Skin cell response to environmental stimuli like viruses may predict type 1 diabetes

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(Boston)—Type 1 diabetes is a genetically-driven autoimmune disease of pancreatic beta-cells, whose origins remain unknown. Researchers at the Boston Medical Center (BMC) and Boston University School of Medicine (BUSM) discovered that skin cells from patients with type 1 diabetes display abnormal activity triggered by immune response mechanisms to environmental stimuli like a viral infection. These findings currently appear online in *PLoS One*.

They found that these cells acquire elevated levels of calcium when exposed to either cytokines or fat. In humans, cytokines or cell signalling molecules essential to the body's immune response, increase with the onset of infection as does an excess of fatty acids when people are sick and stop eating, a common occurrence in children when they get viruses.

"This is significant as it is known that a viral illness usually precedes the development of type 1 diabetes in children but no one knows why it should be related," says Barbara Corkey, PhD, Zoltan Kohn Professor of Medicine at BUSM and vice chair for Research in the Obesity Research Center at BMC. "Our findings that diabetic cells have a different sensitivity as indicated by higher levels of calcium to an environmental event such as a virus, may help to explain why the onset of type 1 diabetes might be triggered by an environmental stimulus as well as a genetic predisposition."

In fact, their data showed that skin cells from relatives of people with type 1 diabetes who are not afflicted with type 1 diabetes themselves

display an intermediate calcium response to circulating signaling molecules. These data suggest that a unique environmental stimulus may interact with a genetic trait to initiate diabetes.

"Determination of this trait before development of diabetes could help to identify susceptible individuals prior to disease onset," adds Corkey.

Provided by Boston University Medical Center