Frontiers in the understanding of peri-implant disease

Perez, Alexandre; Lombardi, Tommaso

How to cite

PEREZ, Alexandre, LOMBARDI, Tommaso. Frontiers in the understanding of peri-implant disease. In: Periodontal frontiers. H. Tal (Ed.). [S.l.] : IntechOpen, 2025. p. 1–26. doi: 10.5772/intechopen.1011935

This publication URL: https://archive-ouverte.unige.ch/unige:187592
Publication DOI: 10.5772/intechopen.1011935

Chapter

Frontiers in the Understanding of Peri-Implant Disease

Alexandre Perez and Tommaso Lombardi

Abstract

Peri-implant diseases are plaque-associated pathologic condition characterized by the inflammatory process affecting hard and soft tissues around endosseous dental implants. These frequent complications are the result of an imbalance between the host response and bacterial challenge, which may affect the peri-implant mucosa "mucositis" or also involve the supporting bone "peri-implantitis". Clinical aspects/signs of peri-implant diseases should be detected at each recall visit as well as swelling, erythema, bleeding on probing and suppuration. Moreover, many risk factors are involved in the development of peri-implant disease and most patients may have multiple risk factors including smoking, substance use disorders, socio-economic status, mental health disorders, old age and poor home dental care. In addition, iatrogenic, local, systemic, type of prosthesis and implant site-specific factors have been involved in the development of such complication. Clinicians must be aware of and recognizes the importance of such risk factors in order to manage them appropriately. This chapter aims to provide clinicians with an up-to-date understanding of peri-implant disease.

Keywords: peri-implantitis, peri-implant mucositis, dental implants, periodontal disease, treatment

1. Introduction

1.1 The growing penetration of dental implant therapy

Dental implant therapy has been established as a predictable and effective treatment option for replacing missing teeth. Cumulative survival rates of implant-borne restorations range from 90.5–100% after 5 years and 85.5–100% after 10 years, reflecting remarkably low failure rates—as low as 0 to 1.56 or 2 per 100 placed implants, respectively [1–6]. Recent systematic reviews have estimated a total survival rate of 96.4% [6]. Furthermore, by carefully adopting patient and case selection criteria, immediate implant protocols—traditionally considered challenging—may today be realized with the comparable survival rates to those of conventional delayed approaches [7–9]. Encouraging long-term success rates and evolving treatment strategies have contributed to the rapid growth and global acceptance of dental

implant therapy, with an estimated 12–15 million implants placed worldwide in 2023 [10]. Similarly, the prevalence of U.S. citizens rehabilitated with dental implants has increased at an annual rate of approximately 14%, rising from 5.7% in 2016 to a projected 23% by 2026 [11]. This substantial and growing proportion of the population rehabilitated with dental implants underscores the significance of implant therapy as a topic of increasing epidemiological and public health relevance.

The growing adoption of dental implant therapy has prompted an evolution in its evaluation criteria. Early success benchmarks, as defined by Albrektsson et al. in 1986, primarily focused on functional outcomes such as implant survival, prosthetic stability, radiographic bone loss and absence of peri-implant infection [12]. More recently, these criteria have expanded to include peri-implant soft tissue health, natural esthetics, prosthodontic performance and patient-centred outcomes such as satisfaction—reflecting a modern, multidimensional approach to assessing long-term implant success [13–16].

1.2 Dental implant complications, biological complications and their interplay

Despite their established long-term success, dental implant restorations are not complications-free. Dental implant complications are commonly classified into biological, technical and esthetic complications [5, 17, 18]. They may arise at any stage of treatment—whether during planning, surgery or implant function—and are influenced by various factors, including material and prosthetic design, anatomical constraints, unforeseen treatment plan deviations and patient-related systemic or behavioral aspects [17, 19–22]. For example, implant positioning and prosthetic design factors have been associated with long-term biological complications, suggesting a causal interplay between different types of implant-related complications [21–23].

Biological complications, that is peri-implant disease, represent the most common type of implant-related complication resulting in implant loss, followed by implant fracture and prosthetic overload. Notably, a potential association—regardless of causal direction—between mechanical and biological complications cannot be excluded [24].

1.3 Understanding and defining the clinical picture and diagnostic criteria of peri-implant disease

Peri-implant disease encompasses a spectrum of progressive inflammatory conditions of the peri-implant soft and/or hard tissues [25, 26]. Notably, the disease often progresses silently, remaining asymptomatic and undetectable by the patient [27]. Compounding this issue, many implant patients remain largely uneducated about potential complications, limiting early self-recognition outside of clinical examinations [28]. Despite the lack of overt symptoms, peri-implant disease is characterized by gradual and progressive tissue deterioration that can lead to esthetic and functional impairments up to the complete loss of the restoration [26].

Peri-implant disease is classified into two stages: peri-implant mucositis and peri-implantitis. Peri-implant mucositis is characterized by reversible inflammation of the peri-implant soft tissues. Peri-implantitis, on the other hand, involves inflammation of both soft and hard tissues accompanied by progressive and accelerating loss of supporting bone [25, 29].

Over the past decades, the scientific community has made the repeated efforts to standardize and refine the diagnostic criteria for peri-implant diseases.

