Direction of Associations between Added Sugar Intake in Early Childhood and Body Mass Index at Age 7 Years May Depend on Intake Levels\textsuperscript{1,2}

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Abstract

Dietary factors, especially during early childhood, have been discussed as potentially critical for the development of childhood overweight. This study evaluated associations between added sugar intake during early childhood and BMI and body fat at age 7 y. Analysis was based on data from 216 participants of the Dortmund Nutritional and Anthropometric Longitudinally Designed (DONALD) Study. Life-course plots were constructed to evaluate the association between added sugar intake at different ages (0.5, 1, 1.5, and 2 y) and BMI SD score (BMI-SDS) and % body fat (%BF) at age 7 y. Multivariable analyses were performed for the periods identified as critical for later BMI and body fat. Added sugar intake at age 1 y and the change in intake levels during the second year of life emerged as potentially critical. At age 1 y, a higher total added sugar intake was related to a lower BMI-SDS at age 7 y \((\text{adjusted } \beta \pm \text{SE} = -0.116 \pm 0.057 \text{ BMI-SDS/percent energy (%En) added sugar}; P = 0.04)\). Conversely, an increase in total added sugar in the second year of life \((\Delta \%\text{En between age 1 and 2 y})\) tended to be associated with a higher BMI-SDS \((\text{adjusted } \beta \pm \text{SE} = 0.074 \pm 0.043 \text{ BMI-SDS/}\Delta\%\text{En added sugar}; P = 0.09)\). No associations were observed with %BF. In conclusion, added sugar intake at low intake levels during early childhood does not appear to be critical for BMI and body fat at age 7 y. However, detrimental effects on BMI development may emerge when added sugar intakes are increased to higher levels. \textit{J. Nutr.} \textbf{141}: 1348–1354, 2011.

Introduction

The prevalence of overweight and obese children is increasing worldwide and it is estimated that its prevalence will have doubled between 1990 and 2010 (1). Besides a lack of physical activity, dietary factors contributing to an energy imbalance are considered critical for the development of later overweight and obesity. Because added sugar intake has increased over the past decades among children and adolescents in Europe and the US (2,3), added sugar has been proposed as one of these dietary contributors (3). Mechanisms linking added sugar intake to body composition include the postprandial rises in glucose and insulin levels induced by many foods rich in added sugar and the contribution of added sugar intake to a higher energy density of the meals (4). Sugar-sweetened beverages (SSB)\textsuperscript{7} in particular are considered to contribute to additional energy intake due to a less accurate energy compensation after ingestion of liquids compared with solids (5,6) and a lower and shorter feeling of satiety after ingestion of sugar in liquid form (7).

Most prospective studies to date have focused on the relevance of SSB consumption yielding somewhat inconsistent findings: Two meta-analyses (8,9) revealed an increased obesity risk for children and adolescents, whereas another meta-analysis (10) reported a risk close to zero. Some of the more recently published cohort studies have confirmed a risk-increasing effect of SSB consumption (11–15); however, other studies reported no associations or relations confined to subgroups (16–19). Intervention studies suggest that a reduction in SSB consumption may be beneficial primarily for children and adolescents at high risk for obesity (e.g. with familial predisposition to obesity) (20–22). Controversial findings were also reported from 4 prospective studies investigating sugar intake in relation to body composition in children and adolescents (23–25,26).

Concerning the development of childhood overweight and obesity, the existence of a vulnerable window of the first 2 y of life has been discussed (27). It has furthermore been proposed that early exposure to sugar may predispose to later preference for that taste (28). Hence, intake of added sugar during early life could be particularly critical for later body composition. We thus examined whether certain time points or periods of added sugar intake in infancy and early childhood (0.5, 1, 1.5 or 2 y) may be particularly relevant for BMI and body fat percentage (%BF)
levels at age 7 y and, based on these results, whether added sugar intake from distinct sources (sweets, beverages, and other sources) could be responsible for potential associations between early added sugar intake and later BMI and body fat.

