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Original article

The association of sweetened beverage intake with risk of type 2 diabetes in an Australian population: A longitudinal study

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ABSTRACT

Aim: Globally, sugar intake from sugar-sweetened beverages (SSBs) exceeds the daily recommended limits for intake levels of free sugar. Artificially sweetened beverages (ASBs), widely used to replace SSBs, are increasingly linked to adverse health outcomes. Hence, we assessed the association of sweetened beverage intake (SSBs and ASBs) with the risk of type 2 diabetes (T2DM).

Methods: Data from the Melbourne Collaborative Cohort Study (MCCS) on 36,608 individuals aged 40 to 69 years were used. Self-reported data on diabetes were collected. The frequency of SSBs and ASBs consumption was categorized as: never or < 1 time/month; 1–3 per month; 1–6 times per week; ≥1 time / day. The association of sweetened beverage intake with the incidence of T2DM was assessed using modified Poisson regression, adjusted for lifestyle, obesity, socioeconomic, and other confounders.

Results: Intakes of SSBs and ASBs were associated with an increased risk of T2DM. A high intake (≥ 1 time/day) compared to a low intake (never or < 1 time / month) was associated with increased risk of T2DM for SSB intake (incidence risk ratio (IRR) = 1.23; 95 % CI: 1.05–1.45; P for trend = 0.006) and for ASB intake (IRR = 1.38; 95 % CI: 1.18–1.61; P for trend < 0.001). Further adjustment for body mass index (BMI) and waist-to hip ratio (WHR) eliminated the association for SSBs, but not ASBs intake.

Conclusions: Both sugar and artificially sweetened beverages were linked to an increased risk of T2DM. The findings highlight the need for public health measures to control the intake of sweetened beverages.

Introduction

The global prevalence of diabetes was 10.5 % (537 million) among adults aged 20 to 79 years in 2021; of these, 90 % of the disease burden is type 2 diabetes [1]. The prevalence and incidence of type 2 diabetes are high and expected to increase globally, mainly geared by the rising rate of obesity due to unhealthy lifestyle [2]. Currently, an estimated 1.3 million (one in twenty) people live with diabetes in Australia [3]. However, research findings indicate underreporting of the actual burden, with many living with undiagnosed diabetes [4,5].

Globally, the consumption of sugar-sweetened beverages (SSBs) contributes to free sugar intake exceeding daily recommended limits [6]. High added sugar intake, especially from beverages, is linked with an increased risk of cardiovascular disease and all-cause mortality [7]. Previous population-based observational studies have reported an association between the consumption of SSBs and type 2 diabetes risk [8,

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9]. Accordingly, the World Health Organization (WHO) recommends reducing the intake of free sugars to <10 % of total energy intake in adults and children [10].

Artificially sweetened beverages (ASBs) were first introduced to replace SSBs and reduce calorie intake by reducing free sugar intake [11]. Evidence indicates that the consumption of carbonated soft drinks containing artificial sweeteners in Australia has increased since 1994 [12]. Recent growing evidence also shows high habitual intake of artificial sweeteners is linked to various adverse health outcomes such as type 2 diabetes [13] and mortality [14]. Several mechanistic studies have revealed the impacts of artificial sweeteners on the gut microbiome (i.e., artificial sweetener-induced dysbiosis [15]) and intestinal glucose absorption [16], which are implicated in the deterioration of glucose homeostasis. A recent systematic review and meta-analysis of prospective studies by the WHO on the health effects of non-sugar-sweetened beverages showed a short-term reduction in weight and adiposity but increased long-term risk of obesity, type 2 diabetes, and cardiovascular diseases [17].

Although findings on the link between SSB intake and the risk of type 2 diabetes are relatively consistent [13], the level and type of sugar used in these beverages vary across different regions of the world [18]. For instance, sucrose, a disaccharide made from 50 % glucose and 50 % fructose, is widely used in Australia, whereas high fructose corn syrup is widely used in the United States [18]. Metabolic effects of glucose and fructose are different; fructose overconsumption seems a stronger driver of visceral central fat accumulation [19]. The effect of Australian sugar-sweetened beverages containing high glucose on the risk of type 2 diabetes has not been explored. A previously published report on the sugar contents of SSB highlighted the need to explore the link between SSBs and health outcomes, including type 2 diabetes in the Australian context [18].

A recent review article on sugar and artificially sweetened drinks in Australia reported that no studies have examined its effects on indices of glycaemic control, and the evidence for the health impact of intense-sweetened drinks is limited, thereby highlighting the need for local evidence on the role of SSBs on cardio-metabolic health outcomes including type 2 diabetes [20]. Similarly, the link between ASBs and health outcomes, including type 2 diabetes, is less explored in an Australian setting. Hence, we aimed to assess the association of sweetened beverage intake with the risk of type 2 diabetes using the Melbourne Collaborative Cohort Study (MCCS) data.

