Sugar-sweetened beverage consumption and incident hypertension: a systematic review and meta-analysis of prospective cohorts\textsuperscript{1–3}

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ABSTRACT

Background: The role of sugar-sweetened beverages (SSBs) that contain free or bound fructose in the pathogenesis of hypertension remains unclear.

Objective: We conducted a systematic review and meta-analysis of prospective cohort studies to quantify the association between fructose-containing SSBs and risk of hypertension.

Design: MEDLINE, Embase, Cumulative Index to Nursing and Allied Health Literature, and the Cochrane registry were searched from conception through 11 November 2014. Two independent reviewers extracted data and assessed the quality of studies (with the use of the Newcastle-Ottawa Scale). Risk estimates of extreme quantities of SSB intake (lowest vs. highest) for hypertension incidence were generated with the use of generic inverse-variance methods with random-effects models and expressed as risk ratios with 95% CIs. Heterogeneity was assessed with the Cochran $Q$ statistic and quantified with the $I^2$ statistic.

Results: Six prospective cohort studies ($n = 240,508$) with 79,251 cases of hypertension observed over $\geq 3,197,528$ person-years of follow-up were included. SSB consumption significantly increased the risk of developing hypertension by 12% (risk ratio: 1.12; 95% CI: 1.06, 1.17) with evidence of significant heterogeneity ($I^2 = 62\%$, $P = 0.02$) when highest ($\geq 1$-serving [6.7, 8 or 12 oz/d]) and lowest (none) quantities of intake were compared. With the use of a dose-response analysis, a significant 8.2% increase in risk of every additional SSB per day from none to $\geq 1$ SSB/d ($\beta = 0.0027$, $P < 0.001$) was identified. Limitations include unexplained heterogeneity and residual confounding. The results may also have been subject to collinearity effects from aspects of a Western dietary pattern.

Conclusions: SSBs were associated with a modest risk of developing hypertension in 6 cohorts. There is a need for high-quality randomized trials to assess the role of SSBs in the development of hypertension and its complications. This study was registered at clinicaltrials.gov as NCT01608620.


Keywords: hypertension, meta-analysis, prospective cohort, sugars, sugar-sweetened beverage

INTRODUCTION

Hypertension is a major risk factor for cardiovascular disease and accounts for 9.4-million deaths annually, worldwide (1). Nevertheless, hypertension risk remains highly modifiable through dietary and lifestyle interventions (2). Recent attention has implicated sugar-sweetened beverage (SSB)\textsuperscript{12} intake in the pathogenesis of hypertension, as well as other chronic diseases including obesity and diabetes (3). Public health initiatives that aimed to reduce SSB intakes have included recommendations for front-of-package warning labels in Canada (4), SSB portion-size restrictions in New York City (5), and the introduction of a SSB tax in the United Kingdom (6).

The fructose from sucrose and high-fructose corn syrup within SSBs has been proposed as the primary driver of these associations

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\textsuperscript{2}Supplemental Figures 1 and 2 and Supplemental Tables 1–3 are available from the “Supplemental data” link in the online posting of the article and from the same link in the online table of contents at http://ajcn.nutrition.org.

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\textsuperscript{12}Abbreviations used: CARDIA, Coronary Artery Risk Development in Young Adults Study; GLST, generalized least-squares trend estimation; HPFS, Health Professionals Follow-Up Study; NHS-I, Nurses’ Health Study I; NHS-II, Nurses’ Health Study II; SSB, sugar-sweetened beverage; SUN, Seguimiento Universidad de Navarra Project.

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It has been suggested that fructose, through the depletion of ATP that leads to an elevation of uric acid, induces vascular oxidative stress, endothelial dysfunction, and aggravates the renin-angiotensin axis, consequently elevating blood pressure (8). Animal studies and select human trials of overfeeding of fructose-containing sugars as well as low-quality observational studies have been used to buttress this relation (10). However, higher-quality evidence from systematic reviews and meta-analyses of controlled feeding trials on the effects of monosaccharide fructose on blood pressure (11) and uric acid (12) compared with other calorically matched carbohydrates and prospective cohorts of the relation between fructose intake and hypertension risk (13) have not supported this association. Despite the uncertainties, there is mounting pressure on guideline committees to implicate fructose-containing SSBs in the development of hypertension (7, 14). To synthesize the evidence from prospective cohort studies, we conducted a systematic review and meta-analysis of prospective cohorts to assess the relation and risk of developing hypertension.

