Intake of Added Sugar and Sugar-Sweetened Drink and Serum Uric Acid Concentration in US Men and Women

Xiang Gao, Lu Qi, Ning Qiao, Hyon K. Choi, Gary Curhan, Katherine L. Tucker, Alberto Ascherio

Abstract—Fructose-induced hyperuricemia might have a causal role in metabolic syndrome, hypertension, and other chronic disease. However, no study has investigated whether sugar added to foods or sugar-sweetened beverages, which are major sources of fructose, are associated with serum uric acid concentration in free-living populations. We examined the relationship between the intakes of added sugars and sugar-sweetened beverages and serum uric acid concentrations in the National Health and Nutrition Examination Survey 2001–2002, a nationally representative sample of men and women. We included 4073 subjects (1988 men and 2085 women) >18 years of age in the current study. Dietary intake was assessed by a single 24-hour recall. We used multivariate linear regression to adjust for age, gender, intake of energy and alcohol, body mass index, use of diuretics, β-blockers, and other covariates. Male subjects in the highest intake quartile of estimated intake of added sugars or sugar-sweetened drinks had higher plasma uric acid concentrations than those in the lowest intake quartiles (P<0.001 for both) after adjusting for potential confounders, whereas we did not observe significant associations for females (P for trend>0.2; P for interaction <0.01). Further research is needed to confirm causality of these associations and the observed difference by gender. (Hypertension. 2007;50:306-312.)

Key Words: uric acid □ beverages □ added sugar □ fructose □ National Health and Nutrition Examination Survey

Increasing consumption of fructose has coincided with the increasing incidence of obesity and the metabolic syndrome over the past 2 decades.1 Unlike glucose and other sugars, fructose intake raises serum uric acid, presumably through induction of catabolism of nucleotides or reduction of uric acid excretion.2–4 Meanwhile, a growing body of evidence has demonstrated an association of hyperuricemia with hypertension, systemic inflammation, insulin resistance, obesity, dyslipidemia, and cardiovascular disease.5–9 Animal studies have suggested a possible causal role of fructose on hyperuricemia10–12 and metabolic syndrome,4,13 whereas hyperuricemia induced by greater fructose intake could be an important mechanism underlying the associations.11,13

In the United States, the largest single source of fructose in the diet is from added sugars, accounting for approximately two thirds of total fructose intake according to the National Health and Nutrition Examination Survey (NHANES) 1999–2000.14,15 Added sugars are sugars and syrups that are not naturally present in foods, including sugar, corn syrup, and high-fructose corn syrup, which are added to foods during processing or preparation.16,17 Half of added sugars consumed in the United States were in the form of high-fructose corn syrup, a major sweetener in sugar-sweetened beverages (soft drinks and fruit drinks).14,18 The NHANES 1999–2000 showed that sweetened soft drinks were the number 1 energy contributor in the United States. Soft drinks and fruit drinks together provided 10.6% of estimated total energy, followed by sweetened pastries (3.6%) and hamburgers (3.1%).14 However, to our knowledge, no study has investigated whether added sugar in foods or sugar-sweetened beverages is associated with serum uric acid concentration in free-living populations.

Therefore, we examined the relationship between reported intakes of added sugars and sugar-sweetened beverages and serum uric acid concentrations in the NHANES 2001–2002. Because earlier evidence has suggested that sex hormones may modulate the metabolic response induced by fructose,19,20 we also examined this relationship by gender.

Subjects and Methods

Subjects

Subjects were 4994 men and women aged >18 years from the NHANES 2001–2002. As we did previously21,22 we excluded the following subjects: (1) those who were pregnant (n=309); (2) those who reported energy intake <2.5 MJ/d (600 kcal/d) or >16.7 MJ/d (4000 kcal/d; n=334); and (3) those who did not have a serum uric acid measurement (n=245). We also excluded subjects who reported use of drugs affecting uric acid (allopurinol or uricosuric agents, n=33). We, therefore, included 4073 subjects (1988 men and 2085 women).
women) in the analysis. Exclusion of subjects with extreme energy intake is commonly used in nutritional epidemiologic studies,23,24 because implausible reported energy intake suggest likely inaccurate estimation of other dietary exposure intakes, ie, added sugar or drinks in this case.

