

EASD—

Novo Nordisk Foundation
Diabetes Prize For Excellence

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Professor
John Andrew Todd



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The EASD–Novo Nordisk Foundation Diabetes Prize for Excellence is being awarded to recognize outstanding research or technology contributions to the understanding of diabetes, its disease mechanisms or its complications.

The Prize is awarded annually to an internationally recognized researcher whose research may focus on prevention, treatment and/or basic research in physiological biochemistry. The research may also be clinically oriented.

In addition, the Prize may be awarded for the “discovery of the decade” within diabetes research.

Established in 2015, the Prize is awarded in collaboration between the European Association for the Study of Diabetes (EASD) and the Novo

Nordisk Foundation. It is accompanied by DKK 6 million – of which DKK 1 million is a personal award and the remaining DKK 5 million is for research purposes.

A special prize committee appointed by the EASD decides the Prize recipient, and the Novo Nordisk Foundation donates the funds accompanying the Prize. Employees of universities, hospitals or other non-profit institutions are considered for the Prize.

Candidates must be highly renowned and may be of any nationality. The Prize is conferred at the EASD Annual Meeting at which the Prize recipient is invited to give a lecture.



Nomination of John Andrew Todd

The 2021 EASD–Novo Nordisk Foundation Diabetes Prize for Excellence is being awarded to Professor John Andrew Todd

By Stefano Del Prato, President, EASD and Stephen O'Rahilly, Committee Chairperson

John Andrew Todd has been leading the genetic and mechanistic understanding of type 1 diabetes since 1986 and translating this knowledge into clinical trials and intervention studies whilst having a major impact on the study of all common multifactorial diseases. In 1994, he co-founded the Wellcome Centre for Human Genetics, in 1999–2005 he had a key role in initiating and developing the internationally successful UK Biobank and in 2003–2009 he founded and co-led the Wellcome Trust Case Control Consortium, which paved the way for a revolution in the genetic understanding of mechanisms in common diseases (including – but not limited to – type 1 diabetes).

Professor Todd is currently Professor of Precision Medicine, University of Oxford and Director of the Wellcome Centre for Human Genetics, University of Oxford, evidencing his seminal and leading role and expertise. His career development has been absolutely outstanding and impressive, as evidenced by his CV. His work has been supported by continuous long-term funding from different sources, enabling the latest state-of-the-art technical and bioinformatics methods to be developed and utilized.

Professor Todd's work has been recognized by a long list of international very prestigious awards and multiple visiting

professorships at the most well-known universities. His research productivity has been outstanding. His bibliography (more than 500 original papers) contains a long list of publications in very top-impact journals. His Web of Science citation numbers are huge, over 45,000, and h-index 101 (2021). His exceptional productivity seems to continue at the outstanding level, as evidenced by 17 recent publications (Science, Nature, Cell, PNAS and others).

Several breakthroughs by Professor Todd have advanced the understanding of the genetic architecture of type 1 diabetes and translated into the molecular mechanisms. Professor Todd was a driving force for the setup of an important genome-wide linkage study reporting interleukin-2 as a risk factor (Nature, 1991). This crucial discovery led to the demonstration of interleukin-2 pathways and its importance in T cell responses as a risk factor for type 1 diabetes. These results led to two key initiatives (2014) in type 1 diabetes: 1) to find a safe and optimal role for interleukin-2 as a replacement therapy to enhance protective immune responses and to prevent type 1 diabetes; and 2) to test whether improved tolerance to insulin can prevent the development of anti-islet antibodies and cell destruction. This concept is being tested in a large international trial – POInT – utilizing daily oral insulin to improve tolerance to

insulin. These key findings have been translated into potential novel pathways to prevent type 1 diabetes and its burden as a chronic disease in children. Another seminal focus has been to build up genetic risk scores utilized in practical tools to enable children at high risk for type 1 diabetes and autoimmunity to be identified – again a seminal and important approach in preventing type 1 diabetes (Nature Genetics, 2007).

His work has advanced the overall understanding of autoimmunity processes in other diseases such as rheumatoid arthritis and systemic lupus erythematosus (Frontiers in Immunology, 2019). His current focus is the role of the gut microbiome in the early development of type 1 diabetes and the potential for options to prevent type 1 diabetes.

