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Effects of Acute Sleep Deprivation on the Physiological Response to Woodsmoke and Exercise

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ABSTRACT

Chronic exposure to woodsmoke presents serious health detriments, yet the acute response to woodsmoke exposure is not well defined. While current literature reports on woodsmoke exposure and exercise, little work has been performed to understand the potential compounding effects of sleep deprivation during exposure. Sleep deprivation could modify physiologic responses to stressors such as exercise and smoke inhalation. Acute sleep deprivation has been shown to depress vagal tone, specifically depressed heart rate variability and stress recovery. The further identification of acute physiologic responses to woodsmoke would likely provide insight into the mechanism behind increased cancer and cardiovascular disease rates. Purpose: To evaluate the effects of sleep deprivation on the acute physiological response to woodsmoke and exercise. Methods: Ten recreationally active male participants (age = 24±4 yrs.; height = 185±4 cm; weight = 85.7±9.4 kg ;VO2max = 46.8±5.7 ml·kg^{-1}·min^{-1}; body fat = 12.6±6.7 %) performed two separate 45-minute stationary bicycle workouts at resistances equivalent to 70% of their VO2 max while inhaling woodsmoke through a facemask in a crossover design. In random order, one trial was performed on 8 hours of sleep (control, slept 22:00-06:00) and the other on 4 hours of sleep (sleep-deprived; slept 00:00-04:00) with heart rate variability (HRV), exhaled breath condensate (EBC), pulse wave velocity (PWV), blood oxidative stress markers, and pulmonary function tests (PFT), analyzed before and after each trial. Trials were separated by one week. Results: EBC volume pre- and post-exercise was 2.4±1.0 and 2.6±1.3 mL, respectively. No significant difference in HRV, PWV, or BP was observed between the control and sleep-deprived groups. However, PWV was significantly different across trials in both groups ($p < 0.01$). Pulmonary data for key dependent measures of PFT (FVC, FEV1%, MVV) displayed no statistically significant differences between trials or across trials. Conclusion: Despite the known harmful effects of smoke inhalation, sleep deprivation did not magnify the physiological response following moderate-intensity aerobic exercise while exposed to woodsmoke particulate matter. Although these findings do not negate the negative impact of wood smoke inhalation, other research approaches are needed to understand better the acute effects of smoke exposure on the cardiovascular system.

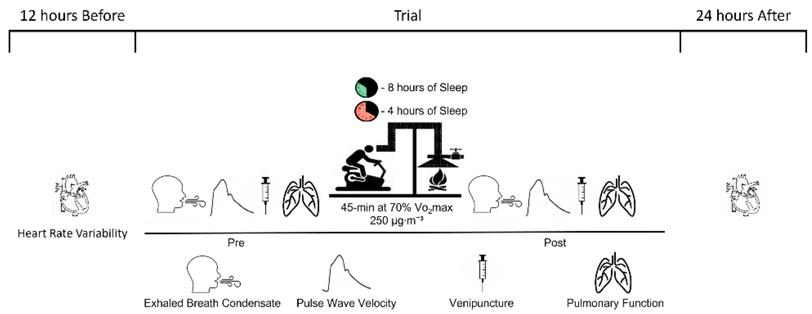
INTRODUCTION

- Acute physiological responses to woodsmoke are not well defined.
- Sleep deprivation is an emerging stressor that may magnify the physiological response to stressors.
- Collectively, acute sleep deprivation, woodsmoke exposure, and exercise may identify a threshold of physiological response to stress in young healthy adults.
- **Purpose:** To evaluate the effects of sleep deprivation on the acute physiological response to woodsmoke and exercise.

