



Review Article

Association between smoking cessation and risk for type 2 diabetes, stratified by post-cessation weight change: A systematic review and meta-analysis

Yifan Yu^{a,*}, Yan Li^a, Thu T. Nguyen^a, Dahai Yue^b, Nedelina Tchangalova^c, Caitlin E. Flouton^a, Hongjie Liu^a

^a Department of Epidemiology and Biostatistics, School of Public Health, University of Maryland, College Park, MD, USA

^b Department of Health Policy and Management, School of Public Health, University of Maryland, College Park, MD, USA

^c The Science, Technology, Engineering, and Mathematics Library, University of Maryland, College Park, MD, USA

ARTICLE INFO

Keywords:

Tobacco cessation
Smoking-related diabetes
Post-cessation weight change
Diabetes risk
Weight gain
Risk factors

ABSTRACT

Objective: While smoking cessation reduces health risks, its impact on type 2 diabetes mellitus (T2DM) remains complex when considering post-cessation weight gain. This systematic review and meta-analysis examined the association between smoking cessation and diabetes risk stratified by weight change and cessation duration.

Methods: We searched seven databases through April 14, 2025. Observational studies examining smoking cessation, weight changes, and T2DM were included. Random-effects models pooled hazard ratios (HRs) comparing recent and long-term quitters to continuous/never smokers, stratified by weight gain.

Results: Among eleven cohort studies, quitters with weight gain showed increased diabetes risk versus continuous smokers (HR = 1.71, 95 % CI: 1.12, 2.62), with recent quitters having greater risk (HR = 2.20, 95 % CI: 1.27, 3.82) but long-term quitters showing reduced risk (HR = 0.91, 95 % CI: 0.87, 0.95). Quitters without weight gain demonstrated no increased risk (recent: HR = 0.99, 95 % CI: 0.81, 1.02) and lower risk (long-term: HR = 0.84, 95 % CI: 0.81, 0.87). Compared to never-smokers, recent quitters had a higher T2DM risk regardless of weight status (with gain: HR = 1.61, 95 % CI: 1.03, 2.50; without gain: HR = 1.25, 95 % CI: 1.05, 1.48), while long-term quitters showed no significant difference.

Conclusions: Smoking cessation temporarily increases T2DM risk, particularly with weight gain, but becomes protective long-term, emphasizing weight management.

1. Introduction

Smoking is an established risk factor for diabetes, with Mendelian randomization studies supporting a causal relationship (Yuan and Larsson, 2019). While smoking cessation reduces diabetes risk (Centers for Disease Control and Prevention, 2024), quitting often leads to weight gain (Tian et al., 2015; Aubin et al., 2012) due to increased appetite and decreased energy expenditure (Harris et al., 2016). Quitters typically gain 4.1–8.8 kg over several years (Bush et al., 2016). Weight gain significantly increases the risk of type 2 diabetes mellitus (T2DM) (Resnick et al., 2000; Ross et al., 2011), with a gain of ≥ 5 kg over 10 years associated with 52 % increased risk (Hu et al., 2018), rising to 96

% among those already overweight (Owora et al., 2022). This raise concerns that post-cessation weight gain might offset smoking cessation's metabolic benefits.

Previous cohort studies examining smoking cessation and diabetes risk typically used never smokers as controls, with few considering weight change (Akter et al., 2017; Pan et al., 2015; Sung et al., 2016; Yeh, 2010; Wannamethee et al., 2001; Luo et al., 2012). Hu et al. (Hu et al., 2018) found that quitting smoking lowers diabetes risk compared to continuing smoking, and this relationship holds regardless of whether the quitter gains weight or not. However, previous studies remain varied and inconclusive (Hu et al., 2018; Owora et al., 2022; Yeh, 2010; Luo et al., 2012; Bar-Zeev et al., 2020; Choi et al., 2021; Maddatu et al.,

Abbreviations: BMI, body mass index; CI, confidence interval; HR, hazard ratios; NOS, Newcastle-Ottawa scale; OR, odds ratio; SE, standard error; T2DM, type 2 diabetes mellitus.

* Corresponding author at: School of Public Health, University of Maryland, College Park, Maryland 20742-2611, USA.

E-mail address: yyu59@umd.edu (Y. Yu).

<https://doi.org/10.1016/j.ypmed.2025.108429>

Received 26 July 2025; Received in revised form 8 October 2025; Accepted 9 October 2025

Available online 10 October 2025

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2017). Some studies have found cessation that is linked to a lower diabetes risk, regardless of weight gain (Choi et al., 2021), while others have found no correlation (Hu et al., 2018; Luo et al., 2012; Sahle et al., 2021). The discrepancy may be due to varying sample sizes, weight gain cutoffs, and participant characteristics. Notably, Hu et al. (Hu et al., 2018) found a 22 % lower diabetes risk among quitters despite 2–5 kg weight gain, which contrast with Luo et al. (Luo et al., 2012), who found that weight gain did not influence the cessation-diabetes relationship.

Given these contradictory findings and absence of comprehensive synthesis, we aim to 1) compare diabetes risk between quitters (with/without weight gain) and continuous smokers, 2) assess diabetes risk between quitters (with/without weight gain) and never smokers, and 3) evaluate how cessation duration modifies these relationships when stratified by weight change status.

2. Methods

2.1. Systematic review and meta-analysis registration

This systematic review and meta-analysis protocol was registered in PROSPERO: CRD42023464859 (Yu and Tchangalova, 2023).

2.2. Eligibility criteria

Studies were included if they met the following criteria: (1) observational study design (prospective or retrospective cohort, case-control, or cross-sectional studies); (2) for cohort studies, adult population (≥ 18 years) without diabetes at baseline; for case-control and cross-sectional studies, adult population (≥ 18 years); (3) smoking cessation as the primary exposure; (4) comparison groups of either never smokers or continuous smokers; (5) type 2 diabetes as the outcome; and (6) for cohort studies, reported risk estimates with 95 % confidence intervals (CI) or standard error (SE) for the association between smoking cessation and diabetes risk in the context of post-cessation weight change; for case-control and cross-sectional studies, reported risk estimates with 95 % CIs for the association between smoking cessation and diabetes. We excluded studies involving non-human subjects, participants with baseline diabetes, randomized clinical trials, qualitative studies, and review articles. All studies included in this systematic review and meta-analysis had institutional ethical approval, and no additional approval was required for analyzing data from published articles.

