Immunopathologia Persa
http immunopathol.com
DOI:10.34172/ipp.2025.43872

Association between diabetes and bladder carcinoma risk: a systematic review and meta-analysis

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Received 23 Mar. 2025
Revised: 26 Jul. 2025
Accepted 28 Jul. 2025
ePublished 9 Aug. 2025

Keywords: Urinary bladder neoplasms, Bladder tumor, Bladder cancer, Diabetes mellitus, Gastroparesis, Diabetes

Abstract

Introduction: Multiple risk factors are raised for bladder carcinoma (BCa) as the 10th most common cancer on the globe. Therefore, this study aimed to evaluate the association between diabetes and BCa through a systematic review and meta-analysis.

Materials and Methods: The sources were searched using the Web of Science, Cochrane, PubMed, Embase, and Scopus databases, as well as the Google Scholar search engine. Data were analyzed with STATA 14 at a significance level of $P<0.05$ for all statistical tests.

Results: Based on HR (1.15, 95% CI: 1.07, 1.24), SIR (1.19, 95% CI: 1.06, 1.34), and OR (2.17, 95% CI: 1.63, 2.89), the risk of BCa increased due to diabetes development. Based on RR, however, no significant relationship was observed between diabetes development and BCa (1.10, 95% CI: 0.90, 1.34). Moreover, BCa risk rose because of diabetes development in the USA, Korea, Lithuania, British Columbia, and Canada. Bladder carcinoma and diabetes were not linked significantly in case-control studies (HR: 1.69, 95% CI: 0.96, 2.97), women (HR: 1.08, 95% CI: 0.94, 1.24), and those with diabetes duration of < 5 years (HR: 1.02, 95% CI: 0.86, 1.22). However, diabetes intensified BCa risk in cohort studies (HR: 1.16, 95% CI: 1.09, 1.23), men (HR: 1.15, 95% CI: 1.06, 1.24), and those with diabetes duration of ≥ 5 years (HR: 1.14, 95% CI: 1.01, 1.27).

Conclusion: Bladder carcinoma risk was worsened by diabetes. We found that male gender and diabetes duration of ≥ 5 years were among the risk factors. Thus, it is recommended to prioritize the screening of these two factors.

Registration: This study has been compiled based on the PRISMA checklist, and its protocol was registered on the PROSPERO (ID: CRD420251004296) and Research Registry (UIN: reviewregistry1960) websites.

Citation: Maghrebi-Ghojogh E, Safari M, Ataei Azimi S, Sheikh A, Mokhtarinia B, Roohinezhad R, Jafari Arismani R, Ahmadipour E, Gandomkar H. Association between diabetes and bladder carcinoma risk: a systematic review and meta-analysis. Immunopathol Persa. 2025;x(x):e43872. DOI:10.34172/ipp.2025.43872.

Introduction

In 2020, bladder carcinoma (BCa) was recognized as the 10th most common cancer globally, with a prevalence of about 4 times in men than in women (1). Multiple risk factors, including high age, smoking, exposure to carcinogens, gender, race, etc, are mentioned for BCa (2). Owing to its high recurrence rate, BCa treatment falls under the most expensive lifelong treatments, hence it influences the healthcare system (3). Thus, primary prevention is of paramount importance to reduce the BCa load (4).

Diabetes is linked to a 17% increase in developing cancer and a 21% increase in cancer-related death (5-7). Type 2 diabetes (T2D) is the most prevalent type of diabetes that comprises almost 90% of all diabetes cases (8). It is the leading cause of stroke, blindness, myocardial infarction, and renal failure (9). Additionally, two diabetes-related causes as the insulin resistance and hyperinsulinemia may be involved in tumorigenesis (10,11). Statistics indicate that the number of diabetics has increased from 180 million people in 1980 to 529 million people in 2021 (12,13), and their number is predicted to multiply to over 100 million people in the next decade (14).