Assessment of Dietary Intakes and Covariates

In the NHANES 2001–2002, dietary intake was measured with a single 24-hour recall from each participant. Data were available from 2 data sets: individual food files25 which provided nutrient data (eg, total energy and alcohol), and pyramid servings intake data for NHANES,26 which provided standard serving size information for 5 major pyramid food groups (grains, dairy, fruit, vegetables, meat, and beans), as well as added sugar and discretionary fat (ie, excess fat). Details about calculation of added sugar are described elsewhere.27

Information on age, gender, race/ethnicity, body measurements (including weight and height), smoking status, vitamin and mineral supplement use, medication use (including diuretics, β-blockers, aspirin, and nonsteroidal anti-inflammatory drugs), and medical conditions (including self-reported diabetes) was collected by self-report. Body mass index (BMI) was calculated as weight (kilograms)/height (meters)². Subjects who had smoked ≥100 cigarettes in their lifetimes were considered never smokers; subjects who had smoked ≥100 cigarettes in their lifetimes were considered former smokers if they answered negatively to the question, “Do you smoke now?” and current smokers if they answered affirmatively.28 Subjects were identified as having diabetes if they reported that they had diabetes or that they used medications for diabetes (insulin or oral medicines). Hypertension was defined as systolic blood pressure ≥140 mm Hg, and/or diastolic blood pressure ≥90 mm Hg, and/or use of medicine for hypertension.

Serum Uric Acid Measurement

Serum uric acid concentrations were measured with a Beckman Synchrontm LX20 using a timed end point method. Uric acid is oxidized by uricase to produce allantoin and hydrogen peroxide. The hydrogen peroxide reacts with 4-aminophthalein and 3,5-dichloro-2-hydroxybenzene sulfonate in a reaction catalyzed by peroxidase to produce a colored product. The system monitors the change in absorbance at 520 nm at a fixed time interval. The change in absorbance is directly proportional to the concentration of uric acid in the sample.29

Statistical Analyses

All of the statistical analyses were performed with SUDAAN, version 9.0 (Research Triangle Institute) using techniques appropriate to the complex survey design of NHANES 2001–2002. All of the analyses used NHANES 2001–2002 sample weights so that results are representative of the US community-dwelling population. All of the P values are 2 sided. Dietary variables were analyzed in quartiles of the whole population. This conservative approach has the advantage that it greatly reduces the chance that a small number of extreme observations will have undue influence on the results. Because a large proportion of subjects did not report sugar-sweetened drinks or fruit juice, we assigned them into the lowest intake quartile. We then evenly divided the rest subjects into 3 categories based on reported intakes and treated them as quartiles 2 to 4. A linear regression model was used to evaluate associations between dietary variables and serum uric acid. Linear trends were tested for significance by using the median value for each quartile of intake and treating this value as a continuous variable. We adjusted for age (year), gender, smoking (never, past, current: 1 to 15 cigarettes per day), BMI, ethnicity, hypertension (yes/no), diabetes (yes/no), intake of total energy (megajoules per day), alcohol (grams per day), dietary vitamin C (milligrams per day), fruit (servings per day), vegetables (servings per day), meat and seafood (servings per day), whole grains (servings per day), and use of antioxidant supplements (vitamin E, vitamin C, and carotenoids), β-blockers, or diuretics (each yes/no).

We also examined whether correlations of added sugar and sweetened drink intakes with serum uric acid were modified by gender, alcohol (none versus >0 g/d), smoking status (never versus ever), and BMI (<25 versus ≥25 kg/m²). To test these correlations, we included multiplicative terms in the linear regression models with adjustment for other potential confounders. To examine robustness of the results, we conducted sensitivity analyses by excluding subjects with hypertension or diabetes. Because losartan has been shown in humans to decrease serum uric acid30 and to prevent fructose-induced hypertension in the rat model,31 we also did a sensitivity analysis excluding subjects who reported use of losartan.

Results

There was a 10-fold difference in the median added sugar intake between the highest and lowest quartiles (Table 1). Those with the greatest added sugar intakes tended to be younger, male, and current smokers; were less likely to have diabetes and hypertension; were less likely to report use of antioxidant supplements or the assessed antihypertensive medicines; and had higher intakes of total energy and meat and lower intakes of fruit and alcohol relative to those in the lowest intake quartile.

