Oral health and diabetes: a systematic review and meta-analysis

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Summary

Background Diabetes is a major public health challenge, affecting nearly 828 million people worldwide. Similarly, oral diseases such as dental caries, periodontal diseases, and oral cancers are highly prevalent, affecting approximately 3·7 billion individuals globally. Diabetes and oral diseases share common risk factors and studies suggest a potential relationship in which poor management of one potentially exacerbates the other. However, the direction, magnitude, and consistency of these associations remain unclear, and oral health remains largely overlooked in diabetes prevention and management strategies. The aim of this study is to systematically assess and synthesise the evidence on the longitudinal association between diabetes and oral diseases.

Methods We conducted a systematic review and meta-analysis reported in accordance with PRISMA guidelines. We searched four electronic databases (Embase, PubMed, Web of Science, and LILACS) and grey literature from database inception to Oct 27, 2025. Eligible studies included longitudinal prospective studies examining associations between diabetes (type 1, type 2, or gestational) and core WHO oral diseases (periodontitis, dental caries, tooth loss or edentulism [complete tooth loss], and oral cancer). Random-effects meta-analyses were performed using the restricted maximum likelihood method to estimate pooled effect sizes with 95% CIs of hazard ratios (HRs) and risk ratio (RR). Methodological quality was assessed using the Newcastle–Ottawa Scale for cohort studies.

Findings We screened 45 477 records and included 28 longitudinal studies from 16 countries, encompassing over 300 000 participants. Most studies were population-based and of moderate-to-high methodological quality. A bidirectional temporal association in incidence patterns was observed between diabetes and periodontitis. Individuals with periodontitis at baseline showed a 18–25% higher occurrence of newly incident type 2 diabetes during follow-up, and baseline diabetes status was associated with a higher newly incident cases of periodontitis, with pooled RRs indicating modest differences between groups. Tooth loss was also associated with newly incident type 2 diabetes over time (HR 1·12–1·20). Quantitative pooling was not feasible for edentulism outcomes; however, individuals who were edentulous at baseline showed a higher occurrence of newly incident type 2 diabetes (RR 1·30). Evidence regarding dental caries was scarce and heterogeneous, precluding meta-analysis. No longitudinal evidence was identified for oral cancer.

Interpretation Findings from this longitudinal synthesis indicate associations between diabetes and oral diseases in both directions; however, the strength and consistency of evidence are asymmetric. Evidence suggests that people with periodontitis and edentulism are more likely to be diagnosed with type 2 diabetes than people without periodontitis and edentulism in the future. People with diabetes are more likely to be diagnosed with periodontitis than people without diabetes, but with less consistent evidence. Our findings support integrating oral health within interdisciplinary diabetes prevention and management strategies.

Funding WHO Oral Health Programme.

Lancet Public Health 2026; 11: e555–66

A previous version of this Article has been retracted. For changes made see appendix 1

See **Comment** page e475

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Introduction

Diabetes affects over 828 million adults worldwide, a figure projected to reach 1·3 billion by 2050.[1,2] Type 2 diabetes accounts for the vast majority of diabetes cases, and is closely linked to a range of chronic complications, including cardiovascular diseases, neuropathy, nephropathy, and retinopathy, which collectively diminish quality of life and increase morbidity and mortality.[3] Among these complications, oral diseases have emerged as a substantial yet under-recognised complication of diabetes, which often remain under-addressed.[3,4]

Despite a growing body of evidence indicating a relationship between diabetes and oral health, the findings remain inconsistent and fragmented, partly due to differences in study designs, populations, and outcomes assessed.[5] Periodontitis often co-occurs with other diabetes complications,[4,6,7] and these conditions share common risk factors through inflammatory and metabolic pathways.[8] Periodontal treatment might also contribute to an additional decrease in A_{1c} glycated haemoglobin (HbA_{1c}) at 12 months post-treatment in people with diabetes;[9] however, the level of evidence remains low for the

Research in context

Evidence before this study

Several studies have explored the association between diabetes and oral health, particularly periodontitis. Although previous reviews suggest a potential link, the overall evidence remains limited by methodological constraints and heterogeneous study quality. We searched PubMed, Embase, Web of Science, LILACS, and grey literature using controlled vocabulary and free-text terms related to diabetes, periodontal disease, dental caries, tooth loss and edentulism, oral cancer, and prospective longitudinal study designs. Search syntaxes were first developed for PubMed and adapted for other databases. Searches covered from database inception to Sept 28, 2024, and were updated on Oct 27, 2025. Despite the abundance of high-quality studies examining the relationship between oral diseases and diabetes, there remains a lack of comprehensive synthesis; specifically, no systematic review to date had integrated prospective longitudinal data across the core WHO oral disease outcomes to investigate the bidirectional nature of these associations.

Added value of this study

To the best of our knowledge, this systematic review and meta-analysis is the first to synthesise exclusively longitudinal evidence on core WHO oral disease outcomes. We included 28 prospective longitudinal cohorts from 16 countries. Our findings show a bidirectional association between diabetes and periodontitis: individuals with periodontitis have a higher incidence of diabetes, and people with diabetes are at increased risk of periodontitis and subsequent tooth loss. Diabetes was also associated with a higher risk of tooth loss and vice-versa, and edentulism was associated with future risk of diabetes onset. For dental caries, the limited number and heterogeneity of eligible longitudinal studies prevented quantitative synthesis, although available evidence suggests a possible association. No longitudinal evidence was identified for oral cancer.

Implications of all the available evidence

The bidirectional relationship between diabetes and periodontitis highlights the need for integrated approaches, whereby oral health professionals and health practitioners collaborate to identify and address these interconnected conditions. Recognising that individuals with periodontitis have a higher incidence of diabetes, and vice versa, highlights the importance of early assessment and preventive strategies to mitigate disease progression and associated complications, such as tooth loss and edentulism. There is a clear need for an integrated, interdisciplinary approach to improve oral health and support people with diabetes worldwide.

