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Q1 You were recently elected to the European Association for the Study of Diabetes (EASD) Board, beginning in January 2026. What does being part of the EASD Board mean to you, and what are you hoping to contribute in this role?

One of the main things I can bring to the Board is the view of a basic researcher. I want to make sure that basic research stays a core part of EASD and its meetings. One of the EASD's greatest strengths is that it brings basic and clinical scientists together in the same arena, so a big part of my role will be making sure we don't lose the basic scientists from the meeting.

I also have a specific role on the Board as the EASD's representative in the BioMed Alliance. This is a policymaking organisation in the EU that brings together societies from many fields, not only from diabetes but also cardiology, obesity, neurology, and others. I think I can play a useful role in bridging the EASD and BioMed Alliance to better support the translation of basic research into clinical outcomes.

Q2 The EASD has an important role in supporting the next generation of diabetes researchers. What opportunities do you see for early-career scientists, and how can EASD help them shape the future of the field?

I think we can do a lot. The EASD has started an Early Career Academy. Through it, we support junior researchers and help them take on leading roles at the meetings. There are also workshops for the younger generation, where they can meet one another and get support from the EASD on how to grow their careers. We have an important role to play as role models, and the EASD puts a lot of emphasis on helping the next generation. This is key. Science is growing very fast,



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and if it is going to keep growing, we need people on board. We especially need to support young researchers so they can be part of this process.

Q3 A lot of your work has been dedicated to understanding how pancreatic α and β cells regulate glucagon and insulin secretion, respectively. What are some of the most important things we now understand about these cells that we didn't know when you began your research?

One thing we definitely know much more about now is the human situation. When I started (and I have been around for quite some time), we couldn't study human islet cells, because we had no access to human material, so we had to rely on mouse models. Getting access to human islets was a very big step forward. We saw that many of the basic features of how α and β cells work were very similar, so animal models are still useful, and that is important. But several things were clearly different.

In my own field, I study small non-coding RNAs called microRNAs. Even when a microRNA is conserved between humans and mice, its targets can differ. They can be the same, but they can also be different. So, it is important to study human islet cells or human β cell lines, or at least to follow up findings in humans. Being able to use human islets has been a breakthrough in understanding what is happening in human islet cells.

More recently, we have also learned much more about paracrine regulation within the islet. We knew about it before, but not in such detail. The heterogeneity of islet cells is another very important factor that we need to understand well.

I also have some personal highlights from very early on, such as our discovery in 1996 that sulfonylureas affect exocytosis. This may be a good story for the younger generation. We were using patch clamping to measure the effect of glucose on exocytosis. Patch clamping already lets us control the membrane potential, but we added sulfonylureas to make doubly sure we closed the ATP-sensitive potassium channels. Then, more or less by serendipity, we saw an increase in exocytosis. We repeated the experiment over and over and got the same result each time. So, we started to investigate the mechanism, and it became an important discovery and paper. That was not too bad for a chance finding!

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Q4 Looking back at the 2026 EASD Annual Meeting, a number of oral presentation sessions focused on β cell biology. Considering your own research in areas such as α and β cell function, microRNAs, and the mechanisms behind impaired glucagon and insulin secretion, were there any key developments discussed, or sessions that made you particularly excited for upcoming research?

There was not much on microRNAs this year, although there were a few studies more connected to Type 1 diabetes, along with some screening studies.

On the physiology side, one of the main oral presentations came from Uppsala University, Sweden. They had looked not at α or β cells, but at another islet cell type, the δ cells. They found that δ cells in islets from people with Type 2 diabetes (T2D) are hypersecreting. That means they put an even stronger brake on both insulin and glucagon secretion in T2D, which leads to even less insulin secretion. I thought those were very interesting findings.

I also enjoyed a very interesting lecture by my colleague Charlotte Ling, Lund University, Sweden, though perhaps I am biased. She spoke about epigenetics and DNA methylation, and about the different pieces of the puzzle in understanding how DNA methylation affects insulin secretion and other aspects of T2D.

There were also several sessions on stem cells, on improving stem cell quality for transplantation, and on islet transplantation. Per-Ola Carlsson, Uppsala University, Sweden, has transplanted human islets in a clinical trial where they managed to suppress the immune response so that the immune system cannot attack the transplanted cells. That is very impressive work. There was also a morning session that opened with Henrik Semb, Helmholtz Munich, Oberschleißheim, Germany. He described a clinical trial of stem cell therapy in Germany that showed improved glycaemic control. They have everything in place, but he also discussed the challenges of making this a reality. Much still needs to be done to set up rules and regulations for how this should be carried out.



Q5 There is a growing emphasis on moving from understanding the biology of diabetes to translating those discoveries into better treatments. From your perspective, where are the biggest opportunities, and challenges, in turning advances in basic diabetes research into benefits for patients?

I think there are several lines. Stem cells are one, which we have already touched on. I don't work in the stem cell field myself, but it was discussed at the EASD Annual Meeting that better protocols are needed and that there is still work to do to get these therapies into the clinic.

In my own area of expertise, microRNAs and epigenetics such as DNA methylation, there are still several challenges to overcome before these can be used as pharmacological treatments in the clinic. To change DNA methylation or other factors, you have to alter the genetic structure in some way, for example with clustered regularly interspaced short palindromic repeats deletions, and then deliver that into the cell in some kind of particle. There are several options:

- Synthetic lipid nanoparticles
- Specially engineered, non-infectious virus-like particles, which have the membrane of a virus but contain no virus; Diego Balboa, University of Helsinki, Finland, gave a talk showing that these can deliver material into stem cells
- More natural exosomes or extracellular vesicles, which can be taken from the patient's own cells, loaded with whatever you like, and put back into the patient

Maybe that is where the future lies. Seeing what has been achieved in the stem cell field, I can imagine it should also be possible to bring these approaches into the clinic. I am a very hopeful person, and I think targeting microRNAs could still have a huge effect on β cells.

On the physiology side, there are new possibilities in pharmacology today. Cryogenic electron microscopy and new ways of designing drugs and compounds make them better and more selective. These are bringing us closer to the end goal, even if we are not there yet, and I think they will increase our ability to influence the physiology of islet cells.

Q6 Finally, looking ahead, what do you think will be the most important questions for the diabetes research community to address over the next 5–10 years, and what gives you the most optimism about the future?

I think we have already touched on the future a little. It would be very interesting to use epigenetic changes, microRNAs, or other tools to manipulate insulin secretion by controlling communication within the β cells. I also think we can do much more at the level of the fusion pore and how it opens, perhaps using similar techniques, although that is also a long way off. It would be very interesting to see whether we could improve insulin secretion, but secreting too much insulin is not good either. Finding the right balance of all the different hormones, and learning how to regulate it, would be fantastic. I think we will get there as our understanding grows. We need something that acts like a rheostat for the cell. Here, I come back to microRNAs, because I think they are one type of regulator that can find an optimal level of protein expression. Many different steps need to work together, and if we find the key factor in all the complexity we are now facing, I think we will be close to a solution.

Take the analogy with AI and neural networks. Many years ago, I listened to a fascinating talk about the brain, and I think we can apply the same idea to islet cells. They used neural network thinking to find the hub gene: the gene that regulates several others and matters most. We can still pick out genes one by one and investigate their function, which is what we have done for many years to study how insulin secretion is regulated. But if we could find the one gene or regulator that is the key regulator in all of this, that would be fascinating.

The blue-sky goal is still to get stem cells working to help people with Type 1 diabetes. I think the work on networks and key regulators is more relevant to people with T2D, helping them achieve better insulin and glucagon secretion.

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