

# MAX PERUTZ

## Science Writing Award



Medical  
Research  
Council

In partnership with **The Observer**

## THE SHORTLIST

### 2022

**25**  
YEARS

[#MaxP22](#)

## 25 YEARS OF IMPACT

This year marks the 25th anniversary of the Medical Research Council (MRC) [Max Perutz Science Writing Award](#). In May, PhD students were invited to enter by writing a compelling piece about their research for the non-scientific reader.

At an inspiring ceremony at the Royal College of Physicians on 20 October, the £1,500 prize was presented to the winner, Emily Cornish, a MRC Clinical Research Training Fellowship at University College London. She wrote eloquently about her PhD research into a rare placenta disorder that can tragically cause pregnancy loss.

Her winning entry brought one of the judges to tears. According to the judging panel (see page 24), "It is powerful writing from a scientist with a genuine empathy for their patients".

This year's shortlist of 10 articles covered a diverse range of research topics including a rare form of dementia, immune therapies for blood cancer, treating a neglected tropical disease in children and how the microbiome develops in babies.

Over the award's 25 years, previous winners and finalists have gone on to use their science communication skills to further their careers and their research, going on to give TED talks, become BBC broadcasters and win national science writing awards.

At the ceremony, guest speaker Professor Andrew Bastawrous told a powerful story of how entering and winning the award in 2012 gave him the opportunity to explain his work in an engaging way. This helped him bring his vision to life, and secure support for launching a social enterprise called [Peek Vision](#) that brings better vision and eye health for the millions of people worldwide who need it in low- and middle-income countries.

25  
YEARS

WINNER



**EMILY CORNISH**

RUNNER-UP



**ANASTASIA THEODOSIOU**

COMMENDED



**REBECCA WILLIAMS**

SHORTLISTED



**ANDREW EDIELU**



**ALICE EVANS**

SHORTLISTED



**KRISTINA HNÍZDILOVÁ**



**EMILY HYDE**



**DOLLY THEIS**



**EMILY WOODCOCK**



**NATALIA ZDOROVTSOVA**

## Hope after Grace: tackling recurrent pregnancy loss

**Amy's third baby was born in the middle of the night. In the split second before he cried, everyone in the operating theatre held their breath. I was lurking in a corner holding a huge bucket of ice, waiting to collect the placenta. The stakes felt incredibly high. Baby William's two sisters were stillborn due to a placental disorder called chronic histiocytic intervillitis, known as CHI.**

Most obstetricians have never heard of CHI. It's a rare condition that affects 1 in 2,000 pregnancies and can only be diagnosed after birth, by examining the placenta under a microscope. The aim of my PhD is to discover the cause of this disease.

In a healthy pregnancy, the mother's blood flows through the placenta in a channel called the intervillous space. Here, it comes into direct contact with tree-like branching structures called placental villi. This is where vital exchange of gases and nutrients between the mother and baby occurs. In CHI, this channel gets clogged with maternal immune cells, impairing the exchange process. This has serious consequences for the baby. Only half of these pregnancies result in a live birth and some babies have to be delivered several months early by emergency Caesarean to give them a chance of survival.

CHI usually affects women who are fit and well, with no medical problems. It comes out of the blue and has a devastating impact on couples hoping to have a baby. The cruellest thing about CHI is that 8 times out of 10, it will recur in a subsequent pregnancy.

Any miscarriage or stillbirth is emotionally shattering for the parents, but for most it is unlikely to happen again. This is where CHI is different. When Amy received the diagnosis after her second daughter was stillborn, she experienced a "double grief": not just for the loss of Grace, but for the loss of the future and the family she'd envisaged.

There are no tests that can reliably predict recurrence of CHI, so these subsequent pregnancies require incredibly close monitoring. Many women will have scans every fortnight, battling to suppress their dread as they return to the same ultrasound departments where their previous losses were diagnosed.



**WINNER**  
**EMILY**  
**CORNISH**

**MRC Clinical Research  
Training Fellowship**  
University College London

The cause of CHI is unknown. However, when you look at a placenta with CHI through a microscope, at a cellular level it looks remarkably similar to a rejected kidney transplant. My hypothesis is that affected mothers make an antibody that attacks the developing placenta. This leads to inflammation of the placenta and an influx of maternal immune cells. Support for this theory comes from the fact that when couples affected by CHI undergo IVF using their own eggs and sperm and transfer the embryo into a surrogate, the pregnancy progresses normally with no signs of CHI. This confirms that the problem comes from the mother's immune system.

Initially, designing a research study focusing on CHI felt daunting – how could I expect to recruit meaningful numbers of women, when the disease is so rare? Luckily, I was rescued by a woman called Claudia and a Facebook group.

Claudia lost four sons to CHI in the space of three years. As she navigated through the heartache of learning to live without her children, she became determined to raise the profile of this mysterious and brutal disease. Since then, she has been a tireless advocate for advancing research into CHI and helps to run a Facebook support group for affected women. The group has over 700 members and is a lifeline for parents struggling to come to terms with their diagnosis.

Despite its rarity, thanks to Claudia's group I have managed to recruit more than 30 women to my study. I collect blood and placental samples from them to look for unusual immune cells, proteins or antibodies that could explain what goes wrong in their pregnancies. I also analyse their DNA to

look for variations in their genetic code that might predispose them to CHI.

By purifying antibodies from their blood and comparing them to women who have had healthy uncomplicated pregnancies, I have shown that women with CHI react abnormally to placental proteins. My next challenge is to determine exactly what triggers this reaction and unravel the molecular basis of how this leads to catastrophic placental damage. And the ultimate goal is to use this knowledge to develop new, targeted treatments that can prevent recurrence.

My team is already making progress on the final question. Claudia lost her children ten years ago and eventually resorted to surrogacy, as nothing her doctors tried could stop the relentless recurrence of CHI. However, over the last few months we have become cautiously optimistic about a new treatment protocol, which involves suppressing the mother's immune system during pregnancy. Once again, we drew on the parallels between CHI placentas and rejected transplants. This treatment is the same one that people with organ transplants take to stop their bodies rejecting the donated organ. It's an intensive cocktail of medication that requires regular blood tests and close vigilance for early signs of potentially dangerous side effects. We have only treated a handful of women so far, but the majority have gone home with a healthy baby.

But until we discover the cause of CHI, these treatments are, at best, an educated guess. I'm only nine months into my PhD and am well aware that finding a miracle cure is an unrealistic dream. However, my vision is to make concrete advances in

our understanding of CHI so that future treatments can be tailored towards the precise defect in affected women's immune systems.

After Amy lost her daughters, she felt like she'd been "kicked out of the motherhood club". She couldn't bear to lose another baby and although the prospect of a heavily medicated pregnancy filled her with anxiety, she decided to take the gamble. Amy took over a thousand tablets and injected herself with a blood thinner more than two hundred times during her pregnancy with William. She travelled to the hospital in commuter trains during the pandemic, came to scans alone because of the restrictive COVID visiting policy, and had blood tests almost every week.

