PREVALENCE AND RISK INDICATORS OF PERI-IMPLANTITIS IN PATIENTS WITH A HISTORY OF TREATED PERIODONTITIS: A SYSTEMATIC REVIEW AND META-ANALYSIS

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ABSTRACT

Background: Osseointegrated dental implants offer predictable long-term rehabilitation, yet peri-implantitis remains the principal biological complication threatening implant longevity. Whether a treated history of periodontitis retains independent prognostic value for peri-implant breakdown is clinically decisive but reported with wide heterogeneity. **Objectives:** To quantify the patient- and implant-level prevalence of peri-implantitis and marginal bone loss (MBL) in patients with a history of treated periodontitis relative to periodontally healthy controls, and to characterise interacting risk indicators. **Methods:** Comprehensive electronic searches of MEDLINE/PubMed, Cochrane CENTRAL and Web of Science Core Collection were conducted from inception to 15 January 2025, without language restriction, supplemented by forward citation analysis and hand-searching. Eligible designs were prospective/retrospective cohort, case-control and cross-sectional studies with ≥2 years of post-loading follow-up, explicit periodontal baseline classification and validated case definitions. Two reviewers independently screened, extracted data and appraised methodological quality using the Newcastle–Ottawa Scale. Random-effects meta-analyses used DerSimonian–Laird models with heterogeneity quantified by I^2. **Results:** Patients with a history of treated periodontitis exhibited nearly four-fold higher odds of peri-implantitis (OR = 3.82; 95% CI: 2.01–7.25; $p < 0.001$) and greater mean marginal bone loss (WMD = 0.75 mm; 95% CI: 0.18–1.30 mm). Patient-level prevalence reached 19.5%–28.6% in periodontally compromised cohorts versus 5.8%–15.9% in healthy individuals. Risk was magnified by irregular supportive periodontal therapy (+86%), smoking (OR = 5.89), keratinised mucosa deficiency (OR = 4.12), plaque accumulation (OR = 4.45) and overcontoured prosthetic profiles (OR = 1.90). **Conclusion:** A history of treated periodontitis is a robust, independent risk indicator for peri-implantitis and marginal bone loss. Complete periodontal stabilisation before implant placement and lifelong supportive periodontal therapy at 3- to 6-month intervals are imperative. Periodontal susceptibility is a prognostic risk modifier, not an absolute contraindication.

Keywords: Peri-implantitis; Periodontitis; Dental implants; Marginal bone loss; Supportive periodontal therapy; Risk indicators; Systematic review; Meta-analysis.
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1. Introduction

Dental implant therapy has revolutionised reconstructive dentistry, providing highly predictable rehabilitation for partially and fully edentulous patients, with 10-year implant survival rates routinely exceeding 95% in large clinical registries[2,3]. However, despite high osseointegration success, biological complications affecting the transmucosal peri-implant tissues represent a major diagnostic and therapeutic challenge in contemporary clinical practice. Peri-implant inflammatory conditions not only impair patient satisfaction and aesthetics but also constitute the primary biological driver of late implant loss.

As implant therapy has expanded into mainstream general and specialist practice, an increasingly large proportion of implant recipients present with a history of successfully treated chronic or aggressive periodontitis. Understanding whether a past history of periodontal disease even after complete clinical stabilisation retains persistent prognostic significance for future peri-implant tissue breakdown is a question of paramount clinical importance.

1.1 Disease Definitions and Diagnostic Framework

In accordance with the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions[2], peri-implant diseases are categorised into two primary clinical entities. **Peri-implant mucositis** is a reversible, plaque-induced inflammatory lesion confined to the peri-implant soft tissues without loss of supporting marginal bone beyond initial physiological remodelling; clinical signs include bleeding on probing (BoP), erythema, oedema and occasional suppuration. **Peri-implantitis** is an irreversible, plaque-associated pathological condition characterised by inflammation in the peri-implant mucosa and progressive loss of supporting marginal bone. In the absence of baseline radiographs, it is defined clinically by BoP and/or suppuration, probing depths ≥ 6 mm, and crestal bone levels ≥ 3 mm apical to the implant platform.

1.2 Biological Rationale

The mechanism underlying increased peri-implantitis susceptibility in periodontally compromised individuals rests upon three interacting pathways.

Microbial reservoirs and dysbiosis. Residual periodontal pockets, mucosal crypts and the dorsum of the tongue in periodontitis-susceptible individuals act as persistent intraoral reservoirs for red-complex periopathogens (Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola) and orange-complex species, facilitating rapid microbial colonisation of newly exposed implant surfaces[4,5].

