MRC Max Perutz Award 2025

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THE SHORTLIST

MRC MAX PERUTZ AWARD 2025 - WRITING

WINNER

Vanessa Drevenakova

RUNNER-UP

Chloe Linham

SHORTLISTED

Anita Valand

Isabel Shand

Patrick Howlett

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Johnny Tam

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Annalise Whines

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Eddie Bullock

Grace Kavanagh

Hou Wang Lam

The sound of youth: can ultrasound delay brain ageing?

If I told you that a gentle pulse of sound could help keep your brain young, would you believe me? Not music. Not meditation. But carefully controlled sound waves, delivered deep into the brain. No surgery. No drugs. Just vibrations - measured, focused, and silent to the ear.

I know, it might sound far-fetched. But the idea that we could delay brain ageing itself with ultrasound might no longer be science fiction. In fact, it's exactly what I am researching.

We're living longer than ever before, but not necessarily better. Dementia is now one of the leading causes of death in the UK. Families are caring for loved ones who slowly fade in front of their eyes. And our treatments? They come late and do little to reverse the damage. That's why we need new approaches. Not just to manage decline, but to prevent it. Not to treat disease after it steals someone's memories, but to protect the brain while there's still time.

I study what happens to the brain as we grow older, and how we might stop some of those changes using ultrasound. Not to live forever. Not to become superhuman. Just to stay ourselves for a little longer. Because ageing doesn't just change our faces and joints. It changes the brain. Quietly at first, misplacing a word, forgetting a detail. Then more deeply. A friend's name disappears. A routine becomes confusing. And for some, even familiar faces begin to feel distant.

For a long time, we thought brain ageing was just slow decay - neurons wearing out, fading away. But now we know the story is more complex. The brain isn't just a tangle of neurons. It's more like a garden, constantly maintained by a team of microscopic caretakers, working tirelessly behind the scenes. These caretakers are microglia, the brain's immune cells. They prune unnecessary connections, clear debris, and quietly patrol for anything out of place. One of their most important jobs is to clear out proteins that naturally build up between cells. In a healthy brain, these are swept away like fallen leaves. But when too many are left to gather, they can form the plaques and tangles seen in diseases like Alzheimer's.

WINNER
for writing
Vanessa Drevenakova
Imperial College London

With age, some microglia begin to falter. They don't disappear, but they stop doing their job well. Scientists call these dysfunctional cells 'senescent'. Instead of clearing out waste, they start adding to it. They release harmful signals, trigger inflammation, and confuse the surrounding cells. This damages the connections between neurons, interfering with the brain's ability to form and retrieve memories. Over time, these changes contribute to declines in learning, memory, and overall cognitive function.

This is where ultrasound comes in. We've all heard of ultrasound. It's what expectant parents see in their first blurry photo of a baby. But the ultrasound I use is a little bit different. It's focused. And focused ultrasound allows us to target precise areas deep inside the brain, using sound waves that converge like sunlight through a magnifying glass. What's even better is that it's safe and non-invasive. You don't hear it. You don't feel it. But the brain does.

Ultrasound is made up of tiny vibrations, waves of pressure moving through the body. Brain cells like microglia are coated in tiny molecular sensors, like trapdoors that respond to these pressure waves. When ultrasound reaches them, it nudges these doors open, allowing a rush of calcium inside. That calcium acts like a spark, igniting a chain reaction inside the cells - nudging them out of their idle daze, and reminding them of their original purpose. Of course, inside the cell, this chain reaction is far more complex, unfolding through a cascade of molecules, genes, and proteins that work together to regulate how the cell behaves.

Sound, like anything powerful, must be tuned carefully. Too little, and nothing changes. Too much, and the delicate cells of the brain may become overstimulated. That's what makes it exciting - we can adjust the ultrasound signal to shape the cellular response.

I study how this process works in aged brain cells, especially microglia. My research asks whether we can encourage these senescent microglia to behave more like their younger selves: active, protective, and calm. I watch how they move under the microscope, track the signals they send, and test the chemical messages they release to see if their behaviour is changing.

