Influence of high glycemic index and glycemic load diets on blood pressure during adolescence

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Keywords
blood, during, adolescence, diets, influence, high, pressure, glycemic, index, load

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Abstract—We aimed to prospectively examine the association between the glycemic index and glycemic load of foods consumed and the dietary intakes of carbohydrates, sugars, fiber, and principal carbohydrate-containing food groups (eg, breads, cereals, and sugary drinks) with changes in blood pressure during adolescence. A total of 858 students aged 12 years at baseline (422 girls and 436 boys) were examined from 2004–2005 to 2009–2011. Dietary data were assessed from validated semiquantitative food frequency questionnaires. Blood pressure was measured using a standard protocol. In girls, after adjusting for age, ethnicity, parental education, parental history of hypertension, baseline height, baseline blood pressure, change in body mass index, and time spent in physical and sedentary activities, each 1-SD (1-SD=7.10 g/d) increase in baseline dietary intake of total fiber was associated with a 0.96-, 0.62-, and 0.75-mmHg decrease in mean systolic (P=0.02), diastolic (P=0.01), and arterial blood pressures (P=0.002), respectively, 5 years later. In girls, each 1-SD increase in dietary glycemic index, glycemic load, carbohydrate, and fructose was concurrently related to increases of 1.81 (P=0.001), 4.02 (P=0.01), 4.74 (P=0.01), and 1.80 mmHg (P=0.03) in systolic blood pressure, respectively, >5 years. Significant associations between carbohydrate nutrition variables and blood pressure were not observed among boys. Excessive dietary intake of carbohydrates, specifically from high glycemic index/glycemic load foods, could adversely influence blood pressure, particularly in girls, whereas fiber-rich diets may be protective against elevated blood pressure during adolescence.

Key Words: adolescents ■ blood pressure ■ carbohydrate ■ fiber ■ glycemic index ■ glycemic load ■ Sydney Childhood Eye Study

Elevated blood pressure (BP) during childhood and adolescence is an independent risk factor for cardiovascular diseases in later life1 and the development of early pathological lesions of atherosclerosis.2,3 Therefore, preventing elevated BP levels in childhood and adolescence may be an important public health measure to limit the overall disease burden caused by hypertension.4,5

More recently, there are data to suggest that dietary factors, such as high consumption of carbohydrates, including sugars and soft drinks, could adversely influence BP levels.6 A recent meta-analysis of intervention studies involving adults7 shows that diets rich in carbohydrates may be associated with slightly higher BP. In contrast, dietary fiber intake was shown to be inversely associated with BP in adults8 and with reduced risk of incident cardiovascular disease.9 To the best of our knowledge, the temporal link between carbohydrate nutrition intake and BP has not been explored in an adolescent population.

In addition, previous adult studies have demonstrated a direct association of sugar-sweetened beverage consumption with systolic/diastolic BP and direct associations of fructose and glucose intake with BP.5,10 In addition, the National Health and Nutrition Examination Survey found that, in a sample of 4867 adolescents aged 12 to 18 years, higher sugar-sweetened beverage consumption was associated with higher serum uric acid levels and systolic BP.11 However, this study had a cross-sectional design, and currently there are no prospective data during adolescence examining this relationship.

To better understand the effects of carbohydrate nutrition variables on BP during adolescence, we used data available from a 5-year longitudinal study of adolescents in Australia to determine the following: (1) the association between baseline dietary intakes of total carbohydrate, sugars, glycemic index (GI), glycemic load (GL), and fiber with temporal changes in systolic and diastolic BP and mean arterial BP; (2) the relationship between baseline consumption of soft drinks/cordials and the principal carbohydrate-containing food groups (eg, breads and cereals, potatoes, and energy-dense nutrient-poor sources of carbohydrates, such as cookies and...
Dietary Data

Dietary data were collected using a 120-item self-administered food-frequency questionnaire, designed for specific use in Australian children and adolescents. An allowance for seasonal variation of fruit and vegetables was made during analysis by weighting seasonal fruits and vegetables. The validity of the food-frequency questionnaire has been reported previously in children. The carbohydrate and total sugars demonstrated a fair level of agreement when compared with 4 days of food records: the weighted κ for carbohydrate was 0.23 and for total sugars was 0.30, using quintiles to categorize the data from each dietary assessment method. In addition, 56% of carbohydrates and 60% of total sugars ranked within 1 quintile. Less than 5% of the results were grossly misclassified (ie, ranked highest by food-frequency questionnaire method and lowest by food record method or vice versa).

