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# Changes in Dietary Magnesium Intake and Risk of Type 2 Diabetes Mellitus in Middle School Students: Using Data from the HEALTHY Study

Manal Naseeb,[1] Michael L Bruneau, Jr,[2] Brandy-Joe Milliron,[3] Deeptha Sukumar,[3] Gary D Foster,[4] Sinclair A Smith,[2] and Stella L Volpe[5]

[1]Department of Clinical Nutrition, Faculty of Applied Medical Sciences, King Abdulaziz University, Jeddah, Saudi Arabia; [2]Department of Health Sciences, College of Nursing and Health Professions, Drexel University, Philadelphia, PA, USA; [3]Department of Nutrition Sciences, College of Nursing and Health Professions, Drexel University, Philadelphia, PA, USA; [4]WW International, Inc., New York, NY, USA; and [5]Department of Human Nutrition, Foods, and Exercise, Virginia Polytechnic Institute and State University, Blacksburg, VA, USA

## ABSTRACT

**Background:** The HEALTHY Study was a multicomponent school-based intervention, designed to prevent type 2 diabetes mellitus (T2DM) in middle-school students.

**Objectives:** We examined whether the difference in dietary magnesium intake, BMI percentile, and plasma glucose and insulin concentrations from 6th to 8th grade were related in the intervention schools and in the control schools that participated in the HEALTHY Study.

**Methods:** A total of 2181 ethnically diverse students, from 11.3 to 13.7 y of age, with completed dietary records, BMI percentile, and plasma glucose and insulin concentrations at 6th and 8th grades were included. Dietary magnesium intake was self-reported using the Block Kids FFQ. A hierarchical multiple regression model was used to determine whether the differences in dietary magnesium intake, BMI percentile, and plasma glucose and insulin concentrations from 6th to 8th grades were related, while adjusting for dietary calcium intake and total energy intake.

**Results:** The difference in dietary magnesium intake was significantly related to changes in BMI percentile from 6th to 8th grade in intervention and in control schools [intervention: $\beta$: −0.07; 95% CI: −0.58, −0.02; $P = 0.03$; $R^2$ (regression coefficient effect size): 0.14; 95% CI for $R^2$: 0.10, 0.17; control: $\beta$: −0.08; 95% CI: −0.63, −0.09; $P = 0.01$; $R^2$: 0.12; 95% CI for $R^2$: 0.08, 0.15]. The difference in dietary magnesium intake was not related to plasma glucose and insulin concentrations in intervention and in control schools.

**Conclusions:** We conclude that a multicomponent intervention was associated with reduced risk of T2DM, and that this association may be modulated, in part, by magnesium. The differences in dietary magnesium intake from 6th to 8th grade were negatively related to changes in BMI percentile among middle-school students. *J Nutr* 2021;151:3442–3449.

**Keywords:** type 2 diabetes mellitus, children and adolescents, HEALTHY Study, body mass index, dietary magnesium, fasting plasma glucose concentrations, fasting plasma insulin concentrations

## Introduction

The incidence of type 2 diabetes mellitus (T2DM) has dramatically increased worldwide and is linked to the rise in childhood obesity. Worldwide, >200 children and adolescents develop T2DM every day (1). The American Diabetes Association reported that 8%–45% of children had newly diagnosed T2DM in 2000. However, there is a limited amount of information about the epidemiology of T2DM in children, because of the relatively recent recognition of its emergence among this population. During 2011 and 2012, the estimated annual number of newly diagnosed children and adolescents with T2DM was 5300 cases in the United States based on findings from the SEARCH for Diabetes in Youth study (2–5). One of the keys to preventing T2DM in children and adolescents is through the prevention of obesity. Further, minimal weight loss

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Abbreviations used: NSLP, National School Lunch Program; SBP, School Breakfast Program; T2DM, type 2 diabetes mellitus.
Address correspondence to SLV (e-mail: stellalv@vt.edu).


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can decrease the risk of T2DM among higher-risk populations (2, 4). It has been shown that in adults, for every 1- to 2-kg/m$^2$ reduction in BMI, there is a 0.72-point reduction in the OR of the risk of T2DM incidence; however, at present, these results have not been demonstrated in children (6). Lifestyle modifications including healthy and well-balanced dietary habits, increased physical activity, and limiting screen time are required to reduce the prevalence of T2DM among children and adolescents (4, 7).