Evidence indicates that diabetes raises the chance of recurrent urinary tract infections, which may be linked to elevated BCa risk (15,16). Therefore, this study aimed to evaluate the association between diabetes and BCa through a systematic review and meta-

Key point

Diabetes elevates the risk of bladder carcinoma (BCa). Male gender and diabetes duration of ≥ 5 years were the risk factors. Therefore, it is recommended to prioritize the screening of these two factors. Additionally, diabetes development increased the risk of this cancer in the USA, Korea, Lithuania, British Columbia, and Canada. Hence, ethnicity and race may also play a role in this regard, which needs further investigation.

analysis because contradictory results were reported in previously published studies. In 2025, a survey in Lithuania revealed that diabetes amplified the BCa risk (17). In 2025, on the other hand, a study in the Netherlands disclosed no significant relationship between diabetes and the risk of BCa (18).

Materials and Methods

This study was designed using Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (19), and its protocol was registered in PROSPERO (International Prospective Register of Systematic Reviews) and research registry websites.

Search strategy

The sources were searched using the Web of Science, Cochrane, PubMed, Embase, and Scopus databases, as well as the Google Scholar search engine, up to 28 February 2025. Medical Subject Headings (Mesh) and their equivalents were used in the search strategy, and (AND, OR) operators were employed to combine keywords. The sources were also searched manually. The PECO (Population, Exposure, Comparison, Outcomes) was the search strategy in the Web of Science database: Urinary Bladder Neoplasms OR Bladder Tumor OR Bladder Cancer (Title) AND Diabetes Mellitus OR Gastroparesis OR Diabetes (Title)

The study population consisted of studies that investigated the association between diabetes and BCa. The exposure was diabetes development. The comparison group included non-diabetics. The main outcome was investigating the relationship between diabetes and the risk of BCa.

Inclusion criterion

Observational studies that examined the connection between diabetes and BCa.

Exclusion criteria

Non-observational studies, letters to the editor-in-chief, repeated studies, studies with poor quality, review studies, studies without accessible full texts and with incomplete abstracts, studies lacking required data for analysis, and abstracts published in conferences.

Quality assessment

Two authors assessed the quality of studies with the Newcastle Ottawa Scale, in which each question was assigned one star at maximum (except for the comparison question to which two stars could be assigned). Thus, the minimum and maximum scores were zero and 10, respectively (20).

Data extraction

Data such as the author's name, indicator, study type, age, the link between diabetes and BCa with a confidence interval of 95%, country, duration of diabetes development, comparison group, year, and sample size were extracted by two authors.

Statistical analysis

Data were analyzed using odds ratio (OR), hazard ratio (HR), relative risk (RR), standardized incidence ratio (SIR), and the studies were combined together. The heterogeneity between the studies was evaluated using the I^2 indicator. The fixed effect and random effect models were used in conditions with low and high heterogeneity, respectively. Data were analyzed with STATA 14 at a significance level of $P < 0.05$ for all statistical tests.

Results

In total, 751 articles were retrieved at the search stage, among which 398 repeated studies were excluded from the study. After reviewing the abstracts, 14 studies without full texts were omitted from this meta-analysis. Of 339 remaining articles, 197 studies lacking the required data for analysis were removed from this review. Of 142 remaining articles, 125 studies were excluded based on exclusion criteria, and 17 articles were included in this study (Figure 1).

Of 17 reviewed studies, 13 and 4 articles were cohort and case-control, respectively (Table 1).

As shown in Figure 2, diabetes development increased the risk of BCa based on HR (1.15, 95% CI: 1.07, 1.24), SIR (1.19, 95% CI: 1.06, 1.34), and OR (2.17, 95% CI: 1.63, 2.89) indicators. Based on RR, however, no significant relationship was found between diabetes development and BCa (1.10, 95% CI: 0.90, 1.34).

Figure 3 shows no significant association between diabetes development and BCa in Italy, Taiwan, UK, Sweden, and the Netherlands. However, BCa risk was amplified because of diabetes development in the USA, Korea, Lithuania, British Columbia, and Canada.

BCa and diabetes were not significantly associated in case-control studies (HR: 1.69, 95% CI: 0.96, 2.97) whereas diabetes escalated BCa risk in cohort studies (HR: 1.16, 95% CI: 1.09, 1.23) (Figure 4).

