

# Sugars and Health Workshop: summary and conclusions<sup>1-4</sup>

David R Lineback and Julie Miller Jones

## INTRODUCTION

Carbohydrates are an important component of diets and food supplies throughout the world. They are involved in physiologic, sensory, textural, and physicochemical roles. Carbohydrates are typically classified into 3 principal groups based on degree of polymerization (DP): sugars (monosaccharides and disaccharides), oligosaccharides (DP: 3–9), and polysaccharides (DP > 10). During discussions of defining and interpreting intakes of sugars, it became readily apparent that 2 major problems existed: 1) a lack of agreement on a common definition of sugars and imprecision when the terms are used in research studies and 2) a limitation in accuracy in estimating actual sugars intake by humans. Because both definition and accuracy are critically important in the consideration of the health effects of sugars consumption, they require attention and clarification.

Physiologic effects of carbohydrates and sugars are defined for research purposes and in some other instances by their effects on blood glucose concentrations. Several different terms are used for this, including glycemic index (GI), glycemic load, and insulinemic index. These are described in the presentations summarized in the following sections. Note that the GI and glycemic load are correctly applied only to carbohydrate foods. In some instances, glycemic load has been used incorrectly with foods that do not contain carbohydrate. For such foods, the glycemic load is zero. Furthermore, sugars are moderate to low in GI and glycemic load. Also, they have a lower GI than do many starch foods. The following sections summarize the discussions held during the workshop.

## DEFINING AND INTERPRETING INTAKES OF SUGARS: PRESENTED BY SIGMAN-GRANT AND MORITA (1)

*Sugar, sugars, added sugars, and caloric sweeteners* are terms used by US government agencies. Different terms are used in other countries, such as *extrinsic* and *intrinsic sugars*, *available* and *unavailable carbohydrate*, and *fermentable carbohydrate*. For this workshop, it was agreed that the term *sugar* would designate sucrose (table sugar); *sugars* would be used for all monosaccharides and disaccharides, including sucrose; and *added sugars* would be defined as by the US Department of Agriculture as “eaten separately or used as ingredients in processed or prepared foods,” with a list of examples including ingredients that may also include oligosaccharides. This lack of precise agreement is regarded as undesirable, unacceptable, and in need of immediate attention; definitions that are commonly accepted by regulators, scientists, manufacturers, dietitians, and other health professionals and consumers are necessary. This need is emphasized by the current discussions on the role of added sugars in the US diet and the health implications, particularly in terms of weight gain and obesity.

Some methods of estimating human consumption of sugars overestimate intake, whereas others underestimate it. Estimation of consumption of sugars usually relies on values obtained from food intake surveys or economic availability data. Because of the tendency for individuals, especially the obese, to underestimate their food consumption, food intake surveys are believed to underestimate actual intake. Foods and beverages that are the major sources of added sugars are known to be differentially underreported, which also contributes to underestimates. Economic availability values for caloric sweeteners are adjusted for losses that occur in the food supply by  $\approx 30\%$ , accounting for the available food supply of sweeteners lost at the retail, consumer, and food-service levels. Some losses of sugars not considered are due to food manufacturing and processing and to uses of these ingredients in pharmaceuticals and alcoholic beverages. This contributes to overestimates of the consumption of sugars.

Another complication is that the amount of sugars ingredients in recipes is calculated from laboratory values rather than by actual determination of the sugars in final food products or foods. This practice may lead to an overestimate of actual consumption. With the analytic methods currently available, the sugars content in food should be analyzed, not calculated. It is not possible to analytically distinguish between naturally occurring and added sugars. This contributes to limitations in databases that rely on recipe calculations for added sugars.

If discussion of the health benefits of sugars intake is not to be based on conjecture leading to continued misunderstandings, the following are needed: 1) commonly agreed on and used definitions of sugars; 2) improved dietary assessment methods for determining sugars intake, with emphasis on reducing underreporting; 3) manufacturing losses and other nonfood and nonalcoholic beverage uses of sugars accounted for to decrease overestimation when using economic availability data; 4) analytic determination of the amounts of sugars in foods rather than data obtained from calculations of ingredients in recipes; and 5) research and literature reviews directed at hypothesized relations between sugars and health to determine biological plausibility and clinical relevance. Research and reviews must precisely define the nature of

<sup>1</sup> From the Joint Institute of Food Safety and Applied Nutrition, University of Maryland, College Park (DRL), and the College of St Catherine, St Paul, MN (MJM).