Healthy eating, characterized by a diet rich in fruit, vegetables, and whole grains, may help prevent T2DM among children and adolescents (8, 9). Magnesium is one of the key micronutrients found in these food sources. Magnesium is involved in the regulation of insulin signaling, the phosphorylation of insulin receptor kinase, and insulin-mediated cellular glucose uptake; a deficit of this element could lead to developing T2DM (10).

The most recent research on the relation between dietary magnesium intake and risk of T2DM has been limited to adult populations. Overall, researchers reported a negative correlation between dietary magnesium intake and the risk of T2DM, establishing it as an important nutrient to study (11–20). Therefore, there is a need to examine the relation between dietary magnesium intake and the risk of T2DM in children and adolescents. Moreover, there have been a limited number of investigations among children and adolescents.

Some researchers have shown that school-based interventions are successful in improving health behaviors of students (21–24). The HEALTHY Study, a school-based intervention, was designed to reduce modifiable risk factors for T2DM in middle-school students from 6th through 8th grades. The HEALTHY Study was a 3-y multicomponent intervention that was conducted in a large population of middle-school students (25–27). One of the HEALTHY Study goals was to increase intake of fruits and vegetables, which are good sources of magnesium. Therefore, we conducted the current study to examine whether the difference in dietary magnesium intake, BMI percentile, and plasma glucose and insulin concentrations from 6th to 8th grade were related in the intervention schools and the control schools that participated in the HEALTHY Study after adjusting for dietary calcium intake and total energy intake.

# Methods

## Study design

The current study was a secondary data analysis from the HEALTHY Study. The HEALTHY Study was conducted in 7 field centers across the United States. Each field center included 3 intervention schools and 3 control schools over 3 school years, starting among 6th-grade students at the beginning of 2006 and continuing through the 8th grade in the 2009 school year. A total of 42 participating schools were included. Eligible schools were required to have ≥50% of children and adolescents attending these schools belonging to an under-represented group (African American, Hispanic/Latino, and/or American Indian) who were at risk of T2DM and/or >50% of students eligible for free or reduced-cost meals. Half of the schools were randomly assigned to either intervention (21 schools) or control (21 schools). Intervention schools received a multicomponent program, which included nutrition, physical education, behavioral knowledge and skills, and communications and social marketing. Control schools received no intervention but participated in data collection activities. The study was approved by the Institutional Review Boards at each of the 7 field centers, and written parental consent and minor child assent were obtained from all participants. Details of the HEALTHY Study design, methods, intervention materials, and results can be found at http://www.healthystudy.org and have been reported elsewhere (27–30).

### *Description of the nutrition intervention.*

The nutrition intervention component was designed to improve the quantity and nutritional quality of the foods and beverages offered to students by changing the total school food environment with emphasis on changes that would likely reduce the risk of overweight, obesity, and T2DM. The total school food environment included federal meal programs such as the School Breakfast Program (SBP), the National School Lunch Program (NSLP), the After-School Snack Program, and the Supper Program. It also included à la carte venues (including snack bars and school stores), vending machines, school fundraisers, and classroom parties and celebrations (27, 29–31).

The nutrition intervention component focused on 5 goals: *1*) increasing the availability and variety of fruits and vegetables; *2*) increasing the availability of high-fiber foods; *3*) lowering the average fat content of the foods offered in schools; *4*) increasing the availability of healthier beverages; and *5*) limiting portion sizes and energy content of desserts and snacks. More details about goals, strategies, and intervention school activities have been reported elsewhere (27, 30, 32, 33).

## Data collection

The primary outcome variables for our study were changes in dietary magnesium intake, BMI percentile, and plasma glucose and insulin concentrations from 6th to 8th grade. All measurements were collected at baseline (6th grade) and the end of the intervention when the same students were in the 8th grade.