Diabetes development and BCa were not related significantly in women (HR: 1.08, 95% CI: 0.94, 1.24). In men, however, the risk of BCa was multiplied by diabetes development (HR: 1.15, 95% CI: 1.06, 1.24) (Figures 5 and 6). No significant relationship was detected between diabetes development and BCa (HR: 1.02, 95% CI: 0.86, 1.22) in diabetics with diabetes duration of <5 years.

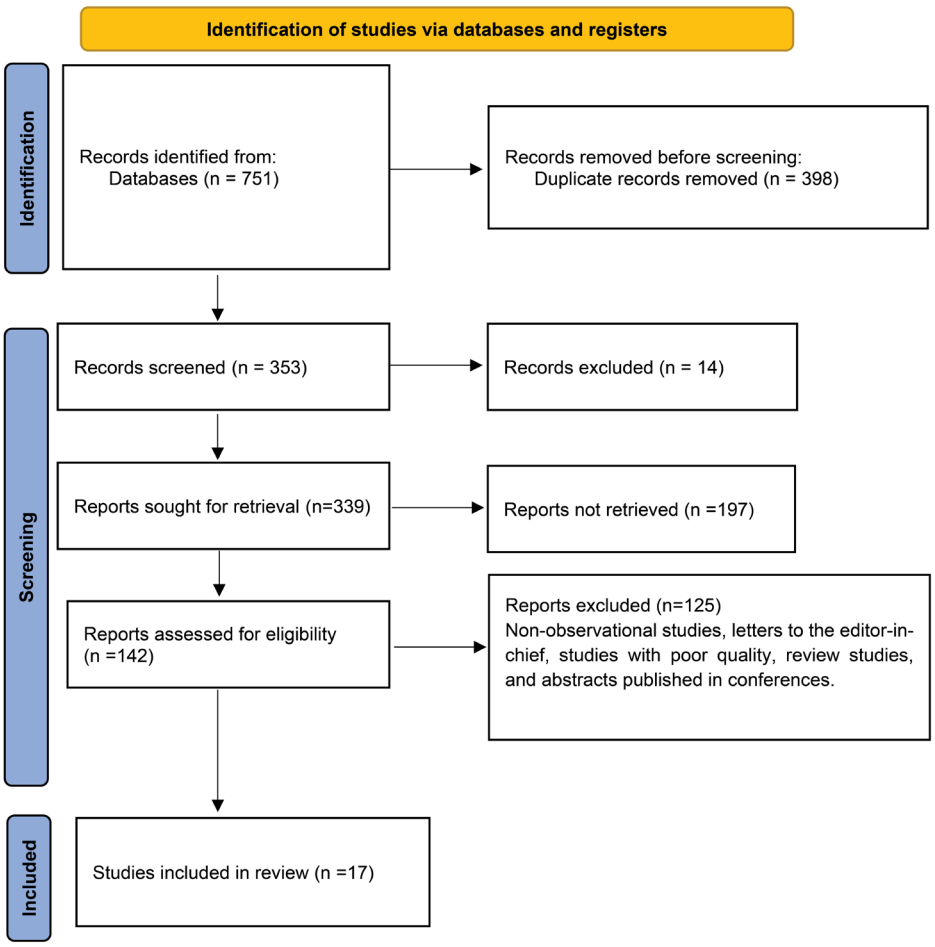

Figure 1. The PRISMA flowchart of study selection.

