BACKGROUND:

Excess dietary fructose intake associated with metabolic syndrome and insulin resistance and increased risk of developing type 2 diabetes. Previous animal studies have reported that diabetic animals have significantly impaired behavioural and cognitive functions, pathological synaptic function and impaired expression of glutamate receptors. Correction of the antioxidant status of laboratory rodents largely prevents the development of fructose-induced plurimetabolic changes in the nervous system. We suggest a novel concept of efficiency of Stevia leaves for treatment of central diabetic neuropathy.

METHODS:

By in vivo extracellular studies induced spike activity of hippocampal neurons during high frequency stimulation of entorhinal cortex, as well as neurons of basolateral amygdala to high-frequency stimulation of the hippocampus effects of Stevia rebaudiana Bertoni plant evaluated in synaptic activity in the brain of fructose-enriched diet rats. In the conditions of metabolic disorders caused by fructose, antioxidant activity of Stevia rebaudiana was assessed by measuring the NOX activity of the hippocampus, amygdala and spinal cord.

RESULTS:

In this study, the characteristic features of the metabolic effects of dietary fructose on synaptic plasticity in hippocampal neurons and basolateral amygdala and the state of the NADPH oxidase (NOX) oxidative system of these brain formations are revealed, as well as the prospects for development of multitarget and polyfunctional phytopreparations (with adaptogenic, antioxidant, antidiabetic, nootropic activity) from native raw material of Stevia rebaudiana. Stevia modulates degree of expressiveness of potentiation/depression (approaches but fails to achieve the norm) by shifting the percentage balance in favor of depressor type of responses during high-frequency stimulation, indicating its adaptogenic role in plasticity of neural networks. Under the action of fructose an increase (3-5 times) in specific quantity of total fraction of NOX isoforms isolated from the central nervous system tissue (amygdala, hippocampus, spinal cord) was revealed. Stevia exhibits an antistress, membrane-stabilizing role reducing the level of total fractions of NOX isoforms from central nervous system tissues and regulates NADPH-dependent O_2^- -producing activity.

CONCLUSION:

Generally, in condition of metabolic disorders caused by intensive consumption of dietary fructose Stevia leaves contributes to the control of neuronal synaptic plasticity possibly influencing the conjugated NOX-specific targets.

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