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Intake of sucrose-sweetened beverages and risk of developing pharmacologically treated hypertension in women: cohort study

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ABSTRACT

Objective To investigate the association between intake of sucrose-sweetened beverages (SSBs) and risk of developing pharmacologically treated hypertension in a population of Norwegian mothers followed up to 10 years after delivery.

Design Women without hypertension at baseline in the Norwegian Mother, Father and Child Cohort Study (n=60 027) who delivered between 2004 and 2009 were linked to the Norwegian Prescription Database to ascertain antihypertensive medication use after the first 90 days following delivery. Diet was assessed by a validated semiquantitative Food Frequency Questionnaire in mid pregnancy. Cox proportional hazard analyses evaluated HRs for the development of hypertension associated with SSB consumption as percent energy by quintiles in multivariable models. Supplemental analyses were stratified by gestational hypertension and by a low versus high sodium-to-potassium intake ratio (<0.78 compared with ≥0.78).

Results A total of 1480 women developed hypertension within 10 years of follow-up. The highest relative to the lowest quintile of SSB intake was associated with an elevated risk for hypertension after adjusting for numerous covariates in adjusted models (HR: 1.20 (95% CI: 1.02 to 1.42)). Consistency in results was observed in sensitivity analyses. In stratified analyses, the high SSB intake quintile associated with elevated hypertension risk among women who were normotensive during pregnancy (HR: 1.25 (95% CI: 1.03 to 1.52)), who had normal body mass index (HR: 1.49 (95% CI: 1.13 to 1.93)) and among women with low sodium to potassium ratio (HR: 1.33 (95% CI: 1.04 to 1.70)).

Conclusions This study provides strong evidence that SSB intake is associated with an increased risk of hypertension in women.

INTRODUCTION

Hypertension represents one of the leading risk factors for cardiometabolic disease and early mortality globally.^{1 2} In the article, 'The wrong white crystals', it was postulated that sugar intake may have greater implications for hypertension development than that of salt.^{3 4} Added sugars to beverages and food reflect

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ High sugar intake has been linked to incident hypertension, but most studies have been conducted in countries where beverages are sweetened with high-fructose corn syrup.

WHAT THIS STUDY ADDS

- ⇒ This study explored prospective associations between sucrose-sweetened beverage intake and incident hypertension in women and accounted for pre-pregnancy and pregnancy-related risk factors.
- ⇒ The results show that sucrose-sweetened beverage intake is associated with developing hypertension in women followed from pregnancy to up to 10 years after delivery and that the association was stronger in low risk than in high-risk groups.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ The study provides additional evidence that sugarsweetened beverage intake is an independent risk factor for hypertension.
- The study adds supporting evidence to the recommendation to limit the intake of added sugars to less than 10% of total energy.

disaccharide sucrose with equal parts of fructose to glucose or high-fructose corn syrup (HFCS) which typically has 50% more fructose relative to glucose.^{5 6} While the current literature remains inconclusive regarding the health implications of sugars, there are several reasons why fructose may be more deleterious than glucose. Both animal and human models, including fructose feeding trials, have shown that high consumption of fructose may increase blood pressure (BP) via activation of the sympathetic nervous system, increases in sodium absorption in the gut and production of uric acid, and via decreases in urinary sodium excretion.^{8–11} Fructose is also suspected of promoting obesity, insulin resistance and dyslipidaemia, all of which would



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impair cardiometabolic health. 12 The majority of epidemiological studies on this topic have been conducted in countries where beverages are sweetened by HFCS.⁵

As beverages in Norway are not sweetened by HFCS, an evaluation of the extent to which sucrose-sweetened beverage (SSB) intake is associated with the development of hypertension in Norway would add meaningfully to the literature. We hypothesised that a habitual high intake of SSBs reported in pregnancy would be a significant risk factor for the development of chronic hypertension among women participating in the Norwegian Mother, Father and Child Cohort Study (MoBa).

METHODS

MoBa is a prospective population-based pregnancy cohort study conducted by the Norwegian Institute of Public Health. ¹³ Recruitment of pregnant participants took place in all regions of Norway from 1999 throughout 2008. The women received an invitation to participate in the study prior to their first ultrasound at 18 weeks gestation; 41% consented to participate. Reasons for not participating were not collected. The cohort includes 114500 children, 95 200 mothers and 75 200 fathers. The current study is based on version eight of the quality-assured data files released for research in 2014. The only MoBa inclusion criterion was that the mothers had to be fluent in Norwegian as all questionnaires were in Norwegian. 13 Therefore, 91% of the women included in the study were of Norwegian ethnicity. MoBa data were linked to the Medical Birth Registry of Norway (MBRN), a compulsory registry of pregnancies (>16 weeks gestation), for relevant information on mothers' health before and during pregnancy, including pregnancy and delivery-related complications. 14 The MoBa data were also linked to the Norwegian Prescription Database (NorPD), which since 2004 registers prescriptions dispensed to non-institutionalised individuals. 15 After delivery, the mothers were followed for up to 10 years (31 December 2013) for the identification of prescriptions for hypertension registered in the NorPD and for migration or deaths registered in the National Registry. Details of this hypertension substudy in MoBa are presented elsewhere. 16 1

A total of 62 746 women delivered in 2004–2009. Women who had hypertension before the index pregnancy were identified by three data sources (MBRN, MoBa, NorPD) and excluded. We also excluded women who had early fetal loss (<22 weeks) (n=1414), who had unrealistically high (>4400 kcal/day), low (<1070 kcal/day) or missing energy intakes (n=1305), leaving 60027 women for analyses. For mothers that participated in MoBa more than once, the last pregnancy was included in the analyses.

