, is essential to explaining how protein synthesis, and ultimately life, first evolved.

Confused? Don't worry, you're not alone – this question has also challenged scientists for decades.

Abiogenesis, or the origin of life theory, proposes that life arose from non-living matter. One key framework of this theory is the "RNA World Hypothesis". This suggests that RNA was the original self-replicating molecule, storing genetic information and catalysing chemical reactions (ribozymes). Later in time, DNA took over as the hereditary molecule. This hypothesis suggested that protein synthesis, controlled by complex machinery and interactions between proteins and RNA, could have evolved in the early world due to RNA's properties.

However, this hypothesis doesn't explain the formation of amino acid-RNA complexes, which allows translation to occur. In water, amino acids preferentially react with other amino acids rather than with RNA. While short peptides may form spontaneously, this does not explain the highly controlled, enzyme-reliant, RNA-directed process seen in modern cells.

An alternative proposal is the “Thioester World Hypothesis”, which suggests that sulphur-containing compounds called thioesters provided both the energy and chemical reactivity needed for early metabolism. Thioesters are still essential to modern biology as important intermediates in biochemical reactions and metabolism.

Recently, chemists at UCL have provided experimental evidence supporting both of these hypotheses. In a study led by Professor Matthew Powner, researchers demonstrated that thioesters could selectively attach amino acids to RNA under prebiotic conditions. Professor Powner explains: “Our study unites two prominent origin of life theories – the ‘RNA world’, where self-replicating RNA is proposed to be fundamental, and the ‘thioester world’, in which thioesters are seen as the energy source for the earliest forms of life.”

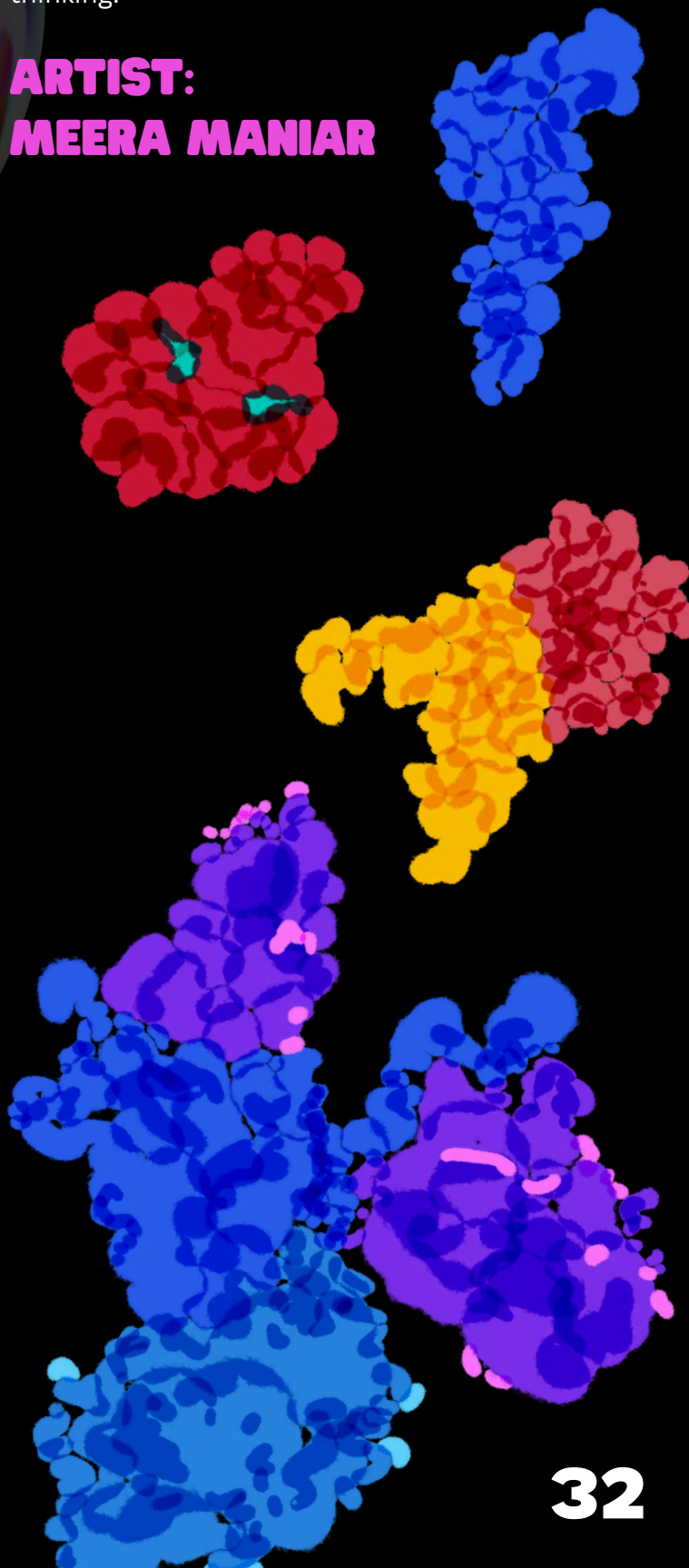
The research shows that two fundamental parts of protein synthesis – attaching amino acids to RNA and linking amino acids into a peptide chain – can be controlled chemically, in prebiotic Earth conditions, without enzymes. Thioesters allow amino acids to selectively bind to other amino acids. However, the presence of thioesters alone did not lead to peptidyl-RNA synthesis at all. A pH change and addition of hydrogen sulfide and ferrocyanide allows a chemical switch to occur, converting thioesters to thioacids, which prevents aminoacylation (a chemical process by which amino acids are attached to RNA), allowing for non-ribosomal peptide synthesis to occur. This suggests that early protein synthesis could have been chemically viable long before the evolution of enzymes or ribosomes.