Material and methods

The Melbourne collaborative cohort study (MCCS)

The Melbourne Collaborative Cohort Study is a prospective study conducted in Melbourne, recruiting 41,513 participants from Melbourne residents between 1990 and 1994 (baseline). The detailed steps and procedures followed during the recruitment of participants, follow-up, and the data collection process have been described elsewhere [21]. Participants were recruited using the electoral roll and a direct approach through clubs, churches, and culturally specific media. Socio-demographic and nutritional data were collected using interviewer-administered questionnaires at the outset. The first follow-up survey (wave 1) was conducted between 1995 and 1998, and the second follow-up was between 2003 and 2007 (wave 2). As shown in Fig. 1, at baseline, 36,608 participants were included after excluding participants with extreme energy intake values, diabetes, history of heart attack, and history of angina. 32,284 were included in the first wave of follow-up and 24,557 in the second wave of follow-up. The average follow-up period was 13.9 years.

During the first follow-up, data were obtained using either a mailed self-administered questionnaire or an interview via phone. In the second follow-up visit, self-administered questionnaires were used to collect data, and anthropometric measures except height were repeated [21].

Dietary assessment

At baseline, a self-administered Food Frequency Questionnaire (FFQ) with 121 items was used to collect dietary consumption data [22]. The sex-specific average portion size was derived for each food item, and the

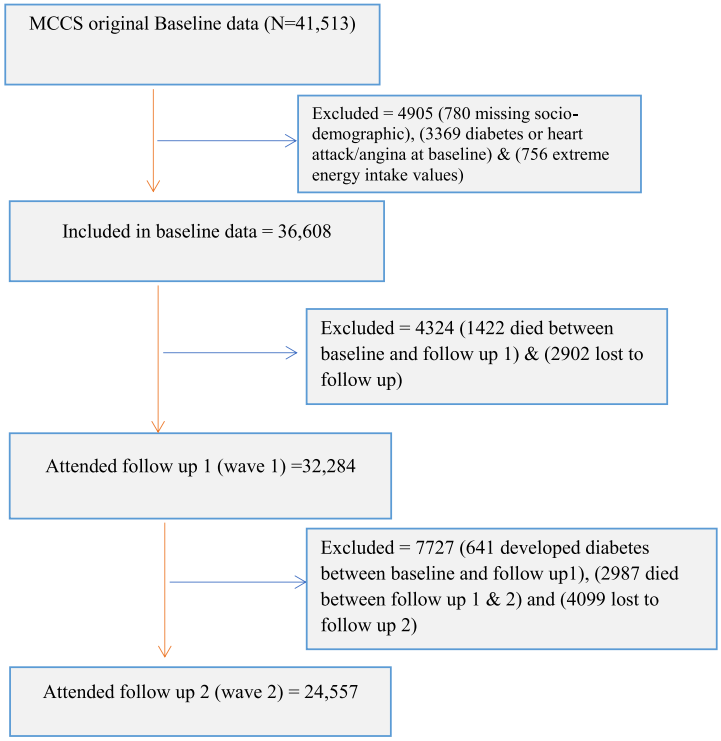

Fig. 1. Flow diagram showing the recruitment and follow-up of the cohort for assessing the association of sweetened beverage intake with the risk of type 2 diabetes.

frequency of intake for some fruits was adjusted to account for seasonality. Mean daily nutrient intakes were calculated by multiplying the daily frequency of each food item by the nutritional composition and portion size. Most of the nutrient composition data came from the Australian food composition tables [23]. Additional data was derived from British tables (folate and vitamin E) [24]. As a measure of overall diet quality to include in multivariable models, we calculated the Alternative Healthy Eating Index 2010 (AHEI-2010) [25], which we have already shown to be associated with weight gain and type 2 diabetes risk in the MCCS [26,27].

Sweetened beverage intake

Data on sweetened beverage intake were derived from the FFQ which included questions on the frequency of consumption of regular (sugar-sweetened) and diet (artificially sweetened) soft drinks ('never or less than once per month'; '1–3 per month'; '1 per week'; '2–4 per week'; '5–6 per week'; '1 per day'; '2–3 per day'; '4–5 per day'; '6 or >6 per day'). We re-categorized frequency of soft drink intake into four categories: never or < 1 time / month; 1–3 per month; 1–6 times / week; ≥ 1 times / day.