For the **protocol** see https://osf.io/gsfn2/overview

relationship between diabetes and dental caries, tooth loss, or oral cancer.[5] Furthermore, oral health problems are often neglected in primary health care and diabetes management frameworks, especially in low-resource settings where health resources are constrained.[10]

See **Online** for appendix 2

Given the expanding evidence base, particularly at the population level, there is an urgent need for a comprehensive and rigorous synthesis of current research. A comprehensive systematic review is crucial to consolidate existing knowledge, clarify the nature of the oral health–diabetes association, and provide a foundation for shaping future policies and public health strategies. A robust, up-to-date evidence base is pivotal to inform integrated care models, shape national prevention frameworks, and support global initiatives (eg, those of WHO) to incorporate oral health into non-communicable disease prevention and control programmes.

We aim to systematically review the longitudinal association between diabetes and core WHO oral diseases (periodontitis, dental caries, tooth loss or edentulism [complete tooth loss], and oral cancer). Focusing on prospective longitudinal studies allows us to derive the highest level of evidence, facilitating a better understanding of a bidirectional relationship and temporal dynamics.

Methods

Overview

The protocol was defined a priori, based on the PRISMA-P guideline,[11] and available publicly on the Open Science Framework. The protocol was appraised by external reviewers and required deviations from the original plan; changes are highlighted in the revised protocol. The results of the present report of findings regarding the association between diabetes and oral diseases are reported following the PRISMA guideline[12] (checklist is included in appendix 2 [pp 3–5]).

Eligibility criteria

To maximise methodological rigour and causal inference, we restricted eligibility to longitudinal studies for all four core WHO outcomes: periodontal diseases, dental caries, tooth loss or edentulism, and oral cancer. Longitudinal designs were selected because they allow evaluation of temporality, reduce risk of reverse causation and selection bias, enable tracking of disease progression, and provide robust and reliable estimates.[13]

Considering the possible bidirectional relationship between diabetes and oral diseases, two population, exposure, comparator, outcome questions were set (appendix 2 p 6): do people (population) with diabetes (exposure) have a higher risk of having an oral disease (outcome: periodontal diseases, dental caries, tooth loss or edentulism, or oral cancer) over time compared with healthy counterparts (comparator); and do people (population) with an oral condition (exposure: periodontal diseases, dental caries, tooth loss or edentulism, or oral cancer) have a higher risk of developing diabetes

(outcome) over time compared with healthy counterparts (comparator).

The exclusion criteria were as follows: retrospective studies, cross-sectional studies, case–control studies, case reports, reviews, commentaries, conference abstracts; studies reporting data about periodontal healing after periodontal treatment (initial and supportive therapy); studies using imprecise or non-validated diagnostic criteria for diabetes or oral disease; and studies without methodological details or insufficient description of setting, population, or analytical approach.

Search strategy and study selection

Syntaxes were developed for PubMed and adapted for Embase, Web of Science, LILACS, and grey literature searches. Searches were carried out from database inception to Sept 28, 2024, and updated on Oct 27, 2025, with no language restrictions. Grey literature was searched on the same dates on OpenGrey, and an additional search was made on WHO reports, government publications, and conference proceedings. For Google Scholar, to maintain feasibility and reproducibility we screened the first 100 results sorted by relevance, following common practice in systematic and mapping reviews. No additional eligible studies were identified through this source. The search strategy was designed by two experienced researchers (JB and VM) and is shown in detail in appendix 2 (pp 7–11).

Two reviewers (JB and VM) independently screened titles and abstracts. Any paper classified as potentially eligible was appraised by a full-text reading, and the reasons for exclusion were fully detailed (appendix 3 p 1). Any discrepancies were resolved through discussion or consultation with a third reviewer (LP). We assessed the inter-examiner agreement through Cohen's kappa and an excellent level of agreement was found (k=0·86, 95% CI 0·83–0·89).

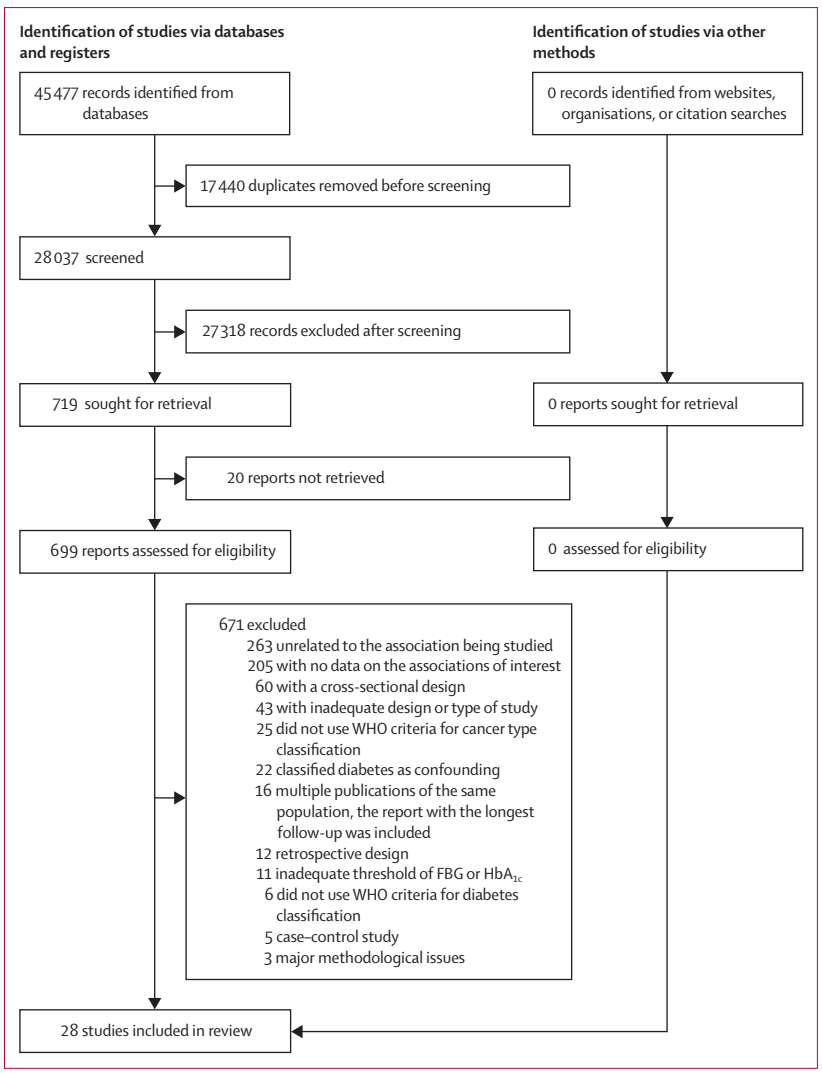

Figure 1: **Study selection**
FBG=fasting blood glucose. HbA_{1c}=A1c glycated haemoglobin.