Previously, Professor Powner’s lab also demonstrated that pantetheine, a sulphur-containing compound and a key fragment of Acetyl-CoA, can be synthesised from nitriles in water. Pantetheine is essential in completing the synthetic pathway from nitriles to aminoacyl-thiols. This allowed them to aminoacylate RNA and synthesise peptidyl-RNA in water.

By uniting the RNA World and Thioester World Hypotheses, the research conducted by Professor Powner’s lab offers a glimpse into the origins of life on Earth, highlighting the possibility of its gradual emergence through a few key chemical reactions.

As UCL celebrates its 200th anniversary, pioneering research like this highlights its enduring role as a home for disruptive thinking.

**ARTIST:  
MEERA MANIAR**



# BICENTENNIAL CELEBRATIONS, COLONIAL ENTANGLEMENTS



# UCL, ARMS AND PALESTINE



In 2026, University College London will celebrate its bicentenary with a year of events highlighting two centuries of “groundbreaking work and people.” Yet as UCL curates this narrative, it continues financial, research, and governance relationships with arms companies whose weapons are used in wars and military occupations — including the ongoing assault on Palestine. These ties link UCL’s research, investments, and prestige to a global military-industrial system that profits from civilian destruction and the collapse of healthcare. This contradiction matters not only for what UCL celebrates, but for what it enables.

## Palestine and the destruction of healthcare

The occupied Palestinian territory exposes the human cost of militarised “global impact.” Decades of occupation and blockade have fragmented healthcare, restricted movement, and created chronic shortages of medicines, electricity, and equipment. Since October 2023, organisations such as Médecins Sans Frontières have documented hundreds of attacks on hospitals and medical workers in Gaza and the West Bank — facilities bombed, ambulances fired upon, patients dying at checkpoints, staff detained or killed.

Healthcare is not collateral damage but a central target: disabling hospitals and emergency systems turns treatable injuries into death sentences. Meanwhile, these events are converted into datasets and case studies within global health research, often without addressing the political structures — occupation, siege, and impunity — that sustain such violence. Physical harm is mirrored by epistemic harm, as Palestinian suffering is analysed while its causes remain untouched.

## Extraction and responsibility in global medicine

This pattern reflects long-standing critiques of global health. Clinical trials and epidemiological research are often conducted in low- and middle-income countries under the banner of “partnership,” while risk is borne by populations with weaker health systems and fewer protections. Universities and corporations in the Global North retain the gains: publications, patents, and prestige.

Debates around HIV prevention trials in Africa exposed how weak ethical standards let sponsors exploit inequality. The question became whether participants gained lasting access to proven interventions or were left with nothing once researchers departed. Gaza, though not a conventional “research site,” shows a similar logic: while its healthcare is systematically destroyed, international bodies extract data, expertise, and moral authority from the crisis. When universities embedded in arms-linked industries celebrate their “impact” in global medicine, solidarity becomes extraction.

## UCL’s arms industry ties

UCL’s connections to arms manufacturers are well documented. In 2019, Pi Media revealed that its Centre for Ethics & Law received around £10,000 a year from BAE Systems, one of the world’s largest weapons producers. A Freedom of Information request showed that between 2010 and 2015 UCL received £3.6 million in grants from arms companies including BAE Systems, Lockheed Martin, and QinetiQ.