Other covariates

At baseline, interviewer-administered questionnaires were used to collect information on age, self-reported sex, country of origin, smoking, alcohol intake, and physical activity. Participants were categorized into three groups based on their region of origin: 1. Australia/New Zealand, 2. Northern European (mainly British) and 2. Southern European (Greek and Italian). Deciles of the Socioeconomic Indexes for Areas (SEIFA) Index of Relative Socioeconomic Disadvantage based on postcode at baseline were used to indicate socioeconomic standing [28]. SEIFA deciles were recoded into quintiles, with the first quintile being the most disadvantaged and the fifth quintile being the most prosperous. A standardised questionnaire was used to assess how often participants spent doing low, moderate, and high levels of physical exercise, and these data were combined to give an overall score that weighted time spent doing vigorous activity twice that of less vigorous activity. The score was divided into four categories: 0; > 0–4; > 4–6, and > 6.

Height, weight, and waist and hip circumferences were all measured using standard procedures, and body mass index (BMI) (in kilograms per meter squared) and waist-to-hip ratio (WHR) were calculated.

Outcome measurement

A self-administered questionnaire was mailed to individuals around four years after baseline and included questions on type 2 diabetes diagnosis. Participants were asked "Has a doctor ever told you that you have diabetes?" Those who answered yes were asked to state the year of their diagnosis. Subjects who indicated a diagnosis date before baseline were excluded. Of the subjects reporting a diabetes diagnosis since baseline, 76 % had their diagnosis confirmed by their doctor and, unless specified, were considered to have type 2 diabetes due to the age of onset. At the second wave of follow-up, similar questions on diabetes were repeated to identify incident cases.

Data analysis

The lowest intake category (never or < 1 time / month) was used as the reference category in models for both sugar and artificial sweetened beverage intakes. At the first and second follow-ups, the cumulative incidence of type 2 diabetes was compared across predictor categories. Multivariable Generalized estimated equation modified Poisson regression model [29] with robust error variance [30] was used to investigate the associations of sugar sweetened beverage intake with the incidence of type 2 diabetes after adjusting for confounders. Survival analysis was not used as we did not have specific data on the time of type 2 diabetes diagnosis in this cohort.

Previous scientific literatures were used to determine potential predictor variables. Three models were used to calculate the incidence rate ratio (IRR) of type 2 diabetes incidence. Model 1 adjusted for age, sex, SEIFA (quintiles 1–5), smoking status (never, former, and current), alcohol drinking status (never, former, and current), family history of diabetes, physical activity level, AHEI-2010 quintiles, comorbidity status, energy intake (KJ/day), and region of origin. These factors were considered as potential confounders based on previous literature and scientific evidence. Model 2 was fitted using model 1 variables plus BMI, while Model 3 was fitted using model 2 variables plus WHR. Trends in soft drink intake across categories were calculated by assigning a median score to each person in that category, and P for trend was reported. Stata version 18 (StataCorp, College Station, TX, USA) was used for all statistical analyses.

Based on the literature, obesity could be considered a mediator for SSB but a confounder for ASBs. Thus, we interpreted the appropriately adjusted model, which is not adjusted for obesity (BMI/WHR) in SSBs intake and adjusted for obesity in ASBs intake.

Additional analyses: A sensitivity analysis by excluding cases at the first follow-up was also conducted to examine whether the observed association reflects a possible reverse causality.

Results

Baseline characteristics by frequency of sweetened beverage consumption are presented in Table I. Individuals with the highest SSB intake tended to have higher BMI, higher central obesity, higher total energy intake, higher total sugar intake, were more likely to be male, to be socio-economically disadvantaged, to be smokers, to be less physically active, have Australia/New Zealand origin, more likely to drink artificially sweetened beverages, lower overall diet quality and likely to have comorbidity.

The most frequent consumers of ASBs tended to have higher, BMI, higher central obesity, higher total energy intake, higher total sugar intake, were more likely to be female, be socio-economically disadvantaged, to be smokers, to be less physically active, have Australia/New Zealand origin, less likely to drink sugar-sweetened beverages, lower overall diet quality and likely to have comorbidity.

During the first follow-up period, 641 cases of type 2 diabetes were reported and 1141 cases in the second follow-up period, giving a total of 1782 incident cases of type 2 diabetes. At both wave 1 and wave 2, a relatively higher incidence of diabetes was observed among older individuals, men, socio-economically disadvantaged individuals, southern Europeans, current smokers, alcohol abstainers, and those with higher BMI, higher WHR, low AHEI-2010 quintile, family history of diabetes, history comorbidity, and higher intake of SSBs and ASBs. Both at wave 1 and wave 2, all variables showed a significant association ($P < 0.05$) with the risk of type 2 diabetes, except for energy intake quintiles at wave 2 ($P = 0.13$) (Table II).

