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Title: Pulmonary vasoreactivity in spontaneously hypertensive rats - Effects of leptin and endothelin-1

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Body: Systemic hypertension may be associated with increased pulmonary vascular resistance in absence of demonstrable increased left ventricular filling pressures (Olivari et al. Circulation. 1978; 57: 1185-90). There has been report of enhanced pulmonary vasoreactivity to hypoxia in patients with systemic hypertension (Guazzi et al. Circulation. 1987; 75: 156-62). Chronic hyperleptinemia has been shown to contribute to systemic hypertension associated to obesity. We investigated pulmonary vascular reactivity to endothelin(ET)-1 and leptin in normotensive Wistar (WKY) and spontaneously hypertensive rats (SHR). Pulmonary (PA) and thoracic artery (TA) rings were sampled in SHR and WKY rats. Vascular reactivity to phenylephrine (PHE), ET-1 and leptin was evaluated in endothelium-intact and -denuded rings mounted in organ baths. In controls, ET-1 induced a higher level of contraction in PA than in TA. After PHE precontraction, leptin relaxed intact PA and TA, while no response was observed in denuded arteries. After ET-1 precontraction, leptin induced a greater time-dependent relaxation in intact TA than PA. In SHR, PHE- and ET-1-induced contraction was enhanced in intact PA. Contraction to PHE and ET-1 was respectively 51% and 32% higher in denuded than intact control PA, while this was respectively reduced to 16% and abolished in SHR. After PHE precontraction, leptin induced a similar endothelium-dependent relaxation in SHR and controls. After ET-1 precontraction, endothelium-dependent relaxation to leptin was abolished in SHR PA. In SHR, pulmonary vascular reactivity is altered with a loss of endothelial blunting effect to vasoconstrictors and of leptin-induced vasodilation.