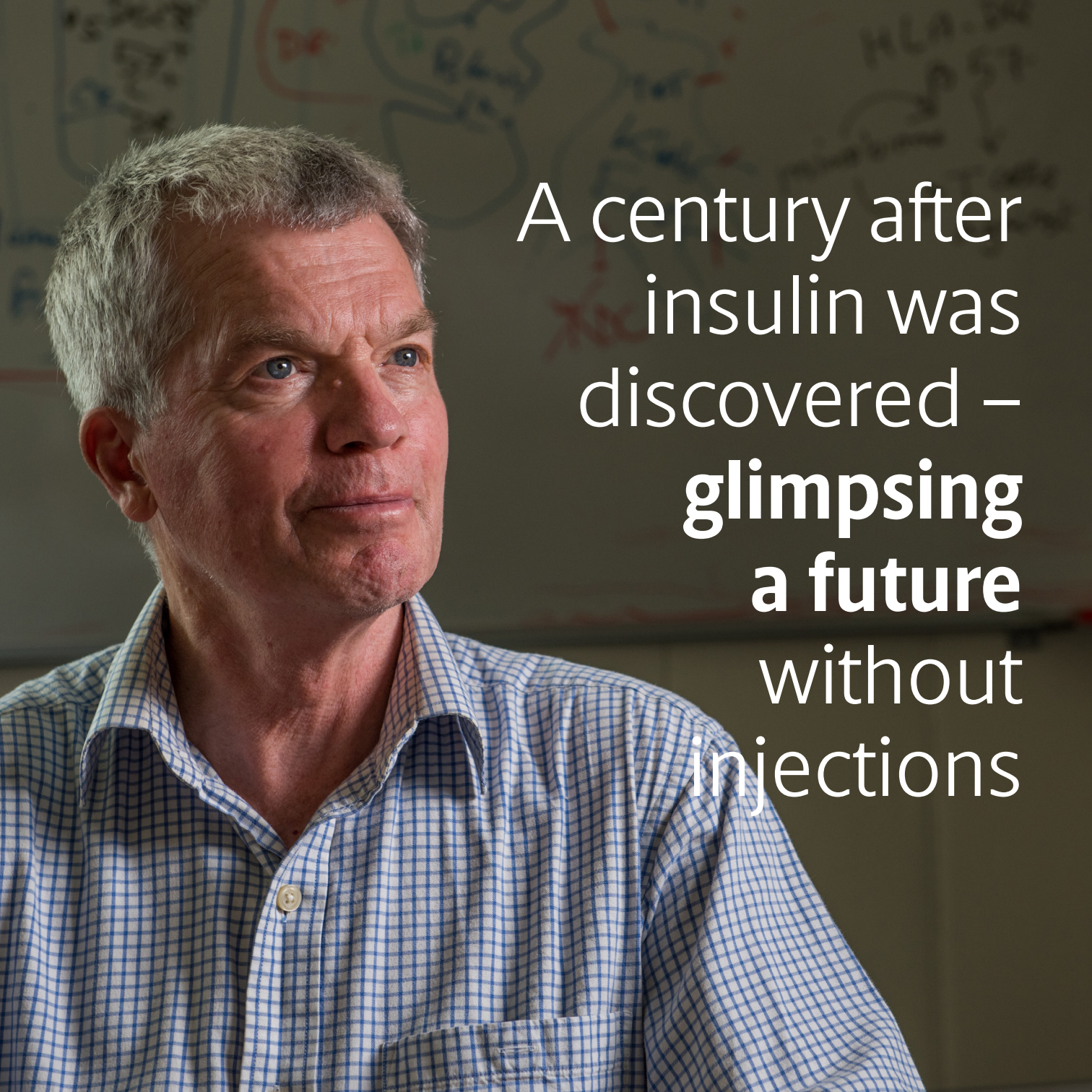
Professor John Todd is a true pioneer and visionary, definitely standing in the forefront of his field, the pathophysiology of type 1 diabetes. His work is characterized by innovative thinking, successfully translated into novel research targets. His numerous original and pioneering discoveries highlight his scientific excellence. He has fostered international collaborations with leading scientists to test the novel concepts in difficult clinical trials in children at high risk for type 1 diabetes.