I am inspired and humbled by the extraordinary courage and determination of the women who embark on this regime. CHI has been neglected by the obstetric research community, but thankfully, the women who have experienced it are highly motivated and desperate to educate us. If my research can draw attention to the massive impact of CHI and give affected families some hope, then spending my evenings crouching in an operating theatre with an ice bucket seems entirely worthwhile.





## Star-struck: exploring the secret universe of bacteria

You are teeming with life. Indeed, you are home to about 40 trillion bacteria. That's more than the number of human cells in your body, and 100 times as many as all the stars in our galaxy.

This complex ecosystem is called the microbiome, and the vast majority of bacteria in your microbiome are not only harmless but utterly indispensable. From the moment you were born, your bacteria have trained your immune system, digested your food, and helped keep more dangerous infections away. Your body is a veritable Tardis: unfathomably bigger on the inside, and capable of transporting you back and forth in time.

Because studying your microbiome can reveal clues about your past (like when you last had antibiotics) and even your future (like your risk of developing diabetes). But the most exciting bit is how much there is still left to learn from studying the microbiome. The technology required is cutting-edge and is becoming more advanced and affordable every day. Researching the microbiome is like an intrepid space voyage, as humbling as mapping an infinity of stars. And so I thank these lucky stars for my PhD, because I get to research the microbiome every day.

My PhD is about how the microbiome develops in newborn babies, and whether we can change it for the better. Babies are born with virtually no bacteria (compared with your 40 trillion-strong army of bugs). Within minutes of life, they become covered (inside and out) with bacteria, mostly from their mothers. I'm particularly interested in the baby's nose and throat microbiome: the bacteria living here are mostly harmless, but can sometimes cause infections of the lungs (pneumonia) and brain (meningitis), which are amongst the commonest causes of death in young children worldwide.

Although vaccines protect against some of these infections, they do not protect the very youngest babies who are most at risk of severe infections. And it is becoming harder to treat such infections, because bacteria are becoming more resistant to antibiotics. The baby's microbiome even appears related to their risk of developing asthma, allergies and other health problems, even much later in life.



**RUNNER-UP**  
**ANASTASIA**  
**THEODOSIOU**

**MRC Clinical Research**  
**Training Fellowship**  
University of Southampton

We know that a healthy, resilient microbiome is more likely in babies who are delivered vaginally and breastfed, and that a more unstable microbiome is seen in babies born by caesarean section or who receive antibiotics or formula milk, highlighting the crucial link between the mother's microbiome and that of her baby.

So that got us thinking: if a baby's microbiome comes mostly from their mother, could we change the bacteria that are passed on from mum to baby? And, if particular microbiome signatures seem to predict future health problems, could changing these signatures provide a new way of protecting babies and young children? That is exactly what we are trying to do. We recruit pregnant research volunteers and, about a month before giving birth, they each receive nose drops containing 100,000 live 'good' bacteria (called *Neisseria lactamica*). This type of 'good' bacteria is completely harmless and naturally lives in the throats of most toddlers, but is uncommon in adults and newborns. It can protect against a type of 'bad' bacteria (called *Neisseria meningitidis*), which can cause serious and even fatal meningitis and bloodstream infections, especially in babies.

Our team have previously shown that the 'good' bacteria nose drops can safely and reliably reduce the 'bad' bacteria in healthy adults; but they have never been tried in pregnant women or babies... until now. Our hope is that, after giving birth, the 'good' bacteria will transfer from the mother to the baby, mimicking the natural way that newborns receive much of their microbiome from their mothers.

In practice, this means that I spend much of my time collecting nose and throat swabs from women and their babies, from the third trimester of pregnancy until the babies are 4 months old. Come day, night or weekend, I wait for the phone call announcing that one of our participants has given birth, and I visit her to swab (and coo over) the new baby within 24 hours. Over the next 4 months, I drop in on them at home, loading my car up with swabs, gloves, sample pots and a rainbow of children's books. I am continually awe-struck by the amazing generosity of our participants in sharing these special early family moments with our research team.

In the lab, I get to work trying to grow the bacteria. The swabs are applied to plates of brightly coloured jelly (the bacteria's favourite food), and left overnight in a warm, cosy incubator. I thoroughly pamper my precious bacteria until they've blossomed into thriving communities of millions of bugs. The next step is to sequence the bacteria's DNA. DNA is essentially an instruction manual telling the bacteria what to do; the genetic sequence is a string of thousands of letters making up this instruction manual. This is where laboratory meets laptop. Using bespoke software, we can identify the bacteria based on their genetic sequence. Just like tracing constellations to make sense of countless stars, we build intricate maps showing how the bacteria in mothers and babies are related. These maps tell us which bacteria have been passed on from mum to baby, and how they've changed over time.

I am only about half-way through my PhD, so there are many questions left to address. So far, we have

shown that the 'good' bacteria in the nose drops do indeed set up home in the mothers' throats, and we can still detect them 4 months later. And, more importantly, we have not encountered any safety concerns in mothers receiving the 'good' bacteria nose drops, nor in their babies. The next step is to find out whether this intervention has caused any detectable change in the babies' microbiome signatures. If it has, this could pave the way to exciting future research. For example, could 'good' bacteria be given directly to the baby, rather than the mother? And do mothers or babies receiving 'good' bacteria have fewer infections or other health problems? Could live bacteria one day offer a new tool for fighting infections, alongside vaccinations and antibiotics? The best thing about research is that the work is never done; there are as many questions to ask as there are bacteria in your body, or stars in the night sky.



## Apathy research: why should we bother?

There are tales of a philosopher who, on hearing of his son's death, calmly noted: "I knew that I had begotten a mortal". History did not record his wife's reaction, but I'm not sure she would have been thrilled. Nevertheless stories of men like him are celebrated in certain philosophical circles. These individuals seek an elusive and virtuous state referred to as apatheia.

A state free from passion which was thought critical to achieve true peace of mind, and peace with the world. From Buddhism to Ancient Greek philosophy, apatheia was considered the key to moral and spiritual success. Perhaps with a little more apatheia I wouldn't feel the urge to fling my computer out of the window whenever it stops working.

When my Grandpa was told that his mother had died, he expressed the admittedly less articulate response: "Oh". His face didn't change, and he didn't shed a tear. Doctors said his apparent lack of feeling was due to a clinical condition called apathy. Researchers describe apathy as a loss of goal-directed actions, but I prefer the explanation that 'his get up and go, got up and went'. He was no longer interested in the things he used to enjoy and instead was perfectly content to sit in his chair for days on end. Why then was my Grandpa's condition not considered a moral and spiritual success? Why was he not sought out by scholars and pilgrims seeking this enviable state of neutrality? And, if my Grandpa

was perfectly content, why have I dedicated my PhD to understanding and treating apathy?

