American Journal of Biomedicine and Pharmacy
https://biojournals.us/index.php/AJBP
ISSN: 2997-7177

The Importance of Glucose in Medicine

Qurbonov Og'abek Khayrulla o'g'li
Second-year student of Tashkent State Medical University

Ne'matova Zulayho Jamol qizi
First-year student of Samarkand State Medical University

Received: 2025, 15, Oct
Accepted: 2025, 21, Nov
Published: 2025, 10, Dec

Annotation: This article provides an overview of glucose, including its properties, associated disorders, and medical applications. The physiological role of glucose in the human body is discussed, along with the consequences of its imbalance, such as hypoglycemia and hyperglycemia. The article also highlights the clinical use of glucose solutions in medicine, including their application for energy supply, hydration, detoxification, parenteral nutrition, and treatment of hypoglycemia. Different concentrations of glucose solutions (5%, 10%, 20%, and 40%) and their specific indications are explained. The study emphasizes the importance of glucose as a vital substrate for metabolic processes and its critical role in maintaining homeostasis and supporting patient care in various clinical situations.

Keywords: Glucose, energy source, hypoglycemia, hyperglycemia, hydration.

Introduction

Glucose is a 6-carbon structure with the chemical formula C6H12O6. Carbohydrates are ubiquitous energy sources for every organism worldwide and are essential to fuel aerobic and anaerobic cellular respiration in simple and complex molecular forms. Glucose often enters the body in isometric forms such as galactose and fructose (monosaccharides), lactose and sucrose (disaccharides), or starch (polysaccharides). Excess glucose is stored in the body as glycogen, a glucose polymer, utilized during fasting. In addition, glucose can be produced through gluconeogenesis, a process involving the breakdown of fats and proteins. Given the paramount importance of carbohydrates in maintaining homeostasis, numerous sources contribute to glucose production. The sugar molecule travels through the blood to energy-requiring tissues when glucose is in the body. Glucose undergoes a series of biochemical reactions, releasing energy as

adenosine triphosphate (ATP). ATP derived from these processes fuels virtually every energy-requiring process in the body. In eukaryotes, most energy derives from aerobic (oxygen-requiring) processes, which start with a glucose molecule. Glucose is initially broken down through the anaerobic process of glycolysis, producing some ATP and pyruvate as end products. Under anaerobic conditions, pyruvate converts to lactate through reduction.Conversely, under aerobic conditions, the pyruvate can enter the citric acid cycle to generate energy-rich electron carriers that produce ATP at the electron transport chain.

Glucose reserves are stored as the polymer glycogen in humans. Glycogen is present in the highest concentrations in the liver and muscle tissues. The regulation of glycogen, and thus glucose, is primarily controlled by the peptide hormones insulin and glucagon. These hormones are produced in the pancreatic islet of Langerhans—glucagon from α-cells and insulin from β-cells. The balance between these 2 hormones depends on the body's metabolic state, whether fasting or energy-rich, with insulin in higher concentrations during energy-rich states and glucagon during fasting. Through signaling cascades regulated by these hormones, glycogen is either catabolized, liberating glucose promoted by glucagon during fasting, or synthesized, further consuming excess glucose facilitated by insulin during times of energy abundance. Insulin and glucagon, among other hormones, also control glucose transport in and out of cells by altering the expression of glucose transporter type 4

Impaired Glucose Use. Although the increase in hepatic gluconeogenesis seems to be the driving force behind stress hyper glycemia, there is also impaired glucose use in peripheral tissues. Although total body glucose uptake is increased, insulin-independent tissues such as brain cells and blood cells account for the majority of the uptake. Although the insulin-independent transporters—glucose transporters (GLUTs) GLUT-1, GLUT-2, and GLUT-3—remain active in settings of stress, they cannot match the concurrent increase in hepatic glucose production. In insulin-dependent tissues, such as skeletal and cardiac muscle, as well as adipocytes, insulin is required to stimulate the GLUT-4, which leads to glucose crossing the cell membrane for metabolism. Activation of the GLUT-4 transporter is impaired insepsis and critical illness through the phosphoryla tion of various molecules along the insulin-signaling pathway. Glucocorticoids and epinephrine are known to impair GLUT-4 translocation from its internal membrane stores to the plasma mem brane, with epinephrine having an added effect of inhibiting insulin binding. Despite reduce uptake, once inside the cell, the oxidation of glucose by glycolysis and the tricarboxylic acid cycle seems to be maintained. Rates of glucose oxidation have been shown to be entirely normal in children with burn injury. Inflammatory mediators, specifically, the cyto kines TNF-, IL-1, IL-6, and CRP, also induceperipheral insulin resistance. TNF-was first linked to hyperglycemia in 1994. Hotamisligil et al36 demonstrated that TNF-mediates insulin resistance in animal models of obesity. Locally elevated TNF-directly interferes with insulin signal transduction, creating a local desensitization to insulin. Neutralization of TNF-with soluble TNF-recep tor restores insulin sensitivity in animal models, but reversal of insulin resistance by anti-TNF-has not been reproduced in humans. Other cytokines have also been linked to the development of diabetes in a correlative way. In healthy women, elevated IL-6 and CRP predict the development of type 2 diabetes. From these observations, Pickup and col leagues describe type 2 diabetes as a disease of the immune system, particularly the acute-phase response, evidenced by the finding that elevations in CRP strongly correlate with diabetes.

