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Systematic Review

Prostate Cancer and Dietary Sugar Intake: A Systematic Review

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Simple Summary

Prostate cancer is a leading cancer among men worldwide. While some risk factors like age and genetics are unchangeable, lifestyle choices, including diet, may influence the risk. Recent studies have explored whether consuming high amounts of sugar, especially from sweetened foods and drinks, could be linked to an increased risk of developing prostate cancer. In this review, we examined the existing research to understand this potential connection. We found that some studies suggest a possible link between high sugar intake and increased prostate cancer risk, while others do not show a clear association. These mixed findings highlight the need for more detailed and consistent research. Understanding how sugar consumption affects prostate cancer risk could help in developing dietary recommendations and public health strategies aimed at prevention.

Abstract

Background: Prostate cancer is a leading malignancy among men globally, with its incidence expected to rise due to aging populations and shifting lifestyles. While established risk factors include age, ethnicity, and genetics, the role of modifiable dietary factors, particularly sugar intake, remains less clear. Emerging evidence suggests that high sugar consumption may promote carcinogenesis through insulin resistance, chronic inflammation, and hormonal dysregulation. This systematic review aimed to evaluate the current evidence on the association between dietary sugar intake and prostate cancer risk. **Methods**: A systematic search was conducted across six databases for observational studies published between January 2005 and April 2025. Eligible studies assessed the associations between quantitative sugar intake and prostate cancer outcomes. Screening, data extraction, and a risk of bias assessment (using ROBINS-E) were performed independently by multiple reviewers. **Results**: Six studies met the inclusion criteria, comprising four prospective cohorts, one case–control study, and one cross-sectional study, with a combined sample of 11,583 men from the USA, Canada, Sweden, and France. Three studies reported a significant positive association between a high intake of dietary sugars and prostate cancer risk, two found no association, and one showed mixed findings depending on the type of sugar. Heterogeneity in the exposure assessments and confounder control limited the comparability. **Conclusions**: This review suggests a possible association between high dietary sugar intake and increased prostate cancer risk, especially from added sugars and sugar-sweetened beverages. However, inconsistent findings and methodological limitations highlight the need for robust, prospective studies with standardized assessments to understand this relationship better.

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1. Introduction

Prostate cancer is among the most frequently diagnosed malignancies in men worldwide, with approximately 1.4 million new cases and over 375,000 deaths reported in 2020 [1]. Projections indicate that the global incidence of prostate cancer will nearly double by 2040, reaching an estimated 2.9 million cases annually, primarily due to aging populations and lifestyle transitions [2,3].

While the established risk factors include age, ethnicity, and genetic predisposition, increasing scientific attention is being directed toward the role of modifiable lifestyle factors, particularly diet, in prostate cancer's development and progression [4]. Among dietary components, the intake of added sugars and sugar-sweetened beverages (SSBs) has garnered interest due to their association with obesity, insulin resistance, and chronic inflammation, which are conditions implicated in carcinogenesis [5].

Several biological mechanisms explain the link between high sugar intake and prostate cancer development. First, the excessive consumption of dietary sugars, particularly fructose and high-glycemic-load foods, can lead to chronic hyperinsulinemia and insulin resistance [6]. Elevated insulin levels may promote cancer development by enhancing the bioactivity of insulin-like growth factor 1 (IGF-1), a mitogen that stimulates cellular proliferation and inhibits apoptosis in the prostate tissue [7]. Second, a high sugar intake has been shown to induce chronic low-grade inflammation by increasing circulating levels of pro-inflammatory cytokines such as TNF-α, IL-6, and CRP, which can create a tumor-promoting microenvironment [8]. Third, excessive sugar consumption may contribute to obesity and visceral adiposity, which in turn can alter levels of sex hormones, including reductions in testosterone and increases in estrogen, thereby influencing prostate cancer risk through hormonal modulation [9]. Moreover, evidence from in vitro and animal studies has suggested that cancer cells, including prostate cancer cells, exhibit increased glucose uptake and metabolism (the Warburg effect) and may rely heavily on sugar as a fuel source to sustain their rapid proliferation [10]. Thus, high availability of sugar could theoretically support tumor growth and progression.

