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# Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis

David S Ludwig, Karen E Peterson, Steven L Gortmaker

## Summary

**Background** The rising prevalence of obesity in children has been linked in part to the consumption of sugar-sweetened drinks. Our aim was to examine this relation.

**Methods** We enrolled 548 ethnically diverse schoolchildren (age 11.7 years, SD 0.8) from public schools in four Massachusetts communities, and studied them prospectively for 19 months from October, 1995, to May, 1997. We examined the association between baseline and change in consumption of sugar-sweetened drinks (the independent variables), and difference in measures of obesity, with linear and logistic regression analyses adjusted for potentially confounding variables and clustering of results within schools.

**Findings** For each additional serving of sugar-sweetened drink consumed, both body mass index (BMI) (mean 0.24 kg/m<sup>2</sup>; 95% CI 0.10–0.39; p=0.03) and frequency of obesity (odds ratio 1.60; 95% CI 1.14–2.24; p=0.02) increased after adjustment for anthropometric, demographic, dietary, and lifestyle variables. Baseline consumption of sugar-sweetened drinks was also independently associated with change in BMI (mean 0.18 kg/m<sup>2</sup> for each daily serving; 95% CI 0.09–0.27; p=0.02).

**Interpretation** Consumption of sugar-sweetened drinks is associated with obesity in children.

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See commentary page 505

## Introduction

The prevalence of obesity among children in USA increased by 100% between 1980 and 1994.<sup>1</sup> Recent national estimates indicate that 24% and 11% of children are above the 85th and 95th reference percentiles of body mass index (BMI), for age and sex, respectively. Various environmental and social factors relating to diet and physical activity have been identified that could contribute to obesity.<sup>2</sup> One such factor, which has received little attention, is the consumption of sugar-sweetened drinks.

According to data from the US Department of Agriculture (USDA), per capita soft-drink consumption has increased by almost 500% over the past 50 years.<sup>3</sup> From 1989–91 to 1994–95, soft-drink intake rose from 195 to 275 mL in the general population, and from 345 to 570 mL among adolescent boys.<sup>4</sup> Half of all Americans and most adolescents (65% girls and 74% boys) consume soft drinks daily,<sup>5</sup> most of which are sugar-sweetened, rather than artificially sweetened.<sup>6</sup> Currently, soft drinks constitute the leading source of added sugars in the diet,<sup>7,8</sup> amounting to 36.2 g daily for adolescent girls and 57.7 g for boys.<sup>7</sup> These figures approach or exceed the daily limits for total

added sugar consumption recommended by the USDA. Among children of school age, total energy intake is positively associated with soft-drink consumption, ranging from an adjusted mean of 7650 kJ daily for non-consumers to 8435 kJ for those drinking an average of 265 mL or more every day.<sup>6</sup>

Although this increase in soft-drink consumption coincides with secular increases in obesity prevalence in children, the long-term effects of sugar-sweetened drink consumption on measures of body weight need to be prospectively examined. We aimed to determine the association between change in sugar-sweetened drink consumption and change in BMI, and incidence of obesity among school-age children, over 2 academic years.

## Methods

### Patients

Data for our study were obtained as part of the Planet Health intervention and evaluation project, which took place in schools in four communities in the Boston, Massachusetts, metropolitan area between October, 1995, and May, 1997. We enrolled children from five randomly assigned control schools that did not take part in the lifestyle intervention programme designed to reduce obesity prevalence.<sup>9</sup> The median household income of zip-code areas where the control schools were located, averaged US\$34 200, according to 1990 census data. This median is lower than that for all households in Massachusetts in the 1990 census (\$41 000), but similar to the USA figure (\$33 952).<sup>10</sup> After excluding individuals who changed schools at baseline, were in special education classes, were in grades other than sixth (11 years) or seventh (12 years) or did not complete the English-language version of the questionnaire, a total of 780 people (64.5% of those eligible) completed the baseline evaluation in October, 1995. Follow-up data were obtained in May, 1997 (19 months later, SD 0.14), for 84% (654) of the baseline sample, indicating a drop-out rate of 18% (69) for girls and 14% (57) for boys. The main reason for lack of follow-up anthropometric data was school transfer (half those not followed-up) and school absence (a quarter). Complete data on all variables were available for 571 children. We excluded an additional 23 children because of implausible daily energy intakes ( $\leq 2090$  kJ or  $\geq 29\,260$  kJ), leaving a cohort of 548 individuals for analysis. At baseline, characteristics of the cohort were: mean age 11.7 years (SD 0.8); 48% (263) female, 64% (351) white, 15% (82) Hispanic, 14% (77) Afro-American, 8% (44) Asian, and 8% (44) American Indian or other; and 38% (208) reported exercise to lose weight. Further details of the school recruitment process, the sampling plan, and a comparison of those followed-up and not are described elsewhere.<sup>9</sup> The study was approved by the Committee on Human Subjects at the Harvard School of Public Health. Informed consent was obtained from all individuals, as previously described.<sup>9</sup>

