Sweetened beverage consumption and risk of coronary heart disease in women[1–4]

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ABSTRACT

Background: Previous studies have linked full-calorie sugar-sweetened beverages (SSBs) with greater weight gain and an increased risk of type 2 diabetes.

Objective: We prospectively examined the association between consumption of SSBs and the risk of coronary heart disease (CHD) in women.

Design: Women ($n = 88{,}520$) from the Nurses' Health Study aged 34–59 y, without previously diagnosed coronary heart disease (CHD), stroke, or diabetes in 1980, were followed from 1980 to 2004. Consumption of SSBs was derived from 7 repeated food-frequency questionnaires administered between 1980 and 2002. Relative risks (RRs) for CHD were calculated by using Cox proportional hazards models and adjusted for known cardiovascular disease risk factors.

Results: During 24 y of follow-up, we ascertained 3105 incident cases of CHD (nonfatal myocardial infarction and fatal CHD). After standard and dietary risk factors were adjusted for, the RRs (and 95% CIs) of CHD according to categories of cumulative average of SSB consumption (<1/mo, 1–4/mo, 2–6/wk, 1/d, and ≥2 servings/d) were 1.0, 0.96 (0.87, 1.06), 1.04 (0.95, 1.14), 1.23 (1.06, 1.43), and 1.35 (1.07, 1.69) (P for trend < 0.001). Additional adjustment for body mass index, energy intake, and incident diabetes attenuated the associations, but they remained significant. Artificially sweetened beverages were not associated with CHD.

Conclusion: Regular consumption of SSBs is associated with a higher risk of CHD in women, even after other unhealthful lifestyle or dietary factors are accounted for. *Am J Clin Nutr* 2009;89:1037–42.

INTRODUCTION

Sugar-sweetened beverages (SSBs), or soft drinks, include carbonated and noncarbonated beverages that contain sugar-based caloric sweeteners and are flavored with fruit juice or natural or artificial flavors. These beverages currently contribute 9.2% of total energy intake in the United States—an increase from 3.9% in the late 1970s (1). In fact, on average, SSBs are the top energy contributor in the US diet (2). Previous epidemiologic studies have found a positive association between SSBs and weight gain and obesity in both children and adults (3). In addition, a higher consumption of SSBs has been linked to an increased risk of developing type 2 diabetes (4, 5). Because obesity and type 2 diabetes are important risk factors for coronary heart disease (CHD), we hypothesized that regular consumption of SSBs is associated with an increased risk of CHD. SSBs can also influence the risk of CHD, independent of obesity, as a potential contributor to a high glycemic load, which has been linked to higher concentrations of the inflammatory marker C-reactive protein and an increased risk of diabetes and CHD (6). Short-term trials that changed the dietary fiber or sucrose content or body weight have been shown to change the concentrations of inflammatory markers (7, 8). Inflammation is not only involved in atherosclerosis, but also affects plaque stability and thrombosis (9), which may respond to lifestyle changes more quickly than atherosclerosis. Therefore, both recent and cumulative measures of SSB might affect CHD risk.

To examine whether SSB consumption is associated with CHD, and whether the relation is independent of obesity and diabetes, we prospectively assessed the intake of sweetened beverages and CHD in middle-aged women with detailed measures of lifestyle and dietary factors. Because soft drinks were the major SSB consumed in this cohort, we particularly emphasized this type of beverage.

SUBJECTS AND METHODS

Study population

The Nurses' Health Study (NHS) cohort began in 1976 when 121,700 female nurses aged 30–55 y living in 11 US states responded to a questionnaire regarding medical, lifestyle, and other health-related information (10). Questionnaires have been sent biennially to update this information. Follow-up was complete for >95% of the potential person-time up to 2004. In 1980,

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the participants completed a 61-item food-frequency questionnaire (FFQ). In 1984, the FFQ was expanded to 116 items. Similar FFQs were sent in 1986, 1990, 1994, 1998, and 2002.

For this analysis, we included women who completed the 1980 FFQ with <70 missing items and total energy intake (as calculated from the FFQ) between 500 and 3500 kcal/d. At baseline, we excluded those with a history of CHD, stroke, or diabetes because the diagnosis of these conditions may lead to changes in diet. After these exclusions, 88,520 women followed up from 1980 to 2004 were included in the analyses. This study was approved by the Institutional Review Board of Brigham and Women's Hospital, Boston, MA.

