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Metabolic control of hepatic gluconeogenesis in response to sepsis.

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Abstract

The regulation of hepatic gluconeogenesis was studied in rats made septic by cecal-ligation and puncture technique. Blood glucose was not significantly different in septic rats, but lactate, pyruvate, and alanine were markedly increased. Conversely, blood ketone body concentrations were markedly decreased in septic rats. Both plasma insulin and glucagon were markedly elevated in septic rats. The maximal activities of glucose 6-phosphatase, fructose 1,6-biphosphatase, pyruvate carboxylase, and phosphenolpyruvate carboxykinase were decreased in livers obtained from septic rats suggesting a diminished hepatic gluconeogenesis. Hepatic concentrations of lactate, pyruvate, and other gluconeogenic intermediates were markedly increased in septic rats, whereas those of fructose 2,6-bisphosphate and acetyl-CoA were decreased. The rate of gluconeogenesis from added lactate, pyruvate, alanine, and glutamine was decreased in isolated incubated hepatocytes from septic rats. It is concluded that the diminished capacity of hepatic gluconeogenesis of septic rats could be the result of changes in the maximal activities or regulation of key nonequilibrium gluconeogenic enzymes or both but do not exclude other factors (e.g., toxins).