Beverage Intake During Pregnancy and Childhood Adiposity

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OBJECTIVES: To examine associations of sugar sweetened beverages (SSBs) and other beverage intake during pregnancy with adiposity in midchildhood (median age of 7.7 years).

METHODS: We studied 1078 mother-child pairs in Project Viva, a prospective prebirth cohort study. Exposures were sugary and nonsugary beverage intake assessed in the first and second trimesters of pregnancy by using a food frequency questionnaire. Main outcome measures were offspring overall adiposity (BMI z score, fat mass index [FMI, kg/m²] from dual-energy radiograph absorptiometry, and sum of subscapular [SS] and triceps [TR] skinfold thicknesses) and central adiposity (SS:TR ratio and waist circumference).

RESULTS: In the second trimester, mean (SD) SSB intake was 0.6 (0.9) servings per day. Thirty-two percent of mothers were multiracial or people of color, 68% were college graduates, and 10% smoked during pregnancy. In midchildhood, mean (SD) BMI z score was 0.38 (1.00), and the FMI was 4.4 (1.9) kg/m². In multivariable models adjusted for multiple maternal and child covariates, each additional serving per day of SSB was associated with higher BMI z scores (0.07 U; 95% confidence interval [CI]: −0.01 to 0.15), FMI (0.15 kg/m²; 95% CI: −0.01 to 0.30), SS + TR (0.85 mm; 95% CI: 0.06 to 1.64), and waist circumference (0.65 cm; 95% CI: 0.01 to 1.28). Stratified models suggested that the associations were due primarily to maternal, not child, SSB intake and to sugary soda rather than fruit drinks or juice. We did not find differences between boys and girls, nor did we observe the effects of sugar-free soda or of first-trimester beverage intake.

CONCLUSIONS: Higher SSB intake during the second trimester of pregnancy was associated with greater adiposity in midchildhood.

WHAT’S KNOWN ON THIS SUBJECT: Maternal diet during pregnancy may entrain offspring obesity. One potential dietary factor is sugar-sweetened beverages, intake of which appears to cause obesity in children and adults.

WHAT THIS STUDY ADDS: In this prospective prebirth cohort study, school-aged children of mothers who consumed more sugary beverages in midpregnancy had higher levels of adiposity, measured by BMI, skinfold thicknesses, and dual-energy radiograph absorptiometry.

One set of solutions for the worldwide childhood obesity pandemic emphasizes prevention in the early, plastic stages of human development, when the right cues could set lifelong trajectories of optimal health.1 Decades of animal experiments show that perturbations during early, sensitive periods of development affect fat deposition and development of cardiometabolic dysfunction.2 Many of these experiments comprise alterations in the diet of pregnant mothers, including reductions in energy or protein intake or, more recently, high fat diets, westernized diets, and high-sugar diets.3–5 Although these experiments provide proof of principle, the experimental energy and protein restriction tend to be out of the physiologic range of humans, many of the components of “overnutrition” diets in animal models do not have analogs in the human diet, and the variation in experimental paradigms make translation to the human condition indirect at best.6

In humans, it has been more challenging to demonstrate robust associations between the diets of pregnant women and obesity in their offspring. One reason is a focus on nutrients, akin to the animal studies, rather than foods. Although nutrient-based studies7–10 may more closely approach causal mechanisms, foods are what people buy, cook, and eat, and thus food-based recommendations are easier to “absorb.”7–10 In addition, dietary assessment begins with asking about foods; nutrient calculations rely on external databases as well as reported food intake, thus introducing multiple sources of error. Another reason is that few cohort studies with accurate dietary assessment in pregnancy have child follow-up to school age,11 when obesity becomes a good predictor of later adverse health outcomes.12 and none, to our knowledge, have examined maternal beverage intake. Sugar-sweetened beverage (SSB) intake is a prime target of obesity prevention and treatment strategies in nonpregnant adults and children. Numerous observational studies and meta-analyses in adults, and some among children, demonstrate that intake of SSBs predicts excess weight gain, obesity, metabolic syndrome, and type 2 diabetes.13–15 National and local policies are geared toward reducing consumption of sugary beverages, and in recent years in the United States (but not abroad), sugary soda consumption has begun to decline.15–18 Despite the clarity from observational studies, 2 recent intervention studies to reduce SSBs among children and adolescents, especially 1 among already overweight or obese adolescents, revealed only modest effects on weight-related outcomes, perhaps because physiologic plasticity is already waning at those ages.19–21 Once obesity takes hold, physiologic, behavioral, and social forces tenaciously fight against weight control, even in childhood.22,23

The purpose of this study was to investigate associations of sugary and nonsugary beverage consumption during pregnancy with obesity-related outcomes in midchildhood among mother-child pairs participating in the prebirth cohort study Project Viva.

