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Review

Consumption of Sweet Beverages and Cancer Risk. A Systematic Review and Meta-Analysis of Observational Studies

Fjorida Llaha[1,#], Mercedes Gil-Lespinard[1,#], Pelin Unal[1], Izar de Villasante[1], Jazmín Castañeda[1], and Raul Zamora-Ros [1,*]

[1] Unit of Nutrition and Cancer, Cancer Epidemiology Research Programme, Catalan Institute of Oncology (ICO), Bellvitge Biomedical Research Institute (IDIBELL), Barcelona, Spain. fllaha@idibell.cat (FL), mgill@idibell.cat (MG-L), pelin.uenal@dkfz-heidelberg.de (PU), idevillasante@idibell.cat (I.deV), acastaneda@idibell.cat (JC), rzamora@idibell.cat (RZ-R).

[#] Both authors equally contributed

*Correspondence: rzamora@idibell.cat; Tel.: +34 932607401

Abstract: The consumption of sweet beverages, including sugar-sweetened beverages (SSB), artificially sweetened beverages (ASB) and fruit juices (FJ) is associated with the risk of different cardiometabolic diseases and probably with some tumors as well. We carried out a systematic review and meta-analysis of observational studies aimed at evaluating the association between sweet beverage intake and different types of cancer risk. Suitable papers published up to June 2020 were searched through PubMed, Web of Science and SCOPUS databases, using relevant keywords. Overall, 64 studies were identified for the systematic review, of which 27 were selected for the meta-analysis. This was performed by analyzing the multivariable-adjusted OR, RR or HR of the highest compared with the lowest sweet beverage intake categories. Random effects showed significant positive association between SSBs intake and breast (RR: 1.14, 95% CI: 1.01 – 1.30) and prostate cancer risk (RR: 1.18, 95% CI: 1.10 – 1.27), also between FJs and prostate cancer risk (RR: 1.03, 95% CI: 1.01 – 1.05). Associations between SSBs and colorectal and pancreatic cancer risk, FJs and breast, colorectal and pancreatic cancer risk, ASBs and pancreatic cancer risk tended to be positive but did not reach the statistically significant threshold. This study supports the recommendation to limit the consumption of SSBs and FJs for cancer prevention and proposes to further investigate the potential harmful role of ASBs intake in cancer risk.

Keywords: systematic review; meta-analysis; cohort; case-control; sugar sweetened beverages; artificial sweetened beverages; fruit juice; cancer.

1. Introduction

Consumption of sweet beverages, including sugar sweetened beverages (SSB), artificial sweetened beverages (ASB) and fruit juices (FJ) has increased worldwide in the last decades [1]. Both SSBs and FJs contain high amounts of free sugar. Sugar present in SSBs usually comes from added sucrose or high fructose corn syrup (HFCS). Although FJs are rich in natural antioxidants and micronutrients, they have much higher content of free sugar and lower levels of fiber compared to whole fruits. High sugar consumption may contribute to excessive energy intake, leading to long-term weight gain [2] and higher risk of type 2 diabetes [3] and cardiovascular diseases [4].

It has been demonstrated that obesity and type 2 diabetes are well-known risk factors for cancer [5–7]. Diets high in added sugar (mainly in the form of sucrose and HFCS) result in an excessive intake of fructose and 'empty' calories. This usually causes an increase in weight gain and in adiposity-related metabolic parameters, insulin resistance, bioactivity of steroid hormones, oxidative stress and inflammation, which leads to cancer development and progression [7]. The International Agency for Research on Cancer (IARC) Working Group, reported as strong evidence

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that excess body fat is a major risk factor for many cancers, including esophageal, pancreatic, colorectal, postmenopausal breast , endometrial, renal, ovarian, gallbladder, hepatic and gastric cardia, among others[8].

High sugar intake impairs glucose and insulin tolerance and augments insulin and insulin-like growth factor (IGF) levels. Insulin and IGF are major determinants of proliferation and apoptosis, and may therefore influence carcinogenesis [9]. The beverages high in sugar, including the FJs present a high glycemic index [10], which is also suggested to be linked to cancer [11]. Moreover, both caloric and non-caloric sweet palatable substances have been demonstrated to activate the dopaminergic reward system. This can trigger addictive-like behaviors which might be responsible for increased body fat and obesity [12]. ASBs contain low or non-caloric sweeteners (e.g. aspartame) and have been marked as healthier alternatives to SSBs. However, some studies have suggested that ASB are also deleterious for obesity [13] and type 2 diabetes risk [3]. Moreover, some studies showed that long-term consumption of aspartame, used in many ASBs, might be carcinogenic [14]. Aspartame in liquids can quickly break down into methanol, and the subsequent metabolized formaldehyde is a documented carcinogenic substance [15]. Both caloric and non-caloric sweet palatable substances have been demonstrated to activate the dopaminergic reward system. This can trigger addictive-like behaviors which might be responsible for increased body fat and obesity [12]. Another factor present in numerous sweet beverages is the 4-methylimidazole (4-MEI), used as colorant, classified by IARC as possibly carcinogenic to humans [16].

In light of all this evidence, the increased consumption of sweet beverages and its association with cancer risk has been investigated and reviewed by different studies. A meta-analysis from 2014 studied the association between SSB and ASB consumption and overall and specific cancer site and no links were found [17]. Likewise, a 2019 meta-analysis did not find any significant association between SSB and ASB intake and pancreatic cancer risk [18]. However, the two mentioned studies did not perform a separate analysis of SSBs and ASBs which might have elucidated their particular role on cancer. A pooled analysis from 2012 [19] suggested a modest positive association between SSB intake alone and the risk of pancreatic cancer, while another one from 2010 [20] showed no significant association with colon cancer risk. A qualitative review of longitudinal studies from 2018 [21] reported inconsistent results for SSB and FJ intake and cancer risk. A recent French publication [22] reported a positive association between FJs and overall cancer risk. Their results for breast, colorectal and prostate cancer risk were negative regarding ASB intake. However, another study [23] showed an increased risk for leukemia in the total population and for Non-Hodgkin lymphoma and multiple myeloma in men only.

Evidence suggests that the link between sweet beverages consumption and cancer onset is biologically plausible. However, each type of beverage may have different mechanisms of action and different role on cancer risk. Therefore, our study aimed to investigate these associations, by conducting separate analysis for SSB, ASB and FJ intake and site-specific or overall cancers. We analyzed case-control and cohort studies and performed a meta-analysis when feasible. Through this study we intend to update and develop a better understanding of the association between the consumption of sweet beverages and cancer risk, a disease that caused 9.6 million deaths in 2018 and is projected to nearly double these figures by 2040 [24].

2. Materials and Methods

2.1. Search Method for Identification of Studies

This study was conducted according to the Preferred Reporting Items for Systematic Reviews Meta-Analysis (PRISMA) guidelines. To identify the suitable articles, we searched in PubMed, Web of Science and SCOPUS databases up to June 31st, 2020, using the following keywords: (((((("soft drinks"[All Fields] OR "sugary drinks"[All Fields]) OR "sugary beverages"[All Fields]) OR "fruit juice"[All Fields]) OR "sugar-sweetened beverages"[MeSH Terms]) OR "artificially sweetened beverages"[MeSH Terms]) AND ((("neoplasms"[MeSH Terms] OR "neoplasm"[All Fields]) OR

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"cancer"[All Fields]) OR "cancers"[All Fields]) OR "tumor"[All Fields]). We also applied search filters by article type (excluding books, reviews, systematic reviews and meta-analysis) and by species (including only humans). Moreover, reference lists of included manuscripts and relevant reviews were examined for any possible unidentified study. Search process was limited to English and Spanish languages.

2.2. Eligibility Criteria and Data Extraction

Eligible cohort and case-control studies were selected if they met the following criteria: 1) included adult participants free of cancer (if prospective) or with no history of previous cancer (if case-control) at recruitment, except for non-melanoma skin cancer, 2) overall or site-specific cancer incidence as an outcome, 3) estimated and reported hazard ratio (HR), risk ratio (RR) or odds ratio (OR) with 95% confidence interval (CI) for the link between any type of sweet beverage intake and any type of cancer incidence. The exclusion criteria were: 1) participants with previous cancer history or currently undergoing cancer treatment, 2) cancer survival and cancer mortality as an outcome, 3) duplicated studies. The following data were extracted: first author's name, publication year, study name, country, age and sex of the participants, study sample size, number of cases and controls, follow-up duration, cancer site, type of exposure and amount of intake, dietary assessment methods, adjust cofounders and HR/RR/OR with 95% CI for the larger degree of adjustment. When time-varying results were reported, those related to baseline data were extracted.

Three review authors independently performed the literature search, study selection and data extraction (FL, MG-L and PU). Disagreements were discussed between all authors until a consensus was reached.

2.3. Quality Assessment of Included Studies

Two independent reviews (FL and MG-L) examined the methodological quality of the individual studies by using the Risk Of Bias In Non-randomized Studies - of Exposures (ROBINS-E) [25] tool for cohort studies and the Newcastle-Ottawa Scale (NOS) [26] tool adapted for case–control studies. ROBINS-E evaluates the risk of bias by assessing different domains: confounding, selection of participants into the study, classification of exposures, departures from intended exposures, missing data, measurement of outcomes and selection of the reported result. Low, moderate or serious risk of bias was established in each study considering all domains. The NOS assesses the selection of groups (0–4 stars), adequacy of comparability between groups (adjustment for confounders) (0–2 stars) and ascertainment of the exposure of interest for case–control studies (0–3 stars). For selection domain, we considered studies with 0-1, 2-3 and 4 stars as serious bias risk, moderate and high-quality, respectively. For comparability between groups, we considered those with 0, 1, and 2 as serious bias risk, moderate and low, respectively. For ascertainment of exposure, we considered those 0, 1-2, and 3 as serious bias risk, moderate and low, respectively. In both tools, when data was not enough for judgment, the domain was classified as "no information".

2.4. Data Synthesis and Statistical Analysis

The first obstacle that we had to overcome is the lack of a unique definition of beverages and the use of a variety of terms. In this text, the following group terms are used to generalize these products: SSB for sugar-sweetened beverages, (regular soft drinks/sodas, and non-diet soft drinks/sodas); ASB for artificial-sweetened beverages (low and non-caloric soft drinks/sodas, diet

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soft drinks/sodas); FJ for fruit juices. In addition, two other terms are used: SB for sweetened beverages that includes SSB together with ASB; SFJ for high-sugar (added or natural) beverages that includes SSB together with FJ. The quantity of each beverage was provided mostly as categories of frequency of consumption, either in amount (ml or gram/day) or serving sizes (cans for SSB, ASB, glasses for FJ/day). In order to unify the data, we converted the categories to ml/day, based on the study-specific serving size for each beverage. When the serving size was not reported, we referred to the national data of each study. Thus, we considered one can equal to 330 ml and one glass equal to 200 ml for European countries [27], one can equal to 360 ml and one glass equal to 240 ml for the United States [28], and one can equal to 375 ml for Australia [29]. One US study [30] expressed consumption as grams of sugar and we weighed up an average of 10.5 g of sugar per 100 ml of SSB and an average of 9.6 g of sugar per 100 ml of FJ. This was calculated based on the sugar content of different commercially available products of popular brands [31].

