

PERIODONTITIS AND CARDIOVASCULAR DISEASE: A NARRATIVE
REVIEW OF EVIDENCE FROM PUBLISHED META-ANALYSES,
CONSENSUS REPORTS AND INTERVENTION STUDIES

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DOI: <https://doi.org/10.5281/zenodo.22914027>

Keywords

periodontitis; cardiovascular diseases; coronary heart disease; stroke; heart failure; Mendelian randomization; narrative review

Article History

Received: 01 June 2026

Accepted: 23 August 2026

Published: 23 September 2026

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Abstract

Background: Periodontitis affects more than one billion people worldwide and has been linked to cardiovascular disease (CVD) in many observational studies. Whether the link is causal, and whether periodontal treatment reduces cardiovascular risk, remains debated.

Aim: To review published evidence on the association between periodontitis and cardiovascular outcomes, the proposed biological mechanisms, the effect of periodontal therapy, and the evidence for causality.

Methods: A structured narrative review was conducted. Meta-analyses, consensus reports, Mendelian randomization (MR) studies, randomized-trial syntheses and global burden estimates were identified through seven structured web-based queries returning Pub Med/PMC and publisher pages (September 2026), plus earlier exploratory queries and citation searching. Effect estimates were extracted and synthesized thematically; overlapping meta-analyses were compared rather than re-pooled.

Results: The global burden of severe periodontitis was about 1.07 billion cases in 2021. Meta-analyses of cohort studies report a 13–34% higher relative risk of cardiovascular events in people with periodontitis; the largest (39 cohorts, 4,389,263 participants) found RR 1.24 for major adverse cardiovascular events, 1.26 for stroke and 1.42 for cardiac death. Case-control and cross-sectional studies gave larger estimates than cohorts (OR up to 2.93 vs 1.18 for myocardial infarction). Periodontitis was also associated with heart failure (RR 1.58) and atrial fibrillation (HR 1.03–1.31).

Periodontal therapy lowered systolic blood pressure by 4.64 mmHg and C-reactive protein by 0.58 mg/L in a meta-analysis of 12 trials, but no adequately powered trial has shown reduced cardiovascular events. Most MR studies found no evidence of a causal effect.

Conclusion: Periodontitis is a consistent marker of higher cardiovascular risk, and professional bodies recommend informing patients accordingly. Causality remains unproven, and trials of periodontal therapy with hard cardiovascular endpoints are needed.

1. INTRODUCTION

Periodontitis is a chronic multifactorial inflammatory disease in which microbial dysbiosis triggers destruction of the tooth-supporting tissues, and its current classification rests on staging and grading [4]. It is the most common cause of tooth loss in adults [3] and a major public health problem: the Global Burden of Disease (GBD) Study estimated 1.1 billion prevalent cases of severe periodontitis in 2019 [1], and more than one billion again in 2021, with a projected rise to over 1.5 billion by 2050 [2].

Atherosclerotic cardiovascular disease is the leading cause of death globally [5]. Two lines of reasoning connect it with periodontitis. First, biological pathways may link the conditions directly, through bacteraemia from ulcerated periodontal pockets, or indirectly, through chronic systemic inflammation [5,6]. Second, the two diseases share risk factors such as age, smoking and obesity, so an association may reflect confounding rather than causation [5,7].

A 2012 American Heart Association (AHA) statement concluded that periodontal disease is associated with atherosclerotic cardiovascular disease but called for more robust evidence on clinical outcomes and treatment effects [5]. Since then, new cohort data, meta-analyses, MR studies and intervention trials have accumulated, and the AHA updated its statement in 2025 [5]. Because these sources use different designs, exposure definitions and outcomes, a thematic synthesis is useful.

This narrative review therefore aims to summarise published evidence on (i) the burden of periodontitis, (ii) its epidemiological association with cardiovascular outcomes, (iii) the

biological mechanisms proposed, (iv) the effect of periodontal therapy on cardiovascular risk markers and events, and (v) the evidence for and against causality.

2. Methods

2.1 Review design

A structured narrative review was undertaken, guided by the SANRA quality criteria for narrative reviews [26]. Because a narrative design aims to interpret a broad body of evidence rather than to answer a single pooled question, no formal meta-analysis of primary studies was performed.

2.2 Literature identification

Seven structured queries (Table S1), each combining periodontitis with a cardiovascular or evidence-type term, were run on 19 September 2026 in a web search engine that returns PubMed/PMC, publisher and repository pages, and the first ten results of each query were logged. Sixteen further sources were retained from earlier exploratory queries on the same topics and from citation searching of consensus reports and key reviews. Bibliographic databases were not searched directly.

2.3 Selection criteria

- Included: systematic reviews and meta-analyses of cohort, case-control or cross-sectional studies; meta-analyses of randomized trials; MR studies; consensus statements; and GBD burden analyses, all in humans and in English.
- Prioritized: the most recent and largest syntheses, plus earlier landmark meta-analyses for historical comparison.

- Excluded: animal and in vitro studies (except where cited within consensus reports), case reports and conference abstracts without data.
- Screening exclusions: news items, blogs, commercial or index pages, citation lists, secondary summaries, narrative reviews without new synthesis, trial protocols and single primary trials.
- Overlap rule: where several syntheses covered the same cohorts or outcome, the largest or most recent was prioritized and the others were recorded as excluded with reasons (Table S2).

2.4 Data extraction and synthesis

For each source, the design, sample size, outcome, and effect estimate (relative risk (RR), hazard ratio (HR) or odds ratio (OR)) with 95% confidence interval were extracted. Findings were grouped into five themes: burden, epidemiological association, mechanisms, intervention effects and causal inference. Because published meta-analyses share many of the same primary cohorts, their summary estimates were compared side by side and were not re-pooled, to avoid double counting.

Source selection is displayed in a PRISMA 2020-style flow diagram [27] adapted for a narrative review (Figure 1); query-level counts are in Table S1 and sources assessed but not included are listed in Table S2. Numerical estimates are also displayed graphically (Figures 2–9). All charts plot values published in the cited sources; no new pooled analysis was performed. Derived

quantities (for example, the projected 2050 case count) are identified in the figure legends.