METHODS

Between 1999 and 2002, we recruited women into Project Viva in early pregnancy from 8 obstetric offices of Atrius Harvard Vanguard Medical Associates, a multispecialty group practice in eastern Massachusetts.24 Details of recruitment and retention are published.24 Of the 2128 women who delivered a live singleton infant, we excluded from this analysis 59 participants with previous type 1 or type 2 diabetes mellitus or gestational diabetes in a previous pregnancy, 44 participants who were born before 34 weeks’ gestational age, and 947 participants who did not attend the midchildhood in-person visit. Thus, our sample size for analysis was 1078 mother-child pairs. Compared with the 1078 participants in this analysis, the 947 nonparticipants were somewhat less likely to have college-educated mothers (62% vs 68%) and to have annual household income exceeding $70,000 (56% vs 61%), and the mean maternal age was slightly lower (31.5 vs 32.1 years). Maternal race and/or ethnicity (33% vs 32% multiracial or people of color) and BMI (mean of 24.8 vs 24.6) and intake of second-trimester SSBs (mean of 0.69 vs 0.63 servings per day), however, were similar.

After obtaining written informed consent, we performed in-person study visits with participating mothers at the end of the first and second trimesters of pregnancy and with mothers and children during the first few days after delivery and in infancy (median age of 6.3 months), early childhood (median age of 3.2 years), and midchildhood (median age of 7.7 years). Mothers completed mailed questionnaires at 1, 2, 4, 5, and 6 years of age. The institutional review boards of Harvard Pilgrim Health Care, Brigham and Women’s Hospital, and Beth Israel Deaconess Medical Center approved the study protocols.

Exposures

We obtained data on consumption of beverages during pregnancy from semiquantitative food frequency questionnaires (FFQs) that expectant mothers completed after the first and second research visits, at the mean (SD) gestational ages of 11.9 (3.5) and 29.2 (2.6) weeks. Each of the 2 FFQs was slightly modified for use in pregnancy from a commonly used adult FFQ from which SSB intake
predicts a number of cardiometabolic outcomes.13−15,25,26 Participants endorsed categories of frequency of beverage consumption from “never/less than 1 per month” to a maximum of “2 or more glasses per day” for some fruit juices, “4 or more cans per day” for soda, and “6 or more glasses per day” for water. The time referent for the first-trimester FFQ was “during this pregnancy,” that is, from the woman’s last menstrual period until she completed the FFQ. For the second-trimester FFQ, the time referent was the previous 3 months. The FFQ included 3 questions on regular (sugary) soda intake, 3 questions on sugar-free soda, 5 questions on fruit juice, 1 question on fruit drinks, and 2 questions on water. We defined SSBs as regular soda and fruit drinks. We also included fruit juice, sugar-free soda (diet soda), and water in our analyses. Although the FFQ included 4 questions regarding the type of milk consumed, it did not include specific information about sweetened and flavored milk. Therefore, we did not include dairy products in this analysis, nor did we include tea or coffee, although these were queried on the FFQs.

