

AHA Scientific Statement

Dietary Sugars Intake and Cardiovascular Health A Scientific Statement From the American Heart Association

Rachel K. Johnson, PhD, MPH, RD, Chair; Lawrence J. Appel, MD, MPH, FAHA;
Michael Brands, PhD, FAHA; Barbara V. Howard, PhD, FAHA;
Michael Lefevre, PhD, FAHA; Robert H. Lustig, MD; Frank Sacks, MD, FAHA;
Lyn M. Steffen, PhD, MPH, RD, FAHA; Judith Wylie-Rosett, EdD, RD;
on behalf of the American Heart Association Nutrition Committee of the Council on Nutrition,
Physical Activity, and Metabolism and the Council on Epidemiology and Prevention

Abstract—High intakes of dietary sugars in the setting of a worldwide pandemic of obesity and cardiovascular disease have heightened concerns about the adverse effects of excessive consumption of sugars. In 2001 to 2004, the usual intake of added sugars for Americans was 22.2 teaspoons per day (355 calories per day). Between 1970 and 2005, average annual availability of sugars/added sugars increased by 19%, which added 76 calories to Americans' average daily energy intake. Soft drinks and other sugar-sweetened beverages are the primary source of added sugars in Americans' diets. Excessive consumption of sugars has been linked with several metabolic abnormalities and adverse health conditions, as well as shortfalls of essential nutrients. Although trial data are limited, evidence from observational studies indicates that a higher intake of soft drinks is associated with greater energy intake, higher body weight, and lower intake of essential nutrients. National survey data also indicate that excessive consumption of added sugars is contributing to overconsumption of discretionary calories by Americans. On the basis of the 2005 US Dietary Guidelines, intake of added sugars greatly exceeds discretionary calorie allowances, regardless of energy needs. In view of these considerations, the American Heart Association recommends reductions in the intake of added sugars. A prudent upper limit of intake is half of the discretionary calorie allowance, which for most American women is no more than 100 calories per day and for most American men is no more than 150 calories per day from added sugars. (*Circulation*. 2009; 120:000-000.)

Key Words: AHA Scientific Statements ■ cardiovascular diseases ■ carbohydrates, dietary ■ diet ■ beverages ■ carbonated beverages ■ lipids

New evidence on the relationship between intake of sugars and cardiovascular health has emerged since the last American Heart Association (AHA) scientific statement was published in 2002.¹ In 2006, the AHA published revised diet and lifestyle recommendations that recommend minimizing the intake of beverages and foods with added sugars.² The present statement expands on that recommendation by reviewing the evidence for recommending a specific upper limit of intake for added sugars. Because the focus of the

present statement is on added sugars, recommendations for intake of naturally occurring sugars and complex carbohydrates are beyond its scope.

Consumption of Sugars in the United States

Sugars are a ubiquitous component of our food supply and are consumed as a naturally occurring component of many foods and as additions to foods during processing, preparation, or at the table.³ There are various definitions for *sugar*. Table 1

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on June 26, 2009. A copy of the statement is available at <http://www.americanheart.org/presenter.jhtml?identifier=3003999> by selecting either the "topic list" link or the "chronological list" link (No. KJ-0727). To purchase additional reprints, call 843-216-2533 or e-mail kelle.ramsay@wolterskluwer.com

The American Heart Association requests that this document be cited as follows: Johnson RK, Appel LJ, Brands M, Howard BV, Lefevre M, Lustig RH, Sacks F, Steffen LM, Wylie-Rosett J; on behalf of the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity, and Metabolism and the Council on Epidemiology and Prevention. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. *Circulation*. 2009;120:000-000.

Expert peer review of AHA Scientific Statements is conducted at the AHA National Center. For more on AHA statements and guidelines development, visit <http://www.americanheart.org/presenter.jhtml?identifier=3023366>.

Permissions: Multiple copies, modification, alteration, enhancement, and/or distribution of this document are not permitted without the express permission of the American Heart Association. Instructions for obtaining permission are located at <http://www.americanheart.org/presenter.jhtml?identifier=4431>. A link to the "Permission Request Form" appears on the right side of the page.

© 2009 American Heart Association, Inc.

Circulation is available at <http://circ.ahajournals.org>

DOI: 10.1161/CIRCULATIONAHA.109.192627

Table 1. Common Definitions

Simple carbohydrates (sugars) refers to monosaccharides and disaccharides. Monosaccharides include glucose, galactose, and fructose. Dextrose is synonymous with glucose. Fructose is the most common naturally occurring monosaccharide, found in fruits and vegetables. Common disaccharides include sucrose (glucose plus fructose), which is found in sugar cane, sugar beets, honey, and corn syrup; lactose (glucose plus galactose), found in milk products; and maltose (glucose plus glucose), found in malt.

Complex carbohydrates refers to glucose-containing polysaccharides, such as starch.

Naturally occurring (intrinsic) sugars refers to sugars that are an integral part of whole fruit, vegetable, and milk products.

Added (extrinsic) sugars refers to sugars and syrups added to foods during processing or preparation and includes sugars and syrups added at the table.

Total sugars are defined as all sugars (naturally occurring and added) in foods and beverages.

High-fructose corn syrup is produced from corn syrup (nearly all glucose), which undergoes enzymatic processing to increase the fructose content and is then mixed with glucose.

lists the common descriptions and definitions of sugars used in the literature.

As expected, a healthy, well-balanced diet contains naturally occurring sugars, because monosaccharides such as fructose and disaccharides such as sucrose and lactose are integral components of fruit, vegetables, dairy products, and many grains. In addition, sugars add desirable sensory effects to many foods, and a sweet taste promotes enjoyment of meals and snacks. In fact, when sugars are added to otherwise nutrient-rich foods, such as sugar-sweetened dairy products like flavored milk and yogurt and sugar-sweetened cereals, the quality of children's and adolescents' diets improves,^{4,5} and in the case of flavored milks, no adverse effects on weight status were found.⁶ However, deleterious health effects may occur when sugars are consumed in large amounts.

Food availability data (also called *disappearance data*), used in conjunction with self-reported food consumption data from nationwide surveys, provide information on consumption of sugars in the United States. According to the US Department of Agriculture's Economic Research Service, between 1970 and 2005, sugars and sweeteners available for consumption increased by an average of 76 calories per day, from 25 teaspoons (400 calories) to 29.8 teaspoons (476 calories), which corresponds to a 19% increase.⁷ Bray et al⁸ estimated that intake of added sugars increased from 235 kcal/d per person in 1977 to 1978 to 318 kcal/d per person in 1994 to 1995, and high-fructose corn syrup intake increased from 80 to 132 kcal/d per person during the same time period.

Added sugars are defined as sugars and syrups that are added to foods during processing or preparation, including sugars and syrups added at the table. Between 1994 and 2002, Americans' intake of added sugars remained high and unchanged among 6- to 19-year-olds and increased among those 20 years old and older (personal correspondence from A.J. Moshfegh, US Department of Agriculture, Food Surveys Research Group, October 2007). The National Cancer Institute recently estimated usual intakes of added sugars on the basis of data from the 2001 to 2004 National Health and

Table 2. Usual Intake of Added Sugars (in Teaspoons),* 2001–2004

Age, y	n	Mean	SE
1–3	1515	12.2	0.33
4–8	1701	21.0	0.54
Males 9–13	1061	29.2	0.92
Males 14–18	1424	34.3	1.03
Males ≥19	4650	25.4	0.48
Females 9–13	1112	23.2	0.82
Females 14–18	1362	25.2	0.71
Females ≥19	5063	18.3	0.37
All persons ≥1	17 888	22.2	0.29

n Indicates number of persons in sample; SE, standard error of the mean (degrees of freedom=30).