### Protocol

In this prospective observational analysis, the primary hypotheses were that baseline, and change in, consumption of sugar-sweetened drinks could directly predict a rise or fall in BMI over 2 academic years. Demonstrating that change in an independent variable predicts change in a dependent variable could provide stronger evidence for causality than

Department of Medicine, Children's Hospital, Boston, MA 02115, USA (D S Ludwig MD); and Departments of Maternal and Child Health and Nutrition (K E Peterson ScD), and Department of Health and Social Behaviour (S L Gortmaker PhD), Harvard School of Public Health, Boston

**Correspondence to:** Dr D S Ludwig  
(e-mail: david.ludwig@tch.harvard.edu)

predictions involving the independent variable measured at just one point in time—eg, baseline.<sup>11</sup> Obesity incidence was the dependent variable in secondary analyses.

We obtained anthropometric data and student surveys at the beginning of grades six and seven in October, 1995, and follow-up measurements about 19 months later, in May, 1997. Height without shoes was measured to the nearest 0.1 cm using a Shorr stadiometer (Irwin Shorr, Olney, MD, USA) and weight in light clothes was measured to the nearest 0.1 kg on a portable electronic scale (Seca Model 770, Seca Corporation, Hanover, MD, USA) calibrated with the Seca standard weights step-up test. BMI was calculated by dividing weight by height, and was expressed as kg/m<sup>2</sup>. We defined obese students with a composite indicator,<sup>12</sup> on the basis of both BMI and triceps-skinfold thickness greater than or equal to the 85th percentile of age-specific and sex-specific reference data.<sup>13</sup> Triceps-skinfold thickness was measured to the nearest 0.2 mm by trained project staff, with calibrated Holtain calipers (Holtain Ltd, Crymch, Pembrokeshire, UK),<sup>14</sup> but without rigorous control of the children's clothing. To improve precision, more than one measurement was done; if two measurements differed by more than 2 mm, a third was taken, and the average was used.

Sexual maturity ratings are recommended to interpret and control for differences among individuals in the maturational tempo not indicated in reference growth curves for BMI and triceps-skinfold thickness.<sup>15</sup> Use of self-reported or clinical sexual maturity rating assessment in either boys or girls was not allowed by school systems. We therefore obtained baseline self-reports of menarcheal status in girls.

Measures of dietary intake, physical activity, and television viewing were obtained with a student food and activity questionnaire. Students completed this questionnaire independently, in class, and under the supervision of teachers who participated in a 1-h training session before administration. The youth food-frequency questionnaire (YFFQ), adapted and validated for use in ethnically and socioeconomically diverse populations,<sup>16,17</sup> was used to assess average intake of drinks, percentage energy intake from dietary fat, and total energy intake. Sugar-sweetened drink consumption was calculated from responses to the YFFQ that inquired as to how often in the past 30 days three items were consumed: soda (never or less than one can per month, one to three per month, one per week, two to six per week, one per day,  $\geq$ two per day); Hawaiian punch, lemonade, Koolaid, or other sweetened fruit drink (never or less than one glass per month, one to three per month, one per week, two to four per week, five to six per week, one per day,  $\geq$ two per day); and iced tea, not artificially sweetened (never or less than one glass per month, one to three per month, one to four per week,  $\geq$ five per week). These three items were added to measure the total daily intake of sugar-sweetened beverage. One question, concerning diet soda (categorised as per soda, above), was used to establish the amount of diet-soda intake every day. Fruit juice (100%) consumption was calculated from responses to two questions about orange, apple, and other fruit juices (quantified as never or less than one glass per month, one to three per month, one per week, two to six per week, one per day,  $\geq$ 2 per day). The category two or more cans per day was coded as two cans per day; the category five or more glasses per week was coded as five glasses per week. For the other drink items, ranges were coded to the midpoint.

