

Calorie Control Council Response to Miller & Adeli

“Dietary fructose and the metabolic syndrome”

Miller A, Adeli K. *Curr Opin Gastroenterol.* 2008 Mar;24:204-9.

Background

This review article originates from the Molecular Structure & Function Research Institute, Department of Biochemistry and Division of Clinical Biochemistry, The Hospital for Sick Children, University of Toronto.

Hypothesis

This review focuses on the mechanisms whereby fructose advances the components of the metabolic syndrome, including the effect of dietary fructose on plasma uric acid, advanced glycation end products (AGEs), inflammation and alterations in metabolic hormones.

Justifications

- Fructose, a naturally found sugar in many fruits, is now commonly used as an industrial sweetener and is excessively consumed in Western diets.
- Fructose consumption has increased dramatically over the past several decades, and with it the incidence of the metabolic syndrome. The metabolic syndrome is a constellation of pathologies including obesity, insulin resistance, dyslipidemia and hypertension.
- Fructose has been shown to be involved in the progression to metabolic syndrome, through dysregulation of many molecular signaling factors.
- Recent animal studies have confirmed the link between fructose feeding and increased plasma uric acid, a potentially causative factor in metabolic syndrome.
- Advanced glycation end products are also implicated because of their direct protein modifications and indirect effects on inflammation and oxidative stress.
- Human studies have demonstrated fructose's ability to change metabolic hormonal response, possibly contributing to decreased satiety.

Subjects Covered

- Evidence for fructose contribution to metabolic syndrome
- Inflammation
- Hyperuricemia
- Advanced glycation end products
- Hormonal response

Author Conclusions

- High dietary fructose consumption is increasingly being recognized as a causative vector in the development of components of the metabolic syndrome, including dyslipidemia, insulin resistance and hypertension.
- Recent advances have uncovered some of the physiologic and molecular mechanisms and have implicated hyperuricemia, inflammation, AGEs and oxidative stress in these fructose-induced metabolic defects.
- Recent studies continue to highlight the key role played by fructose as an important lipogenic and diabetogenic nutrient, contributing to this growing epidemic.

Critique

- The authors have confused fructose and high fructose corn syrup – a fatal flaw in a review article.
- The authors of this review completely ignore the composition of commercial sweeteners in their review of the literature. There is a tacit assumption that the diet is very rich in fructose — based on what others have reported — but no testing of that assumption or presentation of data to support that position. And there certainly is no discussion of what consumers are actually eating in the way of fructose and glucose, or the ratios of one to the other in the diet.
- There is also no critique of amounts of sweeteners in the diet vs. amounts of specific sugars — fructose and glucose — used in experiments.
- What is most disappointing about this review is that no attempt has been made to apply this very academic research, conducted with pure sugars at very high concentrations, to the common diet. So someone reports that pure fructose at high levels elevates triglycerides: what does this mean for the typical human diet? An academic review of the literature is certainly needed, but with no attempt to put the literature into perspective the review falls far short of its primary objective.