Can a few pulses of ultrasound reduce inflammation in the brain? Can we help microglia support brain function and slow down memory loss? And can we do all of this without drugs, just using ultrasound?

Early findings suggest we can. After a single session of ultrasound, microglial behaviour shifts to be more attentive and responsive - their inflammatory signals quiet down, their clean-up systems switch on and their shape changes. But it's not about turning back time. It's about restoring balance. If we can learn how to gently nudge the brain back on track, before too much damage sets in, we could delay or reduce the impact of age-related diseases like Alzheimer's.

Right now, my research is in the pre-clinical stage, working with animal models, testing how microglia respond, refining how ultrasound could one day be used to keep the brain in balance. And the potential is enormous. In just the past few years, focused ultrasound has moved from niche technology to clinical reality. In fact, it is already being tested in patients with Alzheimer's and Parkinson's. In our lab, we're exploring whether it could take things even further - whether ultrasound might help us intervene earlier, by targeting the root causes of brain ageing before symptoms ever appear.

There is so much to explore. How long do the effects last? Can we reach the right cells without affecting the wrong ones? Are aged microglia as easy to shift as younger ones? These are the questions I ask every day. Not just out of scientific curiosity, but because behind every result is someone's story: a daughter trying to reconnect with her mum, a carer stretched to the edge, a family hoping for more time. And in some way, I carry those hopes with me too. They remind me every day why I love this research.

My PhD is part of a wider shift in how we think about brain health - not just in terms of treatment, but of timing. About how we care for the brain before decline sets in. About keeping the garden tended, the balance steady, the spark alive.

Ageing begins in silence. Perhaps healing can, too.

Urinary tract infections: relieving the pain, fighting the resistance

RUNNER-UP
for writing
Chloe Linham
University of East Anglia

How many times a day do you go for a wee? Probably more than you realise. Now imagine that every single time, it feels like burning hot lava. And the moment you're done, you feel like you need to go again. But there's nothing there, just pain. You're trapped in a relentless cycle of discomfort, exhaustion, and frustration.

This is what it feels like to have a urinary tract infection (UTI). It's not just a minor inconvenience; it can dominate your life. It follows you to work, to the bathroom, to bed, and to every moment you wish you could just get on with your day. UTIs are incredibly common: half of all women will experience one at some point in their lives. For some, it's a single unpleasant episode. For others, it becomes chronic and recurring, an ongoing condition that chips away at their quality of life.

But despite how widespread they are, we don't talk about UTIs much. They're private. Embarrassing. Dismissed as "just one of those things". But they're also real and relentless for those who suffer from them. These people cancel plans, avoid intimacy, and go to bed crying because the burning won't stop. UTIs affect more than your bladder. They affect your confidence, your freedom, and your sense of control. You start planning your life around bathrooms and avoiding things you love.

And the situation is even worse for people with urinary catheters, like those living with disabilities, recovering from surgery, or residents in care homes. For them, catheter-associated UTIs (CAUTIs) are painfully common, and they often lead to longer hospital stays, complications, and in some cases, life-threatening consequences.

CAUTIs are often much harder to treat. Why? Because the bacteria that cause them form biofilms on the catheter surface, sticky shields that make them incredibly resistant to treatment. Think of it as bacteria bunkering down behind a defensive wall, waiting out the antibiotics that are supposed to kill them. And to make matters worse, the antibiotics we usually use are delivered through the bloodstream. They trickle through your body and are slowly drip fed into the bladder. This makes it difficult for them to reach a high enough concentration to tackle those resilient, biofilm-protected bacteria. It's like throwing a cup of water on a house fire.