*Includes white, brown, and raw sugar; syrup; honey; and molasses, eaten separately or used as ingredients in processed or prepared foods such as breads, cakes, soft drinks, jams, and ice cream.

One teaspoon of added sugars has the same amount of total sugars as 1 teaspoon (4 g) of table sugar (sucrose).

Adapted from National Cancer Institute.⁹

Nutrition Examination Survey (NHANES). The mean intake for all persons was 22.2 teaspoons per day (355 calories); 14- to 18-year-old children had the highest intakes at 34.3 teaspoons per day (549 calories⁹; Table 2). On the basis of principles elucidated in the 2005 US Dietary Guidelines,¹⁰ the MyPyramid World Wide Web site^{11,12} estimates that these intakes far exceed the allowance for discretionary calories, regardless of energy needs (Table 3).

Increases in the intake of soft drinks, fruit drinks, desserts, sugars and jellies, candy, and ready-to-eat cereals largely account for the increased energy intake from sugars/added sugars. Soft drinks and other sugar-sweetened beverages are the primary source of added sugars in Americans' diets¹³ (Table 4). Between 1970 and 2000, per-person daily consumption of caloric soft drinks increased 70%, from 7.8 to 13.2 ounces.¹⁴

Currently, US food labels contain information on total sugars per serving but do not distinguish between sugars that are naturally present in foods and added sugars. Thus, it is difficult for consumers to determine the amount of added sugars in foods and beverages. In 2006, the US Department of Agriculture published a database for the added sugar content of selected foods.¹⁵ In addition, several voluntary food-labeling systems are in place or are being developed, some of which include criteria for limiting added sugars. These include the Smart Choices Program,¹⁶ the Hannaford's grocery chain Guiding Stars program,¹⁷ the Overall Nutrient Quality Index,¹⁸ and the Nutrient Rich Foods Coalition nutrient density score.¹⁹

Fructose

Originally proposed as the ideal sweetener for people with diabetes mellitus because of its inability to stimulate insulin secretion, fructose consumption has been indirectly implicated in the epidemics of obesity and type 2 diabetes mellitus.^{20–22} Fructose is a monosaccharide naturally found in fruits and honey. Many consumers mistakenly believe that

AQ:6

T2,AQ:8

T3,AQ:9

T4,AQ:10

AQ:7

Table 3. Discretionary Calories Allowance and Examples of How These Calories May Be Divided Between Solid Fats and Added Sugars on the Basis of the US Department of Agriculture Food Guide

	Food Guide Daily Calorie Level									
	1200	1400	1600*	1800	2000	2200	2400	2600	2800	3000
Discretionary calories†	171	171	132	195	267	290	362	410	426	512
Discretionary calories as a % of total calories	14	12	8	11	13	13	15	16	15	17
Solid fats, g‡	14	14	11	15	18	19	22	24	24	29
Added sugars, g (tsp)§	16 (4)	16 (4)	12 (3)	20 (5)	32 (8)	36 (9)	48 (12)	56 (14)	60 (15)	72 (18)

*The discretionary calorie allowance suggested for the 1600-calorie level is lower than the 1400-calorie pattern because it traditionally represents a calorie recommendation for young children (4 to 8 years of age). To accommodate all of the food groups to meet nutrient requirements for this age group, fewer calories are available for a discretionary calorie allowance.

†Total discretionary calories should be limited to the amounts shown in the Table at each calorie level. The calories assigned to discretionary calories may be used to increase intake from the basic food groups; to select foods from these groups that are higher in fat or that have added sugars; to add oils, solid fats, or sugars to foods or beverages; or to consume alcohol.

‡Amounts of solid fats listed in the Table represent approximately 7% to 8% of calories from saturated fat. Foods in each food group are represented in their lowest-fat forms, such as fat-free milk and skinless chicken. Solid fats shown in this Table represent the amounts of fats that may be added in cooking or at the table, as well as fats consumed when higher-fat items are selected from the food groups (eg, whole milk instead of fat-free milk, chicken with skin, or cookies instead of bread) without exceeding the recommended limits on saturated fat intake. Solid fats include meat and poultry fats eaten either as part of the meat or poultry product or separately; milk fat, such as that in whole milk, cheese, and butter; shortenings used in baked products; and hard margarines.

§Added sugars are the sugars and syrups added to foods and beverages in processing or preparation, not the naturally occurring sugars in fruits or milk. The amounts of added sugars suggested in the example are not specific recommendations for amounts of added sugars to consume but rather represent the amounts that can be included at each calorie level without overconsuming calories. The suggested amounts of added sugars may be helpful as part of the Food Guide to allow for some sweetened foods or beverages without exceeding energy needs. This use of added sugars as a calorie balance requires 2 assumptions: (1) That selections are made from all food groups in accordance with the suggested amounts, and (2) that additional fats are used in the amounts shown, which, together with the fats in the core food groups, represent approximately 27% to 30% of calories from fat.

Adapted from US Department of Health and Human Services and US Department of Agriculture, *Dietary Guidelines for Americans, 2005*.¹⁰

high-fructose corn syrup is pure fructose. High-fructose corn syrup is composed of either 42% or 55% fructose and is similar in composition to table sugar (sucrose).^{23,24} High-fructose corn syrup is the sweetener commonly used by the beverage industry.

Cross-sectional studies in humans link soft drink consumption with higher energy intake, greater body weight, and poor nutrition^{25,26} and suggest that excessive fructose consumption is playing a role in the epidemics of insulin resistance, obesity, hypertension, dyslipidemia, and type 2 diabetes mellitus in humans.^{20,27–30} For example, a recent, albeit small, metabolic study in overweight and obese adults suggests that consumption of fructose-sweetened beverages leads to dyslipidemia, increased fasting blood glucose, decreased insulin

sensitivity, and increased visceral adiposity.³¹ Other trials have documented that limiting soft drink consumption has modest beneficial effects on weight in children.^{32,33}

Glucose-Insulin Response

Many factors influence the body’s glucose response to foods, including the composition of the food (fat, protein, sugar, starch, and fiber content), the method of food processing and preparation, the combination of foods eaten, and physiological factors including age and body composition.^{34–36} Glucose control is the net effect of metabolic processes that remove glucose from the blood for either glycogen synthesis or energy production and of gluconeogenesis and glycogenolysis, which return glucose to the blood. The rise in blood glucose after consumption of a carbohydrate triggers the release of insulin and at the same time reduces the secretion of glucagon.³⁷

There is a common belief that consumption of sucrose results in higher blood glucose levels than consumption of starch; however, the form of starch (cooked versus uncooked) results in different glucose responses. Cooked starch, such as bread, rice, and potatoes, evokes glucose responses similar to glucose, whereas uncooked starch is more slowly absorbed than cooked starch, resulting in a lower glycemic response.³⁸ However, cooked whole oats produce a relatively low glucose response, whereas most raw processed cereals produce very high responses. Thus, processing and preparation are key factors in the body’s glucose response to food consumption.

