

Institute of Immunity and Transplantation Pears Building



“It is exciting to lead a biomedical research institute focused on improving health and developing new therapies that exploit the natural power of the immune system.”

Professor Hans Stauss, Director

UCL Institute of Immunity and Transplantation (IIT)



The IIT will be a world-class centre of excellence dedicated to the study of the human immune system. While the immune system plays an important role in protecting people against infection by viruses and bacteria, it is also critical in maintaining lifelong health. At the IIT we aim to discover how defects of the immune system lead to disease and to use this knowledge to develop new forms of immunotherapy. These biological therapies will exploit the natural power of the immune system to restore and maintain health.

The IIT will bring together scientists, clinicians, nurses and patients to gain novel insights into the underlying causes of disease. It will combine the clinical excellence of the Royal Free London's hospitals and the research excellence of scientists at UCL in the most modern facilities in the new Pears Building.

My own research has focused on the study of the immune system in cancer patients. In the past 10 years cancer immunology has made tremendous progress and the development of immunotherapy has revolutionised the treatment of certain types of cancer. We now have the exciting opportunity to learn from the progress in cancer therapy and develop new immunity-based treatments for chronic infection, autoimmunity and for patients after transplantation. IIT research will provide our patients with cutting-edge therapies that may not be available elsewhere.

I am grateful to all the donors who have supported the IIT and the construction of the Pears Building. We have received generous donations from the Pears Foundation, the Wolfson Foundation, the N. Sethia Foundation, Fidelity Foundation and the Catherine Cookson Charitable Trust. We look forward to working closely with our local community to make the IIT a world leader in human immunology and immunotherapy.

Professor Hans Stauss, Director





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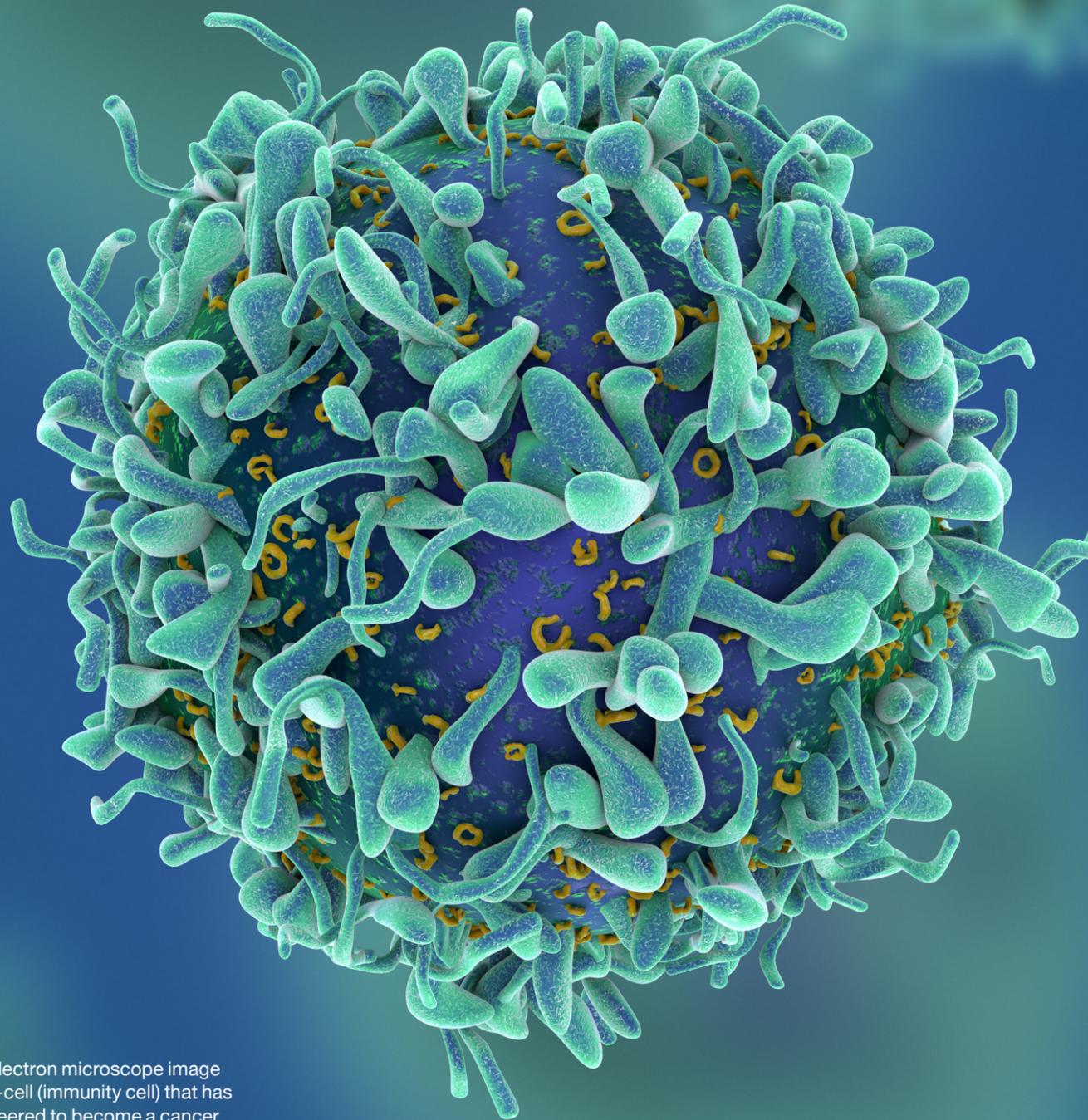
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“At last, somewhere for people with rare conditions like myself. This ground-breaking facility will improve and save lives.”

Kim Fligelstone, patient



Scanning electron microscope image of 'super' T-cell (immunity cell) that has been engineered to become a cancer fighting T-cell.

What is the IIT?

Who are we?

We are one of only a few centres in the world dedicated to collaborative immunity research, bringing together teams of more than 200 experts and more than 10,000 patients.



Researchers



Doctors



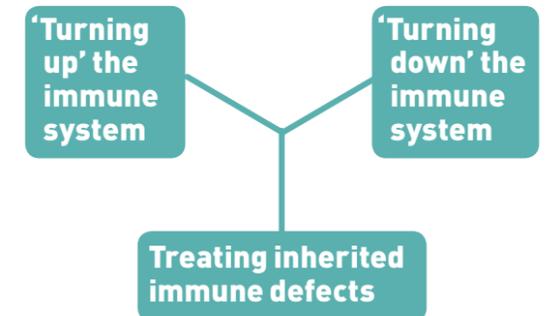
Nurses



Patients

What is the purpose of our research?

Our goal is to improve human health by understanding the human immune system: how it works, why it can stop working properly and how can it be fixed. We pursue three distinct but related goals:



How do we work?

We do translational research. This means that our research teams take information discovered in the laboratory and use it to work with clinical colleagues – doctors, nurses and other healthcare professionals – to find new therapies for patients.



Why is our work important?