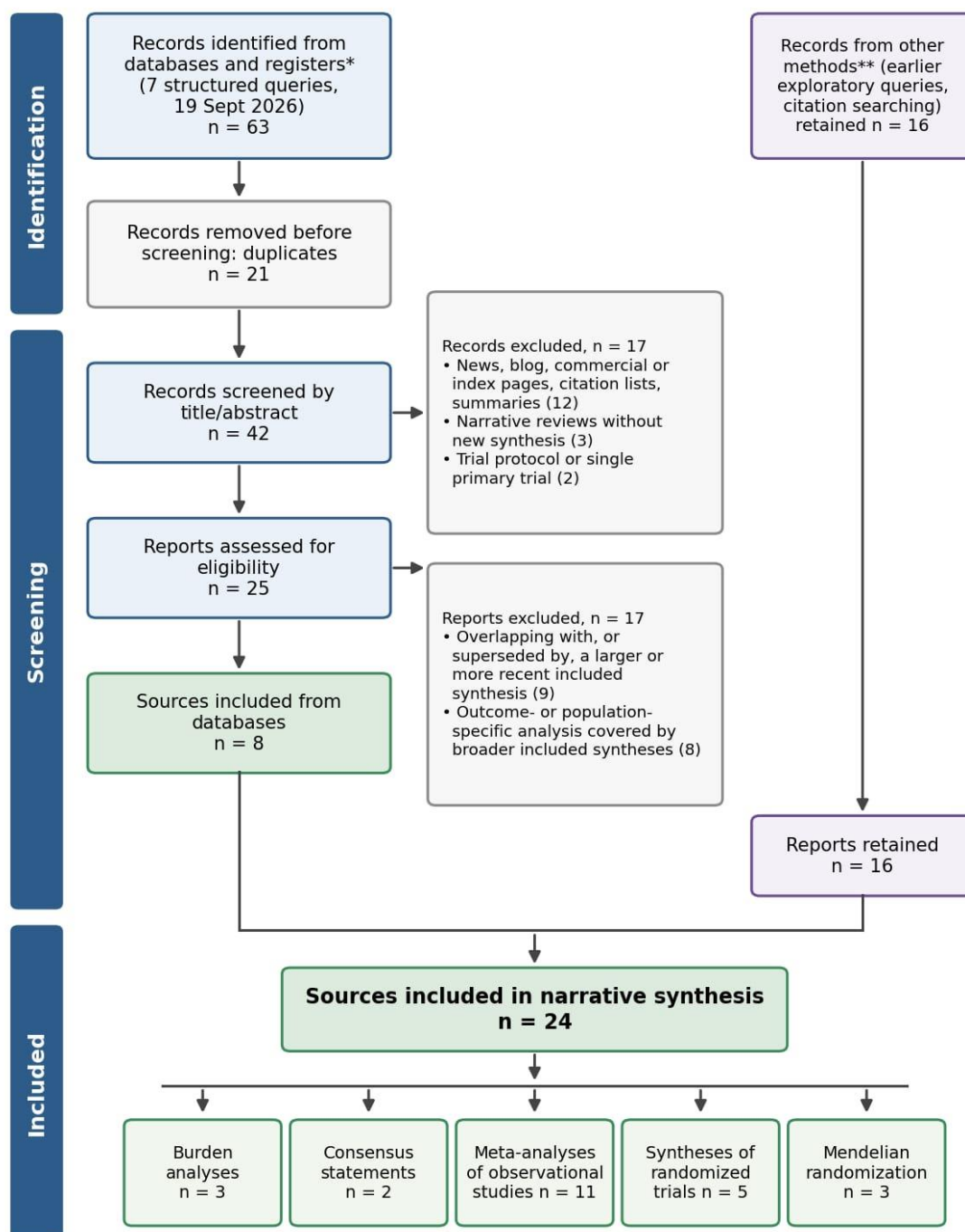
2.5 Ethical considerations

Ethical approval was not required because the review used only published data.

3. Results

3.1 Source selection and characteristics

The seven structured queries returned 63 records, of which 21 were duplicates. Of the 42 unique records screened by title and abstract, 17 were excluded (news, blogs, commercial or index pages, citation lists and summaries, $n = 12$; narrative reviews without new synthesis, $n = 3$; a trial protocol or single trial, $n = 2$). Of the 25 reports assessed for eligibility, 8 were included and 17 were excluded because they overlapped with or were superseded by a larger or more recent included synthesis ($n = 9$) or were outcome- or population-specific analyses covered by broader included syntheses ($n = 8$). Sixteen sources retained from earlier exploratory queries and citation searching brought the total to 24 included sources (Figure 1; Tables S1 and S2). Meta-analyses of observational studies were the largest group ($n = 11$), followed by syntheses of randomized trials ($n = 5$), Mendelian randomization analyses ($n = 3$), burden analyses ($n = 3$) and consensus statements ($n = 2$) (Figure 2A). The cohort-based meta-analyses included between 4 and 39 primary studies (Figure 2B).



* Web search engine returning PubMed/PMC, publisher and repository pages (up to ~10 records per query); queries in Table S1.

** Records not retained from earlier exploratory queries were not logged and are not counted.

Figure 1. PRISMA 2020-style flow diagram of source selection for the narrative review. Counts in the database stream come from the seven structured queries in Table S1. Sources retained from earlier exploratory queries and citation searching are shown as a separate stream because records not retained from those queries were not logged.

