Total sleep deprivation alters endothelial function in rats: a nonsympathetic mechanism

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Résumé en anglais	STUDY OBJECTIVES: Sleep loss is suspected to induce endothelial dysfunction, a key factor in cardiovascular risk. We examined whether sympathetic activity is involved in the endothelial dysfunction caused by total sleep deprivation (TSD). DESIGN: TWO GROUPS: TSD (24-h wakefulness), using slowly rotating wheels, and wheel control (WC). PARTICIPANTS: Seven-month-old male Wistar rats. INTERVENTIONS: Pharmacological sympathectomy (reserpine, 5 mg/kg, intraperitoneal), nitric oxide synthase (NOS) inhibition (N (G)-nitro-L-arginine, 20 mg/kg, intraperitoneally 30 min before experiment) and cyclooxygenase (COX) inhibition (indomethacin, 5 mg/kg, intraperitoneally 30 min before experiment). MEASUREMENTS AND RESULTS: In protocol 1, changes in heart rate (HR) and blood pressure were continuously recorded in the sympathectomized and non-sympathectomized rats. Blood pressure and HR increased during TSD in non-sympathectomized rats. In protocol 2, changes in skin blood flow (vasodilation) were assessed in the sympathectomized and non-sympathectomized rats using laser-Doppler flowmetry coupled with iontophoretic delivery of acetylcholine (ACh), sodium nitroprusside (SNP), and anodal and cathodal currents. ACh- and cathodal current-induced vasodilations were significantly attenuated after TSD in non-sympathectomized and sympathectomized rats (51% and 60%, respectively). In protocol 3, ACh-induced vasodilation was attenuated after NOS and COX inhibition (66% and 49%, respectively). Cathodal current-induced vasodilation decreased by 40% after COX inhibition. In TSD compared to WC a decrease in ACh-induced vasodilation was still observed after COX inhibition. No changes in SNP- and anodal current-induced vasodilation were detected. CONCLUSION: These results demonstrate that total sleep deprivation induces a reduction in endothelial-dependent vasodilation. This endothelial dysfunction is independent of blood pressure and sympathetic activity but associated with nitric oxide synthase and cyclooxygenase pathway alterations.

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