When the infection isn't fully cleared, it returns. The cycle continues, and with each new round of treatment, bacteria learn. They adapt. They become more resilient. That's the process we call antimicrobial resistance (AMR), and it's one of the biggest health threats we face today. We're watching antibiotics slowly lose their power. Infections that once took a few tablets to cure are now sending people to hospital. In some cases, they're becoming untreatable altogether. It means surgeries becoming riskier, hospital stays becoming longer, and conditions we once cured becoming fatal again.

My research aims to break this cycle by rethinking how we deliver antibiotics. Instead of relying on systemic delivery, I'm developing a treatment to send the antibiotic directly to the bladder, fighting the infection at the source. I use a technique called electrospinning, where a high-voltage current pulls polymer solutions into tiny threads that solidify as they fly through the air to produce dry, dissolvable fibres that look like a thin sheet of paper. But hidden inside is something powerful: gentamicin, a strong antibiotic. When mixed with saline, this paper-like sheet transforms into a simple, fast treatment that can be delivered through a specially designed catheter. Once inside the bladder, it releases the drug exactly where it's needed without going through the bloodstream at all.

This approach offers several key advantages. First, it targets the infection directly. By delivering the antibiotic to the bladder we can achieve much higher concentrations than would ever be possible through the bloodstream, without exposing the rest of the body to harmful side effects. For patients, this means faster relief and more effective treatment. It's also safer. Gentamicin, while powerful, can be toxic to the kidneys and ears when it circulates systemically. Local delivery keeps the drug where it's needed and reduces the risk to other organs.

Beyond effectiveness and safety, the treatment is designed with sustainability in mind. Because the fibres are dry, they don't require refrigeration, making them easier to store, transport, and distribute, even in care homes or for at-home use. This also means reduced packaging, lower energy demands, less waste, and lower costs for healthcare systems. Most importantly, it empowers patients. Managing a chronic infection like this often means frequent hospital visits, which can be disruptive and distressing. This system opens the door to self-administered, daily intravesical treatment under clinical guidance, offering not just better infection control, but a better quality of life.

In the lab, we've shown that these fibres kill E. coli, the most common cause of UTIs, and remain effective over 72 hours, suggesting they could be used for once-daily, targeted treatment. I'm still in the early days of my PhD, but as I continue my research, hopefully we will see even more potential in this new treatment method. It's a small fibre with big potential: to help patients recover faster, reduce antibiotic use, and relieve pressure on overloaded hospitals. By rethinking how we treat infection at its source, we're opening the door to better care, fewer side effects, and a real step forward in the fight against AMR.

And next time you go to the toilet, without pain, without fear, spare a thought for those who can't. And know that behind the lab bench, someone is working on a solution. One tiny fibre at a time!

Rewriting the breath script

SHORTLISTED
Anita Valand
University of Nottingham

We breathe without thinking. It's one of the most automatic and essential things we do. But for people with Idiopathic Pulmonary Fibrosis (IPF), every breath becomes a conscious effort, a painful struggle against lungs that are slowly turning into thick scarred tissue.

IPF is a devastating lung disease that affects around 3 million people worldwide. It has no known cause, no cure, and only limited treatment options which help by slowing down the progression of the disease. Life expectancy after diagnosis is usually just three to five years. Despite being as deadly as some cancers, it remains under-recognised and poorly understood.

But what if the key to understanding and treating IPF lies not just in our DNA, but in how our DNA is controlled?

That's the question at the heart of my research.

Unravelling the mystery of IPF

IPF causes progressive scarring of the lungs, making it harder and harder to breathe. Over time, the lung tissue becomes stiff and thick, preventing oxygen from moving into the bloodstream. The condition tends to strike people in their 50s or 60s, and its progression is often unpredictable and cruel.

One of the hallmarks of IPF is the behaviour of certain cells in the lung called fibroblasts. In a healthy person, fibroblasts are part of the repair team which help the body heal when there's damage. But in people with IPF, these do not function properly. These fibroblasts become overactive and instead of healing, they keep laying down scar tissue, even when it's no longer needed. The result of this is irreversible lung damage.

