

Postulated Fructose Influence on Myocardial Infarction is Unconvincing

Author J. S. White
Affiliation White Technical Research

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Correspondence

J. S. White, PhD
 White Technical Research
 8895 Hickory Hills Drive
 Argenta IL 62501
 USA
 Tel.: +1/217/795 4437
 Fax: +1/217/795 3348
 white.tech.res@gmail.com

Potential expert reviewers

David Klurfeld, PhD
 National Program Leader
 (Human Nutrition)
 USDA-ARS
 Room 4-2180
 5601 Sunnyside Ave
 Beltsville, MD 20705
 Tel.: (301) 504-4675
 david.klurfeld@ars.usda.gov

John P. Foreyt, PhD

Professor, Department of
 Psychiatry and Behavioral
 Sciences
 Department of Medicine
 Baylor College of Medicine
 6655 Travis
 St., Suite 320
 Houston, TX 77030
 Tel.: 713-798-5757
 jforeyt@bcm.edu

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In a recent paper, Gul et al. [1] suggested that increased fructose concentration induces the aging process and myocardial infarction through production of advanced glycation endproducts (AGEs), however, their supporting evidence is unconvincing.

First, it must be recognized that the dietary sources of fructose cited by the authors – honey, fruits, sucrose, and high fructose corn syrup – all contain essentially equal amounts of both fructose and glucose; crystalline fructose does not, but is a relatively minor dietary constituent, accounting for less than 2% of added sugars [2]. It follows, then, that any increase in dietary fructose must include an equivalent increase in glucose.

Second, increased exposure to added sugars – and fructose – is far lower than the authors believe. While total energy intake increased 515 kcal/d (24%) between 1970 and 2008 (USDA-ERS data), energy from added sugars (including honey, sucrose, high fructose corn syrup and fruit juice concentrates) increased only 58 kcal/d; energy from fructose accounted for no more than half this amount. By contrast, energy from flour/cereal products and added fats increased by 185 and 235 kcal/d, respectively. And loss-adjusted availability of added sugars has been in serious decline since 1999 [3].

Third, the authors fail to demonstrate that the positive correlation with advanced glycation endproducts (AGEs) is a unique feature of fructose. Indeed, based on the significantly increased fasting blood glucose in senile diabetic subjects with myocardial infarction vs. senile controls reported in Table 1, a similar positive and significant correlation between serum glucose and AGEs must surely be present.

And fourth, the authors' nonspecific test for AGEs fails to prove that those reported are due solely

or even materially to serum fructose. They could as well be formed from several alternate substrates or endogenous mechanisms continuously at work in the human body: directly from reversible reactions of substantial glucose, other carbohydrates or metabolic intermediates with amines; oxidative stress that converts glucose to dicarbonyls which further bind to proteins; lipid peroxidation; or metabolically, through fragmentation and elimination of phosphate from glycolytic intermediates [4].

Thus, the suggestion by Gul et al. that increased fructose concentration induces the aging process and myocardial infarction through production of AGEs is unconvincing, as it demonstrates neither a substantial recent increase of fructose in the diet nor a unique role for fructose in the development of AGEs.

Potential Conflict of Interest: The author is a consultant to the food and beverage industry in the area of nutritive sweeteners. His clients include research institutes, food industry councils, trade organizations and individual companies.

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