2.3. Information sources

Our study was conducted in accordance with the Meta-analysis of Observational Studies in Epidemiology guidelines (Stroup, 2000) and the Preferred Reporting Items for Systematic Reviews and Meta-analyses statement (Page et al., 2021). We searched seven electronic databases from inception through April 14, 2025: Academic Search Ultimate, APA PsycINFO, Cumulative Index of Nursing & Allied Health, Health Source: Nursing/Academic Edition, Medline (all through EBSCO), Scopus (Elsevier), and Web of Science (Clarivate).

2.4. Search strategy

The search strategy was structured around three main concepts, guided by the Population, Exposure, Comparison, and Outcome framework: (1) smoking cessation terms (cigarettes, nicotine, smoking, tobacco combined with abstinence, cessation, quitting, stopping, reduction); (2) weight-related terms (weight change, gain, loss, obesity, overweight, body weight); and (3) diabetes outcomes (diabetes mellitus, type 2 diabetes, glycemic control, T2DM). It was adapted for each database's syntax and controlled vocabulary (Supplementary Table S1). The search was restricted to published, peer-reviewed articles in English. Reference lists of included studies were manually screened to identify additional relevant articles.

2.5. Selection process

The public health librarian (NT) searched databases, compiled the records in Zotero for de-duplication, and exported unique records in Rayyan for screening. Two reviewers (YY, CF) independently screened titles and abstracts for eligibility, followed by a full-text review of potentially eligible studies. Discrepancies were resolved through discussion and, when necessary, in consultation with a third subject matter expert (HL). Reasons for exclusion at the full-text stage were documented in Supplementary Table S2.

2.6. Data collection process and data items

Using a standardized form in Microsoft Excel, one reviewer (YY) independently extracted the data, and a second reviewer (CF) verified its accuracy. The extracted data included the following information: (1) publication details (first author, year); (2) study characteristics (design, location, study name, target population, follow-up duration); (3) participant characteristics (sample size, age, sex, race/ethnicity); (4) smoking assessment methods; (5) weight change assessment method and weight change classification criteria; (6) diabetes outcome assessment methods; (7) statistical approaches; and (8) adjusted risk estimates with 95 % confidence intervals, including details of covariate adjustment. Disagreements were resolved through consensus with the review team and a third expert (HL). The final dataset was imported into Review Manager 5.4 for analysis (Cochrane, 2020).

2.7. Quality assessment of the included studies

Study quality was evaluated using the Newcastle-Ottawa Scale (NOS) for cohort studies (Wells et al., 2025). The NOS comprises nine criteria across three domains: selection (maximum four stars), comparability (maximum two stars), and outcome (maximum three stars). Total scores ranged from zero to nine stars, with higher scores indicating better methodological quality. Studies were classified as high quality (≥ 6 stars), moderate quality (four to five stars), or low quality (zero to three stars).

2.8. Statistical analysis

For meta-analysis, we combined hazard ratios (HRs) and odds ratios (ORs) as a single HR. This approach was methodologically appropriate, given relatively low diabetes incidence across follow-up periods (< 10 % in most studies), where HRs and ORs provide approximately equivalent estimates (VanderWeele, 2020). For weight gain categorization, we selected estimates from the highest weight gain category for the “with weight gain” group. When studies presented separate estimates for weight loss and no weight change, we used no weight change estimates for the “without weight gain” category, as these groups typically comprised larger populations. For studies reporting separate risk estimates for recent and long-term quitters, we applied recent quitter estimates in the primary analysis to maintain consistency and capture immediate cessation effects, as we were primarily interested in weight gain effects occurring shortly after cessation. We then conducted duration-stratified analyses using both recent and long-term estimates.

For subgroup analyses by cessation duration, studies were categorized into recent and long-term quitter groups. Studies not specifying cessation duration were classified as long-term quitters to avoid overestimating acute effects, as supported by most studies having an average/median follow-up exceeding six years.

Statistical heterogeneity was assessed using Cochran's Q test ($p < 0.10$) and quantified using I^2 statistics (Higgins, 2003). Random-effects models were employed when significant heterogeneity was detected ($p < 0.10$ or $I^2 > 50$ %); otherwise, fixed-effects models were used. Chi² test ($p < 0.05$) was used to test subgroup differences (DerSimonian and Laird, 1986). Publication bias was evaluated using Egger's test ($p <$

0.10) (Lin and Chu, 2018) and trim-and-fill analysis due to the limited number of studies (Shi and Lin, 2019).

All meta-analyses were performed using Review Manager 5.4 (Cochrane, 2020), which generated forest plots displaying both individual and pooled effect estimates, along with 95 % confidence intervals. Statistical significance was set at $p < 0.05$ (two-tailed) unless otherwise specified.

2.9. Sensitivity analysis

We repeated meta-analyses excluding the largest sample study to assess whether this administrative database study substantially influenced pooled estimates. We then excluded the pregnancy study to determine whether this specific population affected overall results. Finally, we performed analyses restricted to high-quality studies (NOS scores ≥ 6 stars) to examine whether methodological rigor influenced findings. All sensitivity analyses were conducted separately for comparisons between continuous smokers and never smokers, and for groups with and without weight gain, where data permitted.

3. Results

3.1. Study selection

The database search, supplemented by a Google Scholar search,

yielded 5032 records. After removing 2522 duplicate records and three retracted articles, 2507 unique records remained for screening. During the title/abstract screening, 2472 records were excluded, leaving 35 studies for full-text review (Fig. 1). No case-control or cross-sectional studies met the inclusion criteria after full-text review, resulting in 11 cohort studies for final inclusion.