Outcome Measures

At the midchildhood visit (median age of 7.7 years, range of 6.6−10.9 years), trained research staff measured each child’s height to the nearest 0.1 cm and weight to the nearest 0.1 kg with a calibrated stadiometer (Shorr Productions, Olney, MD) and calibrated scale (Tanita model TBF-300A; Tanita Corporation of America, Inc, Arlington Heights, IL). We calculated age- and sex-specific BMI z scores by using US national reference data.27 We also measured total fat mass with dual-energy radiograph absorptiometry (DXA) (Discovery A model; Hologic Inc, Marlborough, MA), and we calculated fat mass index (FMI) as fat mass in kilograms divided by height in meters squared, analogous to BMI. We measured waist circumference to the nearest 0.1 cm with a Hoechstmass measuring tape (Hoechstmass Balzer GmbH, Sulzbach, Germany), and subscapular skinfold thickness (SS) and triceps skinfold thickness (TR) to the nearest 0.1 mm with Holtain calipers (Holtein LTD, Crosswell, UK) and calculated their sum (SS + TR) and ratio (SS:TR). Research assistants performing all measurements followed standardized techniques28 and participated in in-service training to ensure measurement validity (I J Shorr; Shorr Productions). Inter- and intrarater measurement errors were within published reference ranges for all measurements.29

Covariates

At each in-person and mailed visit from early pregnancy through the midchildhood outcome visit, as well as from outpatient and hospital medical records, we collected information on many potentially confounding, mediating, and moderating variables. We collected sociodemographic and medical data at enrollment, with regular updates. Mothers reported their age, race and/or ethnicity, education, parity, prepregnancy weight and height, and paternal weight and height. We calculated total gestational weight gain by subtracting self-reported prepregnancy weight from the last prenatal weight from medical records. We calculated gestational age by using the date of the last menstrual period, but if the early second-trimester ultrasound assessment differed from the calculated gestational age by more than 10 days, we used the ultrasound dating instead. We obtained birth weight from medical records. At appropriate ages, mothers reported the number of hours their children spent in child care, timing of solid food introduction, breastfeeding duration, child diet, television viewing, and physical activity habits.30−32

To explore mediation or moderation by child’s beverage intake, we used data from a beverage frequency questionnaire that mothers completed at the midchildhood visit. This questionnaire, with 7 response categories from “never” to “5 or more times per day” for each beverage, included 11 questions on milk intake, including flavored milk (which typically contains added sugars); 1 question each on regular and sugar-free soda; 2 questions on fruit juice; 1 question on fruit drinks; and 1 question on water. To facilitate consistency with the maternal prenatal report of beverage intake, in our primary analysis we did not include sweetened flavored milk in our calculations of child intake of SSBs. In a secondary analysis, inclusion of sweetened flavored milks did not materially change the findings.

Data Analysis

The main elements of our analytic approach included multivariable linear and logistic regression to account for confounding and to explore mediation, stratification to examine effect modification, and multiple imputation to compensate for missing data. Our primary exposure was beverage consumption in the second trimester of pregnancy, expressed as a continuous variable in regression models, but displayed as <0.5, 0.5 to <1, 1 to <2, 2 to <3, or at least 3 servings per day in tables. We also examined first-trimester intake and, additionally, the change from first to second trimester because preliminary data showed that changes in SSB consumption predicted gestational weight gain during the same period.33 Gestational weight gain itself is a predictor of offspring obesity in this and other populations.34 Main midchildhood outcomes included BMI z score, obesity, FMI, sum of SS + TR skinfolds
for overall adiposity, and the ratio of SS:TR and waist circumference for central adiposity.

To examine exposure-outcome relationships, we initially examined exposures as categorical variables (quartiles). Because trends across quartiles were linear, we expressed exposures as continuous variables in regression models. We built multivariable linear regression models in which we adjusted for maternal age, race and/or ethnicity, education, smoking, parity, and prepregnancy BMI; household income; and child sex and exact age at the time of the midchildhood visit. Adding other potentially confounding variables, including maternal intake of fried food away from home, carbohydrates, fatty acids, total energy intake, fetal growth, breastfeeding duration, midchildhood sleep duration, television viewing, and physical activity, did not materially change the observed associations, so we did not include them in our final models. We did not adjust for gestational weight gain and fetal growth because they might be on the causal pathway and would be likely to attenuate the total associations. We also considered whether lack of covariate overlap between exposed (maternal SSB intake ≥2 servings per day) and unexposed (<2 servings per day) drove results. We used propensity scores to define overlapping covariate values, or “common support.” We ran common-support regression after excluding 11 participants for whom 1 or the other exposure group provided few data, and results were similar. To examine effect modification, we stratified adjusted models by maternal prepregnancy BMI, child sex, and child race and/or ethnicity in separate models. To examine the mediating or moderating effect of child beverage intake, we dichotomized both maternal and midchildhood sugary beverage intake at cut points defined post hoc by examination of the data and examined effects within each of the 4 resulting strata; as an alternative approach, we added child intake as a covariate.