Prior to the analysis, the selected studies were classified by outcome (cancer incidence by site) and exposure (SB, SSB, ASB, FJ and SFJ). Data were synthetized in a narrative manner and a meta-analysis was performed only if at least three studies reported data for the same exposure and outcome. In the meta-analysis, results for the total number of participants were considered. Subgroups of participants were considered only when the article did not report the analysis for the total number of participants. In the same manner, if studies reported data for specific beverages (e.g. caffeinated and non-caffeinated SSB), results for the total beverage group (e.g. total SSBs) were considered. Even thought, we extracted data for fruit and vegetables juices together, for the meta-analysis we only considered the studies that indicated FJs as the predominant beverage consumed. The meta-analysis was performed by pooling the multivariable-adjusted RR/HR/OR of the highest category of the exposure versus the lowest one. The random-effect model was considered because of the high variability in study design and because of the low number of studies included in the meta-analysis, although we have also performed the fix-effect model. A second analysis was performed excluding outliers when their 95% confidence interval lies ouside the 95% confidence interval of the pooled effect. The I^2, Tau^2 test and the prediction intervals were used to evaluate the heterogeneity across studies. The statistical analysis was performed with the Metafor package [32] of the R software, version 4.0.1.

3. Results

3.1. Literature Search and Study Characteristics

The study selection process according to PRISMA guidelines is reported in Figure 1. In total, 869 potential publications were identified from the databases (PubMed, Web of Science and SCOPUS) and other sources. After removing duplicates, 596 articles were selected, from which 435 were excluded based on titles and 26 on abstracts. Of 135 eligible articles, 71 were excluded due to the following reasons: 59 did not report risk index for sweet beverages and cancer incidence, 3 full-texts were not available, 7 considered other outcomes, 1 case-control study included controls with cancer at recruitment and 1 publication was not in English or Spanish. Finally, 64 studies were included in the systematic review, 27 cohort [22,23,27,30,33–55] and 37 case-control studies [56–92]. Of these, 27 studies were used in the meta-analyses.

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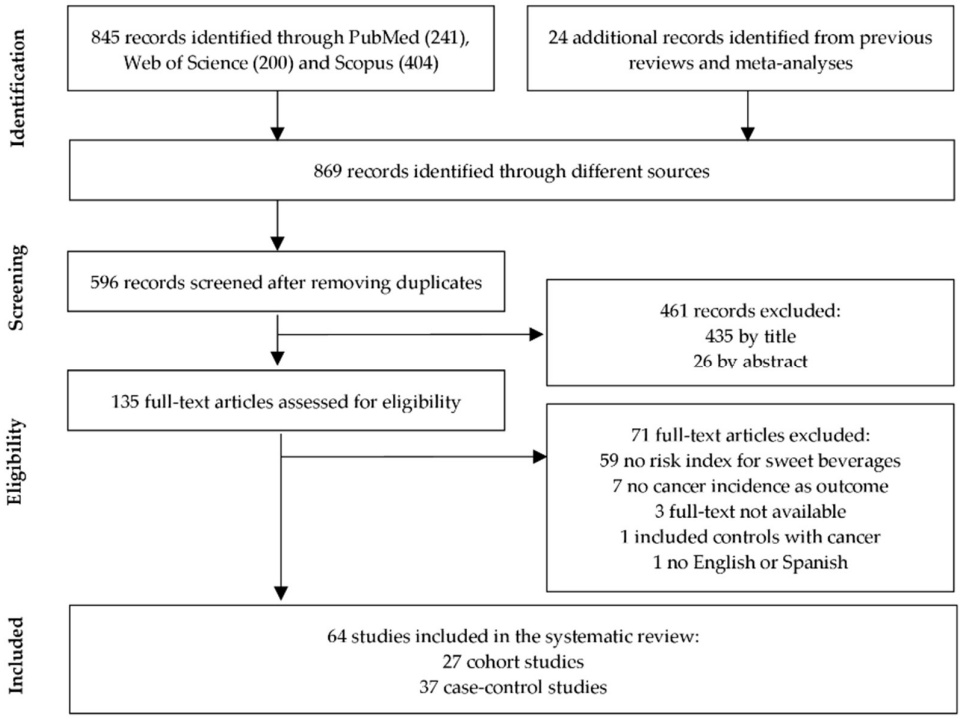

Figure 1. Prisma diagram.

Among the included studies in this review, 29 were performed in the US, 17 in Europe, 6 in Asia, 5 in Canada, 3 in Australia, 2 in Latin-America, 1 in Egypt and 1 was multinational (Italy, Spain, Poland, Northern Ireland, India, Cuba, Canada, Australia and Sudan). They usually included male and female participants with an age range from 18 to 97 years. The 27 cohort studies were published between 2003 and 2020 and enrolled 4,458,056 participants in total, of which 30,646 developed cancer. Mean duration of the follow-up in cohort studies varied from 2 to 20 years. The 37 case-control studies were published between 1985 and 2019. In total, they enrolled 20,827 cancer cases and 34,315 controls. Most of the controls were selected from the general population.

Sweet beverage consumption in both cohort and case-control studies was expressed as categorical or continuous variables. Exposure assessment was collected using food frequency questionnaires (FFQ), 24-hour dietary recalls (24-H DR), dietary questionnaires (DQ), interviews or surveys. Among all the studies, 37 types of cancer were considered as an outcome and 4 cohorts reported data for overall cancer risk, including different types of cancers [22,49,51,53]. In most of the studies, the outcome was confirmed by a medical diagnosis. Overall characteristics of the included studies are summarized in Table 1.

3.2. Sweet Beverages and Risk of Breast Cancer

The results of the meta-analysis for the random-effect model are summarized in Table 2. Nine publications reported data on breast cancer, four case-control [56,57,68,79] and five cohort studies [22,50–52,54]. In the random-effect meta-analysis with six publications, including 4 cohort studies [22,51,52,54] and 2 case-controls [56,79], a significant positive association between the highest vs. the lowest group of SSB consumers and breast cancer risk (RR: 1.14, 95% CI: 1.0 – 1.3) was observed. No associations were found for FJ intake (Table 2). Marzbani et al. [57] reported a positive association with SBs (OR: 2.8, 95% CI: 1.9 – 4.3), but no associations were found for ASBs.

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Table 1. Overall characteristic of the included studies.

Breast cancer (breast, pre and postmenopausal)											
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake+**	**HR/RR/OR (95% CI)**	**Adjustments**
Chandran et al, 2006 [56]	US, WCHS	Breast	PB case-control	3148	1558	20-75	F (100)	125-item FFQ	SSB: ≥152 vs <152 ml/d	OR: 0.97 (0.74-1.27) (AA) OR:1.31 (0.91-1.89) (EA)	Age, ethnicity, country, education, age at menarche, menopause and first birth, MS, parity, BF status, history of benign breast disease, family history of BC, HRT, OC use, BMI and study site.
		Pre-M			797				SSB: ≥152 vs <152 ml/d	OR: 1.17 (0.79-1.74) (AA) OR:0.95 (0.58-1.56) (EA)	
		Post-M			761				SSB: ≥152 vs <152 ml/d	OR: 0.76 (0.51-1.12) (AA) OR:2.05 (1.13-3.7) (EA)	
Chazelas et al, 2019 [22]	France, NNS	Breast	Cohort	101257 5.1 (median)	693	42.2/14.4	F (78)	24H-DR	SFJ: >123 vs <38.1 ml/d (cut-off) SFJ: increase by 100 ml/d SSB: >57.1 vs <13.6 ml/d (cut-off) SSB: increase by 100 ml/d ASB: >11.6 vs <4.6 ml/d (cut-off) ASB: increase by 10 ml/d FJ: >81.9 vs <17.0 ml/d (cut-off) FJ: increase by 100 ml/d SFJ: >123 vs <38.1 ml/d (cut-off) SFJ: increase by 100 ml/d	HR: 1.37 (1.08 -1.73) HR: 1.22 (1.07- 1.39) HR: 1.10 (0.87-1.39) HR: 1.23 (1.03-1.48) HR: 1.33 (0.98-1.75) HR: 0.97 (0.86-1.09) HR: 1.13 (0.91-1.39) HR: 1.15 (0.97-1.35) HR: 1.28 (1.09 -1.83) HR: 1.26 (1.04 - 1.51)	Smoking, education, PA, BMI, height.
		Pre-M			283				SSB: >57.1 vs <13.6 ml/d (cut-off) SSB: increase by 100 ml/d ASB: >11.6 vs <4.6 ml/d (cut-off) ASB: increase by 10 ml/d FJ: >81.9 vs <17.0 ml/d (cut-off) FJ: increase by 100 ml/d SFJ: >123 vs <38.1 ml/d (cut-off)	HR: 1.68 (1.45-1.74) HR: 1.34 (1.15-1.70) HR: 1.23 (0.52-2.53) HR: 0.95 (0.81-1.13) HR: 0.98 (0.67-1.43) HR: 1.10 (0.85-1.41) HR: 1.44 (1.05 -1.99)	

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									SFJ: increase by 100 ml/d SSB: >57.1 vs <13.6 ml/d (cut-off) SSB: increase by 100 ml/d ASB: >11.6 vs <4.6 ml/d (cut-off) ASB: increase by 10 ml/d FJ: >81.9 vs <17.0 ml/d (cut-off) FJ: increase by 100 ml/d	HR: 1.19 (0.98-1.44) HR: 0.99 (0.72-1.39) HR: 1.08 (0.79-1.47) HR: 1.10 (0.55-2.12) HR: 1.01 (0.86-1.18) HR: 1.24 (0.95-1.61) HR: 1.19 (0.96-1.48)	
		Post-M			410						
Hirvonen et al, 2006 [50]	France, SUVIMAX	Breast	Cohort	43966.6	95	35-60	F (100)	24H-DR	FJ: >150 ml/d vs none	RR: 1.29 (0.80-2.09)	Age, smoking, nº of children, OC use, family history of BC and MS.
Makarem et al, 2018 [51]	US	Breast	Cohort	31844	128	54.3	F (53)	FFQ	SFJ: >324 vs <135 ml/d (cut-off) SSB: >51.4 ml/d vs none FJ: >180 vs <38.6 ml/d (cut-off)	HR: 1.00 (0.65-1.57) HR: 1.04 (0.64-1.71) HR: 1.03 (0.67-1.62)	Age, smoking, BMI, EI, alcohol, PA, education, MS, nº of live births, WC, DM and CVD, antioxidant use, energy from fat and diet soda intake.
Marzbani et al, 2019 [57]	Iran	Breast	HB case-control	620	212	40.2	F (100)	11-item healthcare form	SB[7]: favourable intake vs ≤1 time/month	OR: 2.8 (1.9-4.3)	Age, education, BMI
McLaughlin et al, 1992 [68]	US	Breast	PB case-control	3234	1617	56.7	F (100)	SQ-interview	SB[2]: ever vs never	OR: 1.08 (0.92-1.26)	Age, alcohol, country, race, MS, age at first live birth, diagnosis of benign cancers, family history of BC.
Potischman et al, 2002 [79]	US	Breast	PB case-control	2019	568	20-44	F (100)	100-item FFQ	SSB: ≥ 320 ml/d vs none	OR: 1.09 (0.8-1.5)	Age at diagnosis, study site, race, education, alcohol consumption, years of OC use, smoking, BMI and EI.
Romanos-Nanclares et al, 2019 [52]	Spain	Breast	Cohort	107132	100	33.0 (median)	F (100)	FFQ	SSB: >47.1 vs <11 ml/d	HR: 1.36 (0.74-2.50)	Age, height, family history of BC, smoking, PA, BMI, age at menarche and menopause, MS, HRT, nº of pregnancies >6 month and before 30 years old, months of BF, alcohol, education, DM, GI, EI, U-P food and coffee consumption, Med-diet adherence.
		Pre-M			57				SSB: ≥11 ml/d vs none	HR: 1.16 (0.66-2.07)	
		Post-M			43				SSB: >47.1 vs <11 ml/d	HR: 2.12 (1.01-4.41)	