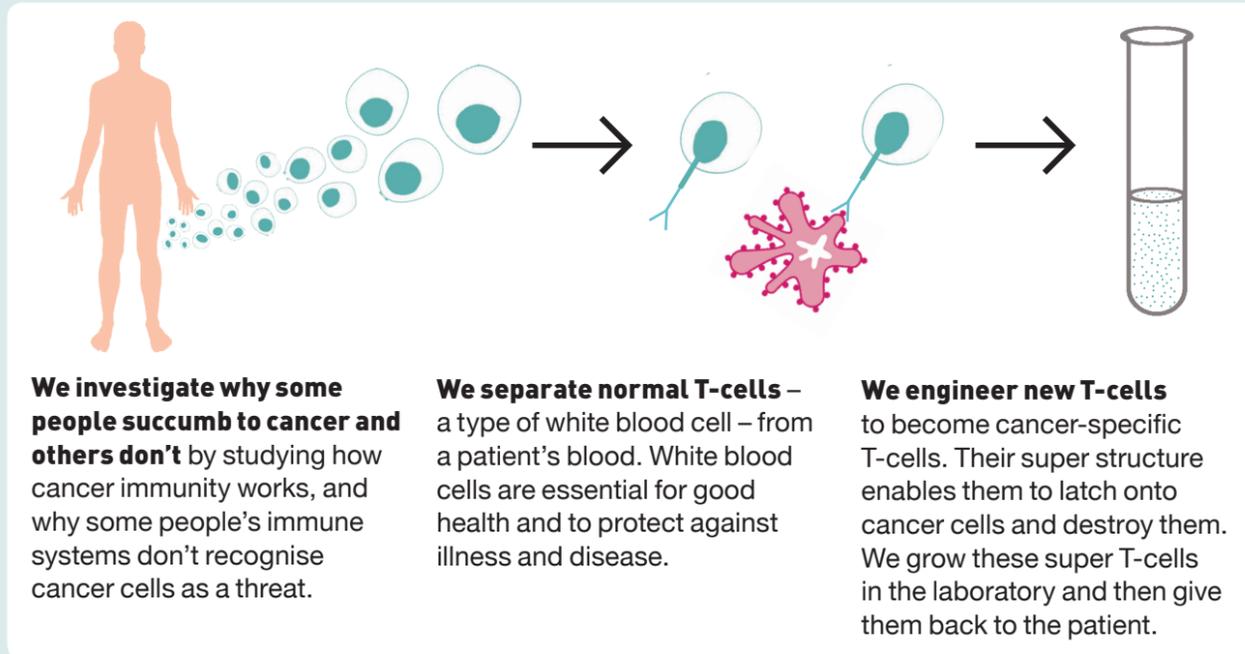
Our research enables us to develop new vaccines and to use cell and gene therapy to treat many serious immune-related diseases. It also enables us to improve the long-term outlook for transplant patients.

- Cancer
- HIV
- Hepatitis B
- Haemophilia
- Liver disease
- Leukaemia
- Kidney cancer
- Diabetes
- Organ regeneration

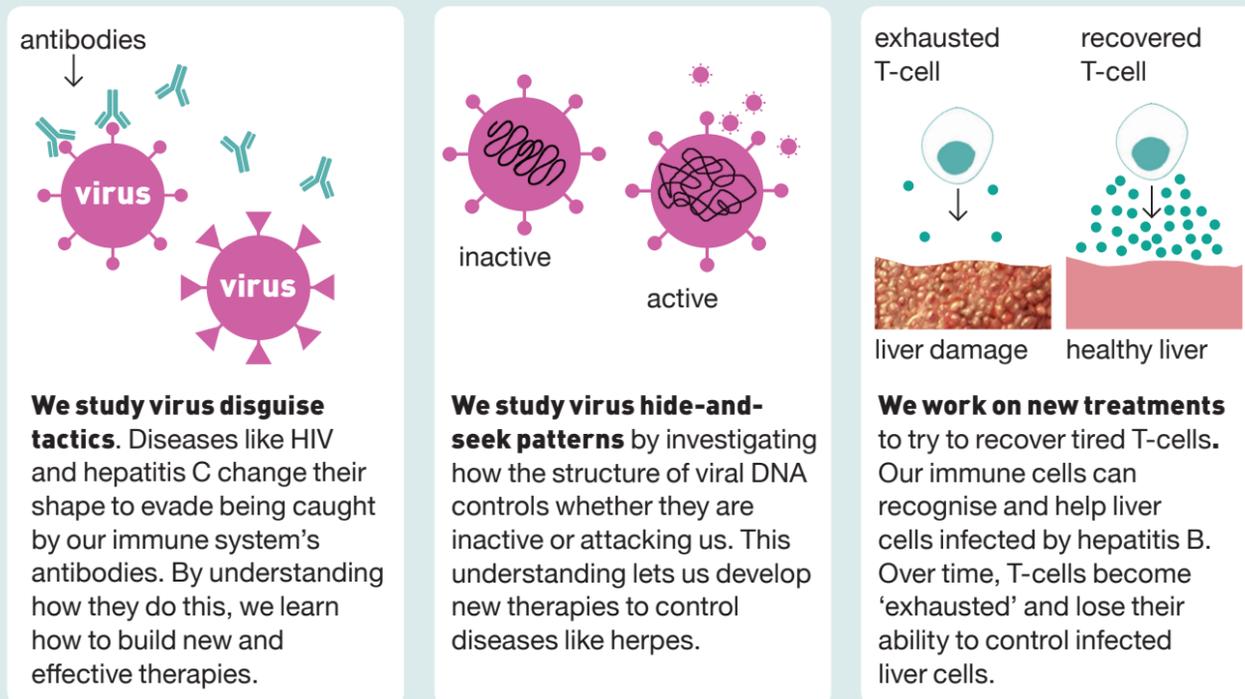
'Turning up' the immune system

We research two programmes within this area of our work:

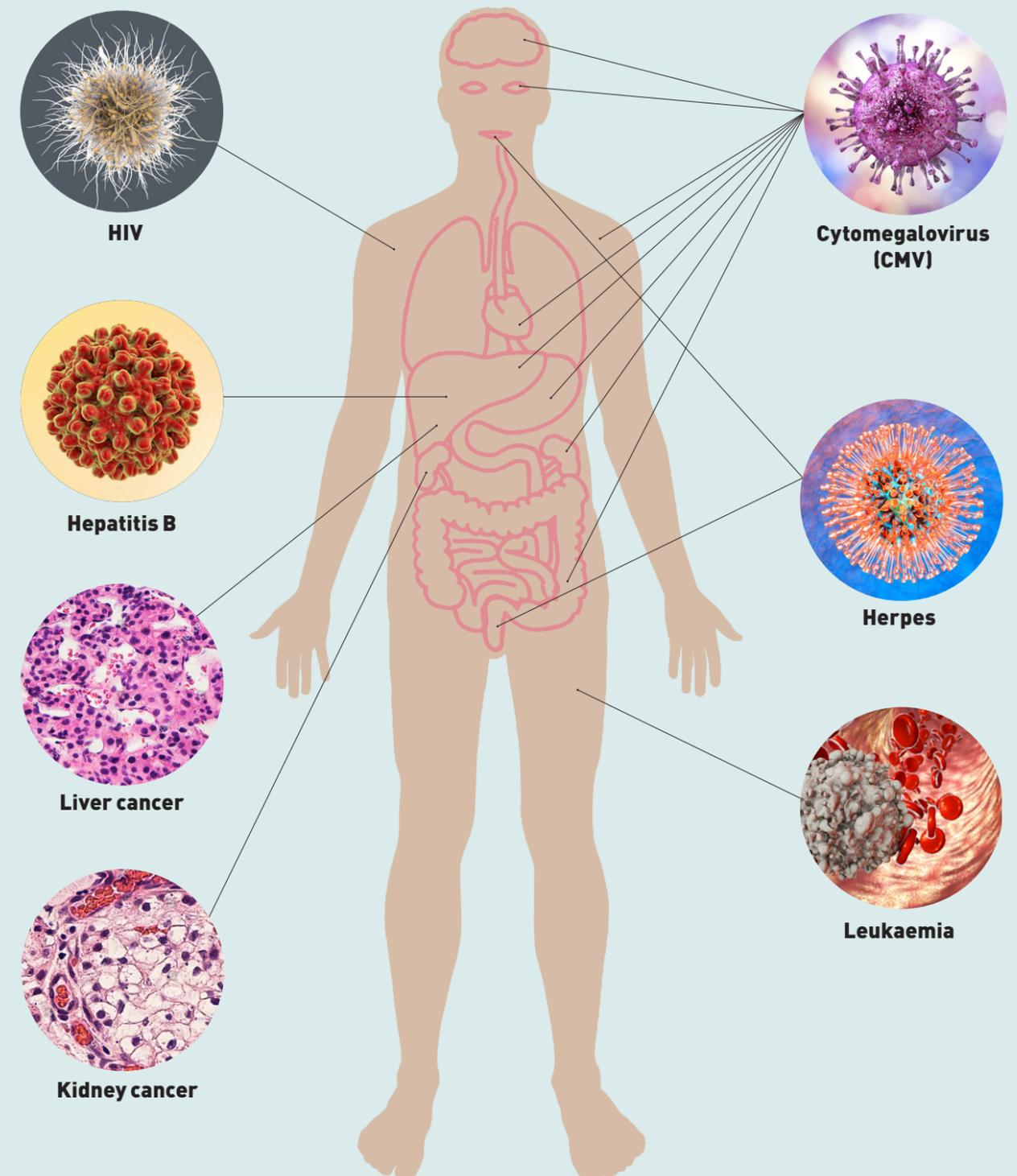
Using the immune system to treat cancer



Developing immunity against viral infections



Sometimes a person's immune system fails to respond to a threat from, for example, a bacteria, virus or fungus. We look at how the immune system works so we can develop treatments to boost the immune response and avoid the need for long-term medication for many serious conditions.