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<sup>4</sup> Address reprint requests to D Lineback, Joint Institute of Food Safety and Applied Nutrition, University of Maryland, 0220 Symons Hall, College Park, MD 20742. E-mail: [lineback@umd.edu](mailto:lineback@umd.edu).

the sugars to delineate any clinical relations. Evidence-based research is needed to evaluate the totality of evidence about dietary recommendations.

#### SCIENTIFIC BASIS OF RECENT US GUIDANCE ON SUGARS INTAKE: PRESENTED BY MURPHY AND JOHNSON (2)

The findings recently reported by 2 expert panels—the 2000 Dietary Guidelines Advisory Committee and the Institute of Medicine report on dietary reference intakes for macronutrients—were reviewed and the state of the science undergirding these recommendations for sugars was discussed. The guideline pertaining to sugars in the *Dietary Guidelines for Americans* does not mention added sugars, but the supporting text of the current guideline distinguishes between added and naturally occurring sugars. The scientific rationale used in developing the guideline for sugars in the 2000 *Dietary Guidelines for Americans* was discussed during this workshop and the following questions were addressed:

- 1) Should added sugars be distinguished from naturally occurring sugars when offering dietary guidance to the public? Chemically and physiologically, the 2 types of sugars are indistinguishable. There was not sufficient evidence for including the term *added sugars* in the actual guideline, but it was included in the text of the *Dietary Guidelines*. More research was recommended to determine whether there are reasons to distinguish between the 2 types of sugars in the guidelines.
- 2) What health effects can be directly attributed to sugars in the diet? No health effects, other than an association with dental caries, have been conclusively shown.
- 3) Is the intake of added sugars increasing? Food availability data and food intake surveys use different methods and are not necessarily comparable with each other. However, data from both of these sources suggest that consumption of added sugars rose in the United States between the 1980s and the mid-1990s. Food availability data indicate no further increase between 1995 and 2000, but survey data are not available to confirm this trend.
- 4) Could added sugars play a role in the obesity epidemic? There is no direct connection between the intake of added sugars and obesity. Obesity is caused by intake of more energy than is expended. One postulate is that excessive consumption of sugars leads to energy imbalance and a resultant weight gain. However, the data show that high fat and low carbohydrate or low sugar intakes are associated with higher body mass indexes.
- 5) Does sugars intake negatively affect nutrient adequacy? Research emerging since the 2000 Dietary Guidelines Advisory Committee deliberations seems to indicate that very high intakes of foods with added sugars has a displacement effect on the intakes of foods that are important sources of some critical micronutrients (on the basis of a select sample of micronutrients). Diets with very low intakes of added sugars also can be associated with low nutrient content.

The 2002 report from the Institute of Medicine on the dietary reference intakes for macronutrients did not set a tolerable upper intake level for sugars because no specific endpoint of adverse effect was identified. However, a maximal intake level of 25% of energy from added sugars was suggested, based on a decline in

micronutrient intake (especially calcium) associated with very high sugars intakes.

#### HEDONIC ASPECTS OF SUGARS, NEUROREGULATION, AND ENERGY BALANCE: PRESENTED BY LEVINE ET AL (3)

Ingestion of sugars is in part regulated by a variety of brain areas, including those involved in energy and reward. For example, opioid antagonists have very robust effects, decreasing intake of preferred diets (such as high-sucrose diets) when injected into a reward area of the brain but decreasing intake of both preferred and nonpreferred diets when injected into a region of the brain involved in energy regulation. Relations have been shown between the liking of sweet-tasting compounds and drug-taking behavior in rats and nonhuman primates. Sucrose ingestion also affects energy metabolism through the central nervous system, perhaps by some of the neuroregulators that affect sugars intake. Although behavioral and neurochemical data from the animal literature support the notion that sucrose ingestion results in neural changes in brain areas affected by drugs of abuse, one cannot conclude that sugars consumption is addictive.

These are fascinating preliminary results. Many questions have yet to be addressed, for example, Does sucrose stimulate food intake? Does the use of sucrose increase the palatability of fat-containing foods? Does sucrose play a role in overeating? Does sucrose play a role in regulation of body weight?

#### CONSUMPTION OF SUGARS AND THE REGULATION OF SHORT-TERM SATIETY AND FOOD INTAKE: PRESENTED BY ANDERSON AND WOODWARD (4)

The goal of this discussion was to address the short-term (1–2 h) effect of the consumption of sugars on satiety and food intake. The review provides clear evidence that sugars produce satiety and decrease food intake in the short term. Both children and adults reduce food intake at meals eaten shortly after the consumption of sugars, and the reduction is remarkably precise for the energy content of the sugar consumed. The paper also challenges the assumptions that the glycemic response to sugars or other carbohydrates predicts their effect on subsequent food intake. However, the mechanisms involved do not solely result from their effect on blood glucose. Considerable discussion centered on the relevance of the results of short-term studies to longer-term (over the day or several days) responses in food intake and body weight control, a topic that was expanded in the next presentation.