Without question, Professor John Todd belongs to the list of outstanding recipients of the EASD–Novo Nordisk Foundation Diabetes Prize for Excellence.

We are therefore very pleased to inform you that, during the recent committee meeting of the EASD–Novo Nordisk Foundation Diabetes Prize for Excellence, a unanimous decision was made to award the 2021 EASD–Novo Nordisk Foundation Diabetes Prize for Excellence to Professor John Andrew Todd, a highly distinguished and stimulating speaker whom we are confident will deliver an exceptional lecture during the virtual 57th EASD Annual Meeting.

About John Andrew Todd

- 2016 Professor of Precision Medicine at the University of Oxford
- 2015 Helmholtz International Fellow Award
- 2011 JDRF David Rumbough Award for Scientific Excellence
- 2009 Fellow of The Royal Society
- 2003 Co-founder of the Wellcome Trust Case Control Consortium
- 2002 Professor of Medical Genetics at the University of Cambridge
- 1999 Co-initiator of the UK Biobank
- 1994 Co-founder of the Wellcome Centre for Human Genetics
- 1985 Postdoctoral fellow, Stanford University
- 1980 BSc in biological sciences, University of Edinburgh



A century after
insulin was
discovered –
glimpsing
a future
without
injections

When insulin was discovered 100 years ago, the cause of type 1 diabetes had only just been discovered. Since then, there has been a constant effort to understand why the immune system of people with diabetes attacks the body. Today, we can finally glimpse an everyday life for children with type 1 diabetes without daily insulin injections. For his 35 years of efforts to understand, prevent and combat type 1 diabetes, Professor John Andrew Todd is receiving the EASD–Novo Nordisk Foundation Diabetes Prize for Excellence.

Despite multiple promises to cure type 1 diabetes, especially in the 1990s, the only treatment for type 1 diabetes for a century has been to inject insulin every day for the rest of the person's life. The autoimmune disease that destroys the insulin-producing β cells in the islets of the pancreas has remained mysterious. "We can't go back to the 1990s, when the JDRF called it the 'decade of the cure'. No one is there yet. But we learned a lot of lessons in the 1990s. Now a lot of things are coinciding positively because of both new knowledge and new technologies. We are entering a new era of optimism in both prevention and treatment, so the level of genuine anticipation and optimism has increased," explains John Todd Professor of Precision Medicine at the University of Oxford.

Choosing science

Studying diabetes was not on John Todd's list when he started his career as a biology student at Edinburgh University in the late 1970s. His idea was to study penicillin-binding proteins in bacteria. This required becoming fascinated by and dependent on two disciplines that have served him faithfully since these very first scientific studies.

"I learned two things. If you are going to study biology, and all biology is complex, you need to use genetics. And then I went to lectures on immunology and I got hooked. And that was an immunology seed that has lasted until today."

John Todd thought he could use penicillin-binding proteins as biomarkers of bacterial development. Some bacteria turn from rods into spores.

"I visited the Biochemistry laboratory of David Ellar in Cambridge. And I said, 'David, I have an idea and I have a fellowship from Northern Ireland.' He said, 'That's interesting, come and join me.'"

Into the pigeonhole

It turned out that John Todd's idea was right. His work resulted in a paper in *Nature* in 1982. He wanted to do more genetics, not just in relation to bacteria but also humans. After he learned recombinant DNA technology for a year at the famous MRC Laboratory of Molecular Biology in Cambridge, he was ready to conquer the world.

"I wanted to combine human genetics and human immunology. There was one premier lab in the world that was really doing that: Hugh McDavitt's lab at Stanford University. So, I wrote to him out of the blue and said, 'I am a microbiologist, but I really want to do human biology, human immunology and genetics. And he didn't reply.'"

Instead John Todd got an offer from another premier lab – Leroy Hood's lab at the California Institute of Technology.