### *Dietary intake measurements.*

Dietary magnesium intake was self-reported by the students using the Block Kids FFQ (32). Members of the HEALTHY Study staff were trained to administer the Block Kids FFQ to clarify any questions and to encourage students to answer all questions. The Block Kids FFQ is a semiquantified questionnaire that assesses intake from the previous week. The Block Kids FFQ asks about both intake of and portion sizes consumed of 100 food items using serving size visuals. The questionnaire included questions related to our research. Examples of these questions included: *1*) frequency of eating whole-grain cereals; *2*) number of bananas consumed per day; *3*) frequency and amount of greens; and *4*) number of days vitamins/mineral supplements were taken. The questionnaire was provided in both English and Spanish. Nutrient analyses were conducted by Block Dietary Data Systems in Berkeley, California, to determine estimates of usual intake for a variety of nutrients, including calculations of daily frequency and amounts for individual food items, and by food group. Dietary magnesium intake was measured in milligrams per day (27, 31). The actual questionnaire was not available for release. However, the list of variables included in the questionnaire is available in the HEALTHY Study protocol. The responses to the questionnaire are available in the HEALTHY Study data ST5.SAS file. The original questionnaire was validated in numerous studies and in a variety of populations and has been redesigned for use with children, a population which is similar to the population recruited and enrolled in the HEALTHY Study (34–38).

### *Anthropometric measurements.*

Body weight and height were measured to the nearest 0.5 pounds and 0.5 inches (0.23 kilograms and 12.7 millimeters), respectively, with participants wearing light clothing and without shoes, by trained, certified HEALTHY Study staff. Body weight and height were measured, and BMI (in kg/m$^2$) was calculated and converted to an age- and sex-specific BMI percentile from the CDC reference BMI-for-age and -sex growth charts. BMI percentile was categorized as follows: underweight (<5th); healthy weight (5th to <85th); overweight (85th to 94th); or obese (>95th) (26, 29, 31). To protect participant confidentiality, very low or high BMI percentiles were recoded in the publicly available data set, in accordance with NIH repository regulations. Based on the

HEALTHY Study analyses, those with very low BMI percentiles were coded as "1" and those with very high BMI percentiles were coded as "3." In our analyses, we imputed the value "30" for those who were coded as 1, and a value of "98" for those who were coded as 3.

### *Laboratory measurements.*

The biochemical parameters included in our study were fasting plasma glucose and insulin concentrations. Approximately 20 mL (~4 teaspoons) of venous blood were taken from fasting students. Detailed information about the analyses of the plasma glucose and insulin concentrations has been reported elsewhere (26, 29). Fasting plasma glucose concentrations were measured in milligrams per deciliter (26, 29). According to the HEALTHY Study procedures, a blood glucose concentration <100 mg/dL was considered normal, concentrations between 100 and 125 mg/dL indicated prediabetes, and concentrations >125 mg/dL indicated diabetes mellitus (26, 29). Fasting plasma insulin concentrations were measured in microunits per milliliter. According to the HEALTHY Study procedures, blood insulin concentrations ≥30 $\mu$U/mL were considered high, whereas concentrations <30 $\mu$U/mL were considered low. Demographic variables included age, sex, and race/ethnicity, and were self-reported by students. Detailed methods outlining data collection have been published elsewhere (26–30).

## Statistical analyses

Overall, 4603 ethnically diverse students participated in the HEALTHY Study. Of these, 2181 students (47%) were included in our analyses. These participants had completed 6th- and 8th-grade data for all outcome variables of interest, which included changes in dietary magnesium intake, BMI percentile, plasma glucose and insulin concentrations, and dietary calcium intake. Students with variables of interest falling outside the range of ±3 SD, high leverage points, and high influential points were considered outliers and excluded from the analyses to account for possible errors associated with under- and over-reporting. Nonetheless, our study is well-powered owing to the large sample size. A post hoc power analysis (G*Power version 3.1.9.2, 2009, Faul F) was conducted to estimate the power of the study using linear multiple regression, fixed model. Based upon the effect size f(V) = 0.1, an $\alpha$ = 0.05, and sample size = 2181, the detected power was 1. Continuous variables included changes in dietary magnesium intake, BMI percentile, and fasting plasma glucose and insulin concentrations from 6th to 8th grade. Changes in dietary calcium intake and total energy intake were included as covariates. Only those covariates which presented significant correlations with changes in dietary magnesium intake, BMI percentile, and plasma insulin and glucose concentrations were included. Demographic variables included age, sex, race and ethnicity, and school's designation (intervention and control). Descriptive statistics, including adjusted means ± SEs for all variables, were calculated and reported unless otherwise noted.

