Dietary Energy Intake Is Associated With Type 2 Diabetes Risk Markers in Children

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OBJECTIVE

Energy intake, energy density, and nutrient intakes are implicated in type 2 diabetes risk in adults, but little is known about their influence on emerging type 2 diabetes risk in childhood. We examined these associations in a multiethnic population of children.

RESEARCH DESIGN AND METHODS

This was a cross-sectional study of 2,017 children predominantly of white European, South Asian, and black African-Caribbean origin aged 9–10 years who had a detailed 24-h dietary recall and measurements of body composition and provided a fasting blood sample for measurements of plasma glucose, HbA_{1c}, and serum insulin; homeostasis model assessment of insulin resistance was also derived.

RESULTS

Energy intake was positively associated with insulin resistance. After the removal of 176 participants with implausible energy intakes (unlikely to be representative of habitual intake), energy intake was more strongly associated with insulin resistance and was also associated with glucose and fat mass index. Energy density was also positively associated with insulin resistance and fat mass index. However, in mutually adjusted analyses, the associations for energy intake remained while those for energy density became nonsignificant. Individual nutrient intakes showed no associations with type 2 diabetes risk markers.

CONCLUSIONS

Higher total energy intake was strongly associated with high levels of insulin resistance and may help to explain emerging type 2 diabetes risk in childhood. Studies are needed to establish whether reducing energy intake produces sustained favorable changes in insulin resistance and circulating glucose levels.

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Type 2 diabetes is a major global public health problem, requiring concerted preventive efforts (1). Diet appears to play an important role in the etiology of type 2 diabetes, although the importance of specific dietary components has not been completely resolved (2). In adults, diets with a high energy intake (3,4) and a high energy density (5) have been implicated in type 2 diabetes risk. Specific aspects of

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dietary nutrient intakes (including both macronutrients and micronutrients) have also been associated with increased diabetes risk (6,7).

Although there has been considerable concern about the emergence of type 2 diabetes in adolescence and childhood (8) and about the health implications of current childhood dietary patterns (9), very few studies have reported on the associations between dietary nutrient intakes and markers of emerging type 2 diabetes risk in childhood. Such studies may define the elements of diet important in the early stages of the development of type 2 diabetes risk and before confounding by adult lifestyle factors (for example, cigarette smoking and alcohol consumption) become important.

We therefore examined the associations between total energy intake, energy density, dietary nutrient intakes, and risk markers for type 2 diabetes in a large, cross-sectional, multiethnic population of 9–10-year-old U.K. children, predominantly of white European, South Asian, and black African-Caribbean origin. We also examined the extent to which the higher insulin resistance seen in the South Asian and black African-Caribbean children (10) could be explained by ethnic differences in nutrient intakes. Analyses were designed to take account of implausible estimates of energy intake, and to assess the extent to which associations between energy intake, insulin resistance, and glycemia could be explained by effects on body fat.

RESEARCH DESIGN AND METHODS

Participants

This investigation was based on the Child Heart And health Study in England (CHASE) (10), which examined markers of type 2 diabetes risk and their determinants in a multiethnic population of children aged 9–10 years. Balanced numbers of children of South Asian, black African-Caribbean, and white European origin were invited to take part, drawn from a stratified random sample of 200 primary schools in London, Birmingham, and Leicester. Ethical approval was provided by the relevant multicenter research ethics committee; parents or guardians provided informed written consent. Data were collected between October 2004 and February 2007. In the last 85 schools (visited between February 2006 and February 2007), dietary assessments were also made.