Methods

Study population. The Dortmund Nutritional and Anthropometric Longitudinally Designed (DONALD) Study is an ongoing open cohort study conducted at the Research Institute of Child Nutrition. Details of the study design have been published elsewhere (29). Briefly, since 1985, detailed information concerning diet, development, growth, and metabolism between infancy and adulthood has been collected from over 1200 participants. Every year, infants are newly recruited and first examined at the age of 3 mo. Each child is scheduled to return for 3 more visits in the first year, 2 in the second, and then annually until early adulthood. The noninvasive assessments include the completion of a 3-d weighed dietary record, anthropometric measurements, interviews on lifestyle, and a medical examination. The study was approved by the Ethics Committee of the University of Bonn and all examinations are performed with parental written consent.

The inclusion criteria for this analysis were fulfilled by 368 term (37-42 wk) singletons with a birth weight > 2500 g who had a minimum of 2 anthropometric measurements at both age 0.5 and 7 y. Among these, 223 children had complete, plausible (30), 3-d weighed dietary records at 0.5, 1, 1.5, and 2 y of age. Of these, 216 children (48.6% female) had complete information on potential confounders, i.e. socioeconomic characteristics.

Nutritional assessment. Dietary intake is assessed by using 3-d weighed dietary records. The parents of each child are instructed by trained dieticians to weigh and record all foods and beverages consumed, including leftovers, to the nearest 1 g over 3 consecutive days. With regard to breastfeeding, test weighing is performed, i.e. the intake of breast milk is assessed by weighing the infant before and after each meal to the nearest 10 g with an infant-weighing scale (Soehnle multina 8300). To correct for the systematic error in test-weighing due to insensible water loss, 5% was added to the test-weighing results (31). Semiquantitative recording with household measures (e.g. number of spoons) is allowed when exact weighing is not possible. Information on recipes or the types and brands of food items is also requested and at the end of the 3-d record period, a dietician visits the family and checks the record for completeness and accuracy. The dietary records are analyzed by using the continuously updated in-house nutrient database LEBTAB (32), which incorporates information from standard nutrient tables (e.g. mature human milk), product labels (e.g. most infant or follow-on formula), or recipe simulation based on the labeled ingredients and nutrients (e.g. commercial weaning foods).

Data on total daily energy (kJ/d) and nutrient intakes (g/d) were derived for each participant from the mean of the 3 d of recording. Total energy intake was used to exclude potentially implausible records by relating it to the basal metabolic rate (33) applying age- and sex-specific cutoffs (30).

Added sugar intake. Added sugars are sugars and syrups that are added to foods or beverages during processing or preparation at home and/or by the food industry (34). In LEBTAB, the following foods were defined as added sugars: white sugar, brown sugar, raw sugar, corn syrup, corn-syrup solids, high-fructose corn syrup, malt syrup, maple syrup, pancake syrup, fructose sweetener, liquid fructose, honey, molasses, anhydrous dextrose, and crystal dextrose. Fruit syrups commonly used as sweeteners in Germany were also considered. Conversely, naturally occurring sugars such as lactose in milk/formula or fructose in fruits were not included. The sugar content of maltodextrin was defined as 50% of its carbohydrate content, because maltodextrin consists of a mixture of monomers, dimers, oligomers, and polymers of glucose. Sugar substitutes were excluded from the definition because of their low metabolic effect. Added sugar in medicine was neglected.

In this study, 3 subgroups of added sugar intakes were also examined: added sugar from beverages (defined as added sugar from regular and diet soft drinks, and fruit juices), added sugar from sweets (defined as added sugar from candy, chocolate, jam, and ice cream), and added sugar from other sources (i.e. the remainder of food sources, e.g. breakfast cereals, pastries, milk, and milk products). Because added sugar intake from beverages and added sugar intake from sweets are low during the first 2 y of life, we combined these 2 groups to “added sugar from beverages and sweets” for analysis.