Food-frequency questionnaire items were translated into daily food and nutrient intakes using a purpose-built query in Microsoft Access 2007 with various nutrient databases. GI values were assigned to individual food items in the food-frequency questionnaire based on methods published previously. The GI of each food item was calculated as the corresponding GI (as %)×amount (in grams) of available carbohydrates in a serving of that food. The daily dietary GI of each subject was calculated as ∑GI; GL, and the dietary GI was obtained by (dietary GI/total available carbohydrate intake in the day)×100%. Analytic data on fructose were available for >90% of the foods used in the nutrient database. Fructose values were subjectively assigned to the remaining foods based on their compositions, where recipes were used to determine the ingredients of foods wherever available. There is currently no analytic method to distinguish between sugars that are intrinsic to foods and sugars that are added. We have imputed added sugar contents based on a 5-step methodology. Briefly, lactose was assumed to be natural in all foods. All of the sucrose in processed foods was assumed to be added (except where ingredients contain naturally occurring sucrose, eg, peanuts). Less than 10% of the 243 foods used in the nutrient database had subjectively imputed added sugar content.

We also extracted data on the fiber contribution from vegetables, fruit, and bread and cereals and on the consumption of main carbohydrate-containing food groups, vegetables, potatoes, fruit, and bread and cereals (composed of breakfast cereals, bread [white or other], pasta, and rice), as well as foods high in refined sugars (soft drinks, cordials, sweet biscuits, cakes, nuts, scenes, pastries, confectionary, sugar, honey, jams, and syrups), which we term as energy-dense, nutrient-poor sources of carbohydrates. Data on the frequency of soft drinks, cordials (a sweet-flavored concentrated syrup that is mixed with water to taste), and fruit juice consumption were also obtained from the food-frequency questionnaire.

Assessment of BP Measures

Data were collected during a preorganized visit to each school. BP was measured on the school premises according to a standard protocol. After 5 minutes of resting, BP was measured in a seated position using an automated professional sphygmomanometer (HEM 9070; Omron Healthcare, Inc) with appropriate cuff size. We followed general recommendations on selecting cuff size to ensure that the bladder length was ~80% and width was ~40% of the arm circumference, covering the upper arm without obscuring the antecubital fossa. Three separate BP measurements were taken and averaged for analysis. Mean arterial BP was calculated as one third of the systolic plus two thirds of the diastolic BP.

Collection of Data on Covariates

Parents completed a comprehensive 193-item questionnaire. Sociodemographic information covering ethnicity, country of birth, education, occupation, and parental age was collected. Parents were also asked to mark all of the medical conditions that the child’s biological mother and father may have had or currently had, with hypertension being one of these conditions. The child was classified as having a positive parental history of hypertension, if the parent marked hypertension for either or both biological mother and father.

Subjects and Methods

Study Population

The Sydney Childhood Eye Study is a population-based survey of eye conditions and other health outcomes in school children living within the Sydney Metropolitan Area. It was approved by the Human Research Ethics Committee (University of Sydney), the Department of Education and Training, and the Catholic Education Office (New South Wales, Australia). We obtained informed written consent from ≥1 parent of each child, as well as verbal assent or written consent from each child/adolescent before the examinations. Study methods have been described previously. Briefly, students with a mean age of 12.7 years in a stratified random cluster sample of 21 high schools across Sydney were eligible to participate. Stratification was based on socioeconomic status data and led to a proportional mix of public, private, or religious high schools. Of the 3144 eligible 12-year-old children, 2367 were re-examined (51.7% of baseline participants). Participants versus non-participants of the 5-y Follow-Up Survey were less likely to be older and white (Table 1).