What causes this shift in behaviour? We know that the DNA or genetics that we're born with plays a role. But increasingly, scientists are looking beyond our genes to understand how they are controlled and expressed. This is where epigenetics comes in.

What is epigenetics?

Every cell in your body contains the same DNA, like an instruction manual. But different cells use different parts of that manual. A skin cell doesn't need to behave like a brain cell, and that's thanks to epigenetic controls.

Epigenetics refers to changes that affect how genes are switched on or off without changing the DNA sequence itself. One of the most studied types of epigenetic change is DNA methylation. This involves adding small chemical tags (called methyl groups) to DNA. These tags act like dimmer switches, dialling and controlling gene activity up or down.

One key factor is that unlike DNA mutations, epigenetic changes are reversible. That means if we can understand which epigenetic changes are driving diseases like IPF, we might be able to "reset" them and stop the disease in its tracks.

Zooming in on lung cells

My PhD project investigates the role of cell-type specific DNA methylation in IPF. In simple terms, I'm studying how epigenetic changes differ between types of lung fibroblasts in people with and without the disease.

Why does this matter? Well, not all fibroblasts are the same. They vary depending on where they are in the lung and what function they serve. Some might contribute to scarring more than others. By comparing fibroblasts from healthy donors and IPF patients, specifically from both the airways and the deeper parts of the lungs, I aim to uncover which cell types and epigenetic changes are driving the disease.

To do this, I use advanced laboratory techniques that allow me to look at DNA methylation across the entire set of DNA. Essentially scanning millions of sites where chemical tags might be influencing gene behaviour. I also study gene expression, which tells me which genes are active or inactive in these cells.

By combining this information, I can build a picture of how epigenetic changes affect gene activity in different fibroblast types. The goal is to identify specific chemical tags or unique patterns that distinguish healthy from diseased cells.

Why my research matters

In the short term, my research helps us better understand what's going wrong in IPF at the cellular level. That alone is valuable because without understanding a disease, we can't treat it effectively.

But the long-term possibilities are even more exciting

If we can identify specific epigenetic patterns associated with harmful fibroblast behaviour, we could develop biomarkers that help diagnose IPF earlier, when treatment might be more effective. We might also be able to group patients by separating them based on their molecular profile to offer more personalised treatment.

This is especially important in IPF, where the disease progresses differently between patients, and some patients respond to certain drugs and others don't.

Eventually, we may be able to target epigenetic changes directly, using drugs that reset the gene expression patterns in fibroblasts, halting or even reversing the disease process.

Because epigenetic changes are reversible, this approach offers real hope.

The bigger picture

IPF doesn't just impact patients, it affects families, healthcare systems, and economies. Patients often require oxygen support, hospital stays, and long-term care. The disease also places a significant emotional burden on those living with it.

Better treatments could dramatically improve quality of life. Earlier diagnosis could extend survival, and targeted therapies could reduce healthcare costs by avoiding a "one-size-fits-all" approach.

But beyond the statistics and science, my motivation is deeply personal

I've always been fascinated by the human body, how it functions, how it fails, and how we might help it heal. My love for medicine and research has only grown as I've spent time in the lab, growing cells, analysing data, discovering patterns, and realising just how much is still unknown.

Behind every sample I study is a real person, someone who struggles to breathe, who wonders if they'll still be here in a year. Science, to me, isn't just about knowledge. It's about hope. Hope that one day, people with IPF won't be told there is nothing more that can be done. Hope that with every discovery, we're one step closer to giving them their breath and their future back.

Breathing should be effortless. By rewriting the biological script behind this disease, we may one day rewrite the stories of those living with it.

Protrudin: training neurons to get traffic moving again

SHORTLISTED

Isabel Shand

King's College London

You're in your 20s, feeling unstoppable, riding your motorcycle, feeling the wind whip past your face, or singing in the car with your friends. Life feels like freedom. But in one split second, it all changes. An accident. A crash. A wrong angle. Suddenly you're in a hospital bed, being told you may never move or feel, your arms or legs again. This is a spinal cord injury (SCI). And it can happen to anyone, at any time.