Use of Glucose Solutions in Medicine.Glucose solutions are typically produced in concentrations of 5%, 10%, 20%, and 40%. The choice of concentration depends on the physiological needs of the body and the patient’s clinical condition. In medicine, glucose solutions are widely used for energy supply, detoxification, hydration, parenteral nutrition, correction of hypoglycemia, and as a solvent for medications.

1. As a source of energy.Glucose solutions are a rapidly absorbable source of energy for metabolism. A 5% glucose solution is isotonic and provides energy without exerting osmotic effects on cells and tissues. Hypertonic solutions (10–40%) are used in conditions with high

energy demands, such as severe infections, burns, sepsis, and postoperative recovery. Increased glucose levels in the body are converted into pyruvate via glycolysis, which then enters the Krebs cycle and oxidative phosphorylation processes to generate ATP, providing the necessary substrate for energy production.

2. In hydration and rehydration therapy. A 5% glucose solution is used to correct fluid deficits (dehydration). It restores the body's water balance, does not impose osmotic load on the blood, and, when combined with electrolytes, helps stabilize plasma volume.

3. In the treatment of hypoglycemia. Hypoglycemia (a sharp decrease in blood glucose levels) can be life-threatening. Therefore, in medical practice, a 40% glucose solution is administered intravenously as emergency care. This provides rapid energy necessary for the central nervous system, preventing complications such as loss of consciousness or seizures.

4. In detoxification therapy. Glucose solutions play an important role in detoxification therapy. They: Stimulate the detoxifying function of the liver. Provide substrates necessary for glucuronidation processes in hepatocytes. Restore metabolic processes disrupted by intoxication.

Pathophysiology. Glucose metabolism is intricately regulated to meet cell energy demands while preventing hyperglycemia or hypoglycemia. The dysregulation of glucose homeostasis underlies the pathogenesis of several metabolic disorders, posing significant health challenges worldwide. Understanding the pathophysiology of glucose dysregulation provides insights into the molecular mechanisms driving metabolic disorders.

Type 1 diabetes: Type 1 diabetes results from autoimmune destruction of pancreatic β-cells, leading to insulin deficiency. Insulin secretion loss impairs peripheral tissue glucose uptake, resulting in hyperglycemia and metabolic derangements.

Type 2 diabetes: Type 2 diabetes primarily arises from insulin resistance, characterized by diminished cellular response to insulin. Peripheral tissues fail to efficiently utilize glucose, leading to compensatory hyperinsulinemia, β-cell exhaustion, and eventual pancreatic dysfunction.

Reactive hypoglycemia: Reactive hypoglycemia occurs postprandially due to excessive insulin secretion in response to carbohydrate-rich meals. This exaggerated insulin release leads to rapid glucose clearance, resulting in hypoglycemic episodes.

Fasting hypoglycemia: Fasting hypoglycemia can stem from various etiologies, including hormonal deficiencies, liver disease, or metabolic enzyme deficiencies, disrupting the balance between glucose production and utilization during fasting periods.

Hypoglycemia. Hypoglycemia is most commonly observed iatrogenically in patients with diabetes mellitus secondary to glucose-lowering drugs. This condition occurs, especially in the inpatient setting, with the interruption of the patient's usual diet. The symptoms are non-specific, but clinical findings such as relation to fasting or exercise and symptom improvement with glucose administration make hypoglycemia more likely. Hypoglycemia symptoms can be described as either neuroglycopenic, due to a direct effect on the central nervous system, or neurogenic, due to sympatho-adrenergic involvement. Neurogenic symptoms can be further broken down into either cholinergic or adrenergic. The following are some common symptoms of hypoglycemia.

Neuroglycopenic: Fatigue, behavioral changes, seizures, coma, and death.

Neurogenic: Adrenergic such as anxiety, tremor, and palpitations.

Neurogenic: Cholinergic such as paresthesias, diaphoresis, and hunger.

Hyperglycemia. Hyperglycemia can induce pathology, both acutely and chronically. Type 1 and 2 diabetes are both disease states characterized by chronically elevated blood glucose levels that, over time and with poor glucose control, lead to significant morbidity. Both types of diabetes

have multifocal etiologies—type 1 is associated with genetic, environmental, and immunological factors and most often presents in pediatric patients, whereas type 2 is associated with comorbid conditions such as obesity in addition to genetic factors and is more likely to manifest in adulthood. Type 1 diabetes results from the autoimmune destruction of pancreatic β-cells and insulin deficiency, whereas type 2 diabetes results from peripheral insulin resistance due to metabolic dysfunction, often in the setting of obesity. In both cases, the result is inappropriately elevated blood glucose levels, which can lead to pathology through various mechanisms.

Osmotic damage: Glucose is osmotically active and can cause damage to peripheral nerves.

Oxidative stress: Glucose participates in several reactions that produce oxidative byproducts.

Non-enzymatic glycation: Glucose can form complexes with lysine residues on proteins, leading to structural and functional disruption.

Conclusion

Glucose is a fundamental carbohydrate essential for maintaining energy homeostasis in the human body. Its metabolism provides the primary substrate for ATP production through glycolysis, the Krebs cycle, and oxidative phosphorylation, supporting virtually all energy-requiring cellular processes. Dysregulation of glucose levels can lead to pathological conditions such as hypoglycemia and hyperglycemia, which are central to metabolic disorders including type 1 and type 2 diabetes. In clinical practice, glucose solutions of varying concentrations (5%, 10%, 20%, 40%) play a crucial role in medical therapy, providing rapid energy, supporting hydration and rehydration, aiding detoxification, and serving as a solvent for medications. Understanding the physiological roles and clinical applications of glucose underscores its critical importance in patient care and metabolic regulation.

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