The associations between SSB and ASB intake and the risk of type 2 diabetes are presented in Table III. In model 1 (adjusted for age, sex, socioeconomic index (SEIFA), smoking status, lifetime alcohol drinking status, physical activity score, family history of diabetes, history of comorbidity, quintiles of energy intake, region of origin, alternative healthy eating index quintiles and total sugar intake) higher intake of SSBs (≥ 1 time / day) showed a 23 % increase in the risk of type 2 diabetes (IRR=1.23, 95 % CI: 1.05 −1.45, *P*-value for trend = 0.006). Similarly, higher intake of ASB (≥ 1 time / day) showed an 83 % increase in the risk of type 2 diabetes (IRR=1.83, 95 % CI: 1.57- 2.13, *P*-value for trend < 0.001).

In model 2 and model 3, we additionally adjusted for obesity (BMI) and waist-to-hip circumference ratio (WHR), respectively. In model 2, a higher intake of ASBs (≥ 1 time / day) showed a 43 % increase in the risk of type 2 diabetes (IRR=1.43, 95 % CI: 1.23–1.67, *P* for trend < 0.001).

Table I
Baseline descriptive analysis of variables by categories of sweetened beverage intake.

	Frequency of sugar sweetened beverage intake				
	Never or < 1time/ month (n = 19,202)	1–3 times/month (n = 6354)	1–6 times/week (n = 8036)	≥1 time/day (n = 3016)	*P*-value
Age (years)(mean, SD)	55.75±8.53	53.74±8.51	53.53±8.65	54.90±8.66	0.32
BMI (kg/m2)(mean, SD)	26.51±4.43	26.56±4.11	27.05±4.30	27.82±4.42	< 0.001
Waist (cm) (mean, SD)	83.37±12.69	84.40±12.43	86.73±12.48	89.24±12.75	0.09
Energy (kj/day) (mean, SD)	8312±2868.97	8897.3 ± 2894.2	9417.77±3143.35	10,065±3341.65	< 0.001
Sugar intake, g/day (mean ± SD)	115.83±61.48	123.16±60.83	133.15±63.58	164.92±71.77	< 0.001
Alcohol intake, g/day (median, IQR)	2.14 (0–14.89)	3.72 (0–14.23)	3.72 (0–15)	2.14 (0–15)	< 0.001
Female, n (%)	13,009 (67.8)	3762 (59.2)	4068 (50.6)	1424 (47.2)	< 0.001
SEIFA Q5 (least disadvantaged), n (%)	5270 (27.45)	1890 (29.75)	2271 (28.26)	621 (20.59)	< 0.001
Current smoker, n (%)	2098 (10.9)	577 (9.1)	877 (11.0)	445 (14.8)	< 0.001
Physical activity score ≥ 6 (physically active), n (%)	4476 (23.3)	1478 (23.3)	1861 (23.2)	527 (17.5)	< 0.001
Region of origin, n (%)					
Australia/New Zealand	13,275 (69.1)	4648 (73.2)	5919 (73.7)	1735 (57.5)	< 0.001
Northern Europe	1363 (7.1)	387 (6.1)	467 (5.8)	151 (5.0)	
Southern Europe	4564 (23.8)	1319 (20.8)	1650 (20.5)	1130 (37.5)	
Artificially sweetened beverage >1/day, n (%)	1052 (5.5)	199 (3.1)	348 (4.3)	504 (16.7)	< 0.001
AHEI Q5, n (%)	5028 (26.2)	1187 (18.7)	894 (11.1)	125 (4.1)	< 0.001
Comorbidity status (Yes), n (%)	10, 431 (54.3)	3140 (49.4)	4086 (50.9)	1637 (54.3)	< 0.001
	Frequency of artificially sweetened beverage intake				
	Never or < 1time/ month (n = 27,460)	1–3 times/month (n = 3011)	1–6 times/week (n = 4034)	≥1 time/day (n = 2103)	*P* - value
Age (years)(mean ± SD)	55.29±8.65	53.57±8.51	53.29±8.40	53.90±8.50	0.007
BMI (kg/m2)(mean ± SD)	26.36±4.22	27.39±4.28	27.93±4.54	28.67±4.91	< 0.001
Waist (cm) (mean ± SD)	84.04±12.61	85.95±12.60	86.79±12.80	88.12±13.44	0.001
Energy (kj/day) (mean ± SD)	8747.56±3006.95	8861.63±3093.56	8955.60±3049.35	9112±3225.94	< 0.001
Sugar intake, g/day (mean ± SD)	125.08±64.35	121.76±63.70	123.77± 62.29	130.00 ± 67.1	0.001
Alcohol intake, g/day (median, IQR)	2.74 (0–15)	2.14 (0–12.86)	2.74 (0–12.86)	2.14 (0–13.03)	< 0.001
Female, n (%)	16,619 (60.5)	1786 (59.3)	2483 (61.6)	1375 (65.4)	0.031
SEIFA Q5 (least disadvantaged), n (%)	7579 (27.6)	880 (26.8)	1155 (28.6)	621 (24.3)	
Current smoker, n (%)	3066 (11.2)	303 (10.1)	399 (9.9)	239(11.4)	0.002
Physical activity score ≥6 (physically active), n (%)	6180 (22.5)	696 (23.3)	1861 (23.2)	527 (17.5)	0.001
Country/region of birth, n (%)					
Australia/New Zealand	19,142 (69.7)	2031 (67.5)	2897 (71.8)	1507 (71.7)	< 0.001
Northern Europe	1859 (6.8)	154 (5.1)	239 (5.9)	116 (5.5)	
Southern Europe	6459 (23.5)	826 (27.4)	898 (22.3)	480 (22.8)	
Sugar-sweetened beverage >1/day, n (%)	2179 (7.9)	145 (4.8)	188 (4.7)	504 (24.0)	< 0.001
AHEI Q5, n (%)	5600 (20.4)	549 (18.2)	730 (18.1)	355 (16.9)	< 0.001
Comorbidity status (Yes), n (%)	14,352 (52.6)	1599 (53.1)	2123 (52.6)	1220 (58.0)	< 0.001

