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IMPACT OF SMOKING CESSATION ON GLYCAEMIC CONTROL IN PATIENTS WITH TYPE 2 DIABETES MELLITUS: A SYSTEMATIC REVIEW

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ABSTRACT

Objective: To systematically evaluate the effect of smoking cessation on glycaemic control — indexed by HbA1c, fasting blood glucose and insulin resistance — in adults with T2DM.

Methods: A PRISMA 2020-compliant search of PubMed/MEDLINE and Google Scholar was conducted through March 2025. Of 56 records identified, 24 underwent full-text review; six studies (N = 15,744; five countries) met all inclusion criteria. Evaluation of quality using the Cochrane RoB 2 tool for RCTs and ROBINS-I for observational studies.

Results: the number of records included were six studies: two RCTs, two cohort studies, one retrospective cohort, and one cross-sectional observational study. Two observational studies documented a transient short-term HbA1c increase post-cessation, primarily attributed to weight gain. One large cross-sectional study provided supportive associative evidence of a dose- and time-dependent inverse connection among cessation duration, HbA1c, and insulin resistance; however, its design precludes causal inference. One retrospective cohort showed significant HbA1c reduction at six months despite weight gain. Both RCTs reported null findings, reflecting critically insufficient cessation rates.

Conclusions: Cessation follows a temporally biphasic glycaemic trajectory: a transient early deterioration followed by sustained long-term improvement. Proactive weight management — including GLP-1 receptor agonist therapy where indicated — is essential to optimise post-cessation metabolic outcomes. Adequately powered long-term RCTs are urgently required.

KEYWORDS: smoking cessation; type 2 diabetes mellitus; HbA1c; glycaemic control; insulin resistance; PRISMA 2020; systematic review

1. INTRODUCTION

Diabetes melilitus (T2DM) influence many people around the globe with current data around 537 million and expected to jump in 2045 to around 783 million (International Diabetes Federation, 2021) [(1)], with sustained HbA1c reduction constituting the cornerstone of complication prevention. Tobacco smoking — the leading stoppable cause of premature mortality (World Health Organization, 2023) [(2)], converges deleteriously on glucose homeostasis through multiple pathways. Nicotine stimulates catecholamine secretion, augmenting hepatic glucose output and peripheral [(3)(4)]. Activation of nicotinic acetylcholine receptors impairs IRS-1 insulin signal transduction via the MAPK and mTOR pathways [(5)], while smoke-derived reactive oxygen species deplete Nrf2-mediated antioxidant defence and disrupt endothelial nitric oxide synthase activity [(6)].Systemic inflammation characterized by elevated IL-6 and TNF-α further impairs beta-cell function [(7)(8)].Active smoking in T2DM independently elevates cardiovascular mortality [(9)] and worsens cardiometabolic profiles [(10)].

Smoking cessation is universally recommended in T2DM guidelines (American Diabetes Association, 2024) [(11)(12)(13)]. Nevertheless, cessation presents a clinical paradox: patients commonly experience a rise in transient HbA1c, predominantly attributed to post-cessation weight gain [(14)] and alterations in appetite-regulating hormones. Unique patient-level barriers — including diabetes-related depression, perceived metabolic benefits of smoking, and weight-gain apprehension — further complicate cessation management [(15)]. Emerging pharmacological strategies combining varenicline with glucagon-like peptide-1 receptor agonists (GLP-1 RAs) offer integrated solutions that address cessation, weight, and glycaemia simultaneously [(16)(17)]. This systematic review synthesises the available evidence on the temporal pattern and magnitude of glycaemic change following smoking cessation in adults with T2DM.

2. METHODS

This review was executed and written in according with the PRISMA 2020 statement [(18)]. Registration in PRSPERO with ID CRD420261338679 was performed.

2.1 Search Strategy

PubMed/MEDLINE and Google Scholar were searched from inception through March 2025 using: ("smoking cessation" OR "tobacco cessation" OR "quit smoking") AND ("type 2 diabetes" OR "diabetes mellitus type 2") AND ("HbA1c" OR "glycaemic control" OR "blood glucose" OR "insulin resistance"). Searches were conducted independently by both authors; results were merged, de-duplicated, and supplemented by manual reference screening, yielding 56 total records.

2.2 Eligibility Criteria

Inclusion: Adults (≥18 years) with confirmed T2DM; smoking cessation as an intervention or exposure; a comparator of continued smokers or usual care; at least one quantitative glycaemic outcome (HbA1c, FBG, or HOMA-IR); study designs of RCT, cohort, or cross-sectional observational study (the last accepted for associative evidence only). **Exclusion:** Type 1 DM, gestational DM, paediatric samples, studies reporting only cessation rates without glycaemic data, review articles, conference abstracts, and studies at critical risk of bias.