Table 1. Characteristics of studies

Index	Author, year	Location	Design	Duration of study	Sample size	Age (year)
SIR	Ladukas A, 2025 (17)	Lithuania	Cohort	2001-2012	76818	≥40
HR	Van den Brandt PA, 2024 (18)	Netherlands	Cohort	NR	120852	55-69
HR	Bogumil D, 2023 (21)	USA	Cohort	1993-1996	185059	45-75
HR	Choi YH, 2022 (22)	Korea	Cohort	2009	NR	NR
HR	Pan Y, 2020 (23)	Taiwan	Cohort	1997-2013	NR	NR
HR	Li Y, 2020 (24)	USA	Cohort	1993-1998	NR	NR
OR	Turati F, 2015 (25)	Italy	Case-Control	2003-2014	NR	NR
HR	Goossens ME, 2015 (26)	UK	Cohort	1987-2013	NR	NR
HR	Colmers IN, 2013 (27)	British, Columbia, Canada	Cohort	1996-2006	185100	60.7
RR	Newton CC, 2013 (28)	USA	Cohort	1992-2007	NR	NR
HR	Prizment AE, 2013 (29)	USA	Cohort	1986-2010	NR	NR
OR	MacKenzie T, 2011 (30)	USA	Case-Control	1998-2001	NR	NR
RR	Tseng CH, 2011 (31)	Taiwan	Cohort	2003-2005	1000000	NR
RR	Larsson SC, 2008 (32)	Sweden	Cohort	1998-2007	45906	45-79
OR	Ng Y, 2003 (33)	UK	Case-Control	2000-2001	NR	NR
RR	La Vecchia C, 1994 (34)	Italy	Case-Control	1983-1992	431	75<
HR	Ko SH, 2019 (35)	Korea	Cohort	Jan. 1, 2009-Dec. 31, 2009	402752	56.26

NR: Not reported; OR: Odds ratio; HR: Hazard ratio; RR: Relative risk; SIR: Standardized incidence ratio.

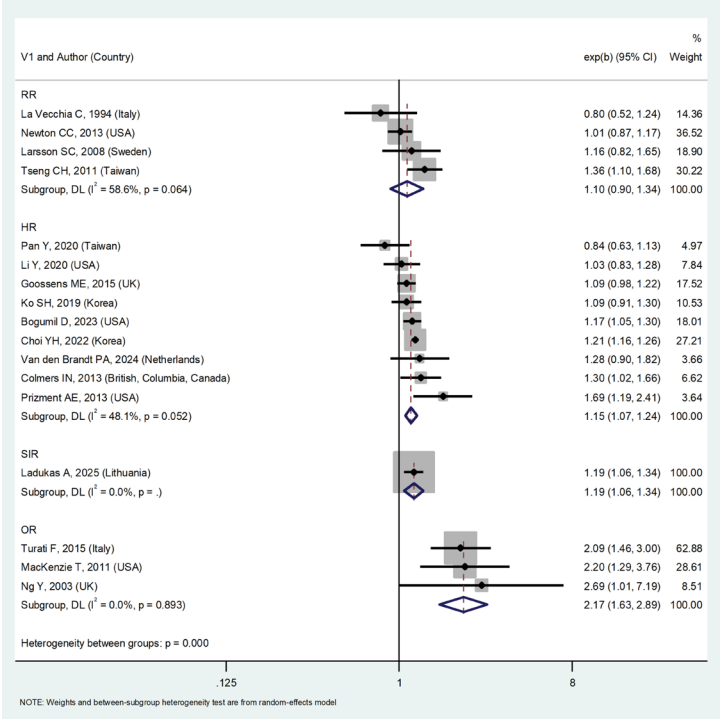

Figure 2. Forest plot showing the association between diabetes and risk of bladder carcinoma by index.