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Hodge et al, 2018 [53]	Australia, MCCS	Post-M	Cohort	35593 19	946	54.6	F (100)	121-item FFQ	SSB: ≥200 vs <6.7 ml/d ASB: ≥200 vs <6.7 ml/d	HR: 1.11 (0.85-1.45) HR: 0.95 (0.73-1.25)	Socio economic indexes, country of birth, alcohol intake, smoking, PA, Med-diet score, sex. ASB also for SSB consumption and WC.
Nomura et al, 2016 [54]	US, BWHS	Breast Pre-M Post-M	Cohort	49103 13.8	1827 678 826	21-69	F (100)	FFQ	SSB: ≥250 ml/d vs none SSB: ≥250 ml/d vs none SSB: ≥250 ml/d vs none	HR: 0.71 (0.50-1.02) HR: 1.72 (0.91-3.23) HR: 1.11 (0.77-1.61)	Age, geographic region of residence, EI, smoking, family history of BC, education, MS, OC use, parity, HRT, BMI, alcohol, PA and sedentary time.
Colorectal and rectal cancer											
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake⁺**	**HR/RR/OR (95% CI)**	**Adjustments**
Bener et al, 2010 [87]	Qatar	Colorectal	HB case-control	428	146	53.4	M (58)	DQ	SB: ≥330 vs ≤47.1 ml/d	OR: 1.62 (1.19-2.17)	Not reported
Chazelas et al, 2019 [22]	France	Colorectal	Cohort	101257 5.1 (median)	166	42.2 (14.4)	F (78)	24H-DR	SFJ: >123 vs <38.1 ml/d (F); >141.7 vs <46.1 ml/d (M) (cut-off) increase by 100 ml/d SSB: >57.1 vs <13.6 ml/d (F); >65.5 vs < 14.0 ml/d (M) (cut-off) increase by 100 ml/d ASB: >11.6 vs <4.6 ml/d (F); >7.9 vs < 2.7 ml/d (M) (cut-off) increase by 10 ml/d FJ: >81.9 vs <17.0 ml/d (F); >97.8 vs <19.9 ml/d (M) (cut-off)	 HR: 1.07 (0.63-1.80) HR: 1.10 (0.84-1.46) HR: 1.01 (0.59-1.71) HR: 1.11 (0.72-1.71) HR: 0.80 (0.44-1.46) HR: 1.02 (0.94-1.10) HR: 1.19 (0.78-1.82)	Smoking, education, PA, BMI, height.

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									increase by 100 ml/d	HR: 1.05 (0.75-1.46)	
Hodge et al, 2018 [53]	Australia, MCCS	Colorectal	Cohort	35593 19	1055	54.6	M/F	121-item FFQ	SSB: ≥200 vs <6.7 ml/d ASB: ≥200 vs <6.7 ml/d	HR: 1.28 (1.04-1.57) HR: 0.79 (0.60-1.06)	Socio economic indexes, country, alcohol, smoking, PA, Med-diet score, sex. ASB also for SSB consumption and WC.
Makarem et al, 2018 [51]	US	Colorectal	Cohort	3184 4	68	54.3	F (53)	FFQ	SFJ: >362.6 vs <154.3 ml/d (cut-off) SSB:>180 vs <25.7 ml/d (cut-off) FJ: >180 vs < 48.9 ml/d (cut-off)	HR: 1.39 (0.68-2.82) HR: 0.96 (0.51-1.82) HR: 1.66 (0.88-3.12)	Age, smoking, BMI, EI, alcohol, PA, education, MS, nº of live births, WC, DM and CVD, antioxidant use, energy from fat and diet soda intake.
Mahfouz et al, 2014 [88]	Egypt	Colorectal	HB case-control	450 1	150	<20- >60	F (52)	DQ	SB: daily vs not daily FJ: daily vs not daily	OR: 4.6 (1.9-11.01) OR: 0.18 (0.09-0.36)	Not reported
Pacheco et al, 2019 [55]	US	Colorectal	Cohort	99798 20.1 (median)	1318	52.0 (13.5)	F (100)	FFQ	SSB: ≥60 ml/d vs never/rare	HR: 1.14 (0.86-1.53)	Age, BMI, EI, smoking, alcohol, family history of CR polyps, multivitamin use, HT.
Tayyem et al, 2018 [89]	Jordan	Colorectal	HB case-control	501 2	220	52	F (51)	Q-DQ	SB: daily vs rarely OJ: daily vs rarely	OR: 1.39 (0.73-2.63) OR: 1.07 (0.45-2.55)	Age, sex, work status, income, PA, marital status, EI, education, other diseases and history of CR cancer.
Theodoratou et al, 2014 [90]	Scotland	Colorectal	PB case-control	4838 7.0	2062	64.3	M/F	FFQ	SSB: increase by 330 ml/d FJ: increase by 200 ml/d	OR: 1.12 (1.05-1.19) OR: 1.19 (1.11-1.27)	Age, sex, BMI, PA, family history of CR cancer, EI, NSAIDs, eggs, FJ, SSB, white fish, coffee and magnesium intake.
Murtaugh et al, 2004 [91]	US	Rectal	PB case-control	2157 4	952	30-79	M (57)	Interview	SSB: yes vs no (M) SSB: yes vs no (F) ASB: yes vs no (M) ASB: yes vs no (F) J: >449 vs ≤58.3 ml/d (M); J: >596.6 vs ≤44.6 ml/d (F)	OR: 1.00 (0.80-1.26) OR: 0.96 (0.73-1.27) OR: 1.28 (0.98-1.68) OR: 0.90 (0.67-1.22) OR: 0.92 (0.63-1.34) OR: 1.56 (1.00-2.41)	Age, PA, EI, dietary fibre and calcium intake.

Esophageal cancers (esophagus-gastric junction, esophageal adenocarcinoma, squamous cell carcinoma)

Source	Country, Study name	Cancer type	Study design	Population Follow-up (years)	Cases	Age (mean/SD or range)	Sex (%)	Dietary assessment method	Type and amount of beverages intake+	HR/RR/OR (95% CI)	Adjustments

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Ibiebele et al, 2008 [92]	Australia	AEGJ	PB case-control	23414	325	18-79	M (71)	FF	SB[7]: ≥375 ml/d vs none SSB[7]: yes vs no ASB[7]: yes vs no	OR: 1.07 (0.67-1.73) OR: 0.63 (0.43-0.92) OR: 0.77 (0.46-1.29)	Age, sex, BMI, EI, alcohol, smoking, education, heartburn and acid reflux symptoms.
		EAC			294				SB[7]: ≥375 ml/d vs none SSB[7]: yes vs no ASB[7]: yes vs no	OR: 0.94 (0.53-1.66) OR: 1.20 (0.79-1.81) OR: 0.71 (0.37-1.37)	
		SCC			238				SB[7]: ≥375 ml/d vs none SSB[7]: yes vs no ASB[7]: yes vs no	OR: 0.40 (0.20-0.78) OR: 0.70 (0.47-1.03) OR: 0.46 (0.25-0.85)	
Mayne et al, 2006 [58]	US	EAC	PB case-control	1782	228	65 Q1, 59.3 Q4	M (78 Q1, 82 Q4)	Proxy and self-interviewed	SSB[7]: ≥355 vs 10.7 ml/d	OR: 0.47 (0.29-0.76)	Age, sex, centre, race, proxy interview status, BMI, EI, alcohol and meat intake, cigarettes/day, education, income and frequency of reflux symptoms.
		SCC			206				SSB[7]: ≥355 vs 10.7 ml/d	OR: 0.85 (0.48-1.52)	
Ren et al, 2010 [33]	US, NIH-AARP-DHS	EAC	Cohort	481563 2	305	50-71	M (59)	124-item FFQ	SB: ≥355 vs ≤355 ml/d	HR: 1.11 (0.66-1.85)	Age, sex, smoking, alcohol, EI, BMI, education, ethnicity, PA, and daily intake of fruit, vegetables, red meat and white meat.
		SCC			123				SB: ≥355 vs ≤355 ml/d	HR: 0.85 (0.46-1.56)	
Stomach cancers (gastric cardia, gastric non-cardia)											
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake⁺**	**HR/RR/OR (95% CI)**	**Adjustments**
Hodge et al, 2018 [53]	Australia, MCCS	Gastric cardia	Cohort	35593 19	165	54.6	M/F	121-item FFQ	SSB: ≥200 vs <6.7 ml/d ASB: ≥200 vs 6.7 ml/d	HR: 1.17 (0.73-1.89) HR: 1.03 (0.53-1.98)	Socio economic indexes, country, alcohol, smoking, PA, Med-diet score and sex. ASB also for SSB consumption and WC.
Mayne et al, 2006 [58]	US	Gastric cardia	PB case-control	1782	255	65 Q1, 59.3 Q4	M (78 Q1, 82 Q4)	Proxy and self-interviewed	SSB[7]: ≥355 vs <10.7 ml/d	OR: 0.74 (0.46-1.16)	Age, sex, centre, race, proxy interview status, BMI, EI, alcohol and meat intake cigarettes/day, education, incomes and frequency of reflux symptoms.
		Gastric non-cardia			352				SSB[7]: ≥355 vs <10.7 ml/d	OR: 0.65 (0.43-0.98)	
Ren et al, 2010 [33]	US, NIH-AARP-	Gastric cardia	Cohort	481563 2	231	50-71	M (59)	124-item FFQ	SB: ≤355 vs ≥355 ml/d	HR: 0.89 (0.55-1.45)	Age, sex, smoking, alcohol, EI, BMI, education, ethnicity, PA and daily intake of

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	DHS	Gastric non-cardia			224			SB: ≥355 vs ≤355 ml/d	HR: 0.75 (0.45-1.24)	fruit, vegetables, and white meat.
Pancreatic cancer										
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake⁺**	**HR/RR/OR (95% CI)**	**Adjustments**
Bao et al, 2008 [41]	US, NIH-AARP-DHS	Pancreatic	Cohort	487922 7.2	1258	50-71	F (41)	124-item FFQ	SB: 816.9 ml/d (median) vs none SSB: 512.8 ml/d (median) vs none ASB: 816.9 ml/d (median) vs none	RR: 1.07 (0.86-1.33) RR: 1.01 (0.77-1.31) RR: 1.11 (0.86-1.44)	Age, sex, race, education, BMI, alcohol, smoking, PA, EI, foliate intake. SSB and ASB were mutually adjusted.
Chan et al, 2009 [75]	US, SFB	Pancreatic	PB case-control	2233	532	21-85	M (53)	131-item FFQ	SB: ≥355 ml/d vs none SB[7]: ≥355 ml/d vs none SSB[7]: ≥355 ml/d vs none ASB[7]: ≥355 ml/d vs none SSB[4]: ≥355 ml/d vs none	OR: 1.0 (0.7-1.3) OR: 1.1 (0.8-1.5) OR: 0.9 (0.6-1.3) OR: 1.5 (1.1-2.1) OR: 1.0 (0.6-1.8)	Age, sex, EI, BMI, race, education, smoking, history of DM, PA, red and white meat, fruit and vegetables, eggs, dairy, whole and refine grained, sweets, SSB and ASB were mutually adjusted.
Gallus et al, 2011 [76]	Italy	Pancreatic	HB case-control	978 7	326	63 (median)	M (53)	FFQ	SB[7]: ≥150 vs <150 ml/d	OR: 1.02 (0.72-1.44)	Age, sex, study centre, education, BMI, smoking, alcohol, EI, family history of pancreatic cancer and DM.
Gold et al, 1985 [77]	US	Pancreatic	HB, PB case-control	676	274	66.1	F (53)	Interview	ASB: ever vs never	OR: 0.66 (0.38-1.2)	Religion, occupation, smoking and alcohol.
Larsson et al, 2006 [40]	Sweden, SMC, COSM	Pancreatic	Cohort	77797 7.2	131	60.8	F (45)	FFQ	SB: ≥500 ml/d vs none	HR: 1.93 (1.18-3.14)	Age, sex, education, smoking, BMI, EI.
Lyon et al, 1992 [78]	US	Pancreatic	PB case-control	512	149	40-79	M/F	DQ	SB (caff): ever vs never	OR: 1.31 (0.89-1.94)	Unadjusted.
Mack et al, 1986 [80]	US	Pancreatic	PB case-control	980	490	18-65	M (58)	Proxy and direct Interview	SB[7]: ≥1650 vs <1320 ml/d	RR: 2.6 (0.9-7.4)	Not reported
Mueller et al, 2010 [42]	China and Singapore,	Pancreatic	Cohort	60524 14	140	56.5	F (56)	FFQ	SB: ≥ 67.7 ml/d vs none J[5]: ≥ 67.7 ml/d vs none	HR: 1.87 (1.10-3.15) HR: 1.31 (0.74-2.30)	Age, sex, smoking, BMI, alcohol, EI, PA, DM, education, added sugar and candy. SB