Epidemiological studies examining the association between dietary sugar intake and prostate cancer risk have yielded inconsistent results. Some prospective cohort studies have reported a positive association between a high intake of SSBs and increased prostate cancer risk [11]. Conversely, other studies have found no significant association or have highlighted the complexity of dietary patterns and their interactions with other lifestyle factors [12].

Given the conflicting evidence and its potential public health implications, a comprehensive synthesis of the existing research is warranted. This systematic review aims to critically evaluate and synthesize the findings from studies published between 2005 and 2025 on the association between dietary sugar intake and the risk of prostate cancer.

2. Methods

2.1. The Protocol and Registration

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [13]. The protocol was developed and was registered with the International Prospective Register of Systematic Reviews (PROSPERO) (Registration ID: CRD420251039770) [14].

2.2. The Search Strategy

A comprehensive literature search was conducted from 1 March 2025 to 23 April 2025 across the following databases: MEDLINE Complete, Embase, Scopus, Web of Science, Cochrane Library, and ScienceDirect. The search strategy was framed using the PEO model (Table 1). Keywords and index terms were identified and refined and then combined using Boolean operators (AND/OR). The search was limited to human studies published in English between January 2005 and April 2025. The search terms included "sugar intake," "added sugars," "sugar-sweetened beverages," and "prostate cancer" and associated Medical Subject Headings (MeSH) or database-specific subject headings (e.g., CINAHL headings).

Table 1. Search strategy for dietary sugar intake and prostate cancer in men.

PEO	Keywords with Boolean Operators and Truncation
Population (Men)	M#n OR male OR males OR "adult m#n" OR "human males" OR "middle-aged m#n" OR "elderly m#n"
Exposure (Sugar)	sugar* OR "dietary sugar*" OR "added sugar*" OR "free sugar*" OR sucrose OR fructose OR glucose OR "sugar-sweetened beverage*" OR "soft drink*" OR soda OR "sugary drink*" OR "sweetened food*" OR "high-sugar diet*" OR "sugar-rich diet*"
Outcome (Prostate Cancer)	"prostate cancer" OR "prostatic cancer" OR "prostate neoplasm*" OR "prostatic neoplasm*" OR "prostate carcinoma" OR "prostate malignanc*" OR "prostate tumor*" OR "prostate adenocarcinoma"
Combined Search	(men OR male OR males) AND (sugar* OR "added sugar*" OR "sugar-sweetened beverage*" OR soda) AND ("prostate cancer" OR "prostate neoplasm*" OR "prostate tumor*")

The searches were supplemented by a manual review of the reference lists of the included studies and relevant reviews. The authors of unpublished or incomplete studies were contacted when additional data were required.

2.3. Selection of the Studies

The primary and secondary reviewers (K.K. and H.J.) screened titles and abstracts for relevance. A full-text screening of potentially eligible studies was then conducted. All excluded full texts were independently reviewed to confirm the exclusion decisions. Discrepancies between reviewers were resolved through discussion or with the input of a third reviewer (O.A.), where needed. Eligibility decisions were based on the criteria described below.

2.4. The Inclusion and Exclusion Criteria

Studies were included if they

(i) Included adult men (≥18 years);
(ii) Measured sugar intake quantitatively (e.g., total sugars, added sugars, sugar-sweetened beverages);
(iii) Reported prostate cancer risk, incidence, or mortality as a primary or secondary outcome;
(iv) Reported sugar intake as an independent exposure or measured it in combination with other nutrients without disaggregation;
(v) Were observational studies (cohort, case–control, or cross-sectional);
(vi) Were published in English in peer-reviewed journals.

Studies were excluded if they

(i) Did not report the effect estimates or necessary data (e.g., odds ratios, hazard ratios, relative risks);

(ii) Focused on animal or in vitro models;
(iii) Were editorials, reviews, conference abstracts, or theses;
(iv) Included participants with diagnosed cancer at the baseline (except in mortality outcome studies).

2.5. The Data Extraction

Data extraction was completed by the lead reviewer and the secondary reviewer (K.K. and H.J.) using a structured template. The information extracted included author, year, country, study design, sample size, participant age range, duration of follow-up (if longitudinal), method of sugar intake assessment (e.g., an FFQ, 24 h recall), the type of sugar assessed, the prostate cancer outcome definitions and assessment tests, confounders adjusted for in the statistical analyses, and the effect estimates (including 95% confidence intervals).