In the UK, a spinal cord injury leading to paralysis occurs every 2 hours. Beyond paralysis, SCI can take away bladder and bowel control, the ability to breathe unassisted, and even the simple sensation of touch, things we never think twice about. It irreversibly changes people's lives, ability to work and can be emotionally devasting to sufferers. Life completely changes after an SCI.

Today, recovery from SCI often ends in hope. Therapy might help a little, and emerging technologies might make daily life easier. But why can't the spinal cord just heal itself? What if the body could repair itself? Those are the questions at the heart of my PhD research.

Think of the spinal cord as your body's central superhighway. Every signal your brain sends, whether to make your fingers type, your lungs breathe, or your feet walk, must travel down this pathway made up of cells called neurons. These neurons are long, branch-like structures, and they pass signals along electrical "roads" called axons, which can stretch over long distances through the body. When the spinal cord is damaged, it is as if a section of the superhighway has collapsed. The roads are ripped apart. Wreckage piles up. Messages can't get through. Muscles go silent. Sensation disappears.

To restore function, two things are needed:

1. Clear the wreckage: get rid of the debris and scar tissue.

2. Rebuild the road: help those broken axons regrow and reconnect.

The tragedy is spinal cord neurons are terrible at both: they are among the worst regenerators in the body. Unlike skin, blood, or even other types of neurons, they simply don't know how to heal after injury. Scientists think it's because these neurons are missing something crucial: some internal tools or

instructions that other cells have. This is why many SCIs result in permanent paralysis, and this is the issue we are trying to overcome.

One reason spinal cord neurons don't recover well is that they are terrible at moving important materials to the places they are needed: to the injured axons. Imagine a damaged road. Construction workers are sent to fix it, but their tools are stuck in a traffic jam miles away. If the necessary tools don't get to where they are needed, the road doesn't get repaired. That's what happens inside spinal cord neurons. Since the axons extend over a large distance, the building blocks needed for repair (like energy, proteins, and raw materials) can be located far away from the injury. And neurons in the spinal cord can't get those supplies to where they're needed. So, we need someone to direct traffic in order to deliver the materials.

Enter Protrudin, a molecule that acts like a traffic warden. It organises the cell's internal roads, so materials are actually delivered to the injury site. When added to ordinary, non-neuronal cells, Protrudin caused something strange to happen: the cells grow long, branch-like extensions, or protrusions, that look like axons. This was a clue to how we could improve the growth of axons in cells that should have them, such as spinal cord neurons. Next, researchers tested Protrudin in neurons. After injuring neurons that received Protrudin, the axons started robustly growing back. Damaged neurons could regrow their lost connections. It turns out that the neurons that naturally regenerate well already have lots of Protrudin. By comparison, the neurons in the spinal cord have very little Protrudin. So, all we need to do is put more Protrudin into the neurons, sounds easy right? This is where things get tricky. We can't just inject the molecule; it disappears too quickly to have any real impact. Especially since axon repair over a long distance can take a long time. Instead, it was necessary to teach the neurons to make Protrudin themselves. That means getting a genetic "instruction manual" into the cell using a method called gene therapy. Once the cells have the manual, they can make plenty of Protrudin themselves and start their regeneration journey.

Perhaps surprisingly, we use viruses to do this. Viruses are nature's experts at sneaking into cells and inserting "code". Although we usually associate this viral code with being detrimental to our cells and our health, we can change that. By removing their harmful sections of code, we can re-engineer them into harmless delivery vehicles. These modified viruses carry the genetic instructions to make more Protrudin and the delivery system to get it into the neuron. Once inside the neuron, the cell reads the instructions and starts making the protein on its own. It's like giving the workers their own handbook and equipment, so they can keep repairing the road, even without supervision.

