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# Longitudinal associations between dietary fructose and sodium and systolic and diastolic blood pressure among US Black and White adults in the CARDIA study

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Hypertension is one of the leading risk factors for major adverse cardiovascular events (MACE). Likewise, suboptimal dietary habits, including high fructose and sodium intake, are major contributors to mortality. High dietary fructose alone predisposes to salt-sensitivity of blood pressure, which is an independent, significant cardiovascular risk factor. We previously showed in our rat model of fructose-salt-sensitive hypertension that even a short term (4 weeks) feeding with 20% fructose and 4% salt leads to salt sensitivity of blood pressure. Furthermore, male rats fed fructose and salt had increased aortic stiffness, impaired diastolic function and longitudinal left ventricular strain. Most recently, we showed that when young rats are fed fructose and salt and then switched to a normal diet, subsequent fructose feeding contributes to the salt sensitivity of blood pressure later in life. The present investigation was designed to test the hypothesis that increased fructose and sodium intake in humans leads to increases in systolic and diastolic blood pressure. The study subjects were derived from the CARDIA study, which is a prospective follow-up study aimed to investigate the evolution of cardiovascular risk factors in a biracial cohort of young adults aged 17-35 years at enrollment. Dietary data was obtained from the diet questionnaires which quantified fructose and sodium. Blood pressures obtained at enrollment, years 7, 15 and 30 of follow-up were used in the present study. Multivariate analysis was completed for the factors contributing to the development of hypertension. Moreover, serum uric acid and C-reactive protein were regressed upon high blood pressure diagnosis at year 15 of follow up. Additionally, echocardiography data were regressed upon high blood pressure diagnosis at year 30 of follow up. Dietary fructose and sodium at year 7 of follow up significantly predicted systolic (SBP)($F(2,2922) = 11.760$, $p < 0.001$) and diastolic (DBP) ($F(2,2922) = 12.425$, $p < 0.001$) two decades later. C reactive protein was significantly associated with being diagnosed with HTN at year 15 of follow up (OR=1.049, 95% CI [1.020-1.079], $P < 0.001$), as was serum uric acid (OR=1.510, 95% CI [1.407-1.621], $P < 0.001$), female sex (OR=2.028, 95% CI [1.610-2.554], $P < 0.001$), and race (favoring non-African American race, OR = 0.475, 95% CI [0.393-0.574], $P < 0.001$). Uric acid measured at year 15 was independently associated with fructose intake at year 7 of follow up ($F(1,3234) = 24.765$, $p < 0.001$). The variables that were significantly associated with HBP diagnosis at year 30 of follow up were longitudinal peak strain (OR = 1.098, 95% CI [1.057-1.141], $P < 0.001$), left ventricular (LV) end-diastolic volume (OR 1.030, 95% CI [1.003-1.059], $P = 0.032$), and LV end-systolic volume (OR=0.939, 95% CI [0.882-0.999], $P = 0.045$). Our data indicate that higher fructose and sodium intake during adolescence may significantly impact increases in both SBP and DBP later in life.