Diabetic potential to create own insulin

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Researchers from the Peninsula Medical School, working in collaboration with colleagues from Glasgow Royal Infirmary and the University of Brighton, have used a unique collection of pancreas specimens taken from patients who died soon after diagnosis of type 1 diabetes to show that they respond to the ongoing process of destruction by inducing their islet cells to proliferate.

The research is published on-line at *Diabetologia* and is funded by Juvenile Diabetes Research Foundation.

The findings are important because, until now, it has been generally believed that, in humans, beta cells divide only very infrequently after the first year or so of life and that they do not readily proliferate once type 1 diabetes is diagnosed. By studying the same unique collection of pancreas specimens that last year led the research team to conclude that some cases of type 1 diabetes may have a viral cause, this current study presents evidence that there is a 10-fold increase in islet cell replication in patients recently diagnosed with type 1 diabetes. A similar response had been seen previously in an animal model of type 1 diabetes by a member of the team, Professor Adrian Bone (University of Brighton) but it was not known if accelerated β-cell replication also occurs in human patients.

The factors that trigger the replication process in patients with type 1 diabetes are still unclear, although the study shows a correlation with the infiltration of immune cells (which suggests that an immune mediator is likely to be involved).

The results of the research offer the hope that, in future, it might be possible to encourage a newly diagnosed type 1 diabetes patient's own beta cells to reproduce as a means of replacing those being destroyed by the disease. The development of such a therapy could mean that some patients with type 1 diabetes would be able to produce their own insulin for a longer period, thereby reducing the need for pharmaceutical interventions.

Professor Noel Morgan, Director of the Institute of Biomedical and Clinical Sciences at the Peninsula Medical School, commented: "Our findings are significant because they challenge current thinking and offer the hope that, at some point in the future, a therapy could be developed that would allow individuals who are developing type 1 diabetes to retain their own insulin. We are a long way from this point, but the first steps have been taken with this research."

He added: "We could not have come to our current conclusions without access to the unique collection of pancreas specimens from patients who died soon after being diagnosed with type 1 diabetes, which was collected by our colleague Dr. Alan Foulis from the Department of Pathology at Glasgow Royal Infirmary. The specimens are allowing us to investigate the cellular processes that occur in the beta cells of people with type 1 diabetes in a way that has not been possible before."

Karen Addington, CEO at JDRF said: "Interventions to halt and reverse the onset of type 1 diabetes are a fundamental part of JDRF's objective to cure, treat and prevent type 1 diabetes and its complications, and these results inform our global research programme. These are early stage findings, but we are positive about the impact this could have for people diagnosed with type 1 diabetes in the future."

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