Combating hepatitis B

Hepatitis B is one of the top ten killer diseases in the world. A preventative vaccine exists, but this doesn't benefit the millions of people who already have the infection. Many of them will die from liver cirrhosis and cancer if left untreated. **Professor Mala Maini** tells us how she and her team are tackling this deadly infection.



Mala Maini, Professor of Viral Immunology

and providing new insights into local immune responses that could be applied to the treatment of other liver diseases.

How we work

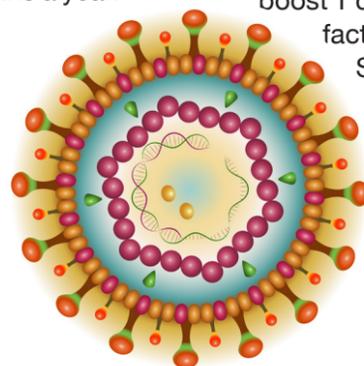
New research techniques allow us to use small numbers of cells from patients' blood and liver to study their immune response in great detail. We work closely with hepatologists (liver physicians), oncologists (doctors specialised in diagnosing and treating cancer) and liver surgeons at the Royal Free Hospital. I also work as a consultant physician in a viral hepatitis clinic. My research has always been closely informed by patients I see in my clinics and the samples they generously donate for our work.

Our progress so far

In patients with persistent infections like hepatitis B, the specific T cells become 'exhausted' - so stop working properly. By defining the mechanisms driving this, we try to identify ways to restore effective T-cell control. One of the ways we can do this is by protecting T-cells from regulatory cells and signals that inhibit them. We can also boost T cells by giving them additional growth factors or nutrients they depend on.

Saving T-cells in these ways supports their recovery and their response to hepatitis B. This knowledge has informed the development of new therapies for liver diseases like hepatitis B.

The infectious hepatitis B virus has a spherical, double-shelled structure



What is hepatitis B?

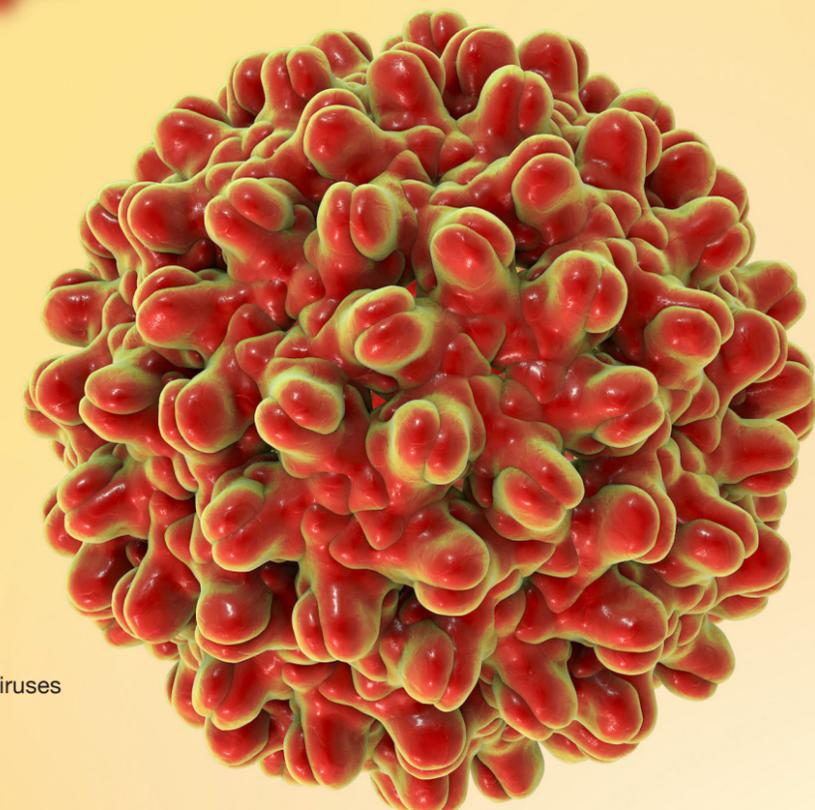
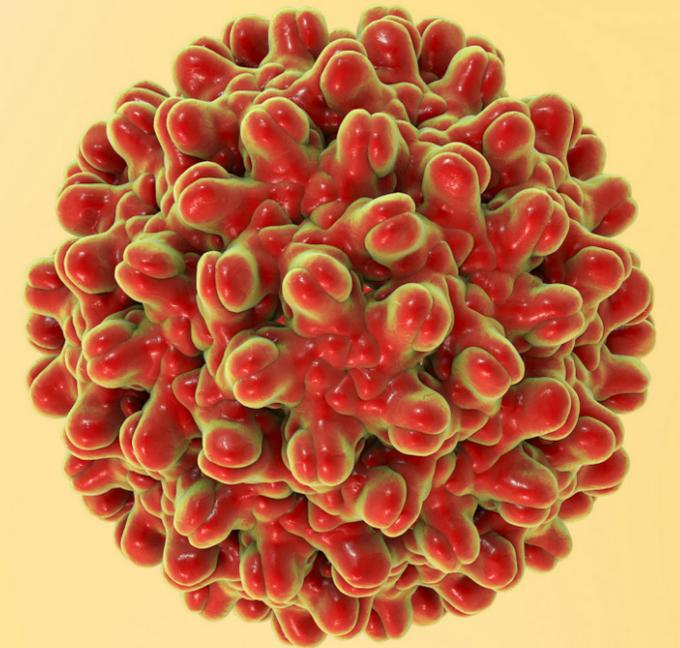
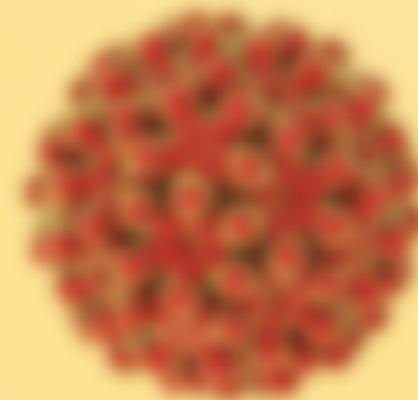
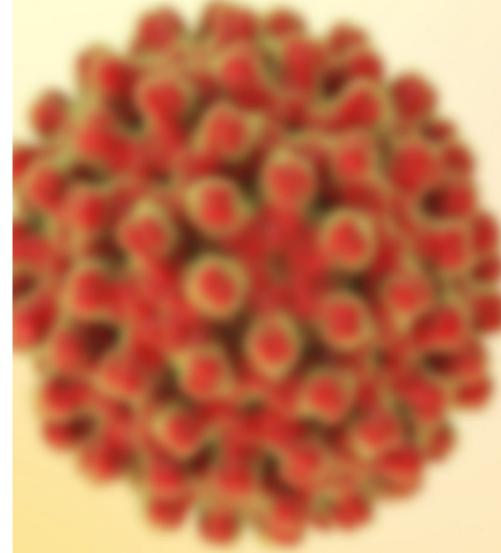
Hepatitis B is an infection of the liver caused by a virus that is spread through blood and body fluids. Most adults infected with hepatitis B fully recover within a few months and keep traces of the virus under efficient lifelong control through their immune responses. But for those who can't control the virus, there's a risk of developing life-threatening problems such as scarring of the liver (cirrhosis) or liver cancer. One third of the world's population has been infected with hepatitis B. Around 240 million people have a long-term infection, causing over 700,000 deaths a year.