Estimation of dietary intake

Dietary intake was estimated using a 255-item semiquantitative Food Frequency Questionnaire (FFQ) developed for MoBa. The FFQ was administered the 22nd week of pregnancy and asked about the average diet since the

beginning of pregnancy.¹⁸ The questionnaire contained details regarding the intake of beverages, food items and dietary supplements. The FFO used in MoBa was validated in 119 participants using a 4-day weighed food diary, 4-day activity monitoring and numerous biomarkers of food and nutrient intakes as reference methods. 19 Furthermore, the relative validity of key food groups was also evaluated. 20-22 Results from the validation studies showed good agreement for total energy intake, nutrients and foods. Energy adjusted intakes of SSBs by the FFQ and the food diary correlated with Spearman's rho=0.51 (95% CIs: 0.36 to 0.63). 19

In this study, the independent variable of interest was SSB intake (ie, any sweetened sodas, colas, energy drinks or cordial beverages (concentrated fruit syrup mixed with water)). Participants listed the amount consumed in glasses, which was converted into mL. Energy (kcal/day) and nutrient intakes were calculated using FoodCalc and the Norwegian food consumption table. The equation for

$$kcal/day \text{ in SSBs} = \frac{mL/day \times 4 \text{ (kcal in 10 mL SSB)}}{10}$$

The SSB variable was then energy-adjusted (as a percentage of total energy intake, %E).²

Outcome

The outcome variable was defined as the use of antihypertensive medications after the first 90 days following delivery. Women's dispensed prescriptions were evaluated through to 31 December 2013. If women only used antihypertensives within the first 90 days postpartum, they were coded as non-hypertensive. Hypertension was considered present if hypertension was listed as the underlying indication for a prescription for any of the following medications: Anatomical Therapeutic Chemical classification codes: antihypertensives (C02), diuretics (C03), beta-blockers (C07), calcium channel blockers (C08) and renin-angiotensin system medications (C09).²⁴

Covariates

Baseline characteristics were obtained from questionnaire 1, administered at 15–17 weeks of pregnancy, while questionnaire 4, administered at 6 months postpartum, provided supplemental information on breastfeeding and maternal weight gain. The baseline questionnaire included self-reported information about maternal prepregnancy smoking, recreational physical activity,²⁵ body weight and height for calculating body mass index (BMI) (kg/m²), health conditions and medication usage and the highest educational level attained (any university or college vs less education). The ratio of sodium to potassium intake was calculated from the FFQ and was included among the covariates as a marker of poor diet quality. A high sodium to potassium ratio is associated with development of hypertension. 16 17

MBRN provided information on maternal age at delivery, parity, gestational age at the time of delivery and maternal health status prior to and during pregnancy (ie, diabetes mellitus including pregestational, gestational and type unspecified; and gestational hypertension with or without proteinuria).

Handling of missing data

For the majority of variables, the percent missing data were very low (0-3%), except for 6-month postpartum smoking (17% missing), breastfeeding status (14% missing) and weight gain (18% missing). An evaluation of the distribution of covariates by missing and non-missing categories of these variables identified that the 6-month postpartum data were not missing at random. For example, the prepregnancy smoking prevalence was notably and significantly higher when breastfeeding, weight gain and smoking information was missing at 6 months postpartum than when available. Therefore, values for missing data for these variables were not imputed.

Statistical analysis

Trends in continuous and bivariate descriptive characteristics by low to high quintiles of SSB intake (as %E) were evaluated in linear and logistic regression, respectively. The median intake of SSB %E for each quintile was entered as the independent variable to facilitate trend analyses of the descriptive characteristics. Cox proportional hazard analyses evaluated HRs for the development of hypertension associated with increasing intake of SSBs using three models with an increasing number of covariates to facilitate the evaluation of stability in coefficients and consistency in results. Variables were selected for the models if they were potential confounders or known predictors of hypertension. Model 1 adjusted for maternal age and total energy intake; model 2 included model 1 covariates and pre-pregnancy BMI (kg/m²), smoking, parity (0, 1, >2 prior births), diabetes mellitus (prior to or during pregnancy or type unspecified), gestational hypertension (with or without proteinuria), preterm delivery (<37 weeks gestation) and physical activity (≤3 times/ week, ≥3times/week, missing). Model 3 included model 2 covariates plus intake of dietary fibre (g/day) and added sugar from food only (E%). Dietary fibre was chosen as a marker of a healthy diet as it reflects intake of unrefined grains, vegetables and fruits and is highly correlated with healthy eating indices.

Additional analyses considered the following variables with model 3 covariates: dietary intake of magnesium and calcium (mg/day), artificially sweetened beverages (mL) and juice/nectar (%E), educational level and breast-feeding status at 6 months. As the inclusion of these variables did not materially alter the results in the main model or in the sensitivity analyses, they were not included in the final models presented.

Three a priori sensitivity analyses were conducted limiting analyses to non-smokers, nulliparous women, and those with minimal weight gain (<7 kg) noted at 6 months postpartum, and to those without diabetes mellitus prior to or during pregnancy. Also, results were compared and contrasted between women with and without gestational hypertension/preeclampsia; and between women with a

high versus low sodium to potassium ratio (≥ 0.78 vs < 0.78 using the median intake as cut-off value).

The Cox assumption of proportionality of hazards was assessed using the Schoenfeld test.²⁶ As the Schoenfeld test indicated deviation from proportionality, we conducted an additional sensitivity analysis limited to the first 90 months of follow-up. Finally, supplemental analyses evaluated SSB intake as servings per day (<1, 1–1.49, >1.5).

Statistical analysis was performed using the software Stata V.16 (StataCorp LP).

RESULTS

Descriptive characteristics

The mother's mean (\pm SD) age at delivery was 31 (\pm 5) years and the mean pre-pregnancy BMI was 24.0 (\pm 4.3) (kg/m²). Further, 43.4% were nulliparous, 28.1% had any college or university education, 16.5% smoked daily and 47.9% were physically active (\geq 3 times/week) prior to pregnancy. During pregnancy, 56.646 (94%) were normotensive while 3381 (6%) were registered with gestational hypertension with or without proteinuria. The mean duration of follow-up was 7.1 (\pm 1.6) years, with a maximum follow-up of 10 years.

The mean (\pm SD) daily energy intake for all participating women was 2293 (\pm 593) kcal (median 2217 kcal), and the mean energy intake from SSBs was 53 (\pm 93) kcal/day. In the lowest quintile of SSB consumption, 6629 (55%) of the mothers reported no SSB intake. On average, SSBs accounted for 2.2 (\pm 3.6) E%.

