e-ISSN: 2637-0611

Available online at
https://journal.uitm.edu.my/ojs/index.php/COS

Compendium of Oral Science 13 (1) 2026, 58 - 73

Compendium of Oral Science

Determinants of Peri-implant Disease: From Biofilm Aetiology to Modifiable Risk Factors and Anatomical Predisposition

Mohamad Shamim Abu Ani[1], Mohd Faizal Hafez Hidayat[1*], Hafizul Izwan Mohd Zahari[1]

[1] *Centre of Studies for Periodontology, Faculty of Dentistry, Universiti Teknologi MARA Sungai Buloh Campus, Jalan Hospital, 47000 Sungai Buloh, Selangor, Malaysia*

ARTICLE INFO

Article history:
Received 04 August 2025
Revised 19 February 2026
Accepted 14 April 2026
Online First
Published 30 April 2026

Keywords:
peri-implantitis
peri-implant mucositis
biofilm
dental implants
risk factors
microbiota

DOI:
10.24191/cos.v13i1.7262

ABSTRACT

Objectives: This review critically evaluates the multifactorial determinants of peri-implant diseases, hypothesising that microbial biofilms, systemic and behavioural factors, and anatomical distinctions collectively influence disease onset and progression. **Materials and Methods**: A comprehensive analysis of current literature was conducted, focusing on microbiological studies, epidemiological reports, and clinical trials related to peri-implant mucositis and peri-implantitis. Special emphasis was placed on evaluating evidence related to bacterial plaque formation, systemic conditions (diabetes mellitus), behavioural habits (smoking, oral hygiene), anatomical differences between implants and natural teeth, and prosthetic complications. **Results**: Bacterial plaque is established as the primary etiological factor in peri-implant mucositis and significantly contributes to peri-implantitis. Disease severity and progression are modulated by systemic conditions such as diabetes mellitus, behavioural factors including smoking and inadequate oral hygiene, and prosthetic factors like residual cement. Individuals with previous periodontitis exhibit heightened susceptibility due to persistent periodontal pathogen colonisation. Anatomical differences such as the lack of periodontal ligament, reduced vascularity, and weaker soft tissue integration also increase vulnerability to peri-implant disease. Microbial analysis highlights a diverse microbiota associated with peri-implant diseases, including opportunistic pathogens such as Staphylococcus epidermidis.

[1*] Corresponding author. *E-mail address*: faizalhafez@uitm.edu.my

Conclusions: Peri-implant diseases are multifactorial, requiring tailored preventive strategies focused on meticulous plaque control, systemic and behavioural risk mitigation, and routine implant maintenance. Future research should prioritise improved diagnostic biomarkers, detailed biofilm characterisation, and innovative therapies targeting the unique biological challenges of peri-implant tissues.

INTRODUCTION

Dental implants have become widely recognised as the gold standard solution for tooth replacement (Schnitman & Han, 2015; Wang et al., 2015). However, their sustained success is frequently threatened by peri-implant diseases, biological complications that can severely compromise both implant functionality and longevity. Among these diseases, peri-implantitis poses particularly challenging diagnostic and therapeutic dilemmas due to its intricate pathogenesis involving numerous contributing factors (Heitz-Mayfield et al., 2018; Berglundh et al., 2018; Schwartz et al., 2018; Herrera et al., 2023).

Although bacterial plaque is an established trigger for mucosal inflammation, the precise reasons why some patients experience aggressive peri-implantitis while others remain unaffected remain inadequately understood. This inconsistency underscores a more extensive interplay among microbial factors, individual host susceptibility, and environmental influences, many aspects of which require further elucidation (Listgarten, 1965; Mombelli et al., 2002; Mombelli & Décaillet, 2011; Berglundh et al., 2024). Furthermore, distinct anatomical variations between natural teeth and implants can significantly influence tissue responses to microbial challenges (Gruber & Bosshardt, 2015; Coli et al., 2017). As the global prevalence of dental implant placement continues to rise, clinicians face increased complexities in managing peri-implant diseases among patients presenting with varied systemic conditions, oral hygiene compliance, and previous clinical backgrounds. Despite expanding research efforts, substantial discrepancies exist regarding crucial risk factors and the most effective strategies for their mitigation (Derks & Tomasi, 2015; Lee et al., 2017).

This review comprehensively examines the multifactorial contributors to peri-implant diseases, including microbial biofilms, systemic modifiers of disease risk, prosthetic designs, and inherent anatomical susceptibilities. By integrating current evidence, this article aims to clarify underlying pathogenic mechanisms and enhance the development of tailored preventive and therapeutic strategies.

Dental biofilm and peri-implant microbiota

The concept of "aetiology" involves a direct causal relationship between exposure to a specific factor and the consequent clinical manifestation, necessitating that the causative agent be consistently present and temporally precede the resulting condition. On the other hand, a "risk factor" influences the probability of an event occurring but is not inherently required for its initiation. Criteria for establishing causation, extensively described by Hill (1965) and Rothman & Greenland (2005), demand confirmation through prospective and interventional research methodologies.

Bacteria in dental plaque biofilm are widely acknowledged as the principal etiological agent linked to peri-implant diseases in periodontal pathology. The 2017 World Workshop on Periodontal and Peri-implant Diseases and Conditions underscored robust evidence identifying bacterial plaque as the main causative factor in peri-implant mucositis (Berglundh et al., 2018). Human experimental models analogous to the classical gingivitis studies by Löe et al. (1965) have consistently confirmed this causative relationship. For instance, deliberate plaque accumulation around dental implants over 21 days invariably

https://doi.org/10.24191/cos.v13i1.7262

resulted in peri-implant mucositis, manifesting as gingival inflammation, swelling, and bleeding upon probing (Heitz-Mayfield & Salvi, 2018; Meyer et al., 2016; Zitzmann et al., 2001; Pontoriero et al., 1994; Löe et al., 1965). Significantly, the reintroduction of meticulous plaque control reliably resolved these inflammatory symptoms (Meyer et al., 2016; Salvi et al., 2011).

Peri-implant mucositis frequently precedes peri-implantitis, thus proposing a potential causal role for bacterial plaque in peri-implantitis progression (Jepsen et al., 2015). Ethical restrictions preclude direct experimental induction of peri-implantitis in humans; however, animal studies employing ligatures to disrupt supracrestal soft tissues and encourage plaque accumulation have reliably replicated pathological features, such as biofilm proliferation, soft tissue inflammation, and bone resorption (Carcuac et al., 2019; Zitzmann et al., 2004; Albouy et al., 2008).

Epidemiological investigations further affirm the critical role of bacterial plaque in peri-implantitis aetiology. Retrospective analyses, such as those by Schwarz et al. (2018), have observed increased peri-implantitis prevalence among individuals with inadequate plaque control and inconsistent oral hygiene routines. Moreover, longitudinal studies demonstrate that consistent plaque management effectively halts disease progression, reduces inflammatory reactions, and preserves peri-implant bone structure (Carcuac et al., 2017; Roccuzzo et al., 2017; Schwarz et al., 2016; Berglundh et al., 2018). Collectively, these data emphasise the necessity of rigorous plaque control practices for preventing and managing peri-implant conditions.