Physical activity was assessed with the youth activity questionnaire (YAQ), which consists of 16 items that together estimate the amount of hours per day spent doing

moderate and vigorous activities ( $\geq$ 3.5 mets)<sup>18</sup> over the past month. Walking was excluded because of the low validity found for this activity.<sup>19</sup> The YAQ is based on a 14-item physical activity questionnaire shown to have good reproducibility and validity in adults,<sup>20,21</sup> and children of highschool age.<sup>19</sup> In a validation study among participants in Planet Health, with repeat 24-h recalls one month apart, the YAQ correlated (deattenuated) with the average of these two 24-h recalls ( $r=0.80$ , with equivalent means).<sup>9</sup> (Deattenuation adjusts for random error seen in the measures, providing a more accurate assessment of the relation between variables of interest.<sup>22</sup>) Although repeat 24-h recalls do not constitute a gold standard of dietary intake, the results provide validity evidence for YAQ.<sup>22</sup>

Time spent watching television and videos was measured with the 11-item television and video measure (TVM).<sup>9</sup> Questions were asked about hours of television typically viewed during every day of the week, as well as use of video cassette recorders, and video and computer games. Items were appropriately weighted and summed to obtain a total viewing hour-per-day estimate. In the validation sample ( $n=53$ ), we found a deattenuated<sup>22</sup> correlation of television viewing via the TVM and the 24-h recall of  $r=0.54$ , with equivalent means.

Age was calculated on the basis of birth date and date of anthropometric examinations; in a few cases of missing birth date, self-reported age from the FAS survey was used. Sex was established at the time of examination, apart from a few missing cases for whom it was obtained from school lists. Ethnic origin was established on the basis of responses of students to a multiple choice question. Participants indicating black on the questionnaire were classified as Afro-American. The self-report questions about exercising to lose weight were adapted from national surveillance indicators.<sup>19,23</sup>

#### Statistical analysis

Because students are clustered within schools, we used SUDAAN software (version 1996) for analysis of correlated data to estimate regressions taking into account the clustered sample. SUDAAN estimates use an implicit Taylor linearisation method. For dichotomous outcomes (obesity incidence), the generalised estimating equation (GEE) method was used<sup>24</sup> with software written for use with SAS data sets. Both estimation approaches take into account the intraclass correlation of responses within schools.

The analyses contained terms for baseline consumption of sugar-sweetened drinks and change in consumption (follow-up minus baseline). After examination of relations between independent variables to ensure lack of multicollinearity, we sequentially adjusted for sets of variables that might confound the associations between intake of sugar-sweetened drinks and measures of obesity. Model 1 included baseline anthropometrics (BMI and triceps-skinfold thickness); demographics (age, sex, ethnicity<sup>13,15</sup>); and indicator variables for schools (the largest as the omitted category). Model 2 included the variables in model 1 plus other factors that might affect body weight, including diet (percent energy from fat at baseline, energy-adjusted fruit-juice intake at baseline, and change in these variables from baseline to follow-up); physical activity (whether exercising to lose weight,<sup>9</sup> physical activity  $\geq$ 3.5 met, change in physical activity  $\geq$ 3.5 met, number of physical education classes per week); and time spent watching television and videos,<sup>9</sup> and change in time spent watching television and videos. In model 3, we controlled further for total energy intake (kJ daily) by replacing the sugar-sweetened drink variables with energy-adjusted sugar-

sweetened drink variables (baseline and change from baseline to follow-up). Although total energy intake might be a causal factor relating obesity to sugar-sweetened drink intake, this variable could also confound our associations if beverage consumption is a marker for increased consumption of other foods. Therefore, we included this adjustment in our last model. All p values are two-tailed.

## Results

Table 1 shows baseline and follow-up anthropometric and dietary data. Intake of sugar-sweetened drinks increased from baseline to follow-up: only 38 (7%) children showed no change in sugar-sweetened drink intake whereas 57% (312) showed increased intake, with a quarter drinking more than one extra serving daily. BMI also increased. The baseline prevalence of obesity was just over a quarter, and the cumulative incidence of new cases over the 19 months was 9.3%. Children reported moderate to vigorous activity for roughly 1–2 h per day. Table 2 shows the associations between sugar-sweetened beverage consumption and BMI at follow-up, controlling for baseline BMI, with further adjustment for potentially confounding variables in three different models. In the fully adjusted model 3, BMI was increased for each serving per day at baseline, and further increased for every additional serving. Table 3 shows the association between sugar-sweetened drink consumption and obesity incidence, controlling for potentially confounding variables. In the fully adjusted model 3, the odds of becoming obese increased significantly for each additional daily serving of sugar-sweetened drink. There was no independent, significant association between baseline consumption and obesity incidence, though the direction of the association was the same as that for change in sugar-sweetened drink consumption.

