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Sodium molybdate prevents hypertension and vascular prostanoid imbalance in fructose-overloaded rats

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Abstract

(1) Fructose (F) overload produces elevated blood pressure (BP), hyperglycaemia, hypertriglyceridemia and insulin resistance, resembling human metabolic syndrome. Previously, we found altered vascular prostanoid (PR) production in this model. (2) Sodium molybdate (Mo), as well as sodium tungstate, causes insulin-like effects and normalizes plasma glucose levels in streptozotocin-treated diabetic rats. We studied the effects of Mo on BP, metabolic parameters and release of PR from the mesenteric vascular bed (MVB) in F-overloaded rats. (3) Four groups of male Sprague-Dawley rats were analysed: Control, tap water to drink; F, F solution 10% W/V to drink; CMo, Mo 100 mg kg day(-1) and FMo, both treatments. After 9 weeks, the animals were killed and MVBs removed and the released PRs measured. (4) F increased BP, glycemia, triglyceridemia and insulinemia. Mo treatment prevented the increases in BP and glycemia, but did not modify triglyceridemia or insulinemia. In addition, Mo decreased BP in controls. (5) Prostaglandins (PG) F2 alpha and E2, PG 6-ketoF1 alpha and thromboxane (TX) B2 , as well as inactive metabolites of prostacyclin (PGI2) and TXA2 were detected. F decreased the production of vasodilator PRs PGI2 and PGE2 in MVB. Mo prevented these alterations and increased PGE2 in controls. Vasoconstrict or PRs PGF2 alpha and TXA2 release was not modified. (6) Mo treatment, beyond its known lowering effect on glycemia, prevents the reduction in the vascular release of vasodilator PR observed in this model. This could be one of the mechanisms by which Mo avoids the increase in BP caused by F overload in the rat.

Keywords: fructose; hypertension; metabolic syndrome; molybdate; prostanoids.

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