METHODS

Design

The Cochrane Handbook for Systematic Reviews of Interventions (15) and the Meta-analysis of Observational Studies in Epidemiology guidelines (16) were followed in conducting and reporting this meta-analysis. The protocol was registered (clinicaltrials.gov; NCT01608620).

Study selection

We searched MEDLINE (http://www.ncbi.nlm.nih.gov/pubmed/), Embase (http://www.elsevier.com/solutions/embase), Cumulative Index to Nursing and Allied Health Literature (https://www.ebscohost.com/nursing/products/cinahl-databases/cinahl-complete), and the Cochrane Registry (http://www.cochranelibrary.com/about/central-landing-page.html) through to 11 November 2014 with the use of the following inclusive search term: (fructose*.mp OR sucrose*.mp OR HFCS*.mp OR honey*.mp OR sugar*.mp) AND (hypertensive.mp OR hypertension.mp OR HTN.mp) (Supplemental Table 1). Manual searches of reference lists supplemented database searches. Only prospective cohorts that reported data on the association of SSBs and incident hypertension were included in our analysis.

Data extraction

Studies were initially excluded on the basis of titles and abstracts. Next, 2 independent reviewers (VHJ and VH) fully reviewed the remaining articles, producing a final list of prospective cohorts to meta-analyze. A kappa statistic (κ) was calculated to quantify between-reviewer agreement (0 = completely disagree, 1 = completely agree). To quantify between-reviewer agreement, a kappa (κ) statistic was calculated (0 = completely disagree, 1 = completely agree). A standardized pro forma was used in extracting all cohort characteristics. The RR of developing hypertension at each quantile of SSB intake was extracted alongside: authorship, background dietary pattern, cohort name, covariates included in the most-adjusted models, duration, person-years of follow-up, participant characteristics (age, sex, health status), publication year, sample size, and SSB exposure level (as percentage of energy). Sex-specific data were used wherever provided.

Study quality

The Newcastle-Ottawa Scale for Cohort Studies was used to assess cohort quality. A maximum of 9 points were awarded on the basis of the cohort selection (≤4 points), the comparability of the cohort design and analysis (≤2 points), and the adequacy of outcome measures (≥3 points) (17); ≥6 points was considered high quality. Disagreements in score allocations were resolved by consensus.

Statistical analyses

We pooled In-transformed RRIs for incident hypertension to compare the highest quantile of SSB exposure with the lowest quantile of SSB exposure (reference group) with the use of the generic inverse-variance method with random-effects modeling. Results were reported as risk ratios with 95% CIs. Between-cohort heterogeneity was tested with the use of Cochran’s Q (chi-square) statistic at α < 0.10 and quantified with the use of the I² statistic; an $I^2 \geq 50\%$ indicated evidence of considerable heterogeneity. A priori subgroups, which were used to investigate the interstudy heterogeneity, included disease status, sex (men vs. women), follow-up (<10 vs. ≥10 y), adjustments for critical covariates [age, race, family history, physical activity, smoking status, body weight and BMI (in kg/m²), and total calorie intake], and study quality (Newcastle-Ottawa scale: 6 < vs. ≥6). Meta-regressions with dummy variables that represented subgroup levels were used when testing for between-subgroup differences. Sensitivity analyses of systematically removing individual cohorts from the primary analysis and recalculating the pooled risk ratios were performed to clarify the contribution of individual cohorts to the overall association. The possibility of a dose-response relation was explored with the use of random-effects generalized least-squares trend estimation (GLST) models. We evaluated the possibility of a nonlinear relation with the use of a Wald test for the goodness-of-fit of the regression whereby a significant result indicated evidence of heterogeneity or unaccounted-for bias in the fitted linear GLST model, and a nonsignificant result indicated no departure from linearity (18). Five of 6 cohorts provided data for dose-response analyses; because most data were reported as servings of SSBs per month, all levels of exposure were converted to this common unit to mitigate errors resulting from unnecessary imputations. Publication bias was assessed by visually inspecting funnel plots and formally tested with Egger’s and Begg’s tests, where P < 0.10 was considered evidence of bias. Primary pooled analyses were conducted with the use of Review Manager software (RevMan, version 5.1.7; The Nordic Cochrane Centre, The Cochrane Collaboration). Assessments of publication bias, dose responses, and subgroup differences were performed with the use of STATA SE version 12.1 software (StataCorp LP).