We also estimated fully adjusted regressions, replacing the sugar-sweetened drink variables with measures of diet-soda consumption and change in diet-soda consumption (model 3 in tables 2 and 3). There were no significant relations with BMI ( $p=0.10$  for both baseline consumption and change in consumption), and the coefficient estimates were negative. Baseline diet-soda intake was not related to obesity incidence ( $p=0.69$ ) but change in diet-soda intake was negatively associated with incidence (odds ratio 0.44,  $p=0.03$ ).

To control for the potential effect of sexual maturity, we

|  | Baseline     | Follow-up    |
|--|--------------|--------------|
| <b>Anthropometric</b>  |              |              |
| Body mass index ( $\text{kg}/\text{m}^2$ )                   | 20.73 (3.99) | 22.23 (4.38) |
| Triceps skinfolds (mm)                                       | 15.87 (6.74) | 17.38 (7.31) |
| Number obese*  | 150 (27.4%)  | 152 (27.7%)  |
| Obesity incidence†   | ..           | 37 (9.3%)    |
| <b>Dietary‡</b>  |              |              |
| Sugar-sweetened drinks (daily servings)                      | 1.22 (1.10)  | 1.44 (1.09)‡ |
| Fruit juice (daily servings)                                 | 1.28 (1.17)  | 1.08 (1.04)  |
| Total energy intake (kJ)                                     | 8950 (4500)  | 9610 (4715)  |
| Energy from fat  | 31.3% (5.4%) | 30.1% (5.1%) |
| Change in sugar-sweetened drink consumption (daily servings) | ..           | 0.22 (1.14)  |
| Change in juice consumption (daily servings)                 | ..           | -0.20 (1.21) |
| Change in energy intake from fat                             | ..           | -1.8% (5.6%) |
| <b>Physical activity and inactivity</b>                      |              |              |
| Daily television viewing (h)                                 | 3.32 (2.10)  | 3.11 (2.08)  |
| Daily reported h of activity ( $\geq 3.5$ met)               | 1.34 (1.09)  | 1.28 (1.03)  |
| Weekly number of physical education lessons                  | 2.00 (1.20)  | 2.09 (1.03)  |
| Change in television viewing                                 | ..           | -0.21 (2.07) |
| Change in h of activity                                      | ..           | -0.06 (0.97) |

\*BMI and triceps-skinfold measurements  $\geq 85$ th reference percentiles. †Obesity incidence=number of the 398 individuals not obese at baseline, who became obese at follow-up. ‡ $p<0.001$ . All values are mean (SD) unless otherwise indicated.

Table 1: Baseline (October, 1995) and follow-up (May, 1997) anthropometric, dietary, and activity data (n=548)

|   | Baseline consumption* |      | Change in consumption† |      |
|---|-----------------------|------|------------------------|------|
|   | Mean (95% CI)         | p    | Mean (95% CI)          | p    |
| <b>Model 1</b><br>Baseline anthropometrics, demographics                        | 0.12 (0.03–0.21)      | 0.06 | 0.20 (0.11–0.30)       | 0.01 |
| <b>Model 2</b><br>Plus dietary variables, physical activity, television viewing | 0.13 (0.05–0.21)      | 0.03 | 0.20 (0.09–0.30)       | 0.02 |
| <b>Model 3</b><br>Plus total energy intake                                      | 0.18 (0.09–0.27)      | 0.02 | 0.24 (0.10–0.39)       | 0.03 |

\*BMI ( $\text{kg}/\text{m}^2$ ) per daily serving at baseline. †BMI ( $\text{kg}/\text{m}^2$ ) per one daily serving increase.

Table 2: Relation between intake of sugar-sweetened drinks (baseline consumption and change in consumption from baseline to follow-up) and BMI in May, 1997, controlling for baseline BMI (October, 1995) and other covariates, among the 548 children

added self-reported menarcheal status to the regressions. When this adjustment was made to model 3 in table 2, the coefficients for baseline consumption and change in consumption were unchanged (0.18,  $p=0.02$ ; and 0.24,  $p=0.03$ , respectively).