3.2. Characteristics of included studies

Table 1 presents the characteristics of the eleven included cohort studies published between 2001 and 2024. Among them, five studies were conducted in Asia (Choi et al., 2021; Oba et al., 2012; Park et al., 2021; Wu et al., 2022; Xie et al., 2024), four in the United States (Hu et al., 2018; Yeh, 2010; Luo et al., 2012; Bar-Zeev et al., 2020), one in Australia (Sahle et al., 2021), and one in the United Kingdom (Wannamethee et al., 2001). Sample sizes ranged from 6559 (Xie et al., 2024) to 5,198,792 (Park et al., 2021), with average follow-up durations ranging from 3.7 years (Sahle et al., 2021) to 19.6 years (Hu et al., 2018). All studies relied on self-reported smoking status assessments. The mean participant age ranged from approximately 43 to 59 years. Five studies focused on single-gender populations: three on exclusively male cohorts (Wannamethee et al., 2001; Oba et al., 2012; Xie et al., 2024) and two on exclusively female cohorts (Luo et al., 2012; Bar-Zeev et al., 2020). Five studies were conducted in Asian populations (Choi et al., 2021; Oba et al., 2012; Park et al., 2021; Wu et al., 2022; Xie et al.,

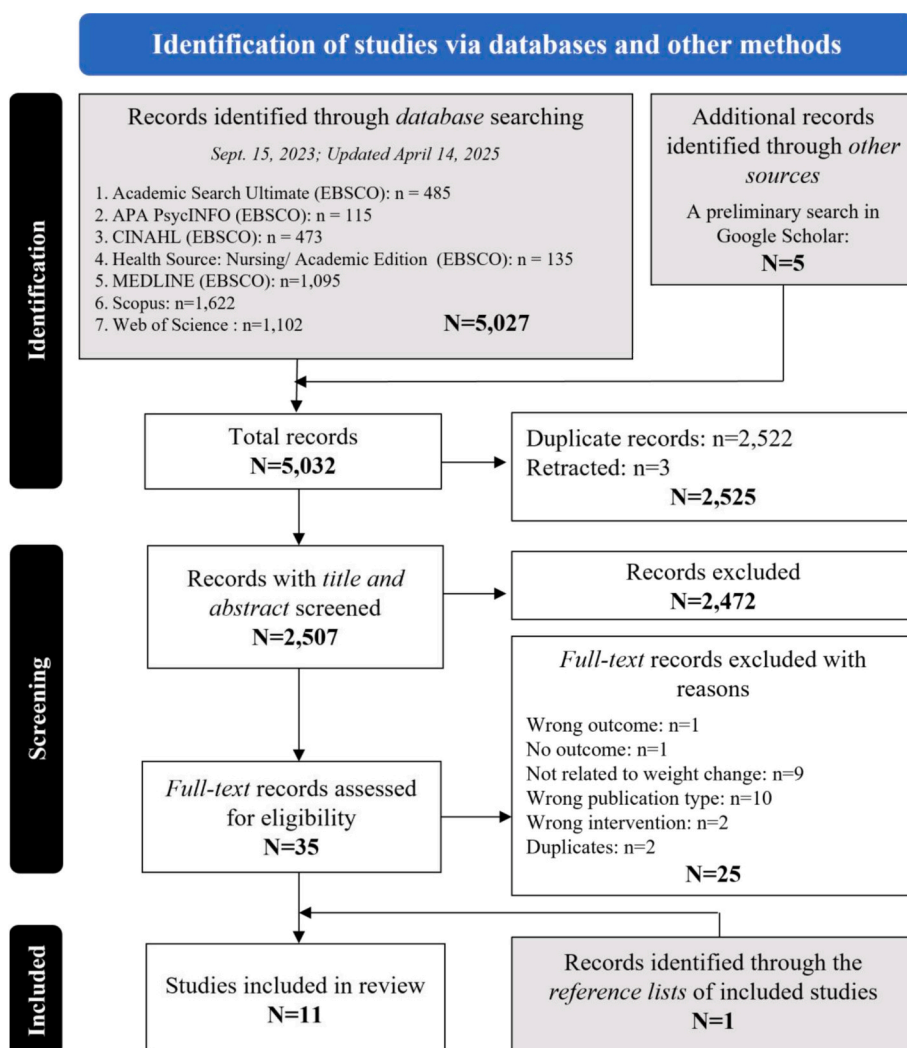


Fig. 1. Study selection flowchart for systematic review and meta-analysis of smoking cessation and type 2 diabetes risk.

Table 1
 Characteristics of eleven cohort studies examining smoking cessation and type 2 diabetes mellitus risk across Asia, Australia, Europe, and North America (2001–2024).