Early microbiological investigations into peri-implantitis emphasised the prominence of spirochetes, absent in healthy implant sites yet abundant in advanced peri-implant lesions, mirroring electron microscopy observations in necrotising ulcerative gingivitis (Listgarten, 1965; Berglundh et al., 2024). Initial bacterial culture-based studies identified predominantly Gram-negative anaerobes, specifically Fusobacterium species and Prevotella intermedia, in implants with deeper probing depths and related bone loss. In contrast, healthy implant sites typically harboured fewer bacteria, primarily Gram-positive cocci, suggesting microbial parallels between peri-implantitis and chronic periodontitis (Mombelli et al., 1987; Berglundh et al., 2024).

Advances in microbiological analysis methods, including checkerboard DNA–DNA hybridisation, quantitative polymerase chain reaction (qPCR), fluorescence in situ hybridisation (FISH), and 16S rRNA gene sequencing, have significantly deepened our understanding of peri-implant microbial communities. Pathogens commonly linked to periodontitis, notably Fusobacterium species and *P. intermedia*, frequently appear in peri-implantitis lesions, whereas *Aggregatibacter actinomycetemcomitans* remains relatively infrequent (Mombelli et al., 2002; Mombelli & Décaillet, 2011; Berglundh et al., 2024).

The microbial profile of peri-implantitis is complex and predominantly comprises Gram-negative anaerobic organisms. A recent meta-analysis identified key peri-implant pathogens, including *Fusobacterium nucleatum*, *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia, Treponema denticola*, and notably, the Gram-positive organism *Staphylococcus epidermidis*. *S. epidermidis* infections notably mirror dental implant-related infections rather than periodontitis lesions on natural teeth (Mombelli & Décaillet, 2011; Carvalho et al., 2023; Berglundh et al., 2024).

Additionally, microbial diversity changes significantly with increasing pocket depth, forming niches favourable for pathogenic growth, particularly at depths ≥5 mm, thereby elevating disease progression risks. Thus, consistent monitoring of probing depths remains crucial for assessing peri-implant health (Mombelli & Décaillet, 2011; Kröger et al., 2018; Berglundh et al., 2024).

Traditional microbiological techniques often inadequately represent the complexity of in vivo microbial biofilms, primarily due to analyses involving disrupted biofilms, limiting comprehensive

https://doi.org/10.24191/cos.v13i1.7262

understanding of intact biofilm dynamics on implant surfaces. Consequently, advanced research targeting native-state peri-implant biofilms is imperative (Mombelli et al., 2018; Berglundh et al., 2024).

Biofilm-associated infections pose substantial challenges across dentistry and broader medical disciplines, including orthopaedics, complicating treatment outcomes and increasing antibiotic resistance risks. Effective management of peri-implantitis thus requires improved diagnostic approaches for biofilm detection, facilitating early interventions, enhancing therapeutic efficacy, and ultimately reducing clinical complications and healthcare expenditures (Ronin et al., 2021; Berglundh et al., 2024).

History of periodontitis

Extensive research consistently demonstrates that patients with a history of periodontitis exhibit increased vulnerability to peri-implant infections (Hardt et al., 2002; Karoussis et al., 2003, 2004; Heitz-Mayfield, 2008; Ong et al., 2008; Roccuzzo et al., 2010; Schwarz et al., 2018). This heightened susceptibility presumably results from shared etiological components between periodontitis and peri-implant diseases. Individuals predisposed to periodontitis remain especially susceptible to biofilm-associated peri-implant infections if periodontal pathogens successfully colonise the peri-implant environment. Supporting this hypothesis, investigations have shown that periodontal pathogens persist even after tooth removal and subsequent dental implant placement in patients previously suffering from advanced periodontal diseases (Quirynen & Van Assche, 2011).

A landmark clinical investigation assessed outcomes in ten patients diagnosed with advanced periodontitis who underwent comprehensive tooth extraction and subsequent implant placement after a six-month healing period. Implant abutments were placed three to six months post-implantation. Biofilm samples were systematically collected at baseline and throughout the first year following abutment connection from the dorsum of the tongue, saliva, and the peri-implant sulcus and submucosal sites. Although substantial reductions in total aerobic and anaerobic colony-forming units (CFU/mL), including pathogens such as *Porphyromonas gingivalis* and *Tannerella forsythia*, were noted in saliva and tongue samples, rapid recolonisation by these pathogens was observed around the peri-implant sulcus. However, *Aggregatibacter actinomycetemcomitans* levels remained stable. These results emphasise that despite significantly reducing bacterial load following extraction of periodontally compromised teeth, complete elimination of pathogens is rarely achieved, thus allowing persistent colonisation around implant sites (Quirynen & Van Assche, 2011).

Once periodontal pathogens colonise the peri-implant biofilm, the risk of peri-implant diseases substantially increases, particularly in susceptible patients. Clinical studies have shown that patients retaining residual periodontal pockets measuring 6 mm or deeper around natural teeth experience notably higher peri-implantitis prevalence, indicated by marginal bone loss and probing depths ≥5 mm with concurrent bleeding on probing (BOP), compared to periodontally healthy individuals or those without deep periodontal pockets (Cho-Yan Lee et al., 2012). Additionally, a longitudinal cohort study conducted over approximately eight years found that patients developing peri-implantitis were significantly more likely to maintain residual periodontal pockets ≥5 mm after periodontal therapy than patients who did not develop peri-implantitis (Pjetursson et al., 2012). These findings underscore the importance of clearly communicating increased peri-implant disease risks to patients previously treated for periodontitis, emphasising diligent oral hygiene and regular periodontal and peri-implant maintenance visits.

Strong evidence consistently indicates the adverse influence of previous periodontitis on implant therapy success, significantly elevating peri-implantitis risk. Two significant Scandinavian cross-sectional studies highlighted this correlation. In research involving 109 participants observed over an average period of 8.4 years, individuals predisposed to periodontitis showed significantly higher peri-implantitis risks, with an odds ratio of approximately six (Koldsland et al., 2010b; Koldsland et al., 2010a). Derks et al. (2016),

https://doi.org/10.24191/cos.v13i1.7262

in a comprehensive study of 596 patients evaluated over a comparable duration, similarly reported a robust association between prior periodontitis and subsequent peri-implantitis development (Derks et al., 2016; Derks et al., 2015).

Further longitudinal studies have reinforced the negative impact of previous periodontitis on implant survival and peri-implantitis incidence. A prospective study spanning 10 years with 53 patients receiving 112 ITI implants reported significantly reduced implant survival rates of 90.5% among periodontitis patients compared to 96.5% in those without a history of periodontitis (Karoussis et al., 2003). Additionally, peri-implantitis was significantly more prevalent in individuals with a prior periodontitis diagnosis (28.6%) than those without (5.8%). A complementary meta-analysis by Sgolastra et al. confirmed these findings, demonstrating that patients previously diagnosed with periodontitis exhibited elevated plaque index scores, increased implant loss, and significantly greater marginal bone resorption compared to periodontally healthy controls (Sgolastra et al., 2013).