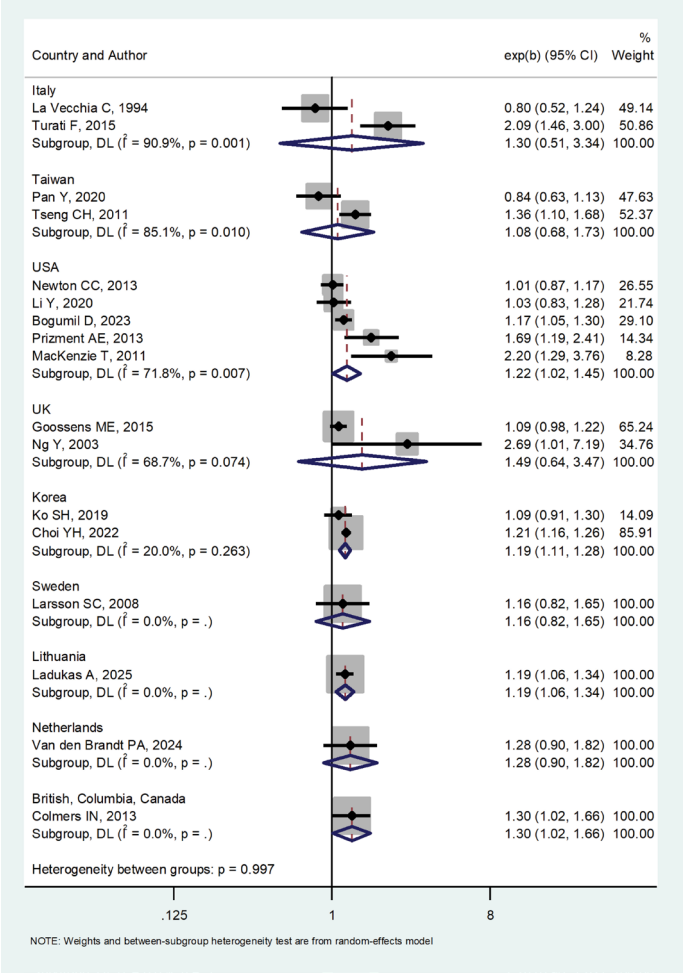

Figure 3. FForest plot showing the association between diabetes and risk of bladder carcinoma by location.

Nonetheless, the risk of BCa rose (HR: 1.14, 95% CI: 1.01, 1.27) in diabetics with diabetes duration of ≥5 years (Figures 7 and 8).

Figure 9 depicts no publication bias in this study ($P = 0.656$), hence the sources were searched comprehensively.

Discussion

Based on HR, SIR, and OR, diabetes was a risk factor for BCa. Diabetes elevated the risk of BCa in the USA, Korea, Lithuania, British Columbia, and Canada. BCa risk was intensified by diabetes development in cohort studies (16%), men (15%), and those with diabetes duration of ≥ 5 years (14%).

The results of a meta-analysis showed that diabetes elevated the risk of BCa compared to non-diabetics (RR: 1.23, 95% CI: 1.12, 1.35). In addition, diabetes-induced BCa risk rose in men (RR: 1.23, 95% CI: 1.06, 1.42) contrary to women (RR: 1.24, 95% CI: 0.95, 1.61) (36). Although their

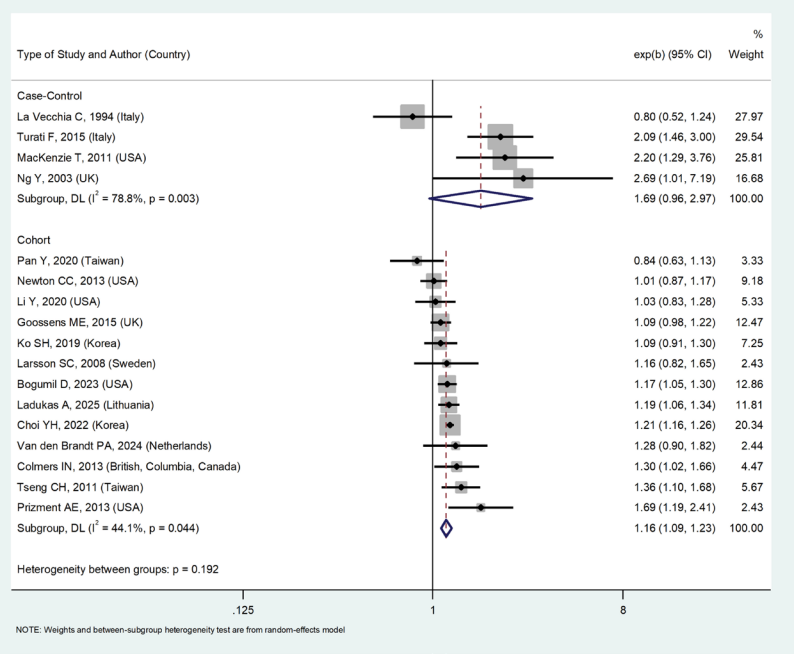

Figure 4. Forest plot showing the association between diabetes and risk of bladder carcinoma by design.