In a feeding study of 10 healthy adults, glucose and insulin responses were determined for high-sugar refined-grain snacks (chocolate-coated candy bar, cola drink with crisps) and whole-food snacks (raisins and peanuts; bananas and

Table 4. Major Sources of Added Sugars in the American Diet

Food Categories	Contribution to Added Sugars Intake (% of Total Added Sugars Consumed)
Regular soft drinks	33.0
Sugars and candy	16.1
Cakes, cookies, pies	12.9
Fruit drinks (fruitades and fruit punch)	9.7
Dairy desserts and milk products (ice cream, sweetened yogurt, and sweetened milk)	8.6
Other grains (cinnamon toast and honey-nut waffles)	5.8

Food groups that contribute more than 5% of the added sugars to the American diet are listed in decreasing order.

Data derived from Guthrie and Morton.¹³

peanuts) of similar energy and fat content. Consumption of the sugary refined-grain snacks resulted in higher glucose and insulin levels than consumption of the whole-food snacks, which suggests that the glycemic response to food is influenced by the carbohydrate content and physical state of the food, such as processed or whole, liquid or solid.³⁹ Fiber content was also higher in the whole-food snacks than in the high-sugar refined-grain snacks, which contributed to a reduced postprandial glucose response with the whole-food snacks compared with the refined-grain snacks.⁴⁰

Effects of Dietary Sugars on Blood Pressure, Lipids, and Inflammation

An emerging but inconclusive body of evidence suggests that increased intake of added sugars might raise blood pressure.^{41–43} Studies include animal studies in which rats were fed high doses of fructose, acute ingestion studies in which humans were fed high doses of different sugars, and more recently, epidemiological studies, such as the Framingham Heart Study, in which consumption of ≥ 1 soft drink per day significantly increased the odds of developing high blood pressure.³⁰ Nonetheless, results from studies in humans are inconsistent,^{44,45} and the chronic effects of a high intake of simple sugars on blood pressure remain uncertain.

It is well established that when used to replace dietary fats, carbohydrates can elevate plasma triglyceride levels and lower levels of high-density lipoprotein cholesterol^{46–48}; however, the type of carbohydrate appears to influence lipid responses. A diet such as the DASH (Dietary Approaches to Stop Hypertension) diet that replaces fat with carbohydrate from fruits, vegetables, whole grains, and nonfat and low-fat dairy products does not increase triglycerides but still lowers high-density lipoprotein cholesterol somewhat.^{36,49} In the Women's Health Initiative, the higher-carbohydrate diet had no effect on triglycerides or high-density lipoprotein cholesterol.⁵⁰

Elevations in fasting plasma triglycerides, principally very-low-density lipoproteins, are a consistent feature of diets high ($>20\%$ of energy) in sucrose, glucose, and fructose.^{20,31,51} The effects of sucrose or fructose on fasting triglycerides may be more marked in men than women,⁵² in sedentary overweight people or those with the metabolic syndrome,⁵¹ and in those eating low-fiber diets.⁵¹ Sucrose and fructose also increase postprandial triglyceride levels and may augment the lipemia associated with fat-containing meals.^{53,54} There are several mechanisms by which fructose increases fasting and postprandial triglyceride levels. These include increased de novo lipogenesis in the liver,^{31,51,55} increased hepatic triglyceride synthesis, and secretion of very-low-density lipoproteins,⁵³ as well as reduced lipoprotein lipase activity at the adipocyte, which decreases the rate of peripheral triglyceride clearance.^{51,53}

Compared with other carbohydrate sources (starch or glucose), recent studies have not demonstrated an added effect of either sucrose or fructose to lower high-density lipoprotein cholesterol levels^{52,56,57}; however, these studies have not agreed on the effects on low-density lipoprotein cholesterol. In summary, although the mechanisms are unclear, relative to other carbohydrate sources, sugar intake

appears to be associated with increased triglyceride levels, a known risk factor for coronary heart disease; however, relative to other sources of carbohydrate, the effects of sugar intake on high-density lipoprotein and low-density lipoprotein levels remain unclear.

In some^{58–60} but not all studies,^{61,62} a higher consumption of high-sugar beverages and foods is associated with evidence of increased inflammation and oxidative stress. Few studies have assessed the effects of long-term sugar consumption on inflammatory and oxidative stress markers.

Dietary Sugars and Obesity

Recent studies report a significant increase of energy intake with increased sugar-sweetened beverage consumption among children, adolescents, and adults^{63–65}; however, evidence is inconsistent regarding the positive association between sugar-sweetened beverage consumption and obesity.⁶⁶ Because overweight and obesity are complex metabolic conditions, it is unlikely that a single food or food group is primarily causal. Many epidemiological studies, including those with cross-sectional and prospective study designs, have shown a positive relationship between higher intake of sweetened beverages and risk of overweight or obesity^{63,64,67}; however, other studies have shown evidence against this hypothesis.^{65,66}

Consumption of sugar-sweetened beverages ingested with meals has doubled.⁶⁸ In a feeding experiment, increasing the size of sugar-sweetened beverages increased total energy intake from solid food.⁶⁹ When the size of a regular cola was increased from 12 to 18 ounces, energy intake from food increased by 10% in women and by 26% in men.⁷⁰ All things being equal, a small, persistent energy imbalance of 50 calories per day could result in up to a 5-pound weight gain over the course of 1 year.⁷¹

Randomized clinical feeding trials have shown inconsistent results from testing the effects of sugar-sweetened beverages on weight gain.^{33,63,64} In a recently published intervention study to reduce sugar-sweetened beverage consumption among adolescents, there was no significant change in body mass index between intervention and control groups.³³ However, in subgroup analysis, there was a net change in body mass index between intervention and control groups of -0.75 (0.34) kg/m^2 ($P=0.03$) in adolescents who had a body mass index >25.6 kg/m^2 at baseline. Differences in study design, population studied, and study instruments and methods may have contributed to these inconsistent findings.^{63,64} Because *added sugar* was recently included as a *nutrient* or *food compound* in the US Department of Agriculture nutrient database, the number of studies examining added sugar as an exposure will most likely increase in the future.

A recent meta-analysis examined 88 cross-sectional and prospective studies exploring the relationship between soft drink intake and nutrition or health outcomes.²⁵ Higher intake of soft drinks was associated with greater energy intake, higher body weight, lower intake of other nutrients, and worse health indices. Subsequent analyses from a large trial confirmed these findings, namely, greater weight loss as sugar-sweetened beverage intake decreased.⁷²

The Hedonic Pathway of Food Reward

The ventral tegmental area and nucleus accumbens, also referred to as the pleasure center of the brain, are the limbic structures central to the hedonic pathway that motivate the reward of food intake. The palatability of available food can undermine normal satiety signals, motivating energy intake independent of energy need.^{73,74} Sucrose infusion directly into the nucleus accumbens alters dopamine and opioid neurotransmission, increasing food intake.⁷⁵ Both sweet and high-fat foods mobilize opioids and dopamine within the nucleus accumbens, establishing hard-wired pathways for craving in these areas.^{76,77} Chronic hyperinsulinemia may also contribute to increased caloric intake by preventing dopamine clearance from the nucleus accumbens, thus fostering pleasure derived from food in situations in which energy stores are replete, contributing to excess energy intake.⁷⁸ Obesity results in a decreased density of striatal D₂ receptors,⁷⁹ which may lead to a compensatory increased strata dopamine neurotransmission through mass action.⁸⁰ Chronic amygdala activation by stress, which increases cortisol secretion, promotes palatable food consumption as a form of self-medication.^{81–83} Several studies in children have observed relationships between stress and increased intake of sugared beverages, sweets, and snacking.^{84,85} In a controlled study of 9-year-olds, children who were both high on dietary restraint and felt more stressed by laboratory challenges tended to eat more sugar-containing comfort food.⁸⁶