A subset of 25% of the extracted data was verified by a second reviewer (O.A.) to ensure accuracy and completeness. Discrepancies were resolved through discussion between the three reviewers.

2.6. The Study Exposure

The primary exposure of interest was dietary sugar intake, assessed in terms of total sugars, added sugars, or intake of sugar-sweetened beverages (SSBs) or sweet food groups (e.g., sweets intake), as defined by the original studies. Sugar intake was considered irrespective of the dietary assessment tool used, including but not limited to food frequency questionnaires (FFQs), 24 h dietary recall, dietary records, or validated diet history interviews.

2.7. The Study Outcomes

The primary outcome was the risk of prostate cancer. The secondary outcomes included prostate cancer incidence or mortality (where reported). Studies reporting any histologically confirmed prostate cancer endpoint, or registry-confirmed mortality, were considered eligible.

2.8. Quality Evaluation

Risk of bias was assessed using the Risk of Bias in Non-randomized Studies of Exposure (ROBINS-E) tool [15]. The domains evaluated included bias due to confounding factors, bias arising from the measurement of the exposure, bias in the selection of participants into the study, bias due to post-exposure interventions, bias due to missing data, bias in the measurement of the outcome, and bias in the selection of the reported results. Each study was independently assessed by two reviewers (K.K. and H.J.), with discrepancies resolved through discussion or referral to a third reviewer (O.A.).

3. Results

The initial database search yielded 12,625 results, of which only 273 had relevant titles (Figure 1). After screening the titles and abstracts, 229 articles were excluded as irrelevant, leaving 44 articles for full-text review. Of these, 38 studies were further excluded for not meeting the inclusion criteria. Ultimately, six studies were included in the review (Figure 1).

3.1. The Characteristics of the Included Studies

Table 2 summarizes the main characteristics of the included studies. The publications span from 2012 to 2021, with the sample sizes ranging from 291 to 6403 participants. Geographically, three studies were conducted in the USA [11,12,16], one in Canada [17], one in Sweden [18], and one in France [19]. The study designs included four prospective cohort studies, one case–control study, and one cross-sectional study. The follow-up

durations ranged from 9 to 22 years for prospective studies. The recruitment of the participants varied across studies, where the majority included data on participants previously enrolled in cohort studies and clinical trials such as NHANES [11]; the French NutriNet-Sante Cohort [19]; the Framingham Offspring Cohort [16]; the Prostate, Lung, Colorectal, and Ovarian (PLCO) Cancer Screening Trial [12]; and the Malmo Diet and Cancer Cohort Study [18].

