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2nd International Conference on

DIABETES, ENDOCRINOLOGY, NUTRITION AND NURSING MANAGEMENT

June 24-25, 2019 | Philadelphia, USA

J Diabetol 2019, Volume 3

INSULINE RESISTANCE–PATHOGENESIS, PREVENTION AND TREATMENT

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The mechanism of development of insulin resistance (IR) is not clear. This makes it difficult to develop adequate ways to prevent and treat type 2 diabetes mellitus (2D). There is no free glucose in the muscle tissue, upon admission it is immediately phosphorylated to glucose-6-phosphate (G-6-F), which prevents its return. With a decrease in the rate of G-6-F conversion, hexokinase is inhibited and the intake of glucose into the muscles decreases. This regulatory process can be considered as the first mechanism of development of IR. The next phosphorylation already produces fructose-6-di-phosphate, which is also a regulatory molecule, and its accumulation will also inhibit the absorption of glucose (the second stage of development of IR). The next regulatory step is the process of assimilation of pyruvic acid or the so-called pyruvate block, since a decrease in anaerobic or aerobic conversion of pyruvate promotes inhibition of glycolysis and the development of IR. The next step in the regulation of glucose conversion is ATP or the level of utilization of the energy of its oxidation. The most volatile process in the muscle cell is protein synthesis, so the amount of glucose utilization will directly correlate with the rate of protein synthesis. With a decrease in protein synthesis with a substrate deficit or inhibition of the protein of the synthesizing apparatus, the utilization of ATP decreases and the ATP/ADP coefficient increases, which contributes to the inhibition of hexokinase and the development of IR. Such mechanism of development of IR will allow developing effective ways of developing the principles of prevention and treatment of patients with 2D.