
Current Knowledge of the Health Effects of Sugar Intake

Anne L. Mardis, MD, MPH¹
Center for Nutrition Policy
and Promotion

Twenty years ago, the common perception was that sugar intake was associated with several chronic diseases: Diabetes, coronary heart disease, obesity, and hyperactivity in children. Sugar was also thought to be the sole cause of dental caries. Recent advances in scientific knowledge, however, have shed some light on the role of sugar in chronic diseases and dental caries. The evidence indicates that sugar is not in itself associated with the aforementioned chronic diseases and is not the sole offender in the development of dental caries. This research brief discusses current scientific knowledge of the health effects of sugar.

Physiology

Despite having been labeled as “empty calories,” sugars are truly important compounds from the perspective of the human organism. Humans have retained the ability to synthesize all forms of carbohydrates the body needs from simple sugars. This is not the case with the other dietary macronutrients, fats, and proteins. Following ingestion, all digestible complex dietary carbohydrates are broken down in the gut to simple sugars before they are absorbed into the body. Because simple sugars are all identical chemically, the absorption process cannot distinguish simple sugars resulting from the breakdown of complex dietary carbohydrates from corresponding simple sugars occurring naturally in the foods

themselves or from corresponding simple sugars added to foods during processing. Within the body, most dietary sugars are converted to glucose, a major fuel used by all cells and the primary fuel required by brain tissue for normal function. Low levels of glucose in the blood will impair the brain and cause permanent mental impairment or worse—coma or death. The body can store a limited amount of glucose as glycogen, which it can draw upon for less than a day. After this, other sources such as proteins, from the breakdown of body tissues, must be used to synthesize glucose for the cells (15).

Diabetes

The relationship between dietary carbohydrates and insulin resistance (a risk factor for diabetes mellitus, ischemic heart disease, and hypertension) is not clear based on available research (7). In two studies based on a large, prospective study of U.S. women, sucrose and carbohydrate intake were not associated with an increased risk of diabetes (6,27). However, based on the same population, associations were found between a diet with high glycemic load² (26) and high intake of refined grains (21) and the risk of diabetes. The general consensus, based on epidemiological studies, is that sugar intake alone is not

¹Currently at the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Morgantown, WV.

²Glycemic load is a function of the effect of a carbohydrate meal on glucose levels in the blood (3).

associated with the development of diabetes mellitus. Sugars fed at levels equivalent to those consumed by the U.S. population do not produce adverse glycemic effects in non-diabetics (23). The effects of sugar intake on glucose tolerance, insulin levels, and plasma lipids are confounded by other dietary components. The American Diabetes Association has also acknowledged, in its nutrition recommendations for people with diabetes, that there is no evidence that refined sugars such as sucrose behave any differently from other types of simple carbohydrates (1).

Heart Disease

The Sugars Task Force of the U.S. Food and Drug Administration (29) presented a comprehensive review of epidemiological, clinical, and animal studies dealing with the relationship between sugar intake and heart disease or risk factors for heart disease (14). The report concluded that at current levels of consumption, sugar is not an adverse risk factor in heart disease. The same conclusion was made by the National Research Council in its report on chronic disease risk (23). There is no conclusive evidence that dietary sugar is an independent risk factor for coronary artery disease in the general population. However, hypertriglyceridemia³ and central fat distribution,⁴ consequences of abnormal glucose tolerance and diabetes mellitus, are independent risk factors for coronary heart disease (8). A 1996 randomized

³Elevated blood levels of triglycerides: a form of fatty acid found in animal and vegetable fats.

⁴A type of body fat distribution with a high ratio of waist or abdominal circumference to hip or gluteal circumference that is epidemiologically associated with heart disease and diabetes.

study of 32 hypertriglyceridemic patients provided evidence that an “extrinsic sugar-free” diet significantly lowers abnormally elevated plasma triglyceride levels (28). Evidence also suggests that insulin resistance and compensatory hyperinsulinemia⁵ have major roles in the regulation of blood pressure in subjects predisposed to hypertension due to hereditary or environmental factors, possibly mediated by activity of the sympathetic nervous system. But there are multiple metabolic abnormalities associated with hyperinsulinemia in hypertensive patients that increase the risk of coronary heart disease (24).