Table 1. Baseline Study Characteristics of Participants and Nonparticipants of the 5-y Follow-Up Survey

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Participants (n=1216)</th>
<th>Nonparticipants (n=1137)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>12.67 (0.4)</td>
<td>12.74 (0.5)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Male sex, %</td>
<td>609 (50.0)</td>
<td>581 (51.2)</td>
<td>0.54</td>
</tr>
<tr>
<td>Ethnicity, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>694 (57.0)</td>
<td>713 (62.7)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>East Asian</td>
<td>238 (19.6)</td>
<td>114 (10.0)</td>
<td></td>
</tr>
<tr>
<td>Middle Eastern</td>
<td>82 (6.8)</td>
<td>84 (7.4)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>202 (16.7)</td>
<td>227 (19.9)</td>
<td></td>
</tr>
<tr>
<td>Parental education*</td>
<td>524 (48.2)</td>
<td>315 (32.3)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Parental history of hypertension</td>
<td>203 (16.7)</td>
<td>159 (14.0)</td>
<td>0.06</td>
</tr>
<tr>
<td>Height, cm</td>
<td>156.3 (7.6)</td>
<td>155.9 (8.2)</td>
<td>0.26</td>
</tr>
<tr>
<td>Body mass index, kg/m2</td>
<td>20.1 (4.0)</td>
<td>20.8 (4.4)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Time spent in screen viewing, h/d</td>
<td>2.75 (1.5)</td>
<td>2.88 (1.6)</td>
<td>0.05</td>
</tr>
<tr>
<td>Time spent in physical activity, h/d</td>
<td>0.82 (0.7)</td>
<td>0.86 (0.8)</td>
<td>0.18</td>
</tr>
</tbody>
</table>

| Dietary intake of                |                      |                          |           |
| Carbohydrate, g/d               | 261.4 (31.7)         | 261.7 (31.0)             | 0.81      |
| Total sugars, g/d               | 132.1 (29.4)         | 134.3 (31.8)             | 0.13      |
| Total fiber, g/d                | 28.6 (7.1)           | 27.9 (7.8)               | 0.06      |
| Glycemic index                  | 54.4 (3.3)           | 54.4 (3.4)               | 0.94      |
| Glycemic load                   | 142.0 (21.8)         | 142.3 (21.9)             | 0.78      |
| Consumption of                  |                      |                          |           |
| Vegetables, g/d                 | 161.0 (121.8)        | 151.0 (128.9)            | 0.08      |
| Fruits, g/d                     | 238.1 (182.2)        | 220.3 (190.3)            | 0.04      |

Data are presented as mean (SD) or proportions. *Data are for tertiary qualified mother and/or father.

We also extracted data on the fiber contribution from vegetables, fruit, and bread and cereals and on the consumption of main carbohydrate-containing food groups, vegetables, potatoes, fruit, and bread and cereals (composed of breakfast cereals, bread [white or other], pasta, and rice), as well as foods high in refined sugars (soft drinks, cordials, sweet biscuits, cakes, nuts, scenes, pastries, confectionary, sugar, honey, jams, and syrups), which we term as energy-dense, nutrient-poor sources of carbohydrates. Data on the frequency of soft drinks, cordials (a sweet-flavored concentrated syrup that is mixed with water to taste), and fruit juice consumption were also obtained from the food-frequency questionnaire.

Statistical Analysis

Data were analyzed using SPSS, version 16.0 (SPSS, Inc). Differences in continuous variables were tested using analysis of variance with Bonferroni post hoc test. Differences in categorical variables were tested using χ2 or Fisher’s exact tests as appropriate. Linear regression analysis was used to determine the magnitude of the association between changes in dietary variables and BP, adjusting for confounders. Odds ratios and 95% CIs were calculated for the association between dichotomized dietary variables and hypertension and prehypertension status. All models were adjusted for sex, age, and ethnic origin. P values <0.05 were considered significant.
The questions relating to physical activity composed a list of 9 common activities in which Sydney school-aged children typically participated, such as athletics, swimming, soccer, and so forth. Children self-reported the usual number of hours per week that they spent in each of these activities and whether the activity was done outdoors or indoors (hall gym or classroom). The time spent in each activity was summed, and the average hours per day spent in total physical activity was calculated. Total screen time (hours per day) was calculated as the time reported that was spent on the following activities, watching TV, playing video games, and using a computer for both recreational and educational purposes.