"On the day I was going to accept Lee Hood's offer, I went down to my pigeonhole in the department. And there was one of those stripy envelopes that you used to get – from Hugh McDavitt. And he said: 'You know, I do not know why I am making such an offer to a crazy microbiologist, but please come.' And thus, I got an offer to go to Stanford and was awarded an EMBO Fellowship to carry out the postdoc with Hugh," recalls John Todd.

An immune link

The stay at Stanford University changed John Todd's life, and several

amazing things happened. The polymerase chain reaction (PCR) was discovered and developed at this time – right around the corner from where John Todd worked – a revolutionary method used to rapidly make millions to billions of complete or partial copies of a specific DNA sequence.

“I was right at the heart of that because I knew the people at Cetus Corporation very well. I went trout fishing in California with Randy Seiki, who really perfected PCR. He kept telling me about this mysterious method, PCR. I went back to the lab, and I tried it in March 1986. My first experiment worked with PCR; it was a breakthrough moment. Until then, analysing human genetics took months.”

In Stanford, John Todd also met a young Canadian researcher, John Bell. They bonded immediately with equal energy, enthusiasm and determination and brought about fantastic results within a short time. The subject of study was proteins on the antigen-presenting cells in the human immune system, coded by HLA class II genes, which allow T lymphocytes to recognise both foreign and self antigens.

“We could now sequence them, genes from patients, in real time using PCR, and that had never been done before. We had a hypothesis about the mode of inheritance of these very strong HLA associations and how they might relate to different diseases – one being type 1 diabetes.”

The ground-breaking results were published in 1987 – once again in *Nature*.

There must be other genes

“That night in the lab, when I was going through the sequences, I noticed this correlation between between HLA class II gene DQ position 57 and the susceptibility and resistance to developing type 1 diabetes. I couldn’t believe it, and the first thing I did was to go home to my partner and say: ‘I’ve discovered something that could be really, really amazing.’”

The link between HLA class II genes and type 1 diabetes makes sense, since it is an autoimmune disease in which T lymphocytes destroy the insulin-producing beta cells. Despite this obvious link, the mechanism behind how position 57 affects type 1 diabetes has remained a mystery for more than 30 years. However, the discovery helped to launch and steer John Todd’s career towards studying type 1 diabetes.

Following the findings, Peter Morris, Head of Surgery at Oxford University Hospitals, contacted John Todd to invite him come back to Oxford. He got a fellowship from the JDRF, and this gave him a chance to start his own lab at the Department of Surgery in the John Radcliffe Hospital.

“Visiting other hospitals also made me realise that most of the people with type 1 diabetes are children, but also the integration between research and practice at these diabetes hospitals really motivated me to make a difference. I thought if we could find the change in position 57 in HLA class II genes, other genes must be involved.”

Double whammy in methods

Not long after, John Todd read a thought-provoking paper on the expression of genetically-determined diabetes in nonobese diabetic mice. The lead author was Merck researcher Linda Wicker. “Then I picked up the phone and called her and said: ‘Your *Journal of Experimental Medicine* paper is fascinating. Could we work together and work out the genetics of mouse type 1 diabetes, because if we can do that, then we can also determine this for human type 1 diabetes?’”

They immediately started planning their first genetic experiments in Wicker’s nonobese diabetic mouse model. And that led to another seminal *Nature* paper in 1991.

“Not only did we map the major genetic effects in the mice for autoimmune type 1 diabetes, but we developed a PCR-analysable map. So, it was like double whammy in methods development and

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also in using the new methods to discover things,” says John Todd. In the experiments, they discovered that subtle deficiencies in the interleukin-2 (IL-2) pathway influenced type 1 diabetes among many people. Importantly, it was a very mild difference causing significant resistance to type 1 diabetes.

“A two-fold difference protecting against disease by 70–80% in the mouse model was a surprise. IL-2 had already been implicated in regulatory T-cell function and an essential molecule for balance in the immune system, necessary for the functioning of the T cells to sustain self-tolerance and prevent autoimmune diseases. So again, our finding made good sense in connection with the development of type 1 diabetes.”