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	SCHS										and J were mutually adjusted.
Nothlings et al, 2007 [43]	US	Pancreatic	Cohort	1621508	434	59.8	F (55)	FFQ	SSB: ≥ 151.4 ml/2000 kcal/d vs none FJ: ≥120 vs < 9.4 ml/2000 kcal/d	RR: 1.07 (0.82,1.41) RR: 1.08 (0.83,1.41)	Age, sex, smoking, BMI, EI, time on study, race, family history of pancreatic cancer, intake of red and processed meat.
Navarrete-Muñoz et al, 2016 [44]	10 European countries†, EPIC	Pancreatic	Cohort	47720611.4	865	51	F (70)	DQ-country specific	SB: >196.4 vs 0.1–13.1 ml/d SB: increase by 100 ml/d SSB: >121.4 vs 0.1-4.5 ml/d SSB: increase by 100 ml/d ASB: >92.2 vs 0.1-2.0 ml/d ASB: increase by 10 ml/d FJ[6]: >123.1 vs 0.1-8.3 ml/d FJ[6]: increase by 100 ml/d	HR: 0.90 (0.68-1.19) HR: 1.02 (0.98-1.06) HR: 0.90 (0.65-1.25) HR: 1.02 (0.97-1.08) HR: 0.99 (0.61-1.60) HR: 1.02 (0.96-1.08) HR: 0.74 (0.57-0.97) HR: 0.91 (0.84-0.98)	Age, sex, smoking, BMI, alcohol, EI, study centre, PA, DM. FJ and SB were mutually adjusted.
Schernhammer et al, 2005 [45]	US, HPFS, NHS	Pancreatic	Cohort	136587 14 HPFS, 20 NHS	379	53.7	F (65)	FFQ	SSB: <143.6 vs > 11.2 ml/d ASB: <143.6 vs > 11.2 ml/d	RR: 1.13 (0.81-1.58) RR: 1.02 (0.79-1.32)	Age, sex, smoking, BMI, follow-up cycle, PA, DM and other soft drink intake.
Genitourinary cancers (prostate, renal cell, urinary bladder and urothelial cell)											
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake[+]**	**HR/RR/OR (95% CI)**	**Adjustments**
Bruemmer et al, 1997 [59]	US	Bladder	PB case-control	620	215	45-65	M (62)	Interview	SSB: >240 vs < 8 ml/d ASB: >240 < 8 ml/d	OR: 0.4 (0.2-1.1) (M) OR: 5.7 (1.2-26.9) (F) OR: 1.6 (0.7-3.6) (M) OR: 2.3 (0.8-6.3) (F)	Age, country, smoking.
De Stefani et al, 2007 [60]	Uruguay	Bladder	HB case-control	756	255	30-89	M (88)	64-item FFQ	SB: ≥142 vs <142 ml/d	OR: 1.1 (0.7-1.7)	Age, sex, residence, education, familiar history of UBC, BMI, occupation, smoking, intake of mate, coffee, tea, milk.
Hemelt et al, 2010 [61]	China	Bladder	HB case-control	792 3	400	65.8	M (79)	DQ	SB: consumers vs none FJ: daily vs none	OR: 2.01 (1.10-3.68) OR: 0.66 (0.26-1.66)	Age, sex, smoking, frequency and duration of smoking.
Radosavljević et	Serbia	Bladder	HB	260	130	64.9	M (79)	101-item	SB: >15.7 ml/d (mean) vs none	OR: 4.73 (2.72-8.18)	Smoking

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al, 2003 [62]			case-control					FFQ	FJ: >11.6 ml/d (mean) vs none	OR: 0.30 (0.18-0.50)	
Turati et al, 2015 [63]	Italy	Bladder	HB case-control	1355	665	67 (median)	M (76)	DQ	SB²: ≥47 ml/d vs none	OR: 1.04 (0.73-1.49)	Age, sex, study centre, year of interview, smoking, education, alcohol, BMI, family history of UBC and cystitis.
Wang, 2013 [64]	US	Bladder	HB case-control	2306	1007	64.4	M (78)	FFQ	SB: ≥255.6 ml/d vs none SSB: ≥126 ml/d vs none ASB: ≥309.6 ml/d vs none	OR: 1.34 (1.05-1.70) OR: 1.27 (1.02-1.58) OR: 1.06 (0.85-1.32)	Age, sex, ethnicity, EI, smoking.
Chazelas et al, 2019 [22]	France	Prostate	Cohort	101257 5.1 (median)	291	42.2/4.4	M (100)	24H-DR	SFJ: >141.7 vs <46.1 ml/d (cut-off) SFJ: increase by 100 ml/d SSB: >65.5 vs <14.0 ml/d (cut-off) SSB: increase by 100 ml/d ASB: >7.9 vs <2.7 ml/d (cut-off) ASB: increase by 10 ml/d FJ: >97.8 vs <19.9 ml/d (cut-off) FJ: increase by 100 ml/d	HR: 1.39 (0.96-2.02) HR: 1.10 (0.92-1.31) HR: 1.19 (0.83-1.72) HR: 1.24 (0.95-1.62) HR: 1.33 (1.01-1.75) HR: 0.57 (0.24-1.34) HR: 1.04 (0.76-1.42) HR: 0.97 (0.79-1.2)	Smoking, education, PA, BMI, height.
Drake et al, 2012 [34]	Sweden, MDC	Prostate	Cohort	8128 14.9	817	45-73	M (100)	168-item FFQ, 7-d menu book Interview	SSB: 297.8 ml/d (median) vs none FJ: 200 ml/d (median) vs none	HR: 1.13 (0.92-1.38) HR: 0.99 (0.81-1.22)	Age, year of study entry, time of data collection, EI, height, WC, PA, smoking, education, birth in Sweden, alcohol, calcium and selenium intake, risk by death from all causes except PC.
Ellison et al, 2000 [35]	Canada, NCSS	Prostate	Cohort	3400 23	201	50-84	M (100)	FFQ	SB²: ≥ 100 ml/d vs none SB²: ≥ any vs none	RR: 1.29 (0.74-2.26) RR:1.09 (0.78-1.35)	Age, alcohol, smoking, BMI, fibre, EI.
Hodge et al, 2018 [53]	Australia, MCCS	Prostate	Cohort	35593 19	433	54.6	M (100)	121-item FFQ	SSB: ≥200 vs <6.7 ml/d ASB: ≥200 vs <6.7 ml/d	HR: 1.08 (0.78-1.50) HR: 0.81 (0.49-1.33)	Socio economic indexes, country of birth, alcohol, smoking, PA, Med-diet score, ASB also for SSB consumption and WC.
Jain et al, 1998 [65]	Canada	Prostate	PB case-control	1253	617	69.8	M (100)	Q-DH	SB²: >200 ml/d vs none	OR: 0.79 (0.53–1.17)	Age, EI
Makarem et al, 2018 [51]	US	Prostate	Cohort	3184 4	157	54.3	M (100)	FFQ	SFJ: >401 vs <212.1 ml/d (cut-off) SSB: >180 vs <25.7 ml/d (cut-off)	HR: 1.06 (1.03-1.09) HR: 1.38 (0.80-2.38)	Age, smoking, BMI, EI, alcohol, PA, education, WC, DM and CVD, antioxidant

									FJ: >180 vs <48.9 ml/d (cut-off)	HR: 1.03 (1.01-1.06)	use, energy from fat and diet soda intake.
Miles et al, 2018 [30]	US	Prostate	Cohort	227209	1996	65.6 (5.9)	M (100)	FFQ	SSB: >183 vs <6 ml/d (cut-off) FJ: >190 vs <24 ml/d (cut-off)	HR: 1.21 (1.06-1.39) HR: 1.07 (0.94-1.22)	Age, sex, smoking, BMI, EI, DM, education, race, family history of PC, PSA screens.
Sharpe et al, 2002 [66]	Canada	Prostate	PB case-control	875	399	61.5	M (100)	Interviews or DQ	SB[7]: daily drank vs never drank weekly	OR: 1.0 (0.7–1.4)	Age, ethnicity, socio-economic status, BMI, cumulative cigarette smoking, alcohol.
Hodge et al, 2018 [53]	Australia, MCCS	Renal cell	Cohort	3559319	146	54.6	M/F	121-item FFQ	SSB: ≥200 vs <6.7 ml/d ASB: ≥200 vs <6.7 ml/d	HR: 1.48 (0.87-2.53) HR: 0.92 (0.46-1.84)	Socio economic indexes, country of birth, alcohol, smoking, PA, Med-diet score and sex. ASB also for SSB consumption, WC
Hu et al, 2009 [67]	Canada	Renal cell	PB case-control	6177	1138	20-80	M (51)	FFQ	SB: >230 ml/d vs none SB: increase by 230 md J: >236 vs ≤23 ml/d J: increase by 118 ml/d	OR: 1.26 (0.96–1.67) OR: 1.05 (0.97–1.13) OR: 1.53 (1.18–1.99) OR: 1.08 (1.04–1.13)	10-year age groups, province, education, BMI, sex, EI, smoking, intake of alcohol meat, vegetables and fruits.
Lee et al, 2006 [36]	US	Renal cell	Cohort	13658714 HPFS20 NHS	248	53.7	F (65)	FFQ	SB: ≥670 vs <47.9 ml/d SSB: increase by 335 ml/d ASB: increase by 335 ml/d FJ: increase by 335 ml/d	RR: 1.03 (0.64-1.68) RR: 0.95 (0.69-1.31) RR: 0.97 (0.82-1.15) RR: 1.06 (0.88-1.28)	BMI, EI, alcohol, smoking, history of HT, DM, multivitamin use, parity.
Maclure and Willet, 1990 [69]	US	Renal cell	PB case-control	430	203	30 - >80	M (67)	FFQ	SB: >480 vs <68.6 ml/d ASB: >480 vs <68.6 ml/d FJ: ≥ 480 vs ≤ 34.3 ml/d	OR: 2.6 (1.4-4.8) OR: 2.7 (1.1-6.5) OR: 0.56 (0.22-1.4)	Age, sex, body weight/height, EI, education
Ros et al, 2011 [37]	10 European countries[†], EPIC	Urothelial cell	Cohort	2332369.3	513	25-70	F (71)	DQ-country specific	SB: ≥99 vs <8 ml/d (M); ≥20 vs <8 ml/d (F) FJ: ≥72 vs <8 ml/d (M); ≥79 vs 8 ml/d (F)	HR: 1.03 (0.83–1.30) HR: 1.32 (1.05–1.66)	Smoking, EI from fat and non-fat sources. Stratified by age at entry, sex and centre.

