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EDITORIAL

SUGAR-SWEETENED BEVERAGES: A MISSING PIECE OF THE OBESITY PUZZLE?

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Every week there is an article published in the lay press on the current epidemic of obesity and on the wave of overweight/obese children who are at very high risk of beginning their young adult lives with diabetes or at least with their share of cardiovascular disease risk factors. For instance, it is really sad to note that type 2 diabetes has now struck the pediatric population [1].

Obesity is a complex condition as it is quite heterogeneous both in terms of etiology and related complications [2-4]. With the exception of rare genetic causes of obesity and endocrine diseases [5], most common forms of obesity are polygenic [6]. Thus, several susceptibility genes interact with permissive environmental factors leading to weight gain, body fat accumulation and eventually obesity [7, 8]. Obesity obviously results from an imbalance between caloric intake and energy expenditure [9, 10]. There is a debate, however, on the respective roles of increased caloric intake vs. reduced physical activity/exercise as the main driving force behind the epidemic of obesity observed over the last century [11]. On the energy intake part of the equation, it appears that we have recently moved from a technical discussion on the role of the macronutrient composition of the diet (such as the "high fat" diet which was a popular culprit in the eighties) to some key qualitative aspects of our nutritional habits [12]. For instance, it is now fairly well accepted that a low consumption of fruits and vegetables combined with excessive consumption of animal meat and of refined foods with a high energy density (not only rich in fat but also in refined sugar) are factors which promote obesity [12]. However, until recently, little attention had been given to calories consumed in liquid forms. There is now evidence that sugar-sweetened beverages have a low satiety potential [13, 14] and that increased consumption

of sugar-sweetened beverages are not associated with a compensatory reduction in the caloric intake from solid foods [15, 16]. Thus, calories from sugar-sweetened beverages essentially add to daily caloric intake. Although numerous factors contribute to the epidemic of obesity, limiting the intake of sugar-sweetened beverages could contribute to reduce daily caloric intake (even without other adjustments in eating habits) [17]. In a world where dieting is sometimes a "sport" very difficult to practice with problems of adherence and compliance, approaches to limit the intake of sugar-sweetened beverages could represent a very simple recommendation which could have significant clinical and public health implications.

This issue of the **CMReJournal** addresses this original question which identifies a new and potentially important target in our battle against the obesity epidemic. Three international experts cover different angles of the topic. In his paper, George Bray reviews the evidence available on the association between the consumption of sugar-sweetened beverages and obesity. Results from acute and short-term studies are discussed. In addition, results from the few meta-analyses available are presented. Bray concludes that there is indeed evidence of a relationship between the consumption of sugar-sweetened beverages and obesity and identifies fructose as a potentially important mediator of their deleterious effects on cardiometabolic risk variables. Recent evidence suggests that fructose may promote intra-abdominal (visceral) fat accumulation and liver fat deposition [18], two phenotypes associated with the presence of insulin resistance and the metabolic syndrome [2-4]. Clearly, further mechanistic studies are needed to examine this important question.

In his paper, Frank Hu reviews the key epidemiological data on the relationship between consumption of sugar-sweetened beverages and risk of type 2 diabetes and cardiovascular disease. Hu also reaches the conclusion that consumption of sugar-sweetened beverages increases risk of both diabetes and cardiovascular disease in a manner which may be partly independent from body weight gain. The latter finding is also concordant with the results of metabolic studies highlighting the potentially deleterious effects of fructose on cardiometabolic risk.

Thus, there is evidence that sugar-sweetened beverages have deleterious effects on adiposity and the risk of both diabetes and cardiovascular disease. In the paper introducing the issue, Popkin provides data showing that the consumption of sugar-sweetened beverages has increased over the last few decades, a phenomenon which must be a source of concern as it contributes to increase total caloric intake. Popkin endorses the position of Bray and Hu on the hazards of a high consumption of sugar-sweetened beverages and moves on to propose possible solutions. His proposal to consider taxation of sugar-sweetened beverages has been the topic of another paper that he recently co-signed with Dr. Kelly Brownell as the leading author in the NEJM [13]. The "mechanics" of how to possibly apply such tax is further discussed in this recent paper [13]. Furthermore, he suggests that regulations should be put in place so that vending machines selling sugar-sweetened beverages in schools to vulnerable populations and in the workplace should be banned. Of course, actions should also be taken to make sure that children have easy access to safe water.