Effects of Liquid Versus Solid Food Form

The form of dietary intake (solid versus fluid) is related to energy balance.⁸⁷ Over the past several decades, energy intake has increased with a concomitant change in the composition of the US diet, including increasing energy intake from beverages.⁸⁸ Energy intake from beverages more than doubled between 1965 and 2001 (11.8% of 1993 kcal in 1965 versus 21.0% of 2185 kcal in 2002) according to national survey data.⁸⁹ Weight gain may occur with greater caloric intake from fluids than from solid foods because of the weak satiety signals evoked from energy-containing beverages; therefore, total energy intake may be greater with fluid calorie intake than with calorie consumption from solid foods.^{87,90,91}

Supportive evidence comes from short-term feeding studies and a recent large epidemiological study. In a crossover study, 7 males and 8 females, mean age 22 years, consumed 450 kcal daily of a liquid (regular soft drink) or solid (jelly beans) during two 4-week periods separated by a 4-week washout period.⁹¹ Total daily energy intake increased by 17% on the liquid treatment compared with the solid treatment, which resulted in increased body weight during the liquid period only. In another crossover study, the effect of food form on appetite and energy intake was tested in 20 lean and 20 obese adults.⁸⁷ Study participants consumed both a solid food and a beverage with a specified energy intake at lunch according to body mass (125-kcal load for normal weight and 225-kcal load for obese adults). Although there was no difference in energy intake between treatment groups, more energy was consumed after lunch with the beverage than with the solid food (1950 versus 1585 kcal, $P=0.03$), respectively.

Both lean and obese adults responded similarly to beverage and solid food intake; the liquid form resulted in greater energy intake than the solid form of food. Subsequently, in observational analyses from an 18-month trial, a reduction in liquid calorie intake had a stronger effect on weight loss than a reduction in calories from solid food.⁷²

Thus, an increase in calorie consumption from energy-containing beverages is associated with greater energy intake and may be contributing to the increase in overweight and obesity in the US population. Further research concerning these issues is needed.

Energy Density

Short-term, laboratory-based studies repeatedly have documented that individuals consume fewer calories when presented with lower-energy-density foods than with similar higher-energy-density foods.^{92–98} Lower energy intakes have also been associated with lower-energy-density diets in cross-sectional studies.^{99–102} In a recent prospective analysis from a completed trial, reductions in energy density were associated with both weight loss and improved diet quality.¹⁰³ In aggregate, these data support reductions in energy density as a means to lower energy intake and control weight. A reduction in added sugars is one means to achieve a reduction in energy density.

Nutrient Adequacy

The effects of excess intake of sugars on nutrient adequacy are of concern. The association between added sugars and micronutrient intakes were examined with data from NHANES III. Although the trends were not consistent for all age groups, reduced intakes of calcium, vitamin A, iron, and zinc were observed with increasing intake of added sugars, particularly at intake levels that exceeded 25% of energy. Largely on the basis of these data, the Dietary Reference Intakes report on macronutrients suggests that no more than 25% of energy should be consumed as added sugars¹⁰⁴; however, as discussed below, these recommendations were not consistently supported by subsequent literature. More importantly, the Dietary Reference Intakes approach to nutrient adequacy, based on the intake of selected individual nutrients, was subsequently superseded by an alternative construct, discretionary calories, which considers recommendations for all nutrients in the context of energy balance.

A subsequent review, published after the Dietary Reference Intakes report, concluded that there was some evidence that diets that contain a high proportion of added sugars are slightly lower in micronutrients than diets that contain a moderate proportion of added sugars.¹⁰⁵ Another review found that studies examining the percentage of people who achieve the recommended daily allowance for micronutrients across categories of sugar intake often found nonlinear relationships, such that higher levels of intake were observed in the moderate categories of added sugar intake than in the low and high categories of intakes. These authors concluded that there was no clear evidence of micronutrient dilution or of a threshold at which a quantitative amount of added sugars intake could be set.¹⁰⁶

Table 5. Calorie Allowances for Discretionary and Added Sugars Based on a Variety of Age, Sex, and Physical Activity Levels

	Male	Male	Female	Female
Age, y	21–25	46–50	51–55	71–75
Physical activity level*	Active	Sedentary	Moderately active	Sedentary
Energy needs, kcal†	3000	2200	1800	1600
Discretionary calories, kcal‡	512	290	195	132
Added sugars, teaspoons§	18	9	5	3
Added sugars, kcal	288	144	80	48

*Sedentary indicates a lifestyle that includes only the physical activity of independent living; moderately active, a lifestyle that includes physical activity equivalent to walking approximately 1.5 to 3 miles per day at 3 to 4 miles per hour, in addition to the activities of independent living; and active, a lifestyle that includes physical activity equivalent to walking >3 miles per day at 3 to 4 miles per hour, in addition to the activities of independent living.

†Energy needs to maintain current weight; this will not promote weight loss in overweight/obese people.

‡Recommended limit for discretionary calories per the 2005 US Dietary Guidelines.

§Recommended limit for added sugars per the 2005 US Dietary Guidelines.

Data derived from US Department of Agriculture^{10,11} and Britten et al.¹²

The form in which added sugars are consumed appears to be an important modifier of the impact of dilution.¹⁰⁵ Soft drinks, sugar, and sweets are more likely to have a negative impact on diet quality, whereas dairy foods, milk drinks, and presweetened cereals may have a positive impact.⁵ Of particular concern is the relationship of the intake of sugars with fiber intake. Intake of sugars is inversely associated with fiber intake; a majority of intervention studies testing the efficacy of the influence of fiber on weight regulation have shown increased fiber intake to be associated with decreasing energy intake, which translates into weight loss.¹⁰⁷

Discretionary Calories

In 2005, the concept of discretionary calories was introduced to help people meet all of their nutrient requirements while avoiding excess total energy intake.^{10,11} A person’s discretionary calorie allowance can be determined by estimating the calories needed to meet nutrient requirements and then subtracting this amount from the estimated energy requirement needed to maintain weight. The remaining calories are the discretionary calorie allowance.¹⁰⁸ Intakes of added sugars, solid fats, and alcohol are included within the allowance for discretionary calories, which proportionally increases as the total energy allowance increases. For example, an 1800-calorie pattern for a moderately active 51- to 55-year-old woman would have a 195-calorie discretionary allowance, whereas the allowance would be 512 calories for a physically active 21- to 25-year-old male whose total energy allotment was 3000 calories (Table 5). These calculations provide consumers with an energy intake recommendation for weight maintenance based on their sex, height, current weight, and physical activity level. If a lower energy intake is needed to achieve a healthy weight (which is the case for the majority of Americans, two thirds of whom are overweight or obese), then the suggested amount of discretionary calories will be lower.

