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Consumption of Fructose Build High Cholesterol in Young Men and Women

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Introduction

Over the past few decades, the MENA has been witnessing significant changes in food habits paralleled by an important preponderance of metabolite-related diseases. In a region whose traditional diet is known to be healthy due to high vegetable proteins, fibers, minerals, and vitamins with low content of unfavorable food products, the "industrialization/westernization of the diet" is a well-studied and documented phenomenon [1]. The MENA has been losing its traditional diet which was distinguished by its diversity and richness in raw foods, proteins, and multivitamins, in the favor of a more industrial diet which consists of increased preprocessed foods, sugars, fats, alcohol, animal products, saturated- and trans-fatty acids, and relatively less vitamins and minerals with decreased consumption. These changes in dietary and lifestyle patterns contribute to an increase in the rates of micronutrients deficiencies, diet-related chronic diseases, and obesity in all groups of the population in the region.

Epidemiology of Diet-Related Diseases in the Mena

We review the numbers and trends for selected chronic metabolic diseases and micronutrient deficiencies in the different countries of the MENA where data are available. In epidemiological studies, consumption of sugar and/or sugar-sweetened beverages has been linked to the presence of unfavorable lipid levels , insulin resistance, fatty liver , type 2 diabetes, cardiovascular disease , and metabolic syndrome [2] . We have recently reported that consumption of fructose-sweetened beverages at 25% of energy requirements (E) increased visceral adipose deposition and de novo lipogenesis, produced dyslipidemia, and decreased glucose tolerance/insulin sensitivity in older, overweight/obese men and women [3].

Materials and Methods

The subjects who participated in this study are a subgroup of participants from an ongoing 5-yr National Institutes of Health-funded investigation in which a total of eight experimental groups (n = 25/group) will be studied. The objectives include comparing the metabolic effects of fructose, glucose, and HFCS consumption at 25% E and to compare the metabolic effects of fructose and HFCS consumption at 0, 10, 17.5, and 25% E. The results reported in this paper are from the first 48 subjects to complete the study protocol in the experimental groups consuming 25% E as glucose, fructose, or HFCS (n = 16/group) [4]. Participants were recruited through anInclusion criteria included age 18–40 yr and body mass index (BMI) 18–35 kg/m2 with a self-report of stable body weight during the prior 6 months. Exclusion criteria included diabetes (fasting glucose >125 mg/dl), evidence of renal or hepatic disease, fasting plasma TG greater than 400 mg/dl, hypertension (>140/90 mm Hg), or surgery for weight loss. Individuals who smoked, habitually ingested more than two alcoholic beverages per day, exercised more than 3.5 h/ wk at a level more vigorous than walking, or used thyroid, lipid-lowering, glucose-lowering, antihypertensive, antidepressant, or weight loss medications were also excluded [5]. The University of California, Davis, Institutional Review Board approved the experimental protocol for this study, and subjects provided written informed consent to participate [6]. For the 5 wk before study, subjects were asked to limit daily consumption of sugar-containing beverages to one 8-oz serving of fruit juice. Fiftyfi ve subjects were enrolled in the experimental groups consuming 25% E as glucose, fructose, or HFCS. Four subjects withdrew due to unwillingness to comply with the study protocol (two in the HFCS group, two before group assignment), and two were withdrawn due to medical conditions not apparent during screening (HFCS and glucose group) [7]. During the 12-d outpatient phase of the study, the subjects were provided with and instructed to drink three servings of sugar-sweetened beverage per day (one per meal), to consume their usual diet, and to not consume other sugar-containing beverages, including fruit juice [8]. To monitor compliance, the sugar-sweetened beverages contained a biomarker (ribofl avin), which was measured fl uorometrically in urine samples collected at the time of beverage pickup. These measurements indicated that the three groups of subjects were comparably compliant [9].

Primary Outcomes: Comparing Glucose, Fructose Consumption

The effects of the three sugars were significantly different (PROC MIXED two factor analysis with adjustment for BMI, ΔBW and outcomeB) for all primary outcomes except fasting TG (see effects of sugar P values in Table 2). The effects of HFCS compared with fructose consumption on all primary outcomes were not significantly different (P > 0.05, Tukey's) . The increases in 24-h TG AUC (P = 0.0068), late evening TG peaks (P = 0.015), fasting apoB (P = 0.037), and the apoB to apoA1 ratio (P = 0.028) were larger after fructose consumption compared with glucose consumption. The increases in 24-h TG AUC (P = 0.034), fasting LDL (P = 0.0083), non-HDL-C (P = 0.0055), apoB (P = 0.0056), and apoB to apoAI ratio (P = 0.0034) were larger after HFCS consumption than glucose consumption.

Secondary Outcomes: Comparing Glucose, Fructose Consumption

The effects of the three sugars were significantly different (PROC MIXED two factor analysis with adjustment for BMI, ΔBW, and outcomeB) for postprandial LDL, non-HDL-C, apoB, RLP-C, and sdLDL-C. The effects of HFCS compared with fructose consumption on all secondary outcomes were not significantly different (P > 0.05,

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Tukey's). The increases in postprandial RLP-C were larger during consumption of fructose compared with glucose (P = 0.044), and HFCS consumption caused larger increases in postprandial LDL (P = 0.0024), non-HDL-C (P = 0.0007), apoB (P = 0.025), and sdLDL-C (P = 0.014) (Tukey's) than glucose consumption.

Gender

Although there were no significant sugar-gender interactions for any of the primary or secondary outcomes, men exhibited larger increases of fasting TG, non-HDL-C, apoB, and sdLDL-C concentrations and postprandial LDL, non-HDL-C, and sdLDL-C concentrations in response to sugar consumption than women .

Discussion

The current study provides evidence that postprandial TG and fasting and postprandial concentrations of LDL, non-HDL-C, apoB, and the apoB to apoAI ratio, established risk factors for coronary heart disease , are significantly increased in response to 2 wk consumption of 25% of E as fructose and HFCS, but not glucose, in younger, normal-weight, and overweight subjects. In contrast and as was observed in older subjects , fasting TG concentrations were increased in subjects consuming glucose but not in those consuming fructose-containing sugars. The differential effects of fructose and glucose consumption on fasting and postprandial TG responses in subjects from both studies suggest that fasting TG concentrations are not a reliable indicator of the adverse changes in postprandial TG and other lipid/lipoprotein risk factors induced by fructose consumption. There is growing evidence linking increases of postprandial TG concentrations with proatherogenic conditions. It is important to note that for both the current and previous study, the differential effects of fructose and HFCS compared with complex carbohydrate on the 24-h TG profile were most marked in the late evening, approximately 4 and 6 h after dinner. Studies investigating the relationship between this late-evening peak and proatherogenic changes would be of interest, as would investigations into the sources of the TG that contributes to these peaks (de novo lipogenesis, diet, or fatty acids derived from adipose lipolysis).

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None

Conflict of Interest

None

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