

Social Jetlag Inhibits Exercise-Induced Adaptations in the Heart and Alters Markers of Mitochondrial Dynamics

GRAHAM R. MCGINNIS, MICHAEL B. DIAL, ELIAS M. MALEK, GRECO NEBLINA, AUSTIN COOPER, NIKOLETA VASILEVA, & TANNER ERWIN

McGinnis Lab; Kinesiology and Nutrition Sciences; University of Nevada, Las Vegas; Las Vegas, NV

Category: Professional

Presenter: McGinnis, Graham (graham.mcginnis@unlv.edu)

ABSTRACT

Social jetlag (SJL), or the shifting of behavior and sleep times between weekdays and weekends, is a pervasive form of circadian rhythm disruption that affects nearly 70% of the population to some extent. The magnitude of SJL can be determined by the difference in the mid-sleep phase between weekends and weekdays. Higher levels of SJL have been associated with lower levels of cardiorespiratory fitness, and increased incidence of cardiometabolic disease, which may be due, in part, to mitochondrial dysfunction. However, no studies to date have evaluated the effects of long term SJL on cardiac mitochondrial dynamics. **PURPOSE:** To determine the effect of SJL on mitochondrial fission and fusion signaling in the heart, and if exercise protects the heart against SJL. **METHODS:** Male C57BL/6 mice (n = 40) were allocated to four groups (n = 10/group): 1) Control Light:Dark cycle, Sedentary (CON-SED), 2) Control Light:Dark cycle, Exercise (CON-EX), 3) SJL, sedentary (SJL-SED), or SJL, exercise (SJL-EX). SJL was implemented by delaying the LD cycle 4 hours on 'Fridays,' and advancing the LD cycle on Mondays. Exercise was provided ad libitum with a disc. Conditions persisted for 6 weeks at which point hearts were harvested for gravimetric analysis and western blotting of markers of mitochondrial dynamics. **RESULTS:** Exercise caused myocardial hypertrophy in both control and SJL LD conditions (Main Effect – EX, $p < 0.05$), with no difference between CON and SJL conditions. We did not observe any significant differences in mitochondrial content (OXPHOS antibody cocktail, $p > 0.05$), SJL decreased expression of mitochondrial fusion proteins MFN1 and OPA1 (Main Effect – SJL, $p < 0.05$). Importantly, SJL inhibited exercise-induced increases in MFN2 ($p < 0.05$), suggesting that SJL specifically ameliorates some exercise-induced adaptations in mitochondrial dynamics in the heart. **CONCLUSION:** These findings suggest that exercise induces adaptations in mitochondrial dynamics, potentially increasing mitochondrial function, and SJL may disrupt mitochondrial dynamics both in the sedentary and exercise trained states.