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Intranasal insulin effects on brain insulin level and peripheral glucose and insulin concentrations in type 2 diabetes

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Background and Objective: Information from previous studies about insulin concentrations in the brain of diabetic animals is controversial. Insulin action in the brain has been shown to affect the metabolism of the periphery. These observations have led to intranasal insulin (INI) being investigated as a possible therapeutic method.
Materials and Methods: Diabetes was induced by a single intraperitoneal injection (45 mg/kg) of streptozotocin (STZ) on day 1. Insulin and saline were given intranasally from day 4 to 14. We measured the brain insulin concentration on day 15, and the serum insulin and glucose concentrations on days 0, 7 and 14.
Results: In the current study, brain and serum insulin concentrations were ~12 ± 0.38 ng/ml and 1.2 ± 0.06 ng/ml, respectively. In our experimental type 2 diabetes mellitus model (T2DM), we observed that brain and serum insulin levels were ~31± 2.7 ng/ml and ~3.01± 0.38 ng/ml, respectively. Our results demonstrate that INI delivery raises the brain insulin roughly ~12.5 and 6.54 times higher in control and diabetic groups compared to intranasal saline groups. In the current study, as in control rats, repeated INI delivery (11 days) increased serum insulin under diabetic condition (~1.5 folds). INI delivery also decreases the serum glucose in diabetic rats.
Conclusion: The results of this study showed that INI delivery increased the levels of insulin in the brain and in the serum. INI delivery also reduced the serum glucose, probably through central mechanisms, in diabetic animals.
Keywords: Intranasal insulin, type 2 diabetes, Brain, Insulin level, Rats

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The effect of safranal in prevention of cognitive decline in intracerebroventricular streptozotocin model of Alzheimer's disease in the rat

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Background and Objective: Cognitive decline is associated with Alzheimer's disease (AD) that is a chronic and progressive syndrome with a neurodegenerative nature that finally leads to irreversible deterioration of neurons. In this study, we evaluated whether safranal could prevent cognitive decline in intracerebroventricular streptozotocin (STZ)-induced model of AD in the rat.
Materials and Methods: Male rats (n=32) were assigned to four groups, i.e., Sham, lesion (receiving intracerebroventricular STZ bilaterally at a dose of 3 mg), and two lesion groups receiving safranal p.o. at doses of 10 or 50 mg/kg in addition to intracerebroventricular STZ. Finally, performance of rats in passive avoidance and Y-maze tests was assessed to explore cognition.
Results: Our obtained data indicated that intracerebroventricular STZ is associated with significant dysfunction in Y-maze and passive avoidance tasks and administration of safranal to intracerebroventricular STZ group at a dose of 50 mg/kg significantly improves performance of animals in these tests.
Conclusion: Collectively, safranal at a dose of 50 mg/kg could significantly attenuate cognitive dysfunction induced by intracerebroventricular injection of STZ in the rat.
Keywords: Streptozotocin, Safranal, Cognition