

## Liquid calories, sugar, and body weight<sup>1-3</sup>

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### ABSTRACT

The consumption of sugar-sweetened beverages has been linked to rising rates of obesity in the United States. The standard explanation is that energy-containing liquids are less satiating than are solid foods. However, purely physiologic mechanisms do not fully account for the proposed links between liquid sugar energy and body weight change. First, a reevaluation of published epidemiologic studies of consumption of sweetened beverages and overweight shows that most such studies either are cross-sectional or are based on passive surveillance of temporal trends and thus permit no conclusions about causal links. Second, research evidence comparing the short-term satiating power of different types of liquids and of solids remains inconclusive. Numerous clinical studies have shown that sugar-containing liquids, when consumed in place of usual meals, can lead to a significant and sustained weight loss. The principal ingredient of liquid meal replacement shakes is sugar, often high-fructose corn syrup, which is present in amounts comparable to those in soft drinks. Far from suppressing satiety, one such liquid shake is marketed on the grounds that it helps control hunger and prevents hunger longer when consumed for the purpose of weight loss. These inconsistencies raise the question whether the issue of sugars and body weight should continue to be framed purely in metabolic or physiologic terms. The effect of sugar consumption on body weight can also depend on behavioral intent, context, and the mode of use, availability, and cost of sweetened liquids. *Am J Clin Nutr* 2007;85:651-61.

**KEY WORDS** Sweetened beverages, meal replacement shakes, weight gain, weight loss, satiety, cost

### INTRODUCTION

Regular consumption of sugar calories in liquid form is said to be responsible for body weight gain (1-4). That is the conclusion of some epidemiologic and experimental studies that have linked the consumption of sweetened beverages in the United States to the rising rates of obesity and overweight (2, 5, 6). Sugar-sweetened beverages are said to promote obesity by virtue of their low satiety and high added sugar content (4).

In evidence-based medicine, one of the criteria for establishing causality is a biologically plausible mechanism (7). The reported links between sweetened beverage consumption and weight gain (4) rest largely on temporal parallels (1, 3) and cross-sectional studies (8). The similarity in time trends between growing beverage consumption (9, 10) and rising obesity rates in

the United States (11-13) is indeed striking and cannot be denied. However, temporal associations are confounded by myriad factors, including dietary and secular trends, and by more sedentary lifestyles (14). Cross-sectional studies, based on a single point in time, do not allow the drawing of conclusions about causal links between sugar intake and the dynamics of weight change.

The search for a biologically plausible mechanism has come to focus on the notion that liquid calories are not perceived by the body. Despite the fact that short-term satiety signals may have little to do with the long-term homeostatic mechanisms regulating body weight, putative satiety deficits are routinely invoked to bolster associations found in epidemiologic studies (1, 4, 6, 15, 16). In fact, the entire debate about beverages and body weight gain has been framed in physiologic terms, and much attention has been paid to satiety (4, 15, 16), energy compensation (4, 17), glycemic index (18), and the vitamin and micronutrient content of beverages (6). When it comes to soft drinks consumption and body weight change, most researchers have opted to implicate human physiology (1, 4, 6, 15, 16, 18) rather than to explore human dietary behavior or the economics of food choice.

Sugar-containing beverages include still and carbonated soft drinks, juice-based beverages, 100% juices, and flavored milk. The overwhelming emphasis has been on soft drinks, and beverages sweetened with high-fructose corn syrup (HFCS) rather than sucrose have come under particular scrutiny (1, 3). In animal models, the consumption of pure fructose or a fructose-rich diet is reported to suppress insulin secretion and leptin production (19, 20), thereby promoting weight gain. One hypothesis is that fructose, whether consumed in solid or in liquid form, does not stimulate insulin secretion or leptin production in humans and may potentially contribute to weight gain (21). However, the question of whether HFCS promotes human obesity (1) requires further study (4). This review will distinguish between different

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