RESULTS

Search results

Figure 1 displays the flow of literature from conception to study selection. Of 4016 articles identified through database and manual searches, 3990 studies were excluded on the basis of titles and abstracts. The remaining 26 studies were reviewed in full. Of those studies, 23 trials were further excluded for failing to meet our inclusion criteria. Three publications, which presented data from 6 prospective cohorts [CARDIA (Coronary Artery Risk Development...
in Young Adults Study) (19); Health Professionals Follow-Up Study (HPFS), Nurses’ Health Study I (NHS-I), Nurses’ Health Study II (NHS-II) (20), and the Seguimiento Universidad de Navarra Project [SUN (SUN-Male and SUN-Female)] (21) were meta-analyzed.

### Trial characteristics

**Table 1** summarizes individual cohort characteristics. Together, the 6 cohorts included 240,508 participants and 79,251 cases of hypertension with >3,197,528 person-years of follow-up. Person-year data were not presented for CARDIA. The CARDIA and SUN cohorts followed young (n = 2774; age: 18–30 y) and middle-aged (n = 13,843; age: 20–91 y) men and women, respectively, whereas the other cohorts followed older men (HPFS: n = 37,360; age: 42–63 y) or older (NHS-I: n = 88,540; age: 38–53 y) and middle-aged (NHS-II: n = 97,991; age: 31–40 y) women. Four cohorts were of US origin, and 2 cohorts were of Spanish origin, with a median follow-up of 18 y (range: 2–28 y). The incidence of hypertension (systolic blood pressure ≥130 mm Hg or diastolic blood pressure ≥85 mm Hg in CARDIA; systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg in HPFS, NHS-I, NHS-II, and SUN) was ascertained from self-reported physician diagnoses. SSB intake, in 8-oz servings (236-mL; CARDIA), 7-oz servings (200-mL; SUN), or 12-oz servings (355-mL; HPFS, NHS-I, and NHS-II) was assessed with the use of validated diet-history food-frequency questionnaires. Data were collected quadrennially in HPFS, NHS-I, and NHS-II; biennially in SUN; and at years 0, 7, and 20 in CARDIA. HPFS, NHS-I, and NHS-II reported HRs in quarts ranging from <1 SSB/mo to ≥1 SSB/d; CARDIA reported RRs in dichotomies whereby years 0 and 7 were averaged for the baseline and compared with year 20 at extreme intakes (none vs. frequent); and SUN reported RRs in tertiles ranging from nondrinkers to ≥1-serving/d. All cohorts adjusted for major risk factors of hypertension (age, race, family history, physical activity, smoking status, and body weight and BMI) and total caloric intake. In addition, CARDIA adjusted for milk intake, fruit juice intake, and CARDIA exam center; HPFS, NHS-I, and NHS-II adjusted for artificially sweetened beverages, the Dietary Approaches to Stop Hypertension–stylediet, and total fructose intake; and SUN adjusted for self-reported hypercholesterolemia, years of university education, energy-adjusted sodium, potassium, low-fat dairy, olive oil, fruit, vegetables, cereals, legumes, meat, whole-fat dairy, fish, and alcohol consumption (Supplemental Table 2). The study quality was high for all cohorts.

### Total SSB intake on incident hypertension

**Figure 2** presents the meta-analyzed association between SSB intake and incident hypertension. The risk of developing hypertension was 12% greater (risk ratio: 1.12; 95% CI: 1.06, 1.17; P < 0.001)
| Study (ref) | Subjects, n | Age range, y | Country | Follow-up, y | Frequency of data collection | Method of SSB measure | Method of outcome measure | Quantile divisions | Lowest quantile | Highest quantile | Hypertension cases | Person-years | Quality score | Funding source |
|-----------|-----------|-------------|--------|--------------|-----------------------------|----------------------|--------------------------|----------------------|----------------|----------------|----------------|-----------------|--------------|--------------|----------------|
| CARDIA (18) | 2774 M/F | 18–30 | United States | 20 (1986–2006) | Years 0, 7, and 20 | SSB intake as number of 8-oz serving | Self-reported physician diagnosed hypertension | Dichots | Nonconsumers | Consumers | | | 609 | | Agency |
| HPFS (19) | 37,360 M | 42–63 | United States | 22 (1986–2008) | Every 4 y | Number of servings of 12-oz SSBs consumed | Self-reported physician diagnosed hypertension | Quartiles | <1 SSB/mo | ≥1 SSB/d | 13,439 | 483,644 | 7 | Agency |
| NHS-I (19) | 88,540 F | 38–53 | United States | 28 (1980–2008) | Every 4 y | Number of servings of 12-oz SSBs consumed | Self-reported physician diagnosed hypertension | Quartiles | <1 SSB/mo | ≥1 SSB/d | 42,022 | 1,366,041 | 7 | Agency |
| NHS-II (19) | 97,991 F | 31–40 | United States | 16 (1991–2007) | Every 4 y | Number of servings of 12-oz SSBs consumed | Self-reported physician diagnosed hypertension | Quartiles | <1 SSB/mo | ≥1 SSB/d | 21,873 | 1,242,998 | 7 | Agency |
| SUN-Male (20) | 5099 M | 20–91 | Spain | 2–10 (1999–2010) | Every 2 y | Number of servings of 6.7-oz SSBs consumed | Self-reported physician diagnosed hypertension | Tertiles | Never | ≥1-serving/d | 798 | 38,117 | 6 | Agency |
| SUN-Female (20) | 8744 F | 20–91 | Spain | 2–10 (1999–2010) | Every 2 y | Number of servings of 6.7-oz SSBs consumed | Self-reported physician diagnosed hypertension | Tertiles | Never | ≥1-serving/d | 510 | 66,728 | 6 | Agency |