Data extraction

Two reviewers (JB and VM) independently extracted data through the implementation of a standardised data extraction form using a Google Sheet. This worksheet was initially conducted separately for each reviewer, before being combined into a single document. Any discrepancies were resolved through discussion with the third reviewer (LP). The data extraction included: authors, year of publication, country, population details, study design, oral condition, clinical oral examination, case definition of oral conditions, calibration, diabetes type, diabetes case definition or assessment, range of duration of diabetes, diabetes complications, number of participants (eg, detailed by type of diabetes, controls, type of oral conditions, and sex ratio), and metabolic control (eg, HbA_{1c}).

Outcomes were defined according to each study's prespecified clinical or validated diagnostic criteria and were extracted verbatim during data collection. Periodontitis was identified using clinical periodontal assessments or established case definitions reported by individual studies, including measures such as probing depth, clinical attachment loss, bleeding on probing, or composite diagnostic thresholds. Dental caries was assessed using clinical examination indices (eg, decayed, missing, and filled teeth; decayed, missing, or filled surfaces; or components) or registry-based diagnoses. Tooth loss was defined as partial tooth loss (number or proportion of missing teeth) or complete edentulism, either clinically observed or self-reported. Given variation across studies, definitions were not standardised post hoc; when possible, outcomes were mapped to WHO core oral health indicators to improve comparability.

See Online for appendix 3

Methodological quality

We used the Newcastle–Ottawa Scale developed for cohort studies (appendix 2 p 11).[14] We categorised the risk

	Country and continent	Participants, n	Follow-up	Age range, years	Criteria for diabetes diagnosis	Oral disease assessed	Diagnostic criteria for oral disease	Effect size	Confounders and mediators	Funding
Cheng et al (2025)[20]	China (Asia)	446	12 weeks minimum	19–45	FPG	Periodontitis	EFP or AAP (2018)	RR	Age, gestational age, pre-pregnancy BMI, household income, occupation, education, smoking, alcohol consumption, and family history of hypertension and diabetes	Research grant
Yoneda et al (2025)[28]	Japan (Asia)	4071	12 years	35–55	HbA_{1c}	Periodontitis	CPI	HR	Sex, age, family history of diabetes, obesity, smoking habits (non-smoker, ex-smoker, or current smoker), drinking habits (non-drinking, alcohol intake of <20 g/day, or alcohol intake of ≥20 g/day), exercise habits (yes or no), use of antihypertensive medications, use of lipid-lowering drugs, total energy intake (quartiles), dietary fibre intake (quartiles), and fasting blood glucose concentrations	Research grant
Naseer et al (2024)[36]	Ireland (Europe)	2539	4 years	≥50	Self-reported	Periodontitis and tooth loss	CPITN or clinical observation for tooth loss	RR	Age, sex, educational attainment, area of residence, smoking status, and BMI	Research grant
Nabila et al (2023)[21]	South Korea (Asia)	14523	5 years	≥20	ICD-10	Periodontitis	ICD-10	HR	Age, sex, lifestyle factors, dental behaviour, diabetes, and cardiovascular diseases	Research grant
Ju et al (2023)[17]	Australia (Oceania)	775	12 years	≥27	Self-reported	Periodontitis	EFP or AAP (2018)	IRR	Age, sex, country of birth, language spoken at home, residential location, highest education qualification, difficulty paying a US$200 dental bill, equivalised household income, socioeconomic factors, number of teeth (in 2004–06), oral and general health, and related behavioural factors	Research grant
Gibson et al (2023)[43]	Australia (Oceania)	213389	Unclear	>45	Self-reported and ICD-10	Tooth loss	Self-reported	HR	Age and sex, adjusted for age, sex, sociodemographics (education, SEIFA, household income, language, country of birth, and private health insurance), lifestyle (BMI, smoking, diet, and physical activity) and adjusted for all demographic and health-related factors (family history of diabetes, cardiovascular disease, blood pressure, and treatment for high cholesterol)	Research grant
Liu et al (2022)[22]	China (Asia)	3523	12 weeks minimum	≥18	FPG	Periodontitis	EFP or AAP (2018)	RR	Age, socioeconomic status, smoking, alcohol, and pregnancy-related information	Research grant
Chaparro et al (2021)[24]	Chile (South America)	358	12 weeks minimum	18–40	FPG	Periodontitis	EFP or AAP (2018)	RR	Adjusted for age, BMI, and MMP-8	Research grant
Zhang et al (2021)[30]	USA (North America)	5569	19·4 years average	62·4 (SD 5·6)	Self-reported	Periodontitis	PPC stages	HR	Age, sex, BMI, health parameter, and education level	Research grant
Holmlund et al (2021)[26]	Sweden (Europe)	8983	21·3 years average	20–85	ICD-10	Periodontitis	PPD ≥5 mm	IRR	Age, sex, education level, civil status, and smoking	Research grant

