

# Scientists shed new light on link between 'killer cells' and diabetes

January 15 2012

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Killer T-cells in the human body which help protect us from disease can inadvertently destroy cells that produce insulin, new research has uncovered.

The study provides the first evidence of this mechanism in action and could offer new understanding of the cause of Type 1 [diabetes](#).

Professor Andy Sewell, an expert in human T-cells from Cardiff University's School of Medicine worked alongside diabetes experts from King's College London to better understand the role of T-cells in the development of Type 1 diabetes.

The team isolated a T-cell from a patient with Type 1 diabetes to view a unique [molecular interaction](#) which results in the killing of insulin-producing [cells](#) in the pancreas.

"Type 1 diabetes is a result of the body's own immune system attacking and destroying the cells in the pancreas that manufacture the [hormone insulin](#). [Insulin](#) controls [blood sugar levels](#) and a lack of insulin is fatal if untreated," said Professor Sewell.

"The mechanism by which the body attacks its own insulin producing cells in the [pancreas](#) is not fully understood. Our findings show how killer T-cells might play an important role in autoimmune diseases like diabetes and we've secured the first ever glimpse of the mechanism by which killer T-cells can attack our own body cells to cause disease," he

added.

Co-author of the study, Professor Mark Peakman from the National Institute for Health Research (NIHR) Biomedical Research Centre at King's College London and Guy's and St Thomas' NHS Foundation Trust said: "This first sight of how killer T-cells make contact with the cells that make insulin is very enlightening, and increases our understanding of how Type 1 diabetes may arise.

"This knowledge will be used in the future to help us predict who might get the disease and also to develop new approaches to prevent it. Our aim is to catch the disease early before too many insulin-producing cells have been damaged."

The team now hope that by gaining a better understanding of this process it will put them in a much stronger position to devise new ways to prevent or even halt the disease.

The study, funded by the UK Biotechnology and Biological Sciences Research Council (BBSRC), the Juvenile Diabetes Research Foundation (JDRF) using facilities at Diamond Light Source and published in *Nature Immunology*, shows that the killer T-cell receptor utilises an abnormal mode of binding in order to recognise cells producing insulin.

"The results of Dr Sewell's work provide key novel insights into T1D pathogenesis" said Teodora Staeva, Director of JDRF's Immune Therapies Program. "JDRF is pleased to support this kind of research that will accelerate the development of biomarkers and preventive therapies for [Type 1 diabetes](#)."

This unusual binding is thought to allow the T-cell to survive the culling process designed to rid the body of autoreactive T-cells.

The structure of the killer T-cell receptor bound to the insulin peptide shows that the interaction is highly focused on just a small part of the molecule.

In a further study published in the *Journal of Biological Chemistry* the same Cardiff and King's team has shown that this focused binding mode allows this T-cell receptor to respond to over 1.3 million other peptides of different molecular shape.

This ability to bind peptides with a multitude of different shapes may provide a clue as to how [autoimmune diseases](#) are initiated. It is possible that this T-cell was raised to fight an infection via one of the other 1.3 million peptides it can recognise but then inadvertently also recognised insulin once it had been put on 'red alert' by this infection.

Diabetes describes diseases where a person has high blood sugar. Treatment of diabetes and its complications represents a major health burden and accounts for over 10% of the National Health Service's annual budget.

Provided by Cardiff University

Citation: Scientists shed new light on link between 'killer cells' and diabetes (2012, January 15) retrieved 9 April 2024 from <https://medicalxpress.com/news/2012-01-scientists-link-killer-cells-diabetes.html>

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