My research focusses on making this system as effective and safe as possible. I study how Protrudin moves within neurons, what exactly it's doing and what other molecules it's working with. I strive to find out what the building blocks are that Protrudin is delivering to the injury site. Can we improve regeneration by increasing the supply of these building blocks to the cells along with Protrudin? In theory we can, but I'm looking to put this theory into practice. Additionally, I'm working to understand and fine-tune this therapy so it can one day move from lab to clinic and help those that suffered a SCI. Of course, Protrudin-based gene therapy is not a cure yet. It's still early days. But we believe the potential is enormous. Every small improvement in axon regeneration, every neuron that reconnects, brings us closer to restoring movement, sensation, and independence for people suffering with SCI.

The road to recovery after spinal cord injury is long and difficult. But for the first time in a long time, it's beginning to look like there is more than just hope that the roads can be rebuilt. We may not be able to prevent every crash, but with tools like Protrudin, we are learning how to repair the road and restart the traffic. Hopefully, we can all look forward to the journey ahead.

Silicosis and tuberculosis: half the world away

SHORTLISTED

Patrick Howlett

MRC Centre for Environment and Health

Imperial College London

You might be reading this in the comfort of your kitchen, the broadsheet newspaper laid on the sleek, new artificial stone worktop. Anchored by a memory, you roll one of your rings or, perhaps, a necklace. More likely – statistically at least – you are consuming this digitally, scrolling the crisply illuminated story on your phone or tablet. How did you get down this internet rabbit hole, anyway?

I'll get to the point. The thread running through this opening – the artificial stone worktop, the jewellery, the precious trace metals in your phone – is that the production of each item involves exposing workers to silica dust. Silica is the most widespread mineral on earth. Cutting, grinding or crushing it releases tiny, invisible silica dust particles. Breathing these in can cause silicosis, a disease that results in an irreversible hardening of the lungs. Our research has shown that the more silica you breathe in, the higher your risk of silicosis.

In some cases, silicosis develops decades after starting work. But, if large quantities of silica dust are breathed in, a more disabling type of silicosis may develop within a few years – or even months – of starting work. Both silica dust and silicosis markedly increase the risk of a lung infection called tuberculosis. In 2023, tuberculosis caused 1.25 million deaths worldwide, more than any other infectious disease. There is no known treatment for silicosis, but tuberculosis is curable with antibiotics.

My PhD research aims to understand the impacts of silicosis and tuberculosis among a group of small-scale miners in Tanzania. Half the world away, I work in a lung clinic at the Royal Brompton Hospital in London, where, over the last two years, we have seen increasing numbers of workers with silicosis. Most cut or grind artificial stone. Across both settings, I am fascinated by the similarities we see and the challenges the workers face.

Small-scale mining is fully integrated in the global economy, producing one-fifth of the world's gold and a quarter of the tin and tantalum. The estimated

50 million small-scale miners worldwide are often self-employed in locally run companies based low- or middle-income countries. The number of miners has quadrupled in the past decade, driven by growing demand for minerals critical to electronics and the green energy transition, and supportive national and international policies.

Artificial stone, sometimes called “Quartz”, is made by glueing together crushed stone. The resulting material is cheaper and more easily shaped than natural stone. It is a big business globally; in 2023, the US market alone was worth $23 billion.

Artificial stone workers and small-scale miners breathe in very high levels of silica dust, frequently several hundred times above recommended levels. As a result, many young workers are getting sick and, in some cases, dying. We found that, globally, an average of 1 in 4 small-scale miners had silicosis and 1 in 28 had tuberculosis. In our study in Tanzania, 15% of small-scale miners had died six months after starting tuberculosis treatment. In our UK clinic, in keeping with reports from colleagues worldwide, increasing numbers of artificial stone workers with silicosis are dying and needing lung transplants.

An important aspect of our research is to tell the story of workers whose lives are often hidden. Small-scale miners tend to come from lower socioeconomic groups and have had limited educational opportunities. Their working arrangements confer few safeguards. Many small-scale miners are women. In our study, mine tailings were largely panned by women among whom silicosis was common. In the UK, many artificial stone workers are recent migrants and are thinly spread between small companies nestled in expanses of warehouses.