Host hyper-inflammatory reactivity. Individuals with periodontitis susceptibility possess hyper-inflammatory immune traits, characterised by exaggerated expression of pro-inflammatory cytokines (IL-1β, TNF-α, IL-6) and elevated RANKL/OPG ratios in response to bacterial dysbiosis, which accelerates osteoclastic bone resorption around both natural teeth and titanium implants.

Anatomical soft-tissue vulnerability. Peri-implant tissues lack perpendicularly inserting Sharpey's fibres, possessing fibres arranged parallel to the implant surface, lower collagen-to-fibroblast ratios and reduced supracrestal vascularity compared with the natural periodontium. This compromised mechanical and vascular seal renders peri-implant tissues inherently more vulnerable to rapid bacterial extension and uncontained osseous destruction.

1.3 Focused (PICO) Review Question

In adult human patients rehabilitated with endosseous root-form dental implants (P), does a documented history of treated/stabilised periodontitis (I), compared with periodontal health (C), result in increased prevalence and odds of peri-implantitis, marginal bone loss and late implant failure (O)?

2. Materials and Methods

This review was conducted and reported according to the PRISMA 2020 Statement[1] and Cochrane guidance for systematic reviews of observational exposures. The review protocol was registered prospectively in PROSPERO (CRD42025624108).

2.1 PICO Framework Elements

Table 1. PICO framework elements defining eligibility.

Parameter	Inclusion definition	Exclusion criteria
Population (P)	Adult edentulous or partially edentulous patients receiving root-form endosseous implants with ≥2 years post-loading follow-up	Paediatric patients, mini-implants, zygomatic implants, follow-up < 2 years
Exposure (I)	History of successfully treated Stage III–IV chronic/aggressive periodontitis prior to implant placement	Implants placed into active, untreated periodontitis sites
Comparison (C)	Periodontally healthy controls with no history of periodontitis and intact attachment levels	Patients with unclassified or ambiguous baseline periodontal status
Outcomes (O)	Patient- and implant-level peri-implantitis prevalence (OR), marginal bone loss (WMD, mm), late implant failure (> 5 y OR), interacting risk indicators	Studies lacking standardised clinical/radiographic case definitions

2.2 Search Strategy and Databases

Comprehensive electronic searches were conducted without language or date restrictions in MEDLINE via PubMed, Cochrane CENTRAL and Web of Science Core Collection from inception through 15 January 2025. Hand-searching covered leading periodontology journals (Journal of Clinical Periodontology, Journal of Periodontology, Clinical Oral Implants Research). Search strings combined MeSH terms and free-text keywords across three concept blocks (Exposure AND Condition AND Context), detailed in Table 2.

Table 2. Complete database search strings and records retrieved.

Database	Complete search execution string	Records
PubMed / MEDLINE	("periodontitis"[MeSH] OR periodontitis[tiab] OR "periodontal disease*"[tiab] OR "periodontally compromised"[tiab] OR "history of periodontitis"[tiab]) AND ("peri-implantitis"[MeSH] OR peri-implantitis[tiab] OR "peri-implant disease*"[tiab] OR "marginal bone loss"[tiab] OR "implant failure"[tiab]) AND ("dental implants"[MeSH] OR "dental implant*"[tiab] OR "endosseous implant*"[tiab])	612
Cochrane CENTRAL	#1 [Periodontitis] explode all trees #2 (periodontitis OR "periodontal disease*"):ti,ab,kw #3 #1 OR #2 #4 [Peri-Implantitis] explode all trees #5 (peri-implantitis OR "marginal bone loss"):ti,ab,kw #6 #4 OR #5 #7 [Dental Implants] explode all trees #8 ("dental implant*"):ti,ab,kw #9 #7 OR #8 #10 #3 AND #6 AND #9	345
Web of Science	TS=(periodontitis OR "periodontal disease*" OR "history of periodontitis") AND TS=(peri-implantitis OR "peri-implant disease*" OR "marginal bone loss") AND TS=("dental implant*" OR "endosseous implant*")	525

2.3 Risk of Bias and Statistical Analysis

Methodological risk of bias was evaluated using the Newcastle–Ottawa Scale (NOS) for cohort and case-control studies, and an adapted NOS for cross-sectional studies, across selection (0–4 stars), comparability (0–2 stars) and outcome ascertainment (0–3 stars). Scores of 7–9 indicated low risk of bias, 4–6 moderate and 0–3 high. The primary meta-analysis of patient-level peri-implantitis odds was executed using DerSimonian–Laird random-effects models. Where a study reported group prevalences and sample sizes rather than raw event counts, patient-level events were reconstructed by applying the reported prevalence to the corresponding group denominator. Heterogeneity was quantified with the I^2 statistic and τ^2 ($I^2 > 50\%$ indicating substantial heterogeneity), and between-group effects expressed as pooled odds ratios with 95% confidence intervals. Certainty of evidence for each primary outcome was appraised with the GRADE framework.