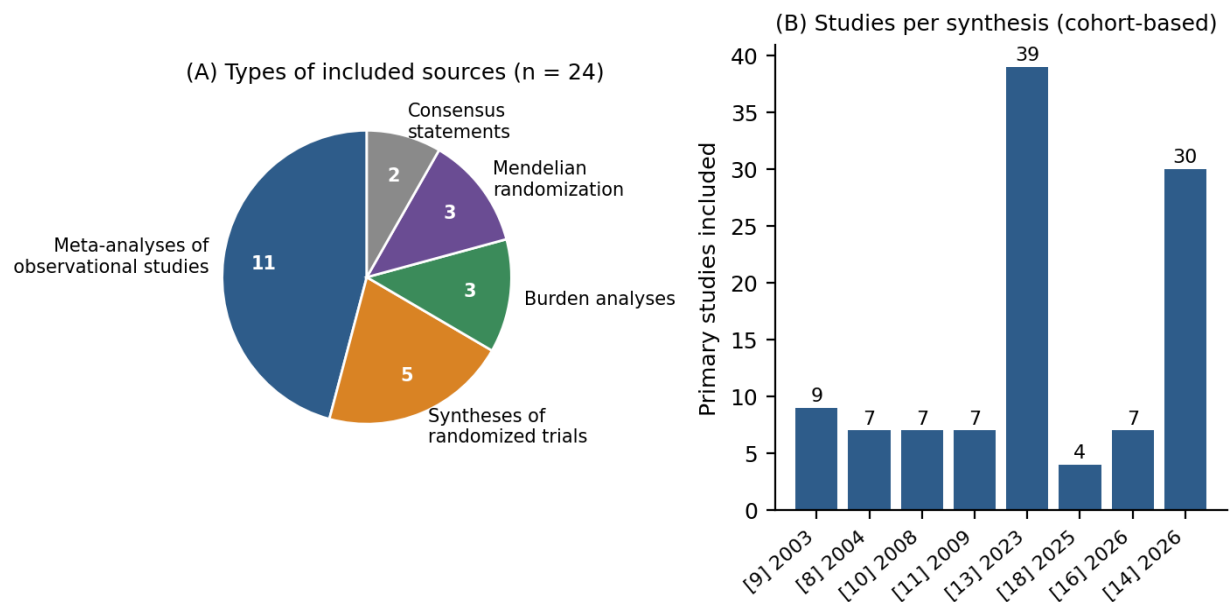


Figure 2. Characteristics of the included sources. (A) Types of source (n = 24). (B) Number of primary studies in cohort-based syntheses, labelled by reference number and year.

3.2 Burden of periodontitis

The GBD 2019 analysis reported 1.1 billion (95% uncertainty interval [UI] 0.8–1.4 billion) prevalent cases of severe periodontitis, with the age-standardized prevalence rising by 8.44% between 1990 and 2019 and higher prevalence in less developed regions [1]. In 2021, 1,066.95 million people (95% UI 896.55–1,234.84) were affected, an age-standardized prevalence of

12.50% (10.53–14.49), highest in South Asia at 17.57% [2]. Cases are projected to exceed 1.5 billion by 2050 (+44.32%) [2]. Between 1990 and 2021, cases rose by 91.54%, driven mainly by population growth (66.15%) and ageing (30.84%) [3]. The economic cost is large: about US\$154 billion in the United States and €159 billion in Europe in 2018 [3]. These figures are plotted in Figure 3.

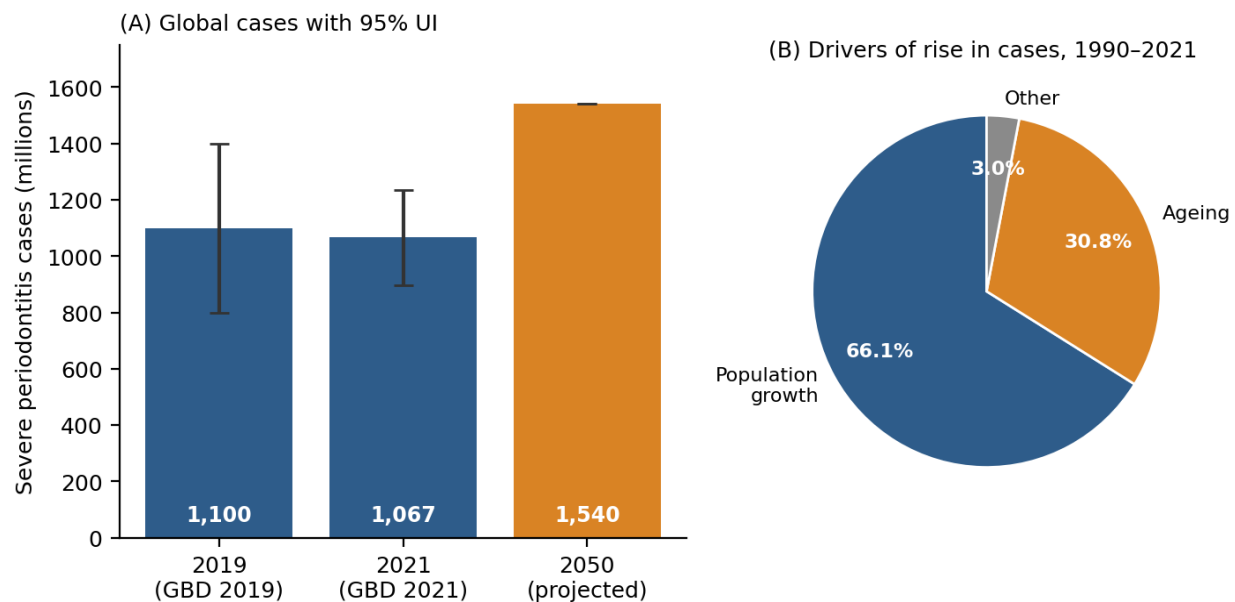


Figure 3. Global burden of severe periodontitis. (A) Prevalent cases with 95% uncertainty intervals for 2019 [1] and 2021 [2]; the 2050 bar is derived from the reported +44.32% projected increase over 2021 [2]. (B) Contributors to the rise in cases from 1990 to 2021 [3]; “Other” is the remainder after the two reported components.

3.3 Epidemiological association with cardiovascular outcomes

Table 1 summarizes the principal meta-analyses. Across cohort-based syntheses, pooled relative risks ranged from 1.13 to 1.34 [8–14]. The earliest meta-analyses found modest associations: RR 1.15 for coronary heart disease (CHD) and

1.13 for cerebrovascular disease [8], and RR 1.19 for future cardiovascular events [9]. Later work reported similar values, with summary RR of 1.24–1.34 for CHD depending on how periodontal disease was defined [10], and RR 1.34 in seven cohorts [11].