Obesity

Despite popular belief that sugar causes obesity, a number of studies show an inverse relationship between reported sugar consumption and degree of overweight (10,11,20,25). An increase in the percentage of calories from sugar is, by definition, associated with a decreased consumption of calories from fat. Obesity is basically a consequence of higher energy intake than energy expenditure, where excess calories are stored as fat (5). The type of calories consumed is the subject of much study in obesity research. For instance, extra calories consumed as sugar cause an appropriate compensatory increase in carbohydrate oxidation (metabolism of carbohydrates for energy), whereas extra calories consumed as fat do not (17). Simply stated, obesity results from energy intake in excess of energy requirements. Many factors contribute to obesity, but evidence does not single out dietary sugar as a cause (25).

⁵An increase in pancreatic secretion of insulin to compensate for cellular resistance to insulin.

Hyperactivity

Many people still believe that sugar intake in children causes hyperactivity. A meta-analysis, however, of 16 different reports from 23 separate studies with 560 subjects showed virtually no effect of sugar intake on the hyperactivity in children (30). In a review of the literature, Krummel et al. (19) reported that in 12 double-blind, placebo-controlled studies, no association was found between sugar intake and hyperactive behavior. Thus, despite numerous anecdotal perceptions to the contrary, systematic, controlled studies show that sugars do not cause hyperactivity.

Dental Caries

Dental caries is a chronic disease that has many causes. Sugar is involved in tooth decay, but it is one of many factors, including oral bacteria, saliva, tooth enamel, food substrate, and host susceptibility. All fermentable carbohydrates are potentially cariogenic. Other dietary factors such as the retention of food in the mouth affect cariogenic potential. Even starches, which may not taste sweet, are chains of glucose and are broken down to glucose in the mouth. Good oral hygiene, good genes, fluoridation of water, and restricting snacks between meals can prevent tooth decay, no matter how high the sugar consumption (13,18,25).

Nutrient Displacement

Research on the effects of sugar intake and nutrient displacement in the diet of children is inconclusive. In a review of the literature, Ruxton et al. (25) found that higher intake of sugar did not negatively affect micronutrient intake. Gibney et al. (10) found, in an analysis

of the 1987-88 USDA Nationwide Food Consumption Survey, that high consumption of sugars was not associated with a poorer quality diet. In a study of 143 children ages 11 and 12 years, a 7-day weighed and recorded food inventory revealed that as the proportion of energy from sugars increased, there was no decline in dietary fiber or micronutrient intake, with the exception of niacin, which exceeded recommended levels (22). However, Linseisen et al. (20) and Gibson (12) did demonstrate intake of many micronutrients below recommended levels in persons in Germany and the United Kingdom who consumed high (energy-adjusted) amounts of sugar. In addition, high consumption of non-diet soft drinks, a significant source of added-sugar intake in children, is associated with lower consumption of milk and fruit juice and lower intake of riboflavin, vitamin A, calcium, phosphorus, and the ratio of calcium to phosphorus, which may be considered markers for milk consumption (16).

In an analysis of the Continuing Survey of Food Intakes by Individuals (1994-96), Bowman found that compared with Americans over 2 years of age with lower added sugar consumption as a percentage of total energy, individuals consuming greater than 18 percent of their total energy from added sugars did not meet the Recommended Daily Allowance (RDA) for many micronutrients (4). Farris et al. reported that as total sugar intake increased, a significant linear decrease occurred in mean intake of protein, fat, saturated fat, starch, cholesterol, sodium, vitamins B₆ and E, thiamin, niacin, iron, and zinc. Also, as total sugar intake increased, a significant linear increase occurred in mean intake of carbohydrate, fructose, lactose, sucrose, vitamin D, and calcium (9).