2.3 articles Selection, Data Extraction, and Quality Assessment

Both authors self-reliant viewed records and resolved disagreements by consensus. Standardised extraction forms captured study design, participant characteristics, cessation intervention details, and glycaemic outcomes. Evaluation of risk of bias utilizing the Cochrane RoB 2 tool [19] for RCTs and ROBINS-I [20] for observational articles. Narrative synthesis was preformed because of substantial methodological heterogeneity.

3. RESULTS

3.1 record Selection

Of 56 papers spotted, seven duplicates were removed, yielding 49 for title/abstract screening; 25 were excluded. Of the 24 full-text articles assessed, 18 were excluded: six review or scoping review articles (Tonstad, 2009[21]; Walicka et al., 2022[22]; Driva et al., 2022[23]); one Chinese-only publication (Su et al., 2017[24]); three reporting cessation rates only (Persson & Hjalmarson, 2006[25]; Canga et al., 2000[26]; Polosa et al., 2025[27]); two non-T2DM cohorts; two with smoking-status data only; two at critical risk of bias; one duplicate; and one conference abstract. Six studies met all inclusion criteria. The PRISMA 2020 flow diagram is presented in Figure 1.

Figure 1. PRISMA 2020 Flow Diagram — Study Selection Process

IDENTIFICATION		
Identifiable records through database searches PubMed/MEDLINE (n = 18) | Google Scholar (n = 38) **Total identified: n = 56**		Additional records from manual searching (n = 0)
▼		
researches after duplicates removed (n = 49)		**Duplicates excluded: n = 7** Identical records across databases
SCREENING		
researches screened (title and abstract) (n = 49)		**Excluded at title/abstract: n = 25** Irrelevant to PICO (n = 12) Incorrect population (n = 5) Ineligible study design (n = 5) No glycaemic outcomes (n = 3)
▼		
ELIGIBILITY		
Full-text researches assessed for eligibility (n = 24)		**Excluded at full-text review: n = 18** Review/scoping review articles (n = 6) Language barrier (n = 1) Cessation rates only (n = 3)

		Type 1 DM or non-diabetic (n = 2) Smoking status only (n = 2) Critical risk of bias (n = 2) Duplicate publication (n = 1) Conference abstract (n = 1)
▼		
INCLUDED		
included articles in qualitative synthesis (n = 6) 2 RCTs | 2 Cohort Studies | 1 Cross-Sectional Observational Study | 1 Retrospective Cohort **Total participants: N = 15,744**		

PRISMA = Preferred Reporting Items for Systematic Reviews and Meta-Analyses. T1DM = type 1 diabetes mellitus. RoB = risk of bias. PICO = Population, Intervention, Comparator, Outcome.

3.2 Characteristics of Included records

Six articles released between 2004 and 2025 spanned Japan (n = 2), Taiwan (n = 1), the UK (n = 1), Malaysia (n = 1), and Hong Kong (n = 1), comprising two RCTs (Ibrahim, 2023[28]; Li et al., 2017[29]), one retrospective cohort (Fu et al., 2025)[30] , one prospective observational cohort (Iino et al., 2004)[31], one primary care database cohort (Lycett, 2015)[32], and one cross-sectional observational article (Ohkuma, 2015)[33] . The cross-sectional study was included because it evaluated glycaemic control according to cessation status and time since quitting; however, due to its design, temporal and causal inferences remain limited. The total sample was 15,744 (range: 25–10,592); participants were predominantly male (87–100%); baseline HbA1c ranged from 6.53% to 8.8%, reflecting heterogeneous clinical contexts. The included articles features are summarised in Table 1.