Compliance to oral hygiene and supportive peri-implant care (SPIC)

The long-term success and durability of dental implants rely extensively on meticulous oral hygiene practices. Ensuring optimal hygiene of both implants and associated prosthetic components necessitates diligent daily oral care coupled with consistent engagement in structured supportive peri-implant care (SPIC) programs. Recent research underscores the strong correlation between the accessibility of implant surfaces for cleaning and the occurrence of peri-implant diseases. Serino and Ström (2008) conducted a comprehensive study involving 23 patients with a total of 109 implants diagnosed with peri-implantitis, highlighting substantial differences attributable to the ease of maintaining implant hygiene. Specifically, implants that posed challenges to effective oral hygiene exhibited a peri-implantitis incidence of 48%, whereas only 4% of readily accessible implants showed indications of peri-implant inflammation. These marked differences accentuate the importance of strategic implant and prosthetic design in enabling efficient oral hygiene practices, significantly mitigating peri-implant disease risk (Serino & Ström, 2008).

Further highlighting the significance of adherence to structured SPIC, Pjetursson et al. (2012) assessed peri-implantitis incidence by comparing outcomes among patients enrolled in structured, university-affiliated SPIC programs against those managed by private practitioners. Although no statistically significant variation in peri-implantitis prevalence was detected between these groups during the observation period, participation rates in private-practice-based SPIC were notably lower (only 12 out of 70 patients), with inadequate documentation of adherence levels. These observations suggest that patients under private practice care potentially had reduced compliance with regular maintenance protocols, influencing their peri-implant health outcomes (Pjetursson et al., 2012).

Emphasising the pivotal role of regular SPIC adherence in peri-implant health, Roccuzzo et al. (2010) presented robust longitudinal evidence derived from a decade-long prospective cohort study. Their findings revealed significantly poorer clinical outcomes among patients who demonstrated inconsistent attendance at SPIC appointments compared to those who consistently adhered to scheduled visits. Irregular attendees exhibited higher plaque accumulation, increased bleeding on probing (BOP), greater probing depths (PD), and elevated tooth loss rates throughout the ten-year follow-up. Furthermore, the maximum probing depths recorded during routine assessments and final examinations were considerably deeper among irregular SPT attendees (Roccuzzo et al., 2010). These results affirm that consistent participation in structured SPT is critical for sustaining peri-implant health and preventing clinical deterioration.

From a clinical perspective, these findings highlight the paramount importance of diligent oral hygiene practices and strict adherence to individualised maintenance regimens, especially in patients with prior periodontitis undergoing implant therapy. Clinicians should rigorously evaluate patients' periodontal

https://doi.org/10.24191/cos.v13i1.7262

histories during implant planning phases and underscore the importance of continuous compliance with tailored maintenance schedules. Adopting this comprehensive strategy markedly enhances long-term implant outcomes, minimises peri-implant complications, and emphasises patient education and motivational interventions as fundamental components of comprehensive implant care management (Valente & Andreana, 2016).

Diabetes

Diabetes mellitus (DM) represents a multifaceted metabolic disorder primarily characterised by sustained hyperglycemia, stemming from defects in insulin secretion, insulin action, or both processes combined (Petersmann et al., 2019). Diabetes is classified into two principal types: Type 1 diabetes, an autoimmune disorder resulting in the destruction of pancreatic β-cells and subsequent absolute insulin deficiency; and Type 2 diabetes, defined by progressive β-cell impairment and enhanced insulin resistance (American Diabetes Association, 2021). Extensive research underscores a reciprocal association between diabetes mellitus and periodontal diseases, attributable to shared underlying chronic inflammatory pathways. Consequently, periodontal disease is frequently identified as a significant complication of diabetes mellitus, often described as the sixth diabetes-associated complication (Stohr et al., 2021; Cowie et al., 2018; Taylor et al., 2001; Loe, 1993).

Individuals diagnosed with diabetes frequently experience significant tooth loss due to periodontal destruction and other dental pathologies, prompting consideration of dental implants to rehabilitate edentulous areas. However, recent literature stresses meticulous evaluation and customised treatment planning for diabetic patients receiving dental implants, mainly due to their heightened susceptibility to peri-implant complications and compromised clinical outcomes (Alzahrani & Abed, 2016; Enteghad et al., 2024).

Systemic conditions, particularly diabetes mellitus, consistently correlate with diminished implant therapy outcomes. These adverse effects primarily stem from compromised tissue regeneration, impaired microvascular integrity, and altered inflammatory-immune responses typically observed in diabetic individuals. Although diabetes has traditionally been regarded as a relative contraindication for dental implant placement, emerging evidence indicates that diabetic patients with effectively managed glycemic levels can attain implant success comparable to non-diabetic populations. Conversely, poorly controlled diabetes significantly elevates the risk of implant complications and diminishes the overall success rate of implant therapy (Verhulst et al., 2019; Almehmadi, 2019; Chrcanovic et al., 2014; Turkyilmaz, 2010; Oates et al., 2014; Dowell et al., 2014).

Several studies have explored the relationship between diabetes mellitus and peri-implant diseases. For instance, Li et al. (2023) performed a comprehensive systematic review and meta-analysis involving observational data from over 24,000 individuals across twenty-one separate studies. Their findings revealed that diabetes mellitus did not significantly heighten the risk of peri-implant mucositis ($p < 0.05$); nonetheless, diabetic patients demonstrated a substantially increased risk of developing peri-implantitis (OR: 1.754, $p = 0.016$). Complementary perspectives provided by Baniulyte and Ali (2023) supported these conclusions, reiterating the significant association between diabetes mellitus and elevated peri-implantitis risk (Baniulyte & Ali, 2023).

Collectively, contemporary research substantiates a bidirectional and potentially causative link between diabetes mellitus and periodontal as well as peri-implant diseases. Effective management and therapeutic control of one condition could positively influence the prevention and treatment outcomes of the other. Furthermore, the progression and severity of periodontal and peri-implant tissue destruction appear intricately associated with glycemic control quality. While diabetic individuals maintaining optimal glycemic regulation can achieve comparable implant outcomes to healthy controls, their enhanced susceptibility to peri-implantitis warrants persistent attention. Therefore, strict glycemic control combined

https://doi.org/10.24191/cos.v13i1.7262

with rigorous oral hygiene protocols is essential for diabetic patients undergoing implant therapy to secure favourable clinical results (Enteghad et al., 2024).

Smoking

Tobacco smoking has been extensively associated with periodontal diseases through numerous pathophysiological mechanisms. These mechanisms include the alteration of host inflammatory and immune responses to periodontal pathogens, modifications of subgingival microbial communities, and impairment in tissue healing processes, collectively disrupting periodontal homeostasis. Evidence indicates that smoking affects periodontal tissues through systemic and localised pathways, influencing clinical outcomes and disease progression significantly (Apatzidou, 2022; Darby, 2022).