AHEI: alternative healthy eating index; BMI: body mass index.

In model 3, a higher intake of ASBs (≥ 1 time / day) showed a 38 % increase in the risk of type 2 diabetes (IRR=1.38, 95 % CI: 1.18–1.61; *P* for trend < 0.001). However, associations for intakes of SSBs were no longer apparent after adjustment for BMI and WHR in models 2 and 3, respectively.

Additional sensitivity analysis was conducted by omitting type 2 diabetes cases on first wave of the follow up. For SSBs intake, a positive association with type 2 diabetes was observed (IRR=1.36, 95 % CI: 1.10–1.68, *P* for trend = 0.001). Similarly, for ASBs intake a positive association with type 2 diabetes was observed (IRR=1.58, 95 % CI: 1.28–1.95, *P* for trend < 0.001). (Table IV).

Discussion

We found that a high intake (≥ 1 time / day) of both SSBs and ASBs was associated with an increased risk of type 2 diabetes in this prospective analysis with average follow up of 13.9 years. The association between SSB intake and type 2 diabetes disappeared when adjusted for obesity (BMI) and central adiposity (WHR). However, the association for ASB intake was independent of BMI or WHR.

Findings of previous studies that assessed the association between SSB intake and type 2 diabetes risk have been mixed [31–33]. A Thai cohort study (n = 39,175) reported a strong positive association of SSB intake (≥ 1 time per day compared with < 1 weekly) with the risk of type 2 diabetes in women but not men [31]. Similarly, the Nurses' Health Study II reported that sugar-sweetened soft drink intake of ≥ 1 time / day compared with ≤ 1 time/month resulted in a 43 % higher risk of type 2 diabetes in women [33]. Sex stratified analysis of our data similarly showed a strong positive association in women only (Table SI; see supplementary materials associated with this article on line). Conversely, another study done among middle-aged Japanese men (n = 2037) followed for 7 years reported SSB intake of ≥1 serving/day results reported no association with type 2 diabetes compared with rare/never intake [32]. The possible reasons for no association in the latter might be reflective of the short follow-up period (7 years), small incident cases, and relatively young participants.

In line with our finding, several previous studies have reported a positive association between SSBs intake and type 2 diabetes [13, 34–36]. A European Prospective Investigation into Cancer and Nutrition (EPIC) study on 15,374 male and female participants reported that one serving per day of SSB compared with < one serving per month was associated with a 22 % increase in the risk of type 2 diabetes [36]. Similarly, a meta-analysis of prospective studies reported a significant positive association between SSB and type 2 diabetes risk [13].

Previous studies exploring the association between ASB intake and the risk of type 2 diabetes also reported mixed findings [32,35,37,38]. Our finding is in line with a French prospective study, a French component of the European Prospective Investigation into Cancer and Nutrition, on 66,118 female teachers [38] and another study done among 2037 middle-aged Japanese men [32] that reported an association of high ASB intake with an increased risk of type 2 diabetes. In contrary, a prospective study from 40,389 health professional men [37]

Table II
Incidence of type 2 diabetes in wave one and two by possible predictor variables.