This presentation also led to considerable discussion of the GI of food and its utility in providing dietary guidance for weight control. It was proposed that both high- and low-GI foods affect satiety and food intake, but the significance of these responses to body weight control remains unresolved.

Additional research is clearly needed to resolve questions concerning sugars and appetite control. These include 1) Is there a difference between solid and liquid forms of sugars or high- or low-GI carbohydrates on appetite, satiety, and subsequent selection of calories? Is this a caloric or sweetness response or one mediated physiologically? 2) What is the duration of the satiety response to sugars and how does this compare with the effect of low- and high-GI carbohydrates? 3) How do sugars added to food affect satiety and food intake compared with sugars and other carbohydrates innately present in foods? 4) What is the effect of daily



consumption of drinks containing sugar compared with the chronic consumption of drinks containing low-GI carbohydrate on body weight and energy intake? Are current studies linking weight and sugars-containing foods and beverages simply due to more calories or are other factors involved? 5) Does a glycemic rebound occur after usual servings of sugars in beverages and other foods and does this cause increased hunger? 6) What is the role of satiety signals from the gut in controlling food intake and satiety after sugars compared with other carbohydrates? and 7) Is there a difference in satiety response to consumption of high-fructose corn syrup and sucrose?

### **SUGARS, ENERGY METABOLISM, AND BODY WEIGHT CONTROL: PRESENTED BY SARIS (5)**

The prevalence of overweight and obesity in recent decades is being termed an epidemic and is increasing in the United States and other countries. Health guidelines concerning the prevention of this phenomenon generally stress an increase in physical activity and a reduction of energy intake by lowered consumption of fat and sugars. The dietary guidelines are the subject of debate and discussion. However, the evidence supports the view that a high ratio of fat to carbohydrate in the diet is the primary dietary factor in promoting overconsumption of energy resulting in weight gain.

The lack of long-term dietary intervention trials makes it difficult to assess the effect of different types of carbohydrates. Evidence is not sufficient to conclude that sugars consumption negatively affects weight control unless an overconsumption of energy occurs. However, the frequent use of highly palatable foods, some of which may be high in sugars or GI (not, however, supported by data), could create a potential risk for energy overconsumption and weight gain. Application of the GI of foods or the glycemic load in relation to body weight control requires further investigation. Currently, the available data concerning sucrose intake and metabolism do not support a specific role of sugars as a major dietary player (with fat) in the development of obesity.

Several issues remain to be clarified on the role of sugars compared with other dietary components in the prevention of weight gain. Information is needed from ad libitum, randomized, controlled, long-term dietary intervention studies on the effect of different types of carbohydrates on body weight control. Special attention should be given to the potential confounding effects of other macronutrient changes as well as the type of foods (fluid or solid). Studies are needed on the effects of sugars consumption on physiologic profiles, including satiety and thermogenesis, from the period between 12 h postconsumption up to 48 or 60 h. Information on compensatory effects at 24 or 48 h is of interest to predict long-term weight control. Validation studies on 24–48-h glucose and insulin profiles are needed to use the glycemic load as a surrogate biomarker in epidemiologic studies. A comparison of caloric sweeteners, such as high-fructose corn syrup, with sucrose through the acquisition of data on thermogenic effect, satiety, blood glucose, and insulin is needed to exclude negative effects on body weight control. In relation to health guidelines to increase physical activity, information is needed about the interaction with food intake, in particular the carbohydrate-fat ratio, type of carbohydrate, and role of sugars in this interaction in relation to weight control.

### **SUGARS AND STARCH IN THE NUTRITIONAL MANAGEMENT OF DIABETES MELLITUS: PRESENTED BY KELLEY (6)**

Current dietary recommendations for the management of type 1 and type 2 diabetes mellitus contain no particular admonitions about the intake of sugars except that sugars should be substituted on a caloric basis for other carbohydrates. The main emphasis is on the total amount of carbohydrate for which the component classifications are sugars, starch, and fiber. Apart from specific recommendations regarding fiber intake, current guidelines emphasize individualization of the carbohydrate content so that, taken together, carbohydrates and monounsaturated fat compose 60–70% of energy intake with protein at 12–20% and saturated and other fat making up the difference. An ongoing area of debate concerns whether the GI of starches and sugars should be considered in the nutritional plan for individuals with diabetes mellitus. Although the issue is not settled, the numerous variables influencing the GI undermine its clinical utility as a specific nutritional principle. Moreover, as discussed in the workshop, sugars per se do not provide the largest contribution to the defined glycemic load of the diet. At this time, the American Diabetic Association has not chosen to enfranchise the use of the GI primarily because of its variability. However, many studies show that substitution of low-GI carbohydrates for high-GI carbohydrates is advantageous in diabetes management.