Dietary assessment

FFQs were designed to assess average food intakes over the preceding year. A standard portion size and 9 possible responses for frequency of consumption, ranging from "never, or less than once per month" to "6 or more times per day," were given for each food item. Total energy and nutrient intakes were calculated by summing energy or nutrient intakes from all foods. Previous validation studies among members of the NHS revealed good correlations between nutrients assessed by the FFQ and multiple weeks of food records completed over the preceding year (11). For example, correlation coefficients between the 1986 FFQs and diet records obtained in 1986 were 0.68 for saturated fat, 0.48 for polyunsaturated fat, and 0.78 for crude fiber. For sweetened beverages, correlations between diet records and FFQs were 0.84 for colas, 0.36 for noncola carbonated soft drinks, and 0.56 for noncarbonated sweetened beverages (12).

The SSBs included in our analysis were caffeinated and noncaffeinated colas [eg, Coke (Coca-Cola, Atlanta, GA), Pepsi (Persico Inc, Purchase, NY), and other colas with sugar], other carbonated beverages with sugar [eg, 7-Up (Dr Pepper Snapple Group, Plano, TX)], noncarbonated sweetened beverages [ie, Hawaiian Punch (Dr Pepper Snapple Group), lemonade, and other noncarbonated fruit drinks]. We summed the consumption of these beverages as total sweetened beverages. Artificially sweetened beverages consisted of all types of low-calorie sweet carbonated beverages, such as diet colas and other diet carbonated beverages.

Endpoint ascertainment

For this analysis, we ascertained incident cases of CHD [nonfatal myocardial infarction (MI) or fatal CHD] that occurred after the return of the 1980 questionnaire in women but before 1 June 2004. We requested permission to review medical records from women who reported having an MI on each biennial questionnaire. Physicians unaware of the self-reported risk factor status systematically reviewed the records. MI was classified as confirmed if the criteria of the World Health Organization were met, specifically, symptoms and either electrocardiograph-detected changes or elevated cardiac enzyme concentrations (13). We included confirmed and probable cases for the analyses. Fatal CHD was confirmed by hospital records or through an autopsy or if CHD was listed as the cause of death on the death certificate, if it was listed as an underlying and most plausible cause of death, and if evidence of previous CHD was available. Deaths were identified from state vital statistics records and the National Death Index or were reported by the families and the postal system. National Death Index search results performed in December 2006 were used, which ensured complete follow-up of fatal CHD up to 2004.

Assessment of covariates

Body mass index (BMI) was calculated from weight reported on each biennial questionnaire and height reported in 1976. In each biennial questionnaire, we also assessed smoking status (including number of cigarettes), frequency and number of aspirin tablets used, multivitamin intake, menopausal status, and use of postmenopausal hormones. Recreational physical activity was measured biennially beginning in 1986 with a validated questionnaire asking about the average time spent on 10 common activities. The information was then summed and calculated as metabolic equivalent hours per week (14). In 1980 and 1982, we measured hours per day of moderate-to-vigorous physical activity.

Statistical analysis

We used Cox proportional hazard modeling to assess the association between sweetened (sugar or artificial) beverage consumption and risk of CHD. SSB intake was classified by frequency: <1/mo (reference), 1–4/mo, 2–6/wk, 1 to <2/d, and ≥2/d. We used cumulative averages of SSB intake from our repeated FFQs to predict CHD risk (15). With this method, sweetened beverage intake in 1980 was used to predict CHD occurrence from 1980 to 1984, and the average intake from 1980 and 1984 was used to predict CHD risk from 1984 to 1986, and so forth. We stopped updating sweetened beverage consumption and other dietary information when a cohort member reported a diagnosis of diabetes, angina, or coronary bypass surgery during follow-up. To separate out changes in SSB intake as a consequence of these 3 intermediate endpoints, we also performed an analysis in which we censored women on report of diabetes, angina, or coronary bypass surgery.

We also examined the consistency of the association between SSB intake and CHD using the most recent intakes of SSBs and artificially sweetened beverages as predictors of CHD risk. With this analysis, intake in 1980 was used to predict CHD between 1980 and 1984, and intake in 1984 was used to predict CHD between 1984 and 1986, and so forth.