Gynaecological cancers (cervical, endometrial, epithelial ovarian, ovarian)											
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake[+]**	**HR/RR/OR (95% CI)**	**Adjustments**
Herrero et al, 1991 [70]	Colombia, Costa Rica,	Cervical	HB, PB case-control	2033	622	46.5	F (100)	FFQ	FJ: >240 vs <0.8 ml/d	OR: 0.90 (0.7-1.2)	Age, study site, age at 1st intercourse, nº of sexual partners and pregnancies, presence

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	Mexico and Panama										of HPV 16/18, interval since last Pap smear, and nº of household facilities.
Verreault et al. 1989 [71]	US	Cervical	PB case-control	416	189	20-74	F (100)	66-items FFQ	FJ: ≥ 355 vs ≤ 48 ml/d	RR: 0.3 (0.2-0.6)	Age, education, smoking, frequency of Pap smears, use of barrier and OC, history of cervical-vaginal infection, age at first intercourse and nº of sexual partners.
Inoue-Choi et al, 2013 [38]	US	Endometrial type I	Cohort	23039 14	506	61.6	F (100)	FFQ	SFJ: >424.3 vs ≤55.7 ml/d SSB:>87.4 ml/d vs none ASB: >144 ml/d vs none FJ: >288 vs ≤20.6 ml/d	HR: 1.48 (1.09-2.00) HR: 1.78 (1.32-2.40) HR: 0.77 (0.59-1.01) HR: 1.16 (0.87-1.56)	Age, smoking, BMI, PA, alcohol, HRT, age at menarche and at menopause, nº of live births, DM, coffee intake.
		Endometrial type II			89				SFJ: >424.3 vs ≤55.7 ml/d SSB: >87.4 ml/d vs none ASB: >144 ml/d vs none FJ: >288 vs ≤20.6 ml/d	HR: 1.09 (0.55-2.15) HR: 1.31 (0.63-2.69) HR: 0.89 (0.48-1.68) HR: 0.97 (0.50-1.88)	
Hodge et al, 2018 [53]	Australia, MCCS	Endometrial	Cohort	35593 19	167	54.6	F (100)	121-item FFQ	SSB: ≥200 vs <6.7 ml/d ASB: ≥200 vs <6.7 ml/d	HR: 1.02 (0.54-1.91) HR: 0.81 (0.42-1.55)	Socio economic indexes, country of birth, alcohol, smoking, PA, Med-diet score, sex. ASB also for SSB consumption and WC.
		Ovarian			130				SSB: ≥200 vs <6.7 ml/d ASB: ≥200 vs <6.7 ml/d	HR: 1.35 (0.71-2.56) HR: 1.37 (0.72-2.61)	
King et al, 2013 [72]	US	Epithelial ovarian	PB case-control	595 7	205	>21	F (100)	FFQ and Interview	SSB: ≥151.2 vs <21.6 ml/2000kcal/d SSB: increase by 360 ml/d	OR: 1.31 (0.77-2.24) OR: 1.63 (0.94-2.83)	Age, education, race, age at menarche, MS, parity, OC use, HRT, BMI, smoking, PA, DM, tubal ligation, intake of fibre, fat, saturated fat.
Leung et al, 2016 [73]	Canada	Epithelial ovarian	PB case-control	2111 11	524	40-79	F (100)	FFQ and Interview	SB: >9.9 ml/d vs none	OR: 0.97 (0.72-1.31)	Age, race, education, BMI, smoking, alcohol, history of ovarian/breast cancer, OC use, parity, MS, HRT, study site.
Song et al, 2008 [74]	US	Epithelial ovarian	PB case-control	2050 3	781	35-74	F (100)	FFQ	SB[3] (caff): ≥720 ml/d vs none SB[3] (not caff): ≥720 ml/d vs none	OR: 1.51 (1.03-2.22) OR: 2.60 (1.25-5.39)	Age, BMI, education, smoking, race, country, years of diagnosis, number of pregnancies, OC use, hysterectomy, family history of breast/ ovarian cancer.

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Hepatobiliary cancers (biliary tract, gallbladder, liver)											
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake+**	**HR/RR/OR (95% CI)**	**Adjustments**
Stepien et al, 2014 [27]	10 European countries†, EPIC	Biliary tract	Cohort	477206 11.4	236	51	F (70)	DQ-country specific	SB: 282.9 ml/d vs none	HR: 0.96 (0.90-1.00)	BMI, alcohol, EI, PA, DM, education.
									FJ[1]: 171.7 ml/d vs none	HR: 0.99 (0.95-1.03)	
		IHBT			66				SB: 282.9 ml/d vs none	HR: 0.97 (0.90-1.06)	
									FJ[1]: 171.7 ml/d vs none	HR: 1.04 (1.00-1.08)	
		HCC			191				SB: 282.9 ml/d vs none	HR: 1.83 (1.11-3.02)	
									SB: increase by 300 ml/wk	HR: 1.05 (1.02-1.07)	
									SSB: increase by 330 ml/wk	HR: 1.00 (0.95-1.06)	
									ASB: increase by 330 ml/wk	HR: 1.06 (1.03-1.09)	
									FJ[1]: 171.4 ml/d vs none	HR: 1.38 (0.80-2.38)	
									FJ[1]: increase by 200 ml/wk	HR: 1.03 (1.01-1.06)	
Larsson et al, 2016 [48]	Sweden, SMC, COSM	IHBT	Cohort	70832 13.4	21	45-83	M (56)	96-item FFQ	SB: ≥400 ml/d vs none	HR: 1.69 (0.41-7.03)	Age, sex, education, smoking, BMI, dietary protein intake and EI.
		EHBT			127				SB: ≥400 ml/d vs none	HR: 1.79 (1.02-3.13)	
		Gallbladder			71				SB: ≥400 ml/d vs none	HR: 2.24 (1.02-4.89)	

Hematologic cancers (leukaemia, lymphoma, myeloma)											
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake+**	**HR/RR/OR (95% CI)**	**Adjustments**
Schernhammer et al, 2012 [23]	US, HPFS, NHS	Leukemia	Cohort	136587 14 HPFS 20 NHS	339	53.7	F (65)	FFQ	SSB: ≥335 ml/d vs none	RR: 1.06 (0.56-2.00)	Age, BMI, EI, PA, alcohol, race, fruit and vegetables consumption, menopause, HT. SSB were adjusted for use of ASB and vice-versa.
									ASB: ≥335 ml/d vs none	RR: 1.42 (1.00-2.02)	
		Multiple myeloma			285				SSB: ≥335 ml/d vs none	RR: 1.47 (0.76-2.83)	
									ASB: ≥335 ml/d vs none	RR: 1.29 (0.89-1.89)	
		NHL			1324				SSB: ≥335 ml/d vs none	RR: 1.34 (0.98-1.83)	
									ASB: ≥335 ml/d vs none	RR: 1.13 (0.94-1.34)	
McCullough et al, 2014 [39]	US, CPS-II NCH	NHL	Cohort	100442 10	1196	47-95	F (57)	Willett FFQ	ASB: >355 ml/d vs none	RR: 0.92 (0.73-1.17)	Education, race, WC, PA, BMI, EI, DM, family history of cancer, HTR and NSAIDs
									SSB: >355 ml/d vs none	RR: 1.10 (0.77-1.58)	

											use, cholesterol-lowering medication, intake of alcohol, read and processed meat, milk, saturated fat, fruits and vegetables, tea and coffee.
Upper aero-digestive cancers (larynx, oral cavity, oral and oropharyngeal squamous cell, pharynx)											
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake⁺**	**HR/RR/OR (95% CI)**	**Adjustments**
Zvrko et al, 2008 [81]	Montenegro	Larynx	HB case-control	216 2	108	59.9 (9.7)	M (82)	DQ	SB: yes vs no	OR: 0.38 (0.16-0.92)	Age, sex, smoking, alcohol, coffee, diet, personal and familiar medical history, education, housing and work conditions, exposure to toxic components.
Ren et al, 2010 [33]	US, NIH-AARP-DHS	Larynx Pharynx Oral cavity	Cohort	481563 2	307 178 391	50-71	M (59)	124-item FFQ	SB: ≥355 vs ≤355 ml/d SB: ≥355 vs ≤355 ml/d SB: ≥355 vs ≤355 ml/d	HR: 0.82 (0.55-1.23) HR: 0.76 (0.46-1.25) HR: 0.77 (0.54-1.09)	Age, sex, smoking, alcohol drinking, BMI, EI, education, ethnicity, PA, intake of fruit, vegetables, red and white meat.
Lissowska et al, 2003 [82]	Poland	Oral cavity	HB case-control	246	122	23-80	M (64)	25-item DQ	FJ: >57 vs <28.6 ml/d	OR: 0.35 (0.15–0.80)	Age, sex, residence, drinking and smoking habit.
Kreimer et al, 2006 [83]	9 countries‡, IARC-MOCS	OOSC	HB case-control	3402	1670	NR	M/F	FFQ	FJ: hight vs low intake	OR: 0.8 (0.6–1.1)	Age, sex, country, education, BMI, smoking, chewing and alcohol.
Other cancers											
Source	**Country, Study name**	**Cancer type**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake⁺**	**HR/RR/OR (95% CI)**	**Adjustments**
Vincenti et al, 2008 [84]	Italy	Cutaneous melanoma	PB case-control	118	59	56	F (53)	188-item FFQ	FJ (no OJ): increase by 10 ml/d OJ: increase by 10 ml/d	RR: 0.95 (0.87–1.03) RR: 0.94 (0.88–1.00)	EI, family history of melanoma, skin type, history of sunlight exposure and sunburns.
Dubrow et al, 2012 [46]	US	Glioma	Cohort	545771 10	904	62.8 (median)	M (60)	FFQ	SB: >720 ml/d vs none	HR: 0.87 (0.65-1.15)	Age, sex, race, EI, height, fruit and vegetables intake, nitrite intake from plants
Luqman et al, 2014 [85]	Pakistan	Lung	HB case-control	1200	400	<40 - >70	M (73)	DQ	J: yes vs no	OR: 0.3 (0.3-0.4)	Not reported