Albrektsson and Isidor defined peri-implant mucositis as a reversible inflammation of the soft tissues as opposed to peri-implantitis being diagnosed by the irreversible and progressive loss of supporting bone [30]. Subsequent consensus reports, including those by Linde and Meyle and Zitzmann and Berglundh in 2008, defined peri-implant disease based on clinical indicators such as bleeding on probing (BoP) and radiographic bone loss [31, 32]. Lang and Berglundh extended these definitions within the broader context of tissue-level changes [33]. The 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases provided the most recent and potentially most comprehensive and clinically applicable criteria to date differentiating Peri-Implant Mucositis and Peri-Implantitis from Peri-Implant Health (**Figure 1**, **Table 1**) [14, 33, 34].

In clinical practice, it is important to recognize that a certain degree of marginal bone remodeling after implant placement and prosthetic loading is part of the normal physiological adaptation and should be expected [12, 35]. Seminal reports on crestal bone changes following prosthetic restoration in two-stage surgical protocols with delayed loading established that up to 1.5 mm of bone loss in the first year after loading and less than 0.2 mm annually thereafter may be considered within the range of physiological remodeling [12, 14, 36]. While these thresholds prevail as general clinical indicators, contemporary consensus emphasizes the importance of temporal stability in peri-implant bone levels rather than strict adherence to absolute values [14]. Progressive bone loss beyond initial remodeling, particularly when associated

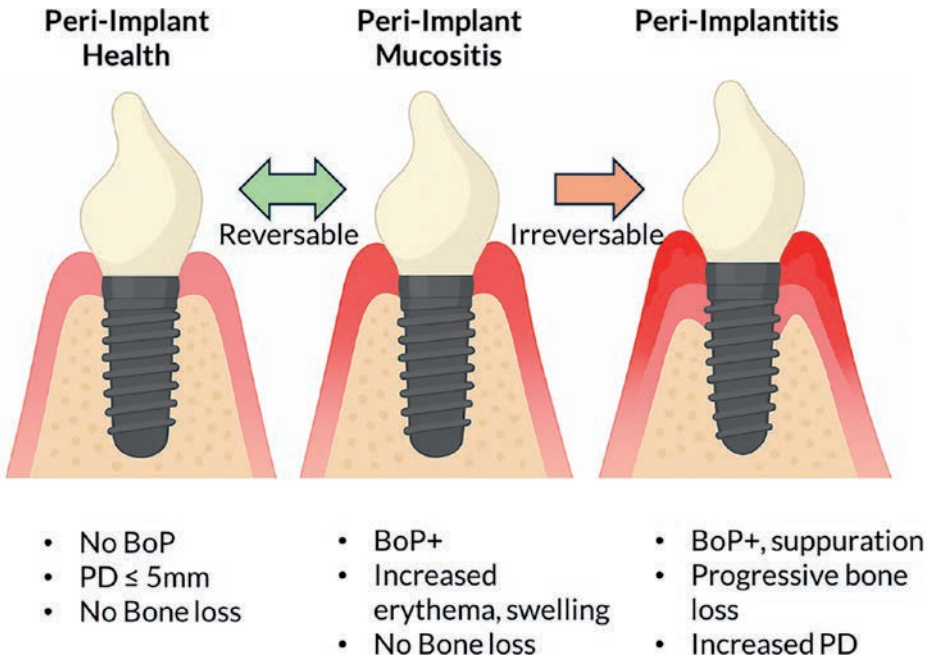

Figure 1.
Schematic illustration comparing peri-implant health, peri-implant mucositis, and peri-implantitis. Healthy peri-implant tissues characterize peri-implant health, no bleeding on probing (BoP), normal probing depth within normal ranges, and no radiographic bone loss. Peri-implant mucositis presents with BoP, increased erythema, and swelling of the soft tissues but without bone loss; this condition is considered reversible upon appropriate intervention. In contrast, peri-implantitis is associated with BoP and/or suppuration, progressive marginal bone loss, and increased probing depths; this condition is considered irreversible and requires surgical intervention.

Authors	Peri-implant mucositis		Peri-implantitis	
	Key features	Clinical parameters	Key features	Clinical parameters
Albrektsson & Isidor [30]	Reversible; only soft tissues	Soft tissue inflammation only	Irreversible inflammation and progressive bone loss	Mobility, radiographic bone loss, inflammation
Lindhe & Meyle [31]	BoP+; inflammation	No radiographic bone loss; BoP+	Inflammation with bone loss	BoP+/ suppuration; radiographic bone loss
Zitzmann & Berglundh [32]	BoP without bone loss beyond normal remodeling	BoP+, stable bone level	Bone loss beyond expected remodeling	Probing depth increase; radiographic bone loss
Lang & Berglundh [33]	Inflammation; reversible upon therapy	BoP+, no additional bone loss; inflammation present	Inflammation of both soft and hard tissues with bone loss	Increased probing depth, recession, bone loss
World Workshop / Berglundh & Armitage [14]	BoP+ and/or suppuration without bone loss beyond normal remodeling	BoP+, Probing depth may increase, but bone levels remain stable	BoP+ / suppuration, increased probing depth, and bone loss ≥3 mm	BoP+, PD ≥ 5 mm, bone level ≥ 3 mm apical of implant shoulder

BoP+: positive bleeding on probing, PD: probing depth.