Dietary Assessment

Full details of the dietary assessment have been reported elsewhere (11). A single, structured 24-h recall was completed and included elements of the U.S. Department of Agriculture multiple pass method (12). Energy and nutrient intakes were calculated by the Medical Research Council Human Nutrition Research center (MRC-HNR) using an in-house food composition database (DINO). Energy density was calculated by dividing the reported total energy intake from food (kJ) by the total weight of food reported (g). Implausible dietary intakes were identified by comparing reported energy intake to estimated minimum and maximum energy requirements. These were calculated using an estimate of basal metabolic rates (BMRs) derived using the Schofield equations for boys and girls aged 3–10 years (13), as follows:

$$\text{Estimated BMR (boys)} = 22.706 \times \text{weight(kg)} + 504.3$$

$$\text{Estimated BMR (girls)} = 20.315 \times \text{weight(kg)} + 485.6$$

To define minimum and maximum limits of feasible energy expenditures, the estimated BMR was then multiplied by lower and upper physical activity levels (PALs) of 0.9 and 2.75, respectively (assuming an average PAL of 1.55) (14). Children with reported energy intakes below their minimum estimated energy requirement or above their maximum estimated energy requirement were classified as implausible reporters.

Physical Measurements and Blood Sampling

Participating children had measurements of height (using a portable stadiometer; CMS Instruments, London, U.K.), weight (Tanita, Tokyo, Japan), waist circumference, multiple skinfold thicknesses, and bioelectrical impedance measured with a Bodystat 1500 body composition analyzer (Bodystat Ltd., Isle of Man, U.K.). Fat-free mass was derived from bioelectrical impedance using a validated equation (15) and fat mass index calculated (kg/m^5), which is independent of height (16). Fat mass index from bioelectrical impedance was used as the principal marker of body fat as it provides valid measurements of body fat in this multiethnic population, in contrast with BMI, which yields biased results (16). Children provided fasting blood samples; serum insulin was measured using an ELISA method (17) and plasma glucose using the glucose oxidase method. HbA_{1c} was measured in whole blood by ion exchange high-performance liquid chromatography. The homeostasis model assessment (HOMA) equations were used to provide an estimate of insulin resistance (18).

Ethnicity and Socioeconomic Position

Ethnicity of the child was categorized using self-defined ethnicity for both parents or by using parental information on the ethnicity of the child. In a small number of participants for whom this information was not available (1%), child-defined place of origin of parents and grandparents was used instead. Socioeconomic position was coded from parental occupation using the U.K. National Statistics Socioeconomic Classification (NS-SEC) as previously described (19).

Statistical Methods

Statistical analyses were carried out using STATA/SE software (STATA/SE 12 for Windows; StataCorp LP, College Station, TX). Multilevel linear regression models were used to quantify the associations between dietary intake (expressed as per 1 SD increase) and type 2 diabetes risk markers, which were all log transformed. All analyses were adjusted for sex, age in quartiles, ethnicity (at the ethnic subgroup level), height (as a proxy for growth), day of week, and month as fixed-effects; school was fitted as a random-effect. Similar multilevel linear regression models were used to estimate ethnic differences in risk markers with additional adjustments for energy intake. Classical measurement error (CME command) was also used to allow for random measurement error in

energy intake in analyses to assess whether ethnic differences in energy intakes could explain ethnic differences in type 2 diabetes risk markers. Estimates for measurement error were based on a sample of repeat 24-h recalls collected in 60 participants after a median interval of 12 months from the initial assessment. In these models, school was fitted as a cluster variable to give robust standard errors as classical measurement error models programmed in STATA will not allow for random effects. Conventional levels of statistical significance ($P < 0.05$) were used in two-sided tests.

RESULTS

Among 3,679 children invited, 2,529 (69%) took part in the current study; participation rates were generally similar across the ethnic groups, although slightly lower in the black African-Caribbeans (66%). Among participants (one child was excluded who had type 1 diabetes), 2,337 children (92%) provided fasting blood samples; 24-h dietary recalls were completed for 2,017 children, mean age 10.0 years, 95% reference range 9.3–10.6 years, with 53% girls. Similar numbers of children of white European, black African-Caribbean, South Asian, and other ethnic origins were included (n = 506, 490, 528, and 493, respectively). The distribution of parental socioeconomic position included 27% in managerial/professional occupations, 26% in intermediate, 33% in routine/manual, 9% economically inactive, and 5% unclassified. The means and standard deviations of the nutrient intakes and type 2 diabetes risk markers of study participants are presented in Supplementary Table 1 for boys and girls separately and combined. After adjustment for covariates, girls had higher levels of insulin resistance and fat mass index and lower levels of fasting glucose; HbA_{1c} levels were similar in girls and boys. Boys had higher energy intake and energy density, and girls had higher polyunsaturated fat intake; intakes of other macronutrients were similar in boys and girls. Boys had higher vitamin B12 and iron intakes but once their higher total energy intakes were taken into account, these differences were not statistically significant.

