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Insulin signaling is distorted in pancreases of Type 2 diabetics

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Insulin signaling is altered in the pancreas, a new study shows for the first time in humans. The errant signals disrupt both the number and quality of beta cells — the cells that produce insulin.

The finding is described in the journal *PLoS ONE*. Franco Folli, M.D., Ph.D., of the School of Medicine at The University of Texas Health Science Center San Antonio, and Rohit Kulkarni, M.D., Ph.D., of the Joslin Diabetes Center, Harvard Medical School, Boston, are principal investigators of the study. In a statement, they said: "People knew there was a lack of beta cells because they die off in type 2 diabetes. Here we show the beta cells attempt to replicate, but this is unsuccessful because of the altered signals."

Inability of the beta cells to replicate themselves results in a major defect in insulin secretion during the late stages of type 2 diabetes, said Drs. Folli and Kulkarni.

Insulin is the hormone that lowers blood sugar after a meal. The study, which examined pancreases from cadaveric organ donors, suggests a potential strategy to prevent beta cells from being depleted — by restoring insulin signals back to normal. This could have important implications for millions of people with type 2 diabetes, a disease marked by poor regulation of blood sugar levels.

Cells in most organs, except the central nervous system, turn over in cell division. One cell dies and another replicates to perform the same

function. This is true in the islets of Langerhans, the area of the pancreas where beta cells and other blood glucose regulators originate.

The study also demonstrated, for the first time in humans, that the insulin receptor is critically important for maintaining beta cell mass. This was previously seen in rodent knock-out models of type 2 diabetes mellitus. A receptor is a molecule on the cell's membrane that receives hormones' signals and transmits them into the cell.

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