Table 1. Published meta-analyses of periodontal disease and cardiovascular outcomes

Ref	Year	Included evidence	Main pooled result (95% CI)
[9]	2003	9 cohort studies	Future CVD: RR 1.19 (1.08–1.32); age ≤65 y: RR 1.44 (1.20–1.73); stroke only: RR 2.85 (1.78–4.56)
[8]	2004	7 cohort + 4 other designs	CHD: RR 1.15 (1.06–1.25), no heterogeneity (P = 0.472); cerebrovascular disease: RR 1.13 (1.01–1.27)
[10]	2008	7 cohorts	CHD: summary RR 1.24 (1.01–1.51) to 1.34 (1.10–1.63) by periodontal exposure category
[11]	2009	29 studies pooled (of 47 observational)	Case-control/cross-sectional: OR 2.35 (1.87–2.96), 22 studies; cohorts: RR 1.34 (1.27–1.42), 7 studies

Ref	Year	Included evidence	Main pooled result (95% CI)
[12]	2017	Cross-sectional, case-control and cohort studies	MI: cross-sectional OR 1.71 (1.07–2.73); case-control OR 2.93 (1.95–4.39); cohort OR 1.18 (0.98–1.42)
[13]	2023	39 cohorts, 4,389,263 participants	MACE RR 1.24 (1.15–1.34); CHD 1.20 (1.12–1.29); MI 1.14 (1.06–1.22); stroke 1.26 (1.15–1.37); cardiac death 1.42 (1.10–1.84); all-cause mortality 1.31 (1.07–1.61)
[15]	2023	26 studies, sex-stratified	CVD: men OR 1.22 (1.12–1.34); women OR 1.11 (1.05–1.17); no significant sex difference ($P > 0.05$)
[14]	2026	30 cohorts (searched 2000–2024)	CVD: HR 1.31 (1.13–1.48); strongest for stroke; dose-response with periodontal severity; substantial heterogeneity

The reported estimates are plotted in Figure 4 without pooling. The largest synthesis, of 39 cohort studies, gave outcome-specific estimates (Figure 5): cardiac death (RR 1.42) and stroke (RR 1.26) showed the largest relative increases, and MI (RR 1.14) the smallest [13]. The 2026 review of 30 cohorts reported a 31% higher risk

(HR 1.31) using a broader periodontal/oral-health exposure definition, with the strongest link for stroke and a dose-response gradient by severity, while cautioning that substantial heterogeneity limits interpretation [14]. Sex-stratified analysis found comparable associations in men and women [15].

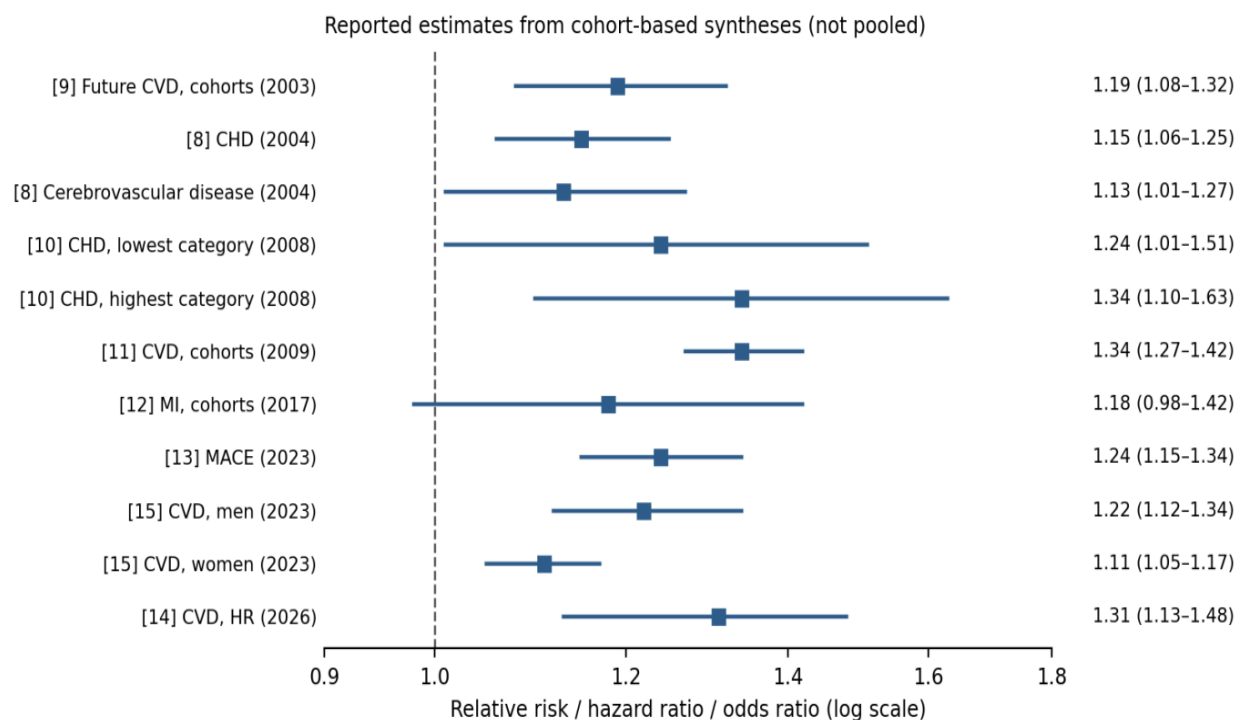


Figure 4. Published effect estimates from cohort-based syntheses. Squares are point estimates and lines are 95% confidence intervals (log scale). Measures are RR, HR or OR as originally reported. No summary estimate is shown because the primary cohorts overlap between reviews.

