Sensory Neurons Control β Cell Stress and Islet Inflammation in Autoimmune Diabetes

By: Curtis Richard

What is Diabetes?

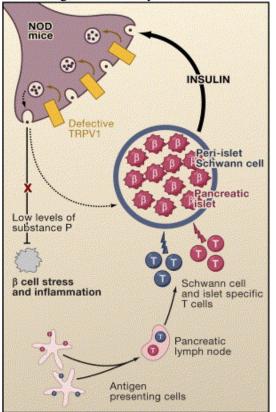
Diabetes mellitus is a metabolic disorder characterized by hyperglycemia (high blood sugar) and other signs, as distinct from a single disease or condition. Three types: type 1, type 2, and gestational diabetes. Type 1 is usually due to autoimmune destruction of the pancreatic beta cells, which produce insulin. Type 2 is characterized by tissue-wide insulin resistance and varies widely; it sometimes progresses to loss of beta cell function. Gestational diabetes is similar to type-2 diabetes, in that it involves insulin resistance.

Current Treatments:

Currently, type-1 diabetes can be treated only with insulin, with careful monitoring of blood glucose levels using blood-testing monitors. Emphasis is also placed on lifestyle adjustments (diet and exercise). Apart from the common subcutaneous injections, it is also possible to deliver insulin by a pump, which allows continuous infusion of insulin 24 hours a day at preset levels and the ability to program doses of insulin as needed at meal times. It is also possible to deliver insulin with an inhaled powder.

New findings:

In normal mice, a feedback loop involving islet β cells and sensory neurons expressing TRPV1 maintains balanced levels of insulin and substance P. In NOD mice, insulin secretion by islet β cells fails to properly stimulate the sensory neurons expressing TRPV1 to release neuropeptides due to the presence of a hypofunctional polymorphism in the trpv1 gene. Suboptimal local levels of neuropeptides lead to insulin resistance and β cell stress as well as a local proinflammatory milieu, while physiological cell death of neurons and islet β cells leads to the presentation of auto-antigens by professional antigen-presenting cells (APCs) in draining lymph nodes. Infiltration of Schwann cell and islet-specific T cells is sustained by the local proinflammatory milieu resulting from defective TRPV1 signals in sensory neurons.



With an increase in the release of the neuropeptides, the stress on the beta cells is reduced, improving function and potential reversal of the disease.

Works Cited:

Journal:

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