Emagrecimento. Dieta pobre em carboidrato e rica em gordura saturada ou jejum prolongado aumenta a atividade da PDH kinase e diminui a atividade da PDH.

No final teremos diminuição da produção de ATP com todos os efeitos colaterais deste tipo de dieta. Temos aqui uma das explicações dos efeitos colaterais da Dieta de Atkins.

Studies of the long-term regulation of hepatic pyruvate dehydrogenase kinase.

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The administration of a low-carbohydrate/high-saturated-fat (LC/HF) diet for 28 days or starvation for 48 h both increased pyruvate dehydrogenase kinase (PDHK) activity in extracts of rat hepatic mitochondria, by approx. 2.1-fold and 3.5-fold respectively. ELISAs of extracts of hepatic mitochondria, conducted over a range of pyruvate dehydrogenase (PDH) activities, revealed that mitochondrial immunoreactive PDHKII (the major PDHK isoform in rat liver) was significantly increased by approx. 1.4-fold after 28 days of LC/HF feeding and by approx. 2-fold after 48 h of starvation. The effect of LC/HF feeding to increase hepatic PDHK activity was retained through hepatocyte preparation, but was decreased on 21 h culture with insulin (100 micro-i.u./ml). A sustained (24 h) 2-4-fold elevation in plasma insulin concentration in vivo (achieved by insulin infusion via an osmotic pump) suppressed the effect of LC/HF feeding to increase hepatic PDHK activity was retained through hepatocyte preparation, but was decreased on 21 h culture with insulin (100 micro-i.u./ml). A sustained (24 h) 2-4-fold elevation in plasma insulin concentration in vivo (achieved by insulin infusion via an osmotic pump) suppressed the effect of LC/HF feeding so that hepatic PDHK activities did not differ significantly from those of (insulin-infused) control rats. The increase in hepatic PDHK activity evoked by 28 days of LC/HF feeding was prevented and reversed (within 24 h) by the replacement of 7% of the dietary lipid with long-chain omega-3 fatty acids. Analysis of hepatic membrane lipid revealed a 1.9-fold increase in the ratio of total polyunsaturated omega-3 fatty acids to total mono-unsaturated fatty acids. The results indicate that the increased hepatic PDHK activities observed in livers of LC/HF-fed or 48 h-starved rats are associated with long-term actions to increase hepatic PDHKII concentrations. The long-term regulation of hepatic PDHK by LC/HF feeding might be achieved through an impaired action of insulin to suppress PDHK activity. In addition, the fatty acid composition of the diet, rather than the fat content, is a key influence.

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