Subjects with the highest added sugar intake had significantly higher serum uric acid concentrations relative to those with the lowest intake quartile (329 versus 317 μmol/L; P=0.05), after adjustment for smoking; BMI; intake of total energy, dairy, and alcohol; hypertension; and other potential confounders (Table 2). Exclusion of participants with diabetes or hypertension did not change the associations (P for trend <0.05 for both). Similar significant associations were seen after excluding subjects who reported use of losartan (data not shown). Consistent with the results on added sugars, subjects in the highest sweetened drinks intake category had significantly higher serum uric acid relative to those in the lowest intake category; the difference in uric acid concentration was 22 μmol/L (P=0.0006). We further examined associations between intakes of 2 major sugar-sweetened drinks, soft drinks and fruit drinks, and serum uric acid. We found a significant positive association for soft drinks (P for trend=0.002) but not for fruit drinks (P for trend=0.30). There was no significant relationship between fruit juice intake and serum uric acid. We further examined the associations between intake of citrus juice and noncitrus juice and serum uric acid, but neither was significant. Intake of apples and pears, which are rich sources of naturally occurring fructose, was also not significantly associated with serum uric acid (data not shown). Further adjustment for coffee intake did not materially change any of the findings. Because sex hormones may modulate the metabolic response induced by fructose, we further explored possible interactions of added sugar or beverage intake with gender in relation to serum uric acid concentrations. Associations between added sugar or beverage intake and plasma uric acid concentration were significantly modified by gender (P for interaction <0.01 for both). Greater intake of added sugar or sweetened drinks was significantly associated with plasma uric acid among men (P for trend=0.0008 for added sugar and 0.0003 for sweetened drinks) but not among women (P for trend >0.2 for both; Figure). We did not see significant associations between intakes of apples and pears or of fruit juice and serum uric acid concentrations in men (P for trend...
No significant interactions were found with age, smoking status, or BMI ($P$ for interaction $<0.05$ for all) compared with serum uric acid.

**Discussion**

In this national sample of men and women, we found that greater intake of added sugars or sugar-sweetened drinks was associated with higher plasma uric acid concentration in men but not in women ($P$ for interaction $<0.01$). Intake of fruit juice, which contains a large amount of naturally occurring sugar, was not associated with plasma uric acid.

These findings could have important clinical and public health implications. High serum uric acid has been suggested as a possible risk factor for hypertension, metabolic syndrome, and cardiovascular disease, independent of the traditional risk factors.\textsuperscript{4,7,32} Associations between hyperuricemia and hypertension have been reported in several studies.\textsuperscript{33–35} Prospective data within 1 study indicated that hyperuricemia precedes onset of hypertension.\textsuperscript{34} Several studies, in vivo and in vitro, have examined the pathophysiological links between hyperuricemia and hypertension. Hyperuricemia has been associated with higher renal vascular and total peripheral resistances.\textsuperscript{36} In 1 study, uric acid infusion in humans led to impaired endothelial NO release, which was reversed by allopurinol, which inhibits uric acid production.\textsuperscript{5} Hyperuricemia may also increase salt retention.\textsuperscript{37} In addition, elevated uric acid concentration is associated with insulin resistance, high triglyceride levels, and chronic inflammation,\textsuperscript{4–6,8,38,39} all of which may affect the development of hypertension.\textsuperscript{40,41}

On the other hand, consumption of added sugars, especially high-fructose corn syrup, has been increasing rapidly during the past few decades in the United States. Economic disappearance data have shown that, from 1970 to 2000, the average intake of added sugars increased from $\approx 64$ to $\approx 80$ kg/y per person, whereas high-fructose corn syrup has increased $>100$-fold, from 0.3 to 33 kg/y per person.\textsuperscript{1,15} However, because economic disappearance data come from measuring the amount of all the foods that were produced or imported and then subtracting all the nonfood uses, consumption of added sugars could be overestimated. Using the same