Table 1.
Key features and clinical parameters of peri-implant mucositis and peri-implantitis.

with clinical signs of inflammation, indicates peri-implant disease. Regular diagnostic follow-ups, including clinical and radiographic examinations compared to baseline post-loading, remain central to early detection [33]. The maintenance of stable, unremarkable peri-implant soft and hard tissue conditions—characterized by the absence of excessive radiographic bone loss, inflammation and the presence of healthy, keratinized epithelium—supports peri-implant health and can be regarded as a leitmotif in the clinical diagnostic for long-term peri-implant health (**Table 2**) [37].

Despite the development of refined diagnostic criteria and their importance in standardizing the research on disease prevalence in epidemiological and disease surveillance studies, the clinical and biological heterogeneity in tissue response, disease progression and defect morphology continues to present challenges—not necessarily in recognizing peri-implant disease itself, but in adequately classifying its severity, progression and clinical therapeutic approaches [38–41]. Unlike, for example periodontitis and other pathological conditions that benefit from established staging systems, current definitions of peri-implant diseases remain largely binary [42, 43]. Future research efforts may contribute to developing more differentiated grading systems, enabling improved prognostic accuracy and refined therapeutic approaches (**Figure 2**).

1.4 Epidemiology of peri-implant disease

In epidemiology, two fundamental metrics are used to characterize disease patterns: prevalence and incidence. Prevalence describes the proportion of individuals affected by a condition at a specific point or over a defined period, while incidence refers to the number of new cases that arise within a specified timeframe. Together, these measures

Time after implant loading	Expected bone level changes
First-year	≤1.5 mm
After the first year	≤ 0.2 mm per year

Table 2.
Time after implant loading and expected level changes.

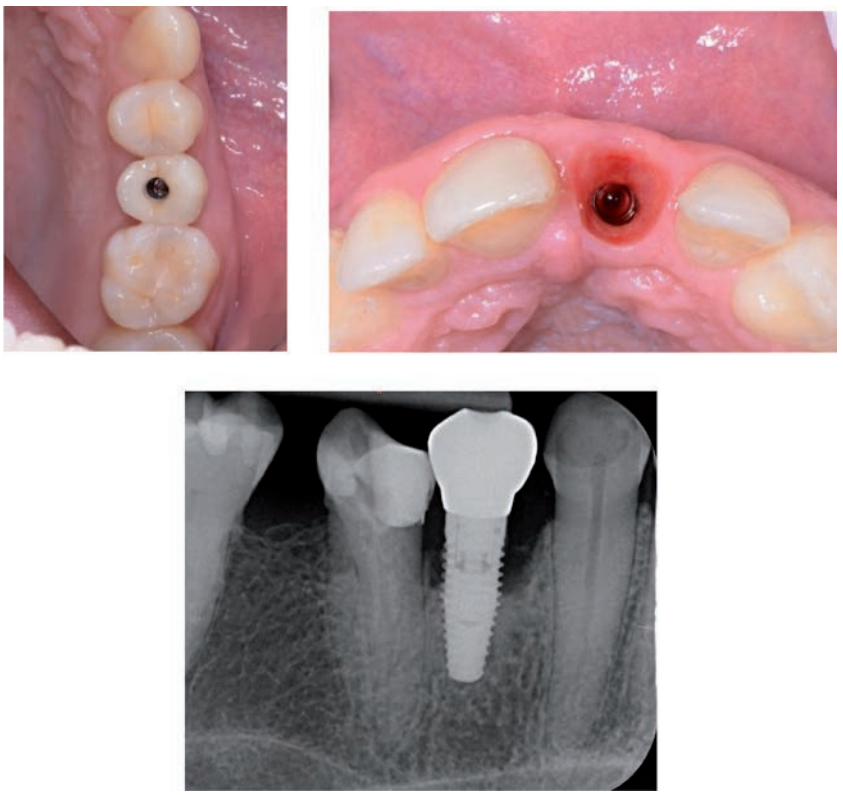

Figure 2.
Screw-retained single-unit crown placement site 25 showing good peri-implant health (top left), peri-implant mucositis highlighted after removal of a crown site 21 (top right) and peri-implantitis site 44 on intraoral radiography.

provide insight into the burden and dynamics of peri-implant disease within a given population. They may be assessed at the implant level (implant-based) or at the patient level (subject-based), offering a more nuanced understanding of both peri-implantitis and peri-implant mucositis across diverse clinical and patient-centred contexts.

The reported prevalence of peri-implant diseases varies considerably across clinical studies. Comprehensive systematic reviews published in 2015 and 2017 reported that the patient-level prevalence of peri-implant mucositis ranges from 19 to 65% and that of peri-implantitis from 1 to 47% [44]. Calculated average and weighted prevalence estimates derived from meta-analytical approaches may offer a more tangible indication of the overall epidemiological burden of peri-implant diseases. Based on aggregated data from multiple cross-sectional studies, peri-implant mucositis affects approximately 1 in 2 patients (43–47%), while peri-implantitis is observed in about 1 in 5 patients (20–22%) [44, 45].

The weighted mean prevalence of peri-implantitis at the implant level is estimated at 9.3%, compared to 19.8% at the patient level [45]. This discrepancy highlights the greater clinical impact of subject-based prevalence, as individual patients often present with multiple implants.

The considerable variability in the reported prevalence rates of peri-implant diseases can be attributed to several methodological and clinical factors. One of the most critical sources of inconsistency is the lack of standardized case definitions and diagnostic criteria across studies—particularly regarding parameters such as marginal bone levels [46]. This gap was rectified by the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, which introduced consensus-based diagnostic criteria for both disease entities [14, 34].