A hierarchical multiple regression was used to determine whether the differences in dietary magnesium intake, BMI percentile, and plasma glucose and insulin concentrations from 6th to 8th grades were related in the intervention schools and the control schools, while adjusting for dietary calcium intake and total energy intake. SPSS version 25.0 (2016, IBM Corporation) was used for analyses. All tests were 2-tailed, and a $P$ value < 0.05 was considered to be statistically significant a priori.

# Results

Table 1 displays descriptive characteristics for age, sex, race and ethnicity, and school's designation. A total of 2181 students (48% intervention, 52% control), ranging from 11.3 ± 0.6 to 13.7 ± 0.6 y of age, were included in this investigation. There were more female students in control schools than in intervention schools. There were more students of racial or ethnic minority in the intervention schools than in the control schools. The percentage of white students was greater in the control schools than in the intervention schools. Table 2 displays mean dietary magnesium and calcium intakes of the student sample and the corresponding RDA. Table 3 displays differences in dietary magnesium intake, BMI percentile, and plasma glucose and insulin concentrations from 6th to 8th grade in the intervention and control schools. The adjusted mean dietary magnesium intake and fasting plasma glucose concentrations were decreased from 6th to 8th grade in intervention schools, but were increased in control schools. The adjusted mean BMI percentile was decreased from 6th to 8th grade in both school designations. The adjusted mean fasting plasma insulin concentration was increased from 6th to 8th grade in both school designations.

**TABLE 1** Descriptive characteristics of the HEALTHY Study sample[1]

| Characteristics | Intervention ($n$ = 1045; 48%) | Control ($n$ = 1136; 52%) |
|---|---|---|
| Age,[2] y | 11.3 ± 0.6 to 13.7 ± 0.6 | 11.3 ± 0.6 to 13.7 ± 0.6 |
| Sex, $n$ (%) | | |
| Female | 563 (53.9) | 627 (55.2) |
| Male | 482 (46.1) | 509 (44.8) |
| Race/ethnicity, $n$ (%) | | |
| Hispanic | 578 (55.3) | 622 (54.8) |
| Black | 206 (19.7) | 169 (14.9) |
| White | 196 (18.8) | 257 (22.6) |
| Other | 65 (6.2) | 88 (7.7) |

[1] $n$ = 2181.
[2] Values are range ± SD.

## Outcome variables for intervention schools

Table 4 reports the results of the hierarchical multiple regression analyses to determine whether changes in dietary magnesium intake, BMI percentile, and plasma glucose and insulin concentrations were related in the intervention schools and in the control schools. When all variables were added into the full model (difference in BMI percentile, plasma glucose and insulin concentrations, dietary calcium intake, and total energy intake from 6th to 8th grades), differences in dietary magnesium intake were related to changes in BMI percentile from 6th to 8th grade [$\beta$ (standardized beta coefficient): −0.07; 95% CI: −0.58, −0.08; $P$ = 0.03]; however, this relation was seen to have only a small practical significance [$R^2$ (regression coefficient effect size): 0.14; 95% CI: 0.10, 0.17]. Differences in dietary magnesium intake were not significantly related to changes in plasma glucose and insulin concentrations from 6th to 8th grade ($\beta$: −0.05; 95% CI: −1.78, 0.06; $P$ = 0.78 and $\beta$: −0.08; 95% CI: −0.22, 1.82; $P$ = 0.54, respectively).

## Outcome variables for control schools

When all variables were added into the full model (difference in BMI percentile, plasma glucose and insulin concentrations, dietary calcium intake, and total energy intake from 6th to 8th grade), differences in dietary magnesium intake were statistically related to changes in BMI percentile from 6th to 8th grade ($\beta$: −0.08; 95% CI: −0.63, −0.09; $P$ = 0.01); however, this relation was seen to have only a small practical significance ($R^2$: 0.12; 95% CI: 0.08, 0.15). Differences in dietary magnesium intake were not significantly related to changes in plasma glucose and insulin concentrations from 6th to 8th grade (glucose: $\beta$: −0.01; 95% CI: −0.99, 0.79; $P$ = 0.83 and insulin: $\beta$: −0.02; 95% CI: −0.99, 0.57; $P$ = 0.60).