\begin{table}
\centering
\caption{Classification of the Characteristics of the Study Population by Estimated Added Sugar Quartiles in the NHANES 2001–2002}
\begin{tabular}{lcccc}
\hline
Characteristic & Q1 (n=1018) & Q2 (n=1019) & Q3 (n=1018) & Q4 (n=1018) \\
\hline
Median intake (range), g/d & 15.0 & 50.0 & 85.8 & 157 \\
Age, y & 50.2±0.7 & 50.0±0.8 & 45.3±1.1 & 38.6±0.6 \\
Female, % & 57.5 & 57.1 & 49.5 & 40.1 \\
BMI* & 28.1±0.3 & 27.8±0.3 & 27.8±0.3 & 28.0±0.2 \\
Ethnicity, % & & & & \\
Non-Hispanic white & 53.2 & 55.7 & 51.9 & 47.8 \\
Non-Hispanic black & 14.5 & 15.6 & 19.8 & 24.6 \\
Mexican American & 23.1 & 20.7 & 21.6 & 21.7 \\
Other Hispanic & 4.4 & 5.2 & 3.8 & 3.4 \\
Asian and other ethnicity & 4.8 & 2.8 & 2.9 & 2.5 \\
Smoking status, % & & & & \\
Never & 57.4 & 60.1 & 57.0 & 50.5 \\
Past & 24.9 & 21.6 & 20.8 & 17.7 \\
Current & 17.7 & 18.3 & 22.2 & 31.8 \\
Total energy, kcal/d* & 1706±29 & 1922±28 & 2085±31 & 2542±26 \\
Alcohol, g/d* & 13.1±1.4 & 11.3±1.0 & 8.0±0.9 & 7.8±1.4 \\
Dietary vitamin C, mg/d* & 85.4±4.0 & 89.7±2.9 & 90.1±4.0 & 93.5±5.9 \\
Fruit, servings per day* & 1.3±0.1 & 1.1±0.05 & 1.0±0.06 & 1.0±0.07 \\
Vegetables, servings per day* & 3.2±0.1 & 3.3±0.1 & 3.0±0.1 & 3.2±0.1 \\
Dairy, servings per day* & 1.3±0.05 & 1.3±0.06 & 1.5±0.09 & 1.5±0.06 \\
Meat and seafood, servings per day* & 4.5±0.1 & 4.6±0.1 & 4.5±0.1 & 4.8±0.1 \\
Whole grain, servings per day* & 0.8±0.07 & 0.8±0.06 & 0.9±0.05 & 0.8±0.04 \\
Use of antioxidant supplement, % & 26.2 & 26.7 & 22.2 & 15.4 \\
Use of $\beta$-blocker, % & 11.5 & 9.4 & 8.0 & 3.9 \\
Use of diuretics, % & 16.4 & 15.0 & 11.8 & 6.5 \\
Diabetes, % & 19.4 & 8.5 & 7.2 & 3.0 \\
Hypertension, % & 49.1 & 39.7 & 33.2 & 23.8 \\
\hline
\end{tabular}
\footnotesize{*Mean±SE, adjusted for age and gender.}
\end{table}
may cause hypertension, whereas a high-fructose diet has a pathogenetic role in hypertension: overloaded fructose serum uric acid are also consistent with previous trials. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose administration of allopurinol, which lowered serum uric acid. "

TABLE 2. Serum Uric Acid Concentration (Micromoles per Liter) According to Intake Quartile of Added Sugar, Sugar-Sweetened Drinks, and Fruit Juice in the NHANES 2001–2002