Incidence data of peri-implantitis are less well documented but indicate a relatively consistent trend [47–52]. It is further important to consider that the incidence of peri-implant disease has been demonstrated to increase with time in function [48, 50, 52]. While longitudinal studies have reported a patient-level cumulative incidence from 14.5 to 22% over 9 to 10 years following implant placement, other investigations have documented higher annual incidence rates of up to 16% per patient-year [50–52].

Finally, clinician surveys confirm peri-implant diseases as a frequently encountered complication in clinical practice [41]. Specifically, surveys among US periodontologists indicated that up to 10% of implants required to be removed due to peri-implantitis. This finding highlights a significant unmet need for effective, evidence-based treatment protocols, underscoring the ongoing clinical challenge of managing peri-implant disease.

1.5 Plaque and biofilm association of peri-implant disease and differences between implant-associated and tooth-associated biofilm

Strong evidence suggests that bacterial biofilms represent the most important etiological factor for the onset and progression of peri-implant disease [14, 33]. Clinical studies have demonstrated a direct causal effect of biofilm accumulation on triggering a peri-implant inflammatory tissue response, with a significant dose-dependent association [53–56]. Full mouth plaque scores above 25% have, for example, correlated with a greater prevalence of peri-implantitis in patients with a history of periodontitis [57]. In contrast, anti-infective therapy has demonstrated efficacious in reducing peri-implant soft tissue inflammation and arresting disease progression [58]. In addition, ligature-induced preclinical models have advanced the understanding of peri-implantitis by clearly demonstrating the role of infectious bacterial biofilms in driving the progressive inflammatory breakdown of peri-implant soft tissues and alveolar bone, specifically on a histological level [59].

Compared to orthopedic implants, osseointegrated dental implants feature a transmucosal design penetrating the oral mucosa, establishing a direct interface between the peri-implant tissues—particularly the alveolar bone—and the bacteria-rich oral environment. This anatomical configuration increases their susceptibility to biofilm accumulation and subsequent inflammatory complications [60]. Like natural teeth, implants are surrounded by a soft tissue seal that protects the underlying bone from microbial challenges. Specifically, the oral, sulcular, and junctional (or junctional-like) epithelium around implants forms a biological barrier against the external environment [33, 61]. However, unlike natural teeth—where gingival connective tissue fibers insert perpendicularly into the cementum—dental implants lack a true

connective tissue attachment [61]. Instead, circumferential collagen fibers aligned parallel to the implant surface stabilize the peri-implant soft tissue seal without, however, inserting into the implant material, thereby reducing the mechanical resilience of the seal to disruption [61].

Despite both bearing effective soft-tissue seals, the biofilm-associated inflammatory response between teeth and implants differs markedly. Specifically, implants show significantly faster tissue breakdown and a markedly faster progression into bone loss upon infection. This difference might be associated with the potential weakening of the epithelial barrier due to ulceration and edematous swelling [29, 33, 62]. Comparative analyses of soft tissue biopsies from teeth and implants revealed notable histopathological differences between peri-implantitis and periodontitis. Specifically, lesions associated with peri-implantitis compared to periodontitis are characterized by a larger inflammatory infiltrate and greater density of immune cells, that is plasma cells, macrophages and neutrophils. Peri-implant inflammatory infiltrates tend furthermore to extend more apically beyond the pocket epithelium compared to those in periodontitis and are not contained by a surrounding capsule of healthy connective tissue, arresting its progression (**Figure 3**) [29, 63].

These observed differences in the inflammatory response may be attributed to the lower vascularisation of peri-implant soft tissues. Specifically, soft tissue blood supply in peri-implant tissues is primarily derived from the periosteum of the crestal bone as opposed to natural teeth, where the richly vascularised periodontal ligament contributes to blood supply significantly [64]. Over time, bone remodeling and recession around implants can further compromise the limited blood supply, affecting the ability of peri-implant tissues to respond to inflammatory challenges further [61].

Despite advances in understanding peri-implant disease, the transition from health to peri-implantitis appears to be driven by a complex and partly self-reinforcing interplay between biofilm maturation on the implant surface and the host's inflammatory response [29]. A key event in disease progression is the shift from a symbiotic to a dysbiotic implant-associated biofilm, which has been described to be lower in quality and less diverse, with a higher accumulation of specific bacteria

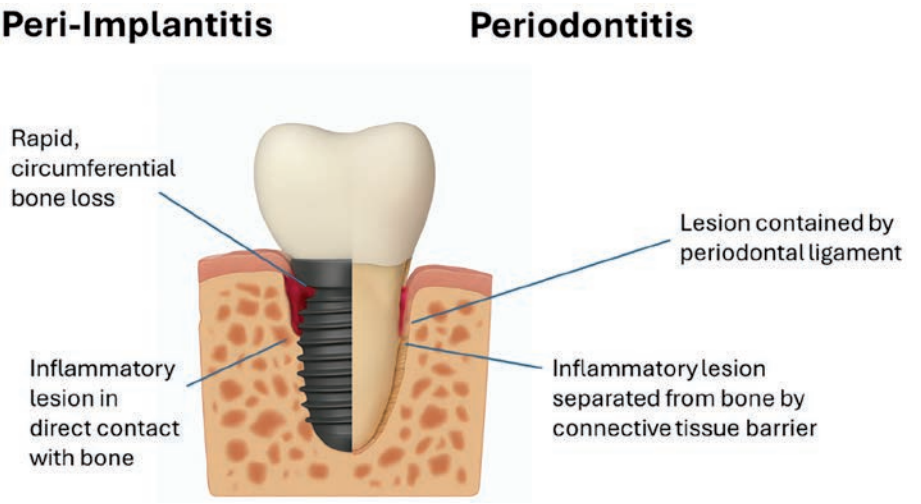

Figure 3.
Anatomical and inflammatory differences between peri-implantitis (left) and periodontitis (right).