The central idea in my lab is that people with apathy lack clear expectations of how the world should be. So, when the world changes (for better or for worse), it still doesn't change enough that they are motivated to act. If the light dims in their living room, it remains light enough. If the temperature drops, it remains warm enough. Whilst you and I may be motivated to fix the light bulb or grab a jacket, people with apathy have a much broader definition of how dark is dark enough, or how cold is cold enough to trigger a course of action. The world fails to violate their brain's expectations and so they sit, apparently uncaring, in the cold and dark.

A former PhD student tested this theory in a scientific experiment which showed people with high levels of apathy have less precise expectations of the world. I am now testing this idea in people with the same diagnosis as Grandpa, people with



COMMENDED  
**REBECCA  
WILLIAMS**

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frontotemporal dementia. Frontotemporal dementia is a rare type of dementia often beginning in middle-age, that causes changes to behaviour and personality, with many people becoming apathetic as a result.

The real problem with apathy in dementia is that it often points to a worse prognosis: people with apathy die sooner. So, every week I meet people with dementia. Some of whom have profound levels of apathy. I talk with them about their daily life, and play computer 'games' which reveal information about their decision making and expectations. By combining theory and data, I hope to identify circuits in the brain which cause someone to become apathetic. Even when applying for my PhD, I knew the reason this was important. To find treatments which can improve apathy, we need to know what to target in apathetic brains. Understanding the mechanisms at fault is a crucial part of finding drugs which can improve apathy and help people. Sounds great. Right?

My doubts began about three months ago during a discussion with my supervisor. In our conversation he asked why it was important we researched apathy and I listed all the very reasonable explanations I just gave you. "But why do we need to cure apathy?", he said. I was a bit shaken by this. Treating things is always good, isn't it?

I thought again: "Because it impacts their quality of life." This did not satisfy. Aren't patients with apathy in a state of contentment with the world as it is? After all, they have achieved the virtuous state sought after by monks and philosophers

for centuries. If you ask apathetic patients with frontotemporal dementia if they're happy, they will often tell you that they are. Is it fair to treat apathy with the risk of changing that answer?

Okay, so not quality of life. "Because they will be more motivated to take vital medicines". He smiled then, a long slow smile. There are currently no cures for frontotemporal dementia. We should absolutely use drugs to try and improve the condition but what medication should they take? As we sat and discussed, I began to question the purpose of my PhD. Perhaps I should have been spending time on worthier pursuits? Watching the new season of Bridgerton, for example.

When my Grandpa was told that his mother had died, he said: "Oh". Perhaps he truly was content with this. But over the years living alongside this condition, years of feeling as though my Grandpa didn't care about anything, my Grandma was definitely not in a state of contentment. Carers often don't get to share their side of the story, yet I see and hear the immense burden of dementia through my research, and the voices of family members. They confide in the spare moments within clinic and research visits. Research which they are rarely the focus of. Families aren't the ones who receive new drug treatments after all.

When I saw that the purpose of this essay was to explain why my research mattered, I knew that to do it justice there were two stories I had to tell. On one hand, it is still true that treating apathy could be beneficial for patients, increasing survival and independence. But treating apathy may have an

even bigger impact on their families. It is a story which is applicable to many neurodegenerative conditions, and which is sadly experienced by many first-hand.

My research is focussed on understanding the brain circuits which cause apathy. Over the next three years, I will study computational models of behaviour, brain scans and experimental medicines to develop better treatments for apathy in dementia. However, in my first year, I've also learned that questioning the motivations behind our research can be as important as the research itself. Apathy is not a philosophical idea. Apathy is a clinical condition that is bad for patients, and awful for their carers. Apathy research may be the key to giving families more time to enjoy each other's company. That's why I bother.



## Schistosomiasis in pre-school aged children in Albertine Region of Uganda: a neglected tropical disease in a neglected community in a neglected population

Mr. Smith stood at the edge of the escarpment, savoring the golden sunset bouncing off the gentle waves of Lake Albert. This is his third visit to Murchison Falls National Park, the game reserve putting Uganda on the global map and keeping a steady flow of the much needed foreign exchange from tourism.

The usually six-hour dusty trip from Kampala has been rather short this time since the road was tarmacked owing to the discovery of oil in the region. There is even rumor of an airport springing up. The latest model of Toyota Landcruiser VX V8 slowly weaved its way through a roadblock of baboons as the tour guide frantically waved to the occupants who barely noticed him. It was the area Member of Parliament, one of the 529 of them shaping the country's policy.

Less than 100meters from the splendid tarmac, Munguromo sits in apathy on the verandah of the mud-and-wattle hut that houses the family; now comprised of eight other siblings and his widowed mother. His father had passed on following a long episode of abdominal swelling and, eventually,

vomiting blood. Munguromo was just a few months old then.

The other children played around him, occasionally trying to get his attention. On a merrier day, Munguromo would have accompanied his mother to fetch water from the lake and played in the water with other children. Not today. Having had bloody diarrhea for over a week, his mother finally decided to take him to the health unit; a trip that had yielded more frustration than answers. She had spent her entire earnings from the previous week on transport alone and all she had to show was a prescription for drugs she couldn't even afford. The medical assistant had diagnosed the child with dysentery. 'I guess I will just have to wait it out. The last one resolved on its own', she thought to herself. 'Various



SHORTLISTED  
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children in the neighborhood experience this, probably due to some trigger in the environment.' But this is just the beginning.

Bilharzia affects approximately 240 million people across 78 countries and Munguromo is just one of over 123 million children; 40% of them pre-school aged children (PSAC) in similar settings across Africa. The disease is caused by a parasitic worm which resides in the blood vessels of the intestines or the bladder from where eggs find their way into the stool or urine depending on the species, and are passed into the environment. In fresh water, they hatch into larvae called miracidia which enter a suitable snail. Another form of larvae (cercariae) emerging from the snail have the ability to penetrate human skin, move to the lungs and eventually settle in the blood vessels of the intestines or bladder.

Nothing seems to happen for many months or even years thereafter. However, the thousands of eggs produced daily poke micro-holes in the walls of the intestines causing bloody diarrhea. Because the eggs are foreign bodies, a frantic immune reaction is mounted and a 'wall' forms around them so that they are can no longer cause havoc. It seems like a problem solved; only that this wall is composed of fibrous tissue that compromises blood flow through organs like the liver. In a bid to continue its role of filtering and re-channeling blood back into circulation, alternate routes are devised after many years (bilharzia worms can live up to 40-years!). One such route is via blood vessels of the esophagus whose capacity is designed for much less than it carries under these circumstances. Like an ocean liner moving through a swamp, disaster is bound to happen and the vessels rupture leading to vomiting

blood. If the patient is lucky to make it to the health facility; and if blood is available, perhaps he will have a few more weeks. However, the relatives have other ideas. They use the last of their resources to make the perilous boat journey across the river to the prominent witchdoctor near the Congo border. By this time, it is too late.