A high intake of added sugars contributes to overconsumption of discretionary calories by Americans. On the basis of data from the 2001 to 2002 NHANES, the average percentage of discretionary calories that different age and gender groups consume is much higher than recommended, ranging between 30% and 42% of total energy intake.¹⁰⁸ Concern about Americans’ intake of added sugars has heightened because

of recommendations that a reduced intake of added sugars (especially sugar-sweetened beverages) may be helpful in achieving the recommended intakes of nutrients and in weight control.¹⁰⁹ In 2003, the World Health Organization stated that excessive consumption of energy-rich foods can encourage weight gain and subsequently recommended limiting the consumption of added sugars to <10% of total energy intake.¹¹⁰

Summary and Recommendations

Over the past 30 years, total calorie intake has increased by an average of 150 to 300 calories per day, and approximately 50% of this increase comes from liquid calories (primarily sugar-sweetened beverages).^{111,112} At the same time, there has been no apparent change in physical activity.¹¹³ Hence, it is likely that weight gain over the same period must be related in part to increased intake of added sugars, even though research tools thus far have been insufficient to confirm a direct link. This likely results both from the fact that obesity is a multifactorial condition and because it is extremely difficult to identify, much less quantify, the relative contributions of each factor in epidemiological studies. Given these considerations, it is prudent to advocate a multifaceted approach, one component of which is reducing energy intake from added sugars.

The AHA’s “Diet and Lifestyle Recommendations Revision 2006” recommended minimizing the intake of beverages and food with added sugars.² The present statement expands on that recommendation by proposing a specific upper limit of intake for added sugars. A prudent upper limit of intake is half of the discretionary calorie allowance that can be accommodated within the appropriate energy intake level needed for a person to achieve or maintain a healthy weight based on the US Department of Agriculture food intake patterns (Table 3). In Table 3, discretionary calories are approximately equally divided between solid fats and added sugars. If a person chooses to consume alcohol as well, then intake of solid fats and added sugars should be further reduced to accommodate the additional calories from alcohol. Depending on the calorie level, recommendations for added sugars vary from 5 teaspoons per day (or 80 calories) for a daily energy expenditure of 1800 calories for an average adult

woman and 9 teaspoons per day (or 144 calories) for a daily energy expenditure of 2200 calories for an average adult man. For reference, one 12-ounce can of cola contains ≈8 teaspoons of added sugar, for ≈130 calories.¹¹⁴

In conclusion, to achieve and maintain healthy weights and decrease cardiovascular risk while at the same time meeting essential nutrient needs, the AHA encourages people to consume an overall healthy diet that is consistent with the AHA’s 2006

diet and lifestyle recommendations.² Most American women should eat or drink no more than 100 calories per day from added sugars, and most American men should eat or drink no more than 150 calories per day from added sugars.

AQ: 16

Acknowledgment

The authors thank Bethany Yon, MS, for her assistance with the preparation of this statement.

Disclosures

Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/Advisory Board	Other
Rachel K. Johnson	University of Vermont	USDA Hatch*	None	None	None	None	Dairy Management Inc–National Dairy Council*; International Dairy Foods Association*	None
Lawrence J. Appel	Johns Hopkins	NHLBI†	None	None	None	None	None	None
Michael Brands	Medical College of Georgia	NIH†	None	None	None	None	None	None
Barbara V. Howard	MedStar Research Institute	None	None	Schering-Plough*	None	None	Egg Nutrition Council*; Merck*	None
Michael Lefevre	Utah State University	None	None	None	None	None	Hershey*; Kraft†; Mars†	None
Robert H. Lustig	University of California, San Francisco	None	None	Canadian Pediatric Endocrine Group*; Children's Hospital of Orange County*; Columbia University*; Endocrine Society Science Writers Conference*	None	None	None	None
Frank Sacks	Brigham & Women's Hospital/Harvard University	NIH†	None	None	None	None	None	None
Lyn M. Steffen	University of Minnesota, School of Public Health	None	None	None	None	None	None	None
Judith Wylie-Rosett	Albert Einstein College of Medicine	AHA; Dr Robert C. and Veronica Atkins Foundation†; NIDDK†; NHLBI†	None	Unilever*, American Diabetes Association*	None	None	New York City Department of Health*; D'Life*; Veterans Administration*; Mt. Sinai School of Medicine*; SencisCorp*	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be “significant” if (1) the person receives \$10 000 or more during any 12-month period, or 5% or more of the person’s gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns \$10 000 or more of the fair market value of the entity. A relationship is considered to be “modest” if it is less than “significant” under the preceding definition.

*Modest.
†Significant.

Reviewer Disclosures

Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/Advisory Board	Other
Penny M. Kris-Etherton	Pennsylvania State University	None	None	None	None	None	None	None
Dariusz Mozaffarian	Harvard Medical School and School of Public Health	None	None	None	None	None	None	None
Susan Roberts	Tufts University	None	None	None	None	None	None	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be “significant” if (1) the person receives \$10 000 or more during any 12-month period, or 5% or more of the person’s gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns \$10 000 or more of the fair market value of the entity. A relationship is considered to be “modest” if it is less than “significant” under the preceding definition.