	Wave 1 (n = 31,150)		Wave 2 (n = 21,265)	
	n/N (%)	P - value	n/N (%)	P - value
Age				
<50 years	113/10,592 (1.1)	< 0.001	261/7856 (3.3)	< 0.001
50–59 years	241/10,494 (2.3)		460/7373 (6.2)	
≥60 years	287/10,424 (2.8)		420/6036 (7.0)	
Sex				
Male	315/12,230 (2.6)	< 0.001	530/8021 (6.61)	< 0.001
Female	326/19,280 (1.7)		611/13,244 (4.61)	
SEIFA quintiles				
SEIFA Q1	185/5391 (3.4)	< 0.001	254/3119 (8.1)	< 0.001
SEIFA Q2	164/6344 (2.6)		261/3764 (7.2)	
SEIFA Q3	95/5003 (1.9)		169/3209 (5.3)	
SEIFA Q4	85/5957 (1.4)		202/4300 (4.7)	
SEIFA Q5	112/8815 (1.3)		255/6873 (3.7)	
Region of Origin				
AUS/NZ	293/22,265 (1.3)	< 0.001	661/15,603 (4.2)	< 0.001
Northern Europe	36/2058 (1.8)		55/1448 (3.8)	
Southern Europe	312/7187 (4.3)		425/4214 (10.1)	
Smoking status				
Never	339/18,819 (1.8)	= 0.001	649/13,068 (5.0)	= 0.002
Current Smoker	84/3201 (2.6)		128/1918 (6.7)	
Former Smoker	218/9490 (2.3)		364/6279 (5.8)	
Alcohol drinking				
Lifetime abstainers	238/8780 (2.7)	< 0.001	374/5608 (6.7)	< 0.001
Ex-drinkers	78/3308 (2.4)		135/2208 (6.1)	
Current drinkers	325/19,422 (1.7)		632/13,449 (4.7)	
Physical activity				
0	195/6752 (2.9)	< 0.001	334/4439 (7.5)	< 0.001
>0 and <4	150/6345 (2.4)		248/4422 (5.6)	
≥4 and <6	212/11,057 (1.9)		389/7247 (5.4)	
≥6	84/7356 (1.1)		170/5157 (3.3)	
Waist circumference				
Normal	215/21,120 (1.0)	< 0.001	465/14,735 (3.2)	< 0.001
High	426/10,390 (4.1)		676/6530 (10.4)	
BMI				
< 25.0	54/12,079 (0.5)	< 0.001	126/8494 (1.5)	< 0.001
25.0–29.9	249/13,452 (1.9)		498/9055 (5.5)	
≥30.0	338/5979 (5.7)		517/3716 (13.9)	
Alternative healthy eating index (AHEI_2010)				
Q1	147/6217 (2.4)	< 0.001	283/4003 (7.1)	< 0.001
Q2	161/6728 (2.4)		274/4525 (6.1)	
Q3	141/6081 (2.3)		219/4119 (5.3)	
Q4	108/6119 (1.8)		215/4195 (5.1)	
Q5	84/6365 (1.3)		150/4424 (3.4)	
Family history of diabetes				
No	425/25,948 (1.6)	< 0.001	789/17,644 (4.5)	< 0.001
Yes	216/5562 (3.9)		352/3621 (9.7)	
Comorbidity				
No	182/15,115 (1.2)	< 0.001	399/10,542 (3.8)	< 0.001
Yes	459/16,395 (2.8)		742/10,723 (6.9)	
Artificially sweetened beverage				
Never or < 1time/ month	440/23,696 (1.9)	< 0.001	764/15,833 (4.8)	< 0.001
1–3 times/month	56/2609 (2.2)		99/1783 (5.6)	
1–6 times/week	87/3432 (2.5)		159/2424 (6.6)	
≥1 time/day	58/1773 (3.3)		119/1225 (9.7)	
Energy intake (Kj/day)				
Q1	145/6250 (2.3)	= 0.005	234/3973 (5.9)	0.13
Q2	113/6289 (1.8)		236/4234 (5.6)	
Q3	110/6357 (1.7)		214/4354 (4.9)	
Q4	116/6294 (1.8)		213/4384 (4.9)	
Q5	157/6320 (2.5)		244/4320 (5.7)	
Sugar-sweetened beverage				
Never or < 1time/ month	334/16,503 (2.0)	< 0.002	527/10,867 (4.9)	< 0.001
1–3 times/month	94/5509 (1.7)		198/3893 (5.1)	
1–6 times/week	137/6950 (2.0)		280/4843 (5.8)	
≥1 time/day	76/2548 (3.0)		136/1662 (8.2)	

SEIFA: socioeconomic index for areas, WHR: waist to hip ratio, BMI: body mass index, AU/NZ: Australia/New Zealand.

and a case–cohort analysis from the European Prospective Investigation into Cancer and Nutrition (EPIC) [36] study reported no association.