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	Country and continent	Participants, n	Follow-up	Age range, years	Criteria for diabetes diagnosis	Oral disease assessed	Diagnostic criteria for oral disease	Effect size	Confounders and mediators	Funding
Schmolinsky et al (2019)[39]	Germany (Europe)	3731	11 years	≥18	Self-reported and HbA_{1c}	Dental caries	WHO criteria; carious defects, fillings, secondary caries, and missing teeth were registered by surface (occlusal, mesial, distal, vestibular, and oral), with the exception of wisdom teeth	OR	Age (cubic splines with four knots), gender, school education, smoking, dental visits, interdental cleaning, tooth brushing frequency, partnership, and waist circumference as fixed effects	Not reported
Poulsen et al (2019)[40]	Finland (Europe)	112	5 years	No-gestational diabetes (39 [SD 6]); gestational diabetes (40 [SD 4])	HbA_{1c} and OGTT	Dental caries	The oral health examination included a full mouth examination and panoramic x-rays of the jaws; DMFT index	Correlation	NA	Research grant
Joshipura et al (2018)[25]	Puerto Rico (North America)	941	2·96 years (IQR 2·88–3·01)	50·6 (SD 6·8)	FPG and HbA_{1c}	Periodontitis and tooth loss	CDC–AAP (2012) or clinical observation for tooth loss	IRR	Adjusted for age, gender, smoking status, family history of diabetes, education, waist circumference, alcohol consumption, and physical activity (METs)	Research grant
Myllymäki et al (2018)[18]	Finland (Europe)	395	15 years	≥70	FPG	Periodontitis and tooth loss	PPD 4–5 mm and PPD ≥6 mm; or clinical observation for tooth loss	RR	Gender, risk of diabetes, physical activity, dietary habits, smoking status, BMI, absolute change in BMI during the follow-up period (continuous variable), IGT at baseline, hypertriglyceridaemia, low HDL-C, and arterial hypertension	None
Kumar et al (2018)[19]	India (Asia)	584	12 weeks	20–35	FPG	Periodontitis	≥4 teeth having ≥1 sites with PD ≥4 mm and CAL ≥3 mm associated with BOP	HR	Age, BMI, anaemia, preterm delivery, and family history of diabetes	None
Kebede et al (2018)[23]	Germany and Poland (Europe)	2047	11·1 years average	56·8 (SD 10·3)	FPG and HbA_{1c}	Periodontitis	PPD ≥3 mm and cumulative PPD (sum of the deepest PPDs ≥4 mm per tooth) and categorised as either Q1–Q4 or analysed continuously; mean CAL (as secondary) and percentage of sites with CAL ≥3 mm were calculated on the subject level and categorised as either Q1–Q4 or analysed continuously	IRR	Age, gender, highest level of general education, marital status, waist circumference, physical activity, smoking status (five categories, including pack-years), dental visits in past 12 months, and follow-up time	Research grant
Winning et al (2017)[29]	Northern Ireland (Europe)	1331	7·8 years average	58–72	FPG	Periodontitis	CDC–AAP (2007)	HR	Age, number of teeth, smoking status, tooth brushing frequency, baseline BMI, cholesterol, history of atherosclerotic cardiovascular disease, history of hypertension, history of education years, dental attendance, marital status, and socioeconomic status	Research grant
Miyawaki et al (2016)[33]	Japan (Asia)	2469	5 years	36–55	FPG and HbA_{1c}	Periodontitis	Self-reported	RR	Age, current smoking habits, BMI, family history of diabetes, hypertension, heavy alcohol consumption (40 g/day), and exercise habits (>30 min, 2 days/week)	Research grant

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	Country and continent	Participants, n	Follow-up	Age range, years	Criteria for diabetes diagnosis	Oral disease assessed	Diagnostic criteria for oral disease	Effect size	Confounders and mediators	Funding
(Continued from previous page)										
Chiu et al (2015)[35]	Taiwan (Asia)	4387	5 years	35–44	FPG	Periodontitis	CPI	HR	Age, sex, years of education, betels consumed, smoking, alcohol, waist circumference, triglyceride concentration, HDL, elevated blood pressure, fruit intake, and physical activity	Not reported
Oluwagbemigun et al (2015)[41]	Germany (Europe)	24 313	13 years	35–64	Self-reported	Tooth loss	Self-reported	HR	Age (continuous), sex, BMI (continuous), education (three categories), occupation (four categories), lifestyle (smoking [never, former, current; number of cigarettes per day], alcohol consumption [continuous], physical activity [Cambridge physical activity index], use of vitamin or mineral supplements, antibiotics and non–steroidal anti–inflammatory drugs, hormone replacement therapy [women], prevalent diseases, and three retained factors from factor analysis of 49 food groups)	Research grant
Liljestrand et al (2015)[42]	Finland (Europe)	7629	13·9 years average	25–74	Self-reported and drug-based	Tooth loss	Clinical inspection	HR	Age, sex, BMI, smoking (yes or no), physical inactivity, parent with diabetes, C-reactive protein (log), and a geographical variable	Research grant
Haas et al (2014)[32]	Brazil (South America)	653	5 years	19–94	Self-reported	Periodontitis	Periodontal AL progression ≥3 mm in ≥2 or ≥4 teeth	RR	Univariate model	Research grant
Morita et al (2012)[31]	Japan (Asia)	5856	5 years	30–69	HbA_{1c}	Periodontitis	CPI	RR	Adjusted for BMI, alcohol consumption, smoking status, sex, and age	Research grant
Jimenez et al (2012)[37]	USA (North America)	35 247	20 years	40–75	Self-reported	Periodontitis and tooth loss	Self-reported or clinical observation for tooth loss	HR	Age, race (White, Black, Asian, or other), smoking (comprehensive smoking index), BMI, fruit and vegetable intake (quintiles), physical activity (quintiles), alcohol consumption (0, 0·1–4·9, 5–14·9, 15–29 and ≥30 g/day), dental profession (yes or no), baseline history of stroke, coronary artery bypass surgery or myocardial infarction (yes or no), and number of teeth at baseline (≥25 *vs* <25)	Research grant
Ide et al (2011)[27]	Japan (Asia)	5848	6·5 years average	30–59	FPG	Periodontitis	CPI	HR	Age, sex, smoking (current, past, or never smoker), high BMI (≥25 kg/m² *vs* <25 kg/m²), high triglyceride concentration (≥150 mg/dL *vs* <150 mg/dL), hypertension (systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg *vs* neither), low high-density lipoprotein cholesterol concentration (<40 mg/dL in males or <50 mg/dL in females *vs* neither), and high γ-glutamyl transpeptidase (>60 IU/L in males or >40 IU/L in females *vs* neither)	Research grant

(Table continues on next page)

of bias as: low (7–9 stars); moderate (4–6 stars), or high (1–3 stars) as previously reported.[15] Two reviewers independently assessed study quality (JB and VM), with any disagreements resolved through discussion with a third reviewer (FVB). We assessed the inter-examiner agreement through Cohen's kappa, and an excellent (k>0·80) level of agreement was found (k=0·92, 95% CI 0·89–0·95).