An L-shaped curve

In the early 1990s, as the Human Genome Project was getting underway, John Todd’s former partner-in-science at Stanford, John Bell, successfully applied for support from the Wellcome Trust to establish the Wellcome Centre for Human Genetics in Oxford. John Todd co-founded the Centre, as the first of several successful genetics projects he helped initiate, followed by the UK Biobank in 1999 and the Wellcome Trust Case Control Consortium in 2003. And in 2000 the JDRF and Wellcome formed a unique partnership to fund the Todd-Wicker laboratory in Cambridge, the JDRF/Wellcome Diabetes and Inflammation Laboratory (DIL) to map, understand the effects of type 1 diabetes genes and take forward the potential therapeutic opportunities.

“We mapped many, many genes in type 1 diabetes, and it is like an L-shaped curve. Thus, very few genes have a great effect, such as the HLA type II genes and the ones affecting insulin and IL-2, but then it tails off quite quickly, so that most genes have very little effect. Although some of these smaller effects and their associated genes have provided great insights into the biology of type 1 diabetes, for example, in the role of viral infections and gut bacteria in the early development of the disease,” explains John Todd.

Many researchers were still stuck on a Mendelian-effect mentality, with either the correct gene or a mutation and then a certain defined phenotype, but type 1 diabetes turned out to be different and more complex.

“In the beginning, we kept trying to think that these genetic changes were fully recessive and fully penetrant. And we kept mapping it and mapping it, until we realised that type 1 diabetes is polygenic, with none of the disease genes being fully penetrant. So, you might have a mutation, but that does not mean you are destined to get the disease, and you might lack some of the mutations but still get it.” The polygenic nature of type 1 diabetes confused and delayed many promising attempts to understand the disorder.

“We stopped doing genome-wide association studies quite early in our lab. We were more interested in the biology and translating it to the clinic and decided to start working on the top genes with the greatest effect without completely approximating that genes with a greater effect might have a larger influence on biology and therefore be ‘easier’ to study, although type 1 diabetes was never an easy disorder.”

Delays the need for insulin

Once the researchers accepted the fact that they would never find a single gene and a single mutation that led to type 1 diabetes, they started looking for obvious candidates to treat it. Based on the identified deficiencies in the IL-2 pathway predisposing to type 1 diabetes among many people, they decided to try to mimic the effect of IL-2.

“If we could very gently physiologically replace IL-2 by injection, then susceptible people could perhaps aspire to having a similar disease-protective effect. We proposed using aldesleukin – a recombinant human IL-2 drug – to compensate for the IL-2 pathway deficiency as a treatment for type 1 diabetes.”

The diagnosis of type 1 diabetes was traditionally considered to be the appearance of such symptoms as weight loss, thirst and



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excessive urination. However, it has become clear that this represents a very late stage of the disease.

“At this stage, lots of beta-cell function and insulin production has already been lost. With genetic risk markers and early markers of autoimmunity, autoantibodies against insulin itself, and against other islet proteins, type 1 diabetes can now be identified at a preclinical presymptomatic phase in which pancreatic beta-cell function is still sufficient to control blood glucose concentrations without the need for insulin therapy. Slowing the loss of beta-cell function delays the need for added insulin.”

The first two clinical trials with aldesleukin were conducted in Cambridge to determine the dose and dosing regimen for people with type 1 diabetes.

“We are running the third trial with children newly-diagnosed with type 1 diabetes. I am very excited and impatient for the outcome of this trial, because the results will help to implement IL-2 therapy for children and young adults who have signs of autoimmunity but are not yet diagnosed with type 1 diabetes and thus prevent the progression and diagnosis of this common and serious disorder that affects over 1 in 400 children per year,” says John Todd.

Global platform for prevention

In the past decade, new research on the onset and progression

of type 1 diabetes has led to the identification of strong familial and genetic associations and islet-specific autoantibodies. These studies have revealed that the islet-specific autoantibody can appear years before clinical diagnosis.

“The first autoantibody to appear is against insulin, with a peak incidence around the age of 1 year. Ninety percent of the children with a single type of islet-specific autoantibody do not progress to type 1 diabetes, but seroconversion to the presence of two or more autoantibodies points to a risk of over 80% of clinically-diagnosed type 1 diabetes by the age of 18 years.”