## Discussion

Excessive bodyweight probably now constitutes the most common paediatric medical problem in USA. Although the cause of this apparent obesity epidemic is likely to be multifactorial, our findings suggest that sugar-sweetened drink consumption could be an important contributory factor. The odds ratio of becoming obese among children increased 1.6 times for each additional can or glass of sugar-sweetened drink that they consumed every day. By contrast, increased diet-soda consumption was negatively associated with obesity incidence. Our prospective analysis also indicates that both baseline sugar-sweetened drink consumption and change in consumption independently predict change in BMI.

There are several limitations to the interpretation of our findings. First, our study was observational in nature and cannot prove causality. Although we attempted to control for the effects of the major identified predictors of obesity in childhood, sugar-sweetened drink consumption could be a marker for unrecognised factors that affect body weight. Furthermore, inaccuracy in measurement of factors included in our models, such as menarcheal status by self-

|   | Baseline consumption* |      | Change in consumption† |       |
|---|-----------------------|------|------------------------|-------|
|   | Mean (95% CI)         | p    | Mean (95% CI)          | p     |
| <b>Model 1</b><br>Baseline anthropometrics, demographics                        | 1.41 (0.62–3.25)      | 0.31 | 1.39 (0.99–1.95)       | 0.05  |
| <b>Model 2</b><br>Plus dietary variables, physical activity, television viewing | 1.46 (0.57–3.77)      | 0.33 | 1.44 (1.22–1.70)       | 0.004 |
| <b>Model 3</b><br>Plus total energy intake                                      | 1.48 (0.63–3.47)      | 0.27 | 1.60 (1.14–2.24)       | 0.02  |

\*Odds ratio per daily serving at baseline. †Odds ratio per one daily serving increase. ‡Incidence rate per 19-month follow up.

Table 3: Odds ratio for relation between intake of sugar-sweetened drinks (baseline consumption and change in consumption from baseline to follow-up) and incidence of obesity‡ from baseline to follow-up in 398 children classified as non-obese at baseline



report or total energy consumption, might mask residual confounding. The possibility of confounding is especially strong for the analyses for diet soda, since these drinks could be preferred by individuals trying to lose weight. Second, for logistical reasons, we used indirect measures of obesity (BMI and a composite indicator of BMI and triceps-skinfold thickness). Although these measures are in widespread use, and provide a good estimate of adiposity in children,<sup>25</sup> we cannot fully control for changes in body composition over time, resulting, for example, from puberty or fitness training. Third, the study has limited statistical power, with 548 children (the entire cohort) in analyses of BMI, but only 37 in estimates of incident obesity. By contrast with these limitations, random error in the measurement of drink consumption, and inaccuracy in the estimation of adiposity by BMI, could lead to underestimation of actual effects.

Why should consumption of sugar-sweetened drinks promote obesity any more than other categories of food? In the short-term, most individuals effectively compensate for excess energy consumption by eating less at subsequent meals.<sup>26</sup> In the longterm, changes in bodyweight elicit physiological adaptations, involving hunger and rate of metabolism, that tend to restore baseline bodyweight.<sup>27</sup> Indeed, there is no clear evidence that consumption of sugar per se affects food intake in a unique manner or causes obesity.<sup>28</sup> However, a meta-analysis of studies done over 25 years suggests that compensation at subsequent meals for energy consumed in the form of a liquid could be less complete than for energy consumed in the form of solid food.<sup>29</sup> For example, De Castro<sup>30</sup> examined 7-day food diaries of 323 adults and found that energy from drinks added to total energy intake and did not displace energy ingested in other forms. Mattes<sup>29</sup> showed that total energy consumption among 16 patients was greater on the day that an energy-containing drink was given at lunch than on the preceding day. Moreover, Tordoff and Alleva<sup>31</sup> found that total energy intake and body weight increased in people given 2215 kJ of sugar-sweetened drink daily for 3 weeks, but decreased when they were given artificially sweetened carbonated drinks for the same period of time, relative to when no such drinks were given. Finally, school children drinking an average of 265 mL or more of soft drinks daily consumed almost 835 kJ more total energy every day than those drinking no soft drinks.<sup>6</sup> Thus, the results of our study are consistent with a plausible physiological mechanism, that consumption of sugar-sweetened drinks could lead to obesity because of imprecise and incomplete compensation for energy consumed in liquid form.

#### Contributors

David Ludwig and Steven Gortmaker designed the study, interpreted the data, and wrote the initial draft of the manuscript. Karen Peterson was co-principal investigator of Planet Health, and assisted in data analysis and manuscript preparation.

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