Research needs in the area of sugars and diabetes mellitus include the question of whether a diet with a high sugars content or a higher GI or glycemic load contributes to the risk of type 2 diabetes mellitus. A related need is the continued examination of whether there is a use for the GI, especially as newer pharmacologic approaches enable improvements in the difficult area of postprandial hyperglycemia. Another area requiring research clarification is the role of sugars and of carbohydrates in inducing triacylglycerol synthesis and affecting the triacylglycerol content of muscle and liver, sites of insulin resistance in type 2 diabetes mellitus.

### **SUGARS, INSULIN SENSITIVITY, AND THE POSTPRANDIAL STATE: PRESENTED BY DALY (7)**

Animal studies have shown that high-sucrose and high-fructose diets decrease insulin sensitivity. However, there is no conclusive evidence showing that humans respond to high-sucrose diets similarly to the way rodents respond.

Considerable attention was given to the potential use of the GI and glycemic load when considering effects on insulin sensitivity. Recent studies showing associations between a high dietary glycemic load and disease (type 2 diabetes and ischemic heart disease) have added to this interest. However, the utility of the terms *GI* and *glycemic load* with respect to sugars and health were strongly questioned. Human studies of diets containing low-GI starches appear to improve insulin sensitivity. However, many question the use of postprandial glucose concentrations as a way to measure insulin sensitivity. Other work showed that different patterns of postprandial glucose and insulin concentrations between sugars and starches varied in a way not reflected in the GI. Minor variations in starch sources have a much greater effect on the area under the insulin curve than are found with sucrose. Therefore, the types of starch in the diet may be more important than the presence of sugars. This is supported by the observation that the major determinants of glycemic load in the American diet are not sugars but high-GI starch sources. Current

data indicate that high-sucrose diets are unlikely to have a detrimental effect on insulin sensitivity.

Several fundamental questions remain with regard to the effects of sucrose on insulin sensitivity and require further investigation. One important question is, "Does the rat model of altered insulin sensitivity with high fructose (>30% of energy) or high sucrose (>60% of dietary energy) have a meaningful parallel in humans?" Studies are needed with the feeding of ad libitum high-sucrose diets (>25% of energy) compared with high-starch diets in the context of high carbohydrate (>55% of energy) and moderate carbohydrate ( $\approx$ 40% of energy). In these studies, insulin sensitivity should be assessed by using a gold standard, dynamic assessment of insulin sensitivity at baseline and posttreatment. These studies should be done in different subject groups [those with a body mass index (in kg/m<sup>2</sup>) > 25 and diabetes, patients with hypertriglyceridemia, and patients with diabetes (type 1 and type 2 separately)]. Results should be interpreted not only as a result of the dietary intervention itself but also as a change from that person's habitual diet. A second question is, "Using a susceptible subject group and a range of sucrose intakes fed in an ad libitum design, do changes in dynamic insulin sensitivity and fasting and postprandial triacylglycerol concentrations change in a dose-response relation?"

#### **SUGARS, HYPERTRIGLYCERIDEMIA, AND CARDIOVASCULAR DISEASE: PRESENTED BY FRIED AND RAO (8)**

Short-term studies show that sugars produce a dose-dependent increase in serum triacylglycerol (sTAG) concentrations. However, the size of the effect of sugars required to raise sTAG concentrations is attenuated by diets that achieve recommended levels of dietary fiber and types of fat (low saturated fatty acids, higher unsaturated fats). Obese individuals with the metabolic syndrome tend to be more responsive to the hypertriglyceridemic effects of diets high in sugars. However, several studies indicate that the modest weight loss (several kilograms) that often accompanies shifts to diets that are rich in fruit, vegetables, and whole grains prevents an increase in sTAG with diets high in sugars (>20% of energy) and total carbohydrates (55–60% of energy). Studies are needed to determine the dose-response effects of dietary sucrose and fructose on sTAG in sedentary and exercising lean and obese men and women. The effects of sucrose on blood lipids may depend on the background diet; for example, 25% of energy from sucrose may have a different effect in diets with different ratios of polyunsaturated to saturated fatty acids.

