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Insufficient sleep is associated with impaired nitric oxide-mediated endothelium-dependent vasodilation

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Background & Aim: Habitual short nightly sleep duration is associated with increased atherosclerotic cardiovascular disease risk and morbidity. Vascular endothelial dysfunction represents an important mechanism that may underlie this heightened cardiovascular risk. Impaired endothelium -dependent vasodilation, particularly NO-mediated vasodilation, contributes to the development and progression of atherosclerotic vascular disease and acute vascular events. We tested the hypothesis that chronic insufficient sleep is associated with impaired NO-mediated endothelium-dependent vasodilation in middle-aged adults.

Methods: Thirty adult men were studied: 15 with normal nightly sleep duration (age: 58±2 y; sleep duration: 7.7±0.2 h/night) and 15 with short nightly sleep duration (55±2 y; 6.1±0.2 h/night). Forearm blood flow (FBF) responses to intra-arterial infusion of acetylcholine, in the absence and presence of the endothelial NO synthase inhibitor NG-monomethyl-L-arginine (L-NMMA), as well as responses to sodium nitroprusside, were determined by strain-gauge venous occlusion plethysmography.

Results: The FBF response to acetylcholine was lower (20%; $p<0.05$) in the short sleep duration group (from 4.6±0.3 to 11.7±1.0 ml/100 ml tissue/min) compared with normal sleep duration group (from 4.4±0.3 to 14.5±0.5 ml/100 ml tissue/min). L-NMMA significantly reduced the FBF response to acetylcholine in the normal sleep duration group (40%), but not the short sleep duration group. There were no group differences in the vasodilator response to sodium nitroprusside.

Conclusions: These data indicate that short nightly sleep duration is associated with endothelial-dependent vasodilator dysfunction due, in part, to diminish NO bioavailability. Impaired NO-mediated endothelium-dependent vasodilation may contribute to the increased cardiovascular risk with insufficient sleep.

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