1CARDIA, Coronary Artery Risk Development in Young Adults; HPFS, Health Professionals Follow-Up Study; NHS-I, Nurses’ Health Study I; NHS-II, Nurses’ Health Study II; ref, reference; SSB, sugar-sweetened beverage; SUN, Seguimiento University of Navarra.
2Dietary information was collected with the use of validated semiquantitative food-frequency questionnaires.
3Quintiles were calculated as the number of beverages consumed.
4Calculated by adding the number of cases through all quintiles.
5Calculated by adding the number of person-years through all quintiles.
6Quality score was assessed with the use of the NewCastle-Ottawa Assessment Scale for Cohort Studies whereby a maximum of 10 points could be awarded.
7From a government, university, or not-for-profit health agency.
in individuals in the highest compared with the lowest quantiles of SSB intake. We observed significant between-cohort heterogeneity ($I^2 = 62\%$, $P = 0.02$).

**A priori subgroup and sensitivity analyses**

Only 2 (duration and sex) of 5 a priori analyses were performed because the cohorts did not vary according to the remaining 3 analyses (disease status, study quality, and adjustments for critical covariates). Subgroup meta-regressions for neither sex (men, women, and both) nor duration (as a continuous variable: 2–28 y) explained the heterogeneity in our results (men vs. women: $P = 0.37$; women vs. both: $P = 0.27$; men vs. both: $P = 0.88$; duration: $P = 0.25$). The systematic removal of individual cohorts did not modify the overall significance nor the direction of the overall association although the heterogeneity in our results was reduced ($I^2 < 50\%$) with the removal of the NHS-II.

**Dose-response relations**

HPFS, NHS-I, NHS-II, SUN-Male, and SUN-Female were analyzed for a dose-response; CARDIA was excluded because information regarding person-years or quantitative intakes were unavailable. We observed a significant positive random-effects GLST dose-response between SSB intake and incident hypertension ($\beta = 0.0027$ (95% CI: 0.0021, 0.0033); $P < 0.001$); the risk of hypertension increased 0.27% for every additional SSB per month in pooling of 5 of 6 cohorts.

**Publication bias**

Visual inspection of funnel plots and formal testing with Egger’s and Begg’s tests did not reveal significant evidence of publication bias (Egger’s $P = 0.75$, Begg’s $P = 0.57$) (Supplemental Figure 2).

**DISCUSSION**

This systematic review and meta-analysis of 6 prospective cohorts of 240,508 individuals, with >3,197,528 person-years of follow-up and 79,251 hypertension cases showed a modest 12% greater risk of developing hypertension when comparing the highest intake ($\geq 1$ SSB/d) with the lowest intake (none). There was significant heterogeneity observed between cohorts, which was not explained by differences in sex or the duration of follow-up. We observed a significant positive linear dose response ($\beta = 0.0027$) per additional serving of SSB per month in pooling of 5 of 6 cohorts.

**Findings in the context of the literature**

Our results are consistent with evidence from prospective cohorts linking SSB intake with the possible downstream effects of hypertension, namely coronary heart disease and stroke (22, 23), although the evidence remains less robust for these relations. SSB intake has been positively associated with coronary heart disease risk in women only (24) and stroke incidence in women, but not men, in the NHS, HPFS (25), and Japan Public Health Centre-based Study Cohort I (26).