The mothers in the highest quintile of SSB intake had a slightly higher carbohydrate intake, slightly lower protein intake and lower fibre intake compared with the lowest quintile of SSB consumption. In contrast, the percent of energy from fat, and the intake of magnesium, calcium and sodium/potassium ratio were relatively stable across quintiles (table 1). The mean intake of total added sugars from food and drinks among women in the highest consumption quintile of SSBs was 15.4 (\pm 5.6) E% which is above dietary recommendations that added sugar in the diet should be under 10% of total energy intake.²⁷

Women in the highest SSB intake quintile smoked more, exercised less and had lower educational attainment compared with those in the lowest SSB intake quintile. There was, however, little or no variation in parity or BMI across quintiles. Diabetes mellitus of any type was more prevalent (3.1%) among women in the lowest quintile than among women in the highest quintile of SSB intake (1.1%).

At 6 months postpartum, 4921 (10%) of the MoBa participants had a weight gain of ≥7kg compared with their pre-pregnancy weight. Women in the highest quintile of SSB consumption had a slightly higher percentage with a high weight gain compared with women in the lowest quintile (table 1).



Table 1 Baseline demographic characteristics of women by quintiles of sucrose-sweetened beverages as percent energy intake: MoBa Cohort Study (n=60 027)*

	Quintile 1 (n=12005)	Quintile 2 (n=12004)	Quintile 3 (n=12006)	Quintile 4 (n=12006)	Quintile 5 (n=12006)	P value†
Intake of SSB, E%	(<0.3)	(0.3-0.7)	(0.7–1.5)	(1.5-3.9)	(>3.9)	
Median SSB intake, E%	0	0.5	1.0	2.4	6.8	
Calories from SSBs,‡ E%	0.1±0.1	0.5±0.1	1.1±0.2	2.5±0.7	9.0±6.2	*
Intake of SSBs, mL	3.0±4.1	23.8±9.0	48.8±15.4	121±48	459±356	*
BMI,§ kg/m²	24.1±4.5	23.8±4.1	23.9±4.0	24.0±4.2	24.4±4.5	*
Maternal age, years	31.4±4.4	30.9±4.4	30.5±4.5	30.1±4.6	29.6±4.8	*
Parity	0.8±0.9	0.8±0.9	0.8±0.9	0.8±0.9	0.9±0.9	*
Nulliparous, %	43.4	45.7	45.2	42.5	40.0	*
Smoking,§ %	14.4	12.3	13.3	17.0	25.6	*
Preterm,¶ %	5.6	5.3	5.6	5.3	5.8	
Diabetes,** %	3.1	1.6	1.0	1.0	1.1	*
Gestational hypertension,†† %	5.6	5.4	5.5	5.8	5.9	
High education,‡‡ %	33.3	33.4	30.4	25.2	18.0	*
Physical activity ≥3 times/week, %	55.6	53.6	48.7	44.9	37.0	*
Weight gain ≥7 kg, %	10.1	9.2	8.6	10.5	11.6	*
Total energy intake, kcal/day	2179±578	2312±574	2229±543	2330±603	2413±627	*
Energy from protein, E%	16.3±2.1	15.8±1.9	15.6±1.8	15.2±1.8	14.1±2.0	*
Energy from fat, E%	31.3±5.0	31.6±4.5	31.9±4.3	31.8±4.3	30.6±4.4	*
Energy from carbohydrates, E%	52.3±5.0	52.4±4.5	52.3±4.3	52.9±4.3	55.1±4.8	*
Added sugar, g/day	42.0±25.4	49.4±25.4	50.7±24.3	61.8±28.2	94.1±46.8	*
Added sugar, E%	7.5±3.4	8.4±3.2	9.0±3.1	10.4±3.2	15.4±5.6	*
Fibre, g/day	32.6±11.4	33.1±10.6	30.5±9.6	30.4±10.0	28.8±10.0	*
Sodium/potassium ratio	0.76±0.18	0.77±0.18	0.78±0.17	0.78±0.17	0.79±0.18	*
Orange juice/nectar, mL	149±178	163±176	160±162	178±182	186±202	*
Artificial sweetened beverages, mL	223±385	143±290	108±229	100±217	118±255	*
Magnesium, mg/day	410±120	423±115	397±105	402±113	384±112	*
Calcium, mg/day	1041±426	1091±428	1032±393	1061±429	1007±419	*
Breastfeeding 6 months post-delivery, %	79.9	83.4	83.0	80.7	74.6	*
Juice/nectar, E%	2.7±3.1	2.8±2.8	2.9±2.8	3.1±2.9	3.1±3.1	*
Years of follow-up	6.9±1.6	6.9±1.6	7.1±1.6	7.3±1.6	7.5±1.7	*

^{*}Values are means±SDor percentages.

Prospective risk of developing hypertension

A total of 1480 (3%) mothers developed pharmacologically treated hypertension three or more months after delivery. As reported previously, the median duration of antihypertensive medication usage in this cohort was 24 months (IQR 9–48 months) with increasing duration of medication usage observed with increasing years of follow-up. The risk of developing hypertension was higher for the highest relative to the lowest quintile of

SSB intake in all three models evaluated (table 2): a 20% greater risk of developing hypertension was observed in the highest compared with the lowest SSB quintile (model 3 HR: 1.20 (95% CI: 1.02 to 1.42)). The sensitivity analyses identified similar HRs associated with the highest SSB intake quintile for non-smokers (HR: 1.20 (95% CI: 0.99 to 1.45)), and first-time mothers (HR: 1.22 (95% CI: 0.91 to 1.64)), although CIs were wider given the smaller number of participants in these sub-analyses.

[†]Trends in continuous and bivariate descriptive characteristics from low to high intake quintiles of SSBs %E were evaluated in linear and logistic regression, respectively, *P trend <0.001.

[‡]Sugar-sweetened soda and cordial.

[§]Before pregnancy.

[¶]Gestational age <37 weeks or when gestational age was missing a birth weight <2500 g.