Anthropometric measurements. At each visit children are measured by trained and regularly monitored nurses according to standard procedures (35). The children are dressed in underwear only and are barefoot. In children until 2 y of age, recumbent length is measured using a Harpenden stadiometer (Holtain) and from the age of 2 y onwards, standing height is measured with a digital stadiometer, each to the nearest 0.1 cm. Body weight is assessed to the nearest 100 g with the use of a supine infant weighing scale (PS 15; Mettler) or an electronic scale (753 E; Seca) for participants in the standing position. Skinfold thicknesses are measured on the right side of the body at the biceps and triceps and subscapular and suprailiac sites to the nearest 0.1 mm with a Holttain caliper.

Sex- and age-independent BMI SD scores (BMI-SDS) were calculated using the German national reference data (36). Overweight was defined according to the International Obesity Task Force BMI cutoffs for children, which correspond to an adult BMI of 25 (37). %BF was calculated using data from the 4 skinfolds (38) and excess body fat at age 7 y was defined according to McCarthy et al. (39).

Potentially confounding factors. On their child’s admission to the study, parents are interviewed about familial characteristics (i.e. parental education, smoking in the household) and are weighed and measured using the same equipment as for the children. Information on the child’s birth characteristics are abstracted from the “Mutterpass,” a standardized document given to all pregnant women in Germany.

Statistical analysis. SAS procedures (SAS, version 9.13; SAS Institute) were used for data analysis. P < 0.05 was considered significant. Because analysis indicated no interactions of sex with the association between added sugar intake and BMI or body fat, data from girls and boys were pooled for all further calculations.

Life-course plots were constructed to evaluate which age or time periods of added sugar intake in early life might be most important with regard to BMI-SDS and %BF at age 7 y. This approach, which was described by Cole (40), allows to simultaneously consider repeated measures of an explanatory variable (e.g. added sugar intake in early life) in relation to an outcome (e.g. BMI-SDS or %BF at age 7 y). In this analysis, the intakes of total added sugar and their sources at the ages of 0.5, 1, 1.5, and 2 y were corrected for total energy intake by expressing them as residuals (41) and were standardized (mean = 0, SD = 1) by age and sex to facilitate comparability. These values were entered into multivariable linear regression models as independent variables with BMI-SDS and %BF at 7 y as the outcomes (adjusted for sex in case of %BF). The resulting regression coefficients were then plotted against age and both their values (representing the strength of the relation at a distinct time point) and their changes (representing the association between outcome and change in the explanatory variables over the corresponding time interval) were evaluated to identify potentially sensitive time periods or points of added sugar intake with respect to later BMI and body fat.

Subsequent analysis concentrated on the critical ages and periods identified by the life-course plot approach only, i.e. intake at age 1 y and change in intake between the first and second year of life. To convey the sense of the actual added sugar intake, a constant was added to the residuals (age 1 y) and studentized residuals (difference between age 1 and 2 y) as described by Willett (42) and the resulting values were converted to percentages of energy intake.

Thereafter, the obtained percentages of energy intake from total added sugar, added sugar from beverages and sweets, and added sugar from other sources, each at age 1 and as change in the second year of life (intake at age 2 minus intake at age 1), were included as independent variables in linear regression models with BMI-SDS and %BF at 7 y as the outcomes.

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In a further step, potentially confounding factors were considered as covariates. These included sex, birth characteristics [gestational age, birth year, anthropometric characteristics, breastfeeding (breastfeeding \(\geq 4\) mo, yes/no)], parental characteristics [maternal age at birth (43,44), maternal overweight (BMI \(\geq 25\), yes/no), maternal and paternal educational attainment (at least 12 y schooling, yes/no), and smoking in the household (yes/no)], and dietary animal protein intake. Only variables that modified the regression coefficients outlined above in the unadjusted models by \(\geq 10\%\) (43) or that had a significant, independent effect on the outcome variable were included in multivariable-adjusted models.