References

1. Howard BV, Wylie-Rosett J. Sugar and cardiovascular disease: a statement for healthcare professionals from the Committee on Nutrition of the Council on Nutrition, Physical Activity, and Metabolism of the American Heart Association [published correction appears in *Circulation*. 2003;107:2166]. *Circulation*. 2002;106:523–527.
2. Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, Franch HA, Franklin B, Kris-Etherton P, Harris WS, Howard B, Karanja N, Lefevre M, Rudel L, Sacks F, Van Horn L, Winston M, Wylie-Rosett J. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee [published corrections appear in *Circulation*. 2006;114:e629 and 2006;114:e27]. *Circulation*. 2006;114:82–96.
3. Murphy SP, Johnson RK. The scientific basis of recent US guidance on sugars intake. *Am J Clin Nutr*. 2003;78(suppl):827S–833S.
4. Johnson RK, Frary C, Wang MQ. The nutritional consequences of flavored-milk consumption by school-aged children and adolescents in the United States. *J Am Diet Assoc*. 2002;102:853–856.
5. Frary CD, Johnson RK, Wang MQ. Children and adolescent's choices of food and beverages high in added sugars are associated with intakes of key nutrients and food groups. *J Adolesc Health*. 2004;34:56–63.
6. Murphy MM, Douglass JS, Johnson RK, Spence LA. Drinking flavored or plain milk is positively associated with nutrient intake and is not associated with adverse effects on weight status in US children and adolescents. *J Am Diet Assoc*. 2008;108:631–639.
7. Wells HF, Buzby JC. Dietary assessment of major trends in US food consumption, 1970–2005. Economic Research Service, US Department of Agriculture; March 2008. *Economic Information Bulletin No. 33*. Available at: <http://www.ers.usda.gov/Publications/EIB33>. Accessed January 4, 2009.
8. Bray GA, Nielsen SJ, Popkin BM. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity [published correction appears in *Am J Clin Nutr*. 2004;80:1090]. *Am J Clin Nutr*. 2004;79:537–543.
9. National Cancer Institute. Usual intake of added sugars. In: Usual Dietary Intakes: Food Intakes, US Population 2001–04. November 2008. Available at: <http://riskfactor.cancer.gov/diet/usualintakes/pop/t35.html>. Accessed January 4, 2009.
10. US Department of Health and Human Services and US Department of Agriculture. *Dietary Guidelines for Americans, 2005*. 6th ed. Washington, DC: US Government Printing Office; January 2005.
11. US Department of Agriculture. MyPyramid.gov (USDA food guidance system Web site). 2005. Available at: <http://www.mypyramid.gov>. Accessed January 4, 2009.
12. Britten P, Marcoe K, Yamini S, Davis C. Development of food intake patterns for the MyPyramid Food Guidance System. *J Nutr Educ Behav*. 2006;38:S78–S92.
13. Guthrie JF, Morton JF. Food sources of added sweeteners in the diets of Americans. *J Am Diet Assoc*. 2000;100:43–51.
14. Frazao E, Allshouse J. Strategies for intervention: commentary and debate. *J Nutr*. 2003;133:844S–847S.
15. Nutrient Data Laboratory, Beltsville Human Nutrition Research Center, Agricultural Research Service, US Department of Agriculture. USDA database for the added sugars content of selected foods. February 2006. Available at: http://www.nal.usda.gov/fnic/foodcomp/Data/add_sug/addsug01.pdf. Accessed January 4, 2009.
16. Smart Choices Program: Guiding Food Choices. Smart Choices Program Web site. Available at: <http://smartchoicesprogram.com/>. Accessed January 4, 2009.
17. Guiding Stars program. Hannaford Brothers Co Web site. Available at: http://www.hannaford.com/Contents/Healthy_Living/Guiding_Stars/index.shtml. Accessed January 4, 2009.
18. NuVal nutritional scoring system. Available at: <http://www.nuval.com>. Accessed January 4, 2009.
19. Drewnowski A. Concept of a nutritious food: toward a nutrient density score. *Am J Clin Nutr*. 2005;82:721–732.
20. Lê KA, Tappy L. Metabolic effects of fructose. *Curr Opin Clin Nutr Metab Care*. 2006;9:469–475.
21. Rutledge AC, Adeli K. Fructose and the metabolic syndrome: pathophysiology and molecular mechanisms. *Nutr Rev*. 2007;65(part 2):S13–S23.
22. Johnson RJ, Segal MS, Sautin Y, Nakagawa T, Feig DI, Kang DH, Gersch MS, Benner S, Sánchez-Lozada LG. Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. *Am J Clin Nutr*. 2007;86:899–906.
23. Takasaki Y. Studies on sugar isomerizing enzyme: production and utilization of glucose isomerase from *Streptomyces* spp. *Agric Biol Chem*. 1966;30:1247–1253.
24. Coulston AM, Johnson RK. Sugar and sugars: myths and realities. *J Am Diet Assoc*. 2002;102:351–353.
25. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health*. 2007;97:667–675.
26. Tordoff MG, Alleva AM. Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. *Am J Clin Nutr*. 1990;51:963–969.
27. Havel PJ. Dietary fructose: implications for dysregulation of energy homeostasis and lipid/carbohydrate metabolism. *Nutr Rev*. 2005;63:133–157.
28. Gross LS, Li S, Ford ES, Liu S. Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the United States: an ecologic assessment. *Am J Clin Nutr*. 2004;79:774–779.
29. Elliott SS, Keim NL, Stern JS, Teff K, Havel PJ. Fructose, weight gain, and the insulin resistance syndrome. *Am J Clin Nutr*. 2002;76:911–922.
30. Dhingra R, Sullivan L, Jacques PF, Wang TJ, Fox CS, Meigs JB, D'Agostino RB, Gaziano JM, Vasan RS. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community [published correction appears in *Circulation*. 2007;116:e557]. *Circulation*. 2007;116:480–488.
31. Stanhope KL, Schwarz JM, Keim NL, Griffen SC, Bremer AA, Graham JL, Hatcher B, Cox CL, Dyachenko A, Zhang W, McGahan JP, Seibert A, Krauss RM, Chiu S, Schaefer EJ, Ai M, Otokozawa S, Nakajima K, Nakano T, Beysen C, Hellerstein MK, Berglund L, Havel PJ. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. *J Clin Invest*. 2009;119:1322–1334.
32. James J, Thomas P, Cavan D, Kerr D. Preventing childhood obesity by reducing consumption of carbonated drinks: cluster randomised controlled trial [published correction appears in *BMJ*. 2004;328:1236]. *BMJ*. 2004;328:1237.
33. Ebbeling CB, Feldman HA, Osganian SK, Chomitz VR, Ellenbogen SJ, Ludwig DS. Effects of decreasing sugar-sweetened beverage consumption on body weight in adolescents: a randomized, controlled pilot study. *Pediatrics*. 2006;117:673–680.
34. McDonald RB. Influence of dietary sucrose on biological aging. *Am J Clin Nutr*. 1995;62(suppl):284S–293S.
35. Chen M, Halter JB, Porte D Jr. The role of dietary carbohydrate in the decreased glucose tolerance of the elderly. *J Am Geriatr Soc*. 1987;35:417–424.
36. Moghaddam E, Vogt JA, Wolever TM. The effects of fat and protein on glycemic responses in nondiabetic humans vary with waist circumference, fasting plasma insulin, and dietary fiber intake [published correction appears in *J Nutr*. 2006;136:3084]. *J Nutr*. 2006;136:2506–2511.
37. Groff JL, Gropper SS, Hunt SM. *Advanced Nutrition and Human Metabolism*. 2nd ed. Minneapolis/St Paul, Minn: West Publishing; 1995.
38. Wolever TM, Miller JB. Sugars and blood glucose control. *Am J Clin Nutr*. 1995;62(suppl):212S–227S.
39. Oettlé GJ, Emmett PM, Heaton KW. Glucose and insulin responses to manufactured and whole-food snacks. *Am J Clin Nutr*. 1987;45:86–91.
40. Weickert MO, Pfeiffer AF. Metabolic effects of dietary fiber consumption and prevention of diabetes. *J Nutr*. 2008;138:439–442.
41. Feig DI, Soletsky B, Johnson RJ. Effect of allopurinol on blood pressure of adolescents with newly diagnosed essential hypertension: a randomized trial. *JAMA*. 2008;300:924–932.
42. Nguyen S, Choi HK, Lustig RH, Hsu CY. Sugar-sweetened beverages, serum uric acid, and blood pressure in adolescents. *J Pediatr*. 2009;154:807–813.
43. Bremer AA, Auinger P, Byrd RS. Relationship between insulin resistance-associated metabolic parameters and anthropometric measurements with sugar-sweetened beverage intake and physical activity levels in US adolescents: findings from the 1999–2004 National Health and Nutrition Examination Survey. *Arch Pediatr Adolesc Med*. 2009;163:328–335.
44. Van der Schaaf MR, Koomans HA, Joles JA. Dietary sucrose does not increase twenty-four-hour ambulatory blood pressure in patients with