When including a single beverage as an exposure, these regression approaches essentially reveal the effect of adding servings of a beverage to one’s diet. We also explored effects of substituting each type of beverage for SSBs by fitting models that also included total beverage intake and all beverage types except SSBs. This type of model may more accurately reflect the common practice of drinking 1 type of beverage instead of another, rather than adding it to total fluid intake.

To account for missing data, we performed multiple imputation for all 2128 mother–child pairs in Project Viva. We then limited the analysis to the 1078 included participants. We used SAS (Proc MI) to impute 50 values for each missing observation and combined multivariable modeling estimates by using Proc MI ANALYZE in SAS version 9.3 (SAS Institute, Cary, NC). An alternative approach, including only participants with all covariate data (complete cases), yielded similar results. Because we observed approximately linear effects across quartiles of intake, we reported regression estimates and their 95% confidence intervals (CIs) for each increment of 1 serving per day of beverage intake as a continuous variable.

Results

Mean (SD) maternal age at enrollment was 32.1 (5.4) years, prepregnancy BMI was 24.6 (5.2), and total gestational weight gain was 15.6 (5.3) kg; 32% of the participants were multiracial or people of color, 10% smoked during pregnancy, 68% were college graduates, and 39% had household incomes ≤$70 000 per year (Table 1).

In the second trimester, mean (SD, range) sugary beverage intake was 0.6 (0.9, 0.0–8.3) servings per day. Over half consumed <0.5 servings per day, and fewer than 10% drank more than 2 servings per day. Mean intakes of juice, diet soda, and water were 1.4, 0.1, and 4.5 servings per day, respectively (Table 2).

Correlates of higher second-trimester consumption of sugary beverages included younger maternal age, higher prepregnancy BMI, multiracial race and/or ethnicity, lower education and household income, smoking during pregnancy, shorter breastfeeding duration, and earlier introduction of solid foods (Table 1). In midchildhood, mean (SD) BMI z score was 0.38 (1.00) and 272/1078 (25.2%) of the children were overweight or obese, that is, BMI exceeding the age–sex–specific 85th percentile. These and the other adiposity outcomes were highest among children whose mothers consumed at least 2 servings per day of sugary beverages (Table 1). The Spearman correlation coefficient was 0.27 between maternal second-trimester and child midchildhood consumption of sugary beverages, 0.07 between maternal second-trimester consumption and child midchildhood BMI z score, and 0.10 between child consumption and BMI z score.

In multivariable models (Table 3), we found that maternal second-trimester intake of sugary beverages was related to all child overall adiposity and central adiposity outcomes. Unadjusted estimates (95% CIs) for each additional serving per day included 0.15 U (0.07 to 0.23) for BMI z score, 0.31 kg/m² (0.15 to 0.47) for FMI, 1.76 mm (0.94 to 2.59) for SS + TR, 2.12 (0.65 to 3.60) for SS:TR, and 1.47 cm (0.79 to 2.15) for waist circumference. These estimates were attenuated by adjusting for sociodemographic variables and maternal prepregnancy BMI and...
smoking. For example, the adjusted estimate (95% CI) for BMI z score was 0.07 U (−0.01 to 0.15) and for FMI was 0.15 kg/m² (−0.01 to 0.30). In logistic regression, the adjusted odds ratio (OR) for childhood BMI exceeding the 85th percentile was 1.14 (0.95 to 1.36).

Only maternal consumption of SSBs (0.07 U; −0.01 to 0.15), not juice, diet soda, or water, was associated with BMI z score (Table 4). Between the 2
types of sugary beverages, maternal intake of soda demonstrated the stronger association (0.11 U; −0.02 to 0.23) than did fruit drinks (0.06 U; −0.05 to 0.17), although CIs were wide and crossed 0. Estimates for juice 0.01 (−0.05 to 0.06), diet soda 0.07 (−0.05 to 0.18), and water 0.00 (−0.03 to 0.03) were null. DXA FMI results (Table 5) were similar to BMI z score results (Table 4).