Data synthesis

Extracted data were organised into evidence tables. A meta-analysis of binary outcomes was conducted using the meta package in R, using a random-effects model based on the restricted maximum-likelihood estimator. This approach accounts for heterogeneity between studies by assuming that the true effect size varies across studies. The pooled effect estimates (eg, risk ratio) and 95% CIs were calculated. Heterogeneity was explored through the I^2 index, Cochrane's Q statistic (p<0·1), and χ^2 test for the overall homogeneity.[13] Substantial heterogeneity was defined as I^2 >50%. All tests were two-tailed, with alpha set at 5%. Publication bias was computed via Egger's significance test.[13]

We conducted a series of sensitivity analyses to examine the effect of the case definition confidence (confident *vs* non-confident) and risk of bias (low *vs* moderate-high) on the overall estimates. Meta-regression was used to explore adjusting particular confounding variables on the prevalence estimates, particularly sex ratio, geographical location (using latitude and longitude), and study sample size.

In the case of limited studies to compute meta-analytical estimates, we reported results following the synthesis without meta-analysis guidelines.[16]

Results

Our search retrieved a total of 45 477 entries. After removing duplicates (n=17 440), a total of 28 037 records were screened for title and abstracts against the eligibility criteria. 671 studies were excluded (figure 1; appendix 3 p 1). A final sample of 28 longitudinal studies were included for qualitative analysis,[17–44] four of which contributed quantitative data for both periodontitis and tooth loss.[18,25,36,37]

Of the 28 studies included (table; appendix 4 pp 1–4), most were conducted in Europe (n=10)[18,23,26,29,36,38–42] and Asia (n=9);[19–22,27,28,31,33,35] others were conducted in North America (n=5),[25,30,34,37,44] Oceania (n=2),[17,43] and South America (n=2).[24,32] Most studies reported data from regional settings (n=11),[18,23,25,27,29,30,32,33,35,39,41] followed by national (n=8)[17,21,36,37,40,42–44] and local (n=8)[19,20,22,24,26,31,34,38] settings, with one study not clarifying the geographical scope (n=1).[28] Most studies focused on population-based epidemiologic studies (n=22)[17,18,20,21,23,25,27–37,39,41–44] rather than university clinic cohorts (n=6).[19,22,24,26,38,40] With respect to diabetes classification, type 2 diabetes was the most reported

	Country and continent	Participants, n	Follow-up	Age range, years	Criteria for diabetes diagnosis	Oral disease assessed	Diagnostic criteria for oral disease	Effect size	Confounders and mediators	Funding
(Continued from previous page)										
Demmer et al (2008)[44]	USA (North America)	2127	4 years	25–74	Medical history and ICD-9	Tooth loss	Clinical inspection	OR	Age (continuous), sex, BMI (continuous), education (three categories), occupation (four categories), lifestyle (smoking [never, former, current; number of cigarettes per day], alcohol consumption [continuous], physical activity [Cambridge physical activity index], use of vitamin or mineral supplements, antibiotics and non-steroidal anti-inflammatory drugs, hormone replacement therapy [women], prevalent diseases, and three retained factors from factor analysis of 49 food groups)	Research grant
Twetman et al (2002)[38]	Sweden (Europe)	1768	3 years	8–16	Medical record	Dental caries	WHO criteria	OR	Unadjusted	Not reported
Taylor GW et al (1996)[34]	USA (North America)	105	2 years	18–67	HbA_{1c}	Periodontitis	Baseline radiographic bone loss of 50% or more for at least one tooth; baseline maximum loss of periodontal attachment of 6 mm or more for at least one index tooth	RR	Unclear	Research grant

AAP=American Academy of Periodontology. AL=attachment loss. BOP=bleeding on probing. CAL=clinical attachment level. CDC=US Centers for Disease Control and Prevention. CPI=Community Periodontal Index. CPITN=Community Periodontal Index of Treatment Needs. DFMS=Decayed, missing, and filled surfaces. DMFT=decayed, missing, and filled teeth. EFP=European Federation of Periodontology. FPG=fasting plasma glucose. Hba1c=A1c glycated haemoglobin. HDL-C=high density lipoprotein cholesterol. HR=hazard ratio. IGT=impaired glucose tolerance. IRR=incidence rate ratio. MET=metabolic equivalent of task. MMP-8=matrix metalloproteinase-8. NA=not applicable. OGTT=Oral glucose tolerance test. OR=odds ratio. PD=probing depth. PPC=periodontal profile class. PPD=probing pocket depth. RR=risk ratio. SEIFA=Socio-Economic Indexes for Areas.