- either essential hypertension or polycystic kidney disease. *J Hypertens*. 1999;17:453–454.
45. Hallfrisch J, Reiser S, Prather ES. Blood lipid distribution of hyperinsulinemic men consuming three levels of fructose. *Am J Clin Nutr*. 1983;37:740–748.
 46. Hellerstein MK. Carbohydrate-induced hypertriglyceridemia: modifying factors and implications for cardiovascular risk. *Curr Opin Lipidol*. 2002;13:33–40.
 47. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am J Clin Nutr*. 2003;77:1146–1155.
 48. Appel LJ, Sacks FM, Carey VJ, Obarzanek E, Swain JF, Miller ER III, Conlin PR, Erlinger TP, Rosner BA, Laranjo NM, Charleston J, McCarron P, Bishop LM; for the OmniHeart Collaborative Research Group. Effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. *JAMA*. 2005;294:2455–2464.
 49. Obarzanek E, Sacks FM, Vollmer WM, Bray GA, Miller ER III, Lin PH, Karanja NM, Most-Windhauser MM, Moore TJ, Swain JF, Bales CW, Proschan MA; DASH Research Group. Effects on blood lipids of a blood pressure-lowering diet: the Dietary Approaches to Stop Hypertension (DASH) Trial. *Am J Clin Nutr*. 2001;74:80–89.
 50. Howard BV, Van Horn L, Hsia J, Manson JE, Stefanick ML, Wassertheil-Smolter S, Kuller LH, LaCroix AZ, Langer RD, Lasser NL, Lewis CE, Limacher MC, Margolis KL, Mysiw WJ, Ockene JK, Parker LM, Perri MG, Phillips L, Prentice RL, Robbins J, Rossouw JE, Sarto GE, Schatz IJ, Snetselaar LG, Stevens VJ, Tinker LF, Trevisan M, Vitolins MZ, Anderson GL, Assaf AR, Bassford T, Beresford SA, Black HR, Brunner RL, Brzyski RG, Caan B, Chlebowski RT, Gass M, Granek I, Greenland P, Hays J, Heber D, Heiss G, Hendrix SL, Hubbell FA, Johnson KC, Kotchen JM. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA*. 2006;295:655–666.
 51. Fried SK, Rao SP. Sugars, hypertriglyceridemia, and cardiovascular disease. *Am J Clin Nutr*. 2003;78:873S–880S.
 52. Bantle JP, Raatz SK, Thomas W, Georgopoulos A. Effects of dietary fructose of plasma lipids in healthy subjects. *Am J Clin Nutr*. 2000;72:1128–1134.
 53. Chong MF, Fielding BA, Frayn KN. Mechanisms for the acute effect of fructose of postprandial lipemia. *Am J Clin Nutr*. 2007;85:1511–1520.
 54. Teff KL, Elliott SS, Tschöp M, Kieffer TJ, Rader D, Heiman M, Townsend RR, Keim NL, D'Alessio D, Havel PJ. Dietary fructose reduces circulating insulin and leptin, attenuates postprandial suppression of ghrelin, and increases triglycerides in women. *J Clin Endocrinol Metab*. 2004;89:2963–2972.
 55. Parks EJ, Skokan LE, Timlin MT, Dingfelder CS. Dietary sugars stimulate fatty acid synthesis in adults. *J Nutr*. 2008;138:1039–1046.
 56. Black RN, Spence M, McMahon RO, Cuskelly GJ, Ennis CN, McCance DR, Young IS, Bell PM, Hunter SJ. Effect of eucaloric high- and low-sucrose diets with identical macronutrient profile on insulin resistance and vascular risk: a randomized controlled trial. *Diabetes*. 2006;55:3566–3572.
 57. Marckmann P, Raben A, Astrup A. Ad libitum intake of low-fat diets rich in either starchy foods or sucrose: effects on blood lipids, factor VII coagulant activity, and fibrinogen. *Metabolism*. 2000;49:731–735.
 58. Liu S, Manson JE, Buring JE, Stampfer MJ, Willett WC, Ridker PM. Relation between a diet with a high glycemic load and plasma concentrations of high-sensitivity C-reactive protein in middle-aged women. *Am J Clin Nutr*. 2002;75:492–498.
 59. Price KD, Price CS, Reynolds RD. Hyperglycemia-induced ascorbic acid deficiency promotes endothelial dysfunction and the development of atherosclerosis. *Atherosclerosis*. 2001;158:1–12.
 60. Scribner KB, Pawlak DB, Ludwig DS. Hepatic steatosis and increased adiposity in mice consuming rapidly vs. slowly absorbed carbohydrate. *Obesity (Silver Spring)*. 2007;15:2190–2199.
 61. Ceriello A, Bortolotti N, Crescentini A, Motz E, Lizzio S, Russo A, Ézsol Z, Tonutti L, Taboga C. Antioxidant defences are reduced during the oral glucose tolerance test in normal and non-insulin-dependent diabetic subjects. *Eur J Clin Invest*. 1998;28:329–333.
 62. Ma SW, Tomlinson B, Benzie IF. A study of the effect of oral glucose loading on plasma oxidant:antioxidant balance in normal subjects. *Eur J Nutr*. 2005;44:250–254.
 63. Bachman CM, Baranowski T, Nicklas TA. Is there an association between sweetened beverages and adiposity? *Nutr Rev*. 2006;64:153–174.
 64. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr*. 2006;84:274–288.
 65. Johnson L, Mander AP, Jones LR, Emmett PM, Jebb SA. Is sugar-sweetened beverage consumption associated with increased fatness in children? *Nutrition*. 2007;23:557–563.
 66. Forshee RA, Anderson PA, Storey ML. Sugar-sweetened beverages and body mass index in children and adolescents: a meta-analysis [published correction appears in *Am J Clin Nutr*. 2009;89:441–442]. *Am J Clin Nutr*. 2008;87:1662–1671.
 67. Palmer JR, Boggs DA, Krishnan S, Hu FB, Singer M, Rosenberg L. Sugar-sweetened beverages and incidence of type 2 diabetes mellitus in African American women. *Arch Intern Med*. 2008;165:1487–1492.
 68. Nielsen SJ, Popkin BM. Changes in beverage intake between 1977 and 2001 [published correction appears in *Am J Prev Med*. 2005;28:413]. *Am J Prev Med*. 2004;27:205–210.
 69. Rolls BJ, Roe LS, Meengs JS. Larger portion sizes lead to a sustained increase in energy intake over 2 days. *J Am Diet Assoc*. 2006;106:543–549.
 70. Flood JE, Roe LS, Rolls BJ. The effect of increased beverage portion size on energy intake at a meal. *J Am Diet Assoc*. 2006;106:1984–1990.
 71. Kumanyika SK, Obarzanek E, Stettler N, Bell R, Field AE, Fortmann SP, Franklin BA, Gillman MW, Lewis CE, Poston WC 2nd, Stevens J, Hong Y. Population-based prevention of obesity: the need for comprehensive promotion of healthful eating, physical activity, and energy balance: a scientific statement from American Heart Association Council on Epidemiology and Prevention, Interdisciplinary Committee for Prevention (formerly the Expert Panel on Population and Prevention Science). *Circulation*. 2008;118:428–464.
 72. Chen L, Appel LJ, Loria C, Lin PH, Champagne CM, Elmer PJ, Ard JD, Mitchell D, Batch BC, Svetkey LP, Caballero B. Reduction in consumption of sugar-sweetened beverages is associated with weight loss: the PREMIER trial. *Am J Clin Nutr*. 2009;89:1299–1306.
 73. Erlanson-Albertsson C. How palatable food disrupts appetite regulation. *Basic Clin Pharmacol Toxicol*. 2005;97:61–73.
 74. Pelchat ML. Of human bondage: food craving, obsession, compulsion, and addiction. *Physiol Behav*. 2002;76:347–352.
 75. Spangler R, Wittkowski KM, Goddard NL, Avena NM, Hoebel BG, Leibowitz SF. Opiate-like effects of sugar on gene expression in reward areas of the rat brain. *Mol Brain Res*. 2004;124:134–142.
 76. Kelley AE, Bakshi VP, Haber SN, Steininger TL, Will MJ, Zhang M. Opioid modulation of taste hedonics within the ventral striatum. *Physiol Behav*. 2002;76:365–377.
 77. Pelchat ML, Johnson A, Chan R, Valdez J, Ragland JD. Images of desire: food-craving activation during fMRI. *Neuroimage*. 2004;23:1486–1493.
 78. Anderzhanova E, Covasa M, Hajnal A. Altered basal and stimulated accumbens dopamine release in obese OLETF rats as a function of age and diabetic status. *Am J Physiol Regul Integr Comp Physiol*. 2007;293:R603–R611.
 79. Wang GJ, Volkow ND, Logan J, Pappas NR, Wong CT, Zhu W, Netusil N, Fowler JS. Brain dopamine and obesity. *Lancet*. 2001;357:354–357.
 80. Stice E, Spoor S, Bohon C, Small DM. Relation between obesity and blunted striatal response to food is moderated by TaqIA A1 allele. *Science*. 2008;322:449–452.
 81. Dallman MF, Pecoraro N, Akana SF, La Fleur SE, Gomez F, Houshyar H, Bell ME, Bhatnagar S, Laugero KD, Manalo S. Chronic stress and obesity: a new view of “comfort food.” *Proc Natl Acad Sci U S A*. 2003;100:11696–11701.
 82. Mietus-Snyder ML, Lustig RH. Childhood obesity: adrift in the “limbic triangle.” *Annu Rev Med*. 2008;59:147–162.
 83. Pecoraro N, Reyes F, Gomez F, Bhargava A, Dallman MF. Chronic stress promotes palatable feeding, which reduces signs of stress: feed-forward and feedback effects of chronic stress. *Endocrinology*. 2004;145:3754–3762.
 84. Nguyen-Michel ST, Unger JB, Spruijt-Metz D. Dietary correlates of emotional eating in adolescence. *Appetite*. 2007;49:494–499.
 85. Oliver G, Wardle J. Perceived effects of stress on food choice. *Physiol Behav*. 1999;66:511–515.
 86. Roemmich JN, Wright SM, Epstein LH. Dietary restraint and stress-induced snacking in youth. *Obes Res*. 2002;10:1120–1126.