We did not observe different effects among offspring of overweight or obese (BMI ≥25) versus normal-weight mothers (interaction P value = .88), or boys versus girls (interaction P value = .25), or children of white versus African American versus other race and/or ethnicity (interaction P value = .12) (Table 4). In addition, including child beverage intake in the models did not appreciably attenuate the estimates (Tables 3–5). For example, the associations of maternal SSB intake and BMI z score (0.07 U; −0.01 to 0.15) and FMI (0.15 kg/m²; −0.01 to 0.30) were the same before and after adjusting for child SSB intake. In these models, each additional SSB serving per day in children was not associated with BMI z score (−0.04 U; −0.13 to 0.05) or total FMI (0.00 kg/m²; −0.17 to 0.17). In logistic regression, the adjusted OR for childhood BMI ≥85th percentile was the same before and after adjusting for child SSB intake (OR: 1.14; 0.95 to 1.36). In this model, including both maternal and child intake, each additional SSB serving per day in children was not associated with BMI ≥85th percentile (OR: 1.01; 0.81 to 1.25).

Furthermore, models stratified simultaneously according to maternal and child intake suggested that the associations were primarily driven by maternal intake, although some of the CIs were wide and overlapping (Fig 1). We examined maternal intake of at least versus <2 servings per day and child intake of at least versus <0.5 servings per week and their interaction. Compared with lower intakes in both mother and child, higher intake in children alone was associated with a higher BMI z score of 0.08 (95% CI: −0.06 to 0.22) whereas higher maternal intake was associated with BMI z score increments of 0.22 (−0.37 to 0.80) and 0.29 (0.01 to 0.56) among children with lower and higher intakes, respectively (Fig 1A).

![Table 2](image2.png)

**TABLE 2** Beverage Intake Among 1078 Mother-Child Pairs Participating in Project Viva, Total and According to Category of Intake of SSBs in the Second Trimester of Pregnancy