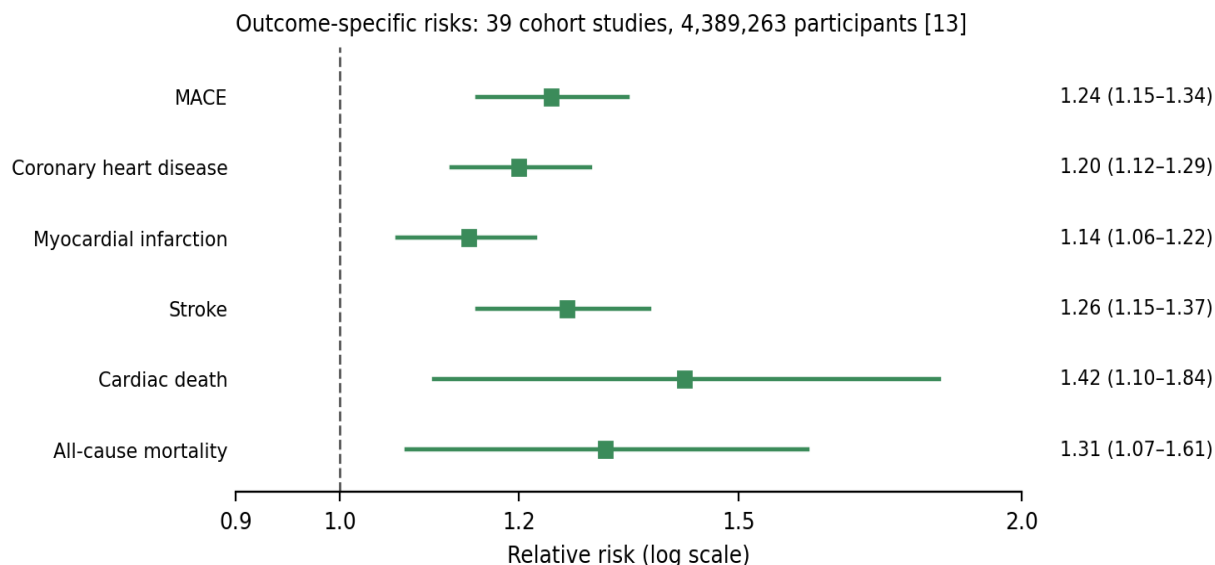


Figure 5. Outcome-specific relative risks in the meta-analysis of 39 cohort studies [13]. MACE, major adverse cardiovascular events.

Study design strongly influenced the estimates (Figure 6). Case-control and cross-sectional studies gave markedly larger values (pooled OR 2.35 for CVD [11]; OR 1.71-2.93 for MI [12]) than cohorts, where the MI estimate fell to OR 1.18 with a confidence interval including 1 [12]. One meta-analysis found a larger effect at ages ≤ 65 years (RR 1.44 vs 1.19 overall) and for stroke

alone (RR 2.85) [9]. Its meta-regression estimated that residual confounding inflated the pooled result by 12.9%, whereas using a proxy measure of periodontal disease deflated it by 29.7% [9]. Across the cohort-based syntheses in Table 1, point estimates clustered between 1.11 and 1.42, with a median of 1.23 (Figure 7).

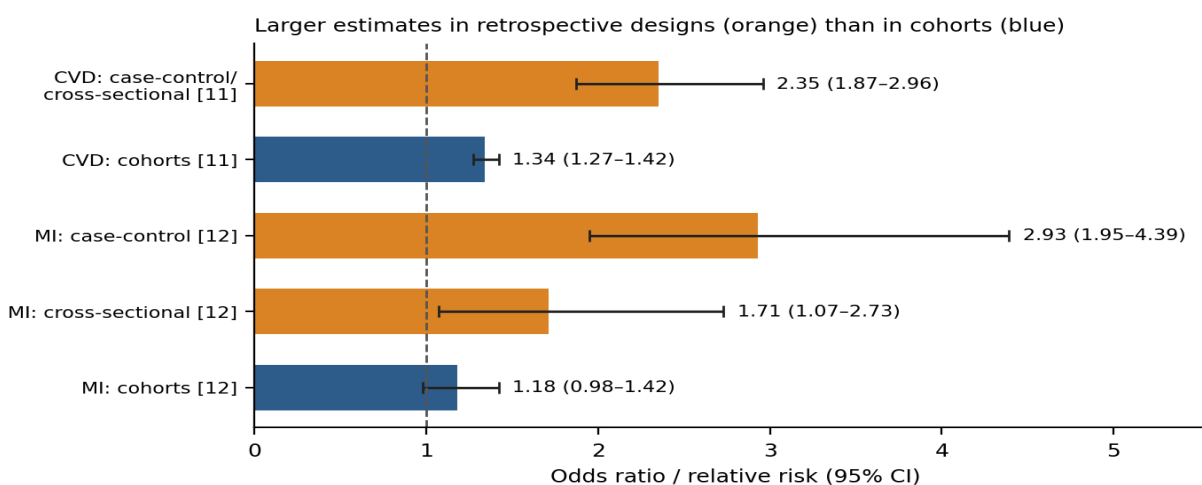


Figure 6. Effect estimates by study design for cardiovascular disease [11] and myocardial infarction [12]. Bars show point estimates (RR or OR) and whiskers show 95% confidence intervals; orange bars are case-control or cross-sectional designs and blue bars are cohorts.

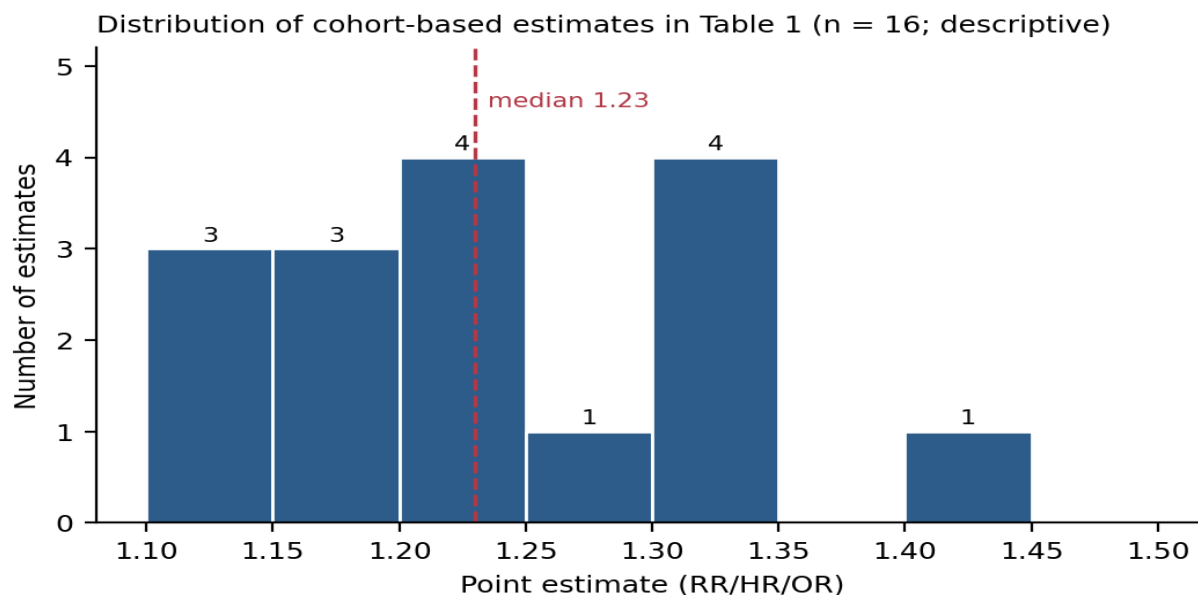


Figure 7. Histogram of cohort-based point estimates listed in Table 1 (n = 16). Case-control, cross-sectional and stroke-only or age-restricted estimates are excluded. The display is descriptive; the estimates come from overlapping cohorts and are not independent.