In the current epidemic of type 2 diabetes and considering that patients with type 2 diabetes are younger than ever because of their obesity, a robust action plan should be put in place to battle the epidemic of obesity underlying the huge prevalence figures reached by this metabolic disease (type 2 diabetes) which have been suggested to limit our ability to successfully battle cardiovascular disease in the 21st century. The question is complex as numerous societal, individual and biological factors are at interplay. However, on top of all the appropriate nutritional guidance that our patients should receive, it appears that a focus on the consumption of sugar-sweetened beverages may represent a simple initial step in an attempt to reduce caloric intake. Thus, limiting the consumption of sugar-sweetened beverages represents one element of a healthy lifestyle to prevent or manage obesity and its related complications.

References

1. Rosenbloom AL, Joe JR, Young RS, et al. Emerging epidemic of type 2 diabetes in youth. Diabetes Care 1999; 22: 345-54.

2. Després JP and Lemieux I. Abdominal obesity and metabolic syndrome. Nature 2006; 444: 881-7.

3. Després JP, Lemieux I, Bergeron J, et al. Abdominal obesity and the metabolic syndrome: contribution to global cardiometabolic risk. Arterioscler Thromb Vasc Biol 2008; 28: 1039-49.

4. Van Gaal LF, Mertens IL and De Block CE. Mechanisms linking obesity with cardiovascular disease. Nature 2006; 444: 875-80.

5. O'Rahilly S and Farooqi IS. Human obesity as a heritable disorder of the central control of energy balance. Int J Obes (Lond) 2008; 32 Suppl 7: S55-61.

6. Rankinen T, Zuberi A, Chagnon YC, et al. The human obesity gene map: the 2005 update. Obesity (Silver Spring) 2006; 14: 529-644.

7. Bouchard C, Tremblay A, Després JP, et al. The response to long-term overfeeding in identical twins. N Engl J Med 1990; 322: 1477-82.

8. Bouchard C, Després JP and Mauriège P. Genetic and nongenetic determinants of regional fat distribution. Endocr Rev 1993; 14: 72-93.

9. Doucet E and Tremblay A. Food intake, energy balance and body weight control. Eur J Clin Nutr 1997; 51: 846-55.

10. Hill JO. Understanding and addressing the epidemic of obesity: an energy balance perspective. Endocr Rev 2006; 27: 750-61.

11. Jeffery RW and Utter J. The changing environment and population obesity in the United States. Obes Res 2003; 11 Suppl: 12S-22S.

12. Gidding SS, Lichtenstein AH, Faith MS, et al. Implementing American Heart Association pediatric and adult nutrition guidelines: a scientific statement from the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism, Council on Cardiovascular Disease in the Young, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, and Council for High Blood Pressure Research. Circulation 2009; 119: 1161-75.

13. Brownell KD, Farley T, Willett WC, et al. The public health and economic benefits of taxing sugar-sweetened beverages. N Engl J Med 2009; 360: 1599-605.

14. Mourao DM, Bressan J, Campbell WW, et al. Effects of food form on appetite and energy intake in lean and obese young adults. Int J Obes (Lond) 2007; 31: 1688-95.

15. De Castro JM. The effects of the spontaneous ingestion of particular foods or beverages on the meal pattern and overall nutrient intake of humans. Physiol Behav 1993; 53: 1133-44.

16. Harnack L, Stang J and Story M. Soft drink consumption among US children and adolescents: nutritional consequences. J Am Diet Assoc 1999; 99: 436-41.

17. Mattes RD and Popkin BM. Nonnutritive sweetener consumption in humans: effects on appetite and food intake and their putative mechanisms. Am J Clin Nutr 2009; 89: 1-14.

18. Stanhope KL, Schwarz JM, Keim NL, et al. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. J Clin Invest 2009; 119: 1322-34.