**TABLE 2** Dietary magnesium and calcium intakes of the HEALTHY Study sample[1]

| Characteristics | Control school | | Intervention school | | RDA, mg/d[2] |
|---|---|---|---|---|---|
| | 6th grade | 8th grade | 6th grade | 8th grade | |
| Dietary magnesium intake, mg/d | 195.80 ± 104.02 | 196.22 ± 100.67 | 196.71 ± 107.34 | 193.62 ± 100.36 | 9–13 y: 240; 14–18 y (females and males): 360–410 |
| Dietary calcium intake, mg/d | 697.55 ± 375.34 | 715.90 ± 377.50 | 688.68 ± 376.51 | 708.87 ± 372.55 | 1300 |

[1] $n = 2181$. Values are mean ± SD unless otherwise indicated.

### Dietary magnesium intake relation with covariates

Differences in dietary magnesium intake were significantly related to changes in dietary calcium intake from 6th to 8th grade in intervention and control schools (intervention: $\beta$: 0.37; 95% CI: 0.10, 0.13; $P < 0.001$ and control: $\beta$: 0.33; 95% CI: 0.09, 0.12; $P < 0.001$) when added into the model. In addition, when total energy intake was added into the model, the differences in dietary magnesium intake were significantly related to changes in the total energy intake from 6th to 8th grade in intervention and control schools (intervention: $\beta$: 0.10; 95% CI: −0.001, 0.003; $P < 0.001$ and control: $\beta$: 0.15; 95% CI: 0.001, 0.002; $P < 0.001$). Table 5 shows the equations for the full models.

## Discussion

We examined whether the differences in dietary magnesium intake, BMI percentile, and plasma glucose and insulin concentrations from 6th to 8th grade were related in the intervention schools and the control schools that participated in the HEALTHY Study, after controlling for dietary calcium intake and total energy intake. BMI percentile and plasma glucose and insulin concentrations have been established as risk factors for T2DM. Surprisingly, we found that differences in dietary magnesium intake were related to changes in BMI percentile, dietary calcium, and total energy intake from 6th to 8th grade in the intervention schools and in the control schools (intervention: $\beta$: −0.07, $P = 0.03$, $P < 0.001$, and $P < 0.001$; control: $\beta$: −0.08, $P = 0.01$, $P < 0.001$, and $P < 0.001$, respectively). Although there were significant relations between dietary magnesium intake and BMI percentile, the absolute value of the $\beta$ standardized coefficient was small, suggesting a small clinical finding. In addition, differences in dietary magnesium intake were not related to changes in plasma glucose and insulin concentrations from 6th to 8th grade in the intervention schools and the control schools (intervention: $P = 0.78$ and $P = 0.54$; control: $P = 0.83$ and $P = 0.60$, respectively).

Evidence that dietary magnesium intake is involved in body weight regulation is lacking in children and adolescent populations. Some researchers have reported a negative association between dietary magnesium intake and body weight (39), whereas others have reported a positive or no association (40). Jose et al. reported higher mean dietaty magensium intake in overweight children (0.20 ± 0.06 mg/kcal (40). Our findings are consistent with Huerta et al. (39), who reported a significantly lower mean dietary magnesium intake in children who were obese than in those who were lean, after adjusting for energy intake (0.12 ± 0.004 mg/kcal compared with 0.14 ± 0.004 mg/kcal, $P = 0.003$). Furthermore, they reported that serum magnesium concentrations were significantly inversely associated with BMI ($r = -0.44$; 95% CI: −0.65, −0.17; $P = 0.002$). A possible explanation for this association is that magnesium may form a soap with fatty acids in the intestine, consequently reducing the digestible energy content of the diet. Magnesium also acts as a cofactor of lipoprotein lipase, which is an enzyme required to break down lipids. Thus, magnesium deficiency might affect dietary fat and lipolysis in the body, leading to obesity (41).