Each child’s height was measured with shoes off using a free-standing Seca height rod (model 220; Hamburg, Germany). Weight in kilograms was measured using a professional portable weighing machine, after removing any heavy clothing. BMI was calculated as weight divided by height squared (kilograms per meter squared).

### Statistical Analysis

Statistical analyses were performed using SAS (version 9.1, SAS Institute, Cary, NC). Carbohydrate nutrition variables and the main carbohydrate-containing food groups were the independent variables and were each analyzed as continuous variables (per 1-SD increase). Changes in systolic and diastolic BPs and mean arterial BP were the dependent variables and analyzed as continuous variables. Linear regression models were used to estimate the slopes (magnitudes) of the potential linear relationships between the dietary variables and BP. These analyses generated 2 regression coefficients, representing the prospective estimate, the slope of the regression of the dietary variables at baseline on the change in systolic and diastolic BPs and mean arterial BP at the 5-year follow-up, and concurrent estimate, the slope of the regression of the change in the dietary variables over the 5 years on the concurrent change in BP measures. Changes in dietary variables during the study period were calculated by subtracting baseline intakes from the intake recorded at the 5-year follow-up examination. We constructed multivariable linear regression models adjusting for age, sex, ethnicity, parental qualifications, parental history of hypertension, baseline height, baseline BP, change in BMI, recreational screen viewing time, and time spent in physical activity. Dietary GI, GL, carbohydrates, sugar, and fiber variables were adjusted for total energy intake using the residual method.20 Intakes of principal carbohydrate-containing food groups (eg, cereals, fruits, and soft drinks) were energy adjusted by using the energy partition method; that is, consumption was adjusted for the energy intake from all of the other foods.20 After multivariable adjustment, linear regression analyses indicated interactions between sex and the associations of dietary intakes of GL, carbohydrates, total sugars, fiber, soft drinks, fruit juice, and fruit fiber with systolic BP; the \( P_{interaction} \) values were 0.01, 0.03, 0.004, 0.04, 0.03, 0.03, and 0.01, respectively. Subsequent analyses of all of the carbohydrate nutrition variables and the principal carbohydrate containing food groups were, therefore, stratified by sex.

### Results

Of the 1216 children re-examined at the 5-year follow-up, 850 children (418 girls and 432 boys) were used for the current analyses, because these participants had complete data on the following: (1) all of the covariates included in the multivariable model; (2) BP measures at both baseline and 5-year follow-up; and (3) complete nutrient information at baseline. Study characteristics of participants with \((n=850)\) and without complete data \((n=363)\) were also compared. Participants with versus those without complete data were more likely to be white and older; to have tertiary qualified parents, a parental history of hypertension, and lower BMI; to spend less time in recreational screen viewing but more time in total physical activity; and to have higher dietary intake of fiber.

#### Association Between Carbohydrate Nutrition With BP Among Girls Only

The unadjusted associations between baseline dietary intakes of the different types of fiber with change in BP in girls are presented in Table 2; an inverse association between fruit fiber consumption and systolic and mean arterial BPs was observed. After multivariable adjustment, each 1-SD increase in baseline dietary intake of total fiber was associated with a mean decrease in systolic, diastolic, and mean arterial BPs at the 5-year follow-up (Table 3). Similarly, each 1-SD increase in dietary intakes of fruit and vegetable fiber and fruits at baseline was associated with significant decreases in BP measures. Baseline dietary GI, GL, carbohydrate, sugars, and cereal fiber intake, as well as consumption of the principal carbohydrate-containing food groups (eg, soft drinks) at baseline, were not associated with temporal change in mean BP (data not shown).

We analyzed the relationship between changes in the intakes of dietary variables with concurrent change in BP. For these particular analyses, we had 509 participants (278 girls and 231 boys) with complete data on all of the covariates included in the multivariable model, as well as BP and dietary data at both the baseline and 5-year follow-up exams. Significant, unadjusted linear associations were observed between several carbohydrate nutrition variables, such as dietary GI, GL, and sugars with BP (Table 4). Each 1-SD increase in dietary GI, GL, carbohydrates, total sugars, added sugars, and fructose was concurrently related to an increase in BP measures in girls over 5 years (Table 5). We further adjusted for baseline dietary intake of total fiber when examining the association between dietary GI with BP; each 1-SD increase in dietary GI was concurrently associated with a 1.85- and 1.05-mm Hg higher mean systolic BP \((P=0.001)\) and mean arterial BP \((P=0.03)\), respectively. We also con-
Association Between Carbohydrate Nutrition With BP Among Boys Only

In boys, significant associations were not observed between carbohydrate nutrition parameters or the principal carbohydrate-containing food groups (eg, breads and cereals) with changes in BP (Tables S1 and S2, available in the online-only Data Supplement).