Table 1. features of the Included records

article	Title (Abbreviated)	Geography	Design	n	Follow-up	Exposure vs. Comparator	Primary Glycaemic Outcome
Fu et al. (2025)[30]	Smoking cessation: metabolic & renal effects	Taiwan	Retrospective cohort	1,954	6 months	Cessation success vs. failure	HbA1c ↓ 6.59→6.28%; β=−0.258
Iino et al. (2004)[31]	Smoking cessation & glycaemic control in T2DM	Japan	Prospective observational cohort	25	12 months	Quitters (n=15) vs. continued smokers	HbA1c ↑ 6.8→7.8% at 12 months
Lycett et al. (2015)[32]	Smoking cessation & HbA1c: THIN cohort	UK	Database cohort (THIN)	10,592	12 months	Cessation vs. continued smoking	Transient HbA1c ↑ +0.21% (95% CI 0.17–0.25)
Ibrahim et al. (2023)[28]	Brief cessation intervention & glycaemia in T2DM	Malaysia	Randomised controlled trial	126	~6 months	5A's counselling vs. usual care	No significant HbA1c difference
Ohkuma et al. (2015)[33]	Dose/time-dependent cessation & glycaemia: Fukuoka Registry	Japan	Cross-sectional observational	2,490	N/A	Never / past / current smokers; years since quitting	HbA1c ↓ linearly with cessation years; HOMA2-IR dose-response

article	Title (Abbreviated)	Geography	Design	n	Follow-up	Exposure vs. Comparator	Primary Glycaemic Outcome
Li et al. (2017)[29]	Brief cessation intervention & glycaemia in T2DM	Hong Kong	Randomised controlled trial	557	12 months	Stage-matched counselling vs. usual care	No significant HbA1c difference at 12 months

T2DM = type 2 diabetes mellitus. RCT = randomised controlled trial. THIN = The Health Improvement Network. N/A = not applicable. Note: Ohkuma et al. (2015) is cross-sectional; causal and temporal inferences are limited accordingly.

3.3 Risk of Bias

Both RCTs were graded as having as low overall risk on the RoB 2 tool, with adequate randomisation, allocation concealment, and biochemically verified outcomes. The THIN database cohort (Lycett et al., 2015) [32] and the Fukuoka Registry study (Ohkuma et al., 2015) [33] were rated moderate, primarily reflecting potential residual confounding. Iino et al. (2004) [31] was rated as high risk owing to a very small sample (n = 25) and inadequate confounder control. Fu et al. (2025) [30] was rated as moderate due to its retrospective design and telephone-verified smoking status. Assessments are Exhibited in Table 2.

Table 2. Risk of Bias evaluation of Included records

Study	Tool	Selection	Performance	Detection	Attrition	Reporting	Overall Risk
Fu et al. (2025) [30]	ROBINS-I	Moderate	Moderate	Moderate	Low	Low	**Moderate**
Iino et al. (2004) [31]	ROBINS-I	High	High	Moderate	Low	Low	**High**
Lycett et al. (2015) [32]	ROBINS-I	Low	Moderate	Low	Low	Low	**Moderate**
Ibrahim et al. (2023) [28]	Cochrane RoB 2	Low	Low	Low	Low	Low	**Low**
Ohkuma et al. (2015) [33]	ROBINS-I	Moderate	N/A	Moderate	N/A	Low	**Moderate**
Li et al. (2017) [29]	Cochrane RoB 2	Low	Low	Low	Moderate	Low	**Low**

Colour coding: Green = Low; Yellow = Moderate; Red = High. RoB 2 = Cochrane Risk of Bias 2 (RCTs). ROBINS-I = Risk Of Bias in Non-randomised Studies (observational). N/A = not applicable.

3.4 Glycaemic Outcomes

Fu et al. (2025) [30]. In 1,954 Taiwanese adults enrolled in a pharmacotherapy-supported cessation programme, successful abstinence produced a significant reduction in HbA1c from 6.59% to 6.28% at six months ($\beta = -0.258$; 95% CI: −0.502 to −0.014; $p < 0.05$), despite weight gain, alongside improvements in LDL and estimated glomerular filtration rate.

Iino et al. (2004) [31]. Among 25 Japanese patients (high risk of bias), HbA1c increased significantly from 6.8% to 7.8% at twelve months in quitters versus no change in continued smokers; fasting and postprandial glucose were similarly elevated. Weight gain was the proposed primary mediator, although the very small sample substantially limits generalisability.

Lycett et al. (2015) [32]. In 10,592 UK primary care patients, HbA1c increased by a mean of 0.21% (95% CI: 0.17–0.25; $p < 0.001$) during the year one post-cessation. Critically, this persisted after weight adjustment, implicating weight-independent mechanisms including withdrawal-related alterations in leptin, ghrelin, and GLP-1 [34]. HbA1c trajectories converged toward baseline levels over three years of sustained abstinence.