In other cases, heart failure, retarded growth, cognitive deficits and poor vaccine responses accompany the scourge of bilharzia, drastically reducing the quality of life.

A single dose of praziquantel, the only treatment for bilharzia, would have cleared the infection. If only the health worker had interrogated that episode of bloody diarrhea many years back. But what would he have done that early? Praziquantel is not licensed for use in PSAC and only supplied through donations. Even the erratic supply is usually shunned by those that need it most because of side effects like severe abdominal pain, vomiting, diarrhea and terrible taste. Improvement in sanitation facilities also goes a long way in preventing the disease. However, Buliisa district is firmly rooted near the bottom of the national sanitation league table and the fishing communities are even further below the district average.

The sunset is truly beautiful. For the hundreds of tourists on the game drive, it's a golden opportunity to catch a glance of the thousands of game animals as they get their supper. For the oil explorers, it's time to reflect on the day's achievement (a cold drink at hand) and drool at the prospect of millions of dollars beckoning. In the nearby jungle, the lion sleeps and the owls get ready for night life. As for

Munguromo, the hunger pangs (or is it something else?) ensure that it's going to be another restless night. That is most likely the last light he will see till the next sunrise. Praziquantel may leave a bad taste; but looking back, there is no worse taste than the feeling of losing a loved one to such a preventable condition. Enough with the wisdom of hindsight. Who's going to shine the light and provide the foresight for Munguromo to avoid following in his father's footsteps?

I am part of the PIP Trial team whose study aims to determine the optimal dose of praziquantel in PSAC with the goal of including them in routine treatment programs. Preliminary studies showed that it is safe but a higher dose may be required for them. However, even with favorable results, it will take more than a clinical trial to achieve the WHO goal of elimination or the Sustainable Development Goals. There is dire need for funding for more research, diagnostic tools, access to praziquantel and various public health interventions. Perhaps getting the attention of the area parliamentarian will kick start the required chain of action. Perhaps.



## The invisible man: immune therapies for cancer

Picture the most frustrating 'Where's Wally' you've ever encountered. Now scale it up to the size of Wembley stadium and give the entire crowd stripey shirts. This is the kind of challenge our immune system faces in detecting cancer.

The human body encounters thousands of germs every single day. Some are vital to our survival, helping us make vitamins and digest food, while others are potentially life-threatening. It's up to the millions of immune cells that patrol our bodies to guard us against infection and fight off these constant threats. The system is effective, recognising and removing germs before you've even had the chance to tut at the person coughing loudly on the train. The problem comes when the threat isn't coming from outside the body, but from our cells themselves.

Cancer is a disease which hides in plain sight, with tumours masquerading as normal healthy cells. The challenge of my research focuses on helping our immune system unmask and remove these cancerous intruders. The task is so complex that it has taken years of painstaking research to identify a few targets that allow tumours to be attacked without harming the surrounding healthy cells. All this research is now paying off, as these markers are

being used in a new type of treatment, successfully directing the patient's own immune system against their cancer.

Chimeric antigen receptor (CAR) T cell therapy is a revolutionary approach made possible by advances in gene editing technology. The treatment uses T cells, a type of white blood cell key to the body's immune response. Millions of these cells are equipped to recognise a specific marker, allowing them to latch onto the cancer cells they previously couldn't identify. Astoundingly, in only ten minutes each cell can be armed with a protein which acts as a cancer recognition device (that's the 'CAR' part) and delivered back to the patient, ready for action. All it takes is a single visit to a clinic and a blood sample.

Suddenly, that Where's Wally doesn't seem so impossible when an entire army joins your search, trained to spot the tell-tale glasses and bobble hat.



**SHORTLISTED**

**ALICE  
EVANS**

**MRC iCASE Studentship**  
University of Oxford

The therapy has produced incredible results in blood cancer, with patients surviving for years against all odds. Following stories of success, the NHS has set up multiple clinics and is offering the treatment across the country. It's an exciting step which represents a bold new age in the fight against cancer – moving away from harsh chemical therapy and towards more personalised treatments. It is an essential development in improving the lives of cancer patients everywhere, sparing them the gruelling side effects of chemotherapy.

I am researching the application of these potentially transformative immune cell therapies to the much tougher challenge of pancreatic cancer. It's a daunting but vital task, as currently only 7% of pancreatic cancer patients survive five years from their diagnosis and there haven't been any new approved treatments for over a decade. This life-changing illness can appear without warning, often only marked by a simple stomach-ache or a bit of back pain. We've all experienced and dismissed these niggles but, for some, the consequences can be devastating.

Investigation in this area is critical but there are some serious hurdles to overcome. Pancreatic tumours are surrounded by a dense tangle of tough proteins (think *Where's Wally: Amazon Rainforest* edition), making it challenging for immune cells to detect any of the tumour hiding within. Those that do fight through the thicket are met with a barrage of chemical signals which the cancer deploys as a form of camouflage, to escape detection. These factors mean that currently CAR-T cell therapy doesn't work as a treatment for pancreatic cancer.

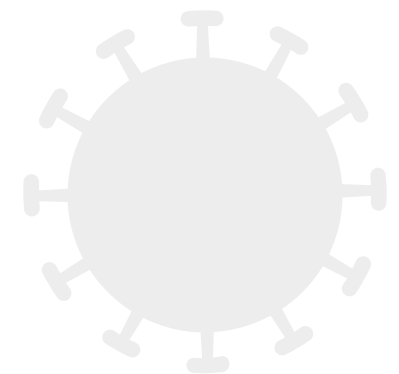
To overcome these challenges, we must find models which allow us to examine how CAR-T cells respond to pancreatic tumours in the most representative way possible. This is tricky using animals or cells grown in the lab, as they don't allow you to recreate the proper structure of a tumour (the forest) or the complex chemical signals (the camouflage) which make treatment so difficult. To tackle this problem, I study tumour samples taken directly from pancreatic cancer patients following surgery. It's quite a surreal process – collecting a tiny piece of pancreas from the hospital and furiously cycling it back to the lab – but it allows some pretty amazing research to happen. I set to work creating tiny slices of the tumour, keeping these alive in dishes and studying how they respond to therapy. All of this can be done in a matter of days, meaning that we can make discoveries about the cancer before the patient has even left their hospital bed.

So far, my project involves developing a method to study how drugs interact with these tumour slices. We've been able to show that chemical treatments can affect how well the tumour slices survive, even linking this to how well the patient responds to the same drug. The next challenge is working out how to do this with immune cells. With the help of fluorescent tags, I can highlight the cells, then use powerful microscopes to follow them through the slices on their search for the cancer. It's often easy to forget what these glowing pixels on a computer screen represent – a chance to make a real difference to patients' lives.