Table: **Main characteristics of the studies included in the systematic review**

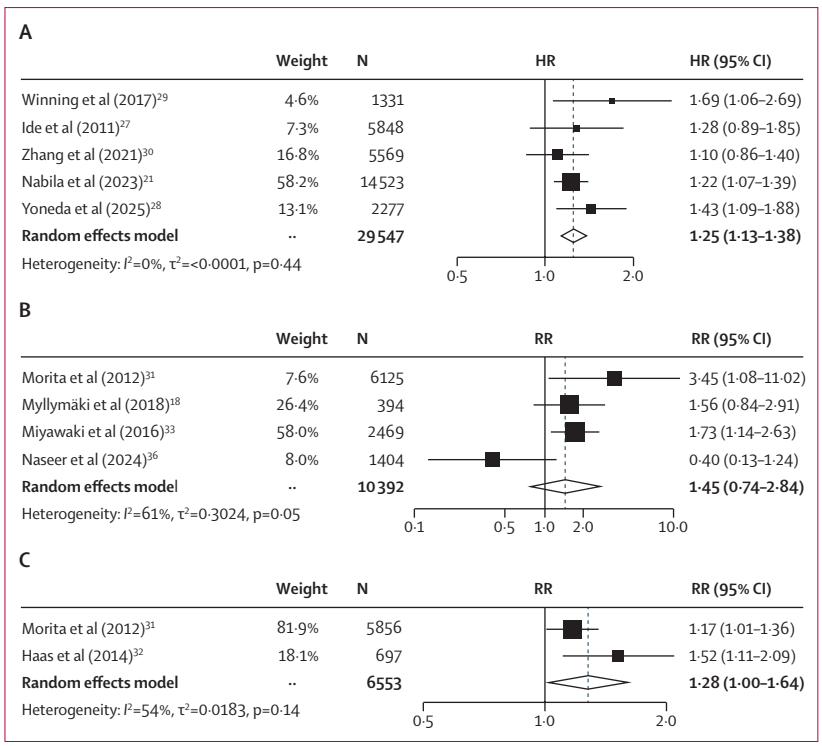

Figure 2: **Association between periodontitis and diabetes**
(A) Forest plot of the association between periodontitis and incident diabetes expressed as HRs, using the severest degree (most severe periodontitis category reported within each study) of periodontitis reported. (B) Forest plot of the association between periodontitis and diabetes expressed as RRs, using the severest degree of periodontitis reported. (C) Forest plot of the association between diabetes and periodontitis expressed as RRs, using the severest degree of periodontitis reported. Pooled effect estimates were calculated using random-effects (mixed-effects) meta-analysis. Squares represent study-specific estimates (size proportional to weight), horizontal lines indicate 95% CIs, and diamonds represent pooled effects. Heterogeneity statistics (I^2, τ^2, and p values) are presented for each analysis. HR=hazard ratio. RR=risk ratio.

See Online for appendix 4
See Online for appendix 5

(n=15),[18,23,25–31,33–37,41] followed by gestational diabetes (n=5),[19,20,22,24,40] and type 1 diabetes (n=1).[38] In seven studies, the diabetes type was undefined (n=7).[17,21,32,39,42–44]

The 28 included studies predominantly enrolled adult populations (89·3%, n=25), with ages typically ranging from 18 to 94 years. Most cohorts comprised mixed-sex populations, although several studies focused exclusively on women (focusing on gestational diabetes)[19,20,22,24,40] or men.[29,33,37] Only one study included children or adolescents.[38] Follow-up duration varied widely, from short-term pregnancy-related cohorts of approximately 12 weeks to long-term population-based cohorts exceeding 20 years, with most studies following participants for 5–12 years. Socioeconomic position and ethnicity data were absent or inconsistently reported; when available, studies adjusted for socioeconomic indicators such as education or income, but detailed stratified analyses were uncommon.

Regarding diagnostic tests, fasting plasma glucose (FPG) was used in 11 studies and HbA_{1c} in eight studies; three studies used both markers. Overall, 16 of the 28 studies used at least one glycaemic biomarker (FPG or HbA_{1c}), whereas the remaining studies relied on self-report, medical records, or diagnostic codes. Across core WHO oral outcomes, periodontal disease was the predominant endpoint (n=21), followed by tooth loss or edentulism (n=8) and dental caries (n=3); there were no studies concerning oral cancer.

No studies were categorised as low methodological quality. 71·4% of the studies (n=20) were judged as being of high methodological quality (ie, low risk of bias), while the remaining (28·6%, n=8) were of moderate methodological quality (ie, moderate risk of bias). The discriminative results are detailed in appendix 5 [p 1]). The most frequent sources of potential bias were related to representativeness of the cohort (39·3%, n=11), exposure ascertainment (35·7%, n=10), outcome assessment (32·1%, n=9), and completeness of follow-up (28·6%, n=8); confounder control and cohort comparability were consistently adequate (100%).

For periodontitis, 21 studies reported quantitative data: eight reported hazard ratio (HR) data,[19,21,27–30,35,37] nine reported risk ratio (RR) data,[18,20,22,24,31–34,36] and four reported incidence rate ratio (IRR; appendix 2 p 13).[17,23,25,26]

For analyses considering periodontitis status at baseline and newly incident cases of diabetes during follow-up, six studies reported HR data from longitudinal studies with a follow-up ranging from 5 to 20 years. Across for 29 265 participants, pooled HR estimates indicated a 18–25% higher occurrence of newly incident type 2 diabetes cases among individuals with periodontitis compared with those without periodontitis (figure 2A; appendix 2 p 14). Male to female ratio (estimate=0·401, SE=0·361, p=0·267) and geographical location (either with latitude [estimate=0·02, SE=0·01, p=0·106] or longitude [estimate=0·000, SE=0·001, p=0·632]) did not significantly influence the estimates, via meta-regression. The direction of this association is also corroborated by meta-analysis of RR (figure 2A; appendix 2 p 14). A higher effect size was found for studies reporting RR, but only using the severest staging reported (p=0·283; figure 2B). For the mildest degree of periodontitis plot and a summarised table, see appendix 2 (pp 14–15).

For analyses considering diabetes status at baseline and newly incident cases of periodontitis, three studies reported HR (50 167 participants) and three reported RR (6553 participants). The HR analysis yielded a pooled, adjusted estimate of 1·29 (95% CI 1·01–1·66), indicating considerable imprecision (appendix 2 p 14). The pooled RR analysis indicated a moderate association between diabetes and periodontitis (RR 1·28 [95% CI 1·00–1·64]), with moderate heterogeneity differences between the studies (I^2=31%; figure 2C).

When examining IRRs for periodontitis status at baseline and diabetes incidence (n=11 971) a pooled rate of 0·97 (95% CI 0·74–1·28; appendix 2 p 15) suggested a very small and not significant effect (p=0·8464).