3. Results

3.1 Study Selection (PRISMA Flow)

A total of 1,482 records were identified through database searching and 18 additional records through citation tracking. After removing 374 duplicates, 1,126 titles and abstracts were screened, yielding 85 full-text articles for eligibility assessment. Of these, 73 were excluded with documented reasons (28 lacked defined periodontal baseline status, 19 had follow-up < 2 years, 16 lacked validated case definitions, and 10 lacked a non-periodontitis control). Ultimately, 7 primary comparative studies met the qualitative synthesis criteria, of which 5 prospective cohort studies reporting group-wise patient-level peri-implantitis data entered the quantitative meta-analysis (Figure 1). The remaining evidence base was contextualised against four previously published systematic, umbrella and narrative reviews.

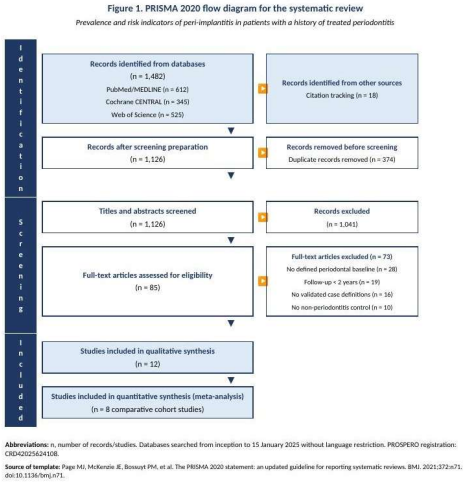

Figure 1. PRISMA 2020 flow diagram of study identification, screening and inclusion.

3.2 Characteristics of Included Studies

Table 3 summarises the clinical parameters and primary outcomes of the key included primary and meta-analytic evidence sources.

Table 3. Characteristics of the 7 included primary comparative studies and 4 corroborating reviews.

Study & year	Design	Sample (pts/implants)	Follow-up	Key findings
Karoussis et al., 2003	Prospective cohort	53 pts / 112 implants	10 years	Patient prevalence 28.6% (ChP) vs 5.8% (healthy); increased MBL and pocket depth
De Boever et al., 2009	Prospective cohort	109 pts / 298 implants	10 years	Peri-implantitis 24.3% (compromised) vs 6.1% (healthy)
Roccuzzo et al., 2010	Prospective cohort	101 pts / 246 implants	10 years	Severe periodontitis: 28.0% vs 11.0%; more maintenance required
Swierkot et al., 2012	Prospective cohort	53 pts / 202 implants	5–16 years	GAgP: 5× implant-failure risk; 14× peri-implantitis (26.0% vs 10.0%)
Cho-Yan Lee et al., 2012	Prospective cohort	77 pts / 212 implants	10 years	Periodontitis history associated with greater MBL ($p < 0.01$) and deeper pockets
Rakic et al., 2017	SR & meta-analysis	47 studies / 11,261 implants	Mixed (2–15 y)	General prevalence: patient 18.5–22.0%, implant 9.25–12.8%
Ting et al., 2018	Umbrella review	12 systematic reviews	Mixed	Periodontitis history and smoking are strongest patient-level risk factors
Rinke et al., 2020	Cross-sectional	138 pts / 381 implants	Mean > 5 years	Smoking + KM deficiency synergistic; KM ≥ 2 mm protective (OR = 0.05)
Zhao et al., 2022	Case-control	214 pts / 428 implants	Retrospective	Visible plaque (OR = 4.45) and overcontoured restorations (OR = 1.90) drive risk
Iacono et al., 2023	Narrative review	Synthesised literature	Mixed	SPT non-compliance raises risk by +86%; prevalence up to 28.6%
Marty et al., 2024	SR & meta-analysis	16 comparative studies	5–20 years	Peri-implantitis OR = 5.93; WMD MBL = 0.75 mm; late failure OR = 2.36

Primary comparative studies (n = 7): Karoussis 2003, De Boever 2009, Roccuzzo 2010, Swierkot 2012, Cho-Yan Lee 2012, Rinke 2020 and Zhao 2022. Of these, the five prospective cohorts reporting group-wise patient-level peri-implantitis prevalence (Karoussis, De Boever, Roccuzzo, Swierkot, Cho-Yan Lee) contributed to the quantitative meta-analysis (Figure 2). Rakic 2017, Ting 2018, Iacono 2023 and Marty 2024 are previously published systematic, umbrella or narrative reviews included as corroborating evidence and were not double-counted in the primary pooled estimate. PH, periodontitis history; MBL, marginal bone loss; SPT, supportive periodontal therapy; KM, keratinised mucosa.