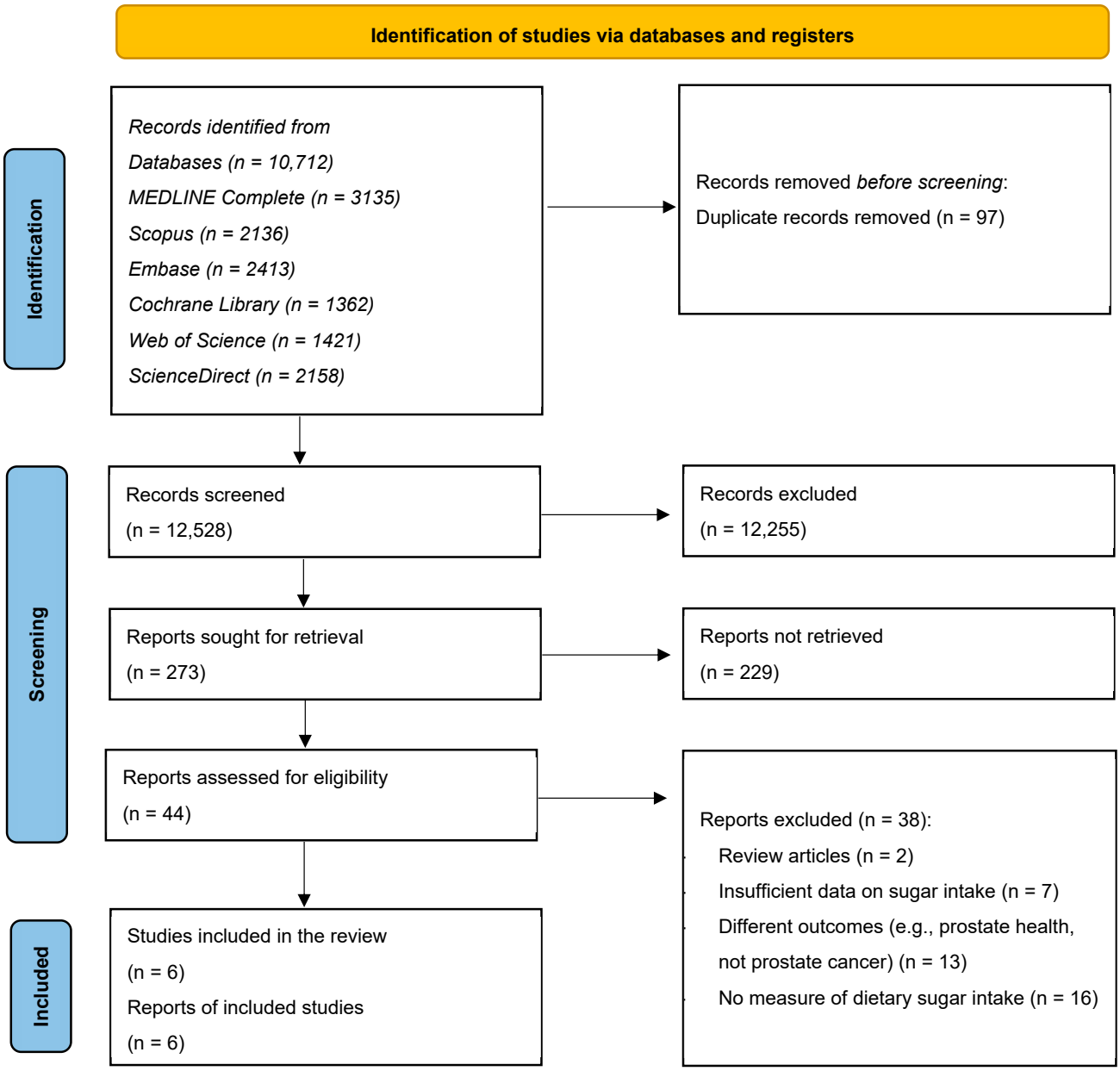

Figure 1. A PRISMA flow diagram of the search strategy and selection process.

Table 2. Study characteristics of the included studies (by year of publication).

Author, Year	Country	Study Design	Age Range or Mean Age	Number of Participants	Participants in Study
Liu et al., 2021 [11]	USA	CS	Mean age: 58.1 (± 13.6)	6403	Men aged >40 years from the NHANES study with no history of malignancy.
Trudeau et al., 2020 [17]	Canada	CC	Mean age: Cases: 64 years Control: 65 years	Cases (n = 1919) Controls (n = 1991)	Cases enrolled from 7–9 French language hospitals in Montreal. Controls enrolled randomly from Quebec's permanent electoral list of French electors

Table 2. *Cont.*

Author, Year	Country	Study Design	Age Range or Mean Age	Number of Participants	Participants in Study
Chazelas et al., 2019 [19]	France	LG (9 years follow-up)	Mean age: At baseline: 46.9 At cancer diagnosis: 58.5	291	Adults from the French NutriNet-Sante Cohort not previously diagnosed with any type of cancer.
Makarem et al., 2018 [16]	USA	LG (22 years follow-up)	26–84 (55.4)	157	Adults from the Framingham Offspring Cohort.
Miles et al., 2018 [12]	USA	LG (9 years follow-up)	55–74 years	1996	Men from the general population from the PLCO Cancer Screening Trial
Drake et al., 2012 [18]	Sweden	LG (15 years follow-up)	45–73 (58.5)	817	Cases of prostate cancer diagnosed between 1992 and 2009 from the Malmo Diet and Cancer Cohort Study

CS (Cross-Sectional), LG (Longitudinal), CC (Case–Control), NHANES (National Health and Nutrition Examination Survey), PLCO (Prostate, Lung, Colorectal, and Ovarian).

