INTRODUCTION

The pancreatic islets in mice undergo a significant remodeling in response to taurine. In this experiment, we tested the effect of taurine on the function of the pancreas. Mice and pigeons were administered a high-energy diet (basal diet supplemented with 5% taurine; and with or without taurine). Mice were fed a diet ad libitum; pigeons were force-fed. The results showed a significant increase in plasma glucose levels in the taurine-fed group. In addition, after 24 hours of water consumption, control (taurine-fed) animals showed a significant increase in plasma glucose levels compared to PBS-treated animals. Taurine-fed mice and pigeons showed significantly lower plasma glucose compared to controls. Taurine supplementation in drinking water resulted in a significant increase in the number and size of the pancreas increased in glucagon and somatostatin immunoreactivity levels in the pancreas. Studies showed that mice from taurine-supplemented mice showed a significant increase in the number of PCNA immunopositive cells. This increased proliferation was accompanied by an increase in the incidence of apoptosis in glandular cells, and also a significant increase in the number of immunopositive PCNA/CD cells. Taurine supplementation, which is important for developing pancreatic development, may reduce cytokine-mediated apoptosis in pancreatic β-cells. This in turn increases the size of pancreatic islets and confers tolerance to glucose and resistance to glucose-induced hyperglycemia.

METHODS

Intestinal glucose tolerance test: Mice from both groups were fasted overnight (12h) and then injected intraperitoneally with 0.6 ml/kg body weight of glucose (15% stock solution in saline). Blood samples were taken by tail cannulation at 0, 10, 30, 60, and 120 min intervals after the glucose load. Glucose was measured with Accu-check portable glucose meter (Roche, Germany). Mice were given only water during the test.

Expression of glucose transporter, insulin receptor, and their role in bioenergetics:

RESULTS

Effect of taurine supplementation on glucose homeostasis. Immunoreactive insulin response to overnight fasted control (C), and taurine-supplemented (T) mice are shown in the graph. Control mice received saline solution (1 ml) and taurine mice received 5% taurine solution (1 ml) intraperitoneally. In the blocking step, the slides were rinsed in an antibody dilution cocktail (CAB), consisting of 2% BSA and 1% PBS in EBM-2. Primary antibodies (Chemicon International) were applied directed against insulin receptor (mouse, 1:500) and Glut 3 (mouse, 1:200). The primary antibody was incubated overnight at 4°C and then unbound antibodies were washed with EBM-2. Secondary antibodies were applied in the goat and directed against appropriate primary antibody type, images were obtained using confocal microscopy (Carl Zeiss Axioskop). Nuclei were counter-stained with Hoechst containing DAPI (Invitrogen).

Effect of taurine supplementation on insulin receptor expression in the hippocampus and pancreas. Upper panel depicts the brains reconstruction of the views from representative coronal sections showing insulin receptor (green) immunoreactivity in CA1 region of the hippocampus from control and taurine-fed mice. Lower panel shows an enlargement of a single-diluted frame. Taurine supplementation increases insulin receptor expression in the hippocampus and pancreas from taurine-fed mice show a significant increase in immunoreactivity for insulin receptors.

CONCLUSIONS

- Sustained dietary intake of taurine in drinking water improved glucose handling in both pigeons and mice.
- Pancreas of taurine-fed mice have alterations in proteins relevant to the function of the GABAergic synapses that are common to the brain and pancreas.
- The functional consequences of the altered expression of these proteins in the pancreas is altered glucose homeostasis. In the brain, this altered expression leads to hyperglycemia and other adaptive mechanisms.
- Chronic supplementation of taurine in drinking water results in significant increase in calbindin and Na-K ATPase expression with a concomitant decrease in VSCC expression. These biochemical changes are consistent with protective mechanisms against excitability.

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