Our goal

Our goal is to develop new therapies for treating hepatitis B by harnessing the natural capacity of the body's immune response to control this virus. Our work is also directed towards advancing immunotherapy for liver cancer

"The IIT has saved my life and other patients like me by ensuring that the treatment and therapy match the patient. The research has enabled some 'incurable' conditions to be cured."

Jose Drabwell, patient



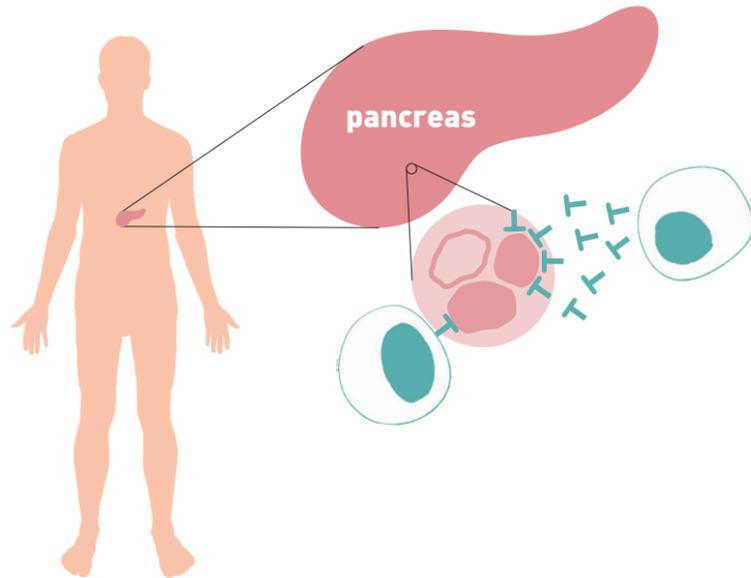
Hepatitis B viruses

'Turning down' the immune system

We research two programmes within this area of our work:

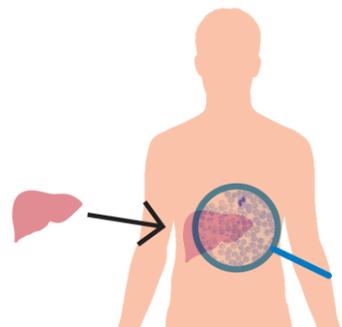
Immune regulation

We study how immune cells interact with each other and our body. An unfortunate side effect of immune protection can be autoimmunity, which occurs when our immune system attacks our healthy cells. We study how these kinds of immune cells are made, how they work and how they can interact with each other. This research helps us to correct defective regulation and control autoimmune diseases such as type 1 diabetes, scleroderma and multiple sclerosis.

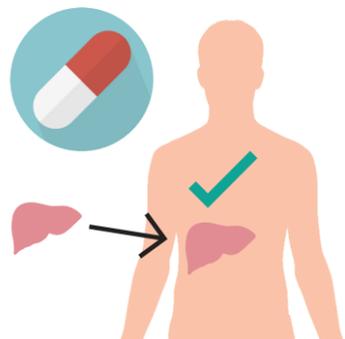


Transplantation and tissue engineering

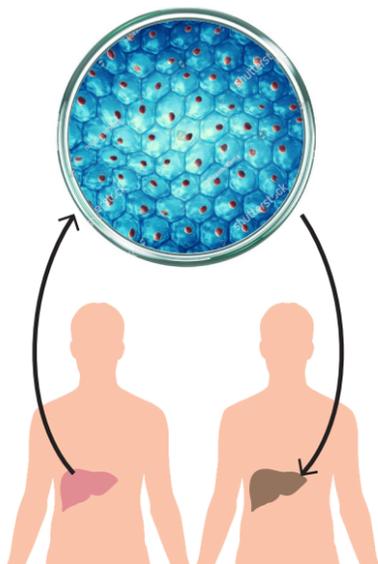
We work on improving transplantation by studying how the immune system responds in liver and kidney transplant patients. Thousands of UK citizens are waiting for an organ transplant. Even after a successful transplant, the body can attack the donor organ.



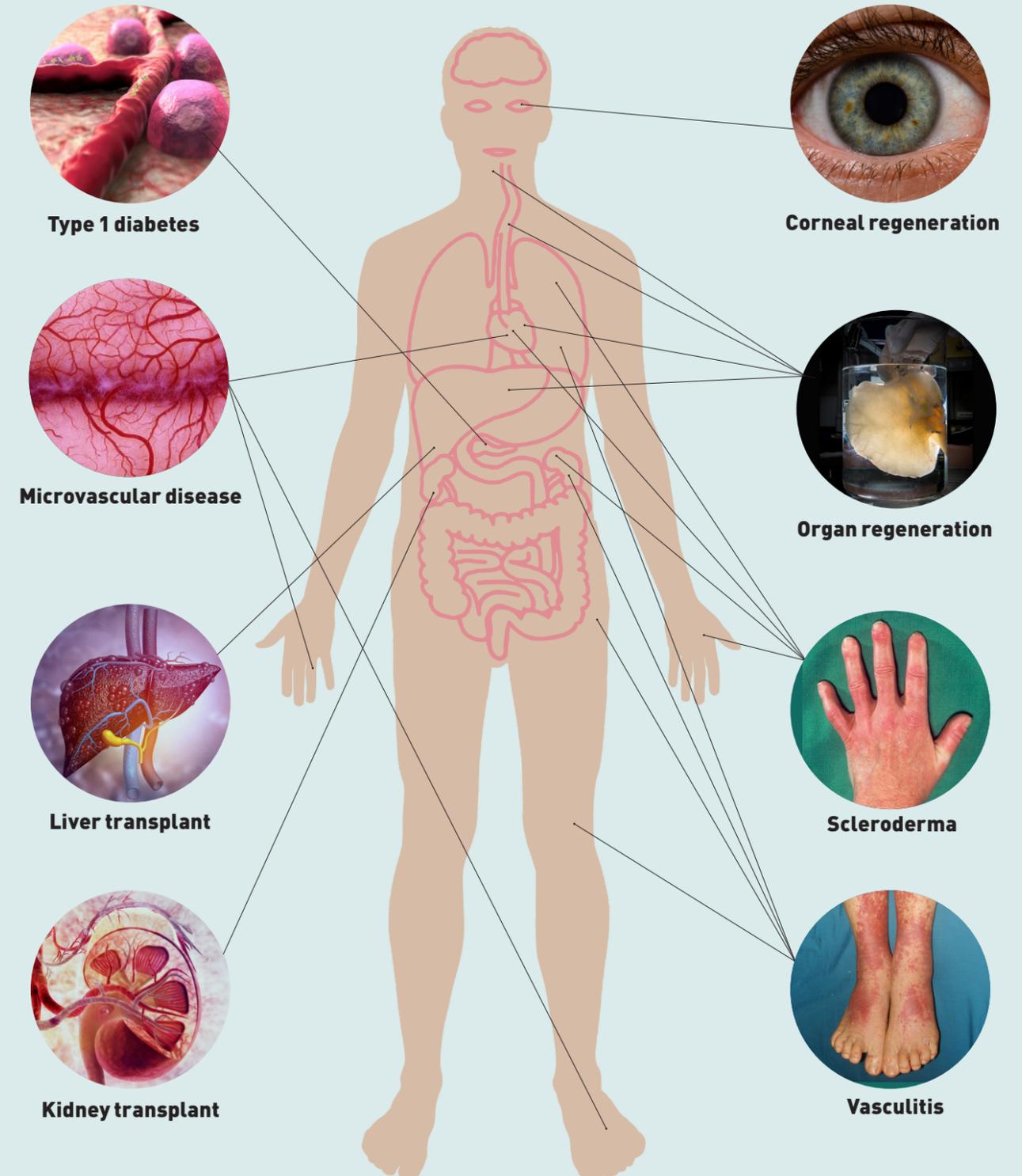
We tailor medication to reduce the chances of organ rejection. After a transplant, a patient needs to take immunosuppressive medicines that control the immune system to prevent the new organ from being rejected.