Associations Between Dietary Intakes and Type 2 Diabetes Risk Markers and Adiposity

Associations between energy intake, energy density, and intakes of specific nutrients and insulin resistance, glycemia markers, and fat mass index are shown in Table 1, expressed as the difference in outcome per 1 SD increase in nutrient intakes, with adjustment for covariates. There was a positive association between energy intake and insulin resistance, but no further associations between energy density, macro- or micronutrient intakes, and insulin resistance or glycemia were observed, although a weak negative association with folate and fat mass index was apparent. After the exclusion of 176 participants with implausibly high (n = 18, 1%) or low (n = 158, 8%) intakes, total energy intake was strongly associated with higher insulin resistance, fasting glucose, and fat mass index; energy density was positively associated with insulin resistance and fat mass index (Table 2). There were no differences in the associations between dietary intakes and type 2 diabetes risk markers by ethnic group or sex (all P values for interaction > 0.05). As total energy intakes and energy density were correlated (r = 0.31, $P < 0.001$), the independent associations between energy intake, energy density, and type 2 diabetes risk markers were examined after mutual adjustment (Table 3). The associations between energy intake and insulin resistance, fasting glucose, and fat mass index were little affected by the addition of energy density (model 2a). In contrast, the associations between energy density, insulin resistance, and fat mass index were greatly attenuated and not statistically significant after adjustment for energy intake (model 2b). Additional adjustment for fat mass index (Table 3, model 3a) reduced the association between energy intake and insulin resistance by about half, although it remained statistically significant. The association for fasting glucose was reduced by about one-fifth, also remaining statistically significant. The associations between mean energy intakes (fifths) and insulin resistance are shown in Fig. 1*A* (all participants) and Fig. 1*B* (excluding participants with implausible energy intakes). These associations are clearly graded; similar patterns are also seen for glucose and fat mass index (data not presented).

Ethnic Differences in Type 2 Diabetes Risk Markers: Contribution of Energy Intake

Compared with white Europeans and excluding implausible reporters, energy intake was 110 kcal (95% CI 51–170) higher among South Asians ($P < 0.001$) and 45 kcal (95% CI −14 to 103) higher among black African-Caribbean children (P = 0.13). Additional adjustment for fat mass index had little effect on these differences, which were then 107 kcal (95% CI 48–166) higher for South Asians and 47 kcal (95% CI −11 to 104) higher for black African-Caribbeans. Ethnic differences in insulin resistance, HbA_{1c}, and fasting glucose and the effect of adjustment for energy intake on these differences are shown in Supplementary Table 2. As previously reported in the whole CHASE population (10), South Asian children had markedly higher insulin resistance, HbA_{1c}, and fasting glucose levels than white European children. After adjustment for differences in total energy intake (particularly taking account of measurement error in energy intake), the South Asian–white European differences in insulin resistance, HbA_{1c}, and glucose were reduced by between one-tenth and one-fifth. Black African-Caribbean children had a less consistent pattern of differences in this study subpopulation (slightly but nonsignificantly higher insulin resistance, higher HbA_{1c}, and slightly lower fasting glucose); these differences were little affected by adjustment for energy intake. Thus, adjusting for differences in energy intake does not appear to explain ethnic differences in diabetes risk markers. These results were not materially affected by including fat mass index in the models.