In Tanzania, I met Robert (*not his real name). Aged 47, he had been a miner for 23 years. Since being diagnosed with silicosis and treated for tuberculosis a year ago, he was too unwell to work. Because he could no longer support them, his wife returned to her family with their daughter. Alone, he sat hunched on a small stool outside his single room mud house. Breathing was difficult and our conversation interspersed with bouts of coughing. Although TB treatment was free, to pay for hospital visits, medications, and other daily costs, he spent all £300 of his savings. He had loaned £300 from neighbours and had sold his livestock, bed and phone, making another £450.

In our UK clinic, I spoke with Qsay (his real name, as he wants his story to be made public). Aged 31, Qsay was diagnosed with silicosis a year ago, after 4 years cutting and polishing artificial stone. Now too ill to work he is being evaluated for a lung transplant. When his breathing is bad, his wife helps him wash. His son, aged 7 and the oldest of three children, often asks “Why don’t you go to work?”. Having spent all his savings, he had loaned £7800 from friends and family to cover his family’s costs of living. He sold their kitchen table, chairs and a mirror to make another £700.

As with Robert, Qsay’s friends were afraid to visit him because they thought he had tuberculosis. Qsay explained that his friends didn’t answer his calls as they thought he would ask for money. Robert and Qsay may be half the world away from each other, but their experiences were remarkably similar. The combined effect of unemployment and illness had led to a loss of identity, income and social connections. Their situations demonstrated why it had been so important for us to study the wider impacts of work-related illness. In our study, 20% of miners with silicosis or tuberculosis had a child who stopped going to school because there wasn’t enough money.

In this situation, what does hope look like? Studies by our US and Tanzania partners have shown that simple, cost-effective measures, such as spraying water, can cut dust levels in mines by 80-99%. We hope to roll these out more widely to prevent silicosis and tuberculosis. Chest x-rays interpreted by artificial intelligence are currently used to diagnose tuberculosis. I am confident the same technology can make diagnosing silicosis cheaper and more accurate. Finally, we must look for better treatments. Our partners at Kibong’oto Infectious Diseases Hospital in Tanzania have shown that lung rehabilitation exercises help miners treated for tuberculosis recover their strength. For Robert these sessions are a lifeline.

Much like the miners in George Orwell’s The Road to Wigan Pier, I am struck by the incredible pride and identity these workers share doing a job that furnishes the world around us. In return, they ask for little. I think they deserve more.

WINNER
for video
Johnny Tam

University of Edinburgh

RUNNER-UP
for video
Annalise Whines

MRC Cognition and Brain Sciences Unit
University of Cambridge

PLAY

Digital speech biomarkers for motor neuron disease

PLAY

Does motion change across the menstrual cycle?

SHORTLISTED

Eddie Bullock

University of Cambridge

PLAY

Supporting autistic people with social interaction through AI

SHORTLISTED

Grace Kavanagh

University of Nottingham

PLAY

A quest to fight antimicrobial resistance

SHORTLISTED

Hou Wang Lam

University College London

PLAY

Cracking the code of treatment-resistant colorectal cancer

THE JUDGING PANEL

Elspeth Oakley
Public Member

Jess Winch
Managing Editor,
The Observer

Dr Roger Highfield
Science Director of the
Science Museum Group

Andy Ridgeway
Associate Head of School of
Applied Sciences, UWE Bristol

Hafsah Iqbal
Public Member
(not pictured)

Dr Joanna Robinson
Director of Research Talent,
Skills and Careers at MRC and
Chair of the Judging Panel

Medical Research Council

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In partnership with The Observer

The MRC Max Perutz Award is our science communication competition for current MRC PhD students. To enter, students need to tell a non-scientific audience why their area of research matters. The award aims to encourage and recognise outstanding science communication and help MRC PhD students build their science communication skills.

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