Historical and contemporary studies have analysed the impact of tobacco smoking on periodontal clinical parameters, subgingival microbiota composition, and inflammatory responses. Traditional microbiological detection methods targeting specific pathogens have revealed changes in smokers' periodontal pockets. More recently, advanced methodologies such as deep sequencing and bioinformatic analyses have enabled the comprehensive characterisation of subgingival microbial ecosystems. Nevertheless, the interpretation of these findings has been complicated by variability in research methodologies and study designs, leading to inconsistent outcomes across different studies (Apatzidou, 2022; Darby, 2022).

While smoking is frequently considered harmful to implant survival, evidence regarding its direct impact on peri-implant disease has shown variability. Koldsland et al. (2010) reported no significant correlation between tobacco smoking and peri-implant conditions (Koldsland et al., 2010). Similarly, Roos-Jansåker et al. (2006) found no significant relationship between smoking and implant loss. However, this study did observe higher frequencies of peri-implant mucositis, peri-implantitis, and marginal bone loss among smokers compared to non-smokers (Roos-Jansåker et al., 2006). These conflicting outcomes were also mirrored in a meta-analysis conducted by Sgolastra et al. (2013), where implant-level analysis revealed a significant increase in peri-implantitis risk among smokers. In contrast, patient-level analysis indicated no significant differences between smokers and non-smokers. Acknowledging the considerable heterogeneity observed in these analyses, underscoring the complexity of establishing a definitive relationship between smoking and peri-implant diseases (Sgolastra et al., 2013).

Nevertheless, recent literature provides more substantial evidence supporting the association between smoking and peri-implantitis. Martinez-Amargant et al. (2023) conducted a study involving 117 patients with 450 implants. They demonstrated a statistically significant correlation between the intensity of smoking and an increased risk of developing peri-implantitis ($p < 0.002$). Moreover, this study highlighted that individuals who had ceased smoking for over 21 years exhibited peri-implant disease risks similar to those who had never smoked, suggesting the beneficial impact of prolonged smoking cessation (Martinez-Amargant et al., 2023). Further corroborating this association, a systematic review and meta-analysis by Ries et al. (2023) evaluated seven studies encompassing 702 patients and approximately two thousand implants. The results significantly favoured non-smokers, demonstrating a pronounced and statistically significant difference in peri-implantitis risk compared to smokers at both implant and patient levels ($p < 0.001$) (Ries et al., 2023).

Further expanding on this topic, Vamos and colleagues (2024) conducted a systematic review and meta-analysis assessing various tobacco consumption forms, including conventional cigarettes, electronic cigarettes, waterpipes, and smokeless tobacco products. Their findings indicated that all nicotine-containing products, excluding electronic cigarettes, significantly contributed to greater marginal bone loss and poorer clinical parameters around implants compared to non-smokers. Interestingly, although the detrimental effects of electronic cigarettes did not reach statistical significance, users demonstrated inferior clinical

https://doi.org/10.24191/cos.v13i1.7262

outcomes relative to individuals with no history of smoking. This highlights the necessity of critically evaluating alternative tobacco products' impact on peri-implant health, despite their perceived safety compared to conventional smoking (Vamos et al., 2024).

A critical appraisal of the existing body of research emphasises the significance of study design and methodological approaches in influencing results. Variations in study protocols, especially between earlier investigations using traditional microbial identification methods and recent studies employing advanced genomic and bioinformatic techniques, contribute to inconsistencies in reported outcomes. Additionally, methodological differences in evaluating inflammatory and immune responses add further complexity to interpretations. Investigations into passive smoking's potential effects on peri-implant health have also provided insights, emphasising the broader implications of tobacco exposure and benefits associated with smoking cessation (Darby, 2022; Apatzidou, 2022).

Clinically, these findings reinforce the necessity for practitioners to advocate smoking cessation actively among patients, emphasising its substantial benefits on periodontal and peri-implant treatment outcomes. Educational interventions should integrate robust evidence-based information, highlighting the risks associated with smoking and emphasising quitting as integral to improving therapeutic outcomes. Despite recent advancements, current evidence remains somewhat limited regarding smoking as a definitive risk factor for peri-implantitis. Future research with robust, controlled study designs is essential to clarify this association further and provide unequivocal clinical guidance regarding tobacco's impact on peri-implant health (Darby, 2022; Apatzidou, 2022).

Anatomical and histological differences of natural teeth and dental implants

Significant anatomical and histological distinctions between natural dentition and dental implants markedly influence implants' susceptibility to peri-implant diseases (Coli et al., 2017; Lang & Berglundh, 2011; Berglundh et al., 1991; Buser et al., 1992; Berglundh et al., 1994). Dental implants functionally replace missing teeth by utilising a fixture that acts as an artificial tooth root, supporting a prosthetic crown that substitutes for the visible tooth structure (Vishwakarma et al., 2015; Gruber & Bosshardt, 2015). Although implant-supported prostheses closely mimic natural teeth clinically and aesthetically, distinct implant components are easily identifiable through radiographic imaging, differentiating them from natural dental structures (Vishwakarma et al., 2015; Gruber & Bosshardt, 2015).

Histologically, there are considerable differences between dental implants and natural teeth. Tooth development involves intricate interactions between ectodermal tissues originating from the first branchial arch and neural crest-derived ectomesenchyme. These interactions result in tooth bud formation, subsequently differentiating into three main structures: the enamel organ, which produces ameloblasts; the dental papilla, responsible for generating odontoblasts and pulp fibroblasts; and the dental follicle, giving rise to cementoblasts, osteoblasts, and periodontal ligament fibroblasts (Vishwakarma et al., 2015; Gruber & Bosshardt, 2015).

These specialised cellular populations collectively produce various mineralised and non-mineralised dental components, including enamel, dentin, cementum, alveolar bone, periodontal ligament (PDL), and dental pulp, resulting in a structurally complex biological entity anchored firmly within alveolar bone (Vishwakarma et al., 2015; Gruber & Bosshardt, 2015). The periodontium, composed of cementum, alveolar bone, periodontal ligament, and gingival tissues, provides a unique anchoring mechanism for natural teeth. Notably, this attachment apparatus fundamentally differs from dental implants due to the periodontal ligament's presence, a structure absent around implants (Vishwakarma et al., 2015; Gruber & Bosshardt, 2015).

https://doi.org/10.24191/cos.v13i1.7262

Conversely, dental implants directly integrate with alveolar bone through osseointegration, a biological process defined by direct structural and functional attachment of bone tissue to the implant surface, devoid of intermediary ligamentous structures (Gruber & Bosshardt, 2015). While osseointegration provides excellent mechanical stability initially, the lack of a periodontal ligament introduces several functional limitations. Implants do not benefit from the ligament's capacity to effectively distribute and dissipate mechanical stress, rendering them vulnerable to mechanical overload from excessive or improperly oriented occlusal forces. Additionally, implants lack the proprioceptive feedback in natural teeth, reducing their sensitivity to occlusal forces and increasing their risk of mechanical stress-induced complications.

