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Erythropoietin-induced hypertension and vascular injury in mice overexpressing human endothelin-1: exercise attenuated hypertension, oxidative stress, inflammation and immune response

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Résumé en anglais	OBJECTIVE: Erythropoietin used to correct anaemia in chronic kidney disease (CKD) has been shown to increase blood pressure (BP) in CKD patients and experimental animals. Endothelin (ET)-1 expression is increased in CKD animals and patients, and enhanced by erythropoietin. Erythropoietin-induced BP rise was blunted by ETA receptor blockers. This study was designed to determine whether preexisting endothelin (ET)-1 overexpression is required for erythropoietin to cause adverse vascular effects and whether this could be prevented by exercise training. METHODS: Eight to 10-week old male wild-type mice and mice with endothelial-specific ET-1 overexpression (eET-1) were treated or not with EPO (100 IU/kg, SC, 3 times/week). eET-1 was subjected or not to swimming exercise training (1 h/day, 6 days/week) for 8 weeks. SBP, mesenteric artery endothelial function and remodelling, NADPH oxidase activity, reactive oxygen species (ROS) generation, vascular cell adhesion protein (VCAM)-1, monocyte/macrophage infiltration, T regulatory cells (Tregs) and tissue ET-1 and plasma endothelin were determined. RESULTS: Erythropoietin increased SBP by 24 mmHg ($P < 0.05$) and decreased by 25% vasodilatory responses to acetylcholine ($P < 0.01$) in eET-1 mice. Erythropoietin enhanced ET-1 induced increase in resistance artery media/lumen ratio (31%, $P < 0.05$), aortic NADPH oxidase activity (50%, $P < 0.05$), ROS generation (93%, $P < 0.001$), VCAM-1 (80%, $P < 0.01$) and monocyte/macrophage infiltration (159%, $P < 0.001$), and raised plasma and aortic ET-1 levels (>/=130%, $P < 0.05$). EPO had no effect in wild-type mice. Exercise training prevented all of the above ($P < 0.05$). CONCLUSION: Erythropoietin-induced adverse vascular effects are dependent on preexisting elevated ET-1 expression. Exercise training prevented erythropoietin-induced adverse vascular effects in part by inhibiting ET-1 overexpression-induced oxidative stress, inflammation and immune activation.
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