These relationships did not end cleanly.

openDemocracy later reported that BAE donated almost £50,000 to the same centre between 2017 and 2021, while a company representative continued to sit on its advisory board – a position that influences research agendas and institutional priorities. Student journalists have identified similar links across UCL: staff from BAE Systems and Leonardo help steer engineering curricula and research, embedding the military-industrial complex within the university's governance and career networks. This is the academic side of the military-industrial complex: a revolving door between university labs, corporate weapons manufacturers, and state militaries.

### Investments and indirect profit

UCL's entanglements extend beyond research. Cheesegrater Magazine reported that the university invests around £1.5 million in HSBC, a bank criticised for holding shares in arms manufacturers including BAE Systems and Raytheon. Weapons from these firms have been used by Israeli forces in Gaza and the Saudi-led coalition in Yemen. As HSBC profits from its arms holdings, UCL profits indirectly, despite its "ethical" investment policy. Such policies risk becoming branding exercises rather than mechanisms of accountability.

### Research for war

UCL also conducts research for military purposes. An Engineering and Physical Sciences Research Council-funded project, "Signal Sensing, Design and Delivery for Electronic Warfare," led by UCL Engineering with Thales and the US Army Research Laboratory, focuses on sensing and signal-processing in contested electromagnetic environments – core capabilities for modern surveillance, drones, and combat systems. Thales, a major defence contractor, markets these technologies to NATO militaries. Even when framed as "dual-use," integration into weaponry is structural, not accidental.

### Contesting UCL's future

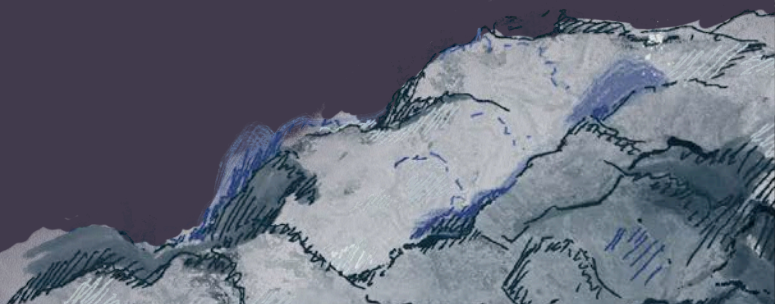
Students and staff have repeatedly challenged these contradictions. A Students' Union policy calls for an end to relationships with arms companies and for transparency across funding, investments, and partnerships. Trade unions and activist groups at UCL have likewise demanded divestment from firms implicated in human rights abuses and alignment with Palestinian civil society's calls for boycott, divestment, and sanctions.

In this context, UCL's bicentenary cannot be reduced to light shows and glossy narratives of global impact. Celebration without accountability risks denial. A just bicentenary requires concrete commitments: divestment from arms manufacturers and their financiers; an end to partnerships advancing weapons systems; material support for Palestinian healthcare; and teaching that confronts the university's role in militarised knowledge production.

UCL's legacy should not rest on the profits and prestige of war but on solidarity with those whose lives and hospitals are treated as expendable – nowhere more visibly than in Palestine.

**AUTHOR: MARIJA JOVCHESKI**

**ARTIST: MEERA MANIAR**



# Neuroscience Nobels at UCL: A History



*Sophie Rogers*

Over the last 200 years, UCL has been home to an incredible five Nobel Prize winners for neuroscience-related discoveries. Here's a closer look at some of their historic work.

## **Dale & Loewi (1936)**

Almost a century ago nerve impulses were thought to be purely electrical, a theory propagated (no pun intended) by Sir John Eccles. Electrical impulses called 'action potentials' do transmit along neurons. However, thanks to the work of Sir Henry Dale and Professor Otto Loewi, we now know that these impulses also cause the release of chemicals called 'neurotransmitters'. Neurotransmitters cross the gap between neurons to generate another action potential on the other side.

This is fundamental to modern neuroscience. However, it took decades of research to be accepted - and some fantastically macabre dream-inspired experiments on frog hearts. Yes, really. Professor Loewi showed that electrical stimulation of the vagus nerve released a mystery substance from the heart, which he named 'vagusstoff'. Transfer of this fluid to a different frog heart slowed its spontaneous beating. 'Vagusstoff' was later identified as acetylcholine, a neurotransmitter which can slow heart rate and lower blood pressure.

Meanwhile, in his research across UCL and Cambridge, Sir Henry Dale succeeded in isolating acetylcholine from a fungus called 'ergot of rye'. Through collaboration the two confirmed that acetylcholine acts on the human nervous system and is

produced by our own bodies too.

These findings were controversial. It took decades to resolve the 'soup versus spark' debates (chemical versus electrical transmission) into our hybrid model. However, Dale & Loewi were ultimately awarded the Nobel Prize for revealing the chemical side of neural communication.

