**HABITUAL SHORT SLEEP DURATION IS ASSOCIATED WITH ENDOTHELIAL FIBRINOLYTIC DYSFUNCTION**

B.R. Weil, **K.J. Diehl**, J.J. Greiner, B.L. Stauffer, C.A. DeSouza

1University of Colorado, Boulder, CO, 2University of Colorado Health Sciences Center, Aurora, CO, USA

Habitual short sleep duration is associated with increased cardiovascular disease morbidity and mortality. The mechanisms responsible for this heightened cardiovascular risk are not fully understood. The capacity of the endothelium to release tissue-type plasminogen activator (t-PA) is a key endogenous defense mechanism against thrombosis. We tested the hypothesis that endothelial t-PA release is impaired in adults who sleep less than 7 hrs/night compared with adults who sleep between 7 and 9 hrs/night. 30 adult men were stratified based on average nightly habitual sleep duration: 15 with normal sleep duration (age: 55±2 yr; 7.6 hrs sleep/night) and 15 with short sleep duration (56±2 yr; 6.1 hrs sleep/night). Net endothelial release of t-PA was determined, in vivo, in response to intrabrachial infusions of bradykinin (BDK) and sodium nitroprusside. Net endothelial t-PA release to BDK was significantly lower (~30%) in the short (from 0.39±0.8 to 41.5±4.3 ng·100 ml tissue–1·min–1) compared with the normal (0.43±0.5 to 64.9±6.7 ng·100 ml tissue–1·min–1) sleep duration group. Furthermore, there was an inverse relation between average nightly sleep duration and peak t-PA release to BDK (r=0.36, P<0.05). In summary endothelial t-PA release is impaired in adults with short habitual sleep duration. This may underlie the increased atherothrombotic risk associated with chronic short sleep.