Authors	Year	Study design	Total population	Study location	Study name	Target population	Age (mean/SD)	% of males	Majority race	Smoking assessment	Follow-up period	Follow-up duration ^a	Weight assessment method	Weight gain classification ^b	Outcome assessment	Event (n) ^c	Total quality scores ^d
Bar-Zeev et al. (Bar-Zeev et al., 2020)	2020	Prospective cohort	222,408	US	Pregnancy risk assessment monitoring system	Pregnant women	25–34	0 %	Non-Hispanic white	Self-reported	2009–2015	NA	Self-reported	Inadequate, Normal, excessive	Self-reported	12,897	5 stars
Choi et al. (Choi et al., 2021)	2021	Prospective cohort	96,524	Korea	NSC	Korean adults aged 20+ without T2DM or hypertension at baseline	42.9 (13.1)	52 %	Asian	Self-reported	2006–2015	6.9	Measured	No weight gain, gain 0.1–5.0 kg, gain 5.1–10.0 kg, gain 10 kg	Laboratory test/medical record/treatment record	18,852	7 stars
Hu et al. (Hu et al., 2018)	2018	Prospective cohort	162,807	US	NHS, NHS II, HPFS	Health professionals	NA	NA	White	Self-reported	1984–2013	19.6	Self-reported	No weight gain, weight gain 0.1–5.0 kg, weight gain 5.1–10.0 kg, weight gain more than 10.0 kg	Self-reported with a validated supplementary questionnaire	2585	6 stars
Luo et al. (Luo et al., 2012)	2012	Prospective cohort	115,092	US	WHI	Postmenopausal women	NA	0 %	NA	Self-reported	NA	8.5	Measured	Weight gain ≥5 kg, weight gain <5 kg	Treatment record/self-reported	11,056	6 stars
Oba et al. (Oba et al., 2012)	2012	Prospective cohort	25,875	Japan	JPHC	Residents aged 40–69	56.5 (7.7)	100 %	Asian	Self-reported	1995–2005	NA	Self-reported	Weight gain ≥3	Self-reported and medical records	1100	6 stars
Park et al. (Park et al., 2021)	2021	Prospective cohort	5,198,792	Korea	NHIS database	Korean residents aged 20+ who had a medical evaluation by NHIS	48.8 (NA)	46.9 %	Asian	Self-reported	2009–2016	4.87	Health check-up	Weight change <−0.5 %, weight change −5–5 %, weight change ≥5 %	Treatment record and ICD-10 codes	164,335	6 stars
Sahle et al. (Sahle et al., 2021)	2021	Prospective cohort	16,663	Australia	HILDA survey	Australian adults	43.7 (16.3)	48.5 %	NA	Self-reported	2006–2014	3.7	Self-reported	Lost weight, no change, gained 0.1–5.0 kg, gained 5.1–10.0 kg, gained more than 10.0 kg	Self-reported	865	6 stars
Wannamethee et al. (Wannamethee et al., 2001)	2001	Retrospective cohort	7735	UK	British regional heart study	Men aged 40–59 from general practices	NA	100 %	NA	Self-reported	NA	16.8	Measured at baseline, self-report at follow-up	Weight loss >4 %, weight stable (−4 %–4 %), weight gain >4 %–10 %, weight gain >10 %	Self-reported and confirmed in the medical record	290	6 stars
Wu et al. (Wu et al., 2022)	2022	Prospective cohort	8951	China	CHARLS	Chinese residents over 45 years old	58.7 (NA)	48.5 %	Asian	Self-reported	2011–2015	NA	Self-reported	Weight gain <2 kg, weight gain ≥2 kg	Self-reported /laboratory test	712	7 stars

(continued on next page)

Table 1 (continued)

Authors	Year	Study design	Total population	Study location	Study name	Target population	Age (mean/SD)	% of males	Majority race	Smoking assessment	Follow-up period	Follow-up duration ^a	Weight assessment method	Weight gain classification ^b	Outcome assessment	Event (n) ^c	Total quality scores ^d
Yeh et al. (Yeh, 2010)	2010	Prospective cohort	9398	US	ARIC	Adults aged 45 to 64 years in 4 U.S. communities	53.8 (NA)	43.3 %	White	Self-reported	1987–2004	NA	Measured	Weight gain \leq 4KG, weight gain $>$ 4 KG, self-reported	Laboratory test/ treatment record/ self-reported	1254	7 stars
Xie et al. (Xie et al., 2024)	2024	Prospective cohort	6559	China	NA	Adults who attended annual health checkups at Zhenhai Lianhua hospital	44.8 (12.7)	100 %	Asian	Self-reported	2007–2014	NA	Health check-up	Weight gain $<$ 0 kg, weight gain 0–1.9 kg, weight gain 2.0–3.9 kg, weight gain \geq 4.0 kg	Laboratory test/ self-reported	363	6 stars

Abbreviations: CHARLS, China Health and Retirement Longitudinal Study; HILDA, Household, Income and Labor Dynamics in Australia; HPFS, Health Professionals Follow-up Study; ICD-10, International Classification of Diseases, 10th Revision; JPHC, Japan Public Health Center-Based Prospective Study; NA, not available; NHIS, National Health Insurance Service; NHS, Nurses' Health Study; NSC, NHIS-Nation Sample Cohort; WHI, Women's Health Initiative; SD, standard deviation; T2DM, type 2 diabetes mellitus; US, United States; UK, United Kingdom.

^a Follow-up duration is presented in years as median or mean, as reported in original studies.

^b Weight change classifications are presented as defined in the original studies.

^c Events (n) represent the number of outcome events during follow-up.

^d Quality scores were assessed using the Newcastle-Ottawa scale (range: 0–9, with higher scores indicating better quality).

2024), and two studies reported a predominantly white population (Yeh, 2010; Bar-Zeev et al., 2020). The remaining studies did not specify racial composition.

Weight measurement varied across studies: five used self-reported weight (Hu et al., 2018; Bar-Zeev et al., 2020; Sahle et al., 2021; Oba et al., 2012; Wu et al., 2022), four employed measured weight (Yeh, 2010; Luo et al., 2012; Choi et al., 2021; Park et al., 2021), one used both methods (Wannamethee et al., 2001), and one study did not report the weight measurement (Xie et al., 2024). Weight gain classifications included absolute weight gain categories (e.g., 0.1–5.0 kg, 5.1–10.0 kg, >10.0 kg) (Hu et al., 2018; Choi et al., 2021; Sahle et al., 2021; Xie et al., 2024), percentage changes (e.g., $<$ –5 %, –5–5 %, \geq 5 %) (Park et al., 2021), and binary cutoffs (e.g., \geq 2 kg (Wu et al., 2022); \geq 3 kg (Oba et al., 2012); \geq 4 kg (Yeh, 2010); \geq 5 kg (Luo et al., 2012)). Diabetes assessment methods included self-reported (Hu et al., 2018; Bar-Zeev et al., 2020; Sahle et al., 2021; Wu et al., 2022), medical records/laboratory tests (Choi et al., 2021; Park et al., 2021), and combined methods (Yeh, 2010; Wannamethee et al., 2001; Luo et al., 2012; Oba et al., 2012; Xie et al., 2024). The number of incident diabetes cases ranged from 290 (Wannamethee et al., 2001) to 164,335 (Park et al., 2021) (Table 1).

