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The maximal activity of phosphate-dependent glutaminase and glutamine metabolism in the colon and the small intestine of streptozotocin-diabetic rats.

Ardawi MS.

Abstract

The effects of short- and long-term diabetes on the maximal activities of phosphate-dependent glutaminase and glutamine metabolism were studied in the colon and the small intestine of streptozotocin-diabetic rats. The maximal activity of colonic phosphate-dependent glutaminase was decreased [44% in mucosal scrapings (p less than 0.01); 29% in whole colon (p less than 0.001)] or unchanged in short- or long-term diabetes respectively. That of the small intestine was increased in both short- (110%) and long-term (200%-500%) diabetes; insulin treatment corrected this increase. Acute insulin-deficiency (using anti-insulin serum) resulted in the increase (18%, p less than 0.05) of the activity of only intestinal glutaminase. Chemically-induced acidosis and alkalosis decreased (46%, p less than 0.001) and increased (24%, p less than 0.001), respectively, the activity of intestinal glutaminase, but had no effect on the colonic enzyme. Changes in glutaminase of the enlarged colon and small intestine were only detectable when activities were measured in whole organ. Arteriovenous-difference measurements showed diminished metabolism of plasma glutamine by the gut which correlated with the duration of the state of diabetes, and was accompanied by enhanced release by skeletal muscle and increased uptake by both kidney and liver. It is concluded that insulin is directly or indirectly involved in the regulation of glutamine metabolism of the gut.