compared to periodontal biofilms [60, 65]. This shift is promoted by changes in nutrient availability, pH alterations, oxygen depletion and the emergence of pathobionts, all fueled by sustained chronic inflammation [66]. As the disease progresses, biofilms mature and become increasingly resistant to host immune responses and chemotherapeutic agents [65]. Notably, mature biofilms are less responsive to antiseptic and antibiotic treatments as opposed to, for example planktonic bacteria and early, immature biofilm communities, which have proven well controllable through established preventive antibiotic protocols during, for example implant provision [66, 67].

1.6 Novel concepts in the understanding of dental implant integration and rejection

Recent research has highlighted the role of immunological factors in implant osseointegration, giving rise to the emerging field of osteoimmunology [68–70]. Within this framework, implant placement induces a transient, acute inflammatory response which—when involving an inert biomaterial such as titanium—may result in a favorable regenerative healing response, that is osseointegration. This process contrasts a situation in which chronic inflammation may lead to fibrous encapsulation of the biomaterial and eventual implant rejection [71].

Unlike natural teeth, dental implants trigger a controlled foreign body reaction that leads to the formation of a cortical bone capsule—commonly referred to as osseointegration. This structure reflects a state of tissue homeostasis, maintained by a balance between osteoclastic and osteoblastic activities [69]. The evolving understanding of osseointegration has been propelled, in part, by insights into macrophage behavior—specifically, their polarization from a pro-inflammatory M1 into a pro-regenerative M2 phenotype [72–74]. Conversely, M1 macrophages play a critical role in the pathogenesis of peri-implant disease by releasing pro-inflammatory cytokines such as interleukin-1 (IL-1) and tumor necrosis factor-alpha (TNF-α), both of which are the potent mediators of osteolysis and inflammatory tissue degradation [75].

Disruption of peri-implant tissue homeostasis—whether by plaque accumulation or other factors—can trigger an inflammatory response leading to peri-implant osteolysis and a reduced capacity for tissue repair. The onset and progression of peri-implantitis may be influenced by a combination of local, systemic and patient-related behavioral factors, rendering it a clinically complex and challenging condition to manage [29].

2. Risk factors

The susceptibility of dental implants to peri-implantitis is shaped by a range of systemic, local, and iatrogenic factors. Accurate prognostic assessment is essential for effective risk management and implementing preventive strategies throughout implant therapy.

From an academic perspective, the determinants of disease development are typically classified as either risk indicators or risk factors. Risk indicators are characteristics or exposures associated with disease presence, primarily identified through cross-sectional studies and denote correlation rather than causation. In contrast, risk factors are established through longitudinal research and imply a temporal and potentially causal relationship with disease onset.

Importantly, most studies on peri-implant disease are observational in nature and do not meet the methodological criteria required to establish the true risk factors. Nevertheless, to provide clinicians with a practical framework for evaluating patient-specific risks during implant placement and maintenance, this chapter will use the term risk factors in a broad sense—encompassing both evidence-based risk factors and strongly associated risk indicators.

In clinical practice, it is often useful to distinguish between risk factors that are intrinsic to the patient and those that are treatment-related or behaviourally driven—and therefore potentially modifiable. A more nuanced classification differentiates among patient-related systemic and local factors, iatrogenic influences, behavioral risks and implant- or site-specific conditions. It is also important to acknowledge that while some risk factors are well-established, others remain under investigation, with current evidence insufficient to confirm a definitive causal relationship (**Table 3**).

Patient-related systemic and local factors constitute the foundational determinants of an individual's susceptibility to peri-implantitis. These factors are often non-modifiable or only marginally amenable to change. In contrast, iatrogenic and behavioral factors represent key opportunities for risk mitigation through appropriate clinical decision-making, patient education and reinforcement of compliance—ideally initiated prior to implant placement as part of primordial prevention concepts [76]. Finally, implant- and site-specific variables, such as implant design and bone quality, must be carefully assessed for their potential influence on case-specific outcomes and long-term treatment success (**Figure 4**).

2.1 Patient-related risk factors

2.1.1 History of periodontitis

Both longitudinal and cross-sectional studies evidence that patients with a history of periodontitis are more susceptible to peri-implantitis [50, 52, 77–79]. Meta-analysis has indicated that a history of periodontitis exposes patients to a 2.2 to 9.2 elevated risk for peri-implantitis [29]. From an epidemiological point of view, it is important to note that the global prevalence of periodontitis, particularly its moderate to severe forms, is high and consistent across populations, with estimates showing that severe

Category	Description	Examples
Systemic	Factors related to the patient's general health influencing susceptibility to inflammation and healing.	Smoking, diabetes mellitus, genetic predisposition, osteoporosis, immunodeficiency
Local factor-related	Conditions in the oral environment affecting plaque control and tissue response.	History of periodontitis, poor oral hygiene, keratinized mucosa deficiency
Behavioral	Patient habits and compliance affecting disease control	Poor plaque control, irregular maintenance visits, smoking
Iatrogenic	Factors related to clinical procedures, surgical techniques and prosthetic design.	Residual cement, implant malposition, inadequate prosthetic emergence profile, overloading
Implant- and site-specific	Implant or anatomical site-related factors affecting peri-implant tissue health.	Implant surface characteristics, bone quality, soft tissue thickness

Table 3.
Risk factors.