Among the eleven included studies, eight applied Cox proportional hazards regression models (Hu et al., 2018; Yeh, 2010; Wannamethee et al., 2001; Luo et al., 2012; Choi et al., 2021; Sahle et al., 2021; Park et al., 2021; Xie et al., 2024), and three applied logistic regression (Bar-Zeev et al., 2020; Oba et al., 2012; Wu et al., 2022) (Supplementary Table S3). Of the ten studies reporting covariate information (Hu et al., 2018; Yeh, 2010; Wannamethee et al., 2001; Luo et al., 2012; Bar-Zeev et al., 2020; Choi et al., 2021; Sahle et al., 2021; Oba et al., 2012; Park et al., 2021; Wu et al., 2022), all adjusted for age and baseline body mass index (BMI) or BMI categories. One study did not provide detailed information on covariates (Park et al., 2021). Seven studies adjusted for physical activity (Hu et al., 2018; Yeh, 2010; Wannamethee et al., 2001; Luo et al., 2012; Choi et al., 2021; Sahle et al., 2021; Oba et al., 2012), and six adjusted for alcohol consumption (Hu et al., 2018; Wannamethee et al., 2001; Luo et al., 2012; Sahle et al., 2021; Oba et al., 2012; Xie et al., 2024). Six studies accounted for hypertension or blood pressure (Hu et al., 2018; Yeh, 2010; Luo et al., 2012; Choi et al., 2021; Oba et al., 2012; Xie et al., 2024), and three included family history of diabetes (Hu et al., 2018; Choi et al., 2021; Oba et al., 2012). Sociodemographic factors were considered in several studies, with four adjusting for race/ethnicity (Hu et al., 2018; Yeh, 2010; Luo et al., 2012; Bar-Zeev et al., 2020) and four including education level or socioeconomic indicators (Yeh, 2010; Luo et al., 2012; Sahle et al., 2021; Wu et al., 2022). Dietary factors were considered in three studies (Hu et al., 2018; Choi et al., 2021; Sahle et al., 2021), while additional anthropometric measures, such as waist circumference, were included in one study (Luo et al., 2012). Three studies adjusted for residential area or study center (Yeh, 2010; Choi et al., 2021; Wu et al., 2022), and one included a psychological factor, adjusting for depressive symptoms (Wu et al., 2022).

3.3. Quality assessment of the included studies

The Newcastle-Ottawa Scale assessment showed that most included studies were of good to high quality, though studies by Bar-Zeev et al. (Bar-Zeev et al., 2020) and Xie et al. (Xie et al., 2024) showed some methodological limitations in representativeness of exposed cohorts and exposure ascertainment, while all studies adequately controlled for key confounders and maintained sufficient follow-up duration (Supplementary Table S4).

3.4. Comparison of quitters versus continuous smokers

Seven studies (Hu et al., 2018; Bar-Zeev et al., 2020; Choi et al., 2021; Sahle et al., 2021; Park et al., 2021; Wu et al., 2022; Xie et al., 2024) examined the relationship between smoking cessation and diabetes risk among individuals who quit smoking compared to continuous

smokers, with analyses stratified by post-cessation weight status (weight gain versus no weight gain) (Fig. 2).

Among those with weight gain, the pooled analysis of these seven studies (Hu et al., 2018; Bar-Zeev et al., 2020; Choi et al., 2021; Sahle et al., 2021; Park et al., 2021; Wu et al., 2022; Xie et al., 2024) showed a non-significant increased risk of diabetes (HR = 1.71, 95 % CI: 1.12, 2.62) with substantial heterogeneity ($I^2 = 99\%$).

When stratified by smoking cessation duration, pooled analysis showed that recent quitters had significantly higher diabetes risk than continuous smokers (HR = 2.20, 95 % CI: 1.27, 3.82) with heterogeneity ($I^2 = 85\%$) (Table 2). For long-term quitters who gained weight, only one study (Choi et al., 2021) presented no increased risk of diabetes compared to continuous smokers (HR = 0.92, 95 % CI: 0.30, 2.58). When studies with unspecified cessation durations were classified as long-term quitters, the pooled analysis revealed a slight reduction in diabetes risk compared to continuous smokers (HR = 0.91, 95 % CI: 0.87, 0.95) with minimal heterogeneity ($I^2 = 0\%$). Significant differences were observed between the two cessation duration subgroups: recent quitters had a significantly increased diabetes risk (HR = 2.20), while long-term quitters had a significantly reduced risk (HR = 0.91) compared to continuous smokers ($\text{Chi}^2 = 9.78, p < 0.01$).

Among those without weight gain, the pooled effect across these seven studies indicated no significant difference in diabetes risk (HR = 1.03, 95 % CI: 0.83, 1.29), with considerable heterogeneity ($I^2 = 93\%$) (Fig. 2).

Stratifying by cessation duration, recent quitters showed no significant difference in T2DM risk compared to continuous smokers (HR = 0.98, 95 % CI: 0.81, 1.20) with moderate to high heterogeneity ($I^2 = 67\%$) (Table 2). Long-term quitters, however, showed a significant reduction in diabetes risk (HR = 0.84, 95 % CI: 0.81, 0.87) with no heterogeneity ($I^2 = 0\%$) (Table 2). No significant subgroup differences were observed between recent and long-term quitters ($\text{Chi}^2 = 2.35, p = 0.13$).

3.5. Comparison of quitters versus never smokers

Six studies (Yeh, 2010; Wannamethee et al., 2001; Luo et al., 2012; Bar-Zeev et al., 2020; Sahle et al., 2021; Oba et al., 2012) compared quitters to never smokers; five (Yeh, 2010; Luo et al., 2012; Bar-Zeev et al., 2020; Sahle et al., 2021; Oba et al., 2012) provided sufficient data for meta-analysis (Fig. 2A).

Among quitters with weight gain, the pooled analysis revealed no significant difference in diabetes risk (HR = 1.20, 95 % CI: 0.86, 1.67) with moderate heterogeneity ($I^2 = 79\%, p < 0.01$) (Fig. 2B).

