



# Increased microparticle production and impaired microvascular endothelial function in aldosterone-salt-treated rats: protective effects of polyphenols

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Résumé en  
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We aimed to characterize circulating microparticles in association with arterial stiffness, inflammation and endothelial dysfunction in aldosterone-salt-induced hypertension in rats and to investigate the preventive effects of red wine polyphenols. Uninephrectomized male Sprague-Dawley rats were treated with aldosterone-salt (1 µg.h(-1)), with or without administration of either red wine polyphenols, Provinols™ (20 mg.kg(-1).day(-1)), or spironolactone (30 mg.kg(-1).day(-1)) for 4 weeks. Microparticles, arterial stiffness, nitric oxide (NO) spin trapping, and mesenteric arterial function were measured. Aldosterone-salt rats showed increased microparticle levels, including those originating from platelets, endothelium and erythrocytes. Hypertension resulted in enhanced aortic stiffness accompanied by increased circulating and aortic NO levels and an upregulation of aortic inducible NO-synthase, NFκB, superoxide anions and nitrotyrosine. Flow-induced dilatation was reduced in mesenteric arteries. These effects were prevented by spironolactone. Provinols™ did not reduce arterial stiffness or systolic hypertension but had effects similar to those of spironolactone on endothelial function assessed by flow-mediated vasodilatation, microparticle generation, aortic NO levels and oxidative stress and apoptosis in the vessel wall. Neither the contractile response nor endothelium-dependent relaxation in mesenteric arteries differed between groups. The in vivo effects of Provinols™ were not mediated by mineralocorticoid receptors or changes in shear stress. In conclusion, vascular remodelling and endothelial dysfunction in aldosterone-salt-mediated hypertension are associated with increased circulating microparticles. Polyphenols prevent the enhanced release of microparticles, macrovascular inflammation and oxidative stress, and microvascular endothelial dysfunction independently of blood pressure, shear stress and mineralocorticoid receptor activation in a model of hyperaldosteronism.

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