For the multivariate analysis, we adjusted for the following potential confounders, which were updated at each 2-y cycle: age (continuous), smoking [never, past, or current (1–14/d, 15–24/d, ≥25/d, or missing)], alcohol intake (0, 1 to <5, 5–15, or >15 g/d), parental history of myocardial infarction before age 60 y (yes or no), physical activity (quintiles), aspirin use (<1, 1–2, 3–6, 7–14, or ≥15/wk), menopausal status and postmenopausal hormone use (premenopausal, never, past, or current hormone use), and history of hypertension and high blood cholesterol. In secondary analyses, we also stratified by major cardiac disease risk factors to evaluate whether associations between SSB consumption and CHD differed by these factors.

We further adjusted for the Alternate Healthy Eating Index (AHEI; quintiles) because sweetened beverage consumption may be a marker of a generally unhealthy diet. The AHEI measures healthfulness of a diet by awarding a maximum of 10 points to the following categories: high intake of fruit, vegetables, cereal fiber; high polyunsaturated-to-saturated fat ratio; moderate alcohol

intake; multivitamin use (maximum of 7.5 points), white-to-red meat ratio; legumes and soy intake; and low *trans* fat intake (16). The AHEI has a maximum of 87.5 points. We also assessed whether any observed association with sweetened beverages was independent of factors that are in the potential causal pathway between SSB intake and CHD by further adjusting for diabetes, BMI (5 categories), and energy intake (quintiles) in separate models. Analysis was conducted with SAS version 9.1 (SAS Institute Inc, Cary, NC).

RESULTS

During 24 y of follow-up, we ascertained 3105 incident cases of CHD. Mean SSB consumption during follow-up decreased from 0.41 servings/d at baseline to 0.24 servings/d in 2002. Women with a higher consumption of SSBs were more likely than those with a lower intake to be current smokers, to have lower levels of physical activity, and to have a higher BMI (**Table 1**). They also tended to consume more energy, sugar, and total fat, but less alcohol, fruit, and vegetables.

After nondietary risk factors for CHD were adjusted for, we observed a significant positive association between SSB intake and CHD risk. Women who consumed ≥2 servings of SSBs per day had an RR of 1.39 (95% CI: 1.11, 1.75; *P* for trend < 0.001) compared with those who consumed SSBs less than once a month (**Table 2**). Because individuals with a high consumption of these beverages tended to consume an unhealthful diet, we also adjusted for the AHEI score. The RR of ≥2 servings/d compared with <1 serving/mo was only slightly attenuated (RR: 1.35; 95% CI: 1.07, 1.69; *P* for trend < 0.001). The association was moderately attenuated (RR: 1.21; 95% CI: 0.95, 1.53; *P* for trend = 0.005 for extreme categories) after further adjustment for diabetes, BMI, and total energy intake.

Among specific SSBs, we observed a positive association with cola-type beverages. After dietary and nondietary risk factors were adjusted for, the RR for every 2 servings/d increase in consumption was 1.35 (95% CI: 1.15, 1.57; *P* for trend < 0.001; **Table 3**). Results were slightly attenuated, but remained significant after adjustment for BMI and total energy intake (RR: 1.25; 95% CI: 1.07, 1.47; *P* for trend = 0.005). Similar associations were observed for fruit drinks and punches, which are noncarbonated. A similar magnitude of risk was associated with sugar-sweetened carbonated noncola type beverages, but it did not reach statistical significance. Results were similar after exclusion of women who reported a diagnosis of diabetes, angina, or coronary bypass surgery during follow-up. Although we observed a weak but marginally significant association with artificially sweetened soft drinks after adjustment for dietary and nondietary risk factors (RR: 1.15; 95% CI: 0.97, 1.38; *P* = 0.07; **Table 4**), the association was greatly attenuated and no longer significant after further adjustment for diabetes, BMI, and energy intake (RR comparing extreme categories: 1.00; 95% CI: 0.84, 1.19; *P* for trend = 0.87).

We also assessed the risk of CHD using recent intake of SSBs, which reflects short-term changes in intake. We observed a slightly weaker but statistically significant association. After standard risk factors, AHEI, BMI, and energy intake were

TABLE 1
Age-standardized baseline (1980) lifestyle, health, and 1990 dietary characteristics of the women by sugar-sweetened beverage consumption[1]

