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Perspective of Channelopathy in Diabetes Mellitus

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ABSTRACT

Ion channels, which are found in the membrane of all cells and several cellular organelles, can malfunction, causing a variety of different diseases known as channelopathies. One such example is ion channels, which are also crucial for controlling electrical activity and the signal transduction pathway that controls insulin. The route will signal for an increase in glucose transporters in the cell membrane if the hormone is required by muscle cells to support higher physical activity. As a result, this review investigates the channelopathy in diabetes mellitus.

Keywords: Perspective; Channelopathy Diabetes Mellitus

Introduction

Ion channel malfunction is a medical ailment known as channelopathy. It is known that ion channels for potassium, sodium, chloride, and calcium are involved in channelopathy. The underlying molecular mechanisms of increased muscle membrane excitability have recently become more understandable in a group of illnesses known as channelopathies [1]. It is a heterogeneous set of diseases brought on by ion channel malfunction. Ion channels are found in the membranes of all cells and many cellular organelles. Channelopathy is connected to endocrine system disorders (for instance, family hyperaldosteronism, thyrotoxic hypokalemic periodic paralysis, familial hyperinsulinemic hypoglycemia, and neonatal diabetes mellitus)[2].The pancreatickatp plays a crucial function in maintaining the homeostasis of glucose by connecting insulin production, electrical excitability, and glucose metabolism. The metabolic modulation of channel activity is mediated by alterations in the intracellular ATP/ADP ratio [3].

By connecting electrical activities and cell metabolism, KATP channels play significant roles in a variety of cellular processes. It is believed that the pancreatic cell's KATP channels play a key role in controlling insulin production that is sulfonylurea- and glucose-induced [4].

Ion Channels

Ion Channels are membrane proteins that play a key part in controlling circular excitability. They are present in almost all cells and, based on the stimuli they respond to, are of negligible physiological significance. Voltage-gated, ligand-gated, and mechanosensitive ion channels are the three families of ion channels.

a. Voltage-gated: Voltage-gated ion channels are highly selective for one particular ion, such as Na+, K+, Ca2+, or Cl-, and they are further categorized into families based on the primary permeate ion. They react to changes in cell membrane potentials.

b. ion channels that are ligand-gated: Anesthetics and other medications target LGIC.

c. They are categorized into three families based on molecular biology and protein, and are named based on the ligand to which they respond.

d. Mechano-sensitive ion channels play a key role in the detection and conversion of external mechanical forces into electrical and chemical intracellular signals. These ion channels respond to changes in mechanical forces acting on the cell membrane [5].

Ion channels that are mechanosensitive control blood pressure, cell volume, the development of muscles and bones, as well as the perceptions of touch and hearing. Studies have revealed that these channels play a role in a number of conditions, including tumor metastasis, polycystic kidney disease, muscular dystrophy, and cardiac arrhythmias [6].

Diabetes Mellitus

This relates to a class of conditions that impact how your body utilizes blood sugar (glucose). Since the cells that comprise our muscles and tissues rely on glucose as a major source of energy, glucose is crucial to your health. Our brains mostly use it as fuel. Diabetes has a variety of underlying causes. However, diabetes can result in an excess of sugar in the blood regardless of the kind that a person has. Serious health issues might result from high blood sugar levels. Type 1 diabetes and type 2 diabetes are chronic illnesses associated with diabetes. Gestational Diabetes (Pregnancy associated Diabetes 190 mg/dl) and Pre-Diabetes (Before Diabetes 100 to 125 mg/dl) [7]. When our blood sugar level is greater than usual but not high enough to be diagnosed as diabetes, pre diabetes is present. And unless proper steps are made to stop progressions, Pre Diabetes frequently leads to Diabetes. Pregnancy-related gestational diabetes can go away once the baby is delivered [8].