^{**}Diabetes mellitus (before or during pregnancy or unspecified).

^{††}With or without proteinuria diagnosed after 20 weeks of gestation, when systolic blood pressure was 140 mm Hg or diastolic blood pressure was 90 mm Hg, or both.

^{‡‡}Any university or college education.

BMI, body mass index; E%, as percent of total energy; SSB, sucrose-sweetened beverage.

Table 2 The risk of incident hypertension within 10 years following delivery by quintile intake of sucrose-sweetened beverages as percent energy: MoBa Cohort Study (n=60027)

Entire cohort quintiles SSB, %E	Participants,	Cases, n	Model 1* HR (95% CI)	Model 2† HR (95% CI)	Model 3‡ HR (95% CI)
Q1	12005	312	Referent	Referent	Referent
Q2	12004	224	0.76 (0.64 to 0.90)	0.88 (0.73 to 1.05)	0.88 (0.73 to 1.05)
Q3	12006	279	0.93 (0.79 to 1.09)	1.05 (0.89 to 1.25)	1.04 (0.88 to 1.23)
Q4	12006	279	0.93 (0.79 to 1.09)	1.01 (0.85 to 1.19)	0.98 (0.83 to 1.17)
Q5	12006	386	1.27 (1.09 to 1.47)	1.25 (1.07 to 1.47)	1.20 (1.02 to 1.42)
Sensitivity analyses					
Non-smokers					
Q1	9952	238	Referent	Referent	Referent
Q2	10187	195	0.85 (0.70 to 1.03)	0.97 (0.80 to 1.18)	0.96 (0.79 to 1.17)
Q3	10099	229	0.99 (0.82 to 1.18)	1.10 (0.91 to 1.33)	1.07 (0.89 to 1.30)
Q4	9637	213	0.96 (0.80 to 1.16)	0.99 (0.82 to 1.21)	0.95 (0.78 to 1.16)
Q5	8673	274	1.34 (1.12 to 1.59)	1.29 (1.08 to 1.55)	1.20 (0.99 to 1.45)
Low weight gain§					
Q1	8730	198	Referent	Referent	Referent
Q2	9096	162	0.82 (0.67 to 1.01)	0.94 (0.76 to 1.16)	0.93 (0.75 to 1.15)
Q3	9145	193	0.95 (0.78 to 1.16)	1.07 (0.87 to 1.31)	1.05 (0.85 to 1.28)
Q4	8953	189	0.95 (0.78 to 1.16)	1.00 (0.81 to 1.23)	0.97 (0.79 to 1.20)
Q5	8465	275	1.42 (1.18 to 1.71)	1.36 (1.13 to 1.65)	1.30 (1.06 to 1.58)
Nulliparous					
Q1	5209	104	Referent	Referent	Referent
Q2	5480	84	0.82 (0.61 to 1.09)	0.92 (0.68 to 1.24)	0.92 (0.68 to 1.24)
Q3	5427	88	0.87 (0.66 to 1.16)	0.96 (0.71 to 1.30)	0.95 (0.70 to 1.29)
Q4	5100	97	1.02 (0.77 to 1.35)	1.09 (0.81 to 1.46)	1.06 (0.79 to 1.43)
Q5	4807	117	1.30 (0.99 to 1.70)	1.28 (0.97 to 1.70)	1.22 (0.91 to 1.64)
First 90 months of follow-up					
Q1	12005	279	Referent	Referent	Referent
Q2	12004	205	0.76 (0.64 to 0.92)	0.89 (0.73 to 1.07)	0.88 (0.73 to 1.07)
Q3	12006	248	0.93 (0.78 to 1.10)	1.05 (0.88 to 1.25)	1.04 (0.87 to 1.24)
Q4	12006	243	0.93 (0.78 to 1.10)	1.00 (0.83 to 1.20)	0.98 (0.82 to 1.18)
Q5	12006	347	1.35 (1.15 to 1.58)	1.32 (1.11 to 1.56)	1.27 (1.06 to 1.51)

^{*}Maternal age at delivery (years), total energy intake (kcal/day) evaluated in Cox proportional hazards analyses.

The association was stronger when restricted to women with BMI in the normal range (18.5–24.9 kg/m²), with a 49% greater risk for the highest compared with the lowest SSB quintile (model 3 HR: 1.49 (95% CI: 1.13 to 1.93)). In the sensitivity analyses limited to women with a low weight gain at 6 months postpartum, the risk of developing hypertension was 42% higher for the highest versus lowest SSB intake quintile when adjusting for maternal age and energy intake in model 1 (HR: 1.42 (95% CI: 1.18 to 1.71)). The HRs were attenuated in model 2 and model 3 but remained statistically significant with a 30%

greater risk of hypertension associated with the highest quintile intake of SSB (model 3 HR: 1.30 (95% CI: 1.06 to 1.58)). Similar findings were observed when analyses were limited to women without pregestational or gestational diabetes mellitus, and in the final sensitivity analysis limited to the first 90 months of follow-up (table 2).

Furthermore, similar results were obtained as those presented when considering additional covariates: educational level, consumption of artificially sweetened drinks, juice/nectar, added sugar from food, dietary calcium, magnesium intake, physical inactivity and breastfeeding.

[†]Maternal age at delivery (years), total energy intake (kcal/day), body mass index, daily smoking prior to pregnancy, parity, diabetes mellitus (prior or during pregnancy or type unspecified), gestational hypertension (with or without proteinuria), preterm delivery (<37 weeks gestation) and physical activity (\geq 3 hours, <3 hours per/week, missing).

[‡]Model 2 covariates plus intake of dietary fibre (g/day) and added sugar from food as percent energy.

^{\$}Low weight gain between prepregnancy and 6 months postpartum weight (<7 kg).

[%]E, as percent of total energy; Q, quintile; SSB, sugar-sweetened beverage.

