



# A Brief Note on Regulation of Blood Glucose Regulation

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## DESCRIPTION

Blood sugar levels are regulated by several pancreatic hormones. Pancreatic hormones regulate blood glucose levels in a variety of ways under normal and abnormal conditions by expressing or suppressing the target of several genes or molecules or cells. Some synthetic drugs and treatments are used to cure glucose regulation problems, but many of them are used to cure other health problems caused by impaired blood sugar regulation. Many new approaches have been used to develop phytochemical-based agents to treat glycemic control problems, treating insulin resistance, intestinal or GLP1 homeostasis or GLP1 homeostasis beta cell function or glucose. Many compounds have been isolated and identified to regulate absorption. More metabolic pathways (eg, cure of hyperglycemia, or increased insulin resistance, or cure of pancreatic beta cell regeneration, or increased GLP1, pancreatic islet cell production, production and insulin receptor signaling and increased insulin secretion, or decreased insulin resistance, or gluconeogenesis and insulin mimicking, or production of  $\beta$ -glucosidase and  $\beta$ -amylase inhibitors or maintenance of pancreatic islet mass or Protein Kinase A (PKA) and Extracellular Signal Regulatory Kinase Activates (ERK) or AMPK and reduces insulin sensitivity or suppresses  $\beta$ -glucosidase and activates AM PK and downstream molecules prevent cell death of pancreatic  $\beta$ -cells and are insulin-like. Activates SIRT1 or hypoglycemia to reduce chemical structure or lipid peroxidation.

## The role of glucagon in blood glucose control

The action of glucagon has the net effect of tricking the liver into releasing glucose stored in cells into the bloodstream, raising blood sugar levels. Glucagon also causes the liver (and other cells such as muscles) to produce glucose from building blocks derived from other nutrients in the body (such as proteins). Our body wants to keep blood glucose between 70mg/dl and 110mg/dl (mg/dl means milligrams of glucose in 100 milliliters of blood). Less than 70 is called "hypoglycemia". If eaten within a few hours, it may be normal to exceed 110. This is why doctors want to measure their blood glucose during a fast. Must be between 70 and 110. But even after eating, the blood sugar level should be less than 180. Above 180, it is called "hyperglycemia". (This means "too much glucose in the blood"). Diabetes is diagnosed if the two blood glucose levels above 200 after drinking a sugar water drink (glucose tolerance test).

## Fetal glucose metabolism

Glucose is the main source of energy for fetal development. Fetal

glucose homeostasis is entirely dependent on the continuous movement of glucose through the placenta. In fact, no significant fetal glucose production has been demonstrated. The fetal glucose pool is in equilibrium with that of the mother. The fetal endocrine environment is characterized by high plasma insulin and low plasma glucagon levels. Circulating insulin can be detected after 13 weeks of gestation. The fetal pancreas can release insulin in response to glucose and amino acids 20 weeks after gestation. Insulin action is regulated by glucocorticoids, which induce the expression of transcription factors. These factors regulate the expression of enzymes involved in glycogen and lipid synthesis. Insulin is inactive until the corticosteroid action begins in the second trimester. This explains why glycogen storage does not occur until 27 weeks. Glycogen synthesis begins late in pregnancy and slowly increases until 36 weeks gestation. The glycogen content in the liver then increases rapidly until it is fully delivered and reaches 50 mg / g tissue. Most gluconeogenic enzymes show significant activity in the fetal liver for a short period of time, but gluconeogenesis is inhibited by the presence of high levels of insulin and does not function in the uterus. Insulin promotes fatty acid synthesis in the liver and glucose uptake in adipose tissue, resulting in triglyceride synthesis. The human placenta is permeable to triglycerides, free fatty acids, and glycerol. Taken together, these processes allow fat to accumulate in adipose tissue during the third trimester. The fat content of term infants reaches 16% of body weight at birth. Glycogen and fat are stores that can be used for metabolic changes at birth.

## Poor regulation of blood glucose

People with diabetes frequently and persistently develop the hyperglycemia that is characteristic of diabetes. In people with type 1 diabetes who do not produce insulin, glucose remains in the plasma without the glucose-lowering effect that insulin requires. Another factor that contributes to this chronic hyperglycemia is the liver. When a diabetic person fasts, the liver secretes excess glucose and continues to secrete glucose even after blood levels reach normal levels. Another cause of chronic hyperglycemia in diabetes is skeletal muscle. After a meal, the muscles of diabetics absorb too little glucose and blood sugar levels remain elevated for extended periods of time. Metabolic dysfunction of the liver and skeletal muscle in type 2 diabetes results from a combination of insulin resistance, beta cell dysfunction, excess glucagon, and a decrease in incretins.

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**Received:** 03-Jan-2022, Manuscript No. 2572-5629-22-15550; **Editor assigned:** 05-Jan-2022, PreQC No. 2572-5629-22-15550 (PQ); **Reviewed:** 20-Jan-2022, QC No 2572-5629-22-15550; **Revised:** 24-Jan-2022, Manuscript No. 2572-5629-22-15550 (R); **Published:** 07-Mar-2022, DOI:10.35841/2572-5629-22.07.108

**Citation:** Jensen M (2022) A Brief Note on Regulation of Blood Glucose Regulation. Diabetes Case Rep. 7:108

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