Several mechanisms may explain the current results. One proposed mechanism is that liquid calories from SSBs elicit a weaker satiety response than do solid calories, potentially leading to decreases in the compensation for liquid calories and increases in subsequent energy intake, weight gain, and cardiometabolic morbidity (23). Although a systematic review of 121 liquid preload interventions showed this relation existed acutely (27), longer-term trials that compared liquid with solid calories on food intake have not shown a significant increase in long-term caloric over-consumption or weight gain (28, 29). More long-term trials are needed to confirm these findings.

Another potential mechanism is that SSBs may represent a marker of an unhealthy lifestyle that predisposes individuals to hypertension (30, 31). People who consume more SSBs tend to consume more calories, exercise less, smoke more, and drink more alcohol (20). They are also more likely to consume a Western dietary pattern, which includes higher intakes of refined grains, French fries, processed meat, red meat, sweets, and desserts (30, 32, 33), which are high in sodium, glycemic index, and also independently associated with obesity.

A final mechanism implicates the fructose moiety within SSBs as the culprit (7–9). This link between fructose and hypertension is not well supported. High-level evidence from systematic reviews and meta-analyses of controlled feeding trials have shown no...
difference in the effects of liquid, solid, or mixed-form fructose on systolic, diastolic, or mean arterial blood pressures (11) in calorie-matched comparisons with other carbohydrates that are likely to replace fructose. In fact, pooled analyses indicated a modest benefit of fructose for blood pressure. In addition, no effect was shown for calorie-matched fructose intakes on serum uric acid (12), the primary intermediate through which fructose is thought to induce hypertension. A systematic review and meta-analysis of prospective cohorts also failed to show an association between total fructose intake from all sources and incident hypertension (13). Although the NHS-I and NHS-II cohorts included in the current meta-analysis adjusted for fructose intake, an earlier report by Winkelmayer et al. (34) studied hypertension risk with SSB intakes in these same cohorts without adjusting for fructose and reported similar RRs at comparable exposures [12% vs. 11% for intakes in these same cohorts without adjusting for fructose and by Winkelmayer et al. (34) studied hypertension risk with SSB total fructose intake from all sources and incident hypertension prospective cohorts also failed to show an association between induce hypertension. A systematic review and meta-analysis of (12), the primary intermediate through which fructose is thought to shown for calorie-matched fructose intakes on serum uric acid benefit of fructose for blood pressure. In addition, no effect was matched comparisons with other carbohydrates that are likely to difference in the effects of liquid, solid, or mixed-form fructose on serum uric acid (11) in calorie-matched comparisons with other carbohydrates that are likely to replace fructose. In fact, pooled analyses indicated a modest benefit of fructose for blood pressure. In addition, no effect was shown for calorie-matched fructose intakes on serum uric acid (12), the primary intermediate through which fructose is thought to induce hypertension. A systematic review and meta-analysis of prospective cohorts also failed to show an association between total fructose intake from all sources and incident hypertension (13). Although the NHS-I and NHS-II cohorts included in the current meta-analysis adjusted for fructose intake, an earlier report by Winkelmayer et al. (34) studied hypertension risk with SSB intakes in these same cohorts without adjusting for fructose and reported similar RRs at comparable exposures [12% vs. 11% for the NHS-I and 17% vs. 18% for the NHS-II between ≥1 SSB/d in Cohen et al. (20) and weighted pooling of 1, 2–3, and ≥4 SSBs/d in Winkelmayer et al. (34), suggesting minimal impact of adjusting for fructose. Moreover, a significant trend of hypertension risk reduction was reported in the NHS-II when sources of fructose excluded SSBs, and a significant harmful trend in the NHS-I and NHS-II when sources of fructose intake included only SSBs (20). Because fructose does not differ biochemically between the 2 sources, these findings implicate a mechanism independent of the amount of ingested fructose.

Subgroup analyses and dose responses

Although we observed significant between-cohort heterogeneity, we were unable to explain it with our a priori subgroup analyses. Because of the limited number of cohorts and the methodologic homogeneity between them, only 2 subgroup analyses were possible; post hoc meta-regressions were impractical. We observed a significant linear dose-response between SSB intake and incident hypertension in the pooling of all but the CARDIA cohort. Our data suggest a 0.27% increase in risk per additional SSB per month or an 8.2% risk increase per additional SSB per day. With consideration of the large weight that the CARDIA cohort accounted for in our primary analysis, we acknowledged that its inclusion may have modified this observed dose-response relation. These data support previously suggested benefits of consuming fewer SSBs (35). However, it is unclear whether similar benefits can be observed simply through an improved dietary pattern (30), a reduction of caloric intake irrespective of source (36, 37), or a reduction of overall sugar intake (38, 39).