We grow new tissues in our labs using human cells, which can replace damaged organs.



Sometimes a person's immune system can attack their own body. These autoimmune diseases include type 1 diabetes, rheumatoid arthritis and multiple sclerosis and can lead to rejection of transplanted organs.



Tackling type 1 diabetes

Around 400,000 people in the UK have type 1 diabetes, 30,000 are children. Overall the number of people in the UK affected by type 1 diabetes is increasing by 4% a year. **Professor Lucy Walker** explains how she and her team are working to prevent this increasingly prevalent disease.



Lucy Walker, Professor of Immune Regulation

How we work

Currently when therapies are tried in people with type 1 diabetes, results become clear only after about a year. However, by looking at biological markers in the immune cells discovered in this research, we can tell far sooner which therapies look promising. Drug trials are usually very long-term and expensive, but by finding a way to monitor the blood's response, new medicines could be developed faster.

Our progress so far

My team has worked closely with the clinical diabetes team and with patients to study immune cells in the blood. We have discovered immune markers in the blood that appear even before a person develops type 1 diabetes. This may help us and other researchers predict who will develop type 1 diabetes before any symptoms occur, and could lead to medicines being given early on in a person's life before any pancreas cells have been destroyed.

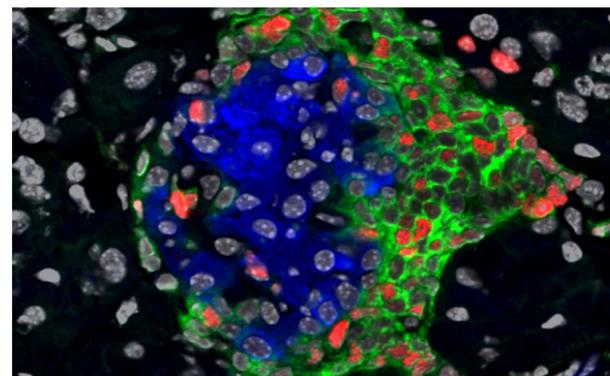
What is type 1 diabetes?

Type 1 diabetes is a lifelong condition caused by the body's immune system attacking the pancreas, which affects insulin production. We need insulin to regulate the blood sugar level in our bodies. Typically, onset of diabetes is between the ages of 9 and 14 years, but more children under 5 are now being diagnosed.

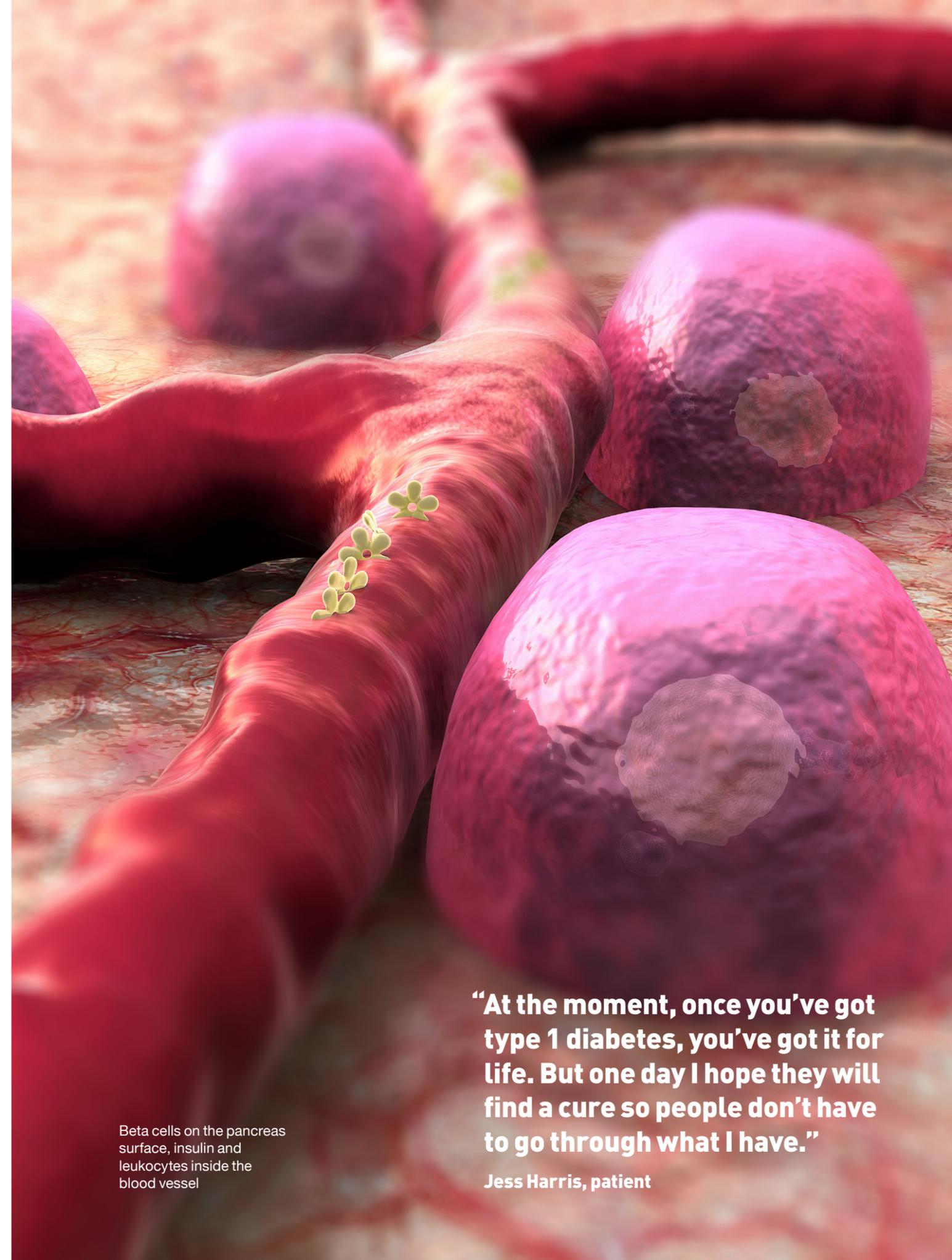
Our goal

As a result of our research, important information is now known about the immune cells that cause type 1 diabetes. My team has two strands of work:

- the development of new therapies that interfere with this type of immune response
- the monitoring of the effectiveness of these therapies



Immune cells (green) infiltrating a pancreatic islet (blue) in autoimmune diabetes



Beta cells on the pancreas surface, insulin and leukocytes inside the blood vessel

“At the moment, once you’ve got type 1 diabetes, you’ve got it for life. But one day I hope they will find a cure so people don’t have to go through what I have.”