Table 3 The risk of incident hypertension within 10 years following delivery stratified by women with gestational hypertension or normotensive pregnancies by quintile intake of sucrose-sweetened beverages as percent energy: MoBa Cohort Study (n=60027)

Quintiles SSBs %E	Participants, n	Cases, n	Model 1* HR (95% CI)	Model 2† HR (95% CI)	Model 3‡ HR (95% CI)
Normotensive					
Q1	11331	212	Referent	Referent	Referent
Q2	11353	144	0.72 (0.58 to 0.89)	0.78 (0.62 to 0.97)	0.77 (0.62 to 0.96)
Q3	11342	190	0.93 (0.76 to 1.13)	1.00 (0.81 to 1.23)	0.99 (0.80 to 1.21)
Q4	11316	204	1.00 (0.82 to 1.21)	1.06 (0.87 to 1.30)	1.04 (0.85 to 1.27)
Q5	11304	289	1.38 (1.15 to 1.66)	1.31 (1.08 to 1.58)	1.25 (1.03 to 1.52)
Gest hypt.§					
Q1	674	100	Referent	Referent	Referent
Q2	651	80	0.84 (0.63 to 1.13)	1.10 (0.81 to 1.50)	1.10 (0.81 to 1.50)
Q3	664	89	0.92 (0.69 to 1.23)	1.16 (0.86 to 1.57)	1.15 (0.85 to 1.56)
Q4	690	75	0.76 (0.56 to 1.02)	0.88 (0.64 to 1.21)	0.86 (0.62 to 1.19)
Q5	702	97	1.00 (0.75 to 1.33)	1.06 (0.78 to 1.43)	1.02 (0.75 to 1.40)

^{*}Maternal age at delivery (years), total energy intake (kcal/day) in Cox proportional hazards analyses.

Stratified analyses

For the women who were normotensive during pregnancy, there was a 25% higher risk of developing hypertension after pregnancy for the women who were in the highest compared with the lowest quintile of SSBs intake when adjusting for multiple covariates (model 3 HR: 1.25 (95% CI: 1.03 to 1.52)) (table 3). In contrast, among women with gestational hypertension/preeclampsia, there was no increased risk of developing hypertension associated with the highest quintile of SSB intake (HR: 1.02 (95% CI: 0.75 to 1.40)). When analyses were stratified by a low and high sodium/potassium ratio, SSB Q5 was associated with significantly elevated HRs compared with Q1 only among those with a low sodium/potassium intake ratio (table 4).

Supplemental analyses

In analyses of SSB intake as servings per day, we observed that those reporting consuming 1–1.49 and 1.5 or more servings per day had an elevated risk of developing pharmacologically-treated hypertension within the follow-up period relative to those reporting under 1 serving per day (online supplemental table 1).

DISCUSSION

The results indicate a greater risk of hypertension development within 10 years after delivery associated with the highest relative to the lowest quintile of SSB intake. Findings persisted in a variety of multivariable models and in the sensitivity and stratified analyses, although

with wider CIs given the smaller number of women in the subanalyses.

The current study adds importantly to the literature given that the sweetened beverages were not sweetened with HFCS. Also, the current study is unique in that the analyses were able to take into account relevant prepregnancy and pregnancy-related risk factors.

The most interesting and unique aspect of this study, however, is that that the association of SSB intake with hypertension was stronger in the low-risk groups: non-smokers, normotensive women, those with pre-pregnancy BMI in the normal range (18.5–24.9 kg/m²), those with a low weight gain at 6 months postpartum, those without pregestational or gestational diabetes mellitus and among those with a low sodium/potassium intake ratio. These results provide meaningful additions to the literature suggesting that SSB intake is an independent risk factor for hypertension.

In comparison with findings from the present study, other prospective cohort studies have found a similar increased risk of hypertension with increased intake of SSBs. ^{28–30} These cohorts also identified a greater prevalence of smoking and low educational level and a lower prevalence of physical activity among those who consumed the most SSBs. Additionally, a dose–response meta-analysis conducted in 2014 found a significantly increased risk ratio for every serving/day increase in SSB consumption. ³¹ There are also observed associations between reductions of SSB consumption over time and reduced BP. ³² As hypertension is a primary risk factor for

[†]Maternal age at delivery (years), total energy intake (kcal/day), BMI (kg/m²), smoking (daily smoking prior to pregnancy), parity, diabetes (prior or during pregnancy), gestational hypertension (with or without proteinuria) preterm delivery and physical activity.

[‡]Maternal age at delivery (years), total energy intake (kcal/day), BMI (kg/m²), smoking (daily smoking prior to pregnancy), parity, diabetes (prior or during pregnancy), gestational hypertension (with or without proteinuria) preterm delivery and physical activity, fibre (g/day) and added sugar from food as percent energy.

[§]Gestational hypertensive with or without proteinuria.

BMI, body mass index; %E, as percent of total energy; Q, quintile; SSBs, sugar-sweetened beverages.

Table 4 The risk of incident hypertension within 10 years following delivery by quintile intake of sucrose-sweetened beverages as percent energy stratified by a low and high sodium/potassium intake ratio: MoBa Cohort Study (n=60027)

	Participants, n	Cases, n	Model 1* HR (95% CI)	Model 2† HR (95% CI)	Model 3‡ HR (95% CI)
Low Sod/Pot ratio (<0.78)					
Q1	6757	145	Referent	Referent	Referent
Q2	6661	128	0.94 (0.74 to 1.20)	1.14 (0.89 to 1.45)	1.13 (0.88 to 1.45)
Q3	6279	142	1.10 (0.87 to 1.38)	1.22 (0.95 to 1.56)	1.21 (0.94 to 1.55)
Q4	6321	153	1.18 (0.94 to 1.48)	1.32 (1.04 to 1.67)	1.30 (1.02 to 1.66)
Q5	6160	178	1.38 (1.10 to 1.72)	1.38 (1.09 to 1.74)	1.33 (1.04 to 1.70)
High Sod/Pot ratio (≥0.78)					
Q1	5248	167	Referent	Referent	Referent
Q2	5343	96	0.60 (0.46 to 0.77)	0.67 (0.52 to 0.87)	0.66 (0.51 to 0.86)
Q3	5727	137	0.78 (0.62 to 0.98)	0.92 (0.73 to 1.17)	0.91 (0.72 to 1.15)
Q4	5685	126	0.72 (0.57 to 0.91)	0.77 (0.60 to 0.98)	0.75 (0.58 to 0.96)
Q5	5846	208	1.15 (0.93 to 1.41)	1.14 (0.92 to 1.42)	1.08 (0.86 to 1.36)

^{*}Maternal age at delivery (years), total energy intake (kcal/day).

