Effect of acute sleep deprivation on vascular function in healthy subjects

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Résumé en anglais	Sleep disorders are associated with inflammation and sympathetic activation, which are suspected to induce endothelial dysfunction, a key factor in the increased risk of cardiovascular disease. Less is known about the early effects of acute sleep deprivation on vascular function. We evaluated microvascular reactivity and biological markers of endothelial activation during continuous 40 h of total sleep deprivation (TSD) in 12 healthy men (29 +/- 3 yr). The days before [day 1 (D1)] and during TSD (D3), at 1200 and 1800, endothelium-dependent and -independent cutaneous vascular conductance was assessed by iontophoresis of acetylcholine and sodium nitroprusside, respectively, coupled to laser-Doppler flowmetry. At 0900, 1200, 1500, and 1800, heart rate (HR) and instantaneous blood pressure (BP) were recorded in the supine position. At D1, D3, and the day after one night of sleep recovery (D4), markers of vascular endothelial cell activation, including soluble intercellular adhesion molecule-1, vascular cell adhesion molecule-1, E-selectin, and interleukin-6 were measured from blood samples at 0800. Compared with D1, plasma levels of E-selectin were raised at D3, whereas intercellular adhesion molecule-1 and interleukin-6 were raised at D4 ($P < 0.05$). The endothelium-dependent and -independent CVC were significantly decreased after 29 h of TSD ($P < 0.05$). By contrast, HR, systolic BP, and the normalized low-frequency component of HR variability (0.04-0.15 Hz), a marker of the sympathetic activity, increased significantly within 32 h of TSD ($P < 0.05$). In conclusion, acute exposure to 40 h of TSD appears to cause vascular dysfunction before the increase in sympathetic activity and systolic BP.
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