Clin Sci (Lond). 1990 Nov;79(5):483-90.

Metabolic regulation of renal gluconeogenesis in response to sepsis in the rat.

Ardawi MS, Khoja SM, Newsholme EA.

Department of Clinical Biochemistry, College of Medicine and Allied Sciences, King Abdulaziz University, Jeddah, Saudi Arabia.

Abstract

1. The regulation of renal gluconeogenesis was studied in rats made septic by a caecal ligation and puncture technique. 2. Blood glucose concentrations were not markedly different in septic rats, but lactate, pyruvate and alanine concentrations were markedly increased, compared with sham-operated rats. Conversely, blood ketone body concentrations were significantly decreased in septic rats. Both plasma insulin and glucagon concentrations were markedly elevated in response to sepsis. 3. The maximal activities of glucose-6-phosphatase (EC 3.1.3.9), fructose-1,6-bisphosphatase (EC 3.1.3.11), pyruvate carboxylase (EC 6.4.1.1) and phosphoenolpyruvate carboxykinase (EC 4.1.1.49) were markedly decreased in kidneys obtained from septic rats, suggesting diminished renal gluconeogenesis. 4. Renal concentrations of lactate, pyruvate and other gluconeogenetic intermediates were markedly elevated in septic rats, whereas those of acetyl-CoA and fructose 2,6-bisphosphate were decreased and unchanged, respectively. 5. The rate of gluconeogenesis from added lactate, pyruvate and glycerol was decreased in isolated incubated renal tubules from septic rats. 6. Sepsis decreased the arteriovenous concentration difference for glucose, lactate, and alanine. Septic rats showed decreased net rates of glucose production and net rates of removal of lactate and alanine as compared with sham-operated controls. 7. It is concluded that the diminished capacity for renal gluconeogenesis in septic rats could be the result of changes in the maximal activities or regulation of key non-equilibrium gluconeogenic enzymes or both, but the effect of other factors (e.g. toxins) has not been excluded