3.2. The Studies' Exposure and Outcome Assessment Methods

The types of dietary sugar intake assessed varied among the studies: Liu et al. [11] assessed total dietary sugar intake, while the rest evaluated specific exposures, such as sugar-sweetened beverages [17], sugary drinks [16,19], sugary foods [16], and individual sugar subtypes, including monosaccharides [18], sucrose [18], cakes and biscuits [18], sweets [17,18], and fruit juice [12,18] (Table 3).

Table 3. The data extracted from the six included studies on dietary sugar intake and prostate cancer (by year of publication).

Author, Year	Type of Sugar Measured	Sugar Intake Assessment Tool	Prostate Cancer Risk Assessment Methods	Confounding Factors
Liu et al., 2021 [11]	Total dietary sugar intake	USDA AMPM	Hybritech PSA method	Age, SES, BMI, smoking, and history of diabetes, hypertension, and coronary heart disease/stroke.
Trudeau et al., 2020 [17]	Western Sweet and Beverage intake	63-item FFQ	- Gleason score - PSA - DRE	Age, ethnicity, education, family history of prostate cancer, timing of last screening.
Chazelas et al., 2019 [19]	Sugary drinks	Three 24-dietary records	Medical records and ICD-10	Age, sex, energy intake, smoking, family history of cancer and diabetes, BMI, physical activity.
Makarem et al., 2018 [16]	- Sugary foods - Sugary drinks	Semi-quantitative 126-item Harvard FFQ	Medical and pathology records	Age, sex, smoking, alcohol, energy intake, BMI, waist circumference, chronic diseases (CVD and diabetes) history, physical activity, antioxidant use.
Miles et al., 2018 [12]	Concentrated sugars (sugar sweetened beverages, desserts, and fruit juices)	DHQ FFQ	- PSA - DRE	Study center, age, race, education, smoking, BMI, history of diabetes and prostate cancer, number PSA screens over the previous three years, energy intake (kcal/day), red and processed meat (g/day), fruit (servings/day), and vegetables (servings/day).
Drake et al., 2012 [18]	- Monosaccharides - Sucrose - Sugar-sweetened beverages - Cakes and biscuits - Sweets and sugar - Fruit juices	- 168-item quantitative diet history questionnaire - 7-d menu book, - 1-h dietary interview	- clinical tumor stage - lymph node metastasis or bone metastasis - Gleason score - PSA	Age, BMI, waist circumference, alcohol intake, selenium intake, calcium intake, smoking, educational level, physical activity, diabetes diagnosis, history of cardiovascular event, born in Sweden, past food habit change, and energy intake.

FFQ (Food Frequency Questionnaire), PSA (Prostate-Specific Antigen), DRE (Digital Rectal Examination), USDA AMPM (The US Department of Agriculture Automatic Multiple Pass Method), ICD-10 (International Classification of Diseases), DHQ (Diet History Questionnaire), FFQ (Food Frequency Questionnaire), BMI (Body Mass Index), SES (Socioeconomic Status).

All of the studies assessed dietary intake using self-reported instruments: four used food frequency questionnaires (FFQs) [12,16–18], and two used 24 h dietary recalls [11,19].

Dietary sugar intake was generally categorized into tertiles, quartiles, or quintiles. The lowest intake category was used as the reference group for calculating the relative risks.

All of the studies assessed prostate cancer incidence, confirmed through medical records or cancer registries (Table 3). Risk or diagnosis was confirmed in four studies using pathology or cancer registry data [16–19] and was supported by clinical diagnostic tools such as PSA levels [12,17,18], Gleason scores [17,18], and digital rectal examination (DRE) [12,17]. Two studies used PSA concentration as a biomarker for cancer diagnosis [11,12].

3.3. Confounding Factors

The studies adjusted for a range of potential confounders, including (1) age, race, and education; (2) family history of prostate cancer; (3) smoking status, alcohol consumption, and physical activity; (4) total energy intake and BMI; and (5) other dietary factors, such as fat, fiber, calcium, and dairy intake. However, the adjustment for potential mediators such as insulin, glucose, or diabetes status was limited.