3.4 Other cardiovascular outcomes

Evidence extends beyond atherosclerotic events (Table 2, Figure 8). A systematic review of seven cohorts (n = 376,166) found periodontal disease associated with incident heart failure (crude RR 1.58, 95% CI 1.20–2.07) [16]. A separate meta-analysis of three publications (21,997 participants) reported OR 1.62 (1.29–2.03) for heart failure, with OR 1.99 (1.22–3.23) for

reduced and 1.36 (1.00–1.86) for preserved ejection fraction, although its MR component did not show a causal effect [17]. For atrial fibrillation, four high-quality studies consistently showed an association, with adjusted HRs from 1.03 to 1.31 [18]. The AHA also notes periodontal disease as a reported risk factor for atrial fibrillation, heart failure and chronic kidney disease [5].

Table 2. Periodontal disease and non-atherosclerotic or additional cardiovascular outcomes

Outcome	Ref	Included evidence	Main result (95% CI)
Heart failure	[16]	7 cohorts, n = 376,166 (2026)	Crude RR 1.58 (1.20–2.07)
Heart failure	[17]	3 publications, 21,997 participants, plus MR	OR 1.62 (1.29–2.03); HFrfEF OR 1.99 (1.22–3.23); HFpEF OR 1.36 (1.00–1.86); MR showed no causal effect
Atrial fibrillation	[18]	4 high-quality studies (NOS ≥ 6), systematic review (2025)	Adjusted HR range 1.03–1.31

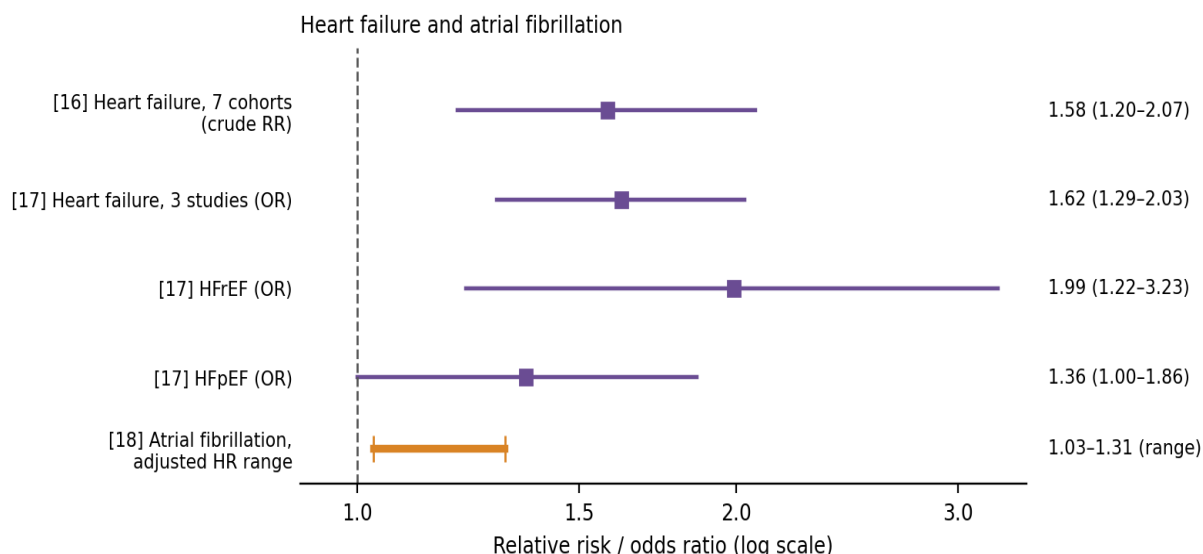


Figure 8. Published estimates for heart failure [16,17] and atrial fibrillation [18]. Heart failure: relative risk or odds ratio with 95% CI; atrial fibrillation: range of adjusted hazard ratios (no pooled CI reported). HFrEF, reduced ejection fraction; HFpEF, preserved ejection fraction.

3.5 Proposed biological mechanisms

Two broad routes are proposed. The direct route involves bacteraemia, as oral bacteria enter the bloodstream through ulcerated periodontal pockets, especially after oral hygiene procedures or clinical interventions [7]. The indirect route involves chronic systemic inflammation, evidenced by raised inflammatory cytokines and altered vascular measures [5,6]. Reviews of mechanistic links and of flow-mediated dilation connect these pathways with endothelial dysfunction, a key step in atherosclerosis [6,25]. Inflammation is a plausible therapeutic target in general: the CANTOS trial showed that lowering inflammation reduced cardiovascular events in patients with established CVD [22]. However, the rise in inflammatory markers seen in periodontal disease is modest compared with other chronic inflammatory conditions, which weakens the argument for a large systemic effect [7].

3.6 Effect of periodontal therapy

Trials of periodontal therapy have concentrated on surrogate markers (Table 3, Figure 9). A 2026

meta-analysis of 12 randomized trials found that periodontal therapy reduced systolic blood pressure by 4.64 mmHg (95% CI -5.99 to -3.30), diastolic pressure by 1.84 mmHg (-2.65 to -1.02) and C-reactive protein by 0.58 mg/L (-0.83 to -0.33), although the certainty of evidence was moderate to low [21]. A review of non-surgical periodontal therapy reported reductions in high-sensitivity CRP, IL-6, TNF- α and HbA1c (particularly in type 2 diabetes), improvements in endothelial micro particles, heterogeneous effects on flow-mediated dilation and pulse-wave velocity, and inconsistent, modest effects on lipids [23]. Meta-analyses restricted to endothelial function, including 14 randomized trials published between 2004 and 2022, and a 2026 analysis of flow-mediated dilation, likewise suggest that treatment improves endothelial function and inflammation [24,25]. Nevertheless, these trials remain inconclusive about direct effects on cardiovascular events [7], and the EFP/WHF workshop reviewed intervention studies for both cardiovascular and surrogate outcomes [6].