I am lucky enough to be working with a company developing a new type of CAR-T cell therapy,

specifically designed for pancreatic cancer. Soon I will be able to take blood samples from patients and help to create the little armies of gene-edited T cells, ready to recognise and fight their cancer. I will then test these cells out on the patients' tumour slices, in the hope that we can improve the results in the lab, overcoming the obstacles that have made pancreatic cancer such a tough nut to crack. If this can be achieved, the final step will be to take the solution directly to patients, with the goal of producing a safer, more effective therapy than ever before.

A few decades ago, many people diagnosed with blood cancer would be faced with harsh, ineffective treatments and little chance of success. Now, thanks to immunotherapy, the outlook is far brighter. I want my research to help to bring this glimmer of hope to pancreatic cancer patients.





## Choosing life: stopping Scotland's climbing drug-related death rate

The National Records Office began recording Scotland's drug-related death rates in 1996, the same year that saw the release of the film adaptation of Irvine Welsh's novel, *Trainspotting*. The iconic piece of Scottish cinema explored the themes of urban poverty and heroin use in 1990s Edinburgh and posters with protagonist Renton's "Choose Life" monologue are still available at any student poster sale to this day.

26 years on, Scotland has the highest drug-related death rate in Europe. Its statistics are three and a half times higher than in the rest of the UK and have been the highest since records began for 6 years in a row.

Drug use in Scotland is mostly driven by poverty, much of which can be traced to the economic and social consequences of deindustrialisation in the 1980s. I am undertaking my PhD at the University of Dundee. Dundee, Scotland's fourth largest city, has invested millions of pounds in the regeneration of its city centre. Formerly the city of jute, jam and journalism, Dundee is now home to the only outpost of the Victoria and Albert Museum and will house the second phase of the Eden Project. In 2015, *GQ* magazine declared that Dundee is becoming the "coolest little city", praising it for its reinvention as a food and art destination. However, Dundee is also the Scottish city with the highest drug-related death

rate at 43.1 per 100,000 people, nearly double the Scottish average. Due to Dundee's relatively small size, issues such as poverty and social deprivation are concentrated. Any drugs arriving in the city spread faster. While millions of pounds have been spent on rejuvenating Dundee's city centre, these concentrated, most deprived areas have not been able to benefit from this funding, deepening the city's inequality.

Much of the public debate around Scotland's drug problem has been clouded by stigma. Slurs regularly appear in tabloid headlines. Discussions swirl around "lifestyle choices", and who is or is not "deserving" of support. These debates demonise people who use drugs and lead to their marginalisation. When treatment is stigmatised, those who need it are less likely to seek support. This discourse leads us away from the long-term solutions to Scotland's drug problem, which lie in reducing and eradicating



SHORTLISTED

**KRISTINA  
HNÍZDILOVÁ**

**MRC iCASE Studentship**

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poverty and inequality. Alongside addressing these issues, more acutely, we must address the climbing drug-related death rate.

In 2018, the Scottish Government launched a national drug and alcohol strategy to fund evidence-supported methods to reduce harms associated with problem substance use, focusing on drug-related deaths. As part of this strategy, the Drug Deaths Taskforce was assembled in 2019 to improve health and quality of life in people who use drugs, by improving treatment, tackling stigma and inequality as well as widening access to naloxone and increasing awareness of new dangerous drugs available on the streets.

While in recent years, illicitly manufactured benzodiazepines and crack cocaine have become more commonly used drugs in Scotland, the leading cause of Scottish drug-related deaths is opioid-induced respiratory depression (OIRD). Deaths from opioid overdose are almost never instant. Opioids, such as heroin, slow down the central nervous system, which in turn slows breathing. Opioids affect the medulla oblongata and the pons, two areas in the brainstem that control respiration. When opioids attach themselves to opioid receptors in these areas, respiration is suppressed, which can lead to the brain becoming starved of oxygen and carbon dioxide building up in the bloodstream, leading to death.

Naloxone, a drug which blocks the effects of opioids, is effective at reversing OIRD, however, there are many barriers to overdose response and naloxone administration, such as failure to recognize the signs of an overdose. Many people are afraid to intervene

due to a lack of training, or fear of prosecution, since drug use may be criminalised, or if individuals have outstanding arrest warrants. Calling an ambulance is often seen as a "last resort". Furthermore, naloxone administration relies on bystander presence. For over half of people who died of drug-related deaths in Tayside in 2019, a bystander was not there, as they passed away in their own homes. Detecting an overdose event and triggering a response in time is an unmet need and would benefit many people, especially those who use drugs alone.

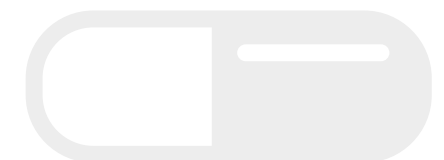
My PhD project explores how we can use digital technology to reduce drug related deaths. We aim to investigate if a sensor attached to the chest can accurately and reliably detect abnormal respiratory patterns in people who use drugs so that we can identify trigger points to send an emergency call. Much like in an activity tracker, the sensor we are using contains an accelerometer, which measures how the participants' chest rises and falls, giving us a reading of the participants' respiration rate. In this trial, we recruit participants from a needle exchange service in Dundee's city centre and monitor their respiration over four weeks to collect baseline data. Collecting data from people who use drugs is important, as this data will inform algorithms which will aid us in defining parameters and trigger points for an emergency call.

Understanding the acceptability of the device to people who use drugs is crucial to its successful implementation. We have been interviewing study participants about their experiences wearing the device, and carrying out focus groups with third sector stakeholders, such as drug services, to assess its suitability and how it could best serve the needs

of people who use drugs. Our goal is to develop a device which will be able to send an emergency call upon detecting abnormal respiratory patterns, so that an emergency contact can intervene in time and administer life-saving naloxone.

So far, our project has been met with enthusiasm and eagerness from people who use drugs and stakeholder groups alike, however, it has not yet been a year into the project, so there is still a long journey ahead of us. While we are very passionate about our project, it is important to recognize that even if the development of this device is successful, it will never be a cure-all.

One size will never fit all, in order to address Scotland's drug-related death rate, there must be options available for all patients. Everyone deserves the right to seek support without demonisation and stigma, whether or not they are ready to stop using drugs. The reasons why drug problems develop are numerous and complex, often involving inequality, poverty and trauma, issues that must be addressed for a long-term solution to Scotland's drug problem, so that no more families will have to lose loved ones to addiction.



## Let the cells do the talking: how babies can tell us how they're feeling

The couple walks through the double doors. The ward is quiet. A backdrop of thrumming ventilators and the rhythmic beeping of life support machines is both soothing and suffocating. The couple take up their vigil at the bedside of their new baby, as they have done every day for the last few weeks. She looked healthy yesterday but today she is full of tubes – helping her breathe, feeding her and giving her life-saving antibiotics. The doctors try to reassure them, saying that this isn't unusual. They promise they're doing everything they can, but babies just fall sick too quickly sometimes. But is this really the truth? Or are we just not looking out for the right signs?