The peri-implant mucosa surrounding implants differs significantly from natural gingival attachments (Buser et al., 1992). Peri-implant mucosa forms a direct epithelial attachment to the implant surface, lacking robust connective tissue fibres characteristic of natural gingival attachments, resulting in reduced mechanical resilience and increased susceptibility to microbial invasion (Berglundh et al., 1991). Furthermore, peri-implant mucosal tissues exhibit notably diminished vascularity compared to the richly vascular periodontal tissues around natural teeth (Berglundh et al., 1994). This limited vascular supply, mainly from alveolar bone and periosteal sources, can hinder immune and regenerative responses during inflammatory or infectious conditions (Berglundh et al., 1994).

These anatomical and physiological variances, particularly the absence of the periodontal ligament, limited vascularisation, and weaker mucosal attachment, substantially heighten implants' vulnerability to peri-implant disorders, especially peri-implantitis (Lang & Berglundh, 2011; Buser et al., 1992). Moreover, compromised vascularity around implants significantly impedes effective immune responses against bacterial challenges, perpetuating inflammation and subsequent tissue degradation. Consequently, precise and timely diagnostic methods are vital for clinicians to manage and mitigate peri-implant disease risks efficiently (Salvi et al., 2016; Carcuac & Berglundh, 2014).

Remnant of excess dental cement

A retrospective study examining 77 patients with 129 implants experiencing mechanical or biological complications identified residual cement in 73 implants, constituting 56% of the cases evaluated. Specifically, excess cement was detected in 11 of 32 implants associated with mechanical issues and 62 of 97 implants presenting biological problems. Critically, peri-implant disease occurred in 85% (62 cases) of implants where residual cement was present. Early-onset peri-implant disease accounted for roughly 12% (7 implants) of these cases, whereas delayed-onset peri-implant disease represented the majority at 88% (55 implants). Notably, all patients who had previously experienced periodontal disease and exhibited residual cement around implants subsequently developed peri-implantitis (Linkevičius et al., 2012; Valente & Andreana, 2016).

These results underscore the considerable etiological impact of residual cement in initiating and promoting peri-implant pathologies, particularly in individuals with an existing predisposition to periodontal diseases. The prevalent occurrence of delayed-onset peri-implantitis highlights the frequently unnoticed nature of cement-related complications, often remaining undetected during the immediate post-restoration phase.

Consequently, dental clinicians should strictly adhere to precise cementation protocols, ensuring complete elimination of excess cement to minimise associated risks. Additionally, rigorous postoperative follow-up through structured, routine examinations is essential to identify and effectively manage emerging complications promptly. These observations align with existing literature, emphasising the intricate interaction between localised etiological elements, such as residual cement, and individual patient susceptibility in peri-implant disease development. Hence, individualised preventive strategies and

https://doi.org/10.24191/cos.v13i1.7262

personalised care plans are vital for achieving sustainable implant success over time (Linkevičius et al., 2012; Valente & Andreana, 2016).

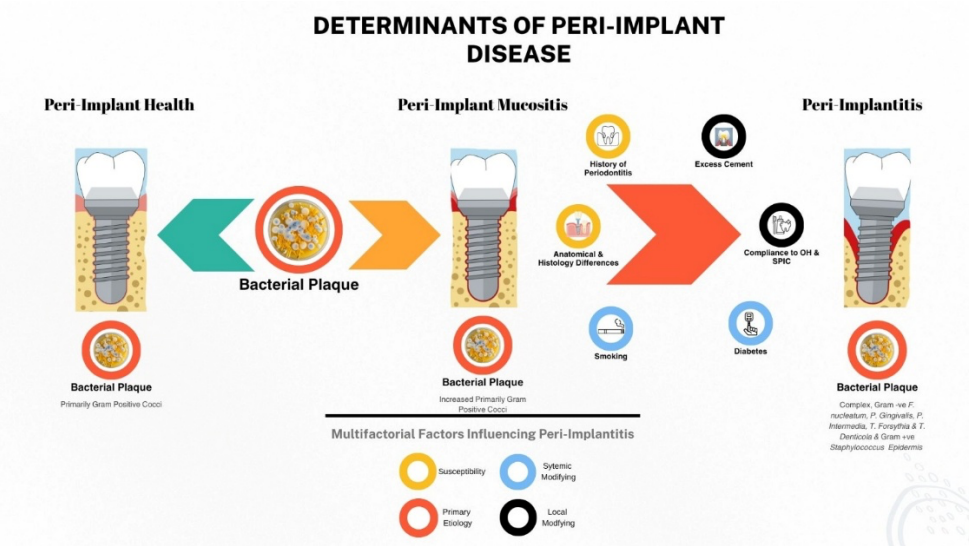

Figure 1. Conceptual determinants of peri-implant disease

CONCLUSION

Peri-implant diseases, encompassing peri-implant mucositis and peri-implantitis, emerge from complex interactions involving microbial biofilms, individual host susceptibility, patient behavioural factors, and intrinsic anatomical distinctions between dental implants and natural teeth. While microbial plaque remains the primary causative factor in peri-implant mucositis and substantially influences peri-implantitis, the transition from mucositis to peri-implantitis is critically mediated by systemic and local risk determinants, such as diabetes mellitus, history of periodontal disease, tobacco use, presence of residual cement, and insufficient compliance with maintenance programs (Figure 1).

Advancements in molecular techniques have significantly deepened the understanding of peri-implant microbiomes, uncovering parallels and critical differences compared to periodontal pathogens. Notably, opportunistic microorganisms, including Staphylococcus epidermidis, suggest peri-implant infections may share characteristics with biofilm-associated infections of other medical devices. Anatomically, the absence of periodontal ligament structures, reduced vascularisation, and relatively weaker mucosal attachment around implants inherently compromise local immune defence mechanisms. These anatomical limitations restrict effective immune responses and regenerative capacities, rendering peri-implant tissues vulnerable to progressive inflammation and bone deterioration.

Therefore, effective management and preventive strategies must adopt a patient-specific, risk-based approach. Essential preventive measures include rigorous oral hygiene practices, behavioural modification strategies, consistent adherence to structured supportive peri-implant therapy, and timely disease identification. It is imperative for clinicians to comprehensively assess systemic and local risk factors during implant planning and maintenance, particularly in patients predisposed to periodontitis.

https://doi.org/10.24191/cos.v13i1.7262

ACKNOWLEDGEMENTS/FUNDING

The authors would like to acknowledge the financial support of Universiti Teknologi Mara (UiTM), Cawangan Negeri Selangor, Kampus Sungai Buloh, for this research.

CONFLICT OF INTEREST STATEMENT

The authors agree that this article was written in the absence of any self-benefits, commercial or financial conflicts and declare the absence of conflicting interests with the funders.