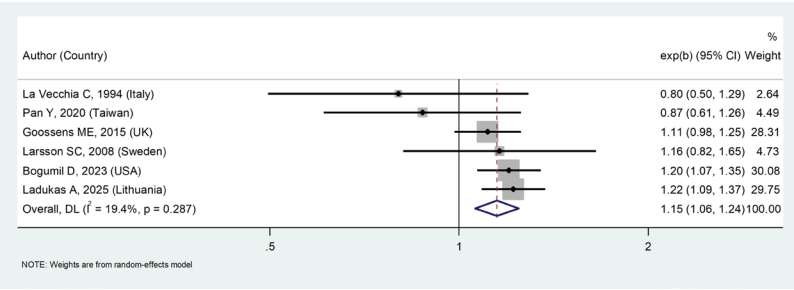

Figure 5. Forest plot showing the association between diabetes and risk of bladder carcinoma in males.

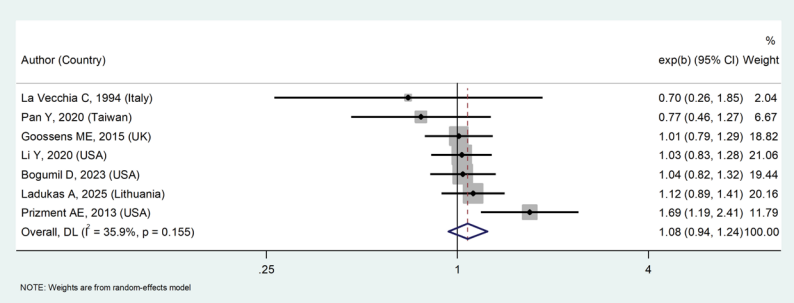

Figure 6. Forest plot showing the association between diabetes and risk of bladder carcinoma in females.

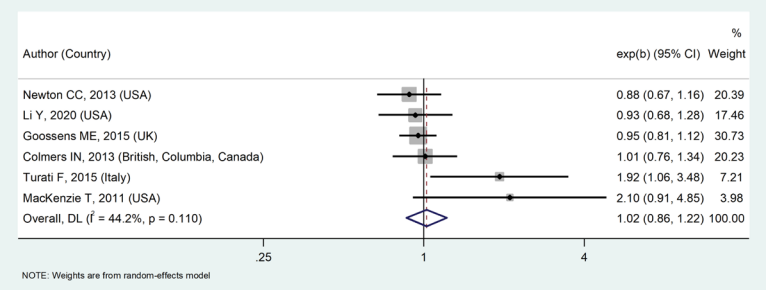

Figure 7. Forest plot showing the association between diabetes and risk of bladder carcinoma in duration <5 year.

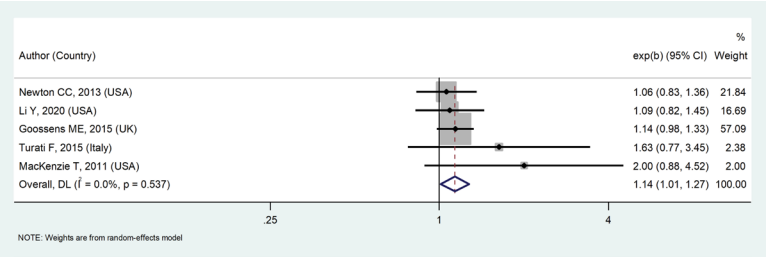

Figure 8. Forest plot showing the association between diabetes and risk of bladder carcinoma in duration ≥5

results agree with those of the present study, our results are more up-to-date because of reviewing recently published studies (2017 onward).

Compared to non-diabetics, diabetes was associated with increased risk of BCa meta-analyses by Xu et al (RR: 1.11, 95% CI: 1, 1.23) (37), Zhu et al (RR: 1.35, 95% CI 1.17, 1.56) (38), Yang et al (OR: 1.68, 95% CI: 1.32, 2.13) (39), Larsson et al (RR: 1.24, 95% CI: 1.08, 1.42) (40), and another meta-analysis by Zhu et al on cohort studies (RR: 1.29, 95% CI: 1.08, 1.54) (41). The meta-analyses mentioned above correspond to the current meta-analysis, suggesting that diabetes is a risk factor for BCa development.

