

Pathology: A Review

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of the nuclei of the medulla oblongata initiates an increase in stroke

- the latter is the GLU.

The formation of this sequence happened back in prokaryotes, mitochondria, and according to the reception of "biological chain of command" and biological "prohibition" of evolution, cannot be changed [43].

ANN will strengthen not only the activated (passive) uptake by cells through the glucose transporters GLU 4 (GLYUT4), but the GLU and oxidation in mitochondria, if not in the cytosol or ketone bodies or fatty acids in the form of polar NEFA. To mitochondrial oxidation started GLU, insulin has to block lipolysis in insulin-dependent cells and lower the levels in the cytosol of the LCD, and their metabolites [44]. In exotrophy biological response when postprandial hyperglycemia and hyperinsulinemia INS: a) inhibits lipolysis b) depriving mitochondria possible to oxidize ketones and short-LCD, c) facilitates cellular uptake of GLU and d) its oxidation in the mitochondria. Simultaneously, the LCD cells are deposited in the form of triglycerides to provide energy for the biological function of locomotion. INS acts only exotrophy biological response. Consequently, the INS activates GLU oxidation in cells by regulating the metabolism of the LCD, so diabetes can reasonably be called metabolic disorders LCD.

The main cause of the "inaction" of ANN is the formation in vivo physiological processes at the level of communities paracrine cells in which the phylogenetically earlier hormones activate lipolysis in cells

the conditions in which there was a formation of biologically conservative foundations metabolism, biological functions and

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