Every year, around 1 in 7 babies are admitted to neonatal intensive care units (NICU). That's over 90,000 babies in the UK alone. In the NICU, doctors face the difficult challenge of monitoring these babies for any signs that they are falling ill. That's easy in adults: we get a headache, complain that we feel rough, pop a few paracetamols, and hope we feel better in the morning. If that doesn't work, it's off to the GP! Babies make a much more difficult patient. They can't tell you when they feel ill, let alone what is wrong with them. Newborn babies can also appear very resilient. If a baby picks up a nasty bug, they may seem healthy as the infection takes hold and only show symptoms when it is too late. In other words, babies can sneak right up to the edge of the cliff without anyone realising, and we only notice once they jump off.

As if this wasn't bad enough, the tests that doctors use to detect infections are a lot less useful in babies. Full blood counts, for example, provide doctors with information on the number of white blood cells (the soldiers in the army of the immune system). Healthy adults only have a small number of white blood cells present in their blood. If we fall sick, these numbers rapidly increase as the immune system gears up to fight the infection. Unfortunately, babies don't show the same pattern in their white cell counts, especially those in the NICU. Being born is a very stressful process and babies are well adapted to bounce back quickly. This means the white cell count can fluctuate drastically in the first few days of life, making it an unreliable tool for identifying sick babies.



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So, how do we help doctors pick up when a baby is sick before they dive headfirst off the metaphorical cliff? It's quite simple really. Why reinvent the wheel when we can adapt the tools we already have? The machines that give full blood counts are capable of measuring a lot more than just the number of cells. Using specialised techniques, these machines can tell us how activated, or 'switched on' our immune cells are. The main way this is done is by flow cytometry – a big word, I know, but let's break it down.

Think of all the cells in your blood as marbles. Each marble is a different size or colour or has a different pattern inside it. You shine a light on these marbles one by one and the light is scattered in different directions, depending on these features. Can this tell us how many marbles are large, how many are red, etc?

This is the concept behind flow cytometry. A full blood count machine uses flow cytometry to count how many of each cell type there is, as well as give us information on how activated these cells are. Cells are passed through a laser beam one at a time, and scatter the beam depending on their size, granularity (how much 'stuff' is inside the cells), and DNA/RNA content (how much the cells are making more 'stuff'). As we fall ill, our white blood cells gear up to fight the infection by making proteins that target and kill invading germs. This is known as cell activation and is marked by a change in the granularity and DNA/RNA content of white blood cells in sick patients.

The ability to measure the activation of white blood cells also allows doctors to distinguish between sterile inflammation and infection. If you knock your

head, the lovely bump that would form is due to sterile inflammation. White blood cells are recruited to help repair the damage but don't become activated as nothing harmful has entered the body. In a full blood count, we may see a slight increase in the number of white blood cells but there would be no evidence of activation.

Let's bring this back to the problem at hand: babies. We have a group of patients who cannot communicate with us and are notoriously good at hiding their symptoms. Most babies in a NICU are likely to have some form of medical equipment in their bodies. Even something as simple as a catheter could affect cell counts by causing sterile inflammation. And as we discussed before, newborn babies are busy recovering from being born. Most NICU babies are going to have an abnormal white cell count but let's dig deeper and look at the activation of their white blood cells. Baby X is on a ventilator and has a raised white cell count but no activation. Is she sick? Or just reacting to the tube in her lungs? Baby Y is being drip fed, his white cell count appears normal, but the activation is rocketing. Do we give him antibiotics?

This is where my research comes into play. Cell activation is an untapped resource in neonatal clinical care, having been neglected in favour of traditional, less effective methods of detecting infection. I plan to look at the results from full blood counts of NICU babies, specifically the activation of their white blood cells. In doing so, I hope to uncover the patterns of activation that are present at the start of and throughout an infection. By doing this, we can target babies as they begin to fall ill and hopefully rid them of any nasty bugs before they can

take hold. While I am only at the start of this journey, I have already seen some promising results. As an additional bonus, we can combat the unnecessary overuse of antibiotics in non-infected NICU babies, reducing the prevalence of MRSA (antibiotic-resistant super bacteria).

Perhaps cell activation can do the talking for babies.

Perhaps cell activation is the sign we've been looking for.



## Breaking the decades-long cycle of failed obesity policy

It's April 2020. The Prime Minister Boris Johnson has just left hospital where he was in intensive care with serious Covid-19-related health problems. Evidence about who is more vulnerable to Covid-related complications and death is still emerging. However, one thing that appears to be clear is that people living with obesity are at a greater risk of Covid-19 related hospitalisations, serious illness and death. Having been sceptical of strong government intervention on diet and obesity just one year prior, the Prime Minister's close encounter with death is catalytic and he decides that the government must do something about obesity. In July 2021, his government publishes an obesity strategy.

This year, 2022, officially marks three decades of government obesity strategies in England. The first was published in 1992 and it included some ambitious population obesity reduction targets. Needless to say, these were not met. In fact, in this time the obesity prevalence has actually increased from 13% of men and 16% of women living with obesity in 1993 to now more than a quarter of adults (27% of men and 29% of women) in 2019. How and why has this happened? How can government obesity policy have failed so badly after all these years? And what are the consequences of this epic policy failure?

Fuelled by these questions, I analysed the 14 obesity strategies for England that have been published

since 1992, which collectively contain no less than 689 policies. My research found that successive governments have failed to successfully reduce the obesity prevalence and related inequalities not only because of the policy ideas proposed, but also because of the way they have been proposed.

The hundreds of different policy ideas to tackle obesity and the related inequalities include school food and curriculum changes, guidance and standards for the food industry, provision of healthy food vouchers for low-income families, and a weighing and measuring programme for primary school-aged children. However, research shows that the largest proportion of the policy ideas are unlikely to be effective or equitable. For example, information



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campaigns have remained very popular with the government. The thinking being that government publishes dietary advice, people engage with it, they change their behaviour, and then ultimately their health and weight improves. However, this is unlikely to work for most people because individual behaviour change is tremendously difficult, especially long-term and especially when you live in conditions or face circumstances that make such change very hard. Evidence shows that shaping the environment and other key external influences to make it easy for people to enjoy a healthy life is much more likely to be effective and equitable. And yet, a much smaller proportion of the government's obesity policies have focused on doing this.

My research also found that the government has tended to propose policies in a way that makes it unlikely they will be implemented. I identified seven key pieces of information necessary for effective implementation, but only 8% of policies fulfilled all seven criteria, versus the largest proportion of policies (29%) that were proposed without a single one. Only 9% of policies were proposed with a cost or allocated budget, 19% with any cited scientific evidence upon which the policy was based, and just 24% were proposed with a monitoring or evaluation plan.