**Results**

The present study included 111 boys and 105 girls (Table 1). More than 80% of the children were fully breastfed for \(>4\) mo. Mean BMI-SDS of the children of our sample at birth was slightly higher than that of the German reference population. At age 7 y, BMI-SDS was close to 0, with a median BMI of 15 and the percentage of children with excess body fat and overweight was \(\sim 14\%\). One-fourth of the children had an overweight mother and \(>60\%\) had parents with a high educational level.

Daily energy intake at age 1 y was \(\sim 3400\) kJ/d and increased by 615 kJ/d until age 2 y (Table 2). At age 1 y, \(-4\%\) of the daily energy intake was provided by total added sugar, the minor part derived from beverages and sweets and the major part from other sources (at age 0.5 y, \(\sim 1\%\) of energy intake was provided by added sugar; data not shown). Between age 1 and 2 y, added sugar from sweets and beverages increased by \(-3\%\), whereas added sugar from other sources increased only minimally; hence, total added sugar increased by \(-4\%\) of energy intake. Common sources of added sugar between age 1 and 2 y were dairy products, sweet spreads, cakes and pastries, and sweets.

The life-course plot analysis consistently indicated that higher intakes of total added sugar and added sugar from beverages and sweets at age 1 y were related to a lower BMI-SDS at age 7 y (i.e., negative regression coefficients; \(P < 0.05\) (Fig. 1)). Between age 1 and 2 y, there was a clear switch in signs of the regression coefficients (46), i.e., increases in added sugar intakes during this time period were related to higher BMI-SDS levels at age 7 y. There were no associations between added sugar intakes (total or from beverages and sweets) at specific time points or periods and %BF at age 7 y. On the basis of the results of the life-course plots, subsequent multivariable analyses concentrated on relations between added sugar intake at age 1 y and the change in added sugar intake between age 1 and 2 y in relation to BMI and %BF at age 7 y.

Covariates that were significantly associated with BMI-SDS or %BF at age 7 y included sex, animal protein intake, paternal educational attainment, and maternal overweight status and were hence considered as potentially confounding factors. A higher total added sugar intake at age 1 y was related to lower BMI-SDS levels at 7 y after adjustment for potentially confounding factors (\(P = 0.04\); model 2) (Table 3). By contrast, an increase in total added sugar intake in the second year of life tended to be related to a higher BMI-SDS at age 7 y (\(P = 0.08\); model 2). Intake of total added sugar at age 1 y or change in added sugar intake in the second year of life were not related to %BF at age 7 y (\(P > 0.05\)). For added sugar from beverages and sweets, a higher intake at age 1 y was associated with a lower BMI-SDS at age 7 y (\(P = 0.02\); model 2) and also tended to be associated with a lower %BF at age 7 y (\(P = 0.1\); model 2). However, the change in intake of added sugar from beverages and sweets between age 1 and 2 y was not related to BMI-SDS or %BF at age 7 y (\(P > 0.05\)). Furthermore, there were no associations between the intake of added sugars from other sources at age 1 y or the change from age 1 to 2 y and BMI or body fat at age 7 y (\(P > 0.05\)).

Similar results were obtained when adjusting for consumption of breast milk (yes/no) at each time point (0.5, 1, and 1.5 y) or for the amount of energy provided by breast milk (data not shown).

**Discussion**

The present study provides new evidence for longitudinal associations between added sugar intake in the first 2 y of life and BMI at age 7 y. At low intake levels, as observed at age 1 y in this study, added sugar intake (particularly from beverages and sweets) was inversely related to BMI-SDS levels at age 7 y. By contrast, increases of total added sugar intakes, as seen during the second year of life in this cohort, may have a potential to adversely affect later BMI-SDS. In the present study, no associations were discernable between added sugar intake during early childhood and %BF at age 7 y.