3.4. The Study Findings

Across the six included studies, most found no significant association between dietary sugar intake and prostate cancer risk or related outcomes [16–18] (Table 4). These studies examined a range of sugar sources, including sugary foods, beverages, and specific sugar types. In contrast, three studies reported positive associations, linking a higher sugar or sugar-sweetened beverage intake to increased prostate cancer risk, PSA levels, and consumption patterns typical of a Western dietary pattern [11,12,17]. For example, in the study by Trudeau et al. [17], the association between Western sweet and beverage pattern scores (in quartiles) and the risk of overall PCa (Prostate Cancer) was higher in the fourth quartiles as compared to that in the first (1.35 [1.10–1.66] (0.002)) (Table 4).

Table 4. Findings of the six included studies.

Author, Year	Association Between Sugar Intake and Prostatic Cancer			Association Direction
	β [95% CI] (p-Value) in Fully Adjusted Multivariable Weighted Linear Regression	**Hazard Ratio [CI] (p-Value)**	**OR [CI] (p-Value) in Adjusted Logistic Regression**	
Cross-sectional study				
Liu et al., 2021 [11]	For each additional 1 g of sugar intake, the PSA concentrations were increased by 0.003 ng/mL [0.001–0.005] (0.0029) (log2-transformed)			↑
Case–control study				
Trudeau et al., 2020 [17]			1.35 [1.10–1.66] (0.002) ‡	↑
Longitudinal studies				
Chazelas et al., 2019 [19]	1.1 [0.92–1.31] (0.31) *			⇌
Makarem et al., 2018 [16]		Sugary foods ^ 1.00 [0.62–1.62] ($p > 0.05$)		⇌
		Sugary drinks ^ 1.36 [0.88–2.09] ($p > 0.05$)		⇌
Miles et al., 2018 [12]		Sugar-sweetened beverages ^ 1.21 [1.06–1.39] ($p < 0.01$)		↑
		Fruit juices ^ 1.07 [0.94–1.22] ($p > 0.05$)		⇌
		Desserts ^ 0.95 [0.83–1.10] ($p > 0.05$)		⇌
Drake et al., 2012 [18]		Monosaccharides 1.18 [0.92–1.52] (0.59)		⇌
		Sucrose 0.9 [0.71–1.15] (0.83)		⇌
		Sugar-sweetened beverages 1.13 [0.92–1.38] (0.22)		⇌
		Cakes and biscuits 1.21 [0.94–1.56] (0.23)		⇌
		Sweets and sugar 0.93 [0.73–1.19] (0.63)		⇌
		Fruit juices 0.99 [0.81–1.22] (0.62)		⇌

PSA (Prostate-Specific Antigen), OR (Odds Ratio), CI (Confidence Interval), ↑ (positive significant association), ⇌ (no significant association). * Competing risk model (the sub-distribution hazard ratio). ^ Multivariable adjusted hazard ratio. ‡ The fourth quartile of the Western sweet and beverage pattern scores compared to the first quartile.

3.5. Quality Assessments

The risk of bias assessment across the six included studies revealed varying levels of methodological quality across different domains (Figure 2, Supplementary Figure S1). Bias due to confounding was generally rated as low-risk in most studies, although approximately 40% presented some concerns. Concerning their measurement of exposure, most studies raised some concerns, with a few identified as high-risk. The selection of participants was low-risk in about half of the studies, while the remainder exhibited some

concerns. Notably, bias related to post-exposure interventions was consistently rated as having some concerns, with no studies classified as low-risk. Regarding missing data, most studies demonstrated low risk, with only a minority showing some concerns. Measurement of the outcomes was predominantly low-risk, but a considerable proportion of studies raised some concerns, and a few were categorized as high-risk. Finally, bias in the selection of the reported results was low-risk in roughly 60 to 70% of the studies, with the rest presenting some concerns.