Sensitivity Analyses

In sensitivity analyses examining the associations between energy intake and type 2 diabetes risk markers using adjustment rather than exclusion of participants who reported energy intakes unlikely to be representative of habitual intake, the results were very similar to those obtained with the exclusion of these participants;

Table 1—Percentage differences in type 2 diabetes risk markers and fat mass index per 1 SD increase in dietary intake in all participants (2,017 children)

	Insulin resistance		HbA_{1c} (%)		Glucose (mmol/L)		Fat mass index (kg/m^5)	
Dietary intake	% change (95% CI)	*P* value*	% change (95% CI)	*P* value*	% change (95% CI)	*P* value*	% change (95% CI)	*P* value*
Energy intake (kcal)	4.40 (1.81–7.05)	<0.001	0.17 (−0.10 to 0.45)	0.22	0.26 (−0.05 to 0.58)	0.10	0.65 (−1.30 to 2.63)	0.52
Energy density (kJ/g)	1.89 (−0.64 to 4.47)	0.14	−0.09 (−0.36 to 0.19)	0.53	0.06 (−0.25 to 0.38)	0.70	0.66 (−1.28 to 2.64)	0.51
Fat (% energy)	−2.02 (−4.44 to 0.47)	0.11	−0.10 (−0.38 to 0.17)	0.46	−0.04 (−0.36 to 0.27)	0.79	−1.81 (−3.71 to 0.12)	0.07
Saturated fat (% energy)	−1.55 (−3.97 to 0.94)	0.22	−0.11 (−0.38 to 0.17)	0.45	0.05 (−0.26 to 0.37)	0.75	−0.77 (−2.67 to 1.17)	0.44
Monounsaturated fat (% energy)	−1.40 (−3.87 to 1.13)	0.27	−0.12 (−0.40 to 0.15)	0.38	0.03 (−0.29 to 0.35)	0.85	−1.53 (−3.44 to 0.42)	0.12
Polyunsaturated fat (% energy)	−0.01 (−2.51 to 2.55)	0.99	0.06 (−0.21 to 0.34)	0.65	0.07 (−0.25 to 0.38)	0.68	0.39 (−1.55 to 2.38)	0.69
Carbohydrate (% energy)	1.79 (−0.73 to 4.36)	0.16	0.14 (−0.14 to 0.41)	0.34	0.03 (−0.28 to 0.35)	0.83	0.52 (−1.41 to 2.49)	0.60
Sugars (% energy)	1.28 (−1.29 to 3.91)	0.33	0.02 (−0.26 to 0.30)	0.88	−0.04 (−0.36 to 0.28)	0.80	0.41 (−1.56 to 2.42)	0.69
Starch (% energy)	1.18 (−1.39 to 3.82)	0.37	0.10 (−0.18 to 0.38)	0.50	0.12 (−0.20 to 0.44)	0.45	0.42 (−1.55 to 2.44)	0.68
Nonstarch polysaccharides (g)	−1.11 (−3.97 to 1.84)	0.46	0.26 (−0.06 to 0.58)	0.11	−0.14 (−0.51 to 0.23)	0.46	0.99 (−1.28 to 3.31)	0.39
Protein (% energy)	−0.01 (−2.52 to 2.56)	0.99	−0.13 (−0.41 to 0.14)	0.34	0.02 (−0.30 to 0.34)	0.90	2.24 (0.26–4.25)	0.03
Vitamin B12 (μg)	−1.68 (−4.24 to 0.95)	0.21	−0.02 (−0.31 to 0.28)	0.91	−0.09 (−0.42 to 0.25)	0.61	−0.79 (−2.81 to 1.27)	0.45
Folate (μg)	−1.72 (−4.43 to 1.07)	0.23	0.27 (−0.04 to 0.58)	0.09	−0.26 (−0.60 to 0.09)	0.15	−2.54 (−4.62 to −0.41)	0.02
Vitamin C (mg)	0.84 (−1.72 to 3.47)	0.52	0.18 (−0.10 to 0.47)	0.21	−0.11 (−0.43 to 0.21)	0.51	1.01 (−0.9 to 3.04)	0.32
Calcium (mg)	−2.62 (−5.58 to 0.43)	0.09	0.13 (−0.21 to 0.48)	0.44	−0.29 (−0.67 to 0.10)	0.15	0.15 (−2.22 to 2.58)	0.90
Iron (mg)	−2.29 (−5.27 to 0.78)	0.14	0.03 (−0.31 to 0.37)	0.85	−0.14 (−0.53 to 0.24)	0.46	−0.36 (−2.72 to 2.06)	0.77