Previous prospective studies in children and adolescents have reported mixed findings for the association between added sugar or sucrose intake and measures of body composition. Similar to our findings, a study conducted among 3- to 4-y-old U.S. children reported that sucrose intake was related to lower BMI levels 4 y later (26). In the Special Turku Coronary Risk Factor Intervention Project, children with consistently high sucrose intakes between 13 mo and 9 y of age weighed more in the first year of the study, but from age 4 to 9 y, their weight and BMI was lower than that of children with average or low sucrose

**Table 1** Birth, anthropometric, and parental characteristics of 216 participants of the DONALD Study

<table>
<thead>
<tr>
<th>Variables</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female, %</td>
<td>48.6</td>
</tr>
<tr>
<td>Birth characteristics</td>
<td></td>
</tr>
<tr>
<td>Gestational age, wk</td>
<td>40 (39,41)</td>
</tr>
<tr>
<td>Birth weight, g</td>
<td>3470 (3220, 3800)</td>
</tr>
<tr>
<td>BMI at birth, kg/m²</td>
<td>12.9 (12.1, 13.7)</td>
</tr>
<tr>
<td>BMI-SDS at birth²</td>
<td>0.22 (−0.40, 0.91)</td>
</tr>
<tr>
<td>Fully breastfed ≥ 4 mo, %</td>
<td>81.9</td>
</tr>
<tr>
<td>Anthropometric characteristics at age 7 y</td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>15.5 (14.8, 17.1)</td>
</tr>
<tr>
<td>BMI-SDS³</td>
<td>−0.07 (−0.47, 0.71)</td>
</tr>
<tr>
<td>Overweight, %</td>
<td>14.4</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>16.6 (14.1, 20.3)</td>
</tr>
<tr>
<td>Excess body fat, %</td>
<td>14.4</td>
</tr>
<tr>
<td>Parental characteristics</td>
<td></td>
</tr>
<tr>
<td>Maternal age at birth, y</td>
<td>31 (29.33)</td>
</tr>
<tr>
<td>Overweight of the mother, %</td>
<td>24.4</td>
</tr>
<tr>
<td>High maternal educational level, %</td>
<td>62.0</td>
</tr>
<tr>
<td>High paternal educational level, %</td>
<td>65.1</td>
</tr>
<tr>
<td>Smoking in the household, %</td>
<td>23.6</td>
</tr>
</tbody>
</table>

1 Values are medians (Q1, Q3) or frequencies.
2 Calculated using the German sex- and age-specific percentiles by Kromeyer-Hauschild et al. (38).
3 Defined according to the International Obesity Task Force BMI cutoffs for children, which correspond to an adult BMI of 25 (37).
4 Estimated according to the equations of Deurenberg et al. (38).
5 Defined according to McCarthy et al. (39).
6 BMI ≥ 25 kg/m².
7 At least 12 y of education.
intakes (23). Conversely, in a group of overweight U.S. Latino youth, change in total or added sugar intake during the 2-y follow-up was not related to changes in BMI or total body, visceral, or subcutaneous fat (25). Similarly, in a previous analysis of carbohydrate quality in relation to the development of body composition between age 2 and 7 y in German children, changes in added sugar intake levels between 2 and 7 y were not related to concurrent changes in BMI or percentage body fat (24).

**Sugar as a slimming agent.** The moderate inverse relation between the intake of added sugar at age 1 y and BMI in later childhood is in accordance with metabolic and epidemiological studies in adults (4) and with cross-sectional studies on children (47,48) describing a lower BMI or risk of overweight among those with higher sugar intakes. Thus, sugar has been called a “slimming agent” (49). In fact, in a metabolic ward study of 20 healthy young men, weight gain by overeating carbohydrates and sucrose during 21 d was not higher than weight gain by overeating fat (50), presumably because conversion of glucose to fat is energetically costly and fecal energy loss is higher under overfeeding with carbohydrates. In addition, short-term metabolic ward studies have consistently shown a satiety-inducing effect of sugar, leading to a suppression of subsequent food intake (i.e. energy compensation) in both adults and children (51). In their review of the role of sugar intake in the regulation of short-term satiety and food intake, Anderson and Woodend (51) concluded that sugar intake appears to suppress food intake even when consumed in small quantities provided that the time periods elapsing between the meals is short. Thus, in diets comprising frequent meals, such as infant and toddler diets, even small amounts of added sugar may contribute to a lower energy intake and therefore, over the longer term, result in lower BMI. Nonetheless, our data also suggest that these associations may not be discernible when only minimal amounts of added sugar are consumed, as was the case at age 0.5 y.