The idea of oral tolerance to insulin therefore arose, so type 1 diabetes can be stopped before the first autoantibody develops, which is usually for insulin. In the trial, children are tested for 47 single-nucleotide polymorphisms that determine the risk of type 1 diabetes. If the newborn child was in the high-risk category, their families were asked whether they would like to have their child randomised and enter the trial.”

“So, with colleagues in Munich and Dresden we formed a consortium to build a Global Platform for the Prevention of Autoimmune Diabetes with the Helmsley Charitable Trust to conduct studies like this with the purpose of preventing or eradicating type 1 diabetes. We then give the children daily oral insulin over 3 years to build up their immune tolerance to insulin. More than 1000 children from five countries have

entered the trial, and we are delighted on how families are continuing to participate, eating their food mixed with the insulin powder,” explains John Todd.

A bit like a vaccine

A second trial underway is investigating using probiotics in the primary prevention of type 1 diabetes. Inspired by a growing number of studies linking type 1 diabetes and gut dysbiosis, the trial involves giving a probiotic to newborn babies who are at high risk of type 1 diabetes.

“We developed an interest in 2017 in probiotics and how they affect the immune system, led by a senior post doc in the lab, Dr Marcin Pekalski. A student in the lab, Arcadio Rubio García, discovered that hidden in the human microbiome is a mimotope of the key insulin sequence, which could be a factor in autoimmunity and type 1 diabetes.”

A mimotope is a molecule that mimics the structure of the antigen that is normally recognised by the immune system.

“Our current theory is that the bacteria containing these mimotopes help to tolerise us against type 1 diabetes. The bacteria could help establish tolerance to insulin and prevent the earliest events in the development of the disease in the first days and weeks of life,” says John Todd.

Bacteria and babies

John Todd’s group has been working on the project for over 3 years together with Marcin Pekalski, an expert in the link between genetic and environmental determinants of autoimmunity, being a key lead in the project’s initiation and development, with Linda Wicker once again lending her wisdom and deep knowledge of the immune system.

“We already have evidence of the cross-reactivity of these mimotopes with the insulin B chain sequence 9–23. This link might

actually be an important reason for the growing number of children with type 1 diabetes. In the United Kingdom, only 80% of women start breastfeeding, and by 24 weeks, only 1% of are still exclusively breastfeeding, and the bacteria that are so important for a healthy gut in the first weeks of life require the sugars in breast milk to metabolise, grow and colonise the intestine.”

These bacteria, known as commensals, when they are thriving and plentiful produce many metabolites that have extraordinary benefits for the functioning of the child’s immune, endocrine and neurological systems.

The DIL aims through the studies to understand the role of mimotopes and understand how early commensal tolerance affects the baby’s immune system, the tolerance to that bacterial mimotope and eventually to the baby’s own insulin.

“Without breast-milk, these bacteria do not do well. So, if you do not breastfeed you should maybe take a probiotic in the first weeks to lower the risk of type 1 diabetes. Especially if you are genetically at risk of type 1 diabetes,” explains John Todd.

However, John Todd’s group first needed to understand in small clinical studies asking very targeted questions, including which are the best probiotic bacteria; when best to begin supplementation of newborn babies; how to compensate in the best ways when breast feeding is stopped; and do the HLA genes, including position 57, influence early-life immunity to insulin and the interactions between the child’s immune and endocrine systems and the healthy and activity of the gut bacteria?

HLA-DQ revisited

Even though John Todd has come a long way since his first diabetes study in 1986, the mechanism behind the discovery of position 57 of the beta chain of the HLA-DQ molecule that helped him launch his career still remained a mystery.

“Solving this mystery that I pondered probably every day of my career, I have developed this obsession. When I see a house number 57. When my phone or watch run out of power, I look down, it’s always 57. It’s a psychological condition. If you start looking for something, you will find it.”

With major advances in single-cell sequencing technology for T-cell receptor antigen sequencing over the past 4 years and careful clinical collections over many years, the researchers are actually beginning to unravel the mechanisms underlying the mystery.