AUTHORS' CONTRIBUTIONS

Mohamad Shamim Abu Ani conducted the research and wrote and revised the article. **Mohd Faizal Hafez Hidayat** conceptualised the central research idea and provided the theoretical framework. **Hafizul Izwan Mohd Zahari** anchored the review and revisions and approved the article submission.

REFERENCES

Albouy, J. P., Abrahamsson, I., Persson, L. G., & Berglundh, T. (2008). Spontaneous progression of peri-implantitis at different types of implants. An experimental study in dogs. I: clinical and radiographic observations. *Clinical oral implants research*, *19*(10), 997–1002. https://doi.org/10.1111/j.1600-0501.2008.01589.x

Almehmadi A. H. (2019). Awareness of population regarding the effects of diabetes on dental implant treatment in Jeddah, Saudi Arabia. *Heliyon,* 5(9), e02407. https://doi.org/10.1016/j.heliyon.2019.e02407

Alzahrani, A. S., & Abed, H. H. (2016). To what extent should dental implant placement be adopted as a standard for diabetic patients?. *Saudi Medical Journal*, 37(11), 1179–1183. https://doi.org/10.15537/smj.2016.11.15512

American Diabetes Association (2021). 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2021. Diabetes care, 44(Suppl 1), S15–S33. https://doi.org/10.2337/dc21-S002

Apatzidou D. A. (2022). The role of cigarette smoking in periodontal disease and treatment outcomes of dental implant therapy. *Periodontology* 2000, 90(1), 45–61. https://doi.org/10.1111/prd.12449

Baniulyte, G., & Ali, K. (2023). How does diabetes mellitus impact on the prognosis of dental implants?. *Evidence-based dentistry*, *24*(3), 114–115. https://doi.org/10.1038/s41432-023-00909-0

Berglundh, T., Mombelli, A., Schwarz, F., & Derks, J. (2024). Etiology, pathogenesis and treatment of peri-implantitis: A European perspective. *Periodontology 2000*, 10.1111/prd.12549. Advance online publication. https://doi.org/10.1111/prd.12549

Berglundh, T., Armitage, G., Araujo, M. G., Avila-Ortiz, G., Blanco, J., Camargo, P. M., Chen, S., Cochran, D., Derks, J., Figuero, E., Hämmerle, C. H. F., Heitz-Mayfield, L. J. A., Huynh-Ba, G., Iacono, V., Koo, K. T., Lambert, F., McCauley, L., Quirynen, M., Renvert, S., Salvi, G. E., … Zitzmann, N. (2018). Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *Journal of clinical periodontology,* 45 Suppl 20, S286–S291. https://doi.org/10.1111/jcpe.12957

Berglundh, T., Lindhe, J., Jonsson, K., & Ericsson, I. (1994). The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. *Journal of clinical periodontology*, *21*(3), 189–193. https://doi.org/10.1111/j.1600-051x.1994.tb00302.x

Berglundh, T., Lindhe, J., Ericsson, I., Marinello, C. P., Liljenberg, B., & Thomsen, P. (1991). The soft tissue barrier at implants and teeth. *Clinical oral implants research*, *2*(2), 81–90. https://doi.org/10.1034/j.1600-0501.1991.020206.x

Borgnakke, W. S., Genco, R. J., & Eke, P. I. (2017). Oral health and diabetes. In C. C. Cowie, C. C. Menke, L. J. Eberhardt, J. B. Gregg, & M. S. Narayan (Eds.), Diabetes in America (3rd ed., NIH Publication No. 17-1468). National Institute of Diabetes and Digestive and Kidney Diseases

Buser, D., Weber, H. P., Donath, K., Fiorellini, J. P., Paquette, D. W., & Williams, R. C. (1992). Soft tissue reactions to non-submerged unloaded titanium implants in beagle dogs. *Journal of periodontology*, *63*(3), 225–235. https://doi.org/10.1902/jop.1992.63.3.225

Carcuac, O., Abrahamsson, I., Derks, J., Petzold, M., & Berglundh, T. (2020). Spontaneous progression of experimental peri-implantitis in augmented and pristine bone: A pre-clinical in vivo study. *Clinical oral implants research*, *31*(2), 192–200. https://doi.org/10.1111/clr.13564

Carvalho, É. B. S., Romandini, M., Sadilina, S., Sant'Ana, A. C. P., & Sanz, M. (2023). Microbiota associated with peri-implantitis-A systematic review with meta-analyses. *Clinical oral implants research*, *34*(11), 1176–1187. https://doi.org/10.1111/clr.14153

Cho-Yan Lee, J., Mattheos, N., Nixon, K. C., & Ivanovski, S. (2012). Residual periodontal pockets are a risk indicator for peri-implantitis in patients treated for periodontitis. *Clinical oral implants research*, *23*(3), 325–333. https://doi.org/10.1111/j.1600-0501.2011.02264.x

Chrcanovic, B. R., Albrektsson, T., & Wennerberg, A. (2014). Diabetes and oral implant failure: a systematic review. *Journal of dental research*, 93(9), 859–867. https://doi.org/10.1177/0022034514538820

Coli, P., Christiaens, V., Sennerby, L., & Bruyn, H. (2017). Reliability of periodontal diagnostic tools for monitoring peri-implant health and disease. *Periodontology 2000*, *73*(1), 203–217. https://doi.org/10.1111/prd.12162

Darby I. (2022). Risk factors for periodontitis & peri-implantitis. *Periodontology 2000*, *90*(1), 9–12. https://doi.org/10.1111/prd.12447

Derks, J., & Tomasi, C. (2015). Peri-implant health and disease. A systematic review of current epidemiology. *Journal of clinical periodontology*, *42 Suppl 16*, S158–S171. https://doi.org/10.1111/jcpe.12334Derks, J., Schaller, D., Håkansson, J., Wennström, J. L., Tomasi, C., & Berglundh, T. (2016). Effectiveness of Implant Therapy Analyzed in a Swedish Population: Prevalence of Peri-implantitis. *Journal of dental research*, *95*(1), 43–49. https://doi.org/10.1177/0022034515608832

, J., Schaller, D., Håkansson, J., Wennström, J. L., Tomasi, C., & Berglundh, T. (2016). Effectiveness of Implant Therapy Analyzed in a Swedish Population: Prevalence of Peri-implantitis. *Journal of dental research*, *95*(1), 43–49. https://doi.org/10.1177/0022034515608832

Dowell, S., Oates, T. W., & Robinson, M. (2007). Implant success in people with type 2 diabetes mellitus with varying glycemic control: a pilot study. *Journal of the American Dental Association* (1939), 138(3), 355–398. https://doi.org/10.14219/jada.archive.2007.0168

Enteghad, S., Shirban, F., Nikbakht, M. H., Bagherniya, M., & Sahebkar, A. (2024). Relationship Between Diabetes Mellitus and Periodontal/Peri-Implant Disease: A Contemporaneous Review. *International*

dental journal, *74*(3), 426–445. https://doi.org/10.1016/j.identj.2024.03.010 Enteghad, S., Shirban, F., Nikbakht, M. H., Bagherniya, M., & Sahebkar, A. (2024). Relationship Between Diabetes Mellitus and Periodontal/Peri-Implant Disease: A Contemporaneous Review. *International dental journal*, *74*(3), 426–445. https://doi.org/10.1016/j.identj.2024.03.010