Discussion

In this prospective study of adolescents, both carbohydrate quantity and quality were independently associated with BP in girls. Specifically, we observed positive associations between changes in the consumption of carbohydrates, GI, GL, total sugars, fructose, and added sugars, with concurrent change in BP among adolescent girls. Conversely, girls who consumed the highest amounts of total fiber at age 12 years had significantly lower systolic BP 5 years later during adolescence.

We demonstrate that each 1-SD increase in dietary GI and GL over 5 years was associated with a concurrent increase of ~2 and 4 mm Hg in mean systolic BP in girls, respectively. Several intervention studies have shown that lowering dietary GI lowers BP in adults; however, independent associations between dietary GI and GL with BP have not been observed previously in adolescents. In our cohort, girls consuming high-GI/GL diets could have had greater postprandial hyperinsulinemia than their peers who consumed lower amounts of refined carbohydrates. Insulin is known to activate the sympathetic nervous system and is a potential mediator of sodium retention and volume expansion and, thus, higher BP. Additional prospective studies of adolescents with larger sample size will, nevertheless, be needed to determine the importance of our observation and to delineate underlying mechanisms.

The independent associations reported here for sugars and BP concur with the hypothesized effects on the uric acid pathway. It appears that fructose is the more damaging sugar component in terms of cardiovascular risk. Fructose might increase BP by raising serum uric acid, which can reduce endothelial NO and activate the renin-angiotensin system. Another pathway could be a reduction in sodium excretion and/or enhancement of sympathetic nervous system activity, after ingestion of sugars.

It was surprising that we did not observe a temporal association between soft drink consumption and BP, particularly because previous population-based studies in adoles-

Table 4. Changes in Dietary Intake of Carbohydrate Nutrition Variables With Concurrent Change in Mean BP in Millimeters of Mercury From Age 12 to 17 y Among Girls (n=278)

<table>
<thead>
<tr>
<th>Nutritional Intake (per SD Increase)</th>
<th>Concurrent Change in Mean Systolic BP*</th>
<th>Concurrent Change in Mean Diastolic BP*</th>
<th>Concurrent Change in Mean Arterial BP*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( \beta ) ( \pm ) SE ( P )</td>
<td>( \beta ) ( \pm ) SE ( P )</td>
<td>( \beta ) ( \pm ) SE ( P )</td>
</tr>
<tr>
<td>Glycemic index (1-SD=3.55)</td>
<td>2.30 ( (0.78) ) 0.003 0.29</td>
<td>1.20 ( (0.61) ) 0.05</td>
<td></td>
</tr>
<tr>
<td>Glycemic load (1-SD=50.89)</td>
<td>5.74 ( (1.67) ) 0.001 0.06</td>
<td>3.56 ( (1.31) ) 0.01</td>
<td></td>
</tr>
<tr>
<td>Carbohydrate (1-SD=92.37)</td>
<td>6.97 ( (2.07) ) 0.001 0.05</td>
<td>4.46 ( (1.62) ) 0.01</td>
<td></td>
</tr>
<tr>
<td>Total sugars, (1-SD=51.70)</td>
<td>3.33 ( (1.35) ) 0.01 0.02</td>
<td>2.73 ( (1.06) ) 0.01</td>
<td></td>
</tr>
<tr>
<td>Fructose (1-SD=14.19)</td>
<td>2.29 ( (0.97) ) 0.02 0.05</td>
<td>1.79 ( (0.76) ) 0.02</td>
<td></td>
</tr>
<tr>
<td>Added sugars, (1-SD=27.63)</td>
<td>1.47 ( (1.04) ) 0.16 0.04</td>
<td>1.64 ( (0.81) ) 0.04</td>
<td></td>
</tr>
</tbody>
</table>

\( \beta \) indicates blood pressure. \( \beta \)-Coefficients refer to each SD increase in baseline nutritional intake with concurrent decrease/increase in mean BP.