<table>
<thead>
<tr>
<th>Category of Maternal Second-Trimester SSB Intake (Servings/d)</th>
<th>Total</th>
<th>0 to &lt;0.5</th>
<th>0.5 to &lt;1</th>
<th>1 to &lt;2</th>
<th>2 to &lt;3</th>
<th>≥3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal first-trimester intake, servings/d</td>
<td>Mean (SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SSB</td>
<td>0.6 (0.9)</td>
<td>0.3 (0.5)</td>
<td>0.7 (0.8)</td>
<td>1.1 (1.0)</td>
<td>1.8 (1.2)</td>
<td>2.6 (1.6)</td>
</tr>
<tr>
<td>Sugary soda</td>
<td>0.3 (0.6)</td>
<td>0.2 (0.3)</td>
<td>0.4 (0.5)</td>
<td>0.7 (0.8)</td>
<td>0.9 (1.1)</td>
<td>1.3 (1.2)</td>
</tr>
<tr>
<td>Fruit drinks</td>
<td>0.3 (0.6)</td>
<td>0.2 (0.3)</td>
<td>0.4 (0.6)</td>
<td>0.4 (0.5)</td>
<td>0.8 (1.0)</td>
<td>1.4 (1.3)</td>
</tr>
<tr>
<td>Juice</td>
<td>1.3 (1.0)</td>
<td>1.2 (0.9)</td>
<td>1.5 (1.0)</td>
<td>1.4 (1.1)</td>
<td>1.7 (1.3)</td>
<td>2.1 (1.7)</td>
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<tr>
<td>Diet soda</td>
<td>0.2 (0.5)</td>
<td>0.2 (0.5)</td>
<td>0.2 (0.5)</td>
<td>0.2 (0.4)</td>
<td>0.3 (0.5)</td>
<td>0.3 (0.8)</td>
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<tr>
<td>Water</td>
<td>3.8 (1.9)</td>
<td>4.1 (1.8)</td>
<td>3.8 (1.8)</td>
<td>3.4 (2.0)</td>
<td>3.2 (1.9)</td>
<td>2.6 (2.0)</td>
</tr>
<tr>
<td>Maternal second-trimester intake, servings/d</td>
<td>Mean (SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SSB</td>
<td>0.6 (0.9)</td>
<td>0.1 (0.1)</td>
<td>0.7 (0.2)</td>
<td>1.3 (0.2)</td>
<td>2.5 (0.3)</td>
<td>4.1 (0.9)</td>
</tr>
<tr>
<td>Sugary soda</td>
<td>0.3 (0.5)</td>
<td>0.1 (0.1)</td>
<td>0.4 (0.2)</td>
<td>0.7 (0.4)</td>
<td>0.9 (1.0)</td>
<td>1.8 (1.3)</td>
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<tr>
<td>Fruit drinks</td>
<td>0.3 (0.6)</td>
<td>0.1 (0.1)</td>
<td>0.3 (0.2)</td>
<td>0.6 (0.4)</td>
<td>1.6 (1.1)</td>
<td>2.4 (1.3)</td>
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<tr>
<td>Juice</td>
<td>1.4 (1.1)</td>
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<td>1.4 (1.0)</td>
<td>1.7 (1.4)</td>
<td>2.0 (1.5)</td>
<td>2.3 (1.7)</td>
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<tr>
<td>Diet soda</td>
<td>0.1 (0.5)</td>
<td>0.1 (0.4)</td>
<td>0.2 (0.6)</td>
<td>0.1 (0.4)</td>
<td>0.1 (0.3)</td>
<td>0.2 (0.5)</td>
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<tr>
<td>Water</td>
<td>4.5 (2.2)</td>
<td>4.8 (2.2)</td>
<td>4.6 (2.0)</td>
<td>4.0 (2.3)</td>
<td>3.9 (2.2)</td>
<td>3.2 (2.3)</td>
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<tr>
<td>Midchildhood intake, servings/wk</td>
<td>Mean (SD)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SSB</td>
<td>2.6 (4.7)</td>
<td>2.1 (4.4)</td>
<td>2.4 (4.0)</td>
<td>3.7 (5.0)</td>
<td>5.0 (7.7)</td>
<td>3.5 (5.6)</td>
</tr>
<tr>
<td>Sugary soda</td>
<td>0.5 (1.2)</td>
<td>0.4 (0.9)</td>
<td>0.6 (1.6)</td>
<td>0.8 (1.3)</td>
<td>0.7 (1.1)</td>
<td>1.1 (1.8)</td>
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<tr>
<td>Fruit drinks</td>
<td>2.1 (4.2)</td>
<td>1.7 (4.0)</td>
<td>1.8 (5.0)</td>
<td>2.9 (4.7)</td>
<td>4.3 (7.4)</td>
<td>2.5 (3.0)</td>
</tr>
<tr>
<td>Juice</td>
<td>7.7 (8.0)</td>
<td>7.4 (7.9)</td>
<td>7.3 (7.9)</td>
<td>8.3 (8.0)</td>
<td>10.2 (9.3)</td>
<td>9.2 (8.0)</td>
</tr>
<tr>
<td>Diet soda</td>
<td>0.3 (1.7)</td>
<td>0.3 (1.4)</td>
<td>0.5 (2.7)</td>
<td>0.3 (0.9)</td>
<td>0.3 (1.2)</td>
<td>0.4 (1.5)</td>
</tr>
<tr>
<td>Water</td>
<td>18.1 (10.8)</td>
<td>17.9 (10.7)</td>
<td>18.6 (10.4)</td>
<td>18.2 (11.8)</td>
<td>17.8 (10.2)</td>
<td>19.4 (12.6)</td>
</tr>
</tbody>
</table>

![Table 3](image3.png)