METHODS

- 10 recreationally active male subjects (24±4yrs, 85.7±9.4 kg, 46.8±5.7 ml·kg^{-1}·min^{-1}) completed two trials
- Subjects were equipped with a modified face mask to inhale 250 µg·m^{-3} while exercising at 70% of VO_2max for 45 minutes.
- Trials differed by amount of sleep the night before the 8:00am trial – 8 hours of sleep (10:00pm to 6:00am) and 4 hours of sleep (12:00am to 4:00am)
- Data were analyzed using 2x2 repeated measures ANOVA and paired sample t-tests using SPSS 26.0

Study Design

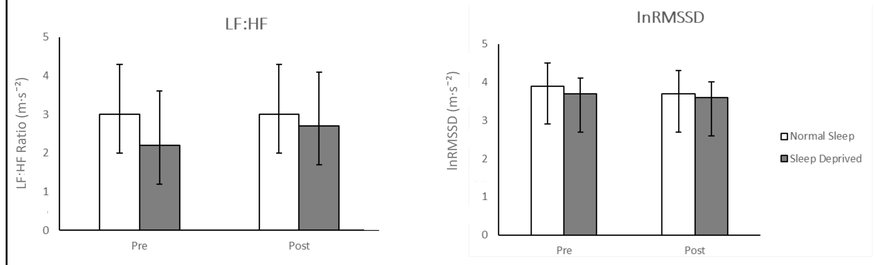


CONCLUSION

- Data indicates significant physiological stress following woodsmoke exposure with exercise.
- Acute sleep deprivation prior to woodsmoke exposure while exercising did not magnify pre/post trial differences in the variables examined.

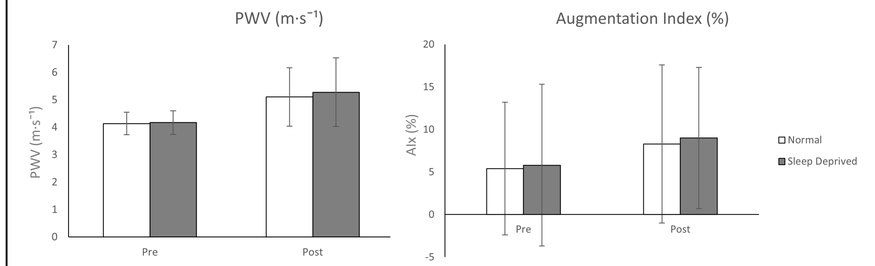
RESULTS

Heart Rate Variability



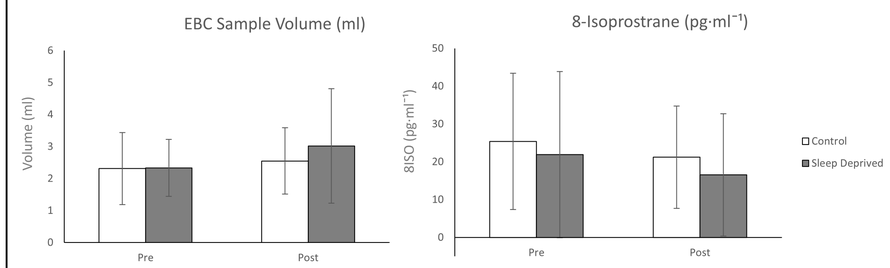
Analysis of lnRMSSD and the LF: HF ratio indicated no time effects (lnRMSSD: *P*=0.442; LF:HF: *P*=0.548) and no between-trial differences (lnRMSSD: *P*=0.412; LF:HF: *P*=0.313).

Arterial Stiffness



Analysis of PWV and AIx indicated no interaction effect (PWV: *P*=0.809; AIx: *P*=0.751), trial effect (PWV: *P*=0.700; AIx: *P*=0.631), indicating that these markers were not significantly different. However, a time effect was noted in PWV (PWV: *P*=0.001; AIx: *P*=0.129).

Exhaled Breath Condensate



No significant differences were observed in EBC pre-trial volumes (Normal sleep = 2.3±1.1 ml; Sleep-deprived = 2.3±0.9 ml, *P*=0.999) or post-trial volumes (Normal sleep = 2.6±1.0 ml; Sleep-deprived = 3.0±1.8 ml; *P*=0.215). For EBC 8-isoprostane, there was no interaction *(P*=0.591), trial *(P*=0.762), or time effect *(P*=0.218).