An increased intake of high-GI carbohydrates has been linked to higher fasting sTAG in men and women. The mechanism by which rapidly absorbed starches increase sTAG probably involves their glycemic and hyperinsulinemic effects. In contrast, fructose increases sTAG independent of insulinemia. Thus, further research is needed to determine whether sugars and high-GI carbohydrates have independent effects on sTAG concentrations in the fasted as well as the fed state. Elucidating the mechanisms involved will also be important.

An key unresolved question is whether the triacylglycerol-rich lipoprotein particles that accumulate in the serum of subjects fed high-sucrose diets are atherogenic and whether they affect cardiovascular risk. Carefully controlled intervention studies with assessment of appropriate biomarkers are needed to address this question.

#### **SUGARS AND DENTAL CARIES: PRESENTED BY TOUGER-DECKER AND VAN LOVEREN (9)**

Sugars and fermentable carbohydrates are acidogenic in dental plaque and, therefore, exert a caries risk. The cariogenic risk is related to the form, retentiveness, duration of exposure, frequency of consumption, nutrient composition, and consumption pattern of the food. However, whether caries develops is modified by many local factors, among which the presence of fluoride is the most important. There are no bad or good foods; all foods can fit into a balanced diet. It is a matter of variety, portion sizes, balance in food groups selected, and integration with oral hygiene, particularly the use of fluorides.

The caries risk of foods may be modified by combining cariogenic foods with dairy products that may reduce the acidogenic effect and promote remineralization. Sugar alcohol-based gums and candies can be a chemotherapeutic agent in the prevention and management of dental caries. Health professionals, in particular dental and nutrition professionals, must recognize the relation between oral health and diet and manage patients accordingly. The primary public health measure from a nutritional perspective is dietary balance and moderation in the adherence to the dietary guidelines, food guides, and dietary reference intakes. The primary public health measure from a dental perspective is the use of topical fluorides and fluoridation of water supplies at appropriate levels.

Further research is needed to 1) determine anticariogenic strategies to reduce risks posed by sugars and other fermentable carbohydrates, 2) explore the use of sugar alcohols (particularly xylitol) and dairy products to prevent caries, 3) determine the cariogenicity of different starches and starch-sugar combinations, and 4) determine the effect of various sugars on plaque pH and specific decay patterns in mixed diets. Longitudinal rather than cross-sectional studies are needed to explore caries risk over time in individuals consuming different sugar and meal-snack consumption patterns. Educational and behavior research is needed to determine strategies to moderate frequency of added sugar intake and increase compliance with the dietary guidelines and dietary reference intakes.

#### **CONCLUSIONS**

Discussions of the role of sugars in diet, health, and weight maintenance are severely hindered by the lack of a common definition of sugars that can be used by the many disciplines and fields involved in this complex area and by the lack of accurate dietary assessment methods. The high current interest in sugars and their potential roles in diabetes mellitus, overweight, obesity, dental caries, and cardiovascular disease has resulted in increased attention to research, much of which is currently in progress, and review of the existing research literature. This is evident in several reports issued by other organizations dealing with various aspects of these issues. These reports were frequently referenced in the various presentations and during the discussions.

Available data show that there are few health concerns for which a direct association with sugar can be established. Confirmatory evidence from a variety of sources supports that sugars intake is associated with increased risk and incidence of dental caries, a decrease in micronutrient intake (especially calcium) only at high levels of intake (>25% of energy), and an increase in sTAG concentrations. The health effect of sTAGs remains to be determined, especially because energy balance and level of physical activity are important in



mitigating the effect of carbohydrates on sTAGs. This is not to say that sugars may not be involved in other health issues cited, particularly when their overconsumption results in an energy imbalance with resulting weight gain, but that currently evidence is not sufficient to validate a direct causative role for sugars consumption.

Throughout this workshop, the research currently underway to obtain scientific evidence bearing on the role of sugars consumption in health and weight maintenance was acknowledged, and additional research needs were identified. Accomplishment of that research is the only way that we will gain insights and answers into these complex health issues. Until then, the recommendations for a balanced diet coupled with sufficient physical activity to maintain energy balance and proper weight should guide the dietary selections and lifestyles of individuals. The presentations in the workshop point out clearly that negative energy balance (eg, sedentary lifestyles) are more important in weight management, insulin sensitivity, and blood lipid concentrations than is the inclusion or exclusion of any particularly dietary component, including sugars. 

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