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Wu A. et al, 1997 [86]	US	Small intestine	PB case-control	1034	36	30–65	M (69)	Interview	SSB[7]: daily vs never	OR: 3.6 (1.3–9.8)	Age, ethnicity and sex.
Zamora-Ros et al, 2018 [47]	10 European countries[†], EPIC	Thyroid	Cohort	477206 11.4	748	51	F (70)	DQ-country specific	FJ[1]: > 94 vs < 1 ml/d FJ[1]: increase by 50 ml/d	HR: 1.23 (0.98-1.53) HR: 1.02 (0.99-1.06)	Age, sex, smoking status, BMI, EI, alcohol, PA, education, centre, menopausal status and type, OC use and infertility problems.
Overall cancers											
Source	**Country, Study name**	**Cancer types**	**Study design**	**Population Follow-up (years)**	**Cases**	**Age (mean/SD or range)**	**Sex (%)**	**Dietary assessment method**	**Type and amount of beverages intake[+]**	**HR/RR/OR (95% CI)**	**Adjustments**
Bassett et al, 2020 [49]	Australia, MCCS	Non-obesity related*	Cohort	35109 19	4789	27-76	F (61)	121-item FFQ	SSB: >375 vs none or < 12.5 ml/d ASB: >375 vs none or < 12.5 ml/d	HR: 1.02 (0.86-1.21) HR: 1.23 (1.02-1.48)	Alcohol, country of birth, Med-diet score, PA, socio-economic position, sex, smoking; ASB also adjusted for SSB intake.
Makarem et al, 2018 [51]	US	Breast, Colorectal, Prostate	Cohort	3184 4	565	54.3	F (53)	FFQ	SFJ: >501 vs <73.2 ml/d SSB:>180 ml/d vs none FJ: >216 vs <23 ml/d (cut-off)	HR: 1.28 (0.97-1.70) HR: 1.00 (0.79-1.27) HR: 1.05 (0.80-1.38)	Age, sex, EI, alcohol, smoking, BMI.
Hodge et al, 2018 [53]	Australia, MCCS	Obesity-related	Cohort	35593 19	3283	54.6	F (100)	121-item FFQ	SSB: ≥200 vs <6.7 ml/d ASB: ≥200 vs <6.7 ml/d	HR: 1.14 (0.93-1.39) HR: 1.00 (0.79-1.27)	Socio economic indexes, country of birth, alcohol, smoking, PA, Med-diet score, sex. ASB also for SSB consumption and WC.
Chazelas et al, 2019 [22]	France, NNS	Breast, Colorectal, Prostate	Cohort	101257 5.1 (median)	2193	42.2/14.4	F (78)	24H-DR	SFJ: >141.7 vs <46.1 ml/d (cut-off) SFJ: increase by 100 ml/d SSB: >65.5 vs <14.0 ml/d (cut-off) SSB: increase by 100 ml/d ASB: >7.9 vs <2.7 ml/d (cut-off) ASB: increase by 10 ml/d FJ: >97.8 vs <19.9 ml/d (cut-off) FJ: increase by 100 ml/d	HR: 1.30 (1.17-1.52) HR: 1.18 (1.10-1.27) HR: 1.06 (1.02-1.21) HR: 1.19 (1.08-1.32) HR: 1.00 (0.84-1.19) HR: 1.02 (0.94-1.10) HR: 1.14 (1.01-1.29) HR: 1.12 (1.03-1.23)	Smoking, education, PA, BMI, height.

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+ expressed in millilitre (ml) per day (d) or week (wk) or none (non-consumers). †Denmark, France, Germany, Greece, Italy, Norway, Spain, Sweden, The Netherlands, and United Kingdom. ‡Italy, Spain, Poland, Northern Ireland, India, Cuba, Canada, Australia and Sudan. * All cancers expect oesophagus, pancreas, colorectum, breast (postmenopausal), endometrium, kidney, ovary, gallbladder, liver, gastric cardia, meningioma, thyroid, multiple myeloma.

1: Fruit juice and vegetables juice. Vegetables juice <2%. 2: Colas. 3: Colas and root beer. 4: not carbonated beverages. 5: Sugarcane juice (20.3%), honeydew melon juice (14.1%), apple juice (12.8%), watermelon juice (9%), carrot juice (9%), pineapple juice (6.4%), star fruit juice (5.1%), and lemon juice drink (5.1%). The remaining canned grape, tomato, prune and juice, along with papaya, plum, and fresh celery juice, each comprised 1.3% to 2.6% of the total juice consumption reported. 6: fruit juice and nectars. 7: Carbonated beverages.

AA: African American; AEGJ: adenocarcinoma of the esophagus-gastric junction; ASB: artificially sweetened beverages; BC: breast cancer; BF: breastfeeding; BMI: body mass index; BWHS: Black Women's Health Study; Caff: caffeinated; CI: confidence interval; COSM: Cohort of Swedish Men; CPS-NCS: Cancer and Prevention Study, Nutrition Cohort Study; CR: colorectal; CVD: cardiovascular disease; EA: European American; EAC: oesophageal adenocarcinoma; EI: energy intake; EPIC: European Prospective Investigation into Cancer and nutrition; DH: Diet history; DM: Diabetes Mellitus; DQ: dietary questionnaire; 24H-DR: 24 hour dietary recall; F: female; FFQ: food frequency questionnaire; FJ: natural fruit juice; GI: glycaemic index; HB: hospital-based; HCC: Hepatocellular Carcinoma; HCS: Hokkaido Cohort Study; HPFS: Health Professionals Follow-up Study; HPV: Human Papilloma Virus; HR: hazard ratio; HRT: hormone replacement therapy; HT: hypertension; IARC-MOCS: International Agency for Research on Cancer, Multicentre Oral Cancer Study; IHBT: intra-hepatic biliary tract; J: natural fruit and vegetable juice; M: male; MCCS: Melbourne Collaborative Cohort Study; MDC: Malmö Diet and Cancer; Med: Mediterranean; MS: menopausal status; NCFD: not carbonated fruit drinks; NCSC: Nutrition Canada Survey Study; NHL: non-Hodgkin lymphoma; NNS: Nutri Net-Santé; NIH-AARP-DHS: National Institute of Health-American Association of Retired Persons, Diet and Health Study; NSAIDs: non-steroidal anti-inflammatory drugs; NHS: Nurses' Health Study; OC oral contraceptive; OJ: orange juice; OOSC: oral and oropharyngeal squamous cell; OR: odds ratio; PA: physical activity; PB: population-based; PC: prostate cancer; Post-M: postmenopausal breast cancer; PSA: prostate specific antigen; Pre-M: premenopausal breast cancer; Q: quantitative; Q1: First quartile; Q4: Quartile four; RR: relative risk; SB: total sweetened beverages, sugar and artificially sweetened beverages; SCC: squamous cell carcinoma SCHS: the Singapore Chinese Health Study; SD: standard deviation; SFQ: structured food questionnaire; SFB: San Francisco Bay Study: SFJ: beverages high in sugar, added or natural, SSB+FJ; SSB: sugar-sweetened beverages; SMC: Swedish Mammography Cohort; SQ: semi qualitative; SUVIMAX: Supplementation en Vitamines et Mineraux Antioxydants Study; UBC: urinary bladder cancer; UP: ultra-processed; US: United States; WC: waist circumference; WCHS: Women's Circle of Health Study.

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3.2.1. Sweet Beverages and Risk of Pre-menopausal Breast Cancer

Three cohort publications [22,52,54] and one case-control [56] were included in the analysis of SSB and pre-menopausal breast cancer and showed a borderline statistically significant positive association using the random-effects (RR: 1.37, 95% CI: 0.99 – 1.88), and statistically significant using fixed-effects meta-analysis (RR: 1.61, 95% CI: 1.48 – 1.76) (Figure S1). A cohort study [22], also reported data for ASB, FJ and SFJ and only indicated a positive association for SFJ (HR: 1.28, 95% CI: 1.09 – 1.83).

3.2.2. Sweet Beverages and Risk of Post-menopausal Breast Cancer

A meta-analysis of 4 cohort studies [22,52–54] and one case-control [56] on SSBs and post-menopausal breast cancer showed null results (Table 2). Chazelas *et al.* [22] investigated the relationships with SFJ consumption and observed a positive association (HR: 1.44, 95% CI: 1.05 – 1.99). No significant results were reported for ASBs.

Table 2. Summary of the results of the meta-analysis (random effects model).

Cancer Type	Exposure	Nº studies		RR (95% CI)	I^2 (%)	Tau²	95% PI
		Cohort	Case-control				
Breast	SSB	4	2	1.14 (1.01-1.30)	0.0	0.0073	0.88, 1.47
Breast	FJ	3	0	1.13 (0.93-1.38)	0.0	0.0017	0.52, 2.46
Breast Pre-M	SSB	3	1	1.37 (0.99-1.88)	55.7	0.058	0.68, 2.76
Breast Post-M	SSB	4	1	1.18 (0.79-1.75)	55.0	0.1080	0.43, 3.23
Colorectal	SSB	4	0	1.18 (0.99-1.41)	0.0	0.0039	0.82, 1.69
Colorectal	FJ	2	2	1.18 (0.79-1.75)	88.5	0.8629	0.01, 73.94
Colorectal*	FJ	2	1	1.29 (0.78-2.12)	0.0	0.0120	0.17, 9.81
Colorectal	SB	0	3	2.02 (0.45-9.01)	62.9	0.2711	0.00, 5757.1
Colorectal*	SB	0	2	1.57 (0.74-3.35)	0.0	0.0010	-
Bladder	SB	0	5	1.66 (0.78-3.56)	83.4	0.3226	0.22, 12.37
Bladder*	SB	0	4	1.27 (0.85-1.90)	25.3	0.0425	0.45, 3.60
Prostate	SSB	5	0	1.18 (1.10-1.27)	0.0	0.0012	1.03, 1.35
Prostate	FJ	4	0	1.03 (1.01-1.05)	0.0	0.0001	0.98, 1.09
Prostate	SB	1	2	0.97 (0.56-1.76)	2.9	0.0241	0.07, 12.7
Renal cell	SB	1	2	1.44 (0.46-4.50)	65.4	0.1559	0.0, 604.16
Pancreatic	SB	4	4	1.28 (0.95-1.72)	58.6	0.0962	0.56, 2.90
Pancreatic	SSB	4	1	1.01 (0.92-1.11)	0.0	0.0016	0.87, 1.17
Pancreatic	ASB	3	2	1.07 (0.77-1.48)	43.6	0.0480	0.48, 2.36

ASB: artificial sweetened beverages; FJ: fruit juice; PI: prediction intervals; Post-M: post-menopausal; Pre-M: pre-menopausal; RR: risk ratio; CI: confidence interval; SB: sweetened beverages (SSB and ASB taken together); SFJ: beverages high in sugar, added or natural; SSB: sugar sweetened beverages.
*Results excluding outliers

3.3. Sweet Beverages and Risk of Intestinal and Colorectal Cancer

Eight publications reported data on colorectal cancer, four case control [87–90] and four cohort studies [22,51,53,55]. A borderline positive association was observed with SBB intake using the random-effect model (RR: 1.18, 95% CI: 0.99 – 1.41), but it reached the significance using the fixed-effect model (RR: 1.19, 95% CI: 1.02 – 1.39) (Figure S1). For SBs, the results were similar:

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non-significant using the random-effects, but positively associated using the fixed model (RR: 1.73, 95% CI: 1.33 – 2.24) (Figure S2). Null results were found for the consumption of both FJs and ASBs. Although a second analysis was performed excluding outliers, results for the random-effect model remained null. With regard to rectal cancer, no associations were observed with either ASBs, SSBs or fruit and vegetables juices [91], A case-control study on small intestine cancer [64] indicated a significant positive association with SSB consumption (OR: 3.6, 95% CI: 1.3 – 9.8).

3.4. Sweet Beverages and Risk of Esophageal Cancer

Three publications, one cohort [33] and two case-control studies [58,92] reported data on different types of esophageal cancers, including esophagus-gastric junction, esophageal adenocarcinoma and squamous cell carcinoma. No significant associations were shown between SB, SSB and ASB consumption and esophageal cancer risk.

