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# Effects of streptozotocin and dietary fructose on delta-6 desaturation in spontaneously hypertensive rat liver.

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## Abstract

We have investigated the effects of hypertension associated with diabetes mellitus on polyunsaturated fatty acid biosynthesis. For this purpose, two rat models for these pathologies have been established: a type 1 diabetic hypertensive model obtained by streptozotocin injection to spontaneously hypertensive rat (SHR), followed or not by insulin treatment (experiment 1); a type 2 diabetic hypertensive model by feeding SHR with a fructose enriched diet (experiment 2). Liver gene expression of delta-6 desaturase (D6D), microsomal D6D activities and fatty acid composition of total lipids were estimated. In experiment 1, an increase of linoleic acid (18:2 n-6) level was observed in the streptozotocin group. D6D gene expression appeared depressed in both experimental groups. Insulin did not reverse the streptozotocin effect in SHR, as it does in insulin-dependent diabetic rats. In experiment 2, the results showed a decrease of 18:2 n-6 and of long chain products of desaturation in rats fed on fructose diet. Delta-6 n-3 desaturase activity was significantly increased, whereas gene expression tended to decrease. Feeding fructose induced a significant increase in delta-9 desaturated products, suggesting a stimulation of stearoyl-CoA desaturase. These changes in monounsaturated fatty acids strongly differ from those observed in the streptozotocin experiment, indicating that the effects on lipogenesis of hypertension linked to diabetes differ according to the type of diabetes. Then, these results indicate that the liver steatosis observed during genetic hypertension was reinforced by fructose feeding. All together, the present results showed that hypertension associated to type 1 or type 2 diabetes exacerbated the damage caused by diabetes or hypertension alone on liver lipid metabolism. The metabolic effects induced by fructose being very similar to those found in human NIDDM, SHR fed a fructose-rich diet appears to be an appropriate model for studying the consequences of the combination of hypertension and NIDDM in the metabolic syndrome diseases.

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