When comparing recent quitters with weight gain to never smokers, one study found no association (Oba et al., 2012), while two found increased risk (Yeh, 2010; Luo et al., 2012). The pooled analysis revealed that recent quitters had a 61 % increased risk of T2DM compared to never smokers (HR = 1.61, 95 % CI: 1.03, 2.50) (Table 2). For long-term quitters, one study reported no increased risk of diabetes (Luo et al., 2012). The pooled analysis, combining two additional studies without reported quitting duration, showed no association between quitting and the risk of T2DM (HR = 0.99, 95 % CI: 0.74, 1.30) (Table 2). No significant subgroup differences were observed between recent and long-term quitters ($\text{Chi}^2 = 3.37, p = 0.07$).

Among those without weight gain, the pooled analysis showed no significant difference in diabetes risk among quitters without weight gain (HR = 1.10, 95 % CI: 0.89, 1.36) with low-to-moderate heterogeneity ($I^2 = 39\%, p = 0.16$) (Fig. 2B).

When examining recent quitters versus never smokers without weight gain, four studies (Yeh, 2010; Wannamethee et al., 2001; Luo et al., 2012; Oba et al., 2012) showed increased point estimates with varying statistical significance. The pooled analysis indicated that recent quitters without weight gain had a significant 25 % increased risk of T2DM compared to never smokers (HR = 1.25, 95 % CI: 1.05, 1.48) (Table 2). For long-term quitters without weight gain, pooled analysis

showed no association between smoking cessation without weight gain and T2DM risk (Luo et al., 2012). Significant subgroup differences were observed between recent and long-term quitters ($\text{Chi}^2 = 7.51, p < 0.01$).

3.6. Sensitivity analysis

Sensitivity analyses were conducted to explore potential sources of heterogeneity and assess the robustness of our findings. First, we examined the impact of excluding specific study characteristics, including a lower-quality study (Bar-Zeev et al., 2020), a study with pregnant populations (Bar-Zeev et al., 2020), a study with the smallest sample size (Xie et al., 2024), and a study with the largest sample size (Park et al., 2021). For quitters versus continuous smokers with weight gain, the hazard ratio shifted from the original 1.71 (95 % CI: 1.12, 2.62) to 1.58 (95 % CI: 1.37, 1.83), with heterogeneity decreasing substantially from $I^2 = 99\%$ to 0% . For quitters versus continuous smokers without weight gain, the hazard ratio changed from 1.03 (95 % CI: 0.83, 1.29) to 1.00 (95 % CI: 0.93, 1.20), with heterogeneity decreasing from $I^2 = 93\%$ to 63% .

Then, a leave-one-out analysis was conducted by sequentially removing individual studies and recalculating pooled effect estimates. For quitters versus continuous smokers without weight gain, removing the study by Bar-Zeev et al. (Bar-Zeev et al., 2020) notably reduced heterogeneity from 99% to 66% (HR = 0.92, 95 % CI: 0.82, 1.04). For quitters versus continuous smokers with weight gain, removing the study by Park et al. (Park et al., 2021) substantially decreased heterogeneity from 99% to 79% . In the comparison of quitters versus never smokers with weight gain, removing the study by Yeh (Yeh, 2010) resulted in the most substantial reduction in heterogeneity (from 79% to 39% ; RR = 0.98, 95 % CI: 0.81, 1.19). In the analysis without weight gain, removing either study by Luo et al. (Luo et al., 2012) or Oba et al. (Oba et al., 2012) completely eliminated heterogeneity ($I^2 = 0\%$), suggesting that these studies contributed substantially to between-study variability. When the study by Luo et al. (Luo et al., 2012) was removed, the estimate increased slightly (HR = 1.24, 95 % CI: 0.98, 1.58), and when the study by Oba et al. (Oba et al., 2012) was removed, the estimate decreased (RR = 0.98, 95 % CI: 0.88, 1.08).

3.7. Publication bias

Publication bias assessment using Egger's test revealed no significant asymmetry for any comparison (all $p > 0.05$). The trim-and-fill method identified one potentially missing study in each analysis; however, after adjustment, pooled hazard ratios changed minimally (0.6–4.3 %) and retained their statistical significance.

4. Discussion

Our primary analysis found that weight gain following smoking cessation significantly increased T2DM risk compared to continuous smokers, particularly among recent quitters. However, long-term quitters, regardless of weight change, were associated with a reduced risk of T2DM. Smoking cessation without weight gain showed no increased T2DM risk compared to continuous smoking, with long-term quitters experiencing a reduced risk. When compared to never smokers, no statistically significant association was found between quitters and never smokers when stratifying by weight change. However, recent quitters with weight gain had significantly higher T2DM risk compared to never smokers, while long-term quitters showed no significant difference regardless of weight changes.

Our systematic review and meta-analysis found that weight gain following smoking cessation significantly increased T2DM risk by 71 % compared to continuous smokers. This aligns with prior research indicating that excess body weight increases the risk of T2DM. One study reported that individuals who are overweight (BMI 25–29.9), obese (BMI 30–39.9), or morbidly obese (BMI ≥ 40) have a substantially high