Ibrahim et al. (2023) [28]. This RCT of a brief 5A's physician-delivered intervention (n = 126) found no significant differences in HbA1c over six months (baseline 8.6% vs. 8.8%; p = NS), with identical 6.3% quit rates in both arms. The null finding reflects insufficient cessation rates rather than an absence of glycaemic benefit (Lindson et al., 2023) [35].
Ohkuma et al. (2015) [33]. This cross-sectional observational study of 2,490 male patients from the Fukuoka Diabetes Registry provided supportive associative evidence: current smoking was interconnected with a multivariate-adjusted rise in HbA1c of +0.19% relative to never-smokers (95% CI: 0.08–0.30; $p < 0.001$), and HbA1c decreased progressively with cessation duration ($\beta = -0.17$ for <10 years, −0.17 for 10–19 years, −0.23 for ≥20 years; p for trend < 0.001), with HOMA2-IR exhibiting a parallel dose-response pattern, all persisting after multivariable adjustment. Because of the cross-sectional design, however, definitive temporal and causal inferences cannot be drawn from these data.
Li et al. (2017) [29]. In 557 Hong Kong patients, HbA1c did not differ significantly between a stage-matched nurse counselling intervention and usual care at twelve months (7.95% vs. 8.05%; p = 0.49), nor between confirmed abstainers and non-quitters (7.96% vs. 7.99%; p = 0.90). Low quit rates (9.2% vs. 13.9%) and the predominance of pre-contemplation stage participants limit the interpretability of this null finding.
Fasting blood glucose was not significantly associated with smoking status after adjustment in any study, consistent with the hypothesis that smoking preferentially elevates HbA1c via post-prandial excursions and glycation kinetics rather than fasting glycaemia (Ohkuma et al., 2015) [33]. Glycaemic outcomes are summarised in Table 3.

Table 3. Summary of Glycaemic Outcomes Across Included records

Study	n	HbA1c Change	FBG / HOMA-IR	Significance	Notes
Fu et al. (2025) [30]	1,954	HbA1c ↓ 6.59%→6.28%; β=−0.258 (95% CI −0.502 to −0.014)	FBG: not reported HOMA-IR: not reported	Significant (p<0.05)	Net benefit despite weight gain; improved eGFR and LDL
Iino et al. (2004) [31]	25	HbA1c ↑ 6.8%→7.8% at 12 months; FBG and PPG also elevated	FBG and PPG elevated post-cessation	Significant deterioration	High RoB; small sample; weight gain implicated
Lycett et al. (2015) [32]	10,592	Transient ↑ +0.21% (95% CI 0.17–0.25) year 1; resolved by year 3	Not separately reported	Significant short-term increase	Weight-independent after adjustment; large primary care cohort
Ibrahim et al. (2023) [28]	126	No significant change (8.6% vs. 8.8%; p=NS)	Not reported	Non-significant	6.3% quit rate both groups; no pharmacotherapy used
Ohkuma et al. (2015) [33]	2,490	Current smokers +0.19% vs. never (95% CI 0.08–0.30); β decreases linearly with cessation years	FBG: NS after adjustment; HOMA2-IR: dose-response decrease	Significant; dose- and time-dependent	Cross-sectional: associative evidence only; male only
Li et al. (2017) [29]	557	No significant difference (7.95% vs. 8.05%; p=0.49)	Not reported	Non-significant	>70% pre-contemplation; 9.2% vs. 13.9% quit rate

HbA1c = glycated haemoglobin. FBG = fasting blood glucose. PPG = postprandial plasma glucose. HOMA2-IR = updated homeostatic model assessment of insulin resistance. NS = not statistically significant. Note: Ohkuma et al. (2015) provides associative evidence only; cross-sectional design precludes causal inference.

4. DISCUSSION

4.1 Principal Findings

This systematic review reveals that the glycaemic response to smoking cessation in T2DM is characterised by a temporally biphasic pattern: a transient short-term HbA1c deterioration in the early post-cessation period, followed by progressive,

dose- and time-dependent improvement with sustained long-term abstinence. This pattern has direct implications for patient counselling, integrated pharmacological management, and the design of future trials.

4.2 Short-Term Glycaemic Deterioration: Mechanisms

Post-cessation HbA1c elevation is predominantly, but not exclusively, mediated by weight gain. Nicotine withdrawal reverses anorexigenic and thermogenic effects, increasing visceral adiposity and insulin resistance in approximately 80–90% of quitters [14]. However, the persistence of HbA1c elevation after weight adjustment in the THIN cohort (Lycett et al., 2015) [32] implicates additional weight-independent mechanisms: withdrawal-related alterations in leptin, ghrelin, and endogenous GLP-1 secretion, as well as cessation of nAChR-mediated modulation of hepatic glucose output [5]. Pharmacotherapy-facilitated cessation may attenuate this effect; Driva et al. (2024) [13] found that varenicline-facilitated cessation was not associated with significant HbA1c deterioration and was accompanied by increased plasma GLP-1 and leptin without significant weight gain at four months.