**TABLE 3** Associations of Second-Trimester Intake of SSBs With Adiposity Outcomes in Midchildhood

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Unadjusted</th>
<th>Adjusted</th>
<th>Adjusted and Child</th>
</tr>
</thead>
<tbody>
<tr>
<td>Difference in Outcome (95% CI) Per 1 Serving/d Increment of Second-Trimester SSB Intake</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI z score</td>
<td>0.15 (0.07 to 0.23)</td>
<td>0.07 (−0.01 to 0.15)</td>
<td>0.07 (−0.01 to 0.15)</td>
</tr>
<tr>
<td>DXA FMI, kg/m²</td>
<td>0.31 (0.15 to 0.47)</td>
<td>0.15 (−0.01 to 0.30)</td>
<td>0.15 (−0.01 to 0.30)</td>
</tr>
<tr>
<td>SS + TR, mm</td>
<td>1.76 (0.94 to 2.59)</td>
<td>0.85 (0.06 to 1.64)</td>
<td>0.85 (0.06 to 1.64)</td>
</tr>
<tr>
<td>SS:TR</td>
<td>2.12 (0.65 to 3.60)</td>
<td>1.28 (−0.22 to 2.77)</td>
<td>1.25 (−0.25 to 2.75)</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>1.47 (0.79 to 2.15)</td>
<td>0.65 (0.01 to 1.28)</td>
<td>0.64 (0.00 to 1.27)</td>
</tr>
</tbody>
</table>

Data are from 1078 mother-child pairs participating in Project Viva. SS:TR models are additionally adjusted for BMI z score.

* Adjusted for maternal age, race and/or ethnicity, education, smoking, parity, and prepregnancy BMI; household income; and child age and sex.
In secondary analyses, first-trimester sugary beverage intake was not associated with any of the midchildhood outcomes (data not shown). However, change in intake from first to second trimester was associated with all of the outcomes, with effect estimates similar to those for the second trimester alone. For example, adjusted estimates (95% CIs) for each increase in 1 serving per day of SSBs were 0.07 (0.00 to 0.15) for childhood BMI z score and 0.19 (0.04 to 0.35) for FMI.

In addition, in substitution analyses, we found that replacing maternal second-trimester SSBs with water, 100% fruit juice, or milk had a greater beneficial effect on child BMI z score (−0.07 [−0.15 to 0.01], −0.08 [−0.19 to 0.03], or −0.10 [−0.19 to −0.01]) per increment of 1 serving per day, respectively) than did replacing SSBs with diet soda (−0.02 [−0.16 to 0.13]).
In this prospective prebirth cohort study, school-aged children of mothers who consumed more sugary beverages in midpregnancy had higher levels of adiposity, measured by BMI, skinfold thicknesses, and the reference standard DXA. After adjustment for multiple covariates, each additional serving of a sugary beverage consumed by a mother during the second trimester was associated with an additional 0.15 kg/m² (95% CI: −0.01 to 0.30) of fat mass. The associations were robust to adjustment for many confounding variables, were independent of the children’s beverage intake, and did not differ by child sex or race and/or ethnicity. We did not find associations with intakes of diet soda or water, or with any first-trimester beverage consumption. These findings suggest that efforts to limit SSB consumption once women become pregnant could help stem the tide of childhood obesity.

The paradigm of developmental origins of health and disease posits that cues during early, plastic phases of human development can have lifelong consequences for obesity and chronic disease. The associations were robust to adjustment for many confounding variables, were independent of the children’s beverage intake, and did not differ by child sex or race and/or ethnicity. We did not find associations with intakes of diet soda or water, or with any first-trimester beverage consumption. These findings suggest that efforts to limit SSB consumption once women become pregnant could help stem the tide of childhood obesity.

The paradigm of developmental origins of health and disease posits that cues during early, plastic phases of human development can have lifelong consequences for obesity and chronic disease. Because such cues can determine a trajectory of long-term health, and because plasticity wanes with age, interventions to alter health trajectories become more difficult as fetuses become children and children become adults. Given the combination of physiologic, behavioral, and social barriers, weight control after the onset of obesity is particularly challenging, implying that early prevention is paramount.

Maternal diet during pregnancy, as 1 element of the “fetal supply line,” may supply sufficient developmental cues to offer effective prevention of obesity in the offspring. The few animal experiments that address beverage or added sugar consumption in pregnancy provide support for our observations, but they may not directly translate to humans. In children and nonpregnant adults, higher consumption of sugary beverages consistently predicts excess weight gain, and randomized trials suggest that reducing their intake can at least modestly contribute to prevention or treatment of obesity.

