How fat could help solve part of the diabetes problem

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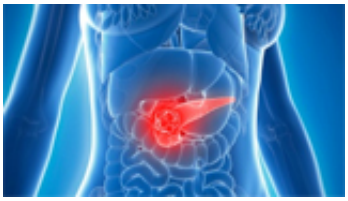

The pancreas is a large organ that wraps around our gut, and produces the exact amount of insulin our bodies need when we eat – except when we start to develop diabetes, and insulin production slows down. Sydney scientists describe how a fat recycling system within pancreatic 'beta cells' determines the amount of insulin they secrete, and so may provide a target for future diabetes therapies.

A small structure inside the beta cell, known as a 'lysosome', behaves like an intracellular recycling unit. It breaks down unwanted fats and proteins in such a way that they can be re-used.

PhD student Gemma Pearson and Professor Trevor Biden from Sydney's Garvan Institute of Medical Research showed that when they prevented lysosomes from breaking down fat, beta cells secreted more insulin. Their study is published in *Diabetologia*, now online.

While this is a very early stage cell biology story, it nonetheless holds promise, and encourages the scientific community to look at diabetes therapies through a fatty lens.

"There are many different ways fats can be used within the beta cell – so if you stop them being recycled, you force them to be used in a different way," said Gemma Pearson, whose PhD examines the "lipid profile" of beta cells.

"When you shift fats from the lysosome, you store them in other parts of the cell, and they become available to participate in various signaling pathways. One of these pathways clearly increases insulin secretion."

"Fat molecules are not the inert blobs you might think – they can bind to proteins and activate them, causing a range of downstream events to occur."

"The good thing about this particular pathway is that it is only stimulated by glucose. That limits the beta cell to producing excess insulin only to deal with food, rather than around the clock. Too much insulin circulating in the blood, or hyperinsulinaemia, can be very detrimental to health in many respects."

"If in future a drug were to be developed to block fat degradation in the lysosome, it would have to be tweaked to affect beta cells only."

More information: link.springer.com/article/10.1007%2Fs00125-013-3083-x

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