4.3 Long-Term Glycaemic Improvement: Evidence and Mechanisms

The most compelling evidence for sustained benefit derives from the Fukuoka Registry (Ohkuma et al., 2015) [33], which demonstrated progressive HbA1c and HOMA2-IR reductions over 20 or more years of abstinence. Although the cross-sectional design inherently limits causal and temporal inference, the dose- and time-dependent gradient — persisting after rigorous multivariable adjustment — provides robust associative evidence for long-term benefit. Underlying mechanisms include progressive resolution of oxidative stress (Klein et al., 2023) [6], restoration of endothelial nitric oxide bioavailability (Bergman et al., 2012) [5], normalisation of systemic inflammatory burden [7][8], and recovery of IRS-1 insulin signalling [3]. That Fu et al. (2025) [30] observed a net HbA1c reduction at six months despite weight gain further suggests that direct glycaemic benefits of cessation can outweigh early adiposity-related deterioration in select contexts.

4.4 Null RCT Findings: Methodological Interpretation

The null HbA1c findings of both RCTs reflect insufficient cessation rates (6–14%), not an absence of glycaemic benefit. Neither incorporated pharmacotherapy, the established determinant of cessation success: a Cochrane network meta-analysis confirmed that varenicline yields substantially higher abstinence rates than counselling alone [35]. Both populations were predominantly pre-contemplative. Future RCTs must be pharmacotherapy-inclusive, powered on cessation rate, and sufficiently long (≥24–36 months) to capture long-term glycaemic trajectories.

4.5 GLP-1 Receptor Agonists: An Integrated Therapeutic Strategy

GLP-1 RAs simultaneously address glycaemia, post-cessation weight gain, and potentially tobacco dependence, offering particular clinical utility in T2DM patients who smoke. Preclinical evidence demonstrates that central GLP-1 receptor activation attenuates nicotine self-administration and reward [16]. Lüthi et al. (2024) [17] showed that dulaglutide combined with varenicline attenuated post-cessation weight gain and reduced new-onset prediabetes in abstinent smokers. Wang et al. (2024) [36] found semaglutide consume was interconnected with reduced incidence of tobacco use disorder in T2DM patients. Lengsfeld et al. (2023) [37] demonstrated comparable metabolic benefits of dulaglutide during active cessation, with attenuation after discontinuation, emphasising the importance of sustained pharmacotherapy.

4.6 Limitations and Research Priorities

Substantial heterogeneity precluded meta-analysis; only six studies were eligible, with variable quality. All cohorts were predominantly or exclusively male, critically limiting generalisability to women with T2DM. The cross-sectional design of Ohkuma et al. (2015) [33] , — although providing important supportive evidence — precludes definitive causal inference. Neither included RCT incorporated pharmacotherapy. Research priorities include: (1) large pharmacotherapy-inclusive RCTs with ≥24-month follow-up, ideally combining varenicline with GLP-1 RA co-therapy; (2) studies enrolling women; (3) mechanistic longitudinal studies characterising post-cessation trajectories of insulin sensitivity, inflammatory biomarkers, and appetite-regulating hormones; and (4) prospective registry studies describing glycaemic trajectories beyond twelve months of cessation.

5. CONCLUSIONS

This PRISMA 2020-compliant systematic review establishes that smoking cessation in T2DM follows a temporally biphasic glycaemic trajectory: a transient early HbA1c deterioration mediated by weight gain and weight-independent hormonal mechanisms, followed by progressive, dose- and time-dependent glycaemic improvement with sustained abstinence. Null RCT findings reflect insufficient cessation rates, not an absence of benefit. Smoking cessation must be an indispensable component of T2DM management, supported by pharmacotherapy, proactive weight management, and — where indicated — GLP-1 RA therapy to optimise concurrent glycaemic, weight, and cessation outcomes. Adequately powered, pharmacotherapy-inclusive, long-term RCTs are urgently needed.

Author Contributions
A.S.A.: Conceptualization, protocol establishment, literature review, data extraction, quality assessment, manuscript preparation, and final approval. A.M.J.: Protocol development, independent search verification, data cross-validation, critical revision, and final approval.

Conflicts of Interest
none conflicts of interest stated by authors.

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All supporting data are available within the cited primary publications. No new primary data were generated.

Ethics Statement
This review was conducted on published data; ethics approval was not required.

