

Failure to suppress postprandial non-esterified fatty acids following high fructose feeding in men of Black African origin but not in men of white European origin

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Populations of Black African (BA) ancestry have historically had cardioprotective lipid profiles⁽¹⁾ but more recent North American data indicates this protection has been for the most part lost⁽²⁾. Epidemiological studies have demonstrated concurrent increases in fructose consumption and cardiovascular risk in the United States⁽³⁾. Fructose tends to be lipogenic because of its insulin-independent metabolism in the liver however there have been no studies of its metabolic effect in people of BA ancestry. The present study investigated the hypothesis that high fructose feeding would potentiate greater post-prandial hypertriglyceridemia in men of BA compared to White European (WE) ancestry.

We conducted a double-blinded pilot study in healthy men of BA ($n = 7$) and WE origin ($n = 8$) in which 25% of total 24hr energy intake was provided as fructose. Regular blood sampling was performed during the postprandial period to determine serum triglyceride (TG), glucose, non-esterified fatty acid (NEFA) and insulin concentrations. The iAUC for each outcome was calculated and compared between ethnic groups by t-test and multivariate ANOVA.

The serum glucose, insulin and NEFA iAUC did not differ between ethnic groups, but a trend towards significance was observed for serum TG iAUC ($p = 0.07$) (Figure 1A–1D). Multivariate ANOVA did demonstrate multiple significant time-point differences showing a lack of suppression of NEFA in BA men versus WE ($p < 0.05$) (Figure 1C).

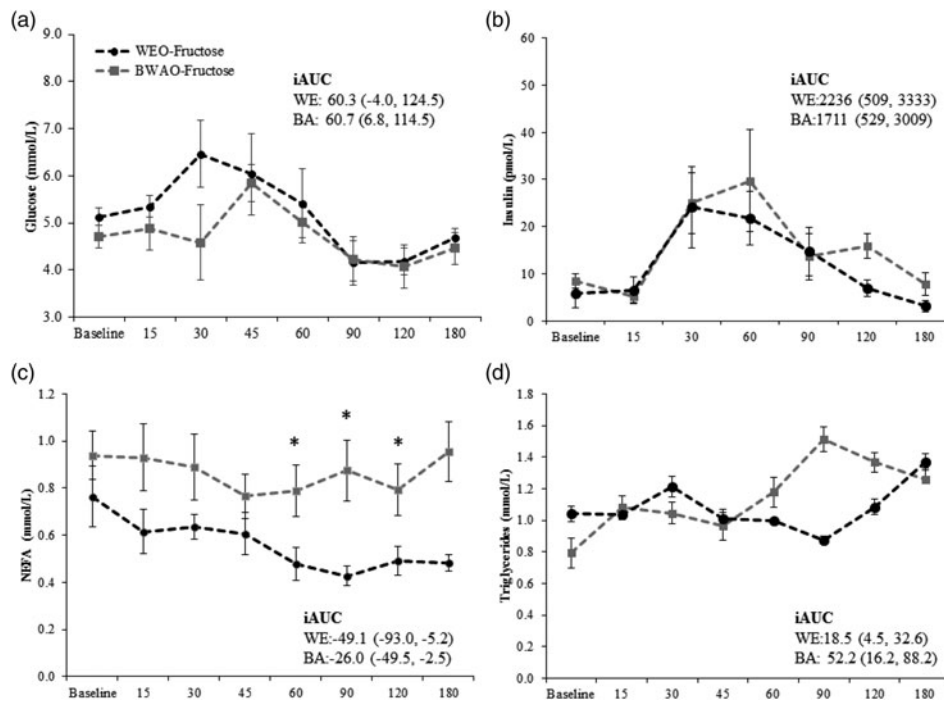


Fig. 1. Postprandial glucose (panel A), insulin (panel B), NEFA (panel C) and TG (panel D) in men of BA and WE origin following a mixed-meal feeding study with fructose intakes.

These data show a trend towards raised postprandial TG and a failure to suppress postprandial NEFA production in BW compared to WE men following acute high fructose feeding. Excessive fructose consumption could drive metabolic changes, similar to those reported here, and may be particularly relevant to cardiometabolic risk factor development in BA populations.

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