3.5. Sweet Beverages and Risk of Gastric Cancer

One case-control [58] and two cohort studies [33,53] reported data on different types of gastric cancer (overall, cardia and non-cardia) and SBs, ASBs and SSBs showing no significant associations.

3.6. Sweet Beverages and Risk of Pancreatic Cancer

Eleven publications, six cohort [40–44] and five case-control studies [75–78,80] reported data on pancreatic cancer. In the random-effect meta-analysis, no significant results were reported for SBs, SSBs or ASBs (Table 2). No association was observed between FJ intake and pancreatic cancer risk.

3.7. Sweet Beverages and Risk of Genitourinary Cancer

3.7.1. Bladder

Six case-control studies [59–64] reported data on bladder cancer. No association between the SB consumption and bladder cancer risk was observed in the random-effect meta-analysis including 5 case-control studies [60–64]. However, using the fixed-effect meta-analysis a positive association was found (RR: 1.43, 95% CI: 1.21 – 1.69) (Figure S2). We performed a second analysis excluding outliers, and, despite we found no positive results for random effects, we did for fixed effects (RR: 1.27, 95% CI: 1.06 – 1.51). Two case-control studies [59,64] also considered SSBs and ASBs separately. SSB intake was suggested as a risk factor for bladder cancer, while the consumption of FJ was not related with bladder cancer risk in a Chinese case-control study [61] and was negatively associated in a Serbian one [62].

3.7.2. Sweet Beverages and Risk of Prostate Cancer

Eight publications, six cohorts [22,30,34,35,51,53] and two case-controls [65,66] showed data on prostate cancer. No significant associations were reported for SBs from quantitative analysis; however, positive relations were observed with SSBs (RR: 1.18, 95% CI: 1.10 – 1.27) and FJs (RR: 1.03, 95% CI: 1.01 – 1.05). Two cohorts [22,53] reported data for ASB intake and only one [22] found an increased risk of 33% (HR: 1.33, 95% CI: 1.01 – 1.75).

3.7.3. Sweet Beverages and Risk of Renal and Urothelial Cell Cancer

Four publications, two case control [67,69] and two cohort studies [36,53] reported data on renal cell cancer. For our meta-analysis, we selected three publications, 2 case-control [67,69] and one control study [36] on SBs, and even though the pooled effect size showed null results using a random-effect meta-analysis (Table 2), a positive association was observed using fixed-effect meta-analysis (RR: 1.33, 95% CI: 1.06 – 1.66) (Figure S2). One case control study [69] reported a positive association with the intake of ASBs (OR: 2.7, 95% CI: 1.1 – 6.5) but not the other two [36,53]. No significant results were reported for SSBs or FJs, despite one case-control [67] found a positive

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association with natural the consumption of fruit and vegetable juices taken together (OR: 1.53, 95% CI: 1.18 – 1.99).

The EPIC cohort study [37] reported data on urothelial cell cancer and its association with SBs and FJs. A significant positive association was found only with the FJ intake (HR: 1.32, 95% CI: 1.05 – 1.66).

3.8. Sweet Beverages and Risk of Gynaecological Cancers

Two case-control studies [70,71] investigated the relationships between FJ intake and cervical cancer risk. Only one of them [71] found an inverse association (RR: 0.3, 95% CI: 0.2 – 0.6). Two cohort studies [38,53] reported data on different types of beverages (SSBs, ASBs, FJs and SFJs) and endometrial cancer risk. Only one of them [38] found significant positive associations with both SSBs (HR: 1.78, 95% CI: 1.32 – 2.40) and SFJs (HR: 1.48, 95% CI: 1.09 – 2.00). Finally, three case-control studies [69, 70,71] reported data on epithelial ovarian cancer risk and only one of them [71] found positive associations for caffeinated (OR: 1.51, 95% CI: 1.03 – 2.22) and non-caffeinated SBs (OR: 2.60, 95% CI: 1.25 – 5.36). No significant associations were reported for ovarian cancer risk [50].

3.9. Sweet Beverages and Risk of Hepatobiliary Cancers

Two cohort studies [27,48] reported data on different types of sweet beverages and various types of hepatobiliary cancers. The EPIC cohort [27] found no significant results between the consumption of either SBs or FJs and biliary tract cancer risk. However, a positive association was observed between both SBs (HR: 1.89, 95% CI: 1.11 – 3.02) and FJs (RR: 1.03, 95% CI: 1.01 – 1.06) and hepatocellular carcinoma risk. The Swedish Mammography Cohort and the Cohort of Swedish Men [48] found significant positive associations with both gallbladder (HR: 2.24, 95% CI: 1.02 – 4.89) and extra-hepatic biliary tract cancer (HR: 1.79, 95% CI: 1.02 – 3.13) cancer risks. No significant results were reported for intra-hepatic biliary tract cancer risk.

3.10. Sweet Beverages and Risk of Hematologic Cancers

One cohort study [21] reported data on leukaemia and multiple myeloma and its association with SSB and ASB intake. Significant associations were found between the consumption of ASBs and leukaemia risk (RR: 1.42, 95% CI: 1.00 – 2.02). Null results were observed in two cohorts [21,36] between drinking SSBs and ASBs and Non-Hodgkin Lymphoma risk.

3.11. Sweet Beverages and Risk of Upper Aero-Digestive Cancers

Four studies [33,81–83] reported data on upper aero-digestive cancers. One US-based cohort [33] showed null results for the association between SB intake and pharynx, larynx and oral cavity cancer risks. A case-control study from Montenegro [81] suggested an inverse relation between SBs and larynx cancer risk. The consumption of FJ was inversely associated with oral cavity cancer risk in one case-control study [82], but not in another one [83].

3.12. Sweet Beverages and Risk of Other Cancers

Single studies reported data on different types of cancer and their link with sweet beverages. No significant associations were reported for cutaneous melanoma [84], glioma [46] or thyroid cancer risk [47] and any type of sweetened beverages. One case-control study [85] reported an inverse association between natural juices (fruit and vegetables) and lung cancer risk (OR: 0.3, 95% CI: 0.3 – 0.4).

3.13. Sweet Beverages and Risk of Overall Cancer

An Australian cohort [49] investigated the association between SSBs and ASBs and the risk of non-obesity-related cancers, and reported a positive association only with ASBs (HR: 1.23, 95% CI:

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1.02 – 1.48). Two cohorts [22,51] assessed the relationships between the intake of several types of sweetened beverages and obesity-related cancer risk. Only one of them [22] showed positive associations with SSBs (HR: 1.06; 95% CI: 1.02 – 1.21), FJs (HR: 1.14, 95% CI: 1.01 – 1.29) and SFJs (HR: 1.30, 95% CI: 1.17 – 1.52) . No association was found for ASBs and obesity-related cancer risk.

3.14. Quality of Included Studies

According to ROBINS-E tool (Figure 2(a), Table S1), 13 of 27 cohort studies presented a moderate overall risk of bias. This is due to some bias detected mostly in the classification of the exposure domain, deviation from the intended intervention and missing data. Missing data bias was not evaluated for 5 cohorts [35,38,42,50,51], as the publications did not report enough information. All studies fulfilled the criteria of low risk of bias for selection of participants' domain. Three [35,36,53] from 27 studies did not adjust the statistical analysis for all potential confounders. Therefore, they were classified at moderate risk of bias. Only one study [49] was classified as moderate bias in the outcome measurement, and another [55] in the selection of reported outcomes.

According to the NOS tool (Figure 2(b), Table S2) most of the case-control studies (29 from 37) presented a moderate overall risk of bias. Seven publications presented a serious risk, whereas one indicated a low risk. The risk of bias due to the selection of the groups was classified as moderate for 35 studies, high for 2 [57,81] and low for another 2 [58,65]. Most of the case-control studies adjusted their results for relevant and additional confounders and were classified at moderate or low risk of bias for comparability between groups. Five were considered as serious risk for this domain, because 4 of them did not adjust for all important confounders [59,62,65,91] and one [78] reported results from an unadjusted analysis. Five studies [80,85,87,88] did not report this information and were classified as 'no information' category. The risk of bias due to ascertainment of the exposure was considered moderate in all case-control studies.

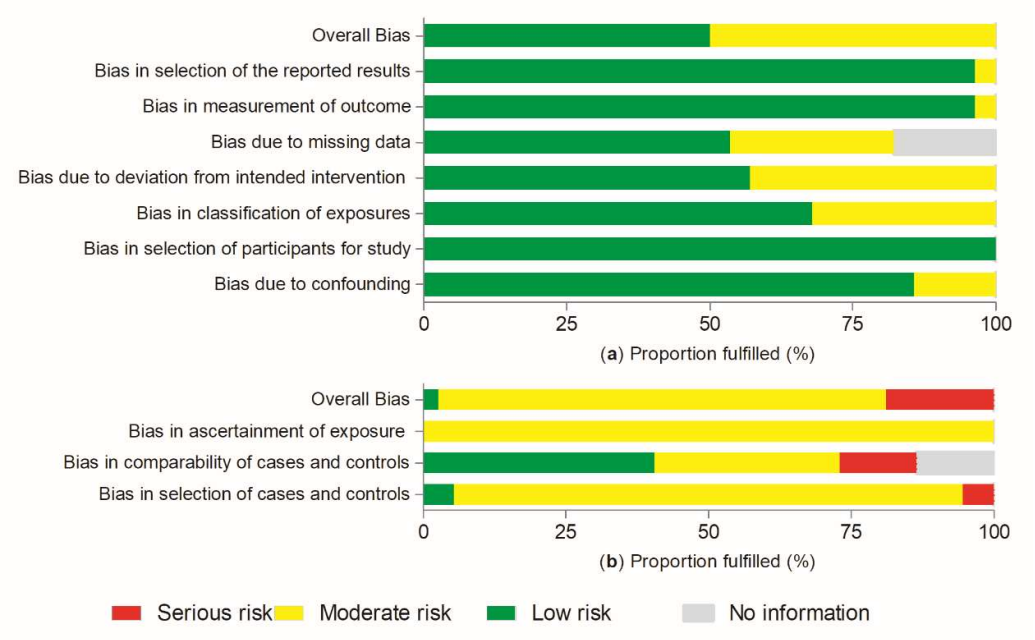

Figure 2. Risk of bias in the included studies. Legend: (**a**) Risk of bias in cohort studies according to the Risk of Bias in Non-randomized Studies - of Exposures (ROBINS-E) tool; (**b**) Risk of bias in case-control studies according to the Newcastle-Ottawa Scale (NOS) tool.

4. Discussion

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4.1. Association Between Consumption of Sweet Beverages and Cancer Risk

The aim of this study was to assess the relationships between different groups of sweetened beverages and site-specific or overall cancer risk. We conducted a meta-analysis when at least three studies reported data for the same exposure (sweetened beverage type) and outcome (cancer type). We found several statistically significant and borderline positive associations between the consumption of SBs, especially SSBs and in some cases ASBs and FJs, and several cancer risks.