The observed association between SSB intake and type 2 diabetes is partly attributable to its effect on weight and body composition [6]. Nurses' Health Study II reported a significant weight gain following increased SSB intake [33]. Similarly, a systematic review and meta-analysis of randomized clinical trials by Ruanpend *et al.* showed a significantly increased risk of obesity due to consumption of SSBs [39]. Intake of excess added sugar due to consumption of SSBs is reported to be responsible for increasing the risk of obesity / overweight [40,41]. Furthermore, high intake of SSB can contribute to high glycaemic load, which is suggested to induce a postprandial insulin spike leading to an increase in appetite, weight gain, and insulin resistance in the long term [42]. Lastly, although the formulation of sugars used in SSBs differs across different countries, for instance, sucrose is common in Australia and high fructose corn syrup (HFCS) in USA [18], based on the findings of our study, their effects on risk of type 2 diabetes appear similar.

The mechanisms linking high habitual consumption of ASBs and the risk of type 2 diabetes are not fully understood. It is suggested that reverse causality between obesity and ASB intake may partly explain the observed association, where individuals with relatively high BMI at baseline might be using ASB to try to reduce weight and follow a healthy lifestyle [35,37]. Our results, showing the attenuation of the association of ASB with type 2 diabetes after adjustment for body size measures, were consistent with supportive of obesity being a confounder of the association. It should be noted that biological fates of commercially available artificial sweeteners are quite different, with some poorly absorbed and excreted in the faeces (e.g. sucralose), some well absorbed and excreted in the urine (e.g. acesulfame potassium), and some rapidly metabolized (aspartame) [43]. Accordingly, they may interact with distinct metabolic tissues to influence glucose metabolism. For example, high intake of aspartame, a commonly used artificial sweetener, resulted

Table III
Association of sweetened beverage intake with risk of type 2 diabetes after controlling for confounders.

Category		Model 1*		Model 2: Model 1 + BMI		Model 3: Model 2 + WHR	
		Adjusted IRR (95 % CI)	*P*-value	Adjusted IRR (95 % CI)	*P*-value	Adjusted IRR (95 % CI)	*P*-value
Sugar sweetened beverage intake	Never or < 1 time/ month	Ref		Ref		Ref	
	1–3 times/month	1.00 (0.88–1.14)	0.99	1.00 (0.88–1.14)	0.97	1.00 (0.88–1.13)	0.96
	1–6 times/week	1.10 (0.98–1.24)	0.09	1.02 (0.90–1.14)	0.73	1.00 (0.90–1.13)	0.87
	≥1 time/day	1.23 (1.05 −1.45)	0.01	1.08 (0.93–1.27)	0.30	1.06 (0.90–1.24)	0.47
	P for trend	0.006		0.34		0.54	
Artificially sweetened beverage intake	Never or < 1 time/ month	Ref		Ref		Ref	
	1–3 times/month	1.11 (0.94–1.30)	0.21	1.00 (0.85–1.18)	1.00	1.00 (0.85–1.18)	0.94
	1–6 times/week	1.40 (1.23–1.60)	< 0.001	1.18 (1.03–1.34)	0.014	1.18 (1.03–1.35)	0.013
	≥1 time/day	1.83 (1.57- 2.13)	< 0.001	1.43 (1.23–1.67)	< 0.001	1.38 (1.18–1.61)	< 0.001
	P for trend	< 0.001		< 0.001		< 0.001	

* adjusted for sex, age, smoking, alcohol, physical activity, family history of DM, sugar intake, comorbidity, AHEI score, Energy intake, socio economic status and region of origin. IRR: incidence rate ratio; BMI: body mass index; WHR: waist to hip ratio; CI: confidence interval.

Table IV
Sensitivity analysis for association of sweetened beverage intake with risk of type 2 diabetes (excluding wave one cases).

Category		Model 1*		Model 2: Model 1 + BMI		Model 3: Model 2 + WHR	
		Adjusted IRR (95 % CI)	*P*-value	Adjusted IRR (95 % CI)	P-value	Adjusted IRR (95 % CI)	*P* -value
Sugar sweetened beverage intake	Never or < 1 time/ month	Reference		Reference		Reference	
	1–3 times/month	1.15 (0.97–1.36)	0.11	1.14 (0.96–1.35)	0.14	1.13 (0.96–1.34)	0.14
	1–6 times/week	1.23 (1.06–1.44)	0.008	1.14 (0.98–1.34)	0.10	1.13 (0.97–1.33)	0.11
	≥1 time/day	1.36 (1.10–1.68)	0.004	1.21 (0.98–1.50)	0.08	1.17 (0.95–1.45)	0.15
	P for trend	0.001		0.042		0.08	
Artificially sweetened beverage intake	Never or < 1 time/ month	Reference		Reference		Reference	
	1–3 times/month	1.14(0.92–1.42)	0.22	1.03(0.83–1.28)	0.80	1.04(0.83–1.29)	0.73
	1–6 times/week	1.47(1.23–1.75)	< 0.001	1.21(1.01–1.45)	0.035	1.23(1.02–1.47)	0.026
	≥1 time/day	2.07(1.69- 2.54)	< 0.001	1.62(1.32–1.99)	< 0.001	1.58(1.28–1.95)	< 0.001
	P for trend	< 0.001		< 0.001		< 0.001	