But, the nutritional quality of the diet of children with higher sugar intake appeared to be adequate regarding vitamin and mineral intakes and were closer to meeting current recommendations for dietary fat. Nevertheless, a relationship between the consumption of sugars and nutrient displacement has not been observed consistently nor has there been consistency among the specific nutrients displaced when a relationship has been found. Thus, this issue remains unsettled and requires additional data from primary research.

Conclusion

Recent evidence shows that aside from dental caries, the intake of added sugars is not directly related to diabetes, heart disease, obesity, and hyperactivity, as was previously thought. This conclusion was also reached in a 1997 review of the literature on the health effects of sugar intake (2). Because high intake of sugars along with other factors can affect oral health and can displace important foods and nutrients in the diets of children when consumed as soft drinks, it seems prudent to limit excessive intake. But the focus on sugar as an independent risk factor for chronic disease and hyperactivity should be de-emphasized.

References

1. American Diabetes Association. 1994. Nutrition recommendations and principles for people with diabetes mellitus. *Diabetes Care* 17:519-522.
2. Anderson, G.H. 1997. Sugars and health: A review. *Nutrition Research* 17(9):1485-1498.
3. Bornet, F.R., Billaux, M.S., and Messing, B. 1997. Glycaemic index concept and metabolic diseases. *International Journal of Biological Macromolecules* 21(1-2):207-219.
4. Bowman, S.A. 1999. Diets of individuals based on energy intakes from added sugars. *Family Economics and Nutrition Review* 12(2):31-38.
5. Bray, G.A. 1996. Obesity. In Ekhard E. Aiegler and L.J. Filer, Jr. (Eds.), *Present Knowledge in Nutrition* (7th ed.) (Chapter 2). International Life Sciences Institute Press, Washington, DC.
6. Colditz, G.A., Manson, J.E., Stampfer, M.J., Rosner, B., Willett, W.C., and Speizer, F.E. 1992. Diet and risk of clinical diabetes in women. *American Journal of Clinical Nutrition* 55(5):1018-1023.
7. Daly, M.E., Vale, C., Walker, M., Alberti, K.G., and Mathers, J.C. 1997. Dietary carbohydrates and insulin sensitivity: A review of the evidence and clinical implications. *American Journal of Clinical Nutrition* 66(5):1072-1085.
8. Eschwege, E., Balkau, B., and Fontbonne, A. 1994. The epidemiology of coronary heart disease in glucose-intolerant and diabetic subjects. *Journal of International Medicine* (Suppl)736:5-11.
9. Farris, R.P, Nicklas, T.A, Myers, L., and Berenson, G.S. 1998. Nutrient intake and food group consumption of 10-year-olds by sugar intake level: The Bogalusa Heart Study. *Journal of the American College of Nutrition* 17(6):579-85.
10. Gibney, M., Sigman-Grant, M., Stanton, J.L.J., and Keast, D.R. 1995. Consumption of sugars [published erratum appears in *American Journal of Clinical Nutrition* 1997 May;65(5):1572-1574]. *American Journal of Clinical Nutrition* 62(1 Suppl):178S-193S.
11. Gibson, S. and Williams, S. 1999. Dental caries in pre-school children: Associations with social class, toothbrushing habit and consumption of sugars and sugar-containing foods. Further analysis of data from the National Diet and Nutrition Survey of children aged 1.5-4.5 years. *Caries Research* 33(2):101-113.
12. Gibson, S.A. 1996. Are high-fat, high-sugar foods and diets conducive to obesity? *International Journal of Food Sciences and Nutrition* 47(5):405-415.
13. Gibson, S.A. 1997. Non-milk extrinsic sugars in the diets of pre-school children: Association with intakes of micronutrients, energy, fat and NSP. *British Journal of Nutrition* 78(3):367-378.
14. Glinsmann, W.H., Iransquin, H., and Park, Y.K. 1986. Report from FDA's Sugars Task Force: Evaluation of Health Aspects of Sugars Contained in Carbohydrate Sweeteners. *Journal of Nutrition* 166(155):51-216.
15. Guyton and Hall. 1996. *Textbook of Medical Physiology*, 9th Edition. W.B. Saunders, Philadelphia.