REFERENCES
1. International Diabetes Federation. (2021). IDF diabetes atlas (10th ed.). International Diabetes Federation. https://www.diabetesatlas.org
2. World Health Organization. (2023). WHO report on the global tobacco epidemic 2023. World Health Organization.
3. Durlach, V., Vergès, B., Al-Salameh, A., Bahougne, T., Canoüï-Poitrine, F., Feve, B., Gourdy, P., Halimi, J.-M., Kanie, J.-P., & Marre, M. (2022). Smoking and diabetes interplay: A comprehensive review and joint statement. Diabetes & Metabolism, 48(6), 101370. https://doi.org/10.1016/j.diabet.2022.101370
4. Śliwińska-Massoń, M., & Milnerowicz, H. (2017). The impact of smoking on the development of diabetes and its complications. Diabetes & Vascular Disease Research, 14(4), 265–276. https://doi.org/10.1177/1479164117701876
5. Bergman, B. C., Perreault, L., Hunerdosse, D., Kerege, A., Playdon, M., Samek, A. M., & Bacchi, E. (2012). Novel and reversible mechanisms of smoking-induced insulin resistance in humans. Diabetes, 61(12), 3156–3166. https://doi.org/10.2337/db12-0418
6. Klein, J., Diaba-Nuhoho, P., Giebe, S., Brunssen, C., & Morawietz, H. (2023). Regulation of endothelial function by cigarette smoke and next-generation tobacco and nicotine products. Pflügers Archiv — European Journal of Physiology, 475(7), 835–844. https://doi.org/10.1007/s00424-023-02824-w
7. Cai, J., & Bidulescu, A. (2023). The association between e-cigarette use or dual use and prediabetes, diabetes, or insulin resistance: Findings from NHANES. Drug and Alcohol Dependence, 251, 110948. https://doi.org/10.1016/j.drugalcdep.2023.110948
8. Rehman, K., & Akash, M. S. H. (2016). Mechanisms of inflammatory responses and development of insulin resistance: How are they interlinked? Journal of Biomedical Science, 23(1), 87. https://doi.org/10.1186/s12929-016-0303-y , Rehman, K., Akash, M. S. H., Liaqat, A., Karim, S., Rasul, S., & Akash, M. (2021). Cigarette smoking and nicotine exposure contributes to aberrant insulin signalling and cardiometabolic disorders. Journal of Hazardous Materials, 416, 125927. https://doi.org/10.1016/j.jhazmat.2021.125927
9. Pan, A., Wang, Y., Talaei, M., & Hu, F. B. (2015). Relation of smoking with total mortality and cardiovascular events among patients with diabetes mellitus: A meta-analysis and systematic review. Circulation, 132(19), 1795–1804. https://doi.org/10.1161/CIRCULATIONAHA.115.017926
10. Kar, D., Gillies, C., Zaccardi, F., Webb, D. R., Seidu, S., Tesfaye, S., Davies, M. J., & Khunti, K. (2016). Relationship of cardiometabolic parameters in non-smokers, current smokers, and quitters in diabetes: A systematic review and meta-analysis. Cardiovascular Diabetology, 15(1), 158. https://doi.org/10.1186/s12933-016-0475-5
11. American Diabetes Association Professional Practice Committee. (2024). Standards of care in diabetes — 2024. Diabetes Care, 47(Suppl 1), S1–S321. https://doi.org/10.2337/dc24-Sint
12. ElSayed, N. A., Aleppo, G., Aroda, V. R., Bannuru, R. R., Brown, F. M., Bruemmer, D., Collins, B. S., Gaglia, J. L., Hilliard, M. E., Isaacs, D., Johnson, E. L., Kahan, S., Khunti, K., Leon, J., Lyons, S. K., Perry, M. L., Prahalad, P., Pratley, R. E., Seley, J. J., … Gabbay, R. A. (2023). Standards of care in diabetes — 2023. Diabetes Care, 46(Suppl 1), S1–S4. https://doi.org/10.2337/dc23-Sint
13. Seidu, S., Cos, X., Brunton, S., Harris, S. B., Jansson, S. P. O., Mata-Cases, M., Neijens, A.-M., Nian, H., & Khunti, K. (2022). 2022 update to the position statement by Primary Care Diabetes Europe. Primary Care Diabetes, 16(2), 223–244. https://doi.org/10.1016/j.pcd.2022.02.002
14. Hartmann-Boyce, J., Theodoulou, A., Farley, A., Hajek, P., Lycett, D., Jones, L. L., Kudlek, L., Heath, L., Hajek, P., Dockrell, M., & Aveyard, P. (2021). Interventions for preventing weight gain after smoking cessation. Cochrane Database of Systematic Reviews, 2021(10), CD006219. https://doi.org/10.1002/14651858.CD006219.pub4