<table>
<thead>
<tr>
<th>Food Items</th>
<th>Quartile of Intake</th>
<th>( P_{\text{trend}} )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Q1</td>
<td>Q2</td>
</tr>
<tr>
<td>Added sugar</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>1018</td>
<td>1019</td>
</tr>
<tr>
<td>Median intake, g/d</td>
<td>15.0</td>
<td>25.0</td>
</tr>
<tr>
<td>Estimated fructose intake, g/d</td>
<td>321 ± 4</td>
<td>319 ± 4</td>
</tr>
<tr>
<td>Model 1*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Model 2†</td>
<td>317 ± 4</td>
<td>319 ± 3</td>
</tr>
<tr>
<td>Excluding subjects with diabetes</td>
<td>316 ± 4</td>
<td>319 ± 3</td>
</tr>
<tr>
<td>Excluding subjects with hypertension</td>
<td>304 ± 4</td>
<td>309 ± 4</td>
</tr>
<tr>
<td>Sugar-sweetened drinks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>1807</td>
<td>748</td>
</tr>
<tr>
<td>Median intake, servings per day</td>
<td>0</td>
<td>1.3</td>
</tr>
<tr>
<td>Estimated fructose intake, g/d¶</td>
<td>0</td>
<td>17.5</td>
</tr>
<tr>
<td>Model 1*</td>
<td>315 ± 3</td>
<td>319 ± 4</td>
</tr>
<tr>
<td>Excluding subjects with diabetes</td>
<td>315 ± 2</td>
<td>321 ± 3</td>
</tr>
<tr>
<td>Excluding subjects with hypertension</td>
<td>303 ± 3</td>
<td>313 ± 5</td>
</tr>
<tr>
<td>Fruit juice</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>3035</td>
<td>340</td>
</tr>
<tr>
<td>Median intake, servings/d</td>
<td>0.66</td>
<td>1.32</td>
</tr>
<tr>
<td>Estimated fructose intake, g/d§</td>
<td>0</td>
<td>5.0</td>
</tr>
<tr>
<td>Model 1*</td>
<td>322 ± 3</td>
<td>317 ± 5</td>
</tr>
<tr>
<td>Excluding subjects with diabetes</td>
<td>321 ± 3</td>
<td>313 ± 5</td>
</tr>
<tr>
<td>Excluding subjects with hypertension</td>
<td>310 ± 3</td>
<td>308 ± 8</td>
</tr>
</tbody>
</table>

*Adjusted for age, gender, and total energy (kilocalories per day).
†Adjusted for age, gender, smoking (never, past, current: 1 to 14 and ≥15 per day), BMI, ethnicity, hypertension (yes/no), diabetes (yes/no), alcohol (grams per day), intake of total energy (kilocalories per day), vitamin C (milligrams per day), fruit (servings per day), vegetable (servings per day), meat and seafood (servings per day), whole grain (servings per day), and use of antioxidant supplement (vitamin E, vitamin C, or carotenoids), \( \beta \)-blockers, or diuretics (each yes/no).
‡Based on regular coca cola.
§Based on regular orange juice. The amount increased to 0, 7.6, 15.2, and 30.6 g/d, respectively, if based on apple juice. 

NHANES data, we have shown previously that careful restriction of added sugar and fat intake is a key for maintenance of appropriate energy intake of Americans.

Our findings that greater intake of foods with high-fructose content correlates with elevated concentrations of uric acid are consistent with a recent animal study in which high-fructose intake induced elevated blood pressure and metabolic syndrome in rats; these effects were partially prevented by administration of allopurinol, which lowered serum uric acid. Several animal studies have demonstrated that fructose has a pathogenetic role in hypertension: overloaded fructose may cause hypertension, whereas a high-fructose diet increased mortality in hypertensive rats. Associations between intakes of added sugar and sugar-sweetened drinks and serum uric acid are also consistent with previous trials, where the administration of a high dose of fructose, either intravenously or orally, led to increases in serum uric acid concentration.

Use of ATP in the phosphorylation of fructose and sequestration of phosphate in fructose-1-phosphate could be an underlying mechanism by which greater intake of added sugar, especially of high-fructose corn syrup in sweetened drinks, is associated with high serum uric acid concentrations. ATP and phosphate are inhibitors of enzymes of adenine nucleotide degradation (eg, 5'-nucleotidase and adenosine monophosphate deaminase). Removal of this inhibition, may, therefore, increase the generation of uric acid. Greater intake of fructose has also been shown to decrease uric acid excretion because of an association with increased lactate production.
sucrose could stimulate long-chain fatty acid synthesis and lead to hypertriglyceridemia and increased insulin resistance. Hypertriglyceridemia and insulin resistance have been shown to be associated with high serum uric acid concentration.