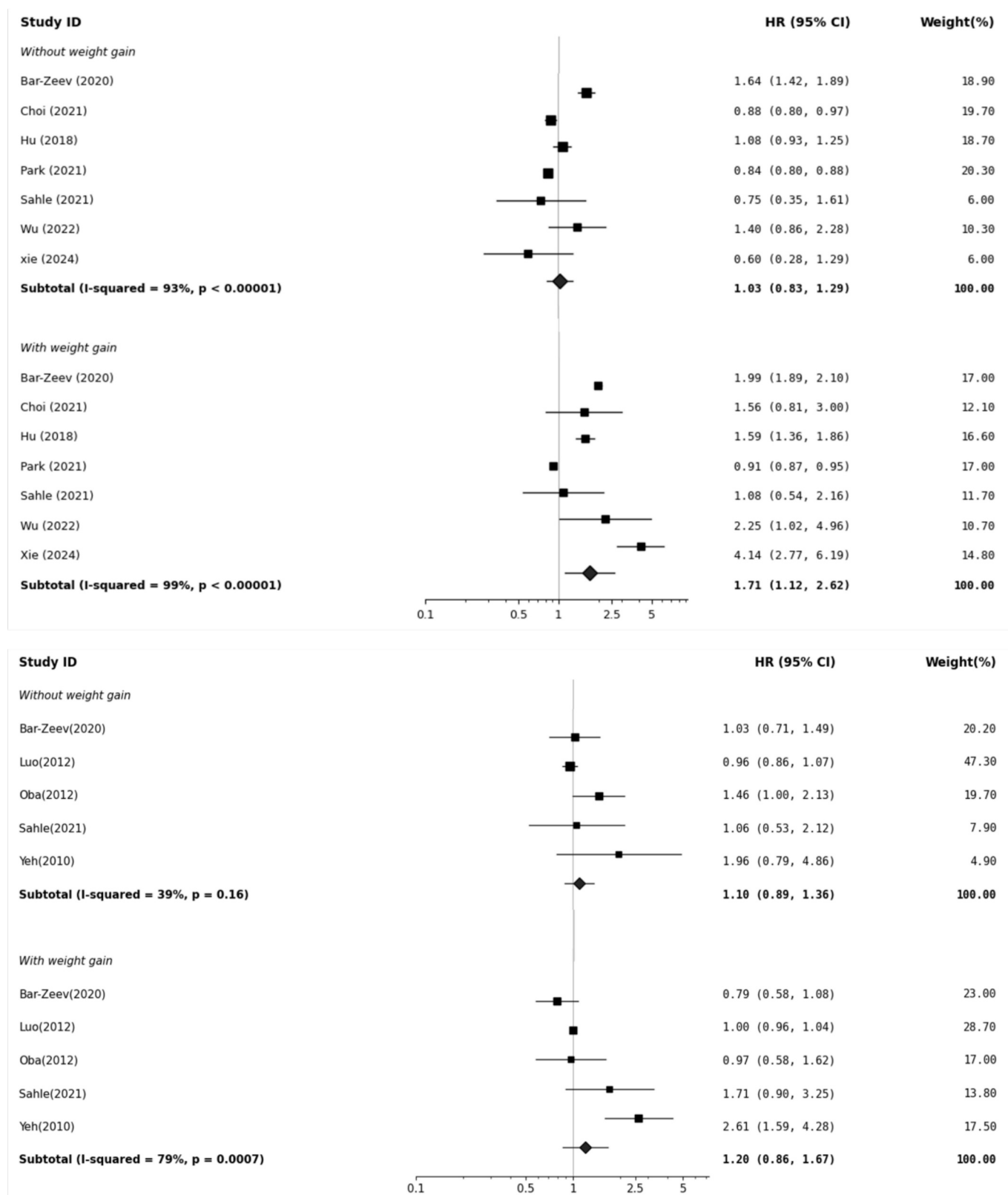


Fig. 2. (A) Hazard ratios and pooled hazard ratios of type 2 diabetes among smoking quitters with weight gain compared to continuous smokers: meta-analysis of seven cohort studies from Asia, Australia, Europe, and North America (2001–2024). (B) Hazard ratios and pooled hazard ratios of type 2 diabetes among smoking quitters with weight gain compared to never smokers: meta-analysis of five cohort studies from Asia, Australia, Europe, and North America (2001–2024).

Table 2

Pooled hazard ratios of type 2 diabetes among smoking quitters stratified by cessation duration and weight gain status: meta-analysis from Asia, Australia, Europe, and North America (2001–2024).

Comparison	With weight gain		Without weight gain	
	Pooled HR (95 % CI)	I ²	Pooled HR (95 % CI)	I ²
Quitters vs. continuous smokers		85		67
Recent quitter	2.20 (1.27, 3.82)	%	0.99 (0.81, 1.02)	%
Long-term quitter	0.91 (0.87, 0.95)	0 %	0.84 (0.81, 0.87)	0 %
Quitters vs. never smokers		57		5 %
Recent quitter	1.61 (1.03, 2.50)	70	1.25 (1.05, 1.48)	5 %
Long-term quitter	0.99 (0.74, 1.30)	%	0.96 (0.90, 1.03)	0 %

risk of developing T2DM (Yashi and Daley, 2025). Another study found that individuals who became obese had a significantly higher T2DM risk (HR 1.49, 95 % CI: 1.26, 1.77) compared to those who maintained a non-obese status, even after adjusting for confounders (Kim et al., 2018). In addition, a large cohort study demonstrated that greater variability in body weight was associated with an increased risk of T2DM (HR 1.10, 95 % CI 1.07, 1.12) when comparing the highest quartile of weight variability to the lowest (Park et al., 2019).

We found different impacts of quitting duration on T2DM risk stratified by weight status. Compared to continuous smokers, recent quitters with weight gain had 210 % higher risk of T2DM. This temporary increase among recent quitters who gain weight is consistent with previous findings. Early smoking cessation can cause acute metabolic changes and possible insulin resistance, which, combined with weight gain, may increase diabetes risk (Bush et al., 2016; Maddatu et al., 2017; Bajaj, 2012). Conversely, long-term quitters with weight gain showed reduced diabetes risk compared to continuous smokers, though based on a single study (Choi et al., 2021). When studies with unspecified cessation durations were included as long-term quitters (Choi et al., 2021; Sahle et al., 2021; Park et al., 2021), a statistically significant 9 % reduction in diabetes risk was observed. This suggests beneficial long-term cessation effects on metabolic health may outweigh adverse weight gain effects (Harris et al., 2016; Chioloro et al., 2008). This difference between recent and long-term quitters underscores the importance of sustained smoking cessation for individuals at risk for T2DM.