Coefficients and CIs are adjusted for age in quartiles, sex, month, height, ethnic group, and school (random-effect); analyses of micronutrients and nonstarch polysaccharides are also adjusted for total energy intake. All metabolic risk markers are log transformed. **P* value depicts no difference.

additional adjustment for physical activity made little difference to the reported results (data not presented). Further analyses investigated associations between energy intake from foods and drinks separately; these variables had similarly positive associations with insulin resistance and glucose (data not presented). The use of body fat measures based on skinfold thicknesses yielded similar results to those based on bioelectrical impedance. The use of fasting insulin instead of HOMA of insulin resistance yielded similar results to those reported here. The inclusion of socioeconomic position in analyses had no material effect on the results.

CONCLUSIONS

In this multiethnic study population, a positive association was found between total energy intake and insulin resistance. After excluding implausible reporters (9% of the total sample), the association was strengthened, energy intake was also positively associated with fasting glucose and fat mass index. These associations persisted after allowing for energy density, which was not associated with type 2 diabetes risk markers once total energy intake was taken into account. No other consistent dietary associations were found between nutrient intakes and risk markers.

Comparison With Previous Studies

There is limited literature on associations between childhood nutrient intakes and type 2 diabetes risk markers (20), and, as far as we are aware, no studies in children have yet been published that have examined total energy intake or energy density and type 2 diabetes risk markers. The results of large studies examining prospective associations between energy intake and type 2 diabetes risk in adults have been conflicting, with some studies reporting positive associations (3,4) and others null associations (21,22). These conflicting findings may reflect the influence of underreporting (more prevalent in overweight participants [23]) on the associations between energy intake and type 2 diabetes risk. Of the two studies reporting positive associations between energy intake and diabetes risk, one

Table 2—Percentage differences in type 2 diabetes risk markers and fat mass index per 1 SD increase in dietary intake in 1,841 participants (excluding implausible reporters)

