

Influence of Evening Binge Alcohol Consumption on Sympathetic Transduction

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ABSTRACT

Evening alcohol consumption elicits an exaggerated increase in morning-after sympathoexcitation and heart rate, yet the effects of evening binge drinking on neural-cardiovascular coupling the morning after alcohol remain unknown. **PURPOSE:** The present study aimed to investigate the effects of evening binge alcohol consumption on sympathetic vascular transduction by examining blood pressure following bursts of muscle sympathetic nerve activity (MSNA). We hypothesized that evening alcohol would exaggerate morning-after blood pressure response to MSNA. **METHODS:** Twenty-nine healthy participants (14 men, 15 women; 28 ± 2 yrs; 27 ± 1 kg/m²) underwent a two-condition, randomized crossover design (i.e. alcohol drinking vs. fluid control [FC]). Based on the NIAAA definition of binge drinking (i.e., 1g/kg men, 0.85 g/kg women), the dose of alcohol administered to the treatment condition was ~4-5 drink equivalency over a two-hour span prior to sleep. An equivalent volume of same fluid (minus alcohol) was provided during the FC. Beat-to-beat blood pressure (BP, finger plethysmography), heart rate (electrocardiogram), venous occlusion plethysmography (VOP), and MSNA were successfully recorded across both conditions during a 10-min rest period the morning after evening alcohol or FC. Mean arterial pressure (MAP) and diastolic blood pressure (DBP) were calculated across 10 cardiac cycles after each MSNA burst. These values were signal averaged and compared between conditions. Forearm blood flow (FBF) and vascular conductance (FVC) were assessed using VOP. **RESULTS:** Resting MAP (FC: 82 ± 1 vs. Alcohol: 82 ± 2 mmHg) and MSNA (FC: 20 ± 2 vs. Alcohol: 21 ± 2 burst/min) were not different between conditions (both $p > 0.05$), while HR was elevated after alcohol consumption (FC: 60 ± 2 vs. Alcohol: 64 ± 2 beats/min, $p = 0.003$). Alcohol consumption elicited a greater change in MAP (FC: $\Delta 1.1 \pm 0.1$ vs. Alcohol: $\Delta 1.4 \pm 0.1$ mmHg, $p = 0.004$) and DBP (FC: $\Delta 0.9 \pm 0.1$ vs. Alcohol: $\Delta 1.1 \pm 0.1$ mmHg, $p = 0.017$) following MSNA bursts. In contrast, FBF (FC: 1.9 ± 0.6 vs. Alcohol: 2.3 ± 0.9 mL/100mL/min, $p = 0.019$) and FVC (FC: 2.2 ± 0.7 vs. Alcohol: 2.7 ± 1.0 mL/100mL/min/mmHg, $p = 0.017$) were elevated following evening binge alcohol consumption. **CONCLUSION:** Our findings demonstrate that evening binge drinking elicits a greater systemic change in blood pressure reactivity after a MSNA burst the morning after alcohol consumption. In contrast, the elevated FBF and FVC suggests a regionalized influence of alcohol on sympathetic regulation of blood pressure. These findings provide new insights regarding potential mechanisms contributing to the known associations between repeated alcohol consumption and cardiovascular risks.