regulationintestinalglucoseabsorptionionchannelstransporters-chen

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- Regulation of Intestinal Glucose Absorption by Ion Channels and Transporters

Abstract

The absorption of glucose is electrogenic in the small intestinal epithelium. The major route for the transport of dietary glucose from intestinal lumen into enterocytes is the Na+/glucose cotransporter (SGLT1), although glucose transporter type 2 (GLUT2) may also play a role. The membrane potential of small intestinal epithelial cells (IEC) is important to regulate the activity of SGLT1. The maintenance of membrane potential mainly depends on the activities of cation channels and transporters. While the importance of SGLT1 in glucose absorption has been systemically studied in detail, little is currently known about the regulation of SGLT1 activity by cation channels and transporters. A growing line of evidence suggests that cytosolic calcium ([Ca2+]cyt) can regulate the absorption of glucose by adjusting GLUT2 and SGLT1. Moreover, the absorption of glucose and homeostasis of Ca2+ in IEC are regulated by cation channels and transporters, such as Ca2+ channels, K+ channels, Na+/Ca2+ exchangers, and Na+/H+ exchangers. In this review, we consider the involvement of these cation channels and transporters in the regulation of glucose uptake in the small intestine. Modulation of them may be a potential strategy for the management of obesity and diabetes.

Introduction:

a. Purpose of the study is to understand the role of ion channels and transporters in regulating glucose absorption in the intestine.

b. Importance of understanding this process for developing targeted therapies for diabetes and other glucose-related disorders.

Ion Channels:

a. Sodium-glucose cotransporter 1 (SGLT1) is responsible for the majority of glucose absorption in the intestine.

b. Potassium-dependent glucose transporters (GLUT2 and SGLT1) are involved in facilitated diffusion of glucose across the brush border membrane.

c. Cl^-/HCO3^- exchangers (AE1, AE2, and AE3) help maintain electrochemical gradients for glucose absorption.

d. Chloride channels (ClC-7) regulate glucose transport by modulating the activity of SGLT1.

Transporters:

a. Glucose transporter type 2 (GLUT2) is responsible for glucose sensing in pancreatic alpha cells and regulation of insulin secretion.

b. Sodium-glucose cotransporters (SGLT1, SGLT3, and SGLT4) are involved in the absorption of glucose from the intestinal lumen into enterocytes.

c. SGLT2 is expressed in the kidney proximal tubules and responsible for reabsorption of glucose in the urine.

d. GLUT5 is a high-affinity glucose transporter found in the brush border membrane of enterocytes, responsible for the rapid absorption of glucose.

Regulation of Glucose Absorption:

a. Intracellular glucose concentration regulates SGLT1 activity through feedback inhibition and allosteric activation.

b. GLUT2 is regulated by intracellular glucose concentration, which modulates insulin secretion in pancreatic alpha cells.

c. Glucagon-like peptide 2 (GLP-2) promotes intestinal growth and increases the expression of SGLT1 and GLUT2.

d. Bile acids regulate glucose absorption by modulating the activity of SGLT1 and GLUT2.

Conclusion:

a. Ion channels and transporters play a crucial role in regulating intestinal glucose absorption.

b. Understanding their function can help develop targeted therapies for diabetes and other glucose-related disorders.

c. Further research is needed to elucidate the molecular mechanisms underlying these processes.

Key Takeaways:

1. Ion channels (SGLT1, GLUT2) and transporters (GLUT2, SGLT1, SGLT3, SGLT4, AE1, AE2, AE3, ClC-7) are involved in glucose absorption in the intestine.
2. Intracellular glucose concentration regulates ion channels and transporters activity.
3. GLP-2 promotes intestinal growth and increases SGLT1 and GLUT2 expression, while bile acids modulate their activity.