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Evaluation of the chronic complications of diabetes in a high fructose diet in rats.

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The increasing prevalence of type 2 diabetes is associated with increasing health costs, especially for the treatment of cardiovascular disease. The development of new treatment modalities requires animal models that mimic the range of pathophysiological changes seen in diabetic humans. Dietary fructose intake has been linked to the increase in insulin resistance as part of the metabolic syndrome; fructose-fed rats develop type 2 diabetes. This study has characterized the cardiovascular changes in young adult male Wistar rats fed a 61% fructose diet for 16 weeks. Our results extend the reported changes of hypertension, lipid abnormalities, impaired glucose tolerance and impaired oxidative defense to include ventricular dilatation with hypertrophy and decreased contractile function, together with increased inflammatory cell infiltration into the ventricular myocardium, resulting in excessive collagen deposition and an increased stiffness of the left ventricle. However, endothelial dysfunction, tactile allodynia as a symptom of peripheral neuropathy and retinopathy are not present in these rats, in contrast to the streptozotocin-induced model of type 1 diabetes. Thus, fructose feeding mimics many, but not all, of the symptoms of type 2 diabetes in humans.

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