Regarding breast cancer, the meta-analysis showed a positive association using random effects, with a 14% higher risk for SSBs, but non-statistically significant results for pre- and post-menopausal breast cancer. However, it showed a 61% higher risk for fixed effect meta-analysis between high amounts of SSBs and pre-menopausal breast cancer. Chazelas *et al* [22] reported a positive linear trend between SSB intake and breast and pre-menopausal breast cancer risk when SSB consumption increased by100 ml/day. In the same line with our results, current evidence supports the WCRF/AICR recommendations, including the reduce of SSB intake, for breast cancer prevention [93]. One US case-control study [56] that conducted a separate analysis for African-American and European-American women showed a positive link between SSB intake and post-menopausal breast cancer risk, but only for European-American women. Likewise, two other cohorts that included mostly Caucasian women [52,53] showed similar results. This evidence suggests that there might be ethnic differences. However, we could not explore this association as no other studies included women of African descent. In fact, evidence on the role of nutritional factors in breast cancer for this population is limited and inconclusive [94]. Our meta-analysis did not find significant associations between FJs and breast cancer risk. With regards to SFJ group, comparing highest versus lowest consumption, Chazelas *et al* [22] reported positive relations for SFJs and total, pre and post-menopausal breast cancer risk. Conversely, Makarem *et al* [51] showed no significant associations. A publication from the US [68] found no positive associations for SBs and breast cancer risk; however, a recent case-control study [57] found positive associations.

For colorectal cancer risk, our meta-analysis found null results using random effects for SB, SSB or FJ intake. However, we did find significant results for fixed effect meta-analysis for both SBs and SSBs with a higher risk of 73% and 19%, respectively. This is in a way consistent with results from a previous meta-analysis which found no association between SSBs and colon cancer risk using a random-effects model [20]. A cohort study from 2014 found a positive association for an increase of 330 ml/day [90]. Likewise, an Australian study that compared extreme categories of SSB intake (≥200 ml/day versus <6.7 ml/day) showed positive results [53]. We included just one study assessing rectal cancer incidence [91]. Here, a separate analysis for women and men was performed. The majority of the results were not significant, and the only positive association was found for juice (fruit and vegetables) consumption in female participants.

In regard to esophageal cancers, publications included in this review were also part of a meta-analysis from 2014 [17]. This meta-analysis reported no association between SBs and esophageal adenocarcinoma and squamous cell carcinoma risk. After extracting separated data for SSB and ASB intake, we found similar results. Despite these observations, positive associations were found in a pooled analysis of US-based case-control studies between sugar dietary intake and Barret's esophagus incidence, as a precursor for esophageal adenocarcinoma tumor [95]. Even though data from the included studies reported null results for stomach cancer incidence, a Japanese cohort study observed that carbonated drinks and juices appeared to be related to an elevated risk of death from stomach cancer [96].

With respect to pancreatic cancer, we performed a meta-analysis for SBs, SSBs and ASBs. The associations, especially for SBs, tended to be positive, but did not reach statistically significant levels using either random or fixed effect models. These results go along with a recent meta-analysis from 2019 [18], which also showed no association between SB drinking and pancreatic cancer risk. Besides that, a pooled analysis from 2012 [19] reported a 56% higher risk of pancreatic cancer only for males

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consuming ≥375 ml/day of SSBs compared to non-consumers. Likewise, a Swedish cohort [40] found a 93% higher risk of pancreatic cancer incidence among those who consumed ≥500 ml/day of SSBs compared to non-consumers. Only one study reported separate results for carbonated and non-carbonated SBs, but no significant results were shown [75].

For bladder cancer risk, three out of six included case-control studies [61,62,64] showed positive associations for highest versus lowest amounts of SB intakes. However, the meta-analysis of these studies together with two other case-control studies [60,63] showed no significant associations for random effects. These results may be explained by the high heterogeneity between studies (I^2 = 83.4%). Conversely, a positive association was found for fixed effects, and after excluding outliers, a 26% higher risk was reported for the fixed effect model. Our meta-analysis of observational studies reported that soft drinks appeared to be unrelated to bladder cancer risk. There is no clear role of SSBs, ASBs or FJs alone, as the evidence is limited. However, the evidence is suggestive but not conclusive for SBs as risk factor for bladder cancer.

With reference to prostate cancer, our meta-analysis demonstrated an 18% higher risk for SSBs comparing the highest with the lowest intake. Similarly, we found a small positive association for FJs (a 3% higher risk). No associations were found for SBs, which may suggest that the role of ASBs might not be relevant; although one study [22] reported a positive relation for ASB intake.

Renal cell cancer appeared to be unrelated to SB consumption according to the random-effect meta-analysis results; although statistically significant positive associations were observed using fixed effect meta-analysis. Indeed, Maclure and Willet [69] reported a similar significant positive association between highest versus lowest intake of SBs and renal cell cancer risk.

The association between SSB consumption and both endometrial and ovarian cancer risk tended to be positive but did not reach statistically significant levels. One study stratifying results by types of endometrial cancer (I and II) [38] reported positive associations between highest versus lowest SSB and SFJ consumption and endometrial type I cancer in postmenopausal women, but not in type II. These might be because subtypes could have different risk factors, even though evidence on this etiologic heterogeneity is quite limited [97]. Data from two studies [70,71] suggested that FJ intake might be a protective factor for cervical cancer. FJ consumption is often part of a healthy diet and lifestyle [98]. However, none of the mentioned studies [70,71] were adjusted by such confounders, thus it is not clear if the protective effect was due to FJ intake or other factors. For epithelial ovarian cancer, one US study [74] stratified the results by caffeinated and non-caffeinated colas. Both results were statistically significant, but non-caffeinated sodas showed a stronger association. Although this might suggest a positive relationship with caffeine, a recent meta-analysis of prospective studies found no link between caffeine intake and ovarian cancer risk [99].

In respect of hepatobiliary cancers, data from the included studies showed a positive association with SB consumption, especially for gallbladder cancer, where the risk was doubled [48]. This might be explained by the detrimental association between sucrose and glycemic load with the increased risk of symptomatic gallstone disease [100], which is strongly correlated with gallbladder cancer [101]. Stepien *et al* [27] showed slightly positive dose-response associations between SBs, ASBs and FJs and HCC incidence.

As regards the hematologic cancers, no associations were found either for sugary or for artificially-sweetened beverages, except for leukemia risk, for which one study [23] reported significant positive associations with ASBs. However, a recent review of clinical trials and observational studies observed no association between artificial sweeteners intake and both leukemia and NHL incidence [102].

The evidence regarding oral cancer, pharynx, larynx, lung, thyroid, glioma and cutaneous myeloma is more limited. The available data mainly showed null results for SB and FJ drinking. Only one study from Montenegro indicated an inverse association between SB intake and larynx cancer risk [81]. However, the results from this study would be treated with caution as they presented some methodological drawbacks and its overall risk of bias was classified as 'serious risk' (Table S2). The incidence of small intestine cancer was 3.6 folded by the consumption of SSBs [86]. Controversially, one study that reported the incidence of overall non-obesity cancers, showed no association for SSBs, although a positive association for ASBs [49]. Moreover, from 3 studies [22,51,53] on overall obesity-related cancer risk, the larger one [22] showed positive relationships for SFJs, SSBs and FJs, but not for ASB drinking. Similarly, a meta-analysis of clinical trials and observational studies showed no association between artificial sweetener intakes and body weight and different types of cancers [102]. We go along with these results, and agree upon the uncertainty of the evidence that links artificial sweeteners with different types of cancer.

4.2. Limitations of the current data

To the best of our knowledge, this is the first systematic review that evaluated the isolated association between different groups of sweet beverages and cancer risk. Some limitations should be considered while interpreting our findings. Few studies included in this systematic review were difficult to compare due to their design (cohort and case-control studies), methods, classification and categories of beverages intake, and therefore, comparing them was challenging. According to the ROBIN-E tool, cohort studies were at low-moderate risk of bias. As per the NOS tool, the case-control studies were at moderate risk of bias and 6 studies [57,59,65,78,81,85] out of 37 presented serious methodology drawbacks. The number of publications included in each meta-analysis was relatively low, maximum 6, except for pancreatic cancer, for which we included 8 studies. Due to the low number of candidate publications for the meta-analysis, the pooled effect size was calculated based on risk ratios of cohort and case-control studies together. The majority of the included participants were from US and European countries. Hence, extrapolating our findings to other geographical areas may not be appropriate. We attempted to classify beverages into specific groups. However, some studies did not report precise information on this topic, which might have given rise to misclassifications. Similarly, we attempted to convert original exposure information into amounts of intake (ml/day) based on national data. Nevertheless, this was not possible in all studies, which did not allow us to perform a dose-response meta-analysis. Another limitation may be the measurement error in collecting dietary data since self-reported questionnaires were used. Moreover, in the longitudinal studies, we were limited to the baseline estimation of beverages consumption and there is a possibility that their consumption changed over time. Although it is suggested that the link between SSBs and FJs and cancer risk is possible due to their high glycemic indexes [11] and to obesity-inducing pathways [2], there were not adequately integrated as confounders in all the studies. Indeed, glycemic index was only considered in one cohort [52]. Despite BMI is a common indicator of obesity and most of the studies considered it as confounder, only 4 of them [34,39,51,53] tested for other obesity indicators such as waist circumference. Most of the studies assessed the association between consumption of SSB and common cancers such as breast, colorectal, prostate and pancreatic cancer. Data were more limited for FJs, ASBs and other types of cancers, especially non-obesity related ones. FJ consumption may coexist with healthy habits, such as healthy diet or exercise [98], so it would have been even better if some studies had adjusted their analysis for such variables. In fact, only three publications [51–53] used diet quality as confounder.

5. Conclusions

The current meta-analysis of cohort and case-control studies indicated a statistically significant positive association between higher consumption of SSB and breast and prostate cancer incidence. Likewise, it showed a statistically significant positive link between higher intakes of FJ and prostate cancer risk. The association between SSB drinking and pre- and post-menopausal breast cancer,

colorectal and pancreatic cancer risk was positive but did not reach statistically significant levels. It was also the case between FJs and breast and colorectal cancer risks, ASBs and pancreatic cancer and SBs and colorectal, bladder, pancreatic and renal cell cancer risks. We did not find statistically significant results probably due to the heterogeneity between studies and the small number of cancer cases. Overall, findings from this systematic review and meta-analysis suggest positive associations between high intakes of SSB, ASB and FJ and risk of some types of cancer. This systematic review supports the recommendations of World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) to limit sugary drinks consumption including FJ for cancer prevention [103] and to raise consumers' awareness of their low nutritional quality and high sugar content.

The onset of cancer by ASB consumption is biologically possible. However, the evidence is limited to make recommendations and this subject requires further investigation. We recommend for the future research in this field consider other obesity-related factors besides body mass index, such as waist circumference, glycemic index and quality of diet as confounders. We also encourage to perform more separated analysis on SSB, ASB and FJ consumption, and to establish a homogeneous classification of beverages in order to better understand their role in carcinogenesis. We could not study the different roles of non-carbonated soft drinks (sport, fruit and tea-based drinks), sometimes used as healthier alternatives to carbonated drinks [104]. Therefore, it would be advisable for future studies to further explore this subject.

Supplementary Materials: The following are available online at www.mdpi.com/xxx/s1, Table S1: Risk of bias in the included cohort studies according to ROBINS-E, Table S2: Risk of bias in the included case-control studies, according to NOS, Figure S1: Forest plot showing the pooled risk ratios with 95% CI for cancer risk, comparing the highest vs. the lowest sugar-sweetened beverages (SSB) intake. Figure S2: Forest plot showing pooled risk ratios with 95% CI for cancer risk, comparing the highest vs. the lowest sugar or artificially-sweetened beverages (SB) intake. Figure S3: Forest plot showing the pooled risk ratios with 95% CI for cancer risk, comparing the highest vs. the lowest fruit juice (FJ) intake. Figure S4: Forest plot showing the pooled risk ratios with 95% CI for cancer risk, comparing the highest vs. the lowest artificial-sweetened beverages (ASB) intake.

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