Table 3. Effect of periodontal therapy on cardiovascular risk markers

Ref	Evidence	Key findings
[21]	Meta-analysis of 12 RCTs (2026)	SBP -4.64 mmHg (-5.99 to -3.30); DBP -1.84 mmHg (-2.65 to -1.02); CRP -0.58 mg/L (-0.83 to -0.33); certainty moderate to low
[22,23]	Systematic reviews of RCTs on CRP and cardiovascular biomarkers	Reductions in hs-CRP, IL-6, TNF- α and HbA1c; lipid effects inconsistent and modest
[24,25]	Meta-analyses of endothelial function (14 RCTs, 2004–2022; flow-mediated dilation, 2026)	Improved endothelial function and inflammation; vascular parameters heterogeneous

Periodontal therapy vs control: 12 randomized trials [21]

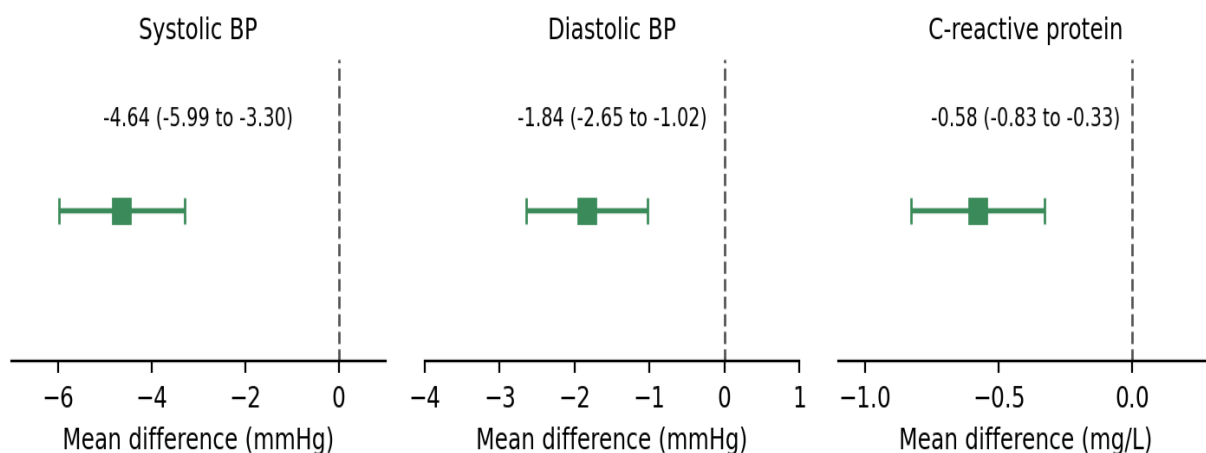


Figure 9. Effect of periodontal therapy on blood pressure and C-reactive protein in 12 randomized trials [21]. Points are mean differences with 95% confidence intervals; negative values favour treatment.

3.7 Causal inference

Confounding by shared risk factors is the central obstacle, so randomized trials and MR designs are informative because MR uses randomly assorted genetic variants as proxies for exposure [5]. Two-sample MR analyses using genetic liability for periodontal diseases found no evidence of a causal effect on coronary artery disease, heart failure, atrial fibrillation or stroke [20], and a systematic review of MR studies likewise found

no evidence of genetic causality for atherosclerotic cardiovascular disease [7]. In contrast, a study combining MR with a randomized trial of non-surgical periodontal therapy reported a causal association between periodontitis and hypertension [19]. That systematic review also noted that biases such as confounding and collider stratification may inflate positive epidemiological findings [7].

3.8 Position of professional bodies

Table 4. Consensus and causal-inference evidence

Ref	Source	Main conclusion
[5]	AHA scientific statement, 2012 (summarised in 2025 update)	Periodontal disease is associated with ASCVD; more robust evidence on clinical outcomes and treatment effects needed
[6]	EFP/WHF consensus report, 2020	Strong epidemiological evidence that periodontitis increases the risk of future atherosclerotic CVD; patients should be advised of higher risk of MI, stroke and heart failure and should manage risk factors
[5]	AHA scientific statement, 2025	New data support an association; bacteraemia and chronic inflammation are candidate mechanisms; disparities in underresourced groups; areas needing research identified
[7,20]	MR analyses and systematic review of MR studies	No evidence of genetic causality for ASCVD, coronary disease, heart failure, atrial fibrillation or stroke
[19]	MR plus randomized trial	Causal association reported between periodontitis and hypertension

The EFP/WHF Perio-Cardio consensus concluded that there is strong epidemiological evidence that periodontitis increases the risk of future atherosclerotic CVD, and recommended that patients with periodontitis be told they face higher risks of myocardial infarction and stroke and should actively manage risk factors including smoking, inactivity, excess weight, blood pressure and diet [6]. It further advised that patients with both conditions may be at higher risk of subsequent cardiovascular complications [6]. The 2025 AHA update reinforces the association while emphasising mechanisms and remaining gaps [5].

4. Discussion

4.1 Principal findings

Published evidence consistently links periodontitis with higher cardiovascular risk, but the strength of that link and its interpretation depend heavily on study design. Prospective cohorts point to a modest 13–34% relative increase across outcomes, with stroke and cardiac death often showing the largest signals [9,13,14]. Estimates from retrospective designs are

considerably larger, and MR studies, which are less vulnerable to confounding, generally do not support a causal effect [7,20].

4.2 Interpreting the association

Several explanations can reconcile these observations. The gradient from case-control (OR up to 2.93) to cohort (OR 1.18) estimates for MI [12] is consistent with recall and selection bias in retrospective designs. Residual confounding by smoking, diabetes and socioeconomic status is likely to inflate cohort estimates, as one meta-regression indicated [9]. Conversely, imprecise exposure definitions may bias results toward the null [9]. Even a modest relative risk may matter at population level, given that more than a billion people have severe periodontitis [2]. The intervention literature adds a further nuance: periodontal therapy improves blood pressure, CRP and endothelial function [21,24,25], which is biologically coherent, yet the certainty of this evidence is only moderate to low [21] and no trial has yet shown fewer cardiovascular events [7].

The MR results and the hypertension study point in different directions [19,20], showing that the

conclusion may vary by outcome, genetic instrument and population. Together, the evidence supports periodontitis as a risk marker, and perhaps a modifiable contributor to specific intermediate risk factors such as blood pressure, without establishing it as a cause of cardiovascular events.