\( ^* \)Data show unadjusted analyses.
There is evidence to suggest that it is the sweetened with high-fructose corn syrup (a mixture of glucose and fructose). In contrast, soft drinks in the United States, unlike in Australia, are primarily sweetened with high-fructose corn syrup component of soft drinks that mediates many of its associations with adverse cardiovascular outcomes. Alternatively, we cannot rule out that the lack of an association could be attributed to greater misclassification of soft drink intake because of differential underreporting of soft drink consumption in this cohort. Other longitudinal adolescent studies with adequate study power are needed to clarify the elevated mechanisms. In addition, the physical activity questions were not validated; however, an alternate validated instrument, such as accelerometry, was not feasible given the large size of our study. In addition, because we had also adjusted for time spent in both physical and sedentary pursuits and still observed an increase in BP for high GI/GL intake. Nevertheless, we cannot exclude residual confounding from unmeasured or unknown factors. Another limitation is that we did not measure blood glucose levels and inflammatory markers, which could have been useful to clarify the elevated mechanisms. In addition, the physical activity questions were not validated; however, an alternate validated instrument, such as accelerometry, was not feasible given the large size of our study. In addition, because we tested a large number of associations, false-positive relations are a possibility. Finally, ~50% of baseline participants were not followed up 5 years later, and participants differed from nonparticipants in study characteristics, such as age, ethnicity, and BMI. Therefore, we cannot rule out the possibility of selection bias, which limits the generalization of our results.

Table 5. Changes in Dietary Intake of Carbohydrate Nutrition Variables With Concurrent Change in Mean BP in Millimeters of Mercury From Age 12 to 17 y Old Among Girls (n=278) After Multivariable Adjustment

<table>
<thead>
<tr>
<th>Nutritional Intake (per SD Increase)</th>
<th>Concurrent Change in Mean Systolic BP*</th>
<th>Concurrent Change in Mean Diastolic BP*</th>
<th>Concurrent Change in Mean Arterial BP*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycemic index (1-SD=3.55)</td>
<td>1.81 (0.55) 0.001</td>
<td>0.79 (0.43) 0.07</td>
<td>1.12 (0.37) 0.003</td>
</tr>
<tr>
<td>Glycemic load (1-SD=50.89)</td>
<td>4.02 (1.42) 0.01</td>
<td>2.63 (0.88) 0.003</td>
<td>3.07 (0.90) 0.001</td>
</tr>
<tr>
<td>Carbohydrate (1-SD=92.37, g/d)</td>
<td>4.74 (1.87) 0.01</td>
<td>3.27 (1.02) 0.001</td>
<td>3.72 (1.13) 0.001</td>
</tr>
<tr>
<td>Total sugars, (1-SD=51.70, g/d)</td>
<td>2.28 (1.17) 0.05</td>
<td>2.15 (0.66) 0.001</td>
<td>2.19 (0.73) 0.003</td>
</tr>
<tr>
<td>Fructose (1-SD=14.19, g/d)</td>
<td>1.80 (0.82) 0.03</td>
<td>1.67 (0.61) 0.01</td>
<td>1.69 (0.56) 0.003</td>
</tr>
<tr>
<td>Added sugars, (1-SD=27.63, g/d)</td>
<td>1.24 (0.73) 0.09</td>
<td>1.31 (0.57) 0.02</td>
<td>1.33 (0.56) 0.02</td>
</tr>
</tbody>
</table>

BP indicates blood pressure. *Coefficients refer to each SD increase in nutritional intake with a concurrent decrease/increase in mean BP.

Data were adjusted for age, ethnicity, parental education, parental history of hypertension, energy intake (residual method), baseline BP, change in height, change in body mass index, screen viewing time, and time spent in physical activity.
adolescence. These associations were independent of the effects of obesity and/or parental history of hypertension. Therefore, our findings suggest that, regardless of weight status or genetic risk, replacing refined carbohydrates and sugars with high-fiber, low-GI foods could be a potentially important dietary strategy to reduce BP during adolescence. Nevertheless, future studies involving larger samples and longer follow-up are required to both confirm and provide additional insights into the clinical significance of our observations.