Decreased serum magnesium concentrations in children with obesity have been observed by several researchers (25, 42). Yakinci et al. (34) conducted a cohort study among children 7–11 y of age living in Turkey. The authors evaluated serum magnesium concentrations and other minerals in children with obesity ($n = 41$) compared with children with a healthy body weight ($n = 41$). They reported that children with obesity had significantly lower serum magnesium concentrations than children with a healthy body weight (1.78 ± 0.03 and 2.14 ± 0.04 mg/dL, respectively; $P < 0.001$). Moreover, Hassan et al. (43) concluded that children 2–14 y of age with overweight and obesity had reduced serum magnesium concentrations (2.08 ± 0.211 mg/dL) compared with children with a healthy body weight (2.55 ± 0.155 mg/dL, $P < 0.001$).

Jose et al. (40) conducted a cross-sectional study and reported that children who were overweight had significantly lower serum magnesium concentrations than children with a healthy body weight, after adjusting for energy intake (2.12 ± 0.33 mg/dL and 2.5 ± 0.24 mg/dL, respectively; $P < 0.001$).

**TABLE 3** Differences in dietary magnesium intake, BMI percentile, and plasma glucose and insulin concentrations from 6th to 8th grade in the intervention and control schools[1]

| Participant demographics | Adjusted mean difference, intervention (8th to 6th grades) | Adjusted mean difference, control (8th to 6th grades) | Adjusted mean difference for the difference (control − intervention) |
|---|---|---|---|
| Difference in dietary magnesium intake, mg | −2.77 ± 4.23 | 0.61 ± 4.05 | 3.38 ± 5.85 |
| Difference in BMI percentile | −0.07 ± 1.05 | −1.92 ± 1.00 | −1.85 ± 1.45 |
| Difference in fasting plasma glucose concentrations, mg/dL | −0.31 ± 0.29 | 0.66 ± 0.27 | 0.97 ± 0.39 |
| Difference in fasting plasma insulin concentrations, $\mu$U/mL | 3.53 ± 0.37 | 3.45 ± 0.36 | −0.08 ± 0.52 |

[1] $n = 2181$. Values are adjusted means ± SEs.

**TABLE 4** Hierarchical multiple regression analysis for changes in dietary magnesium intake from 6th to 8th grade in control and intervention schools[1]

| Outcome | Grade | Variables | $R^2$ | *b* | $\beta$ | $SE_b$ | *t* | *P* |
|---|---|---|---|---|---|---|---|---|
| Changes in dietary magnesium intake—intervention | 6th and 8th | BMI percentile | 0.14 | −0.30 | −0.07 | 0.14 | −2.11 | 0.03* |
| | | Glucose | | −0.86 | −0.05 | 0.47 | −1.82 | 0.78 |
| | | Insulin | | −1.02 | −0.08 | 0.41 | 2.50 | 0.54 |
| | | Dietary calcium | | 0.11 | 0.37 | 0.01 | 12.75 | <0.01* |
| | | Total energy intake | | 0.001 | 0.10 | 0.01 | 6.40 | <0.01* |
| | | Constant | | −9.25 | | | | |
| | | SEE | | 132.88 | | | | |
| Changes in dietary magnesium intake—control | 6th and 8th** | BMI percentile | 0.12 | −0.36 | −0.08 | 0.14 | −2.63 | 0.01* |
| | | Glucose | | −0.10 | −0.01 | 0.46 | −0.21 | 0.83 |
| | | Insulin | | −0.21 | −0.02 | 0.40 | −0.53 | 0.60 |
| | | Dietary calcium | | 0.11 | 0.33 | 0.01 | 11.89 | <0.01* |
| | | Total energy intake | | 0.001 | 0.15 | 0.00 | 9.43 | <0.01* |
| | | Constant | | −1.41 | | | | |
| | | SEE | | 138.22 | | | | |

[1] $n = 2181$. *Correlation is significant at the 0.05 level (2-tailed). **Subtract 8th grade from 6th grade. , standardized $\beta$ coefficient; *b*, unstandardized $\beta$ coefficient; glucose, plasma glucose concentrations; insulin, plasma insulin concentrations; $R^2$, variability of the model explained; $SE_b$, SE for the unstandardized $\beta$.

