

rate (HR) and lowered heart rate variability (HRV). This study aimed to examine the relationship between cardiac reactivity and subjective response following intravenous (IV) alcohol in non-dependent drinkers. **METHODS/STUDY POPULATION:** Non-dependent drinkers (N = 46, average age = 25.2) completed a human laboratory IV alcohol self-administration (IV-ASA) session. Subjective response to alcohol was assessed using the Drug Effects Questionnaire (DEQ) and Alcohol Urge Questionnaire (AUQ). Drinking behavior was assessed using the Alcohol Timeline Followback (TLFB) and Alcohol Use Disorders Identification Test (AUDIT). HR was recorded using the Polar Pro Heart Rate monitor throughout the session. HRV measures were calculated using guidelines determined by the Task Force of the European Society of Cardiology and The North American Society of Pacing and Electrophysiology. **RESULTS/ANTICIPATED RESULTS:** Recent drinking history as measured by the AUDIT and TLFB was not significantly different by sex. Results showed heavier drinking measures (AUDIT and TLFB) were positively associated with HRV measures (all p-values < 0.02). Those who reported a greater increase in alcohol craving (AUQ score) and wanted more alcohol (DEQ) following an alcohol prime, showed a greater change in HRV (p < 0.005). When examining HRV change from baseline throughout the priming session, there was a significant sex interaction for NN50 (p < 0.03) and a trend for PNN50 (p-value < 0.07). **DISCUSSION/SIGNIFICANCE OF IMPACT:** Acute IV alcohol alters cardiac reactivity measures in non-dependent drinkers. Future directions include examining the role of sex in HRV changes during alcohol consumption during IV-ASA. Understanding the effect of alcohol on cardiac reactivity and physiology may help characterize those at risk for alcohol use disorders.

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### Resistant hypertension potentiates the risk of End-Stage Kidney Disease among African-Americans independent of APOL1 genotype in the Million Veteran Program

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**OBJECTIVES/GOALS:** African-Americans have a 3-fold higher risk of end-stage kidney disease (ESKD) compared to Whites due in part to APOL1 risk alleles. Whether resistant hypertension (RH) magnifies the risk of ESKD among African Americans beyond APOL1 is not known. We examined the interaction between RH and race on ESKD risk and the independent effect of RH beyond APOL1. **METHODS/STUDY POPULATION:** We designed a retrospective cohort of 240,038 veterans with HTN, enrolled in the Million Veteran Program with an estimated glomerular filtration rate (eGFR) >30 ml/min/1.73m<sup>2</sup>. The primary exposure was incident RH (time-varying). The primary outcome was incident ESKD during a 13.5 year follow up: 2004-2017. Secondary outcomes were myocardial infarction (MI), stroke, and death. Incident RH was defined as failure to achieve outpatient blood pressure (BP) <140/90 mmHg with 3 antihypertensive drugs, including a thiazide, or use of 4 or more drugs. Poisson models were used to estimate incidence rates

and test additive interaction with race and APOL1 genotype. Multivariable Cox models (with Fine-Gray competing-risks models as sensitivity analyses) were used to examine independent effects. **RESULTS/ANTICIPATED RESULTS:** The cohort comprised 235,046 veterans; median age was 60 years; 21% were African-American and 6% were women, with 23,010 incident RH cases observed over a median follow-up time of 10.2 years [interquartile range, 5.6-12.6]. Patients with RH had higher incidence rates [per 1000 person-years] of ESKD (4.5 vs. 1.3), myocardial infarction (6.5 vs. 3.0), stroke (16.4 vs. 7.6) and death (12.0 vs. 6.9) than non-resistant hypertension (NRH). African-Americans with RH had a 2.6-fold higher risk of ESKD compared to African-Americans with NRH; 3-fold the risk of Whites with RH, and 9.6-fold the risk of Whites with NRH [p-interaction < .001]. Among African-Americans, RH was associated with a 2.2-fold (95%CI, 1.86-2.58) higher risk of incident ESKD in models adjusted for APOL1 genotype and in the subset of African-Americans with no APOL1 risk alleles, RH was associated with an adjusted 2.75-fold (95% CI: 2.00-3.50) higher risk of incident ESKD. **DISCUSSION/SIGNIFICANCE OF IMPACT:** RH was independently associated with a higher risk of ESKD and cardiovascular outcomes, especially among African-Americans. This elevated risk is independent of APOL1 genotype. Interventions that achieve BP targets among patients with RH could curtail the incidence of ESKD and cardiovascular outcomes in this high-risk population. **CONFLICT OF INTEREST DESCRIPTION:** None.

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### Septic Shock Epidemiology and Sociodemographic Predictors of Mortality: Results from One Florida Data Trust Cohort

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**OBJECTIVES/GOALS:** Septic shock is a lethal condition. Research suggests that overall sepsis mortality varies by race, but less is known about demographic differences in septic shock mortality. Our objectives were to describe the septic shock population using a large, statewide data repository and identify demographic predictors of septic shock mortality. **METHODS/STUDY POPULATION:** This was a retrospective review of patients with septic shock in the One Florida Data Trust from 2012-2018. Patients were classified as having septic shock if they received vasopressors and had either 1) an ICD-9 or 10 code for septic shock or 2) an ICD-9 or 10 code for infection and an ICD-9 or 10 code for organ dysfunction. Demographic data and place of residence prior to admission was collected. The primary outcome was 90 day mortality. T-test and chi-square tests were used to test association of individual predictors and mortality. Multiple logistic regression was used to identify predictors of mortality after adjustment for other variables. Level of significance was set at 0.05. SAS v9.4 (Cary, NC) was used for analyses. **RESULTS/ANTICIPATED RESULTS:** There were 11,790 patients with septic shock. The mean(SD) age was 61(16) years. With regard to race/ethnicity 66% identified as white, 27% as black, 3.7% as Hispanic, and 3.5% as other races (non-white, non-black, non-Hispanic). Most