The above has led to an obesity policy merry-go-round where the same or similar policies are proposed again and again by different governments or different secretaries of states, and yet are largely unlikely to be effective and equitable or get progressed fully from implementation right through to monitoring, evaluation and beyond. For example, there has been a Conservative Party Government

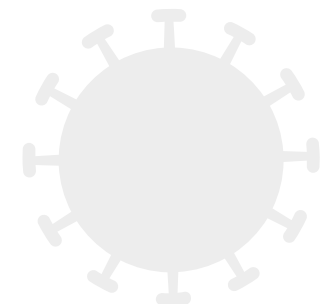
since 2015, which has published not one, but four obesity strategies containing many of the same or similar policies. New prime ministers have come in and instead of seeing through the policies already proposed or in progress, they have all published new strategies. But it's not just new governments that can come in and start again. In the last year, Prime Minister Boris Johnson has scrapped or sought to delay or revoke some of his own 2020 obesity strategy policies.

Meanwhile, problems such as poor diets and rising obesity rates are getting worse. The Covid-19 pandemic revealed how serious the consequences of failed obesity policy can be, as the Prime Minister Boris Johnson so personally experienced. The Global Burden of Disease (2017) found that poor diet is a factor in one in five deaths around the world. Four of the top five risk factors for healthy years of life lost to disease, disability and death are related to poor diet and physical inactivity. The evidence is writ large that poor diets have devastating consequences and there is increasing evidence on likely effective and equitable interventions. So, why does government obesity policy not reflect this?

One major barrier is that governments have tended to favour a less interventionist approach to reducing obesity, regardless of political party. Political decision-making is a primary arena in which scientific evidence comes up against ideology. The influence of neoliberalism, which advocates broad notions of individual responsibility, choice, a market-driven economy, and anti-government intervention, has been found by previous research to clash with more interventionist public health policies. Governments may have avoided stronger interventionist policies,

e.g., legislation and fiscal measures, for fear of being perceived as controlling what people eat. The vilification of such intervention is commonly referred to as "nanny-statism" – the unwelcome interference of the state in people's liberties and choices. Since politicians rely on the electorate to vote them back into power and Government relies on Parliament to support and facilitate policies, maintaining public and political popularity and avoiding potentially unwelcome policies are important. The question that remains is can scientific evidence be viewed as being more compatible with a neoliberal ideology? And if so, then how?

Through my research, I am trying to understand how government policy can more effectively, equitably and rapidly solve major problems like rising obesity rates. Breaking the decades-long cycle of ineffective obesity policies not only has profound implications for population health, but for government and the way it works too. A 2021 National Audit Office report found that the Department of Health and Social Care did not know how much it spent tackling obesity and yet it continues to spend billions of pounds treating the consequences. From our own health to the way that our country is run, improving government obesity policy matters to us all.



## Cancer: It's a hard cell?

**It is not hard to write about why cancer research is important. The statistics speak for themselves. 1 in 2 of us will hear the words "you have cancer" and around half of those will sadly lose their life. Whilst decades of incredible research have helped millions of people obtain earlier diagnosis and better treatment, there is still a very long way to go.**

When I told my friends and family that I would be starting a PhD in cancer research, the most common question was "are you going to cure cancer?" The short answer is "no". There are hundreds of DNA mutations responsible for a whole host of different cancers. No two cancers are alike. The mutations seen in cancer cells will differ between cancer type, between tumours of the same type and even cancers within the same person. This variability makes finding a single "cure" for all cancers almost impossible as treatments may work for some cancers, but not for others.

My research is not focused on a specific mutation but understanding how cancer cells spread throughout the body at the whole cell level, specifically metastatic melanoma (skin cancer). I want you to imagine you are holding a golf ball in one hand and a stress ball in the other. Whilst they are similar in shape and may look similar, they feel very different. A golf ball is hard and rigid, allowing it to travel long distances when hit by a golf club. A stress ball (of which I am much more familiar) is squishy and pliable, helping it to do its job of relieving stress, something I

have needed on more than one occasion during my PhD. The stiffness of each of these items helps it to perform better in its job and a similar theory can be applied to cancer.

Before I go any further, let me take you on the journey of how a cancer cell spreads in the body. Cancer begins with a cell or group of cells which have gained the mutations needed to divide uncontrollably, evade cell death and travel to other parts of the body. Not all cells growing in the tumour will have all the mutations needed to survive and spread. A cell that does get the mutations required, must squeeze through layers of cells, enter the blood stream or lymphatic system, survive the journey, and then grow elsewhere in the body; this is a cancer that has metastasised. Your average, everyday cells would not last five minutes undertaking a journey like this, but cancer cells change and adapt to survive where others could not.

My work looks at whether cancer cells change their stiffness (literally how squishy a cell is) to help them survive this journey. Going back to the golf ball/stress



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ball analogy, imagine you have a tube which is slightly smaller than the diameter of each item. The rigid golf ball will either break in the tube due to the sheer force experienced or the tube will be blocked. The stress ball however, with its elastic texture, can squeeze through the tube and immerge at the other end unharmed. Applying this theory to cancer research, you may assume that a cancer cell may be softer than a non-cancerous cell as they are able to travel in the blood stream, survive and grow at a secondary site in the body.

I know the thought of looking at the stiffness of cancer cells all day is a strange concept. "How on earth do you measure the stiffness of a single cancer cell?" I hear you ask. Three years ago, before I started my PhD, I would have had the exact same question. I now know the answer is with a novel system called Scanning Ion Conductance Microscopy and nanopipettes.

A nanopipette is a tiny glass tube tapered at one end, similar to a needle, with an opening of just 100 nanometres in diameter. That is around 1000 times smaller than the diameter of a human hair. An electrical current is passed through the tip and the nanopipette is lowered to the surface of the cell. As the gap between the nanopipette tip and the cell surface gets smaller, the space the current can flow through gets smaller and so the current drops. A specially designed software then uses this drop in current (and a lot of physics) to calculate the cell stiffness. The nanopipette literally hops along the cell surface, building a picture of the cell's surface and stiffness as it goes, pixel by pixel. From this,

we can investigate the stiffness of melanoma cells through the different stages from non-metastatic to aggressively metastatic.

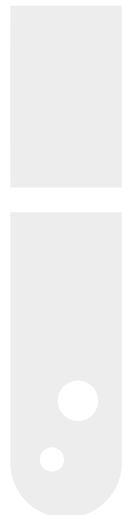
The answer to whether cancer cells are softer than other cells is not a simple one. Scientists have found a link between lower cell stiffness and a higher ability to spread. This has been linked to changes in the cell's cytoskeleton, the scaffolding found in a cell which gives it its structure, shape and stiffness. This has been somewhat true for my research. Metastatic melanoma cells are generally softer than non-metastatic melanoma cells.

However, an aggressive form of metastatic melanoma has been found to be significantly stiffer than all other cell types. This throws the golf/stress ball analogy out of the window because how can a rigid cell survive the journey to metastasise? As with a lot of research, we are not sure yet and requires a lot more investigation. It could be that metastasised cancer cells are able to adapt to their surrounding better as they must survive at a secondary site in the body. As the metastatic cancer cells are grown in a hard plastic dish outside the body, it may be that they are changing their stiffness to mirror this hard environment.

