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Perspective

Assessments of Glycolysis and Mechanism of Cellular Cycle in Type-2 Diabetes

Fernando Moraes*

Department of Toxicology, University of Kentucky, Lexington, USA

DESCRIPTION

Glycolysis is a simple glucose metabolism mechanism that controls insulin secretion and metabolic functions in a variety of cells. Rates of glycolysis are determined at multiple levels of glycolysis that are controlled by major metabolic and regulatory enzymes such as glucokinase, 6-phosphofructo-1-kinase, and 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase, depending on cell type. At the transcription, translation, and post-translational modification levels, these enzymes are regulated by both dietary and hormonal cues. Glycolysis is involved in the regulation of hepatic glucose synthesis in hepatocytes and excessive amounts of the latter cause hyperglycemia in diabetics. Glycolysis links glucose-stimulated insulin secretion in pancreatic cells. Hyperglycemia is caused by high or low amounts of circulating insulin. Glycolysis produces metabolites for lipogenesis in adipocytes and redirects fatty acids from excessive oxidation to triglyceride synthesis, lowering oxidative stress. Adipocytes release pro-hyperglycemic factors when their proinflammatory condition rises, leading to hyperglycemia and insulin resistance. Glycolysis transmits nutrition sensing to hypothalamic neurons, which is linked to eating control. Dysregulation of glycolysis occurs in the presence of insulin insufficiency or resistance, and is caused by insufficient amounts and/or activity of glycolysis' metabolic and regulatory enzymes. Increasing glycolysis by targeting key metabolic and regulatory enzymes could be a potential therapy option for diabetes. Glycolysis is a simple glucose metabolism mechanism that controls insulin secretion and metabolic functions in a variety of cells. Targeting key metabolic and regulatory enzymes to improve glycolysis could be a viable diabetes therapeutic intervention.

Aerobic and anaerobic glycolysis

Diabetes is a worldwide health concern caused mostly by the inability of pancreatic-cells to release enough insulin. The molecular mechanisms underlying Type-2 diabetes's gradual inability of -cells to respond to glucose are yet unknown. Type 2 diabetes is a complicated condition in which decreased insulin secretion and insulin activity contribute to hyperglycemia and a wide spectrum of metabolic abnormalities. The role of glucose metabolic pathways in the etiology of the disease is still unknown. Glucose transport and phosphorylation are the first steps in the cellular destiny of glucose. Aerobic and anaerobic glycolysis, glycogen production, and conversion to various intermediates in the hexose phosphate or hexosamine biosynthesis pathways are the next steps in the glucose use process. In diabetic people, anomalies in each pathway may occur; however, it is unknown whether these disruptions cause diabetes or are a result of the disease's many metabolic abnormalities.

The group of hexose transport proteins located on the cell membrane mediates the energy-independent transfer of glucose along its concentration gradient: facilitative glucose carriers (glucose transporters (GLUTs). GLUT4 has been investigated the most in persons with insulin resistance of all the transport proteins. GLUT4 expression appears to be normal in these participants' muscles, but targeting and trafficking appear to be impaired. Adipocytes, which also have a significant drop in GLUT4 expression in Type 2 Diabetes, show similar changes in protein regulation. Polymorphisms in the GLUT4 gene are extremely rare in Type 2 diabetes patients and have the same prevalence in non-diabetics, implying that these are population variants rather than disease causes. Exercise improves glycemic control in diabetics and lowers the risk of Type 2 diabetes in high-risk groups, while it may not be appropriate for patients with severely poor glycemic control or major underlying cardiovascular disease. By insulin-independent pathways, AMPK enhances GLUT4 translocation in response to insulin and during exercise.

CONCLUSION

Glycolysis plays a critical role in maintaining systemic glucose homeostasis. The opinion covers how glycolysis is regulated in a cell-type-dependent way, as well as major glycolysis metabolic and regulatory enzymes that could be used to treat diabetes. The relevant action mechanisms have also been highlighted. While several targets appear promising, there is still a long way to go.

Correspondence to: Fernando Moraes, Department of Toxicology, University of Kentucky, Lexington, USA, E-mail: Moraes_f@hotmail.com

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