In the United States, in an observational analysis of 249 infants whose mothers took part in a randomized controlled trial...
of moderating gestational weight gain, Phelan et al\textsuperscript{53} found that a greater percent of energy from sweets consumed in early pregnancy predicted higher weight for age among the 6-month-old infants of overweight or obese mothers ($N = 121$) but not among infants of normal weight mothers ($N = 128$). Among 66 mother-infant pairs of Hispanic ethnicity in Texas, Watt et al\textsuperscript{54} found that maternal sugary beverage intake was directly associated with weight for length exceeding the 85\textsuperscript{th} percentile during infancy. However, in a much larger study of Canadian mother-infant pairs, Azad et al\textsuperscript{55} found no association of intake of sugary beverages in late pregnancy with risk of overweight at 1 year of age. In comparison with those 3 studies, the outcomes in our study were in midchildhood, when BMI more strongly predicts adverse adult outcomes.\textsuperscript{12,56} We examined substitution as well as addition effects, and we employed direct measures of adiposity in addition to height and weight.

One key question with any study of maternal behavior and offspring health is the extent to which the same behavior in the child explains the findings. Maternal beverage intake could be related to child intake via 3 pathways. First, in the postnatal period, mothers provide food for their children and serve as role models for behaviors.\textsuperscript{57} Second, an inherited genetic predilection for certain beverages could exist.\textsuperscript{58} Third, maternal intake in pregnancy could program child’s intake, perhaps via mechanisms related to “fuel-mediated teratogenesis.”\textsuperscript{59} Distinguishing among these 3 possibilities is difficult in a single study. Nevertheless, our findings from stratified analyses that indicate that maternal intake was more strongly related to the outcomes than was child intake lend credence to the hypothesis that the observed effects are due to prenatal programming of susceptibility to obesity. In addition, we observed effects for second-trimester beverage intake, when fetal fat accumulation is accelerating, but not for intakes earlier in pregnancy.

Some evidence suggests that artificially sweetened beverages (even those without calories) can lead to increased consumption of food and therefore excess weight gain that is intermediate between unsweetened beverages and SSBs.\textsuperscript{60} In both the Norwegian and Danish cohorts, higher consumption of artificially sweetened beverages was associated with increased risk of preterm birth,\textsuperscript{67,62} and in the Canadian cohort, with risk of overweight at 1 year of age.\textsuperscript{55} However, we did not find that maternal diet soda intake was associated with offspring adiposity.

Strengths of this study include prospective data collection since early pregnancy; robust dietary assessment at 2 points in pregnancy; availability of numerous covariates to address confounding, mediation, and moderation; research-standard adiposity outcomes; and a sample size that allowed precise estimates of effect, at least for continuous outcomes. Limitations include self-report of diet, but resulting misclassification may well be nondifferential leading to bias toward the null, a conservative bias. We did not measure beverage intake in exactly the same way in mothers during pregnancy and in their children, but there was sufficient overlap for consistency, and the 2 were moderately correlated. Loss to follow-up, although regrettable, is common in cohort studies in early life. We observed some differences in baseline covariates between participants and those lost to follow-up, but adjustment for those variables did not alter the conclusions. In addition, we did not observe differences in baseline maternal beverage intake. These factors argue against selection bias as a major threat to validity of the findings.

CONCLUSIONS

Prevention strategies at the earliest stages of human development, including before birth, hold promise for prevention of obesity and noncommunicable diseases across the life course.\textsuperscript{57} In this study, we found that mothers who consumed more sugary beverages in midpregnancy had children with greater levels of adiposity. Women may be more amenable to behavior change when they are pregnant than when they are not.\textsuperscript{63} Combined with our results, this observation provides impetus to examine the long-term effects of interventions to reduce sugary beverage intake during pregnancy. In the United States, reductions in overall sugary beverage intake over the past decade, combined with hopeful results from some randomized trials of behavior change in pregnant women,\textsuperscript{62,63} make efforts to reduce sugary beverages in pregnancy a realistic goal. In low- and middle-income countries, however, where sugary beverage intake continues to rise along with the rapid emergence of obesity, and resources are lacking for individual-level behavior change interventions during gestation, the challenges are great.

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ABBREVIATIONS

CI: confidence interval
DXA: dual-energy radiograph absorptiometry
FFQ: food frequency questionnaire
FMI: fat mass index
OR: odds ratio
SS: subscapular skinfold thickness
SSB: sugar-sweetened beverage
TR: triceps skinfold thickness
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**Beverage Intake During Pregnancy and Childhood Adiposity**

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