BMI, body mass index; Q, quintile; Sod/Pot ratio, sodium/potassium ratio.

stroke, it is noteworthy that three studies observed an elevated risk of stroke in association with the intake of SSBs and added sugar. 33–35 Also, given that hypertension associates with insulin resistance, the current results are compatible with findings from a study identifying a higher risk of mortality due to metabolic syndrome among those consuming high levels of SSB. 36

A central question regarding the findings is whether the increased risk of hypertension is a direct effect of SSB consumption, or if the calories it provides contribute to weight gain, and thereby indirectly increase risk of hypertension and related outcomes. It is hypothesised that liquid calories result in less satiety than solid food, and that liquid calories would thereby contribute to additional caloric intake in contrast to solid food calories. This would lead to an energy surplus and, over time, weight gain.^{37 38} Several studies have found an association between SSB and weight gain. 39-42 In the current study, however, there was little variation in mean BMI across intake quintiles of SSBs, and, when those with the highest weight gain at 6 months postpartum were omitted from the analysis, the association between SSB intake and subsequent hypertension was greater than that observed in the primary analyses. As other studies 43 44 have also found associations between metabolic abnormalities and SSBs irrespective of body weight, these findings suggests that there are likely other pathways than weight gain that could explain the relationship between SSB intake and cardiometabolic health.

Another proposed linkage between SSBs cardiometabolic health is that SSBs may be a marker of an unhealthy lifestyle. For example, consumption of SSBs is associated with a western diet consisting of high intakes of red meats, full-fat dairy products, refined grains as well as sugary treats and desserts. 45 SSB intake may be a lifestyle marker and this is corroborated by the socioeconomic differences observed in the current study. Mothers in the highest SSB quintile were slightly younger, had lower educational attainment, were less active and included more smokers. The fact that the women with the highest SSB intake also consumed higher amounts of added sugar and lower amounts of dietary fibre than the women with lower SSB intake, provides evidence that higher SSB intake is associated with poorer diet quality and lifestyle habits. Interestingly, when the smokers were removed from the analyses, we continued to observe an excess risk associated with the highest SSB intake quintile, suggesting that current findings were independent of smoking status. Further, SSBs intake remained significant in the analyses after adjusting for other dietary characteristics such as intake of dietary fibre, added sugar from food only, fruit juice, and magnesium and calcium.

Strengths and limitations

The strengths of the current study include the large sample size of over 60 000 women, the utilisation of three data sources to identify and remove women with pre-existing chronic hypertension, and the prospective

[†]Maternal age at delivery (years), total energy intake (kcal/day), BMI (kg/m²), smoking (daily smoking prior to pregnancy), parity, diabetes (prior or during pregnancy), gestational hypertension (with or without proteinuria) preterm delivery and physical activity.

[‡]Maternal age at delivery (years), total energy intake (kcal/day), BMI (kg/m²), smoking (daily smoking prior to pregnancy), parity, diabetes (prior or during pregnancy), gestational hypertension (with or without proteinuria) preterm delivery and physical activity, fibre (g/day) and added sugar from food as percent energy.

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cohort design with a complete follow-up of participants through record linkages with the National Registry and the NorPD. The large sample size and the wide range of variables available made it possible to conduct several sensitivity and stratified analyses, and to consider a broad array of potential covariates in multivariable analyses.

This study also has limitations. A primary limitation is that we could not assess incident hypertension treated by lifestyle changes. Also, only 41% of eligible women consented to participate in the MoBa cohort, raising concerns regarding generalisability of the results. However, the relative risks of eight exposure-outcome associations in the MoBa cohort were found to be very similar to those obtained in analyses of the entire birth registry, 46 suggesting that MoBa data provide reliable effect estimates. Another limitation is the use of selfreported anthropometric measures. A systematic review and meta-analysis showed that there were slight differences between self-reported and measured weight and height of women of reproductive age, and concluded that the differences have negligible impact on research.⁴⁷ Intake of SSB was assessed one time, precluding our ability to evaluate dietary changes over time. The FFQ in MoBa assessed habitual diet during the first half of pregnancy and dietary habits may change during pregnancy. Still, in MoBa, dietary components assessed during pregnancy is associated with diabetes mellitus⁴⁸ and hypertension¹⁷ up to ten years after delivery, suggesting that diet assessed during pregnancy may serve as a proxy of long-term intake. Studies, in general, indicate that dietary habits track during life and, while we anticipate some dietary changes during pregnancy, misclassification would likely tend to bias results toward the null. Another limitation is the inherent weaknesses of FFQs which are not able to precisely estimate dietary intakes. Underreporting is a well-recognised problem and among the food items that are likely to be underreported are those high in carbohydrates, such as SSBs. $^{49\,50}$ Further, the FFQ did not assess salt added during food preparation or at table. Although industrially prepared food contributes most to salt intake, the sodium/potassium ratio is likely underestimated. Despite weaknesses, FFQs are recognised as the most efficient and feasible method for evaluating habitual intake in epidemiological studies. 18

CONCLUSION

In summary, this study suggests that SSBs are an independent risk factor for hypertension development in a cohort of women followed from pregnancy to up to 10 years following delivery. The research provides novel contributions to the literature given that the present study was able to consider a wide range of known or suspected risk factors for hypertension, including important pregnancyrelated risk factors, and given that the results were stronger in the low-risk groups evaluated. The results also suggest that the current dietary recommendation to limit intake of added sugars in the diet to less than 10%

of total energy intake is likely sufficient for the prevention of hypertension. Importantly, the study contributes to new knowledge as the sugary drinks consumed were sweetened with sucrose and not HFCS.