	Consumption level				
	<1/mo (*n* = 34,010)	1–4/mo (*n* = 19,874)	2–6/wk (*n* = 21,661)	1 to <2/d (*n* = 8325)	≥2/d (*n* = 4650)
BMI (kg/m^2)	24.1 ± 0.01	24.1 ± 0.01	24.3 ± 0.01	24.5 ± 0.01	25.3 ± 0.02
Smokers (%)	29 ± 0.07	26 ± 0.09	26 ± 0.09	30 ± 0.10	36 ± 0.20
Physical activity (h/wk)[2]	4.1 ± 0.01	3.9 ± 0.01	3.9 ± 0.01	3.8 ± 0.01	3.5 ± 0.01
Family history (%)[3]	30 ± 0.07	29 ± 0.10	28 ± 0.09	29 ± 0.15	29 ± 0.20
Hypercholesterolemia (%)	5 ± 0.03	5 ± 0.04	5 ± 0.04	4 ± 0.07	5 ± 0.09
Hypertension (%)	15 ± 0.06	14 ± 0.07	15 ± 0.07	16 ± 0.11	18 ± 0.15
Dietary intake (1990)					
Alcohol intake (g)	7 ± 0.02	5 ± 0.02	5 ± 0.02	4 ± 0.05	4 ± 0.09
Energy (kcal)	1602 ± 1	1709 ± 1	1831 ± 1	1994 ± 2	2092 ± 5
Glycemic load	101 ± 0.1	111 ± 0.1	124 ± 0.1	147 ± 0.2	167 ± 0.4
Carbohydrates (% of energy)	49 ± 0.02	49 ± 0.02	51 ± 0.01	53 ± 0.04	56 ± 0.07
Total fat (% of energy)	31 ± 0.01	32 ± 0.01	32 ± 0.01	31 ± 0.03	30 ± 0.05
Fiber (g)	19 ± 0.02	20 ± 0.02	20 ± 0.01	19 ± 0.04	17 ± 0.07
Total sugar (g)	89 ± 0.1	98 ± 0.1	114 ± 0.1	145 ± 0.2	179 ± 0.4
Fruit and vegetables (servings)	5.8 ± 0.01	5.8 ± 0.01	5.8 ± 0.01	5.6 ± 0.01	5.2 ± 0.03
Fructose (g)	20 ± 0.03	21 ± 0.03	26 ± 0.02	37 ± 0.06	51 ± 0.11
Sucrose (g)	35 ± 0.04	39 ± 0.04	46 ± 0.04	58 ± 0.10	67 ± 0.18
AHEI score	44 ± 0.02	43 ± 0.02	41 ± 0.02	38 ± 0.05	35 ± 0.10

[1] All values are means ± SEMs and were age-standardized as computed with a generalized linear model. Beverage consumption includes sweetened carbonated and noncarbonated beverages. *n* values reflect the number of women in 1980. AHEI, Alternate Healthy Eating Index. *P* values were <0.05 for trends across increasing categories of sugar-sweetened beverage consumption, except for fiber intake.
[2] Moderate-to-vigorous physical activity.
[3] Parental history of myocardial infarction before age 60 y.

TABLE 2
Relative risks and 95% CIs for sugar-sweetened beverage consumption and risk of coronary heart disease by consumption level (n = 88,520)

	Consumption level					
	<1/mo	1–4/mo	2–6/wk	1 to <2/d	≥2/d	P for trend
Median intake (servings/d)	0	0.1	0.4	1.2	2.6	
No. of cases	883	723	1198	218	83	
Person-years	574,814	494,831	745,176	134,933	52,455	
Age-adjusted	1	0.94 (0.85, 1.03)	1.08 (0.99, 1.18)	1.51 (1.30, 1.75)	1.93 (1.54, 2.43)	<0.001
Multivariate-adjusted[1]	1	0.97 (0.88, 1.07)	1.06 (0.97, 1.16)	1.27 (1.09, 1.47)	1.39 (1.11, 1.75)	<0.001
Multivariate-adjusted + diet[2]	1	0.96 (0.87, 1.06)	1.04 (0.95, 1.14)	1.23 (1.06, 1.43)	1.35 (1.07, 1.69)	<0.001

[1] Adjusted for age (continuous), smoking [never, past, or current cigarette use (1–14/d, 15–24/d, ≥25/d, or missing)], alcohol intake (0, <5, 5–15, or >15 g/d), family history (yes or no), physical activity (quintiles), aspirin use (<1, 1–2, 3–6, 7–14, or ≥15/wk), menopausal status and postmenopausal hormone use (premenopausal, never, past, or current hormone use), and history of hypertension and high blood cholesterol. Relative risks were computed from a Cox proportional hazard model.

[2] Additionally adjusted for the Alternate Healthy Eating Index (quintiles).

adjusted for, the RR of total SSBs, comparing ≥2 servings/d and <1 serving/mo, was 1.38 (95% CI: 1.15, 1.65; P for trend < 0.001).