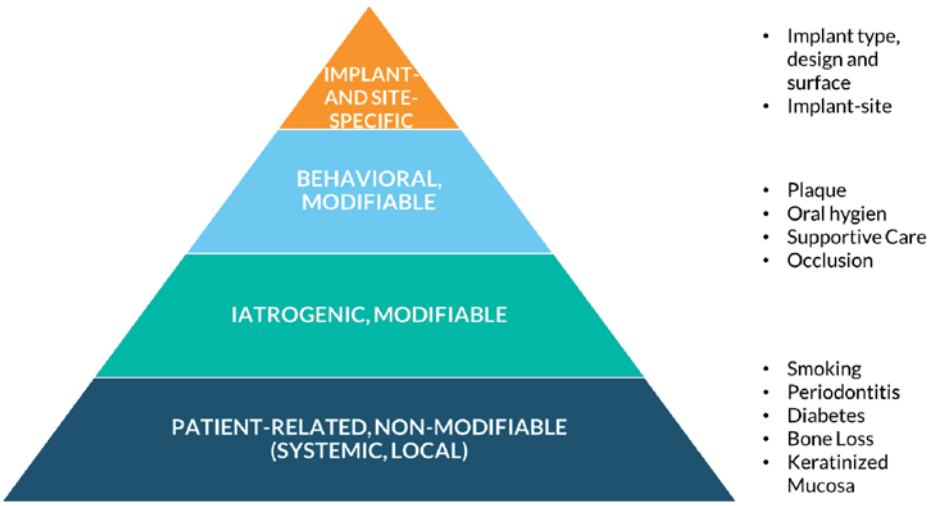

Figure 4.
Hierarchical classification of peri-implantitis risk factors. The pyramid illustrates the layered influence of risk factors, from fundamental patient-related systemic and local factors at the base to implant- and site-specific factors at the top.

periodontitis affects 10–15% of adults worldwide and overall periodontitis (including mild and moderate) affects more than 50% of adults [80–82]. Clinicians planning implant therapy are therefore advised to assess patients for signs of periodontal disease and to identify the indicators of current or past disease activity, such as a history of periodontitis-related tooth loss. Identifying affected patients may necessitate adjustments to the treatment and follow-up plan, such as periodontal treatment prior to commencing implant therapy and implementing supportive periodontal care protocols [78, 83, 84]. Although some studies suggest that implant placement into previously infected periodontal sites may be feasible—provided that extraction sockets are thoroughly debrided and meticulously disinfected—there is currently limited evidence supporting the long-term prognosis of such approaches [85]. There is also currently insufficient evidence to support the notion that only severe forms of periodontitis significantly increase the risk of peri-implantitis [78].

2.1.2 Smoking

Smoking, as well as electronic cigarette use, has been reported to significantly affect implant failure rates, the risk of postoperative infection as well as marginal bone loss [81–83]. In addition, several studies have reported smoking to affect osteogenesis, angiogenesis and plaque accumulation, essential for implant integration and health [83–85]. Individual studies have evidenced that smokers may exhibit a 2.7 to 31.6 times elevated risk for peri-implantitis [29, 86–93] Although numerous studies have demonstrated a strong association between smoking and peri-implantitis, methodological limitations—particularly the inability to control for confounding factors adequately—have so far omitted a consensus on the relation between smoking and peri-implantitis [14, 29]. Clinicians may consider this important factor during treatment provision as a modifiable patient-behavioral factor with relatively high potential importance for treatment prognosis.

2.1.3 Diabetes

With a global prevalence of 8%, type I and insulin-deficient type II (accounting for 90–95% of cases) diabetes comprises a group of metabolic disorders characterized by impaired glycemic control. Evidence supports a causal relationship between diabetes and a moderately elevated 1.9- to 3-fold risk for peri-implantitis and peri-implant bone loss [54, 94, 95]. Specifically, poor glycemic control has been reported to significantly increase the risk of peri-implantitis and implant failure [54, 95–97].

Elevated HbA1c levels exert multiple detrimental effects on peri-implant health, including impairing bone metabolism and osseointegration, compromising the quality and resilience of the peri-implant soft tissue seal and impairing neutrophil function, thereby weakening the host's ability to control biofilm-induced inflammation [98–100]. Importantly, evidence also suggests that patients with adequate glycemic control (HbA1c ≤7%) do not exhibit an increased risk of peri-implantitis compared to non-diabetic individuals [101–104].

However, study inconsistencies remain, particularly regarding the heterogeneous use of diagnostic parameters (e.g. HbA1c levels, fasting blood glucose, or self-reported diabetes status). For example, as opposed to type type 1, only type 2 diabetes has been associated with a higher incidence of peri-implant disease [105–107]. These discrepancies may explain why current consensus reports have not yet recognized diabetes as a definitive risk factor for peri-implantitis or implant loss [14, 29]. In conclusion, the detrimental effect of poorly controlled diabetes on peri-implant health is relatively well established and should be carefully considered during both the planning and maintenance phases of implant therapy.