In a meta-analysis by Fang et al, diabetes was linked to heightened BCa risk (RR: 1.30, 95% CI: 1.18, 1.43). Furthermore, diabetes raised the risk of BCa in analyzing a subgroup in women (RR: 1.23, 95% CI: 1.02, 1.49) whereas no statistically significant relationship was observed in men (RR: 1.07, 95% CI: 0.97, 1.18) (42). In a cohort study by van den Brandt et al, T2D was related to an elevated risk of BCa development (HR: 1.57, 95% CI: 1.04, 2.37). This association was only significant in women (HR: 2.19, 95% CI: 1.10, 4.34) but not in men (HR: 1.42, 95% CI: 0.88, 2.30) (18). The overall results of these studies correspond to our study. However, the aforementioned studies contrast our survey in terms of gender because they reported that female gender was a risk factor for BCa development in diabetics compared to the opposite result in our review.

A cohort study by Ladukas et al revealed that T2D was connected to an amplified risk of BCa (SIR: 1.19, 95% CI: 1.06, 1.34) (17). Similarly, T2D was directly associated with BCa (HR: 1.17, 95% CI: 1.05, 1.30) in a cohort study by Bogumil et al (21). Likewise, our subgroup analysis indicated that diabetes development increased BCa risk in cohort studies.

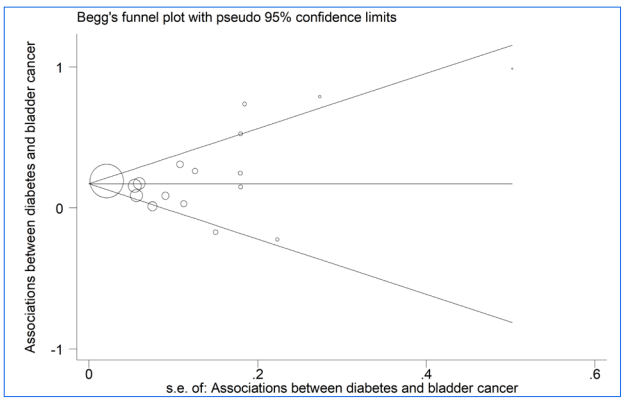

Figure 9. Diagram of publication bias.

Conclusion

Diabetes elevates the risk of BCa. Male gender and diabetes duration of ≥5 years were the risk factors. Therefore, it is recommended to prioritize the screening of these two factors. Additionally, diabetes development increased the risk of BCa in the USA, Korea, Lithuania, British Columbia, and Canada. Hence, ethnicity and race may also play a role in this regard, which needs further investigation.

Limitations of the study

The sample size was not reported in several studies, making it impossible to determine the total number of examined subjects in this meta-analysis. Moreover, the type of diabetes and the mean age of patients were not specified in most studies, preventing subgroup analyses based on these criteria. Besides, the studies were not equally distributed **across** various regions.

Acknowledgments

The authors would like to thank Hosein Mardanparvar (Guissu Research Corporation) and Diana Sarokhani for guidance and editing manuscript registration on the PROSPERO and research registry websites.

Authors' contribution

Conceptualization: Ehsan Maghrebi-Ghojogh.
Data curation: Hossein Gandomkar and Sajad Ataei Azimi.
Formal analysis: Maede Safari and Bahman Mokhtarinia.
Investigation: Ameneh Sheikh and Roozbeh Roohinezhad.
Methodology: Maede Safari and Bahman Mokhtarinia.
Project management: Hossein Gandomkar.
Supervision: Ehsan Maghrebi-Ghojogh.
Validation: Ameneh Sheikh and Rasoul Jafari Arismani.
Visualization: Rasoul Jafari Arismani and Elham Ahmadipour.
Writing–original draft: All authors.
Writing–review and editing: All authors.

Conflicts of interest

There are no competing interests.

Ethical issues

This study has been compiled based on the PRISMA checklist, and its protocol was registered on the PROSPERO website (ID: CRD420251004296) and Research Registry website (Unique Identifying Number (UIN) reviewregistry1960). Furthermore, the authors have fully adhered to ethical standards, including avoiding plagiarism, data fabrication, and double publication.

Funding/Support

No funding.

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