Moreover, the authors indicated that serum magnesium concentrations were significantly inversely correlated with BMI ($P < 0.001$). There appears to be a relation between serum magnesium concentrations and BMI, which may be affected by dietary magnesium intake. Children who are overweight and obese may benefit most from an increased dietary magnesium intake; it has been reported that these children tend to consume fewer whole grains, green leafy vegetables, and other foods rich in magnesium (44).

Our lack of findings of an association between dietary magnesium intake and plasma glucose and insulin concentrations opposes other researchers who have reported a negative association (35, 39, 45). Huerta et al. (39) conducted a cross-sectional study to examine whether dietary magnesium intake is associated with insulin resistance. The authors compared children with obesity (BMI ≥85th percentile) and without T2DM to 24 sex- and puberty-matched, healthy control participants (BMI <85th percentile), 8–17 y of age. They reported that dietary magnesium intake was inversely associated with fasting plasma insulin concentrations [Spearman correlation ($r$) = −0.43, $P = 0.002$] (39). The findings from Huerta et al. (39) are in agreement with Bo et al. (35), who conducted a cross-sectional study to examine the associations between dietary magnesium intake and serum glucose and insulin concentrations among preschool-age Italian children (>2 and <6 y of age). The authors reported that decreased dietary magnesium intake was significantly associated with increased fasting serum glucose and insulin concentrations, and insulin resistance (measured using the HOMA-IR index) ($\beta$: −0.01, $P = 0.005$; $\beta$: −0.009, $P = 0.03$; and $\beta$: −0.012, $P = 0.01$, respectively). However, after adjusting for other confounders and dietary fiber intake, dietary magnesium intake was only inversely associated with fasting serum glucose concentrations ($\beta$: −0.018; 95% CI: −0.026, −0.010) (35). This association may suggest that there is a relation between dietary magnesium intake and risk of T2DM. The relation may differ by other factors we were unable to investigate, such as total energy intake and dietary fiber.

Celik et al. (45) evaluated the relation between serum magnesium concentrations and insulin resistance in 203 children with obesity and insulin resistance (117 children with obesity, 86 controls who were not obese; 11.3 ± 2.4 y of age). The authors measured serum glucose, insulin, and magnesium concentrations. They reported that serum magnesium concentrations, which may be related to dietary magnesium intake, were significantly lower in children with obesity and insulin resistance than in controls (2.14 ± 0.16 compared with 2.22 ± 0.17 mg/dL, $P = 0.01$) (45). A possible explanation for their results is that, in individuals with obesity, insulin may induce magnesium excretion in those with insulin resistance (36). However, Celik et al. (45) reported no significant correlation between serum glucose and magnesium concentrations in children with insulin resistance ($r = 0.12$, $P = 0.33$) (45). Nonetheless, their results provide evidence that low serum magnesium concentrations may contribute to the development of insulin resistance in children with obesity.

Similar to our findings, Muhammad et al. (37) reported that there was no significant association between dietary magnesium intake and insulin resistance, measured using HOMA-IR index, in their cross-sectional study, in 78 adolescent girls (13.80 ± 0.85 y of age) from Indonesia who were

**TABLE 5** Equations for hierarchical multiple regression models for dietary magnesium intake for intervention and control schools[1]

| School designation | Model equation |
|---|---|
| Intervention | Changes in dietary magnesium intake = −9.25 + (−0.30*BMI percentile) + (−0.86*glucose) + (−1.02*insulin) + (0.11*dietary calcium intake) + (0.001*total energy intake) [$r = 0.38$, $R^2 = 0.14$, $F(4,1040) = 42.98$] |
| Control | Changes in dietary magnesium intake = −1.41 + (−0.36*BMI percentile) + (−0.10*glucose) + (−0.21*insulin) + (0.11*dietary calcium intake) + (0.001*total energy intake) [$r = 0.35$, $R^2 = 0.12$, $F(4,1131) = 38.64$] |

[1] $n = 2181$.

obese (BMI ≥95th percentile) ($P = 0.77$, respectively). They also reported that increased magnesium intake was not correlated with decreased fasting blood glucose concentrations ($r = -0.18$, $P = 0.44$), fasting insulin concentrations ($r = -0.004$, $P = 0.46$), and the HOMA-IR index ($r = -0.06$, $P = 0.77$), after adjusting for age, BMI, and waist circumference. However, the girls met 61% of the recommended daily requirement for magnesium, which may have contributed to the authors not finding a significant association (37). Therefore, consumption of >60% of the recommended daily requirement may be needed to create a protective effect.

