Auton Autacoid Pharmacol. 2004 Apr;24(2):29-35.

Fructose overload modifies vascular morphology and prostaglandin production in rats.

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Abstract

1. A fructose (Fru)-enriched diet induces a mild increase in blood pressure associated with hyperglycaemia, hypertriglyceridaemia, and insulin resistance, resembling the human 'syndrome X', being an useful model to study hypertension and type 2 diabetes. 2. A sustained elevation of blood pressure is associated with cardiovascular structural modifications such as left ventricular hypertrophy and increased wall thickness:lumen diameter ratio in blood vessels. 3. Prostanoids (PR), metabolites of arachidonic acid through the cyclooxygenase pathway, include vasoactive substances synthesized and released by the vessel walls. 4. The aim of the present study was to analyse, in Fru-treated rats: (i) the morphology of mesenteric vessels and; (ii) the PR production in aorta and mesenteric vessels, in order to assess whether these parameters are related with the haemodynamic alterations observed in this experimental model. 5. Blood pressure, glycaemia and triglyceridaemia, were significantly elevated in both (4 and 22 weeks) Fru-treated groups. Meanwhile body and heart weight as well as insulinaemia were similar between experimental animals and controls. 6. The mesenteric vessels of Fru-treated rats (22 weeks) showed an increased thickness and area of the media when compared with the controls; meanwhile, the lumen diameter was similar in both groups. 7. The Fru treatment for 4 weeks did not modify PR production in aorta, whereas in the mesenteric bed it diminished prostaglandin (PG) E(2) release significantly compared with the controls. However, in the group treated for 22 weeks, Fru reduced PGI(2) production in the aorta, as assessed by 6-keto-PGF(1)alpha measurements. Meanwhile, in the mesenteric bed, the chronic Fru treatment decreased PGE(2) release but, rather surprisingly, increased the output of PGI(2) when compared with its corresponding controls. 8. In conclusion, the present study shows the existence of an alteration in the morphology of mesenteric vessels in Fru-treated rats, which could be related to an increase in peripheral resistance and the consequent mild hypertension observed in this model. However, a diminished release of vasodilator PRs, such as PGE(2) in mesenteric vessels at 4 and 22 weeks and PGI(2) in aorta at 22 weeks could further impair the vessel response. The increase in PGI(2) observed in the chronic group in mesenteric vessels could be attributed to a compensatory mechanism.

PMID: 15458541 [PubMed - indexed for MEDLINE]