	Insulin resistance		HbA_{1c} (%)		Glucose (mmol/L)		Fat mass index (kg/m^5)	
Dietary intake	% change (95% CI)	*P* value*	% change (95% CI)	*P* value*	% change (95% CI)	*P* value*	% change (95% CI)	*P* value*
Energy intake (kcal)	7.93 (4.83–11.13)	<0.0001	0.17 (−0.16 to 0.49)	0.32	0.58 (0.22–0.95)	0.002	5.34 (3.02–7.72)	<0.0001
Energy density (kJ/g)	2.74 (0.10–5.46)	0.04	−0.08 (−0.37 to 0.21)	0.58	0.19 (−0.13 to 0.52)	0.24	2.34 (0.32–4.41)	0.02
Fat (% energy)	−1.62 (−4.25 to 1.09)	0.24	−0.17 (−0.47 to 0.14)	0.28	0.04 (−0.30 to 0.38)	0.81	0.28 (−1.79 to 2.40)	0.79
Saturated fat (% energy)	−0.27 (−2.88 to 2.41)	0.84	−0.11 (−0.41 to 0.18)	0.45	0.14 (−0.19 to 0.47)	0.42	1.53 (−0.52 to 3.61)	0.14
Monounsaturated fat (% energy)	−1.59 (−4.22 to 1.10)	0.24	−0.17 (−0.47 to 0.13)	0.27	0.06 (−0.28 to 0.40)	0.74	0.01 (−2.04 to 2.10)	0.99
Polyunsaturated fat (% energy)	−0.73 (−3.31 to 1.92)	0.59	−0.02 (−0.31 to 0.27)	0.88	0.11 (−0.21 to 0.44)	0.49	0.28 (−1.71 to 2.32)	0.78
Carbohydrate (% energy)	2.09 (−0.61 to 4.86)	0.13	0.15 (−0.15 to 0.45)	0.31	0.00 (−0.34 to 0.34)	0.99	−0.69 (−2.72 to 1.37)	0.51
Sugars (% energy)	1.98 (−0.78 to 4.82)	0.16	0.04 (−0.27 to 0.34)	0.82	−0.15 (−0.49 to 0.19)	0.38	0.20 (−1.88 to 2.33)	0.85
Starch (% energy)	0.73 (−2.00 to 3.53)	0.60	0.10 (−0.20 to 0.40)	0.52	0.20 (−0.14 to 0.54)	0.25	−0.72 (−2.77 to 1.38)	0.50
Nonstarch polysaccharides (g)	−0.54 (−3.48 to 2.49)	0.72	0.22 (−0.12 to 0.55)	0.20	−0.05 (−0.43 to 0.32)	0.79	1.38 (−0.92 to 3.73)	0.24
Protein (% energy)	−1.90 (−4.61 to 0.89)	0.18	−0.03 (−0.34 to 0.28)	0.83	−0.10 (−0.44 to 0.25)	0.59	0.62 (−1.51 to 2.79)	0.57
Vitamin B12 (μg)	−1.16 (−3.78 to 1.52)	0.39	0.04 (−0.26 to 0.34)	0.79	−0.03 (−0.36 to 0.31)	0.88	−0.40 (−2.43 to 1.67)	0.70
Folate (μg)	−0.75 (−3.56 to 2.14)	0.61	0.29 (−0.03 to 0.61)	0.08	−0.23 (−0.58 to 0.13)	0.22	−2.22 (−4.34 to −0.06)	0.04
Vitamin C (mg)	0.97 (−1.67 to 3.68)	0.48	0.17 (−0.13 to 0.47)	0.26	−0.19 (−0.52 to 0.14)	0.26	0.92 (−1.11 to 2.99)	0.38
Calcium (mg)	−2.01 (−5.06 to 1.14)	0.21	0.15 (−0.20 to 0.50)	0.41	−0.30 (−0.69 to 0.10)	0.14	0.20 (−2.20 to 2.65)	0.87
Iron (mg)	−1.49 (−4.57 to 1.68)	0.35	0.12 (−0.23 to 0.48)	0.49	−0.10 (−0.50 to 0.30)	0.62	0.12 (−2.28 to 2.59)	0.92

Coefficients and CIs are adjusted for age in quartiles, sex, month, height, ethnic group, and school (random-effect). Analyses of micronutrients and nonstarch polysaccharides are also adjusted for total energy intake. All metabolic risk markers are log transformed. **P* value depicts no difference.

observed an association between energy intake and type 2 diabetes risk only when energy intakes calibrated by biomarkers were used (3); the other study was carried out in a population with a low prevalence of overweight and obesity, which may therefore have been less affected by underreporting of energy intake (4). This emphasis on the importance of energy intake would be consistent with previous studies showing strong ecological associations between energy intake and diabetes mortality (24). In one large population-based adult study, energy density showed strong positive associations with insulin resistance (5). However, total energy intake was not taken into account. Evidence on the associations between individual macronutrient and micronutrient intakes and type 2 diabetes risk in adults has been inconsistent, with the weight of previous evidence suggesting that diet quality rather than specific nutrient intakes were related to emerging type 2 diabetes risk (25), as observed in the current study.

Strengths and Limitations

The strengths and limitations of this study warrant consideration. Although the response rates were moderate, the study was sufficiently large to estimate main effects (although not ethnic group–specific effects) with precision. The distribution of socioeconomic position in the study population was close to that observed for England as a whole (26). The study included relevant early risk markers for type 2 diabetes; insulin resistance was assessed using the HOMA method, which has been validated in children, although providing estimates very similar to those of fasting insulin (27,28), as reported here. Assessment of body fat was primarily based on fat mass index derived from bioelectrical impedance, a more valid indicator of body fat than BMI in this multiethnic population (16). The assessment of energy intake was based on a single 24-h diet recall, a practical method for large-scale use that provides estimates of energy intake that are unbiased but imprecise in this age-group (29). However, imprecision in the measurement of energy intake will have reduced the likelihood of detecting any

