CARDIOVASCULAR RESPONSES TO THE EXERCISE-SIESTA TRADITION
Zaregarizi MohammadReza1, Edwards Ben2, George Keith2, Harrison Yvonne2, Atkinson Greg2
(Liverpool John Moores University and Ministry of Health and Medical Education of Iran1, Liverpool John Moores University2, United Kingdom)

Many cultures have a tradition of morning activity followed by an afternoon nap or 'siesta'. Recently, coronary mortality has been found to be lower in siesta takers (Naska et al., 2007), but it is unclear how exercise and siesta, in combination, influence cardiovascular variables (Atkinson et al., 2005). Therefore, we examined cardiovascular changes during a post-exercise period which included an afternoon nap.

Nine normotensive, inactive adults, aged 34±6 years (mean±SD) volunteered for this counterbalanced experiment. Following 4 h of nocturnal sleep, subjects either exercised at 70% VO2max or remained sedentary for 30 min starting at 09:30 hours. At 14:00 hours, subjects were allowed to nap for up to 1 h in our sleep laboratory. Electroencephalographic (EEG) data were analysed to describe three phases of the nap-onset period for each participant (i) a 10-min period of relaxed wakefulness before lights-out, (ii) the time between lights-out and onset of stage 1 sleep, and (iii) the time between onsets of stages 1 and 2 sleep (Carrington et al., 2005). Using repeated-measures general linear models, the changes in blood pressure, heart rate and cutaneous vascular conductance (CVC) over these 3 phases were compared between exercise and no-exercise trials. Following the nap, 24-h measurements of ambulatory blood pressure, heart rate and actigraphy were also compared between trials. Data are described as mean±SE.

Mean arterial pressure (MAP) generally declined by 3-8 mmHg over the three phases of the nap-onset period. However, in the exercise trial, MAP was 3±2 and 2±3 mmHg lower during phases (i) and (ii) of the nap onset period, respectively. Conversely, MAP was 3±2 mmHg higher in phase (iii) of the nap-onset period following exercise (trial x phase interaction: P=0.022). This higher MAP was explained by the fact that the time taken to reach phase (iii) of sleep (and therefore time spent lying supine up to this point) was 3.9±3.2 min shorter in the exercise trial. Exercise did not alter the responses of heart rate and CVC during the nap onsets phases (P>0.20). Ambulatory MAP was lower in the exercise trial by 3.0±1.2 mmHg during the evening after the nap, over the nocturnal sleep period and during the subsequent morning (trial main effect: P=0.037), even though 24-hour actigraphy recordings did not differ between trials (P=0.80).

These results suggest that BP of inactive individuals is decreased during afternoon siesta, especially after a bout of vigorous exercise in the morning. This reduction in BP was apparent 24-h after exercise and did not seem to be related to any exercise-mediated changes in sleep architecture during the nap nor any changes in activity during nocturnal sleep. This lowered BP associated with the exercise-siesta tradition could be one factor in explaining the decreased coronary mortality of siesta takers.

Keywords: Blood Flow, Blood Pressure, Hemodynamics