15. Grech, J., Sammut, H., Sammut, M. R., Fenech, A., Camilleri, L., Gauci, M., Staines, R. J., & Borg, A. (2024). Exploring the smoking cessation needs of individuals with diabetes using the IMB model. Tobacco Prevention & Cessation, 10, TPC/181366. https://doi.org/10.18332/tpc/181366
16. Herman, R. J., Hayes, M. R., Audrain-McGovern, J., Ashare, R. L., & Schmidt, H. D. (2024). Targeting GLP-1 receptors to reduce nicotine use disorder: Preclinical and clinical evidence. Frontiers in Pharmacology, 15. https://doi.org/10.3389/fphar.2024.1396432
17. Lüthi, H., Lengsfeld, S., Burkard, T., Meienberg, A., Vogt, D. R., Bathelt, C., Winzeler, B., Christ, E., Mühlebach, S., & Leuppi-Taegtmeyer, A. (2024). Effect of dulaglutide in promoting abstinence during smoking cessation: 12-month follow-up of a randomised, double-blind, placebo-controlled trial. EClinicalMedicine, 68, 102429. https://doi.org/10.1016/j.eclinm.2024.102429
18. Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., Shamseer, L., Tetzlaff, J. M., Akl, E. A., Brennan, S. E., Chou, R., Glanville, J., Grimshaw, J. M., Hróbjartsson, A., Lalu, M. M., Li, T., Loder, E. W., Mayo-Wilson, E., McDonald, S., … Moher, D. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. BMJ, 372, n71. https://doi.org/10.1136/bmj.n71
19. Sterne, J. A. C., Savović, J., Page, M. J., Elbers, R. G., Blencowe, N. S., Boutron, I., Cates, C. J., Cheng, H.-Y., Corbett, M. S., Eldridge, S. M., Hernán, M. A., Hopewell, S., Hróbjartsson, A., Junqueira, D. R., Jüni, P., Kirkham, J. J., Lasserson, T., Li, T., McAleenan, A., … Higgins, J. P. T. (2019). RoB 2: A revised tool for assessing risk of bias in randomised trials. BMJ, 366, l4898. https://doi.org/10.1136/bmj.l4898
20. Sterne, J. A., Hernán, M. A., Reeves, B. C., Savović, J., Berkman, N. D., Viswanathan, M., Henry, D., Altman, D. G., Ansari, M. T., Boutron, I., Carpenter, J. R., Chan, A.-W., Churchill, R., Deeks, J. J., Hróbjartsson, A., Kirkham, J., Jüni, P., Loke, Y. K., Pigott, T. D., … Higgins, J. P. T. (2016). ROBINS-I: A tool for assessing risk of bias in non-randomised studies of interventions. BMJ, 355, i4919. https://doi.org/10.1136/bmj.i4919
21. Tonstad, S. (2009). Cigarette smoking, smoking cessation, and diabetes. Diabetes Research and Clinical Practice, 85(1), 4–13. https://doi.org/10.1016/j.diabres.2009.04.012
22. Walicka, M., Różycka-Kosmalska, M., Kasznicki, J., Szymoński, K., Świętosławski, J., Sieradzki, J., & Franek, E. (2022). Impact of stopping smoking on metabolic parameters in diabetes mellitus: A scoping review. World Journal of Diabetes, 13(6), 484–497. https://doi.org/10.4239/wjd.v13.i6.484
23. Driva, S., Korkontzelou, A., Tonstad, S., Tentolouris, N., & Katsaounou, P. (2022). The effect of smoking cessation on body weight and other metabolic parameters with focus on people with type 2 diabetes mellitus. International Journal of Environmental Research and Public Health, 19(20), 13222. https://doi.org/10.3390/ijerph192013222
24. Su, J., Qin, Y., Shen, H., Zhao, M., & Liu, P. (2017). [Association between smoking/smoking cessation and glycemic control in male patients with type 2 diabetes]. Zhonghua Liu Xing Bing Xue Za Zhi, 38(11), 1531–1535. https://doi.org/10.3760/cma.j.issn.0254-6450.2017.11.019
25. Persson, L.-G., & Hjalmarson, Å. (2006). Smoking cessation in patients with diabetes mellitus: Results from a controlled study. Scandinavian Journal of Primary Health Care, 24(2), 75–80. https://doi.org/10.1080/02813430500439395
26. Canga, N., De Irala, J., Vara, E., Duaso, M. J., Ferrer, A., & Martínez-González, M. A. (2000). Intervention study for smoking cessation in diabetic patients: A randomized controlled trial. Diabetes Care, 23(10), 1455–1460. https://doi.org/10.2337/diacare.23.10.1455
27. Polosa, R., Robson, A., Marczylo, T., & Forster, M. (2025). Addressing the dual challenge: Managing smoking cessation in patients with diabetes. World Journal of Diabetes, 16(12). https://doi.org/10.4239/wjd.v16.i12.00000
28. Ibrahim, A. K. A., Sulaiman, S. A. S., Awaisu, A., & Shafie, A. A. (2023). Impact of brief smoking cessation intervention on quitting rate and glycemic control in patients with diabetes: A randomized controlled trial. Journal of International Medical Research, 51(1). https://doi.org/10.1177/03000605221148785
29. Li, W. H. C., Wang, M. P., Lam, T. H., Cheung, Y. T. Y., Cheung, D. Y. T., Suen, Y. N., Ho, K. Y., Tan, K. C. B., & Chan, S. S. C. (2017). Brief intervention to promote smoking cessation and improve glycemic control in smokers with type 2 diabetes: A randomized controlled trial. Scientific Reports, 7(1), 45902. https://doi.org/10.1038/srep45902
30. Fu, C.-P., Fu, P.-K., Lu, M.-C., Liao, Y.-Y., & Wang, J.-S. (2025). Impact of smoking cessation on metabolic parameters and renal function in patients with and without diabetes mellitus. Revista Clínica Española (Barc). https://doi.org/10.1016/j.rceng.2025.01.001
31. Iino, K., Iwase, M., Tsuji, N., & Iida, M. (2004). Smoking cessation and glycaemic control in type 2 diabetic patients. Diabetes, Obesity and Metabolism, 6(3), 181–187. https://doi.org/10.1111/j.1462-8902.2004.00330.x
32. Lycett, D., Nichols, L., Ryan, R., Farley, A., Roalfe, A., Mohammed, M., Szatkowski, L., Coleman, T., Morris, R., Farmer, A., & Aveyard, P. (2015). The association between smoking cessation and glycaemic control in patients with type 2 diabetes: A THIN database cohort study. The Lancet Diabetes & Endocrinology, 3(6), 423–430. https://doi.org/10.1016/S2213-8587(15)00082-0
33. Ohkuma, T., Iwase, M., Fujii, H., Kaizu, S., Ide, H., Jodai, T., Kikuchi, Y., Idewaki, Y., Hirakawa, Y., Nakamura, U., & Kitazono, T. (2015). Dose- and time-dependent association of smoking and its cessation with glycemic control and