**Sugar as a weight-gaining nutrient.** Sugar in beverages seems to have a less pronounced effect on onset and duration of satiety among children than sugar intake in solid form (7). Previous studies led to inconsistent results, probably because other variables intervened: consistency, temperature, taste and energy density of the food, psychological factors, physical activity of the individual, or day time (52). Nonetheless, 2 recent meta-analyses suggest that a higher SSB consumption may confer an increased risk for overweight or weight gain in children (8,9); however, both reported small effect sizes and another meta-analysis judged the effect to be close to zero (10). It has been argued that this discrepancy may result from the fact that the association between SSB and BMI in children and adolescents may be discernible only at high levels of intake or in susceptible individuals at higher risk of overweight (53). In addition, Cecil et al. (54) were able to show that the accuracy of energy compensation is age dependent and performed more accurately in young (6–7 y) than in older children (8–9 y).

In the present study in infants and toddlers, SSB made a very small contribution to added sugar intake and could thus not be analyzed as its own food group. Nonetheless, we observed that increases in total added sugar intake during the second year of life may be detrimental for the development of childhood BMI. This finding is novel but should be interpreted with caution, because we observed only a tendency. It could, nonetheless, suggest that rapid increases in added sugar intake may be accompanied by less precise energy compensation, thus contributing to long-term weight gains. Alternatively, consumption of added sugar could be indicative of a general behavioral pattern (55). Under this alternative hypothesis, increases in added sugar intake are not regarded as the physiologically relevant component for subsequent increases in BMI but instead resemble a surrogate marker for other behavioral changes contributing to increases in BMI. In this scenario, public health initiatives directed at the reduction of added sugar intake (56) may improve nutritional quality (57) but not reduce obesity rates.

**Strength and weaknesses of the study.** A clear strength of our analyses lies in the carefully collected, repeated data on growth and the availability of data on several possible confounders such as parental characteristics and other dietary factors. Weighed dietary records may provide limited information about habitual diet when collected for only 3 d; on the other hand, 3–4 d were found to be acceptably accurate for classification of infants and toddlers into groups of macronutrient intake because of the fairly uniform diet of that age group (58).

Our study has several limitations. First, it should be stressed that the socio-economic status (SES) of the DONALD population is relatively high (59), as reflected by the educational level and maternal age at birth. Generally, higher SES levels are related to both lower intakes of added sugar (43,60) and to a lower risk of childhood overweight (61). Indeed, in our study population, the intake of added sugar was found to be substantially lower than in British (62) or U.S. children (63) at comparable...
ages. We aimed to minimize a statistical bias by adjustment for SES, but we cannot preclude that the higher SES and the comparably low variability in BMI-SDS and body fat in our sample may have contributed to an underestimation of the “true” relations. It is also possible that the relative homogeneity of the healthy DONALD sample means that extremes of diet or behavior are not represented. Although the DONALD Study sample is unrepresentative, the dietary data in general are similar to the nationwide German Consumption Study among infants and toddlers, which reported almost identical energy intake levels, slightly higher carbohydrate intakes, and slightly lower protein intakes at age 1 y (64). Furthermore, other unmeasured influences caused by selection of the study population may have interfered. On the other hand, the homogeneity of our sample might have reduced our vulnerability to residual confounding by behavioral or SES factors and nonrepresentation is less relevant for longitudinal analyses and internal validity. Another main limitation of this study was the fact that the body fatness was estimated from skinfold thickness measurements, which are known to be more susceptible to measurement error than specialized research-based techniques, such as DXA, and may underestimate body fatness (65). However, intra- and inter-observer variability are notably reduced when measurements are conducted by trained personnel, as was the case in our study (29).