DISCUSSION

In this large prospective cohort study of women, we observed a significant positive association between regular consumption of SSBs and risk of CHD. This association remained significant even after adjustment for a multitude of dietary and lifestyle factors. Additional adjustment for the BMI and energy intake score somewhat attenuated this association, which suggested that excess calorie intakes and obesity mediate the association. In addition, we observed a small and nonsignificant association between consumption of artificially sweetened beverages and risk of CHD after multivariate adjustment.

TABLE 3
Multivariate relative risks (RRs) for a 2-serving increase in specific sugar-sweetened beverage consumption and risk of coronary heart disease (n = 88,520)

Beverage type	RR (95% CI)	P value
Total sugar-sweetened beverages		
Multivariate-adjusted[1]	1.32 (1.17, 1.48)	<0.001
Multivariate-adjusted + diet[2]	1.28 (1.14, 1.44)	<0.001
Colas		
Multivariate-adjusted[1]	1.40 (1.21, 1.63)	<0.001
Multivariate-adjusted + diet[2]	1.35 (1.15, 1.57)	<0.001
Carbonated noncola		
Multivariate-adjusted[1]	1.33 (0.91, 1.94)	0.24
Multivariate-adjusted + diet[2]	1.27 (0.87, 1.86)	0.22
Fruit drinks and punch[3]		
Multivariate-adjusted[1]	1.32 (1.02, 1.70)	0.04
Multivariate-adjusted + diet[2]	1.33 (1.03, 1.71)	0.03

[1] Adjusted for age (continuous), smoking [never, past, or current cigarette use (1–14/d, 15–24/d, ≥25/d, or missing)], alcohol intake (0, <5, 5–15, or >15 g/d), family history (yes or no), physical activity (quintiles), aspirin use (<1, 1–2, 3–6, 7–14, or ≥15/wk), menopausal status and postmenopausal hormone use (premenopausal, never, past, or current hormone use), and history of hypertension and high blood cholesterol. RRs were computed from a Cox proportional hazard model.

[2] Additionally adjusted for the Alternate Healthy Eating Index (quintiles).

[3] Includes fruit punch, lemonades, and other noncarbonated sweetened beverages.

The positive association that we observed between SSB intake and incidence of CHD is consistent with recent data indicating a relation between soft drink intake and occurrence of the metabolic syndrome. In the Framingham Heart Study, after a mean follow-up of 4 y, the odds ratio for incident metabolic syndrome was 1.44 for those who drank more than one soft drink per day compared with those who drank less than one per day (17). In the Atherosclerosis Risk in Communities Study, the hazard ratio for developing the metabolic syndrome, in a comparison of the top and bottom tertiles of sweetened soft drinks, was marginally significant at 1.09 (P = 0.07) (18). In a cross-sectional study among young adults, those with the metabolic syndrome tended to have higher sweetened beverage intake (19). To our knowledge, no prospective study has addressed the relation of SSB intake to incidence of clinical CHD events.

Serum glucose and insulin concentrations can rise quickly after SSB consumption (20). Therefore, SSB consumption can substantially contribute to the glycemic load of the overall diet. An increase in glycemic load can increase C-reactive protein concentrations (6). Inflammation influences not only atherosclerosis but also plaque stability and thrombosis. Therefore, SSB consumption can affect CHD risk in a relatively short time of a few years. In our cohort, a high glycemic load was shown to be associated with a higher risk of CHD (21). In this analysis, we observed similar results using recent and cumulative SSB intake to predict CHD risk, with perhaps more of an affect of recent intake.

In this analysis, the contribution of BMI did not fully explain the association between SSB intake and CHD, which suggests that other mechanisms are involved. Fructose has been the major sweetener in SSB since the mid-1980s, and it increases triacylglycerol synthesis in the liver (22, 23), which results in elevated triacylglycerol concentrations, which have been associated with a greater risk of CHD (23, 24). Fructose is also the only sugar that can increase blood uric acid concentrations (26–27). High uric acid concentrations may reduce endothelial nitric oxide (28), which could partly mediate a relation between soft drink consumption and risk of CHD.