insulin resistance in male patients with type 2 diabetes mellitus: The Fukuoka Diabetes Registry. PLOS ONE, 10(3), e0122023. https://doi.org/10.1371/journal.pone.0122023
34. Driva, S., Korkontzelou, A., Tonstad, S., Tentolouris, N., Litsiou, E., Vasileiou, V., Vasileiadis, I., & Katsaounou, P. (2024). Metabolic changes following smoking cessation in patients with type 2 diabetes mellitus. Biomedicines, 12(8), 1882. https://doi.org/10.3390/biomedicines12081882
35. Lindson, N., Theodoulou, A., Ordóñez-Mena, J. M., Fanshawe, T. R., Sutton, A. J., Livingstone-Banks, J., Hajek, P., Hartmann-Boyce, J., & Aveyard, P. (2023). Pharmacological and electronic cigarette interventions for smoking cessation in adults: Component network meta-analyses. Cochrane Database of Systematic Reviews, 2023(9), CD015226. https://doi.org/10.1002/14651858.CD015226.pub2
36. Wang, W., Volkow, N. D., Berger, N. A., Davis, P. B., Kaelber, D. C., & Xu, R. (2024). Association of semaglutide with tobacco use disorder in patients with type 2 diabetes: Target trial emulation using real-world data. Annals of Internal Medicine, 177(8), 1016–1027. https://doi.org/10.7326/M23-2718
37. Lengsfeld, S., Burkard, T., Meienberg, A., Bathelt, C., Vogt, D. R., Winzeler, B., Christ, E., Mühlebach, S., & Leuppi-Taegtmeyer, A. (2023). Effect of dulaglutide in promoting abstinence during smoking cessation: A randomized, double-blind, placebo-controlled trial. EClinicalMedicine, 57, 101865. https://doi.org/10.1016/j.eclinm.2023.101865