“The question was how the amino acid aspartic acid at position 57 protects against type 1 diabetes. And now it seems we have the answer: the negatively-charged aspartic acid at position 57 repels negatively-charged amino acids in the peptide-binding bit of the T-cell receptor and in the insulin B:9-23 peptide, a simple charge repulsion model. This causes a reduction in the production of T cells that can be autoreactive against insulin and that could, in the presence of other type 1 diabetes genes, environmental factors and of course the activity of the gut bacteria,” explains John Todd.

So, one single change in the mysterious position 57 changes the interactions in the T-cell receptor binding and insulin and thus contributes critically to this devastating autoimmune reaction. John Todd is now trying to get the results published. I am just so lucky and rejoice in the circularity of it.”

The future of diabetes prevention

After 35 years of research, John Todd still remains equally determined to succeed in treating and prevent type 1 diabetes and the lifelong implications such as blindness, kidney failure, neuropathy and cardiovascular disease. Despite many disappointments, he still feels that the research community is getting closer and closer to understanding the complex interaction of genes and environmental factors, especially in the first year of life.

“Our group takes a unique approach to treating and preventing type 1 diabetes because we aim to understand the genetics of the disease and the relevant pathological mechanisms. We are then able to treat type 1 diabetes using drugs with a mode of action specific to the disease mechanism. This is our personalised or precision medicine approach for type 1 diabetes. And we are confident that we or others will eventually stop the body’s own immune system from destroying the cells in the pancreatic islets that produce insulin,” says John Todd.

Whether it is one of John Todd’s own ongoing trials or one led by his colleagues is less important. Right now, the first prescribed drug since insulin was discovered 100 years ago is close to passing the last clinical trial: an anti-CD3 antibody that binds to the surface of T cells and thus suppresses the immune system and hopefully the destruction of the T cells.

“This drug seems to be able to prevent children with autoantibodies from developing type 1 diabetes. So that is why these anti-CD3 antibodies are creating such a stir. Even though we do not fully understand the mechanism yet, it seems to have a very significant effect and can ensure that these children develop type 1 diabetes later or potentially live the rest of their life without having to inject insulin. We recently had an opportunity to review this current exciting state-of-affairs in the international journal, Science. Perhaps IL-2 will be next? Watch this space!”

John Andrew Todd is receiving the 2021 EASD–Novo Nordisk Foundation Diabetes Prize for Excellence, accompanied by DKK 6 million (€806,000) for his outstanding contributions that have improved knowledge of diabetes.

Year and location of EASD Annual Meeting at which recipients have received the Prize.



The European Association for the Study of Diabetes

The European Association for the Study of Diabetes (EASD) was founded in Montecatini, Italy in 1965.

The mission of the EASD is to promote excellence in diabetes care through research and education. The aims are to encourage and support research, the rapid diffusion of acquired knowledge and to facilitate its application.

EASD membership is open to scientists, physicians, students, postdocs and fellows, allied health professionals and nurses from all over the world who are interested in the field of diabetes or related diseases. Each year, the EASD Annual Meeting brings together over 15,000 medical professionals as well as an online audience of thousands. EASD is the home of diabetes research in Europe.

The Association holds training courses and workshops to attract new talent to diabetes research and to disseminate the latest knowledge. In addition, it has established a large number of study groups focusing on different areas of diabetes research and care and has founded the journal *Diabetologia*.

In 2000, the Association created the European Foundation for the Study of Diabetes (EFSD), which operates on a non-profit basis.

The Novo Nordisk Foundation

The Novo Nordisk Foundation is an independent Danish foundation with corporate interests. Its history goes back more than 90 years.

The objectives of the Foundation are:

1) to provide a stable basis for the commercial and research activities of the companies in the Novo Group; and 2) to support scientific, humanitarian and social purposes.

Our vision is to contribute significantly to research and development that improves people's lives and the sustainability of society.

Since 2010, the Foundation has donated more than DKK 25 billion (€3.3 billion), primarily for research within biomedicine and biotechnology and diabetes treatment at universities and hospitals in Denmark and the other Nordic countries. The Foundation supports the entire research chain – from education to innovation.

In addition to awarding grants, the Foundation annually awards several honorary prizes to recognize and reward individuals for their unique efforts in research, teaching or other efforts relevant to research.

Novo Nordisk Foundation

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