2.1.4 Other systemic conditions

A whole series of other systemic conditions that may be associated with an elevated risk for peri-implantitis comprising, for example cardiovascular disease, rheumatoid arthritis, osteoporosis, specifically when medicated with bisphosphonates, obesity, as well as age and gender, have been researched and discussed throughout the literature [29, 108–112]. Compared to other systemic risk factors, these conditions remain insufficiently studied, and a conclusive association with peri-implantitis has yet to be established [14, 29]. However, certain conditions—such as obesity—may serve as surrogate indicators of other well-established risk factors and could, therefore, contribute to the overall assessment of a patient's risk profile for peri-implantitis. Other conditions, such as osteoporosis, have been associated with increased peri-implant bone loss and may, therefore, be considered modifying factors in the onset and progression of peri-implantitis [112].. Finally, patients may also exhibit a genetic predisposition to peri-implantitis, involving polymorphisms in genes related to immune regulation and inflammatory response—such as interleukin-1 (IL-1), interleukin-6 (IL-6), interleukin-10 (IL-10) and tumor necrosis factor-alpha (TNF-α) [113]. While some evidence suggests a potential role of these genetic factors in the pathogenesis of peri-implantitis, they are not yet considered clinically actionable [29].

2.1.5 Keratinized mucosa

There is growing interest in the role of keratinized mucosa (KM) in maintaining peri-implant health and ensuring a stable peri-implant soft tissue seal [61]. Clinical

studies and systematic reviews suggest that implant sites with a limited width of KM (≤ 2 mm) are more prone to plaque accumulation and present higher plaque and bleeding on probing scores, indicative of increased peri-implant mucosal inflammation [114–117]. Notably, the absence of KM has been associated with patient-reported brushing discomfort and reduced oral hygiene effectiveness, potentially contributing to plaque accumulation and inflammation [118]. Conversely, implants surrounded by an adequate band of KM appear to respond more favorably to peri-implant mucositis treatment, underlining the role of KM in supporting disease resolution [119].

A recent systematic review reported that implants lacking KM have a 2.78-fold higher risk of developing peri-implantitis than those with sufficient KM [120]. However, it should be noted that much of the current evidence stems from observational studies with inherent limitations, and a direct causal relationship remains under debate. Nonetheless, these findings underscore the importance of ensuring an adequate peri-implant soft tissue phenotype—including both KM width and mucosal thickness—as part of comprehensive strategies to promote long-term peri-implant health.

2.2 Iatrogenic and behavioral factors

2.2.1 Plaque control, oral hygiene and professional supportive peri-implant care

Given the plaque-associated nature of peri-implant disease, poor plaque control remains one of the most significant risk factors and predictors for peri-implantitis, with studies reporting a 3- to 14-fold increased risk in patients with inadequate plaque control [54, 57, 121–124]. While plaque scores indicate an elevated microbial burden, the progression to peri-implantitis depends on the host's inflammatory response, highlighting the importance of clinical indicators such as bleeding on probing to assess the individual susceptibility [125]. Plaque accumulation is closely linked to oral hygiene practices but also to factors affecting accessibility for self-care and the provision of regular professional maintenance therapy. Clinical studies have demonstrated that peri-implantitis occurs more frequently at sites with limited accessibility for cleaning compared to well-accessible areas, underscoring the importance of accessibility in prosthetic planning and design [126, 127].

Furthermore, supportive peri-implant care (SPIC) has been shown to significantly reduce peri-implantitis incidence from 44 to 18% over 5 years and the necessity for associated treatment intervention from 41 to 27% over a ten-year period [78, 128]. Importantly, patients adhering to regular SPIC exhibited a sevenfold lower risk of developing peri-implantitis compared to non-compliant individuals, underscoring the dual benefit of professional biofilm removal and early diagnosis of biological complications during recall visits [129]. Likewise, early marginal bone loss around implants has been identified as a significant risk indicator for peri-implantitis development, making its early detection during maintenance visits crucial for preventive intervention [50, 78]. Although not exclusively related to iatrogenic factors or patient behavior, effective plaque control remains crucial for reducing the risk of peri-implantitis. This requires attention from a restorative design, patient education and compliance and clinician awareness standpoint [130].

2.2.2 Occlusal overload

Clinical studies indicate a potential correlation between clinical signs of occlusal overload and an increased incidence of peri-implantitis [123, 131]. Furthermore,

occlusal overload has also been reported to potentially cause peri-implant bone loss increasing the susceptibility for peri-implantits [132]. Likewise, bruxism, although not directly related to peri-implantitis, has been correlated to a significantly higher risk of failure [133, 134].

Systematic reviews remain inconclusive regarding a direct causal relationship between occlusal overload and peri-implantitis, as occlusal overload alone does not appear to induce peri-implantitis but, conversely, may accelerate bone loss in the presence of plaque-induced inflammation [135]. As a result, occlusal overload is potentially best considered a modifying rather than a causative factor in peri-implant tissue breakdown. In the context of primary prevention, clinicians are advised to routinely assess and manage potential malocclusion in implant patients, particularly in those displaying elevated risk profiles.

2.3 Implant- and site-specific factors

Implant- and site-specific factors refer to anatomical, surgical and implant-design-related aspects that may influence a patient's susceptibility to peri-implantitis or modulate the disease's progression. These factors typically indirectly affect plaque accumulation, soft tissue resilience or access for maintenance. Certain aspects—such as implant positioning or the emergence profile—originate from clinical decisions and may, therefore, be considered iatrogenic. Nevertheless, once established, these factors define the long-term anatomical and biomechanical environment in which peri-implant disease may develop or progress.