We found no statistically significant association between quitters without weight gain and the risk of T2DM compared to continuous smokers. This can be explained by persistent metabolic changes (Bergman et al., 2012), slow pancreatic β -cell recovery (Campagna et al., 2023; Driva et al., 2022), and hormone-induced central fat accumulation, which promotes insulin resistance (Śliwińska-Mossoń and Milnerowicz, 2017). However, long-term quitters without weight gain had a 16 % lower risk of T2DM, suggesting that the body gradually recovers from smoking-induced metabolic problems and that the lower T2DM risk in long-term quitters is not solely weight-related. Our findings support the hypothesis that without weight gain following smoking cessation, beneficial metabolic effects of quitting become more evident (Harris et al., 2016; Bush et al., 2016).

Our systematic review also found that recent quitters, regardless of weight status, had an increased risk of T2DM compared to never smokers (HR = 1.61 with weight gain, HR = 1.25 without). These findings are consistent with research suggesting residual smoking metabolic effects persist during early cessation. Tobacco-related inflammation, oxidative stress, and impaired insulin signaling may require prolonged recovery (Walicka et al., 2022). The differential effect between quitters with and without weight gain validates that post-cessation weight gain increases the risk of T2DM, although the

continued excess risk without weight gain suggests additional mechanisms. Importantly, similar T2DM risk among long-term quitters compared to never smokers, regardless of weight status, suggests the body requires extended time beyond recent quitting to fully recover metabolically from smoking effects (Walicka et al., 2022). This emphasizes continuing quit assistance beyond initial cessation and highlights that sustained smoking cessation benefits metabolic health long-term despite short-term adversity. There was some evidence of heterogeneity in our analysis. We were able to detect the sources of this heterogeneity through sensitivity analyses. For instance, the exclusion of the study by Bar-Zeev et al. (Bar-Zeev et al., 2020) significantly decreased heterogeneity in comparing quitters with continuous smokers who did not experience weight gain. This study included pregnant women, while other studies included in the review focused on general populations. Similarly, after further removing the study by Park et al. (Park et al., 2021) from the analysis comparing quitters versus continuous smokers with weight gain, the heterogeneity further decreased to 57 %. The decrease in heterogeneity could be attributed to the study by Park et al. (Park et al., 2021), which had the largest sample size among our included studies and used percentage weight change rather than absolute values as cutoff points. In contrast, many other studies used absolute weight change in kilograms (Hu et al., 2018; Yeh, 2010; Luo et al., 2012; Choi et al., 2021; Sahle et al., 2021; Wu et al., 2022). Additionally, the study by Park et al. (Park et al., 2021) was conducted in a South Korean population, while many others examined Western populations (Hu et al., 2018; Wannamethee et al., 2001; Luo et al., 2012; Sahle et al., 2021). After removing the study by Xie et al. (Xie et al., 2024) from the analysis, the heterogeneity reduced to 0. A possible reason may be that Xie's study included only male participants, while other studies included both genders. These differences in sample size, weight change classification methods, and study populations likely contributed to the observed heterogeneity in our initial analysis.

Our study had several strengths: 1) comprehensive search strategy across multiple databases providing extensive literature coverage; 2) rigorous selection process ensuring inclusion of high-quality research meeting predefined criteria; 3) stratification by post-cessation weight change and quitting duration, clarifying the complex relationship between smoking cessation and T2DM risk; 4) sensitivity analyses determining result robustness across various study populations and methodologies; 5) publication bias assessment using Egger's test and trim-and-fill approach showing no significant asymmetry, reinforcing our results; and comparable sample size to previous meta-analyses examining similar relationships (Wang et al., 2021).

We also acknowledge several limitations. First, substantial heterogeneity was observed in some analyses, though subgroup analyses by cessation duration and sensitivity analyses identified contributing factors (large sample studies, pregnant populations). Second, varying definitions of smoking quitters across studies necessitated stratification by quitting duration. Third, few studies examined long-term quitters; we conservatively classified studies without reported duration as long-term quitters, yielding consistent results with low heterogeneity. Fourth, inconsistent confounder adjustment increased heterogeneity, even though all studies adjusted for key confounders (e.g., age, BMI) and most studies adjusted for physical activity and alcohol consumption. Finally, the limited number of studies restricted the assessment of publication bias, but Egger's test and trim-and-fill analyses showed minimal impact on effect estimates (0.6–4.3 % changes), supporting the robustness of the results. Our meta-analysis combined hazard ratios and odds ratios, potentially introducing imprecision; however, this approach is acceptable when disease incidence is low (<10 %), which was the case for the studies we included.

5. Conclusions

Our systematic review and meta-analysis revealed a complex relationship between smoking cessation and type 2 diabetes risk, influenced

by post-cessation weight changes and cessation duration. While recent quitters with weight gain experience a temporarily increased risk of T2DM compared to continuous smokers, this risk gradually declines over time. After several years of cessation, the risk of T2DM returns to levels similar to never smokers, particularly without significant weight gain. These findings highlight the long-term metabolic benefits of quitting smoking while emphasizing the need for weight management strategies in cessation programs.

Availability of data and materials

All data that were generated or analyzed during this study are included in this published article. Additional information or data is available upon request.

CRediT authorship contribution statement

Yifan Yu: Writing – review & editing, Writing – original draft, Visualization, Validation, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Yan Li:** Writing – review & editing, Validation, Supervision, Methodology. **Thu T. Nguyen:** Writing – review & editing, Validation, Supervision, Methodology. **Dahai Yue:** Writing – review & editing, Validation, Supervision, Methodology. **Nedelina Tchangelova:** Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Conceptualization. **Caitlin E. Flouton:** Writing – review & editing, Validation, Methodology, Data curation. **Hongjie Liu:** Writing – review & editing, Supervision, Methodology, Conceptualization.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or non-profit sectors. All authors have no financial relationships with any organizations that might have an interest in the submitted work, nor any other relationships or activities that could appear to have influenced the submitted work.

Declaration of competing interest

The authors declare no competing interests.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jpmed.2025.108429>.

Data availability

No data was used for the research described in the article.

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