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Ethics approval This study involves human participants. The establishment of MoBa and initial data collection was based on a license from the Norwegian Data Protection Agency and approval from The Regional Committees for Medical and Health Research Ethics. The MoBa cohort is currently regulated by the Norwegian Health Registry Act. The current study was approved by The Regional Committees for Medical and Health Research Ethics (2013/740). Participants gave informed consent to participate in the study before taking part.

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Data availability statement Data are available upon reasonable request. The consent given by the participants does not open for storage of data on an individual level in repositories or journals. Researchers who want access to data sets for replication should apply through helsedata.no. Access to data sets requires approval from The Regional Committee for Medical and Health Research Ethics in Norway and an agreement with MoBa.

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REFERENCES

- Lawes CMM, Vander Hoorn S, Law MR, et al. Blood pressure and the global burden of disease 2000. Part II: estimates of attributable burden. J Hypertens 2006;24:423-30.
- Oparil S, Acelajado MC, Bakris GL, et al. Hypertension. Nat Rev Dis Primers 2018;4:18014.
- DiNicolantonio JJ, Lucan SC. The wrong white crystals: not salt but sugar as aetiological in hypertension and cardiometabolic disease. Open Heart 2014;1:e000167.
- DiNicolantonio JJ, O'Keefe JH, Lucan SC. An unsavory truth: sugar, more than salt, predisposes to hypertension and chronic disease. Am J Cardiol 2014;114:1126-8.
- Walker RW, Dumke KA, Goran MI. Fructose content in popular beverages made with and without high-fructose corn syrup. Nutrition 2014;30:928-35.

- 6 Ventura EE, Davis JN, Goran MI. Sugar content of popular sweetened beverages based on objective laboratory analysis: focus on fructose content. Obesity 2011;19:868–74.
- 7 Nguyen S, Lustig RH. Just a spoonful of sugar helps the blood pressure go up. Expert Rev Cardiovasc Ther 2010;8:1497–9.
- 8 Dupas J, Feray A, Goanvec C, *et al.* Metabolic syndrome and hypertension resulting from fructose enriched diet in Wistar rats. *Biomed Res Int* 2017;2017:2494067.
- 9 Sánchez-Lozada LG, Tapia E, Jiménez A, et al. Fructose-Induced metabolic syndrome is associated with glomerular hypertension and renal microvascular damage in rats. Am J Physiol Renal Physiol 2007;292:F423–9.
- 10 Perez-Pozo SE, Schold J, Nakagawa T, et al. Excessive fructose intake induces the features of metabolic syndrome in healthy adult men: role of uric acid in the hypertensive response. Int J Obes 2010;34:454–61.
- 11 Soleimani M, Alborzi P. The role of salt in the pathogenesis of fructose-induced hypertension. *Int J Nephrol* 2011;2011:392708.
- 12 Hannou SA, Haslam DE, McKeown NM, et al. Fructose metabolism and metabolic disease. *J Clin Invest* 2018;128:545–55.
- 13 Magnus P, Birke C, Vejrup K, et al. Cohort profile update: the Norwegian mother and child cohort study (MobA). Int J Epidemiol 2016;45:382–8.
- 14 Irgens LM. The medical birth registry of Norway. epidemiological research and surveillance throughout 30 years. Acta Obstet Gynecol Scand 2000;79:435–9.
- 15 Furu K. Establishment of the nationwide Norwegian Prescription Database (NorPD) - New opportunities for research in pharmacoepidemiology in Norway. *Norsk Epidemiologi* 2008:18:129–36.
- 16 Egeland GM, Skurtveit S, Staff AC, et al. Pregnancy-Related Risk Factors Are Associated With a Significant Burden of Treated Hypertension Within 10 Years of Delivery: Findings From a Population-Based Norwegian Cohort. J Am Heart Assoc 2018;7. doi:10.1161/JAHA.117.008318. [Epub ahead of print: 13 05 2018].
- 17 Egeland GM, Skurtveit S, Sakshaug S, et al. Low calcium intake in midpregnancy is associated with hypertension development within 10 years after pregnancy: the Norwegian mother and child cohort study. J Nutr 2017:147:1757–63.
- 18 Meltzer HM, Brantsaeter AL, Ydersbond TA, et al. Methodological challenges when monitoring the diet of pregnant women in a large study: experiences from the Norwegian mother and child cohort study (MoBa). Matern Child Nutr 2008;4:14–27.
- 19 Brantsaeter AL, Haugen M, Alexander J, et al. Validity of a new food frequency questionnaire for pregnant women in the Norwegian mother and child cohort study (MoBa). Matern Child Nutr 2008;4:28–43.
- 20 Brantsaeter AL, Haugen M, Rasmussen SE, et al. Urine flavonoids and plasma carotenoids in the validation of fruit, vegetable and tea intake during pregnancy in the Norwegian mother and child cohort study (MoBa). Public Health Nutr 2007;10:838–47.
- 21 Brantsaeter AL, Haugen M, Julshamn K, et al. Evaluation of urinary iodine excretion as a biomarker for intake of milk and dairy products in pregnant women in the Norwegian mother and child cohort study (MoBa). Eur J Clin Nutr 2009;63:347–54.
- 22 Brantsaeter AL, Haugen M, Thomassen Y, et al. Exploration of biomarkers for total fish intake in pregnant Norwegian women. Public Health Nutr 2010;13:54–62.
- 23 Willett WC, Howe GR, Kushi LH. Adjustment for total energy intake in epidemiologic studies. Am J Clin Nutr 1997;65:1220S–8.
- 24 WHO. ATC/DDD index 2020 WHO collaborating centre for drug statistics methodilogy, 2019. Available: https://www.whocc.no/atc_ ddd_index/ accessed 01.25.20
- 25 Brantsaeter AL, Owe KM, Haugen M, et al. Validation of self-reported recreational exercise in pregnant women in the Norwegian mother and child cohort study. Scand J Med Sci Sports 2010;20:e48–55.
- 26 Hernán MA. The hazards of hazard ratios. *Epidemiology* 2010;21:13–15.
- 27 Nishida C, Uauy R, Kumanyika S, et al. The joint WHO/FAO expert consultation on diet, nutrition and the prevention of chronic diseases: process, product and policy implications. Public Health Nutr 2004;7:245–50.
- 28 Cohen L, Curhan G, Forman J. Association of sweetened beverage intake with incident hypertension. *J Gen Intern Med* 2012;27:1127–34.

