Regulatory Effects of Stevia Rebaudiana on NADPH Oxidase-Related Manifestations of Oxidative Stress in Diabetic Rats with Spinal Cord Injury

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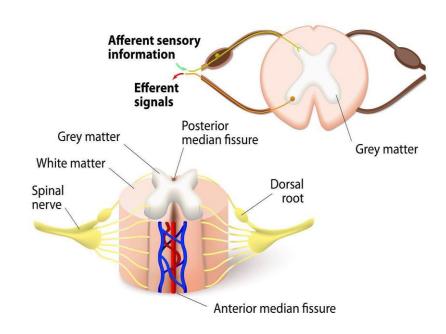
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Introduction

Spinal cord injury induces both acute and chronic inflammation, activation of microglia, invasion of macrophages, and activation of Nox in the cord. This enzyme, intensely expressed in microglia and macrophages after SCI, is an important primary source of reactive oxygen species (ROS).

Prolonged dietary introduction of fructose results in the development of type II experimental diabetes. Stevia preparation was found to significantly modulate the degree of potentiation/depression in the above neurons by shifting the balance (in favor of a depressor type of the responses induced by high-frequency stimulation), which is indicative of its adaptogenic role in the plasticity of neural networks. Stevia also exhibited anti-stress and membrane-stabilizing effects by reducing the level of total fractions of Nox isoforms from the CNS tissues and regulated its NADPH-dependent O2—producing activity.

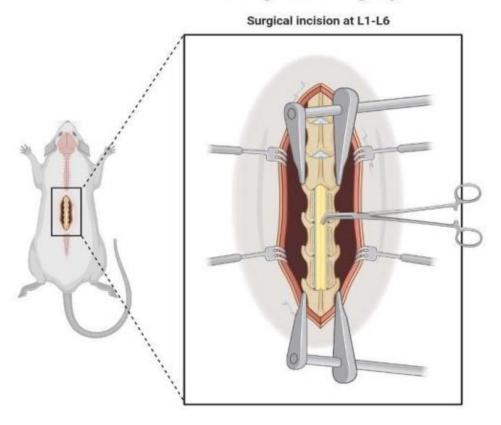
SPINAL CORD

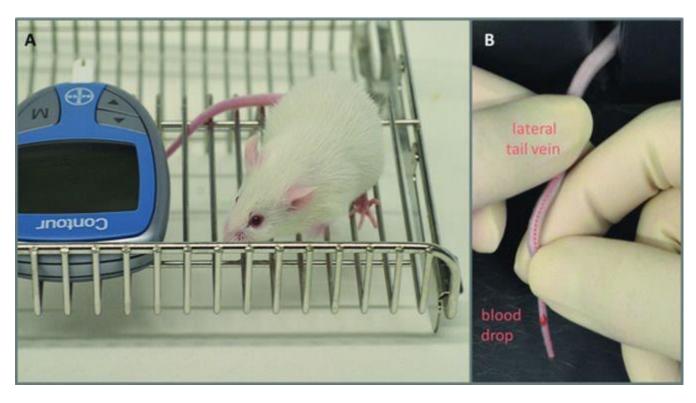


Methods

Lateral hemisection (right half) of the spinal cord was performed at the lumbar level (L4–L5) on the 6th week of the experimental period under aseptic conditions.

Rat Spinal Surgery





Results

It was found that there was a strong positive correlation between the intensity of lipid peroxidation of the spinal cord membranes and elimination of associates and Nox from these membranes. Stevia-enriched diet provides a positive effect by reducing the stationary concentration of O2–



Discussion

Stevia phytopreparation introduced in the given mode significantly reduces the stationary concentration of produced O2— and the intensity of lipid peroxidation provided by these radicals of the spinal cord membranes. Stevia is a dietary supplement with considerable antioxidant action and a corresponding ability to reduce ferrihemoglobin- induced cleavage from the membranes of the spinal cord as O2—producing associates and Nox1+Nox2 isoform.