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16. Harnack, L., Stang, J., and Story, M. 1999. Soft drink consumption among US children and adolescents: Nutritional consequences. *Journal of the American Dietetic Association* 99(4):436-441.
 17. Horton, T.J., Drougas, H., Brachey, A., Reed, G.W., Peters, J.C., and Hill, J.O. 1995. Fat and carbohydrate overfeeding in humans: Different effects on energy storage. *American Journal of Clinical Nutrition* 62(t):19-29.
 18. Kandelman, D. 1997. Sugar, alternative sweeteners and meal frequency in relation to caries prevention: New perspectives. *British Journal of Nutrition* 77(Suppl 1):S121-S128.
 19. Krummel, D.A., Seligson, F.H., and Guthrie, H.A. 1996. Hyperactivity: Is candy causal? *Critical Reviews in Food Science and Nutrition* 36(1-2):31-47.
 20. Linseisen, J., Gedrich, K., Karg, G., and Wolfram, G. 1998. Sucrose intake in Germany. *Zeitschrift für Ernährungswissenschaft* 37(4):303-314.
 21. Liu, S., Manson, J.E., Stampfer, M.J., Hu, F.B., Giovannucci, E., Colditz, G.A., Hennekens, C.H., and Willett, W.C. 2000. A prospective study of whole-grain intake and risk of type 2 diabetes mellitus in US women. *American Journal of Public Health* 90(9):1409-1415.
 22. Naismith, D.J., Nelson, M., Burley, V., and Gatenby, S.J. 1995. Does a high-sugar diet promote overweight in children and lead to nutrient deficiencies? *Journal of Human Nutrition and Dietetics* 8:249-254.
 23. National Research Council, Committee on Diet and Health, Food and Nutrition Board. 1989. *Diet and Health: Implications for Reducing Chronic Disease Risk*. National Academy Press, Washington, DC.
 24. Reaven, G.M., Lithell, H., and Landsberg, L. 1996. Hypertension and associated metabolic abnormalities—the role of insulin resistance and the sympathoadrenal system. *New England Journal of Medicine* 334(6):374-381.
 25. Ruxton, C.H., Garceau, F.J., and Cottrell, R.C. 1999. Guidelines for sugar consumption in Europe: Is a quantitative approach justified? *European Journal of Clinical Nutrition* 53(7):503-513.
 26. Salmeron, J., Manson, J.E., Stampfer, M.J., Colditz, G.A., Wing, A.L., and Willett, W.C. 1997. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women [see comments]. *JAMA* 277(6):472-477.
 27. Shimakawa, T., Herrera-Acena, M.G., Colditz, G.A., Manson, J.E., Stampfer, M.J., and Willett, W.C. 1993. Comparison of diets of diabetic and nondiabetic women [published erratum appears in *Diabetes Care* 1994 Apr;17(4):349]. *Diabetes Care* 16(10):1356-1362.
 28. Smith, J.B., Niven, B.E., and Mann, J.I. 1996. The effect of reduced extrinsic sucrose intake on plasma triglyceride levels. *European Journal of Clinical Nutrition* 50(8):498-504.
 29. Sugars Task Force. 1986. *Evaluation of Health Aspects of Sugars Contained in Carbohydrate Sweeteners*. Health and Human Services, Food and Drug Administration. Washington, DC.
 30. Wolraich, M.L., Wilson, D.B., and White, J.W. 1995. The effect of sugar on behavior or cognition in children. A meta-analysis. *JAMA* 274(20):1617-1621.