4.3 Clinical implications

Consistent with the EFP/WHF consensus, dental teams should advise patients with periodontitis of their elevated cardiovascular risk and encourage management of shared risk factors, and the consensus offers separate recommendations for dentists, physicians and patients [6]. Periodontal care is justified on oral health grounds and may bring cardiometabolic benefits, but clinicians should not promise that it prevents heart attacks or strokes, given the absence of outcome trials [7].

4.4 Strengths and limitations of this review

Strengths include the combined use of meta-analyses, MR studies, trial syntheses, GBD data and consensus reports, giving a multi-perspective view. Limitations are inherent to the narrative design:

1. No formal risk-of-bias assessment or GRADE rating was applied to the included syntheses.
2. Overlapping primary cohorts across meta-analyses prevented valid re-pooling of their estimates.
3. Reliance on published estimates means differences in periodontal definitions, adjustment sets and follow-up could not be harmonised.
4. Source identification relied on seven structured web-based queries (about ten records each) plus earlier exploratory queries rather than direct searches of PubMed, Embase or Scopus, so

eligible syntheses may have been missed; for example, stroke-specific meta-analyses were assessed but not prioritized (Table S2).

Publication bias in the underlying literature also cannot be excluded.

5. Most MR evidence uses European-ancestry data and a limited set of genetic instruments for periodontitis, which restricts generalisability.

4.5 Future research

Priorities include adequately powered randomized trials of periodontal therapy with hard cardiovascular endpoints, harmonized exposure definitions based on the 2018 staging and grading system [4], larger genetic instruments for periodontitis, and cohorts from under-represented regions.

4.6 Conclusion

Periodontitis is consistently associated with a moderately higher risk of cardiovascular disease, stroke, cardiac death, heart failure and atrial fibrillation, and periodontal therapy improves several cardiovascular risk markers. However, confounding, design-dependent heterogeneity and largely null MR findings mean that causality is unproven. Periodontal assessment should be integrated into cardiovascular risk conversations, while outcome trials are awaited.

Declarations

Ethics approval: Not applicable; the review used published data only.

Data availability: All data are from the cited published sources.

Supplementary tables

Table S1. Search log: structured queries run on 19 September 2026

#	Query	Records	Duplicates	Excluded at screening	Assessed for eligibility	Included
1	periodontitis cardiovascular disease meta-analysis cohort studies	9	1	3	5	2

#	Query	Records	Duplicates	Excluded at screening	Assessed for eligibility	Included
2	periodontitis coronary heart disease stroke myocardial infarction meta-analysis risk	9	2	4	3	0
3	periodontitis heart failure atrial fibrillation systematic review	9	2	3	4	1
4	periodontal treatment randomized trials meta-analysis blood pressure C-reactive protein endothelial function	9	4	2	3	0
5	periodontitis cardiovascular disease Mendelian randomization causal	9	4	1	4	2
6	periodontitis cardiovascular consensus report scientific statement American Heart Association EFP	9	6	1	2	1
7	Global Burden of Disease severe periodontitis cases 2021 projections	9	2	3	4	2
	<i>Total</i>	63	21	17	25	8

Duplicates are counted where a source appeared again after its first occurrence in query order. The two journal versions of the EFP/WHF consensus

report were treated as one report. A further 16 included sources came from earlier exploratory queries and citation searching.

Table S2. Sources assessed for eligibility but not included (n = 17)

Source (as retrieved)	Year	Design / main outcome	Reason
Risk of incident cardiovascular disease in people with periodontal disease: SR and meta-analysis (Clin Exp Dent Res)	2021	32 cohorts; CVD RR 1.20 (1.14–1.26)	A
Meta-analysis of periodontitis and myocardial infarction (Front Physiol)	2016	Myocardial infarction	A
Periodontitis and number of teeth in the risk of coronary heart disease: updated meta-analysis (Med Sci Monit)	2021	11 prospective studies; CHD RR 1.18 (1.10–1.26)	A
Meta-analysis of periodontitis and myocardial infarction (ijscardiol.org)	—	Myocardial infarction; 4 studies pooled	A
Periodontal disease and atrial fibrillation/flutter: SR and meta-analysis (PMC10692853)	—	AF/atrial flutter; 4 studies	A

Source (as retrieved)	Year	Design / main outcome	Reason
Influence of anti-infective periodontal treatment on C-reactive protein: SR and meta-analysis of RCTs (PLoS One)	2013	RCTs; CRP	A
Periodontal therapy and treatment of hypertension: SR and meta-analysis (Pharmacol Res)	2021	8 RCTs; SBP -4.3 mmHg (-9.10 to 0.48)	A
Effects of non-surgical periodontal treatment on CRP in coronary artery disease: meta-analysis (Int Sci Technol J)	2017	4 RCTs; CRP	A
Periodontitis and atherosclerotic cardiovascular disease: consensus report of the Joint EFP/AAP Workshop (J Clin Periodontol)	2013	Consensus report	A
Is periodontitis a risk factor for ischemic stroke? SR and meta-analysis (PMC11994202)	—	Ischaemic stroke; OR 2.59	B
Periodontitis and risk of stroke: SR and meta-analysis of observational studies (PMC12620239)	—	22 studies; cohort RR 1.49 (1.23–1.80)	B
Periodontitis and atrial fibrillation recurrence after ablation: SR (Front Cardiovasc Med)	2026	3 studies, n = 1,016	B
Effects of oral inflammatory diseases and oral hygiene on atrial fibrillation: SR (doi 10.1155/2023/1750981)	2023	Atrial fibrillation	B
Is periodontitis a risk factor for ischaemic stroke, coronary artery disease and subclinical atherosclerosis? A Mendelian randomization study (Atherosclerosis)	2020	Two-sample MR	B
Mendelian randomization of periodontitis and ischemic stroke (Evid Based Dent)	2023	Two-sample MR	B
Global, regional and national burden of periodontal diseases 1990–2021 and predictions to 2040 (PMC12332980)	—	GBD 2021; all periodontal diseases	B
Burden of periodontal disease and edentulism, 1990–2021: GBD 2021 framework (In Vivo)	—	GBD 2021	B

SR, systematic review. Reason A: overlapping with, or superseded by, a larger or more recent included synthesis. Reason B: outcome- or population-specific analysis covered by broader included syntheses. Years are shown only where visible in the retrieved record.

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