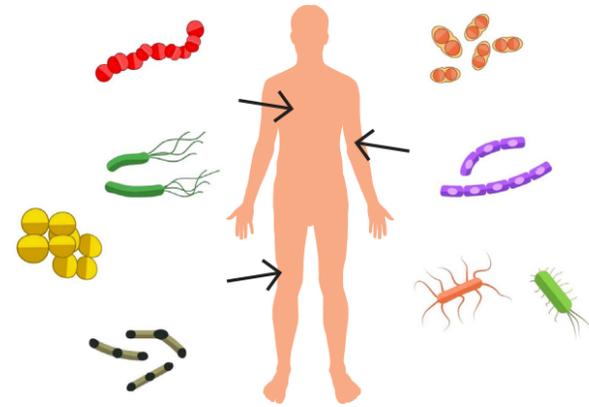
Jess Harris, patient

Treating inherited immune defects

Primary immunodeficiencies (PIDs) are a group of more than 350 different inherited conditions in which faulty genes affect how the body's immune system works. As genetic screening improves, we hope to improve current stem cell transplant therapies and develop less invasive gene therapy and drug treatments.

What are primary immunodeficiencies?

Some people are born with intrinsic defects in their immune system. This is called primary immunodeficiency, or PID. Those affected by PID have a reduced natural defence against bacteria, fungi and viruses.



How are people affected?

Patients with PID have more infections, take longer to recover from them and often get them again. They can have a higher risk of developing blood cancers. In addition, the poor regulation of the immune system can also lead to overstimulation, resulting in chronic inflammation.



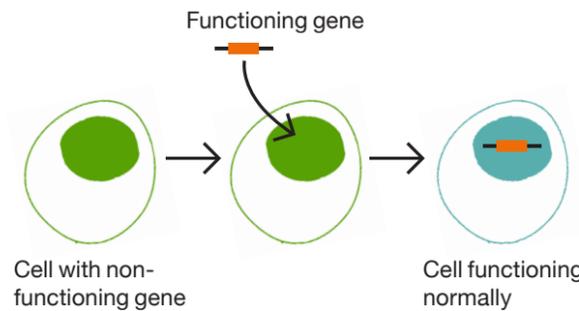
Severe chicken pox

How are people with PID treated?

Treatment development is a balancing act between managing the immune system so that it is not over-stimulated (which can cause autoimmunity and chronic inflammation) and not under-stimulated (which can lead to infections and malignancy).



Treatment may involve stem cell transplantation – in effect a bone marrow transplant – or gene therapy.



Treatment development

We know there are more than 300 genes that can cause immune deficiency. We are trying to understand how those genetic mistakes in patients affect immune cell function. Diagnosis is key in enabling treatment to be tailored to the individual. At present, only people who develop immune deficiency in childhood can be properly diagnosed.

Having access to patient cells is a crucial part of developing new therapies. It means that scientists can design experiments to test the function of specific genes. This is leading to the identification of new genes as causes of immune deficiencies in adults. As research into genomic analysis continues, it is thought that more genes causing PID will be discovered.



IIT starts operating in limited space within the Royal Free Hospital

First gene therapy in the world for an adult with PID performed at the IIT

IIT moves into the Pears Building

2013

2015

2020

2025

2030

2035

2040

Stem cell transplant treatment

Gene therapies

Nearly 300 genes identified which can cause PID

More genes predicted to be identified

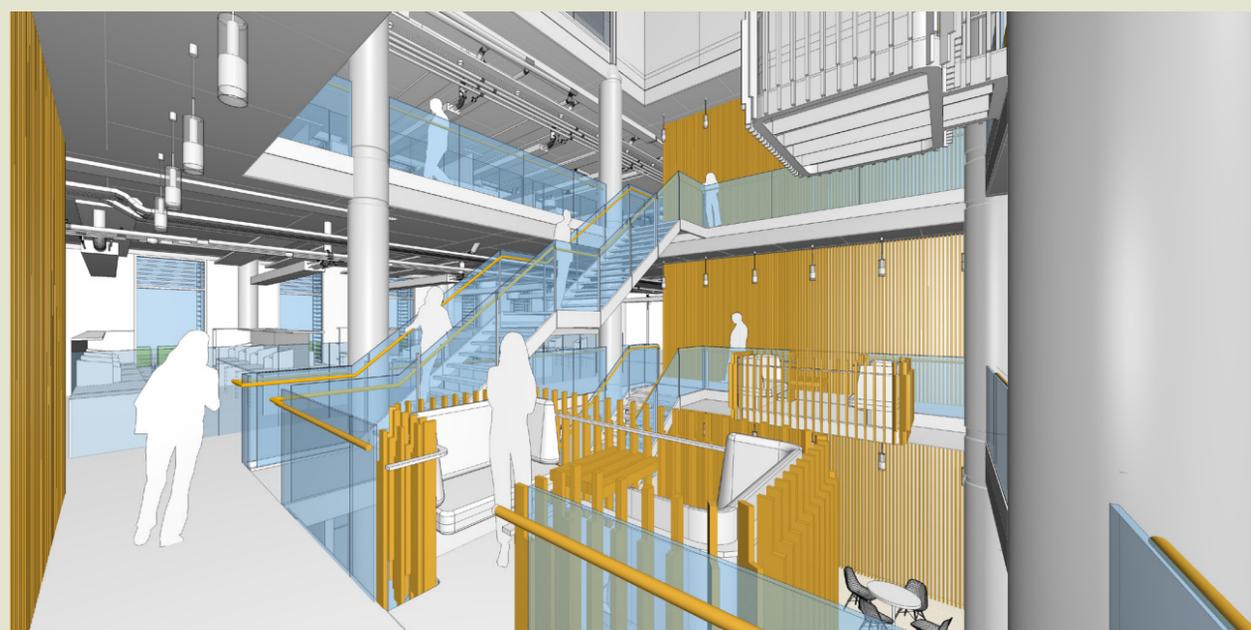
Genetic screening research

New drug treatments

Treating inherited immune defects

A translational research story

The key to success in treating patients who develop immune deficiency is the ability of clinicians to work alongside scientists who have insights into particular parts of the immune system. This is exactly what happens at the IIT, and what our new Pears Building is designed to facilitate.



Curing immune deficiency

I was a music student at Leeds when I was first admitted to the IIT. I had been unwell for several years and, unusually, had developed lymph gland cancer, not once but twice. My immune system was so weak that it made me very prone to infections and increased the risk of my lymph glands becoming cancerous. The IIT team found that I had a gene mutation (in a gene called CD27) that led to my white cells not working properly. Doctors at the IIT discussed with me the possibility of correcting my genetic disease with a bone marrow transplant. They quickly identified a donor from the Anthony Nolan register and I was able to undergo a bone



Patient Jacob Cracknell

marrow transplant in 2015. Three years later, I am completely well. I have just started the final year of my music course and am now looking forward to the rest of my life.

Bone marrow transplantation for PID

As bone marrow transplant (BMT) consultants and scientists at UCL, we have been working with our colleagues at the IIT to develop a bespoke clinical and scientific transplant programme for adult primary immunodeficiency (PID) patients. As these diseases are so rare, the complex, novel transplants are performed only after detailed consideration. We also carry out stem cell gene therapy to correct serious defects in immunity, previously used only in babies and children. We use detailed genomic analysis, studies of the mechanisms of the immune system and combine these with comprehensive clinical data to inform



Professors Ronjon Chakraverty and Emma Morris

decisions about patient care. By bringing together researchers and the clinical teams in one space, the Pears Building accelerates and supports these critical interactions.

Gene therapy for PID

I run a research team at the IIT focusing on understanding the underlying causes of PID. Our team focuses on improving early diagnosis and treatment and works closely with the largest clinical service for people with PID in the UK. We have treated the largest group in the world of adult PID patients with allogeneic BMT and are the only team to have treated two older adults with gene therapy for PID.



Dr Siobhan Burns

In collaboration with Professor Emma Morris I have recently expanded my research team and together we are developing new gene editing approaches to treat PID for patients not eligible for BMT. The new Pears Building will provide

the space and equipment to attract world class scientists to join our team and help our patients to access potentially curable cutting edge therapies.