Table 3—Percentage differences in type 2 diabetes risk markers and fat mass index per 1 SD increase in energy intake and energy density in 1,841 children (excluding implausible reporters)

		Insulin resistance		HbA_{1c} (%)		Glucose (mmol/L)		Fat mass index (kg/m^5)	
Dietary intake	Model	% change (95% CI)	*P* value*	% change (95% CI)	*P* value*	% change (95% CI)	*P* value*	% change (95% CI)	*P* value*
Total energy (kcal)	1a	7.89 (4.79–11.08)	<0.0001	0.17 (−0.16 to 0.49)	0.32	0.58 (0.21–0.95)	0.002	5.39 (3.05–7.77)	<0.0001
	2a	7.58 (4.37–10.90)	<0.0001	0.21 (−0.13 to 0.55)	0.23	0.55 (0.17–0.94)	0.005	5.07 (2.65–7.55)	<0.0001
	3a	3.97 (1.18–6.85)	0.01	0.10 (−0.24 to 0.44)	0.57	0.46 (0.08–0.85)	0.02		
Energy density (kJ/g)	1b	2.74 (0.09–5.45)	0.04	−0.09 (−0.38 to 0.20)	0.55	0.20 (−0.12 to 0.53)	0.22	2.23 (0.20–4.30)	0.03
	2b	0.94 (−1.75 to 3.69)	0.50	−0.14 (−0.44 to 0.16)	0.36	0.07 (−0.27 to 0.41)	0.68	1.02 (−1.05 to 3.14)	0.34
	3b	0.19 (−2.20 to 2.62)	0.88	−0.17 (−0.46 to 0.13)	0.28	0.05 (−0.28 to 0.39)	0.76		

Model 1, coefficients and CIs are adjusted for age in quartiles, sex, month, height, ethnic group, and school (random-effects); model 2, model 1 adjustments and energy density for model with energy (kcal) as explanatory variable and total energy for model with energy density as explanatory variable; model 3, coefficients are adjusted for covariates in model 1and model 2 plus fat mass index. All metabolic risk markers are log transformed. *P value depicts no difference.

association rather than creating a spurious association. Because the estimates were obtained for only a single day, conservative criteria were used to exclude implausible reports of energy intakes (with 0.9 and 2.75 PAL used as cutoff values), so that only the most extreme values were treated as implausible. The association between energy intake and insulin resistance was apparent both before as well as after exclusion of or adjustment for participants who reported implausible energy intakes. The overall validity of energy and nutrient intakes in the current study are supported by their similarity to estimates in the National Diet and Nutrition Survey data, which used a more detailed method of dietary data collection (7-day weighed food diary) (30), and the expected associations between nutrient intakes and blood lipids observed in this study population (31), although the cross-sectional design limits the strength of evidence on a possible causal association between energy intake and emerging type 2 diabetes risk. However, this design is particularly appropriate for examining short-term associations between dietary composition and type 2 diabetes risk markers, which are likely to be particularly relevant in the present context.

Implications

The results of the current study suggest that high energy intake, rather than specific macro- and micronutrient intakes, is associated with type 2 diabetes risk markers in children. The associations between energy intake and type 2 diabetes risk markers show a clear graded relationship that could feasibly be causal. Further studies, particularly trials examining the effects of reducing energy intake on emerging type 2 diabetes risk, could be particularly informative. The possibility that the association between energy intake and insulin resistance is at least partly independent of body fat is consistent with evidence on the impact of bariatric surgery and calorie restriction on insulin resistance, which in adults is also partly independent of body fat (32). The results are a particular concern in the light of recent evidence that childhood energy intake has