For gestational diabetes (n=4327), pregnant women with periodontitis at baseline showed higher newly

incident gestational diabetes cases during pregnancy (RR 1·90 [95% CI 1·07–3·37], p=0·0280; appendix 2 p 15).

For dental caries, three longitudinal studies were included.[38–40] Owing to substantial clinical and methodological heterogeneity, quantitative pooling was not appropriate, and findings were synthesised narratively (detailed in appendix 2 [p 16]). Two cohort studies reported that poorer glycaemic control at baseline was associated with greater incidence or progression of caries over time, whereas individuals with well controlled diabetes showed caries trajectories comparable to those without diabetes.[38,39] Individuals with well controlled diabetes showed caries trajectories similar to those without diabetes. In contrast, one smaller follow-up study of women with previous gestational diabetes found no adverse differences in caries.[40]

For tooth loss and edentulism, eight studies were included both in the qualitative and quantitative analysis.[18,25,36,37,41–44] Three studies registered missing teeth based on self-reports from participants[37,41,43] and five studies registered missing teeth based on clinical assessment.[18,25,36,42,44]

Meta-analyses of HR (226 866 participants) indicated a higher hazard of incident diabetes among people with higher tooth loss than those with lower tooth loss, with pooled estimates corresponding to 12% (95% CI 8–16) using the lowest threshold of tooth loss and 20% (14–26) using the highest threshold of tooth loss (figure 3A; appendix 2 p 17).

For analyses considering edentulism at baseline and diabetes incidence, edentulous participants experienced a higher occurrence of newly diagnosed diabetes cases compared with dentate individuals (RR 1·30 [95% CI 1·02–1·64], p=0·0329; figure 3B), with low heterogeneity (I^2=6·3%) and minimal variability between studies (τ^2<0·0001).

Conversely, for diabetes at baseline and newly incident cases of tooth loss, Jimenez et al reported a significant association of 1·09 (95% CI 1·01–1·18), despite the the lack of more studies to compute pooled estimates.

There were no longitudinal studies available to produce synthesis on oral cancer.

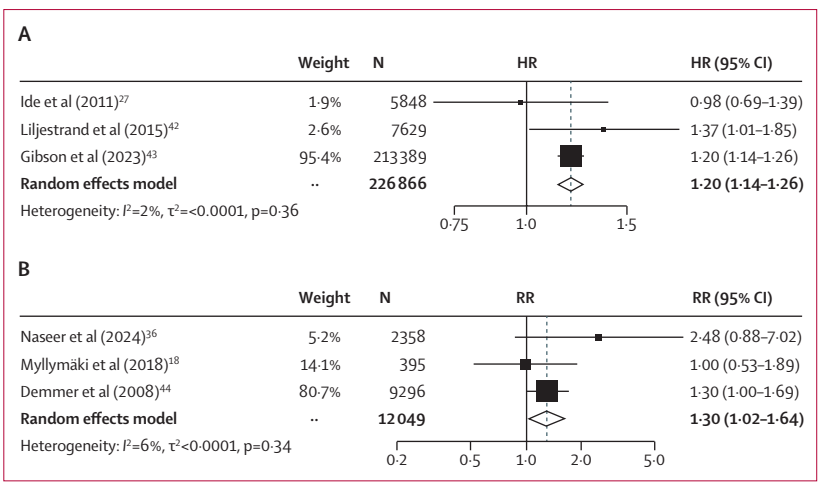

Figure 3: **Association between tooth loss and edentulism with diabetes**
(A) Forest plot of the association between tooth loss and diabetes expressed as HRs, considering the highest thresholds of tooth loss reported. Highest threshold refers to the most severe tooth loss category reported within each study. (B) Forest plot of the association between edentulism and diabetes expressed as RRs. Pooled effect estimates were calculated using random-effects (mixed-effects) meta-analysis. Squares represent study-specific estimates (size proportional to study weight), horizontal lines indicate 95% CIs, and diamonds represent pooled effects. Heterogeneity statistics (I^2, τ^2, and p values) are presented for each analysis. HR=hazard ratio. RR=risk ratio.

Discussion

This meta-analysis of 28 longitudinal studies—encompassing over 300 000 participants from 16 different countries—is, to the best of our knowledge, the first to produce pooled estimates for the bidirectional association of diabetes and core WHO oral diseases. Individuals with diabetes have a higher incidence of new cases of periodontitis and edentulism than those without diabetes, most commonly periodontitis and tooth loss. In pooled analyses, having diabetes was associated with a 28% increased RR of having periodontitis and a 12–20% increased HR of experiencing tooth loss. Individuals with periodontitis had a 18–25% increased risk of developing diabetes than those without periodontitis during a range of 5–20 years of follow-up, and edentulous individuals had a 30% higher risk of developing diabetes than dentate individuals. Although elucidating mechanisms was not the primary aim of this review, these bidirectional associations are probably mediated by chronic low-grade inflammation, dysregulated immune responses, and shared risk factors that link periodontal inflammation with systemic glucose imbalance.[7,8] In summary, these two-way associations suggest that linking oral and systemic health data could enhance how we predict diseases and shape public health strategies. Evidence for dental caries was scarce; however, available data suggest that caries progression was more consistently observed among individuals with poorer glycaemic control.[38,39] This finding is in line with previous evidence showing an association between elevated HbA_{1c} concentrations and caries, measured through decayed, missed, and filled teeth index.[45]

A previous systematic review found that diabetes was associated with an increased risk of onset or progressive periodontitis (RR 1·86 [95% CI 1·30–2·80]).[15] Although our findings are directionally consistent with this previous evidence, we focused on primary studies reporting HRs or RRs, and uniquely assessed bidirectional associations across core WHO oral health outcomes. HRs and RRs can be comparable under highly constrained conditions (eg, identical follow-up periods and risk intervals); however, such assumptions are seldom satisfied. Consequently, in line with Cochrane's methodological guidance,[13] pooling these measures would violate key assumptions and impair interpretability; therefore, HRs and RRs were analysed separately in our study.

