

Skim or whole milk protects against postprandial hyperglycemia-induced impairments in vascular function in adults with prediabetes by limiting increases in oxidative stress that lower nitric oxide bioavailability

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Increases in blood sugar following a meal (i.e. postprandial hyperglycemia; PPH) impair vascular function by inducing oxidative stress to reduce nitric oxide (NO[•]) bioavailability. Co-ingesting dairy milk, independent of fat, protects against PPH, but whether this improves vascular function by reducing oxidative stress to improve NO[•] bioavailability is not fully understood. We hypothesized that skim and whole milk would similarly improve vascular function by protecting against PPH-mediated increases in oxidative stress that otherwise lowers NO[•] bioavailability. A randomized, crossover trial was conducted in adults with prediabetes ($n = 22$) who ingested 75 g glucose dissolved in 473 mL of water (GLU) or 473 mL of skim (SKIM) or whole milk (WHOLE). Before beverage ingestion and at 30 min intervals for 180 min post-beverage ingestion, we assessed vascular function using brachial artery flow-mediated dilation (FMD) and measured circulating biomarkers of glycemic control, oxidative stress, and NO[•] homeostasis. Area under the postprandial curve (AUC_{0-180 min}) for FMD and NO[•] metabolites were lowest in GLU but similarly greater in SKIM and WHOLE. Compared with GLU, AUCs for glucose and oxidative stress responses were similarly lower in milk-based trials. Milk-mediated improvements in vascular function was accompanied by greater levels of plasma arginine and lower levels of asymmetric dimethylarginine and symmetric dimethylarginine. Postprandial insulin, lipids, and tetrahydrobiopterin redox status did not differ among trials. Thus, dairy milk, regardless of fat content, attenuates PPH-induced impairments in vascular function by limiting oxidative stress. This improves arginine metabolism to increase NO[•] bioavailability to the vascular endothelium.