* adjusted for sex, age, smoking, alcohol, physical activity, family history of DM, sugar intake, comorbidity, AHEI score, Energy intake, socio economic status and region of origin.

in a similar postprandial insulin response as sucrose [44]. High habitual intake of saccharin and sucralose were reported to disrupt gut microbiome to impair glucose tolerance in healthy subjects over only two weeks [15].

The findings of this study highlight the need for a firm policy intended to curb the adverse health effects of sugar and artificially sweetened beverages in Australia. Our findings support current moves to reduce the consumption of sugary drinks, such as via sugary drink taxation by the WHO [45] Rethink Sugary Drink Australia [46] Australian Medical Association (AMA) [47]and others [48]. However, most policies to date primarily focus on reducing sugary drink intake by introducing taxation, which might encourage the use of ASB, which, according to our findings, might still have a detrimental effect on health. Hence, further studies on the need and benefits of holistic approaches on reducing the intake of both SSB and ASB are needed.

Strengths and limitations

The strengths of this study include being the first Australian prospective study to assess the association of sweetened beverages and the risk of type 2 diabetes, given that the consumption of Australian SSBs may cause greater elevation in glucose and insulin than formulations in USA [18]. All the results reported were after adjustment for many possible confounders. Anthropometric data used were based on measurements rather than self-reported. In addition, to rule out the likelihood of reverse causality, we interpreted appropriate models and sensitivity analysis was conducted which makes our analysis more robust.

Our study also has some limitations. Self-reported dietary data from a FFQ was used, which is known to measure intake with considerable error. The consumption of sweetened beverages in our data might have some limitation in accurately reflecting the recent intake data [49]. Diabetes was self-reported, albeit the participant's nominated doctor validated the diagnosis at the first follow-up. Given the age of the research participants, we considered that all incident cases were type 2 diabetes [21]. We were unable to obtain further information on the specific artificial sweeteners from the questionnaires. Accordingly, it was not possible to draw future conclusions on the type 2 diabetes risk of specific artificial sweeteners from this study, which requires further validation in properly designed future studies. Although the sample size is large, inference from our study was limited since the study population did not include participants from aboriginals and Torres Strait islanders, Asians and other population groups as well, as we do not have data on these populations groups.

Conclusion

Consumption of both sugar and artificially sweetened beverage intake may increase the risk of type 2 diabetes. Results highlight the need for public health measures to reduce the effects of sweetened beverage intake. Although our results corroborate recent efforts to reduce sugar sweetened beverage intake through taxation, this might potentially lead to shift in ASB use. Further studies are warranted to investigate both the causal effects and the underlying mechanisms of sweetened beverage intake on the risk of chronic health outcomes including type 2 diabetes.

Ethics of human subject participation

This study was conducted according to the guidelines laid down in

the Declaration of Helsinki, and all procedures involving research participants were approved by the Cancer Council Victoria Human Research Ethics Committee. Written informed consent was obtained from all participants. The current study received approval from the Monash University Human Research Ethics Committee.

Data sharing

Details on how to access data for the Melbourne Collaborative Cohort Study are available at: htt ps://ww w.cancervic.org.au/research/epidemiology/pedigree.

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Declaration of generative AI and AI-assisted technologies in the writing process

We have not used any AI at all.

CRediT authorship contribution statement

Robel Hussen Kabthymer: Writing – review & editing, Writing – original draft, Visualization, Software, Methodology, Formal analysis, Data curation, Conceptualization. **Tongzhi Wu:** Writing – review & editing, Validation, Software, Methodology. **Sara Beigrezaei:** Writing – review & editing, Validation, Software, Methodology. **Oscar H Franco:** Writing – review & editing, Validation, Software, Methodology. **Allison M Hodge:** Writing – review & editing, Validation, Software, Resources, Methodology, Funding acquisition, Formal analysis, Conceptualization. **Barbora de Courten:** Writing – review & editing, Validation, Supervision, Software, Resources, Project administration, Methodology, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.diabet.2025.101665.

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