Previous evidence also supported an association between tooth loss and diabetes.[46,47] However, both

syntheses included observational designs without restricting to longitudinal evidence, assessed diabetes only as an exposure, and did not examine edentulism or explore the reverse causal pathway. Both studies explored the effect of diabetes on tooth loss, but none focused also on edentulism. Weijdijk and colleagues[46] reported a higher risk of tooth loss among people with diabetes (RR 1·63 [95% CI 1·33–2·00], p<0·00001) than those without diabetes and Ahmadinia and colleagues[47] reported a similar effect (1·29 [1·07–1·51]) when analysing cohort studies. In our review, we doubled the number of eligible studies compared with the previous two studies,[46,47] focused exclusively on prospective longitudinal designs, and were able to evaluate both directions of the association. The results show that not only is diabetes associated with a higher incidence of tooth loss over time, edentulism is also associated with a higher incidence of new cases of type 2 diabetes. Although evidence suggests that socioeconomic inequalities might amplify the risk of complete edentulism among people with type 1 and type 2 diabetes,[47] none of the included studies reported socioeconomic indicators, warranting their inclusion in future research.

For dental caries, limited longitudinal evidence suggests that poorer metabolic control in people with diabetes might be associated with greater caries progression. However, these associations should be interpreted cautiously, as glycaemic control might act as a proxy for broader behavioural, dietary, and health care-related factors that also influence caries risk. In children and adolescents with type 1 diabetes[38] and in adults followed up over 11 years,[39] higher HbA_{1c} values were associated with a greater increase in caries surfaces over time, whereas those with good metabolic control at baseline did not show this progression. The body of evidence was heterogeneous and did not support meta-analysis, but collectively it suggests that diabetes metabolic control might play a more substantial role in oral health outcomes than the mere presence or history of diabetes. However, these preliminary results require further longitudinal investigation.

This review reinforces that diabetes and oral diseases should not be managed in isolation. Incorporating oral health assessments into diabetes management can enhance patient outcomes, and screening for diabetes risk during dental visits offers an additional opportunity for early detection. Implementing interdisciplinary care models connecting oral health care, primary care, and diabetes care can help reduce complications, improve metabolic outcomes, and support WHO's goals for prevention and management of non-communicable diseases.[48,49]

This study has several strengths and limitations that are crucial to consider when interpreting the findings. The meta-analysis focused exclusively on longitudinal studies, which might contribute to maximising inference and minimising reverse causation. Although residual confounding cannot be entirely ruled out, such an approach helps reduce, but does not eliminate, these effects. The bidirectional assessment is a novel and important finding, based on gold-standard and rigorous approaches, including duplicate screening, extraction and quality assessment with excellent inter-examiner agreement, and builds upon lessons learned from a previous umbrella review.[5] Methodological considerations were transparently addressed through protocol pre-registration, external review, and comprehensive reporting, thereby reducing reporting bias and enhancing reproducibility. The inclusion of core WHO oral health outcomes spanning all four major oral diseases strengthens the comprehensiveness of this study. Additionally, this research draws on a large, geographically diverse evidence base comprising over 300 000 participants from 16 countries, thereby improving the generalisability of the findings.

However, several limitations must be acknowledged. There were very heterogeneous diagnostic criteria for both diabetes (self-report *vs* FPG *vs* HbA_{1c}), periodontitis (multiple case definitions), and caries (decayed, missed, and filled teeth index inconsistencies). In addition, self-reported diabetes or tooth loss in some studies might have introduced measurement error. There was a small number of caries studies and no eligible prospective longitudinal evidence on oral cancer. There was also a diversity of the effect measures (HR, RR, odds ratio) across studies and, as discussed previously, combining them would be statistically inappropriate. Most included studies reported effect estimates from multivariable-adjusted models accounting for major shared risk factors (including age, sex, and smoking). Nevertheless, residual or unmeasured confounding cannot be fully excluded. Also, data on metabolic control were scarce, which prevented pooled estimation of the effect of glycaemic control on oral health outcomes. Despite these limitations, the consistency of associations across populations, analytical approaches, and temporal directions supports the robustness of the findings. In addition, for some outcomes, no longitudinal evidence was identified, reflecting a gap in prospective research rather than the exclusion of eligible studies. Although cross-sectional data exist, restricting to longitudinal data aimed to preserve temporal inference. It is important to mention that most included studies were conducted in high-income countries. This under-representation of socially and geographically diverse populations highlights an important gap and underscores the need for longitudinal studies in low-income and middle-income settings. Importantly, data could not be disaggregated by gender or ethnicity, thus our estimates could not account for these possible confounding factors.

In conclusion, prospective longitudinal evidence indicates associations between diabetes and periodontitis in both directions, although the strength of evidence is asymmetric. Diabetes was also associated with

subsequent tooth loss and vice-versa, whereas edentulism was associated with higher incidence of new cases of diabetes. Longitudinal research remains scarce for dental caries and absent for oral cancer. Integrating oral health within diabetes prevention and management represents an important public health opportunity to address these inter-related conditions.

Contributors

All authors conceptualised the study. JB, LP, VM, and FVB developed the protocol. JB and VM did the literature search and independently screened studies and extracted data. JB did all meta-analyses and LP provided advice on statistical analysis. JB drafted the paper. All authors provided critical input during the writing and revision of the paper, and all authors accept responsibility for the decision to submit the manuscript.

Declaration of interests

We declare no competing interests.

Data sharing

As a systematic review with meta-analysis, this study uses secondary data. We have deposited an open-access dataset of all data during submission. All data generated or analysed during this study are included in the published article (and its online supplementary files).

Acknowledgments

The WHO Oral Health Programme commissioned this research with financial support from the WHO Global Diabetes Compact. The authors alone are responsible for the views expressed in this publication and they do not necessarily represent views, decisions, or policies of WHO. FVB is supported by a research grant from the Danish Diabetes and Endocrine Academy (grant number NNF22SA0079901). We thank Bianca Hemmingsen and Sanjana Marpadga from the WHO Global Diabetes Compact for their valuable input and critical revision of the manuscript, which contributed to improving its clarity and scientific quality.

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