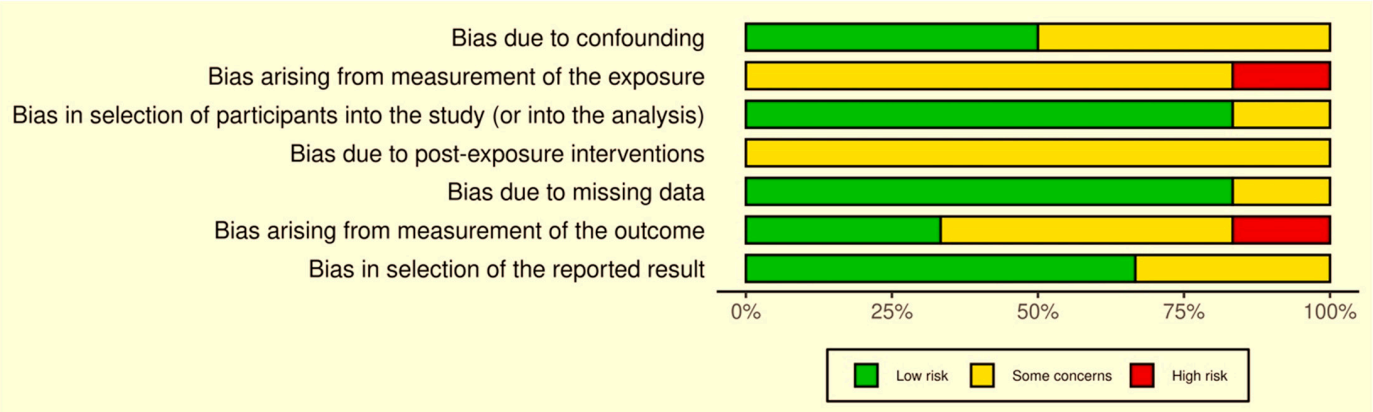

Figure 2. A weighted bar plot illustrating the distribution of the risk-of-bias judgments across different domains for the 6 studies included in this review.

4. Discussion

This systematic review investigated the association between dietary sugar intake and the risk of prostate cancer across observational studies. While individual studies reported inconsistent findings, the dominant conclusion from the overall evidence suggests that there is no statistically significant association between dietary sugar consumption and the risk of developing prostate cancer. These results suggest that dietary sugar intake, in isolation, may not be a meaningful or independent risk factor for prostate cancer, thus challenging the assumptions derived from mechanistic hypotheses that link sugar consumption to hyperinsulinemia, chronic inflammation, and tumor development [20].

In contrast to our findings, some individual studies have reported a positive association between sugar intake, particularly from SSBs and refined carbohydrates, and prostate cancer risk. For instance, a prospective analysis from the PLCO Cancer Screening Trial observed a 21% higher risk of prostate cancer among men in the highest quartile of sugar-sweetened beverage intake compared to those in the lowest (HR: 1.21; 95% CI: 1.06–1.39) [12]. Similarly, a study from the Malmö Diet and Cancer cohort linked a higher consumption of cakes, biscuits, and sugary drinks to symptomatic prostate cancer [18]. These findings support the hypothesis that certain forms of sugar, particularly rapidly absorbable carbohydrates and liquid sugars, may exert metabolic effects that influence prostate carcinogenesis [21]. However, not all studies corroborate this association. A meta-analysis by Zhai et al. [22] that included both cohort and case–control studies reported no significant association between total carbohydrate intake and prostate cancer risk (RR: 1.06; 95% CI: 0.93–1.20), which aligned with the results of this systematic review. Furthermore, another PLCO study found no relationship between dietary glycemic load, glycemic index, or total carbohydrate intake and prostate cancer incidence [23].

These discrepancies highlight the heterogeneity of the available literature and suggest that prostate cancer may be less responsive to dietary sugar exposure compared to other malignancies, such as colorectal or pancreatic cancer, where sugar intake has shown more consistent associations [24,25]. The variability observed may also be due to differences in the study designs; among the included studies, prospective cohort designs offered stronger

temporal inference but often lacked repeated dietary assessments, while case–control studies were more vulnerable to recall and selection bias. Differences in the population characteristics, dietary assessment tools, and adjustment for confounders complicate comparisons further. Moreover, the dominant focus on added or small-molecular-weight sugars (e.g., sucrose, glucose, fructose) may not capture the broader glycemic impact of total carbohydrate intake. Although this limitation is inherent to the primary data, it raises important considerations for future research methodologies, as total carbohydrates (ultimately metabolized into simple sugars) may provide a more comprehensive exposure assessment.