Gruber, R., & Bosshardt, D. D. (2015). Dental implantology and implants—tissue interface. In A. Vishwakarma, P. Sharpe, S. Shi, & M. Ramalingam (Eds.), ***Stem cell biology and tissue engineering in dental sciences*** (pp. 735–747). Academic Press. https://doi.org/10.1016/B978-0-12-397157-9.00078-3

Hardt, C. R., Gröndahl, K., Lekholm, U., & Wennström, J. L. (2002). Outcome of implant therapy in relation to experienced loss of periodontal bone support: a retrospective 5- year study. *Clinical oral implants research*, *13*(5), 488–494. https://doi.org/10.1034/j.1600-0501.2002.130507.x

Heitz-Mayfield, L. J. A., & Salvi, G. E. (2018). Peri-implant mucositis. *Journal of clinical periodontology*, *45 Suppl 20*, S237–S245. https://doi.org/10.1111/jcpe.12953

Hill A. B. (1965). The Environment and Disease: Association or Causation?. *Proceedings of the Royal Society of Medicine*, *58*(5), 295–300. https://doi.org/10.1177/003591576505800503

Karoussis, I. K., Müller, S., Salvi, G. E., Heitz-Mayfield, L. J., Brägger, U., & Lang, N. P. (2004). Association between periodontal and peri-implant conditions: a 10-year prospective study. *Clinical oral implants research*, *15*(1), 1–7. https://doi.org/10.1111/j.1600-0501.2004.00982.x

Koldsland, O. C., Scheie, A. A., & Aass, A. M. (2010). Prevalence of peri-implantitis related to severity of the disease with different degrees of bone loss. *Journal of periodontology*, *81*(2), 231–238. https://doi.org/10.1902/jop.2009.090269

Koldsland, O. C., Scheie, A. A., & Aass, A. M. (2011). The association between selected risk indicators and severity of peri-implantitis using mixed model analyses. *Journal of clinical periodontology*, *38*(3), 285–292. https://doi.org/10.1111/j.1600-051X.2010.01659.x

Kröger, A., Hülsmann, C., Fickl, S., Spinell, T., Hüttig, F., Kaufmann, F., Heimbach, A., Hoffmann, P., Enkling, N., Renvert, S., Schwarz, F., Demmer, R. T., Papapanou, P. N., Jepsen, S., & Kebschull, M. (2018). The severity of human peri-implantitis lesions correlates with the level of submucosal microbial dysbiosis. *Journal of clinical periodontology*, *45*(12), 1498–1509. https://doi.org/10.1111/jcpe.13023

Kröger, A., Hülsmann, C., Fickl, S., Spinell, T., Hüttig, F., Kaufmann, F., Heimbach, A., Hoffmann, P., Enkling, N., Renvert, S., Schwarz, F., Demmer, R. T., Papapanou, P. N., Jepsen, S., & Kebschull, M. (2018). The severity of human peri-implantitis lesions correlates with the level of submucosal microbial dysbiosis. *Journal of clinical periodontology*, *45*(12), 1498–1509. https://doi.org/10.1111/jcpe.13023

Lang, N. P., Berglundh, T., & Working Group 4 of the Seventh European Workshop on Periodontology (2011). Periimplant diseases: where are we now? Consensus of the Seventh European Workshop on Periodontology. *Journal of clinical periodontology*, *38 Suppl 11*, 178–181. https://doi.org/10.1111/j.1600-051X.2010.01674.x

Lee, C. T., Huang, Y. W., Zhu, L., & Weltman, R. (2017). Prevalences of peri-implantitis and peri-implant mucositis: systematic review and meta-analysis. *Journal of dentistry*, *62*, 1–12. https://doi.org/10.1016/j.jdent.2017.04.011Linkevicius, T., Puisys, A., Vindasiute, E., Linkeviciene, L., & Apse, P. (2013). Does residual cement around implant-supported restorations cause peri-implant disease? A retrospective case analysis. *Clinical oral implants research*, *24*(11), 1179–1184. https://doi.org/10.1111/j.1600-0501.2012.02570.x

Listgarten M. A. (1965). Electron Microscopic Observations on The Bacterial Flora of Acute Necrotizing Ulcerative Gingivitis. *The Journal of periodontology*, *36*, 328–339. https://doi.org/10.1902/jop.1965.36.4.328

Loe, H., Theilade, E., & Jensen, S. B. (1965). Experimental Gingivitis in Man. *The Journal of periodontology*, *36*, 177–187. https://doi.org/10.1902/jop.1965.36.3.177

Löe H. (1993). Periodontal disease. The sixth complication of diabetes mellitus. Diabetes care, 16(1), 329–334.

Martinez-Amargant, J., de Tapia, B., Pascual, A., Takamoli, J., Esquinas, C., Nart, J., & Valles, C. (2023). Association between smoking and peri-implant diseases: A retrospective study. *Clinical oral implants research*, *34*(10), 1127–1140. https://doi.org/10.1111/clr.14147

Meyer, S., Giannopoulou, C., Courvoisier, D., Schimmel, M., Müller, F., & Mombelli, A. (2017). Experimental mucositis and experimental gingivitis in persons aged 70 or over. Clinical and biological responses. *Clinical oral implants research*, *28*(8), 1005–1012. https://doi.org/10.1111/clr.12912

Mombelli, A., & Décaillet, F. (2011). The characteristics of biofilms in peri-implant disease. *Journal of clinical periodontology*, *38 Suppl 11*, 203–213. https://doi.org/10.1111/j.1600-051X.2010.01666.x

Mombelli, A., Casagni, F., & Madianos, P. N. (2002). Can presence or absence of periodontal pathogens distinguish between subjects with chronic and aggressive periodontitis? A systematic review. *Journal of clinical periodontology*, *29 Suppl 3*, 10–38. https://doi.org/10.1034/j.1600-051x.29.s3.1.x

Mombelli, A., van Oosten, M. A., Schurch, E., Jr, & Land, N. P. (1987). The microbiota associated with successful or failing osseointegrated titanium implants. *Oral microbiology and immunology*, *2*(4), 145–151. https://doi.org/10.1111/j.1399-302x.1987.tb00298.x

Oates, T. W., Jr, Galloway, P., Alexander, P., Vargas Green, A., Huynh-Ba, G., Feine, J., & McMahan, C. A. (2014). The effects of elevated hemoglobin A(1c) in patients with type 2 diabetes mellitus on dental implants: Survival and stability at one year. Journal of the American Dental Association (1939), 145(12), 1218–1226. https://doi.org/10.14219/jada.2014.93

Ong, C. T., Ivanovski, S., Needleman, I. G., Retzepi, M., Moles, D. R., Tonetti, M. S., & Donos, N. (2008). Systematic review of implant outcomes in treated periodontitis subjects. *Journal of clinical periodontology*, *35*(5), 438–462. https://doi.org/10.1111/j.1600-051X.2008.01207.x