87. Mourao DM, Bressan J, Campbell WW, Mattes RD. Effects of food form on appetite and energy intake in lean and obese young adults. *Int J Obes (Lond)*. 2007;31:1688–1695.
88. Nielsen SJ, Popkin BM. Patterns and trends in food portion sizes, 1977–1998. *JAMA*. 2003;289:450–453.
89. Duffey KJ, Popkin BM. Shifts in patterns and consumption of beverages between 1965 and 2002. *Obesity (Silver Spring)*. 2007;15:2739–2747.
90. Mattes R. Fluid calories and energy balance: the good, the bad, and the uncertain. *Physiol Behav*. 2006;89:66–70.
91. DiMeglio DP, Mattes RD. Liquid versus solid carbohydrate: effects on food intake and body weight. *Int J Obesity*. 2000;24:794–800.
92. Bell EA, Castellanos VH, Pelkman CL, Thorwart ML, Rolls BJ. Energy density of foods affects energy intake in normal-weight women. *Am J Clin Nutr*. 1998;67:412–420.
93. Rolls BJ, Bell EA, Castellanos VH, Chow M, Pelkman CL, Thorwart ML. Energy density but not fat content of foods affected energy intake in lean and obese women. *Am J Clin Nutr*. 1999;69:863–871.
94. Bell EA, Rolls BJ. Energy density of foods affects energy intake across multiple levels of fat content in lean and obese women. *Am J Clin Nutr*. 2001;73:1010–1018.
95. Devitt AA, Mattes RD. Effects of food unit size and energy density on intake in humans. *Appetite*. 2004;42:213–220.
96. Stubbs RJ, Whybrow S. Energy density, diet composition and palatability: influences on overall food energy intake in humans. *Physiol Behav*. 2004;81:755–764.
97. Rolls BJ, Roe LS, Meengs JS. Salad and satiety: energy density and portion size of a first-course salad affect energy intake at lunch. *J Am Diet Assoc*. 2004;104:1570–1576.
98. Rolls BJ, Roe LS, Meengs JS. Reductions in portion size and energy density of foods are additive and lead to sustained decreases in energy intake. *Am J Clin Nutr*. 2006;83:11–17.
99. Cucó G, Arija V, Martí-Henneberg C, Fernández-Ballart J. Food and nutritional profile of high energy density consumers in an adult Mediterranean population. *Eur J Clin Nutr*. 2001;55:192–199.
100. de Castro JM. Dietary energy density is associated with increased intake in free-living humans. *J Nutr*. 2004;134:335–341.
101. Stookey JD. Energy density, energy intake and weight status in a large free-living sample of Chinese adults: exploring the underlying roles of fat, protein, carbohydrate, fiber and water intakes. *Eur J Clin Nutr*. 2001;55:349–359.
102. Kant AK, Graubard BI. Energy density of diets reported by American adults: association with food group intake, nutrient intake, and body weight. *Int J Obes (Lond)*. 2005;29:950–956.
103. Ledikwe JH, Rolls BJ, Smiciklas-Wright H, Mitchell DC, Ard JD, Champagne C, Karanja N, Lin PH, Stevens VJ, Appel LJ. Reductions in dietary energy density are associated with weight loss in overweight and obese participants in the PREMIER trial. *Am J Clin Nutr*. 2007;85:1212–1221.
104. Food and Nutrition Board, Institute of Medicine of the National Academies. *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids*. Washington, DC: National Academies Press; 2002.
105. Gibson SA. Dietary sugars intake and micronutrient adequacy: a systematic review of the evidence. *Nutr Res Rev*. 2007;20:121–131.
106. Rennie KL, Livingstone BE. Associations between dietary added sugar intake and micronutrient intake: a systematic review. *Br J Nutr*. 2007;97:832–841.
107. Howarth NC, Saltzman E, Roberts SB. Dietary fiber and weight regulation. *Nutr Rev*. 2001;59:129–139.
108. Basiotis P, Guenther PM, Lina M, Britten P. Americans consume too many calories from solid fat, alcohol and added sugar. Alexandria, Va: USDA, Center for Nutrition Policy and Promotion; June 2006. *Nutrition Insight 33*.
109. US Department of Health and Human Services. *Nutrition and Your Health: Dietary Guidelines for Americans: Part D: Science Base: Section 5: Carbohydrates*. Available at: http://www.health.gov/dietaryguidelines/dga2005/report/HTML/D5_Carbs.htm. Accessed January 4, 2009.
110. Nishida C, Uauy R, Kumanyika S, Shetty P. The joint WHO/FAO expert consultation on diet, nutrition and the prevention of chronic diseases: process, product and policy implications. *Public Health Nutr*. 2004;7:245–250.
111. Briefel RR, Johnson CL. Secular trends in dietary intake in the United States. *Annu Rev Nutr*. 2004;24:401–431.
112. Nielsen SJ, Siega-Riz AM, Popkin BM. Trends in energy intake in U.S. between 1977 and 1996: similar shifts seen across age groups. *Obes Res*. 2002;10:370–378.
113. French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. *Annu Rev Public Health*. 2001;22:309–335.
114. USDA National Nutrient Database. Agricultural Research Service, US Department of Agriculture. Available at: <http://www.nal.usda.gov/fnic/foodcomp/search/>. Accessed January 4, 2009.