The absence of a consistent, statistically significant association in our review may reflect the multifactorial nature of prostate cancer, which involves complex interactions among genetic predisposition, hormonal regulation, age, and lifestyle behaviors [26–28]. Whilst sugar may contribute to cancer risk indirectly through obesity or metabolic dysfunction [29], most of the included studies adjusted for body mass index or related confounders, potentially attenuating the observed associations. Additionally, it is plausible that the metabolic consequences of sugar intake do not directly affect the prostatic tissue in the same way in which they influence other organ systems more susceptible to insulin-driven growth or chronic inflammation [30].

This systematic review offers several key strengths. First, it represents the most comprehensive synthesis to date examining the association between dietary sugar intake and prostate cancer risk, incorporating data from a broad spectrum of observational studies conducted across diverse populations. Our extensive search strategy, spanning multiple databases and implemented without language or date restrictions, maximized the inclusion of relevant studies and reduced the likelihood of selection bias. Second, this review was conducted in accordance with rigorous methodological standards, including adherence to the PRISMA 2020 guidelines and a protocol developed following the Cochrane Handbook for Systematic Reviews of Interventions, which was prospectively registered in PROSPERO. Independent screening, data extraction, and the risk of bias assessment by multiple reviewers enhanced the reliability, transparency, and reproducibility of the review process further. Third, we applied validated risk of bias tools to evaluating the study quality, offering a clear appraisal of the internal validity of the included studies. Importantly, this review addresses a timely and underexplored topic in nutritional epidemiology, generating insights that may contribute to future dietary guidance and cancer prevention efforts.

Nonetheless, certain limitations must be acknowledged. First, the majority of the included studies were observational in design, which limits establishing a causal relationship. Second, dietary sugar intake was self-reported, typically via food frequency questionnaires, which may be subject to recall bias and measurement errors. Third, inconsistencies in the definitions and quantification of sugar exposure across studies may have introduced misclassification bias. In particular, the focus on small-molecular-weight sugars, without a comprehensive assessment of total carbohydrate intake, may have limited the ability to detect broader dietary effects. Additionally, few studies stratified the outcomes by prostate cancer stage or aggressiveness, potentially obscuring the associations with specific disease subtypes. Finally, although a meta-analytic approach would have allowed for a quantitative synthesis and assessment of heterogeneity, this was not feasible due to the substantial variability in the study designs, exposures, missing data, and outcome measures across the included studies.

Future research should use standardized sugar exposure and repeated dietary assessments to improve the reliability and explore differential associations by cancer subtypes. Moreover, studies should consider the full spectrum of carbohydrate intake (including total and complex carbohydrates) and investigate their metabolic consequences in relation to prostate tissue. Longitudinal designs with sufficient statistical power and rigorous control

for confounders will be essential to clarify the potential role of dietary sugar in prostate cancer etiology.

5. Conclusions

In summary, this systematic review provides the most comprehensive synthesis to date examining the association between dietary sugar intake and prostate cancer risk and found no consistent or statistically significant association. This contributes to the current body of knowledge by challenging the assumption that dietary sugar is an independent risk factor for prostate cancer, based on the available epidemiological evidence.

While reducing sugar consumption remains vital for overall health [31], its impact on prostate cancer appears limited. Given the inconsistent findings, this review underscores the importance of improving the methodological consistency in future studies, especially regarding sugar exposure assessment, prostate cancer phenotyping, and confounder adjustment.

The findings imply that clinicians should continue to emphasize balanced dietary patterns rather than focusing solely on sugar intake for prostate cancer prevention which has been recommended in previous literature [32,33]. Advising patients to limit added sugars remains relevant to general health, but based on the current evidence, promoting sugar reduction specifically for prostate cancer prevention may not be warranted. Public health messaging should reflect this and avoid overgeneralizing the role of sugar in cancer's etiology.

Future research should include standardized, validated methods for dietary assessments, incorporate biomarkers of sugar intake, and explore gene–diet and hormone–inflammation interactions over long-term follow-up. Investigating tumor-subtype-specific effects may clarify sugar's role in prostate cancer's pathophysiology further.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/onco5030031/s1, Figure S1: Risk of Bias domains scoring for the 6 included studies.

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