Transplant immunology

My lab focuses on understanding the key switches that regulate whether or not we make an immune response. If we make an immune response too easily we risk triggering autoimmune diseases such as rheumatoid arthritis or type 1 diabetes. My team works on a protein called CTLA-4 which acts like a tiny vacuum cleaner, removing proteins that stimulate the immune system. If CTLA-4 is faulty the immune system gets overactive. At the IIT we work with doctors who treat patients with defects in the CTLA-4 gene. By applying basic scientific knowledge we're able to help diagnose the problems. Studying patients has also helped us to learn more about how this

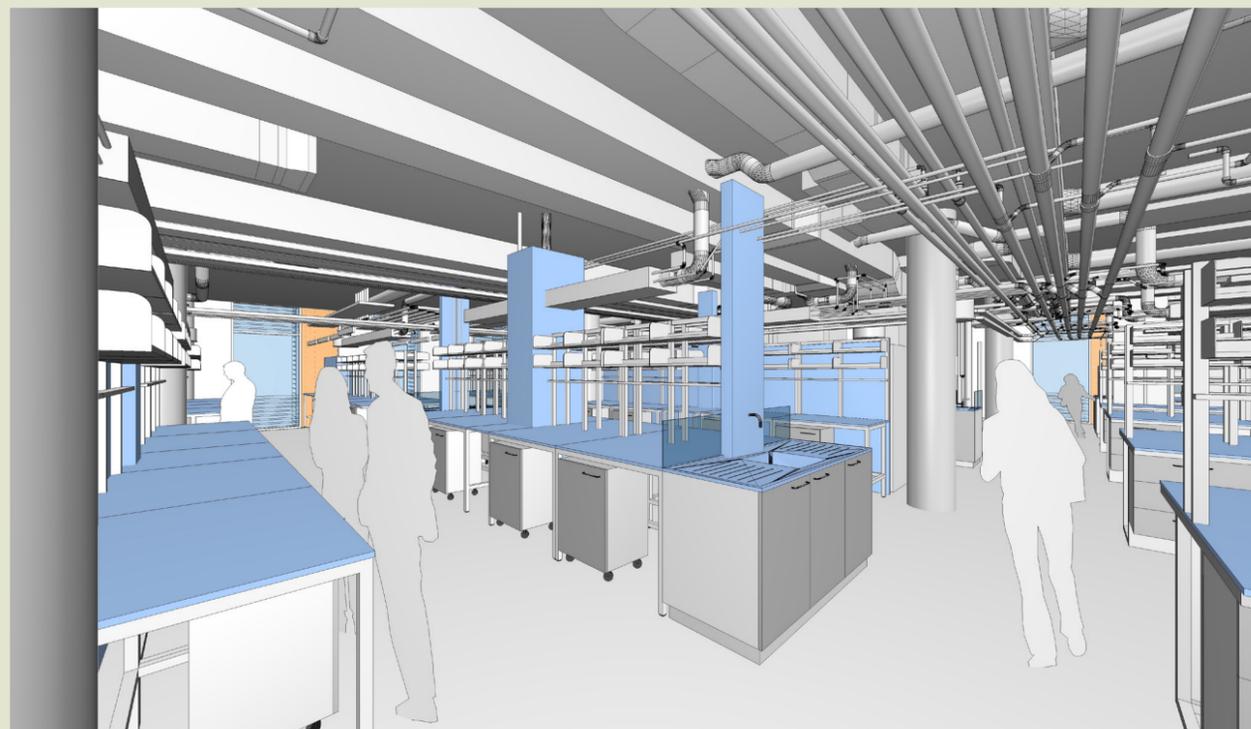


Professor David Sansom

key system works. The new Pears Building will provide outstanding new laboratories for our work and improve the opportunities for us to learn about how the immune system is controlled.

Treating inherited bleeding disorders

A translational research story



Haemophilia B is a genetic disorder caused by a lack of the clotting factor IX. It can cause progressive damage, arthritis and pain, and it can be fatal. There is an effective injection treatment but it must be given every few days for life and is expensive. The new Pears Building will facilitate our research into finding a cure which would avoid the need for this treatment.

World-leading research

The team I lead is at the forefront of worldwide research through our work on gene therapy for monogenetic disorders, ie caused by a single gene. We have had great success with haemophilia B and haemophilia A. Our current plan is to extend the approach to more common diseases such as type 1 diabetes.

We are also working on immunotherapy. We are aiming to harness the same cell technology that has proved so successful in treating leukaemia in children, to find new treatments for solid tumours like pancreatic cancer whose outcomes have changed little in the past 50 years. Another novel type of therapy involves the creation of specific molecules to fight not only cancer but non-malignant conditions



Professor Amit Nathwani

such as chronic infections like tuberculosis and clostridium difficile, caused by antibiotic resistance. When we move to the Pears Building we will be able to collaborate with and harness the expertise of immunologists working on different aspects of what we do.

Living with haemophilia

I'm one of several members of my family with haemophilia B, which means that my blood doesn't clot properly – even a minor bleed could be a disaster. It's like being an insulin-dependent diabetic, having to inject yourself regularly with clotting factor.

As soon as my doctor in South Africa, where I live, started talking about having gene therapy at the Royal Free, I started researching it. I'm medically trained, as a perfusionist for heart/lung machines, so that helped. There were reservations because of all the travelling involved but I knew I wanted to do this, not only for me but also to help research so that other haemophiliacs can benefit from it.

From the day I was treated, I've had no side effects or bleeds. Of course the joints are already damaged, but it's a wonderful relief not to have to worry about injuries. The other day



Patient Martin Lampacher

someone accidentally smashed a door into my arm. Before the treatment, I'd have had to have an infusion and get myself checked over. I didn't even bother to look at my arm and the next day there was no mark.

I was obviously worried about my sons, but they're clear, so in our immediate family the disease will stop with me.

Hijacking a virus

Since my team isolated the genetic cause of haemophilia in the 1980s we've known that gene therapy could be the answer. But it took a while to find the perfect way of transferring the genetic material into the patient. The best method we've found so far is to use a modified virus. We replace its genetic information (which just says how to make more virus) with the genetic information to make the missing clotting factor, be it factor VIII or factor IX, depending on the type of haemophilia. The first successful study was our own in 2010. This effectively turns a severe case of haemophilia to a mild case, making it more manageable. We're now working on a method to achieve normal or near-normal levels of clotting factor.

One challenge is that because we use a virus, some patients' systems recognise it and create



Professor Ted Tuddenham

an immune response. So we need to collaborate with immunologists so that more patients can benefit from the treatment. We're greatly looking forward to the move to the Pears Building. We think more space for people and their equipment will enhance relationships and interactions, making it easier to cross-fertilise ideas, techniques and methods and improve results.

**Stories from our
patients and clinicians**



The story of Noam Tamir's life saving liver donation from his son



Patient: Noam Tamir with his son Jonny



Consultant hepatologist: Prof Douglas Thorburn

NT: After four years of itchiness I developed swelling in my legs. A blood test showed I had end-stage liver failure. It was a terrible shock.

DT: When I first met Noam he had very advanced liver disease (cirrhosis) caused by the rare immune mediated liver disease primary sclerosing cholangitis (PSC). Unfortunately there are no medical treatments that stop the progression of PSC even if it is diagnosed early. We are working hard at the IIT to study the basic mechanisms of the disease and to help design and undertake clinical trials of new treatments for people who have it.

NT: My name was added to the system for deciding who gets a liver transplant. I have one of the less common blood groups so the question of a live donation from a member of the family was raised. It was possible for my son Jonny to donate 61% of his liver to me and in a relatively short time, if all went well, we were told we would both have normal-sized livers again. I was not at all keen for him to do this for me, but my son was determined. The 12-and-a-half hour surgery went well. Within a month it was fully functioning and within three months it had grown to its original size – it's an amazing organ. I have to take immunosuppressive drugs for life and there isn't yet enough research into whether this can be reduced when the

donation is from a family member. But although I had side effects at first they hardly affect me now. They keep an eye on me though. And I'm now walking 11km a day!