Prospective data from the Atherosclerosis Risk in Communities Study (18) and the Framingham Heart Study (17) showed moderate positive associations between artificially sweetened soda intake and incident metabolic syndrome. However, this asso-

TABLE 4
Relative risks and 95% CIs of coronary heart disease by consumption level of artificially sweetened beverages (n = 88,520)[1]

	Consumption level					
	<1/mo	1–4/mo	2–6/wk	1 to <2/d	≥2/d	*P* for trend
Median intake (servings/d)	0	0.1	0.4	1.2	2.5	
No. of cases	1218	424	1040	278	145	
Person-years	730,939	295,253	671,986	202,060	101,970	
Age-adjusted	1	0.82 (0.74, 0.92)	0.88 (0.81, 0.96)	1.01 (0.88, 1.15)	1.28 (1.08, 1.53)	0.002
Multivariate-adjusted[2]	1	0.91 (0.81, 1.02)	0.96 (0.88, 1.05)	1.01 (0.88, 1.15)	1.14 (0.96, 1.36)	0.10
Multivariate-adjusted + diet[3]	1	0.92 (0.82, 1.03)	0.98 (0.90, 1.07)	1.03 (0.90, 1.17))	1.15 (0.97, 1.38)	0.07

[1] Beverage consumption includes low-calorie caffeine- and non-caffeine-containing carbonated beverages. Relative risks were computed from a Cox proportional hazard model.

[2] Adjusted for age (continuous), smoking [never, past, or current cigarette use (1–14/d, 15–24/d, ≥25/d, or missing), alcohol intake (0, <5, 5–15, or >15 g/d), family history (yes or no), physical activity (quintiles), aspirin use (<1, 1–2, 3–6, 7–14, or ≥15/wk), menopausal status and postmenopausal hormone use (premenopausal, never, past, or current hormone use), and history of hypertension and high blood cholesterol.

[3] Additionally adjusted for the Alternate Healthy Eating Index (quintiles).

ciation may be induced by reverse causation, because higher diet beverages intake may be a marker of attempts for those who are prone to weight gain to control body weight. Furthermore, the development of other components of the metabolic syndrome may have prompted individuals to make dietary changes, including switching to artificially sweetened soft drinks before developing the full-blown metabolic syndrome. On the other hand, although diet beverages do not contain calories, they may induce increased energy intakes from other sources. In our study, diet beverages were associated with a small but nonsignificant increased diabetes risk. Adjustment for total energy intake did not materially alter the results. Thus, our findings suggest that intake of diet beverages is unlikely to be associated with CHD through the modification of overall energy intake. Nonetheless, one should be cautious in interpreting the findings concerning diet beverage intake and risk of metabolic diseases and CHD.

Although we have detailed control for confounding, residual confounding remains a possibility because diet and lifestyle information are collected with some degree of error, and SSB intake may be correlated with unmeasured risk factors.

In our cohort and in others, SSB intake was associated with intake of foods higher in saturated and *trans* fats (18, 29), which could potentially confound an association between SSB intake and CHD. Thus, we adjusted not only for standard risk factors that were updated over time but also women's AHEI score, which reflects the overall healthfulness of the diet. This score takes into account the intake of 10 dietary components and has been shown to robustly predict risk of CHD (16, 30).

Our results did not change when we excluded women who developed diabetes or angina during follow-up. Because diabetes is a major risk factor for CHD and SSB consumption has been associated with increased diabetes risk, it can be considered as an intermediate risk factor in the pathway between SSB intake and CHD risk. In addition, people may change their diet after a diagnosis of diabetes. In addition to excluding diabetic patients at baseline, we conducted several sensitivity analyses to address the potential bias caused by a diagnosis of diabetes during follow-up. In our main analysis, we stopped updating diet after participants reported a diagnosis of diabetes. In secondary analysis, we censored women with a diagnosis of diabetes during follow-up. Consistently, we observed significant positive associations between SSB intake and CHD despite diverse approaches to handling SSB intake and diabetes.

In conclusion, we found that consumption of SSB is associated with a higher risk of CHD in women, even after other risk factors for CHD or an unhealthful diet or lifestyle are accounted for. This finding provides further rationale for limiting the consumption of SSBs.

The authors' responsibilities were as follows—TTF: had full access to all of the data in the study, takes responsibility for the integrity of the data and the accuracy of the data analysis, and conducted the statistical analysis; TTF, FBH, and KMR: responsible for the study concept and design; FBH, KMR, and VM: responsible for the acquisition of data; TTF, FBH, KMR, WCW, VM, and JEM: responsible for the analysis and interpretation of data and critical revision of the manuscript for important intellectual content; TTF, KMR, and FBH: responsible for the draft of the manuscript; FBH, KMR, WCW, and JEM: obtained funding. None of the authors had any financial disclosures.

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