Petersmann, A., Müller-Wieland, D., Müller, U. A., Landgraf, R., Nauck, M., Freckmann, G., Heinemann, L., & Schleicher, E. (2019). *Definition, classification and diagnosis of diabetes mellitus* [Clinical practice guidelines]. *Experimental and Clinical Endocrinology & Diabetes, 127*(Suppl. 1), S1–S7. https://doi.org/10.1055/a-1018-9078

Pjetursson, B. E., Helbling, C., Weber, H. P., Matuliene, G., Salvi, G. E., Brägger, U., Schmidlin, K., Zwahlen, M., & Lang, N. P. (2012). Peri-implantitis susceptibility as it relates to periodontal therapy and supportive care. *Clinical oral implants research*, *23*(7), 888–894. https://doi.org/10.1111/j.1600-0501.2012.02474.x

Pontoriero, R., Tonelli, M. P., Carnevale, G., Mombelli, A., Nyman, S. R., & Lang, N. P. (1994). Experimentally induced peri-implant mucositis. A clinical study in humans. *Clinical oral implants research*, *5*(4), 254–259. https://doi.org/10.1034/j.1600-0501.1994.050409.x

Quirynen, M., & Van Assche, N. (2011). Microbial changes after full-mouth tooth extraction, followed by 2-stage implant placement. *Journal of clinical periodontology*, *38*(6), 581–589. https://doi.org/10.1111/j.1600-051X.2011.01728.x

Roccuzzo, M., De Angelis, N., Bonino, L., & Aglietta, M. (2010). Ten-year results of a three-arm prospective cohort study on implants in periodontally compromised patients. Part 1: implant loss and radiographic bone loss. *Clinical oral implants research*, *21*(5), 490–496. https://doi.org/10.1111/j.1600-0501.2009.01886.x

Roccuzzo, M., Pittoni, D., Roccuzzo, A., Charrier, L., & Dalmasso, P. (2017). Surgical treatment of peri-implantitis intrabony lesions by means of deproteinized bovine bone mineral with 10% collagen: 7-year-results. *Clinical oral implants research*, *28*(12), 1577–1583. https://doi.org/10.1111/clr.13028

Ronin, D., Boyer, J., Alban, N., Natoli, R. M., Johnson, A., & Kjellerup, B. V. (2022). Current and novel diagnostics for orthopedic implant biofilm infections: a review. *APMIS : acta pathologica, microbiologica, et immunologica Scandinavica*, *130*(2), 59–81. https://doi.org/10.1111/apm.13197

Roos-Jansåker, A. M., Lindahl, C., Renvert, H., & Renvert, S. (2006). Nine- to fourteen-year follow-up of implant treatment. Part I: implant loss and associations to various factors. *Journal of clinical periodontology*, *33*(4), 283–289. https://doi.org/10.1111/j.1600-051X.2006.00907.x

Rothman, K. J., & Greenland, S. (2005). Causation and causal inference in epidemiology. *American journal of public health*, *95 Suppl 1*, S144–S150. https://doi.org/10.2105/AJPH.2004.059204

Salvi, G. E., Aglietta, M., Eick, S., Sculean, A., Lang, N. P., & Ramseier, C. A. (2012). Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. *Clinical oral implants research*, *23*(2), 182–190. https://doi.org/10.1111/j.1600-0501.2011.02220.x

Schnitman, P. A., & Han, R. K. (2015). Completely Digital Two-Visit Immediately Loaded Implants: Proof of Concept. The Journal of oral implantology, 41(4), 429–436. https://doi.org/10.1563/aaid-joi-D-15-00060Schwarz, F., Becker, K., & Renvert, S. (2015). Efficacy of air polishing for the non-surgical treatment of peri-implant diseases: a systematic review. *Journal of clinical periodontology*, *42*(10), 951–959. https://doi.org/10.1111/jcpe.12454

Schwarz, F., Derks, J., Monje, A., & Wang, H. L. (2018). Peri-implantitis. *Journal of clinical periodontology*, *45 Suppl 20*, S246–S266. https://doi.org/10.1111/jcpe.12954

Serino, G., & Ström, C. (2009). Peri-implantitis in partially edentulous patients: association with inadequate plaque control. *Clinical oral implants research*, *20*(2), 169–174. https://doi.org/10.1111/j.1600-0501.2008.01627.x

Sgolastra, F., Petrucci, A., Severino, M., Gatto, R., & Monaco, A. (2015). Periodontitis, implant loss and peri-implantitis. A meta-analysis. *Clinical oral implants research*, *26*(4), e8–e16. https://doi.org/10.1111/clr.12319

Taylor G. W. (2001). Bidirectional interrelationships between diabetes and periodontal diseases: an epidemiologic perspective. Annals of periodontology, 6(1), 99–112. https://doi.org/10.1902/annals.2001.6.1.99

Turkyilmaz I. (2010). One-year clinical outcome of dental implants placed in patients with type 2 diabetes mellitus: a case series. Implant dentistry, 19(4), 323–329. https://doi.org/10.1097/ID.0b013e3181e40366

Vámos, O., Komora, P., Gede, N., Hegyi, P., Kelemen, K., Varga, G., Mikulás, K., Kerémi, B., & Kispélyi, B. (2024). *The effect of nicotine-containing products on peri-implant tissues: A systematic review and network meta-analysis*. Nicotine & Tobacco Research**,** 26(10), 1276–1285. https://doi.org/10.1093/ntr/ntae085

Valente NA, Andreana S. Peri-implant disease: what we know and what we need to know. J Periodontal Implant Sci. 2016 Jun;46(3):136-51. doi: 10.5051/jpis.2016.46.3.136. Epub 2016 Jun 29. PMID:

27382503; PMCID: PMC4928203.

Verhulst, M. J. L., Loos, B. G., Gerdes, V. E. A., & Teeuw, W. J. (2019). Evaluating All Potential Oral Complications of Diabetes Mellitus. *Frontiers in endocrinology*, *10*, 56. https://doi.org/10.3389/fendo.2019.00056

Wang, H. L., Garaicoa-Pazmino, C., Collins, A., Ong, H. S., Chudri, R., & Giannobile, W. V. (2016). Protein biomarkers and microbial profiles in peri-implantitis. Clinical oral implants research, 27(9), 1129–1136. https://doi.org/10.1111/clr.12708 Zitzmann, N. U., Berglundh, T., Ericsson, I., & Lindhe, J. (2004). Spontaneous progression of experimentally induced periimplantitis. *Journal of clinical periodontology*, *31*(10), 845–849. https://doi.org/10.1111/j.1600-051X.2004.00567.x

Zitzmann, N. U., Berglundh, T., Ericsson, I., & Lindhe, J. (2004). Spontaneous progression of experimentally induced periimplantitis. *Journal of clinical periodontology*, *31*(10), 845–849. https://doi.org/10.1111/j.1600-051X.2004.00567.x