We found that changes in dietary magnesium intake were related to changes in BMI percentile in the intervention schools and the control schools. Possible explanations are that there was no difference in servings per student of high-fiber grain-based foods and legumes in the SBP and NSLP, which are good sources of magnesium ($P = 0.52$ and $P = 0.38$, respectively). Further, servings of vegetables selected by students were similar in intervention and control schools at the end of the study in the SBP and NSLP. This might have been a result of introducing several public health initiatives targeting school food and wellness policies that were mandatory to address childhood obesity within the same time frame as the HEALTHY Study (38). Moreover, Siega-Riz et al. (27) reported a 10% increase in the intake of fruit among students in the intervention schools compared with the control schools in the HEALTHY Study; however, a 10% change may not be enough to show changes in dietary magnesium intake. Moreover, the HEALTHY Study was not designed to target dietary magnesium intake in middle-school students in relation to the risk of T2DM. Furthermore, researchers from the HEALTHY Study reported no significant differences between intervention and control schools in the combined prevalence of overweight and obesity and mean glucose concentrations (27, 33, 38). Lastly, parents received a feedback letter with their child's physical and laboratory values, along with interpretation and recommended follow-up if appropriate, in both intervention and control schools (27). Parents may have changed dietary habits for their children even though they were in control schools.

Our study has several limitations which need to be acknowledged. It was a secondary data analysis using information from the HEALTHY Study. We only focused on the nutrition components of the HEALTHY Study, which made it challenging to measure the effect of a single component from a multicomponent intervention because of a likely synergistic relation (28). Despite the fact that Block's Kids FFQ used in the HEALTHY Study is a validated questionnaire, students' response was subject to social desirability bias (46). Although data were adjusted for potential confounders, such as dietary calcium intake, which is known to attenuate magnesium absorption, the possibility of uncontrolled or unknown confounding variables cannot be ruled out (10, 42, 47, 48). Including all possible confounders may have decreased the power of our analyses. We excluded other confounders such as dietary fiber intake owing to multicollinearity, to avoid overfitting the model and minimize the risk of the model reflecting random error, which may have reduced the generalizability of our findings (49, 50). We did not analyze the findings based on the sex difference in characteristics and stages of sexual maturation. Considering all these unaccounted confounders in our analyses could have provided additional findings. Lastly, our findings cannot establish a causal relation between dietary magnesium intake and the risk of T2DM among children and adolescents.

Our study had several strengths, as well. First, our findings add to the limited knowledge that currently exists. The findings might guide future public health researchers regarding dietary magnesium intake and the risk of T2DM among children and adolescents. The large sample size, which includes a high proportion of under-represented populations who are at high risk of T2DM and obesity, is another major strength of our study. The geographical diversity of the schools included in the HEALTHY Study spanned across the United States, which increased the generalizability of our findings. We also considered the interaction of dietary calcium intake and total energy intake in our statistical analyses. The findings of our study might provide a low-cost approach to reduce the risk of T2DM among children and adolescents.

Further work is required to determine the extent and strength of the relation between magnesium intake and BMI among children and adolescents. In addition, a randomized controlled trial targeting dietary magnesium intake and the risk of T2DM among children and adolescents would be desirable. Despite the fact that some researchers have revealed a link between dietary magnesium intake and obesity, supplementation of the diet with magnesium for the prevention of obesity-related complications should also be considered. Finally, because it is important to promote health and prevent risk in children and adolescents, it is necessary to take notice of research related to school-based intervention as a major priority and recognize the need to fund projects in this research field.

We conclude that a multicomponent intervention was associated with reduced risk in T2DM among children and adolescents, an association which may have been modulated, in part, by magnesium. We found that differences in dietary magnesium intake from 6th to 8th grade were negatively related to changes in BMI percentile among middle-school students. Further research using longitudinal cohort designs should be considered. Our findings support the results from a small number of studies that were conducted among children and adolescents, which showed an association between dietary magnesium intake and high BMI percentile as a risk factor for T2DM. Studies further exploring the causal relation between dietary magnesium intake and the risk of T2DM are warranted.


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