Diabetes Mellitus Symptoms

Depending on how much our blood sugar is high, different diabetes symptoms may be present. Although type 1 diabetes usually manifests more swiftly and severely than type 2 diabetes, with the following symptoms: heightened thirst, extreme hunger, frequent urination, and an unexplained weight loss, fatigue, Ketons Can Be Found in Urine (Ketons are by-products of breakdown of muscles and fat that happens when there is not enough available insulin) and Slow recovery ruins (due to affected platelets by some bacteria). Type 1 diabetes can arise at any age, but most frequently does so in childhood or adolescence. Type 2 diabetes, on the other hand, can appear at any age but is more common in those over 40 [9].

Causes of Type 1 Diabetes

Although the exact cause of type 1 diabetes is unknown, research has revealed that the immune system that protects us from harmful bacteria or viruses instead targets and kills our insulin-producing cells. As a result, when we have little to no insulin, glucose builds up in our bloodstream instead of being transported into the cells. In addition, a mix of genetic predisposition and environmental variables contribute to type 1 diabetes [10].

Causes of Pre Diabetes and Type 2 Diabetes

It is a dangerous condition that has historically been common in western society. One of the main causes of this disease's expression is glucose intolerance, or the inability of glucose to stimulate insulin secretion. This is probably because glucose metabolism does not promote pancreatic beta cell electrical activity, calcium influx, or insulin secretion. The cells become resistant to the action of insulin in pre diabetes, which can progress to type 2 diabetes, and our pancreas is unable to produce enough insulin to overcome this resistance. Glucose accumulates up in our bloodstream rather than entering the cells where it is needed for energy. Although the cause of this is unknown, type 2 diabetes is thought to be influenced by both genetic and environmental factors. Type 2 diabetes and being overweight are significantly correlated, but not all people with type 2 diabetes are overweight [11].

Channelopathy And Diabetes Mellites

Potassium KATP channels connect the metabolism produced inside the cells to changes in the beta-cells membrane potential and ultimately govern insulin production. Ion channels play a crucial function in excitable cells such as cardiac, skeletal and smooth muscles cells, neurons, and endocrine cells. Disorders of glucose homeostasis result from mutations in the genes encoding a few ion transporters and channel proteins (Hyperinsulinemic hyperglycemic and different forms of diabetes mellitus) [12]. Different types of hyperinsulinemic hypoglycemia can be caused by pancreatic KATP and Non KATP, certain calcium channelopathies, and MCT 1 transporter defects (HH). Different types of diabetes, such as Neonatal Diabetes Mellitus (NDN) and Maturity Onset of the Young (MODY), are also caused by mutations in the pancreatic KATP channel gene, and the Falconi-Bickel syndrome is associated with deficiencies in the solute carrier family 2 member 2 (SLC2A2) .In various ion channel genes and transporter genes, variations or polymorphisms have been linked to type 2 diabetes mellitus [13].

Conclusion

Adenosine nucleotides control adenosine-triphosphate-sensitive potassium channels (KATP), which relate cellular metabolism with electrical activity in a variety of organs, including the pancreatic beta-cell.The proof that inactivating and activating mutations in KATP are responsible for persistent hyperinsulinemia and neonatal diabetes mellitus, respectively, in both animal models

and people, supports KATP's crucial role in insulin production. A frequent KATP variation also acts as a risk factor in the development of type 2 diabetes.

References

1. Nnodim JK, Bako H, Ezekwesiri C (2021) Perspective of Ion Channels in Prostate Cancer Sumerianz. Journal of Medical and Healthcare 4(2): 69-72.
2. Marchetti P, Masini M (2009) Autophagy and the pancreatic beta-cell in human type 2 diabetes. Autophagy 5: 1055–1056.
3. Nnodim JK, Tsegba AP (2021) Perspective of membrane potential in medical laboratory diagnosis. EMWPL. International Journal of Medical Physiology and Therapeutics 1(1): 18-28.
4. Mellado-Gil JM, Cobo-Vuilleumier N, Gauthier BR (2012) Islet beta-cell mass preservation and regeneration in diabetes mellitus: four factors with potential therapeutic interest. J Transplant p 230870.
5. Huseyin D, Sonya G, Dogus V, Sara A, Khalid H (2019) Beta Cell and Insulin Secretion. Journal of molecular sciences 20(10): 2590.

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