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Key Biomarkers for Diabetes

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About the Study

Recent studies shows how a new regulatory mechanism serves as an important biomarker for diabetes development as well as a possible therapeutic target for its prevention. The most significant pancreatic hormones in target tissues, such as the liver, are glucagon and insulin, which regulate proper glucose levels in response to food intake. Glucagon is secreted by pancreatic a-cells during fasting to increase blood glucose and protect the body from hypoglycemia. Glucagon has also been related to the development of diabetic hyperglycemia, primarily through the production of increased hepatic glucose, or HGP. Factor that affects transcription, glucagon acts by binding to a receptor known as a G-protein-coupled receptor, or GCGR.

In animals and humans with diabetes, an elevated amount of blood glucagon exists, stimulating excessive HGP and leading to diabetic hyperglycemia. A significant underlying cause for the development of Type 2 diabetes is the disruption of proper hepatic glucose production. HGP is boosted by the pancreatic hormone glucagon, while HGP is reduced by insulin. Insulin involved in gene transcriptional regulation in the liver cell nucleus suppresses the production of glucose and Foxo1 is an important component of insulin signalling cascades that control cell growth, differentiation and metabolism. Role of Foxo1 was known to play in the regulation of HGP by glucagon. Phosphorylation, or the addition of a phosphoryl group to a protein, is essential for its work since it activates or deactivates nearly half of the body's enzymes, controlling their operation.

CRISPR/CAS9 technology is used in the development of Foxo1 'knock-in' mice to determine how this Foxo1 phosphorylation will function in an animal model for their investigation. When insulin is reduced and glucagon is increased in the blood stream, Foxo1 is balanced in the liver of fasting mice. When the GCGR is activated, it activates adenylate cyclase, an enzyme that plays a key regulatory role in almost all cells, and increases intracellular PKA levels. Hepatic Foxo1 deletion in mice resulted in a substantial reduction in hepatic glucose output and blood glucose levels. This was been discovered a novel genetic, cellular, and physiological mechanism by which Foxo1 regulates hepatic gluconeogenesis and blood glucose levels by phosphorylating glucagon signaling.

Foxo1 also acts as a mediator of multiple signaling cascades and integrates different hormones and intracellular protein kinases into the programming that controls insulin sensitivity, HGP and blood glucose. In both type 1 and type 2 diabetes, a high glucagon level is present, and Foxo1 plays a key role in the fundamental process that leads to excess liver gluconeogenesis and results in diabetic hyperglycemia. This indicates that glucagon-mediated HGP may be a major potential therapeutic tool for diabetes management and possible prevention.