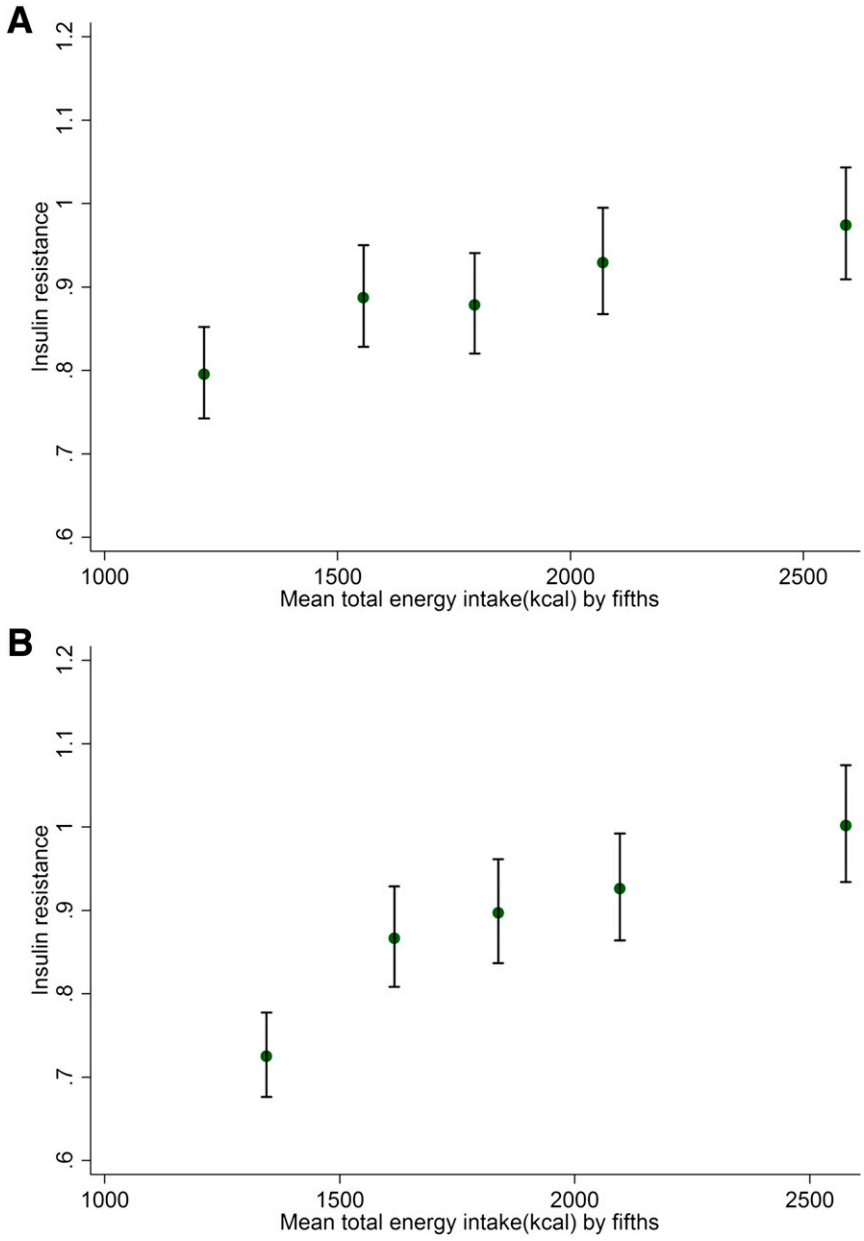

Figure 1—*A*: HOMA of insulin resistance by fifths of mean total energy. *B*: HOMA of insulin resistance by fifths of mean total energy intakes in all participants (2,017 children) in 1,841 children (excluding implausible reporters).

increased over time (33). Efforts to reduce energy intake will need to take account of energy density and diet quality. Although energy density was not independently associated with type 2 diabetes precursors in the current study, those children who consumed the highest amount of energy also tended to consume more energy-dense foods, suggesting that reducing the energy density of foods has an important part to play in reducing energy intake. In light of the findings of the current research, intervention studies examining the effects of reducing energy intake in children on type 2 diabetes risk markers are warranted.

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