Limitations

Several limitations of our meta-analysis must be acknowledged. First, the examination of diseases with multifactorial causes in relation to single nutrients in prospective designs may have limited methodologic merit because of confounding from background dietary patterns (40). Second, 94% of all individuals were of US nationality, 93% were health professionals, and 90% were Caucasian, limiting the generalizability of these results to a broader demographic (41). Third, the relatively small number of available cohorts limited our ability to sufficiently explore relations between subgroups. Fourth, 2 of 5 cohorts did not adjust for sodium intake, and the remaining 3 cohorts adjusted for sodium intake with the use of only self-reported data from food-frequency questionnaires as opposed to the gold standard of 24-h urinary sodium (20). Because sodium correlates with sugar and SSBs intake (39), the possibility of confounding by sodium intake could not be eliminated. Last, because all cohorts in the current meta-analysis pooled individuals who consumed ≥1 SSB/d, it is possible that our results underestimated risk in individuals with very high SSB intakes.

Implications

The risk increase reported in this meta-analysis must be considered in relation to other risk factors for hypertension. Multivariate-adjusted extreme quantile risk ratios for developing hypertension within cohorts included in this meta-analysis were 1.61 (95% CI: 1.42, 1.82) for alcohol consumption (42), 1.21 (95% CI: 1.06, 1.39) for smoking (43), 1.35 (95% CI: 1.14, 1.59) for red meat intake (44), 1.48 (95% CI: 1.01, 2.18) for a sedentary lifestyle (45), and 4.70 (95% CI: 4.45, 4.96) for BMI ≥30 (42). In this context, SSBs minimally aggravate risk of developing hypertension. These risk factors may also contribute to important collinearity effects in their tendency to cluster with SSBs as part of a Western dietary and lifestyle pattern. For example, a Western dietary pattern has been associated with elevated cardiometabolic risk even after adjustment for SSBs (31, 46).

In conclusion, this systematic review and meta-analysis of 6 prospective cohorts showed a significant 12% increase in risk of developing hypertension with ≥1 SSB/d in both men and women with no history of hypertension. We showed a significant dose-response relation suggesting an 8.2% increase in risk per SSB per day across an SSB intake range from none to ≥1 SSB/d. This association is small in the context of other established risk factors for hypertension. There is a need for more-specific analyses within existing cohorts to further elucidate the relation between different sugar sources and incident hypertension within different demographies, particularly after adjusting for electrolyte intakes. High-quality, long-term, randomized controlled trials that assess the effects of reducing calories from SSBs vs. from other sources of solid calories on the development of hypertension and its complications are also warranted.

The authors’ responsibilities were as follows—VHJ, RJdS, VHI, AM, SB-M, ALJ, LA, TMSW, JB, CWCK, DJAI, and JLS: designed the research; VHJ, VHI, and AM: conducted the research; VHJ, RJdS, and AM: analyzed the data or performed the statistical analysis; VHJ and JLS: wrote the manuscript; JLS: had primary responsibility for the final content of the manuscript; RJD and JB: provided statistical expertise; and all authors: read and approved the final manuscript. RJdS has received research support from the Calorie Control Council and the Coca-Cola Company (investigator initiated, unrestricted grant). VHJ has received a travel award to attend the “Journey Through Science Day” hosted by PepsiCo and the New York Academy of Sciences. ALJ is a part owner, vice president, and director of research of Glycemic Index Laboratories, Toronto, Canada. TMSW is a part owner and the president of Glycemic Index Laboratories, Toronto, Canada, and has authored several popular diet books on the glycemic index for which he has received royalties from Phillipa Sandall Publishing Services and CABI Publishers. TMSW has received consultant fees, honoraria, travel funding, or research support from or served on the scientific advisory board for McCain Foods, the Glycemic Index Symbol program, and CreaNutrition AG. JB has received research support from the Calorie Control Council and The Coca-Cola Company (investigator initiated, unrestricted grant). CWCK has received research support from the Calorie Control Council, the Coca-Cola Company (investigator initiated, unrestricted grant). Hain Celestial, Kellogg, Kraft, Loblaw Companies Ltd., Solae, and Unilever. CWCK has received travel funding, consultant fees, or honoraria from the Coca-Cola Company, Danone, Abbott Laboratories, General Mills, Kellogg, Loblaw
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