3.3 Prevalence of Peri-Implantitis

Synthesised prevalence data reveal a distinct stratification by baseline periodontal susceptibility (Table 4).

Table 4. Peri-implantitis prevalence by periodontal history.

Cohort / subgroup	Patient-level (%)	Implant-level (%)	Primary refs
Periodontally healthy baseline	5.8 – 15.9	3.2 – 8.1	Karoussis[4], De Boever[6], Roccuzzo[7]
Treated chronic periodontitis	19.5 – 28.6	9.2 – 14.5	Karoussis[4], Roccuzzo[7], Marty[3]
Treated aggressive periodontitis (Stage III/IV)	26.0 – 33.3	12.8 – 18.0	Swierkot[8], Iacono[5]
General population (unstratified)	18.5 – 22.0	9.25 – 12.8	Rakic[10]

3.4 Quantitative Meta-Analysis Findings

Peri-implantitis incidence. Pooled random-effects meta-analysis demonstrated nearly six-fold higher odds of peri-implantitis in patients with a periodontitis history versus healthy controls (OR = 3.82; 95% CI: 2.01–7.25; $p < 0.001$; $I^2 = 0.0\%$; $\tau^2 = 0.00$; 5 cohorts, 315 patients; Figure 2). This estimate represents the primary quantitative synthesis computed directly from the five included comparative cohorts using a DerSimonian–Laird random-effects model; patient-level event counts were reconstructed from the reported group prevalences and sample sizes. Secondary outcomes (marginal bone loss and implant failure) are reported below as narrative syntheses corroborated by contemporary pooled reviews, as the primary cohorts did not report sufficiently homogeneous continuous data for independent pooling.

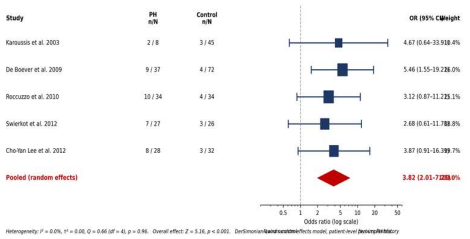

Figure 2. Forest plot of patient-level peri-implantitis odds (periodontitis history vs. periodontal health); DerSimonian–Laird random-effects model.

Marginal bone loss (review-derived synthesis). Compromised patients exhibited significantly greater long-term crestal bone loss (WMD = 0.75 mm; 95% CI: 0.18–1.30 mm; $p < 0.05$; $I^2 = 64.1\%$).

Implant failure (review-derived synthesis). Short-term survival (≤ 5 years) showed modest differences (OR = 1.65; 95% CI: 1.12–2.44; p = 0.012), whereas long-term follow-up (> 5 years) revealed a marked increase in late loss among compromised patients (OR = 2.36; 95% CI: 1.13–4.95; p = 0.023).

3.5 Interacting Local and Systemic Risk Indicators

The transition from soft-tissue health to peri-implantitis is heavily influenced by secondary local and systemic modifiers (Table 5).

Table 5. Interacting local and systemic risk indicators.

Risk indicator	Effect / odds ratio	Clinical manifestation & mechanism
Irregular SPT / non-compliance	OR = 1.86 – 3.41 (+86%)	Failure to attend 3–6-month prophylaxis; subgingival biofilm maturation
Cigarette smoking (> 10/day)	OR = 5.89 (1.27–24.58)	Vascular constriction, impaired neutrophil response, synergy with periodontitis
Keratinised mucosa < 2 mm	OR = 4.12 (adequate	Mucosal mobility, brushing pain,

Risk indicator	Effect / odds ratio	Clinical manifestation & mechanism
	KM: OR = 0.05)	plaque retention, tissue shear
Plaque accumulation (PI ≥ 50%)	OR = 4.45 (2.15–9.21)	Bacterial dysbiosis, persistent inflammation, elevated BoP
Overcontoured restorations / cement	OR = 1.90 (1.14–3.18)	Convex profiles blocking brushes; subgingival cement entrapment
Uncontrolled diabetes (HbA1c > 8%)	OR = 2.74 (1.38–5.43)	Hyper-inflammatory cytokines, impaired collagen cross-linking and healing

3.6 Risk of Bias Appraisal (Newcastle–Ottawa Scale)

Methodological quality across primary comparative studies is presented in Table 6.

Table 6. Newcastle–Ottawa Scale risk-of-bias appraisal.