- 29 Duffey KJ, Gordon-Larsen P, Steffen LM, et al. Drinking caloric beverages increases the risk of adverse cardiometabolic outcomes in the coronary artery risk development in young adults (CARDIA) study. Am J Clin Nutr 2010;92:954–9.
- 30 Kwak JH, Jo G, Chung H-K, et al. Association between sugarsweetened beverage consumption and incident hypertension in Korean adults: a prospective study. Eur J Nutr 2019;58:1009–17.
- 31 Xi B, Huang Y, Reilly KH, et al. Sugar-Sweetened beverages and risk of hypertension and CVD: a dose-response meta-analysis. *Br J Nutr* 2015;113:709–17.
- 32 Chen L, Caballero B, Mitchell DC, et al. Reducing consumption of sugar-sweetened beverages is associated with reduced blood pressure: a prospective study among United States adults. Circulation 2010;121:2398–406.
- 33 Bernstein AM, de Koning L, Flint AJ, et al. Soda consumption and the risk of stroke in men and women. Am J Clin Nutr 2012;95:1190–9.
- 34 Narain A, Kwok CS, Mamas MA. Soft drinks and sweetened beverages and the risk of cardiovascular disease and mortality: a systematic review and meta-analysis. *Int J Clin Pract* 2016;70:791–805.
- 35 Pacheco LS, Lacey JV, Martinez ME, et al. Sugar-Sweetened beverage intake and cardiovascular disease risk in the California teachers study. J Am Heart Assoc 2020;9:e014883.
- 36 Malik VS, Li Y, Pan A, et al. Long-Term consumption of sugarsweetened and artificially sweetened beverages and risk of mortality in US adults. *Circulation* 2019;139:2113–25.
- 37 DiMeglio DP, Mattes RD. Liquid versus solid carbohydrate: effects on food intake and body weight. *Int J Obes Relat Metab Disord* 2000;24:794–800.
- 38 Ranawana V, Henry CJK. Liquid and solid carbohydrate foods: comparative effects on glycemic and insulin responses, and satiety. *Int J Food Sci Nutr* 2011;62:71–81.
- 39 Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. *BMJ* 2012;346:e7492.
- 40 Schulze MB, Manson JE, Ludwig DS, et al. Sugar-Sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. JAMA 2004;292:927–34.
- 41 Malik VS, Pan A, Willett WC, et al. Sugar-Sweetened beverages and weight gain in children and adults: a systematic review and metaanalysis. Am J Clin Nutr 2013;98:1084–102.
- 42 Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. Am J Clin Nutr 2006:84:274–88.
- 43 Green AK, Jacques PF, Rogers G, et al. Sugar-Sweetened beverages and prevalence of the metabolically abnormal phenotype in the Framingham heart study. Obesity 2014;22:E157–63.
- 44 Barrio-Lopez MT, Martinez-Gonzalez MA, Fernandez-Montero A, et al. Prospective study of changes in sugar-sweetened beverage consumption and the incidence of the metabolic syndrome and its components: the SUN cohort. Br J Nutr 2013;110:1722–31.
- 45 Fung TT, Rimm EB, Spiegelman D, et al. Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk. Am J Clin Nutr 2001;73:61–7.
- 46 Nilsen RM, Vollset SE, Gjessing HK, et al. Self-Selection and bias in a large prospective pregnancy cohort in Norway. Paediatr Perinat Epidemiol 2009;23:597–608.
- 47 Seijo M, Minckas N, Cormick G, et al. Comparison of self-reported and directly measured weight and height among women of reproductive age: a systematic review and meta-analysis. Acta Obstet Gynecol Scand 2018;97:429–39.
- 48 Øyen J, Brantsæter AL, Nøstbakken OJ, et al. Intakes of fish and long-chain n-3 polyunsaturated fatty acid supplements during pregnancy and subsequent risk of type 2 diabetes in a large prospective cohort study of Norwegian women. *Diabetes Care* 2021;44:2337–45. doi:10.2337/dc21-0447
- 49 Olafsdottir AS, Thorsdottir I, Gunnarsdottir I, et al. Comparison of women's diet assessed by FFQs and 24-hour recalls with and without underreporters: associations with biomarkers. Ann Nutr Metab 2006;50:450–60.
- 50 Macdiarmid J, Blundell J. Assessing dietary intake: Who, what and why of under-reporting. *Nutr Res Rev* 1998;11:231–53.

Supplemental Table 1: The risk of incident hypertension within 10 years following delivery by servings per day of sucrose-sweetened beverages: MoBa Cohort Study (N= 60,027)

Entire cohort Servings of SSB ¹	Participants N	Cases n	Model 1 ² HR (95% CI)	Model 2 ³ HR (95% CI)	Model 3 ⁴ HR (95% CI)
< 1	51,143	1,179	Referent	Referent	Referent
1-1.49	3728	122	1.40 (1.16, 1.69)	1.37 (1.13, 1.67)	1.34 (1.11, 1.64)
<u>≥</u> 1.50	5156	179	1.55 (1.31, 1.82)	1.29 (1.09, 1.53)	1.23 (1.03, 1.48)

Abbreviations: CI, confidence interval; HR, hazard ratio; SSB, sugar-sweetened beverages.

- One serving =250ml
- Maternal age at delivery (years), total energy intake (kcal/day) evaluated in Cox proportional hazards analyses.
- 3. Maternal age at delivery (years), total energy intake (kcal/day), body mass index (kg/m²), daily smoking prior to pregnancy, parity, diabetes mellitus (prior or during pregnancy or type unspecified), gestational hypertension (with or without proteinuria), preterm delivery (<37 weeks gestation) and physical activity (≥3 hrs, <3 hrs per/week, missing).
- 4. Model 2 covariates plus intake of dietary fiber (g/day) and added sugar from food as percent energy.