We found that added sugar intake was significantly associated with greater serum uric acid in men but not in women, which is consistent with the experimental observation that fructose feeding only leads to hypertension in male rats. This could be because of differences in sex hormones, because fructose-induced hypertension in male rats was prevented by estrogen treatment, whereas ovariectomy of female rats had higher blood pressure when fed with fructose, relative to ovary-intact rats. Several human studies reported a modest hypouricemic effect of estrogen, which may be because of increased uricosuria. Purine nucleotide metabolism is affected by testosterone. Animal studies showed that castration of male rats decreased synthesis of nucleotides, guanosine monophosphate, and adenosine monophosphate and nucleotide catabolism, which was restored by administration of testosterone. In a sample of obese children aged 10 to 16 years, Denzer et al reported a significant positive association between plasma testosterone and uric acid concentrations. Furthermore, animal studies have demonstrated gender differences in uric acid transporters, such as urate transporter 1 and organic anion transport. The expression of these transporters is higher in male mice than in female mice. Therefore, male mice have a higher reabsorption of uric acid than female mice. Interestingly, epidemiologic studies have shown that dietary fructose was associated with disadvantageous plasma lipid profiles in men but not in women. A series of animal studies has demonstrated that effects of fructose on plasma insulin and insulin resistance differed by gender. Fructose feeding had a deteriorative effect only in male rats but not in female rats. As discussed above, blood lipid and insulin status are associated with serum uric acid. These data, together with our study, suggest that sex hormones or other gender-related factors may influence fructose or uric acid metabolism. However, exact mechanisms underlying the gender difference in the association between fruc-

![Graph A](image1.png)

**Difference in serum uric acid concentration according to quartile of intake of added sugar (A) and sugar-sweetened drink (B), stratified by gender. Adjusted for age, smoking (never, past, current: 1 to 14 and ≥15 per day), BMI, ethnicity, hypertension (yes/no), diabetes (yes/no), alcohol (grams per day), intakes of total energy (kilocalories per day), vitamin C (milligrams per day), vegetable (servings per day), meat and seafood (servings per day), whole grain (servings per day), and use of antioxidant supplement (vitamin E, vitamin C, or carotenoids), β-blocker, or diuretics (each yes/no).**

![Graph B](image2.png)
tose and uric acid remain to be elucidated. Further investigation of these may increase our understanding of uric acid metabolism.

We did not observe a significant association between intake of fruit juice, a major source of naturally occurring fructose, and serum uric acid concentrations. One could speculate that fruit juice is also a good source of antioxidants, including vitamin C and carotenoids. Vitamin C has been shown to have an uricosuric effect, ie, increasing uric acid excretion.61,62 These components could offset the deleterious effects of fructose. Another possible explanation for this discrepancy is that, in the current population, variation in fruit juice intake was smaller than that of sugar-sweetened drink intake; the difference between the 2 extreme fruit juice intake quartiles was 2.7 servings per day relative to 5.7 servings per day of sugar-sweetened drinks.

Our study has several limitations. We cannot claim causality because of the cross-sectional nature of this study. Subjects may change their diets because of conditions related to elevated serum uric acid concentrations. However, we obtained similar significant associations after exclusion of those with hypertension or diabetes. A single 24-hour recall was used to assess dietary intake. This method may not accurately estimate the true variation in added sugar intake in the population. Furthermore, it cannot capture the long-term dietary intake pattern for each subject because of high day-to-day variation. The resulting misclassification in exposure measurement may lead to an inaccurate estimation of the associations between added sugars and serum uric acid.

**Perspectives**

We found positive associations between intake of added sugar and sweetened drinks and serum uric acid concentration in men but not in women. If the association is causal, it could imply a potential method to prevent certain chronic diseases, such as gout and cardiovascular disease. In addition, an individual with high dietary fructose intake might need closer screening for the onset of cardiovascular disease. Associations of intake of added sugar and sweetened drinks with serum uric acid and possibly with hypertension and metabolic syndrome are needed to be confirmed by well-designed clinical trials. Further research is also needed to understand the differences in association between hyperuricemia and high dietary intake of fructose in men and women.

**Sources of Funding**

The study was supported by National Institutes of Health/National Institute of Neurological Disorders and Stroke grant R01 NS048517 and US Department of Agriculture contract 58-1950-7-707.

**Disclosures**

None.

**References**


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Hypertension. 2007;50:306-312; originally published online June 25, 2007;
doi: 10.1161/HYPERTENSIONAHA.107.091041

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