## **Huxley (1963)**

The question remained: how do our neurons actually produce electrical signals?

Today, studying neurons is meticulous and painstaking work. In the 1960s studying neuronal impulses in humans or most animals would have been almost impossible. To get around this, Professor Andrew Huxley and colleagues turned to the giant neurons of the longfin inshore squid. These giant neurons can reach up to 1.5mm in diameter: roughly 100 times larger than our own.

Using a 'voltage clamp', the scientists were able to record action potentials inside the giant neurons. Imagine a neuron like a leaky hose containing water. A voltage clamp involves a 'feedback circuit' which holds the neuron at a set water pressure (voltage) to measure any changes in the speed of water moving out of holes in the neuronal membrane (current).

Despite the interruption of their research due to the Second World War, Huxley and colleagues discovered that action

potentials result from charged particles called ions moving in and out of the neuron. In fact, the very same ions that make up table salt – sodium and potassium – drive the electrical language of the brain.

### **Katz & von Euler (1970)**

Sir Bernard Katz and Ulf von Euler were awarded the 1970 Nobel Prize alongside Julius Axelrod for discovering how neurons release neurotransmitters.

Katz worked on the frog neuromuscular junction, the region where a neuron meets muscle. Through painstaking measurements of neurotransmitters released by the neuron, he found that acetylcholine is released in specific or 'quantal' amounts. Rather than a continuous stream of individual molecules being released, this suggests that neurotransmitters are released from neurons in discrete 'packets'.

Von Euler discovered the identity of these 'packets'. Working on noradrenaline, he observed compartments called vesicles which look like tiny bubbles inside neuronal endings. A single vesicle can fuse with the membrane of the neuron to release its neurotransmitter cargo.

### **O'Keefe (2014)**

Professor John O'Keefe has worked at UCL since 1967, and was awarded the 2014 Nobel Prize for his discovery of 'place cells' in the brain.

By recording electrical signals from individual neurons in the rat hippocampus, O'Keefe and colleagues discovered that specific neurons fired only when the rat was in a specific location in its cage. Different neurons fire at different locations,

effectively forming a detailed internal map of the rat's environment. O'Keefe termed this 'cognitive spatial mapping': an incredible example of how the brain makes sense of our world.

This work laid the foundation for subsequent discoveries including 'boundary vector cells', which help to anchor place cells by tracking the location of spatial boundaries like walls or doors.

O'Keefe continues to work at UCL, with some of his recent publications exploring the effects of dementia on place cells.

### **Hinton (2024)**

Two years ago Professor Geoffrey Hinton was awarded the 2024 Nobel Prize in Physics alongside Professor Hopfield. As the founder of the Gatsby Computational Neuroscience Unit situated at UCL, Hinton is known as the 'Godfather of AI'.

Inspired by real brain structures, his research combined neuroscience and machine learning to build artificial neuronal networks. This led to the creation of the 'Boltzmann machine', a system that can learn to find patterns in data autonomously. Hinton's work underpins much of the 'deep learning' that AI models like ChatGPT perform, with wide-ranging effects in modern society.

### **Looking to the future**

UCL's neuroscience legacy is evident across campus from the Andrew Huxley building beside the Student Centre to the Bernard Katz building on Malet Place. Inside these buildings pioneering research continues to drive discoveries that reshape our understanding of the brain – and the future of medicine and technology.

Written by Elizabeth Jovena Sulistyo

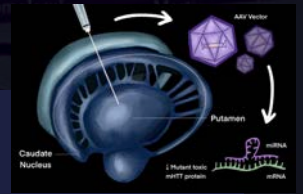
Art by Patrick Marendia

On 10th October 2020, a paper made headlines with the revelation that a species of the tardigrade genus *Paramacrobiotus* can survive harmful radiation by clonally. This discovery

Despite pervading the animal kingdom – scorpions, parrots, chameleons and frogs can autofluorescence – its functional significance is unknown. Photomicrographs

osmohalosis (excessive salinity). Remarkably, they are the first animals found to be able to survive exposure to the vacuum and radiation of outer space. The tardigrade curls up, reducing its surface area for evaporation, with lost water replaced by leiposubstrates such as trehalose that protect cellular macromolecules and internal organs.

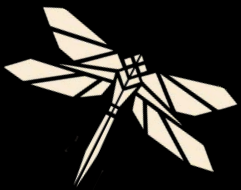
radiation tolerance open for human survival, especially programmes, such as the Life Project, have already studied how they react in various conditions. Possibilities include studying



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