Within three months my liver had grown to its original size – it's an amazing organ

DT: Over 1,000 liver transplants are undertaken within the UK each year, but unfortunately almost 500 patients are on the waiting list and living donor liver transplants are undertaken only rarely. Noam was very fortunate to have a son willing and suitable to donate more than half his liver to him. At the IIT we are exploring alternative means of engineering livers for transplanting into patients. Our hope is that in time these treatments may provide alternative means of liver transplantation.

NT: I can't thank the team who looked after me enough. They know what matters to patients and how to talk to them. I'm sure that in five or 10 years' time, a patient like myself won't have to take drugs to suppress the immune system. Instead, scientists will have worked out how to regenerate organs from stem cells.

The 'game changing' treatments for Fabry disease giving Tony Haymes back his lifestyle



Patient: Tony Haymes



Haematology specialist, Dr Derralyn Hughes

TH: About three years ago I had a serious heart complaint out of the blue. I was rushed to hospital where the doctors suspected I had blocked arteries. After two years and genetic testing, they discovered I had Fabry disease. I was lucky enough to be referred to the IIT at the Royal Free, where I met Dr Derralynn Hughes. She started me on enzyme replacement therapy. Since then my energy levels have increased.

DH: Fabry disease is a rare, inherited metabolic disorder. It results from a deficiency in an enzyme called alpha-galactosidase-A which causes a build-up of a fatty substance within different cells and organs in the body. It can lead to a range of serious symptoms including kidney failure and heart problems. Patients like Tony have a late-onset type of Fabry disease which results predominantly in problems with the heart. Their specific mutation is found more in the UK than anywhere in the world.

TH: It was a big shock to learn that I had a heart problem and then discover I had such a rare condition. I'd always been very fit. Having the implantable cardiac defibrillator meant that I could have a near-normal lifestyle, but I had to be careful in some situations and near certain machines or equipment.

DH: Enzyme replacement therapy was developed as a treatment for Fabry disease about 18 years ago. In the UK we're able to give enzyme replacement therapy in the home. Elsewhere, patients have to come into a hospital or centre every two weeks for an infusion. Around 12 years ago we started working on alternative treatments, and since June last year we've been able to give oral treatment to patients who have the appropriate mutation. For patients like Tony with late-onset Fabry there has been a particularly high acceptance of this treatment, allowing them to convert from having the intravenous treatment.

I will simply have to take one pill every other day, rather than lengthy, fortnightly infusions.

TH: Dr Hughes and the IIT team are about to start me on Migalastat, a new oral treatment that is better suited to my condition as it increases levels of my own enzyme. Best of all, it comes in a pill format. So I will simply have to take one pill every other day, rather than lengthy fortnightly infusions. This is a real game-changer!

Jess Harris' 18-year ordeal with type 1 diabetes and her hopes for a future cure



Patient: Jess Harris

JH: I was diagnosed with type 1 diabetes aged 12. The disease has damaged my kidneys so I have dialysis twice a day, which can take up to an hour each time. I frequently find myself in hospital following a 'hypo' – hypoglycaemia or low blood glucose. I've had some horrendous hypos and find myself in hospital every four to six weeks with something, often related to my renal problems.

MR: 50% of patients who have type 1 diabetes are diagnosed as children and adolescents. People with the disease have to manage their condition 24 /7. This is a big responsibility which is especially difficult to navigate during adolescence. For some patients the teenage years are a time when their diabetes is poorly controlled and they fail to meet required targets. This can lead to long-term damage to the body – especially to the kidneys – which can be life changing.

JH: My condition has dominated every day since I was diagnosed. You never get a day off or a weekend break. There's always something to monitor, whether it's glucose levels, insulin or what you're eating. But I work when I'm well enough. I also find time to campaign for the 'opt-out' organ donation scheme under which everyone would be assumed to have consented to be a donor unless they have said they don't want to in advance.



Endocrinology consultant: Dr Miranda Rosenthal

MR: Managing diabetes well and effectively requires vigilance and integration of information about food, exercise and glucose levels at all times. It also has to be prioritised over all other aspects of daily life. This can be exhausting and challenging, which is why Professor Lucy Walker's research at the IIT is so vital (see p. 12).

I know that research holds the long-term answer

JH: My main hope lies in a kidney and pancreas transplant. The average wait is 12–18 months and I've been on the list for more than a year. I've been called three times but there was always a problem. But I know that research holds the long-term answer. At the moment, once you've got it, you've got it for life. But one day I hope they will find a cure so people don't have to go through what I have.

MR: Working at the clinical end of diabetes, I help run a clinic of over 1,000 adult patients with type 1 diabetes. Our aim is to offer research opportunities by recruiting patients into studies. There is huge enthusiasm by people like Jess among the type 1 community to be involved in the research that is looking at cures and mechanisms.

Andrew Quille's rare case of ANCA-associated vasculitis



Patient: Andrew Quille

AS: Andrew Quille realised something was wrong when first his hearing and then his legs developed problems and he started feeling very ill. He was referred to me at the Royal Free London and diagnosed with a type of vasculitis, a group of disorders which cause inflammation of the blood vessels. We treated Andrew with various drugs including steroids, a type of chemotherapy and immunosuppressants.

AQ: I was very ill and the outlook wasn't good. I ended up on dialysis because my kidneys weren't working properly and at one point it looked as if I might have to have an antibiotic which we knew I was allergic to.

AS: Autoimmune diseases, such as lupus or ANCA-associated vasculitis, are caused by imbalances in the immune system. Current treatment involves suppressing the immune system to improve symptoms and prevent organ failure, but this involves significant side effects and risk of relapse. Rarely, patients with these conditions can become 'cured', so that they can stop taking immunosuppressants and stay well. The way in which the immune system 'reboots' itself to normality is unclear.

AQ: After a number of weeks of treatment, I started to improve. Just over two years later I felt almost back to normal. I'm not receiving any treatment. I'm one of the lucky ones. I'm monitored regularly



Nephrology consultant: Professor Alan Salama

and I know it could come back, but I'm optimistic. Even if it does come back it could be quite a mild response.

We want to understand why the "reboot" doesn't happen to everyone

AS: We are working to understand which important biological pathways are involved in achieving this, why it doesn't happen in everyone and which drugs – or combination of drugs – help or may hinder this process. Knowing about the pathways involved may allow us to find other means of treatment, conventional drugs or novel cell-based therapies, not currently considered for the management of lupus or vasculitis, that could help more patients become symptom free and enable us to identify who is at risk of relapse so that we can customise treatment.

AQ: I am so grateful to Alan and his team for all they did and have been more than happy to agree to take part in a research programme to look at why my body might have responded to treatment the way it did. I'm delighted if this might help prevent others from having to go through what I did.

Thank you to our donors and supporters

Philanthropy is a powerful catalyst that has made the construction of the Pears Building and the development of the IIT possible. These world-class facilities will enable patient-focused research to advance faster and reach further than would otherwise be the case. They will bring together leading scientists, academic clinicians and clinical trials specialists to develop revolutionary treatments and therapies for patients with conditions such as diabetes, HIV and cancer.

We are enormously grateful to the individuals, trusts and foundations, charities and corporate partners who share our passion for harnessing the immune system to tackle the major health challenges that affect many millions of people around the world.

The Royal Free Charity and UCL have established partnerships with our supporters that are genuinely transformative, creating cutting edge infrastructure and new knowledge to develop better and more effective therapies, saving and improving lives.

Our thanks to:

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If you would like to learn more about our pioneering research and how to support our work and our facility please contact:

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or +44 (0)20 3108 3833

The Royal Free Charity at rf.fundraising@nhs.net
or +44 (0)20 7317 7774





In October 2018 members of our IIT team held a 'bottoming out' ceremony on the Pears Building construction site, during which we buried a time capsule in the foundations to be opened in 100 years' time. By this time we hope the research will have transformed lives. Two pupils from the Royal Free Hospital Children's School buried the capsule.

“I’m sure that in 5 or 10 years’ time, a patient like myself won’t have to take drugs to suppress the immune system”

Noam Tamir, patient



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