In conclusion, the present study provides new evidence on the longer term relevance of added sugar intake in the first 2 y of life for body BMI during childhood. At low intake levels, added sugar intake during early childhood may stimulate previously documented satiety mechanisms and thus contribute to lower BMI-SDS levels at age 1 y. However, our data also suggest that detrimental effects of added sugar intake on BMI development may emerge when added sugar intakes are increased to higher levels.

Acknowledgments
We thank Ruth Schaefer and Christa Chada for collecting and coding the dietary records and Sabine Twenhoeven, Birgit Holtermann, and Ute Kahrweg for carrying out the anthropometric measurements. A.E.B. and A.I. conceived the

FIGURE 1 Life-course plots of the multiple linear regression analysis with the BMI-SDS or %BF at age 7 y as the dependent variable and the respective intake of added sugar (standardized residuals by age and sex) as independent variable at different ages (adjusted for sex with %BF). Values are means and 95% CI, n = 216. *Different from zero, P < 0.05.
TABLE 3  Associations between energy intake from added sugar at age 1 y and change from age 1 to 2 y with BMI-SDS \(^1\) and %BF at age 7 y

<table>
<thead>
<tr>
<th>Intake level at age 1 y, %En</th>
<th>Total added sugar</th>
<th>Added sugar from beverages and sweets</th>
<th>Added sugar from other sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1 (^2)</td>
<td>(\beta \pm SD)</td>
<td>(P) Model (R^2)</td>
<td>(\beta \pm SD)</td>
</tr>
<tr>
<td>(-0.087 \pm 0.056)</td>
<td>0.1 (0.04)</td>
<td></td>
<td>(-0.211 \pm 0.102)</td>
</tr>
<tr>
<td>Model 2 (^3)</td>
<td>(-0.116 \pm 0.057)</td>
<td>0.04 (0.07)</td>
<td>(-0.250 \pm 0.103)</td>
</tr>
<tr>
<td>Change in intake level from age 1 to 2 y, (\Delta En)</td>
<td>|</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1 (^4)</td>
<td>(0.062 \pm 0.043)</td>
<td>0.1 (0.06)</td>
<td>(0.046 \pm 0.055)</td>
</tr>
<tr>
<td>Model 2 (^5)</td>
<td>(0.074 \pm 0.043)</td>
<td>0.09 (0.08)</td>
<td>(0.047 \pm 0.055)</td>
</tr>
<tr>
<td>Intake level at age 1 y, %En</td>
<td>|</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1 (^2)</td>
<td>(-0.008 \pm 0.015)</td>
<td>0.6 (0.15)</td>
<td>(-0.044 \pm 0.028)</td>
</tr>
<tr>
<td>Model 2 (^5)</td>
<td>(-0.014 \pm 0.015)</td>
<td>0.4 (0.22)</td>
<td>(-0.044 \pm 0.028)</td>
</tr>
<tr>
<td>Change in intake level from age 1 to 2 y, (\Delta En)</td>
<td>|</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 1 (^4)</td>
<td>(0.002 \pm 0.012)</td>
<td>0.8 (0.15)</td>
<td>(0.002 \pm 0.015)</td>
</tr>
<tr>
<td>Model 2 (^5)</td>
<td>(-0.004 \pm 0.015)</td>
<td>0.8 (0.21)</td>
<td>(-0.004 \pm 0.015)</td>
</tr>
</tbody>
</table>

1 Adjusted for total energy intake by residual method and converted to a percentage of energy as proposed by Willett et al. (42).
2 Model 1 adjusted for baseline characteristics and other nutritional variables: sex and animal protein intake at age 1 (residual).
3 Model 2 additionally adjusted for socioeconomic variables: maternal overweight and paternal education.
4 Model 1 adjusted for baseline characteristics and other nutritional variables: sex and intake from animal protein from age 1 to age 2 (Studentized residuals).
5 Model 2 additionally adjusted for socioeconomic variables: maternal overweight and paternal education. Models include only variables that modified the regression coefficients in the unadjusted models by $\pm 10\%$ or had a significant independent effect on the outcome.

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**Literature Cited**