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Disclosures
None.

References
Online Supplement

Influence of High Glycemic Index and Glycemic Load Diets on Blood Pressure during Adolescence

Running title: Carbohydrate nutrition and blood pressure

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Table S1 Multivariable-adjusted linear association between baseline dietary intake of fiber and fruits with change in blood pressure (BP) from age 12 to 17 years among boys (n=432)

<table>
<thead>
<tr>
<th>Nutritional intake (per SD increase) at baseline, g/day</th>
<th>Change in mean systolic BP *</th>
<th>Change in mean diastolic BP *</th>
<th>Change in mean mean arterial BP *</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β (SE)</td>
<td>P</td>
<td>β (SE)</td>
</tr>
<tr>
<td>Total fiber (1-SD = 7.10)</td>
<td>0.41 (0.46)</td>
<td>0.37</td>
<td>0.38 (0.34)</td>
</tr>
<tr>
<td>Fruit fiber (1-SD = 3.77)</td>
<td>0.46 (0.47)</td>
<td>0.33</td>
<td>0.23 (0.32)</td>
</tr>
<tr>
<td>Vegetable fiber (1-SD = 3.99)</td>
<td>0.73 (0.57)</td>
<td>0.20</td>
<td>0.57 (0.50)</td>
</tr>
<tr>
<td>Fruits (1-SD = 181.7)</td>
<td>0.38 (0.50)</td>
<td>0.45</td>
<td>0.20 (0.35)</td>
</tr>
</tbody>
</table>

SD – Standard Deviation; SE – Standard Error. β-coefficients refer to each SD increase in baseline nutritional intake with a decrease/increase in mean BP 5 years later.

*Adjusted for age, ethnicity, parental education, parental history of hypertension, energy intake (residual method), baseline height, baseline BP, change in body mass index, screen viewing time and time spent in physical activity.
Table S2 Changes in dietary intake of carbohydrate nutrition variables with concurrent change in mean blood pressure (BP) in mm Hg from age 12 to 17 years old among boys (n=231), after multivariable adjustment.

<table>
<thead>
<tr>
<th>Nutritional intake (per SD increase)</th>
<th>Concurrent change in mean systolic BP*</th>
<th>Concurrent change in mean diastolic BP*</th>
<th>Concurrent change in mean arterial BP*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glycemic index (1-SD = 3.55)</td>
<td>0.97 (0.85) 0.22</td>
<td>0.39 (0.62) 0.54</td>
<td>-0.17 (0.47) 0.73</td>
</tr>
<tr>
<td>Glycemic load (1-SD = 50.89)</td>
<td>-1.84 (0.92) 0.06</td>
<td>1.26 (0.91) 0.17</td>
<td>0.18 (0.75) 0.81</td>
</tr>
<tr>
<td>Carbohydrate (1-SD = 92.37), g/day</td>
<td>-1.49 (1.22) 0.22</td>
<td>1.72 (0.97) 0.08</td>
<td>0.61 (0.78) 0.44</td>
</tr>
<tr>
<td>Total sugars, (1-SD = 51.70), g/day</td>
<td>0.75 (0.84) 0.38</td>
<td>0.89 (0.66) 0.18</td>
<td>0.89 (0.55) 0.11</td>
</tr>
<tr>
<td>Fructose (1-SD = 14.19), g/day</td>
<td>0.81 (0.73) 0.27</td>
<td>0.34 (0.60) 0.57</td>
<td>0.54 (0.56) 0.33</td>
</tr>
<tr>
<td>Added sugars, (1-SD = 27.63), g/day</td>
<td>-0.46 (0.93) 0.62</td>
<td>0.18 (0.57) 0.76</td>
<td>-0.05 (0.65) 0.94</td>
</tr>
</tbody>
</table>

SD – Standard Deviation; SE – Standard Error. β-coefficients refer to each SD increase in nutritional intake with a concurrent decrease/increase in mean BP.

*Adjusted for age, ethnicity, parental education, parental history of hypertension, energy intake (residual method), baseline BP, change in height, change in body mass index, screen viewing time and time spent in physical activity