Associations between Types of Dietary Sugar and Risk of Coronary Heart Disease in US Men and Women

Kristine Dennis,[1] Fenglei Wang,[2] Dong Wang,[1] Meir Stampfer,[2] Frank Hu,[2] Yanping Li,[3] Eric Rimm,[2] Walter Willett,[2] and JoAnn Manson[4]

[1]Brigham and Women's Hospital; Harvard Medical School; [2]Harvard T.H. Chan School of Public Health; [4]Brigham and Women's Hospital; and [4]Brigham and Women's Hospital

Objectives: To investigate the associations of dietary sugars, including total fructose equivalents (TFE) and total glucose equivalents (TGE), with coronary heart disease (CHD) risk in two prospective cohort studies, the Nurses' Health Study (NHS) and the Health Professionals Follow-up Study (HPFS). We hypothesized that added sugar, TGE, and TFE intakes were positively associated with CHD risk, but that natural sugar was not associated with higher risk.

Methods: We followed 76,815 women (NHS, 1980–2020) and 39,215 men (HPFS, 1986–2016) who were free of diabetes, cardiovascular disease, and cancer and had completed a semiquantitative food frequency questionnaire (SFFQ) at baseline. Dietary sugar intake was updated every 2–4 years by SFFQs. We defined TFE as fructose monosaccharides and fructose from sucrose, and TGE as glucose monosaccharides and glucose from sucrose, maltose, lactose, and starch. CHD outcomes were ascertained through medical record review and death certificates. Cox proportional hazards models were used to quantify associations between sugar intake and CHD risk with adjustment for potential confounding factors in NHS and HPFS separately. We then pooled risk estimates from the two cohorts using fixed-effects meta-analysis. Secondarily, we examined the associations of sucrose, lactose, starch, and total, added and natural sugars with CHD risk.

Results: We documented 9,683 incident CHD cases during up to 40 years of follow-up. Higher intake of TGE, compared with total fat, was significantly associated with higher CHD risk, whereas TFE intake was not associated with CHD risk. Comparing extreme quintiles of intake, the hazard ratios (HRs, 95% confidence interval [CI]) of CHD were 1.16 (1.06–1.25; $p_{\text{trend}} < .001$) for TGE and 1.06 (0.97–1.16; $p_{\text{trend}} = 0.09$) for TFE. Replacing 5% of energy from total fat with equivalent energy from total and added sugars were associated with estimated increases in CHD risk of 4% (HR: 1.04, 95% CI: 1.02–1.07) and 5% (HR 1.05, 95% CI: 1.01–1.09). Intakes of natural sugar, sucrose, or lactose were not significantly associated with CHD risk. Starch intake was positively associated with CHD risk (HR comparing extreme quintiles: 1.21, 95% CI: 1.11–1.31; $p_{\text{trend}} < 0.001$).

Conclusions: Our findings support limiting dietary intake of starch and suggest that intake of TGE but not TFE is associated with a higher risk of CHD.

Funding Sources: NIH