Cell stiffness may be an important and overlooked marker for cancer cell metastasis. If there was a clear link between cell stiffness and cancer metastasis, there would be potential for it to be used in a diagnostic setting. Similarly, we could identify cell stiffness as an important drug target to help prevent metastatic cancer cells from ever surviving the

journey in the blood stream. However, the road to effective cancer diagnostic tools and treatments is not an easy one. Cancer research is complex and sometimes contradictory. Although there is a lot of work suggesting softer cells spread more effectively, there are also scientists showing that metastatic cells are stiffer, as I have found.

My hope is that my work can contribute this relatively unknown question: is cancer a hard cell?



## When categories fail

Take a look around, and think for a moment about what you see.

Have a list in your head? Me too – I see a laptop and headphones, some houseplants, and a chair tucked neatly underneath a desk. There are paintings hanging on the wall, along with some postcards and photos. An overflowing bookshelf sits in the corner.

In other words, I see collections of objects: electronics, plants, furniture, wall decorations, and things to read. You probably do too. When we look around, we tend to create categories into which different objects can be grouped. When we mentally categorise something, we give it a set of attributes, uses, and appropriate contexts. Whether we're consciously aware of them or not, categories are useful. They help us make sense of a complex world and interact with it in a way that's meaningful to us.

So far, so good. When we're looking at readily-identifiable things in our homes, the task of forming categories is pretty straightforward, and we do it without thinking. But what happens when we start to apply this mental tool to our understanding of other people – a much more complex and ethically-pressing domain?

For a psychiatrist, categorising people is simply part of the job, but it's not an easy one. Most mental conditions aren't like physical illnesses: they don't have clear physical signs, and their causes vary

from person to person. In the UK, psychiatrists standardise this process using diagnostic manuals, such as the DSM-5 or the ICD-10. These manuals list different mental conditions and the symptoms associated with them. This makes categorising patients a bit easier – rather than relying on intuition alone, a psychiatrist has a set of criteria to refer to when assessing someone. In theory, a psychiatrist can look at their manual, see which diagnosis best fits a patient's symptoms, assign the corresponding diagnostic label, and suggest ways to seek further help.

The issue is, diagnostic categories often have fuzzy boundaries. Many conditions share similar features, and individuals with the same diagnosis can differ considerably. This type of ambiguity introduces a host of problems. If our diagnostic labels don't consistently describe similar experiences, then we can't trust their reliability. Even worse, when categories fail to accurately describe patients' symptoms, it means that patients can't access the resources they truly need.



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Inattention and hyperactivity are common across a variety of conditions: Attention Deficit Hyperactivity Disorder (ADHD), autism, and intellectual disability, to name a few. This means that when a child is very hyperactive, or struggles to pay attention in school, there's some uncertainty surrounding the root causes behind their behaviour. As a result, assigning a diagnostic label to that child can be a real challenge. In 2018, Joe Bathelt and other colleagues of mine at Cambridge ran a study of over 800 children with developmental difficulties. Their findings were startling: specific diagnoses weren't predictive of children's traits.

As a neuroscience PhD student, I'm interested in the brain development of children with high levels of inattention and hyperactivity. While I was originally trained to look at things in terms of diagnostic categories, my colleagues' findings changed my thinking on the value of these labels. Rather than only working with children who carry an ADHD diagnosis, I analyse data from a sample of nearly 1,000 children with a broad range of conditions. Having access to a large amount of behavioural, cognitive, and brain data about my participants allows me to find patterns of shared traits, regardless of diagnosis.

The statistical techniques I use have been described as 'data-driven,' or 'bottom-up.' That's because I start my research with very few assumptions – I know that current diagnostic categories don't describe a child's traits very accurately, so I prefer to use cognitive and behavioural questionnaires that do. By looking for robust patterns in my data, I can define new categories that capture the reality of developmental diversity in a more meaningful way.

I began my most recent project with a simple question: do inattention and hyperactivity present themselves separately – with some children only being hyperactive, and others only being inattentive – or do they tend to co-occur? To figure this out, I used a statistical technique called factor analysis. My tests revealed that, more often than not, inattention and hyperactivity go hand-in-hand. Although psychiatrists distinguish between these two behavioural traits, they're highly related, and occur in children regardless of their diagnosis.

Then, I wanted to know more about the brains of children who are particularly inattentive and hyperactive. Specifically, I wanted to find out whether there are different types of brains that produce these behavioural traits – after all, inattention and hyperactivity are common to many developmental conditions.

For this analysis, I took data from MRI brain scans and fed it into clustering algorithm. Clustering methods mimic what humans do when we categorise things in our homes – they detect similarities and group things accordingly. My analysis detected two different 'brain types' across inattentive and hyperactive children. The children belonging to these two groups are neurologically different, but they share the same levels of inattention and hyperactivity. Further tests revealed that the groups don't differ on age, gender, or any of the other twelve behavioural questionnaires we gave to them.

The two groups do, however, differ in one major way: one group performs worse on tests of cognitive ability. Additionally, children from the lower-performing group have fewer

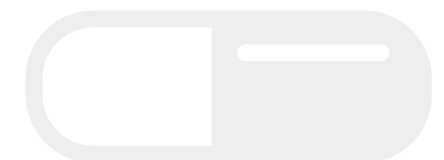
communication pathways in brain areas responsible for logical reasoning.

By applying these methods in a large and diverse sample, I was able to find two new categories of children with high inattention and hyperactivity. One had lower cognitive test scores and less efficient brain wiring. My analyses didn't reveal groupings similar to those in diagnostic manuals. Instead, I found that cognitive ability and brain structure, rather than traditional diagnostic categories, differentiate these children.

Many of the children I work with struggle at school – not only because they have learning difficulties, but because their diagnoses don't capture the full complexity of their needs. As a result, their parents and teachers struggle to find the right ways to support them. When categories fail, many children are deprived of their chance to learn and flourish.

Fortunately, it doesn't have to be this way. By recruiting big samples, collecting different types of data, and staying open-minded, we can change our diagnostic categories so that they reflect reality.

In the future, when a child struggles at school, she won't have to carry a label that barely represents her experiences. With a diagnosis that accurately describes her needs, she'll be empowered to do well at school by teachers who understand how to help.





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## THE JUDGING PANEL

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**SAMIRA AHMED**  
Journalist and  
Broadcaster



**DR JENNIFER ANDERSON**  
MRC Head of Training and Careers  
and Chair of Panel



**DR ROGER HIGHFIELD**  
Science Director of the  
Science Museum Group



**ZARA HUSSAN**  
Student and ABSW  
Young Science Writer  
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**ANDY RIDGWAY**  
Journalist and Senior Lecturer  
in Science Communication at  
UWE Bristol



**IAN TUCKER**  
Science and Technology  
Editor of The Observer



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