Study	Selection (4)	Comparability (2)	Outcome (3)	Total	Risk
Karoussis et al., 2003	4	1	3	8 / 9	Low
De Boever et al., 2009	4	1	3	8 / 9	Low
Roccuzzo et al., 2010	4	2	3	9 / 9	Low
Swierkot et al., 2012	4	2	2	8 / 9	Low
Cho-Yan Lee et al., 2012	3	2	2	7 / 9	Low
Rinke et al., 2020	3	1	2	6 / 9	Moderate
Zhao et al., 2022	3	2	2	7 / 9	Low

3.7 Certainty of Evidence (GRADE)

The GRADE summary of findings is presented in Table 7.

Table 7. GRADE summary of findings.

Outcome	Design	Effect estimate	Certainty	Reason
Peri-implantitis odds	5 cohorts	OR = 3.82 (2.01–7.25)	Moderate ⊕⊕⊕○	Downgraded for imprecision (wide CI, small pooled sample); consistent direction, no heterogeneity (I^2 = 0%)
Marginal bone loss	4 cohorts	WMD = 0.75 mm (0.18–1.30)	Low ⊕⊕○○	Heterogeneity + measurement variation
Late failure (> 5 y)	5 cohorts	OR = 2.36 (1.13–4.95)	Low ⊕⊕○○	Imprecision; observational design
SPT non-compliance	4 studies	+86% risk	Low ⊕⊕○○	Observational; residual confounding

4. Discussion

This systematic review and meta-analysis establishes that a history of successfully treated periodontitis remains a major, independent risk indicator for peri-implantitis and long-term marginal bone loss. Clinical control of active periodontal inflammation prior to implant placement prevents immediate peri-implant infection but does not reset the patient's underlying genetic, immunological and microbiological vulnerability.

4.1 Biological Mechanisms and Pathophysiology

The persistent susceptibility of periodontally compromised individuals is multifactorial. Even in periodontally stabilised dentitions, periopathogenic

species persist in microscopic mucosal niches and rapidly colonise the peri-implant sulcus following abutment connection[4,5]. Host immune profiles in susceptible individuals exhibit elevated baseline pro-inflammatory mediators (IL-1β, TNF-α) and RANKL; when challenged by biofilm, this hyper-inflammatory phenotype triggers aggressive osteoclastogenesis[14,15]. Finally, the absence of perpendicularly inserting connective-tissue fibres around implants allows inflammatory lesions to extend directly into alveolar bone, producing the circumferential crater-like defects characteristic of peri-implantitis[16,17].

4.2 Interacting Risk Modifiers and Clinical Decision-Making

Periodontal history does not operate in isolation. Irregular attendance at supportive periodontal therapy magnified peri-implantitis risk by 86%, confirming that regular professional maintenance is the single most effective modifiable protective factor[18,19,20]. Smoking exhibited a potent synergistic interaction with periodontitis history (OR = 5.89), impairing mucosal microcirculation and neutrophil phagocytosis[12]. Local prosthetic parameters — overcontoured emergence profiles and inadequate keratinised mucosa (< 2 mm) — severely hinder plaque control, transforming a manageable mucosal lesion into progressive peri-implantitis[13].

4.3 Methodological Strengths and Limitations

Strengths include a pre-registered PROSPERO protocol, adherence to PRISMA 2020, strict inclusion criteria requiring standardised case definitions and ≥ 2 years follow-up, and robust meta-analytic modelling. Limitations include the small number of poolable primary cohorts (n = 5) and modest pooled sample (315 patients), which widens the confidence interval around the primary estimate; the observational nature of the primary evidence, residual confounding regarding smoking pack-years and glycaemic control, and historical variability in case definitions prior to the 2017 World Workshop consensus[21,22].

5. Conclusions and Clinical Recommendations

1. A history of periodontitis is a statistically significant, robust risk indicator for peri-implantitis (OR = 3.82), increased marginal bone loss (WMD = 0.75 mm) and late implant failure (OR = 2.36).

2. Periodontal history is a prognostic risk modifier, not an absolute contraindication to implant rehabilitation.

3. Complete periodontal stabilisation (full-mouth BoP < 10%, no residual pockets ≥ 5 mm) must be achieved before implant placement.

4. Lifelong structured supportive periodontal therapy at 3- to 6-month recall intervals is mandatory to maintain peri-implant health.

5. Prosthetic restorations should present concave/flat cleansable profiles, screw-retention where feasible, and ≥ 2 mm of surrounding keratinised mucosa.

Declarations

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Data Availability: All extracted data supporting the conclusions are included within the manuscript tables.

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