Material- and design-related aspects of implant-borne restorations vary widely and include factors such as implant type, surface characteristics, the implant–prosthetic connection type, and the contour and type of the prosthetic restoration, all potentially having a direct causal or potentially modifying effect on the risk of peri-implantitis (**Figures 5** and **6**).

The implant–abutment microcap has long been discussed as a potential reservoir for bacterial colonization and a potentially contributing factor to peri-implant inflammation and marginal bone loss (**Figure 7**) [136].

Tissue-level implants and platform-switching concepts relocate the microgap away from the marginal bone, thereby reducing or eliminating its direct impact on peri-implant tissues [137–141]. Both designs have been demonstrated to reduce crestal bone loss compared to platform-matched variants significantly [142, 143].

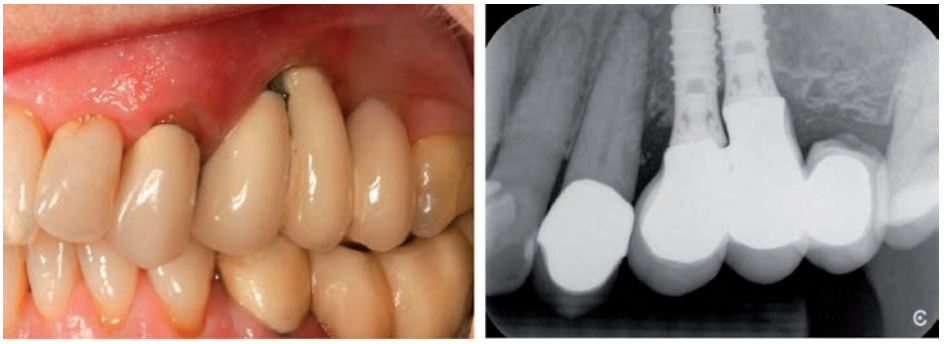

Figure 5.
Iatrogenic peri-implantitis sites 24 and 25 resulting from inadequate inter-implant distances and prosthetic design (on the left clinical image, on the right intraoral radiograph).

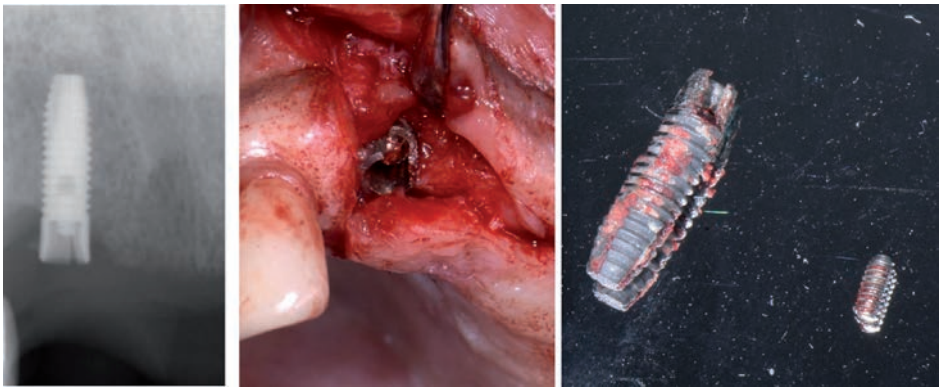

Figure 6.
Peri-implantitis following implant fracture resulting from insufficient implant diameter as a bridge abutment.

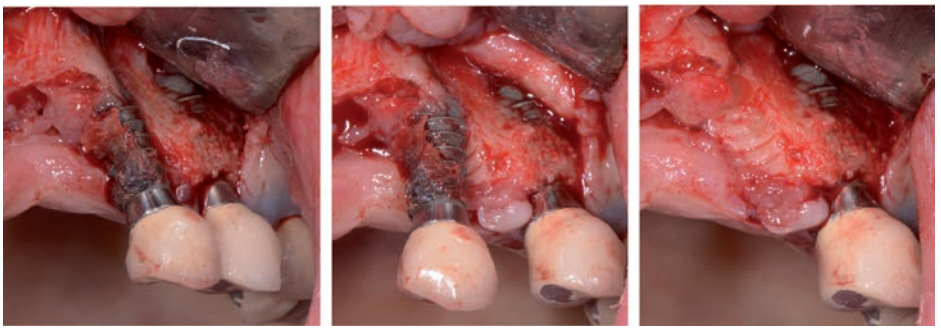

Figure 7.
Severe peri-implantitis: site 24, risk factor is poor fit between abutment and crown; site 25, presence of fenestration. Implant 24 failed: explantation.

Observational studies have further indicated that tissue-level implants may be less prone to peri-implantitis [29, 44, 142]. Nevertheless, the influence of implant or implant abutment connection types on peri-implantitis, irrespective of subcrestal or crestal placement modalities, remains inconclusive [144–147]. Irrespective of the current level of evidence, the biological plausibility of certain implant designs—such as tissue-level implants—in promoting peri-implant tissue stability remains strong. This may justify their preferential use in patients at elevated risk for peri-implant disease despite the lack of conclusive supporting data.

The retention mechanism, that is cement as opposed to screw retention, as a potential risk factor for peri-implantitis, has also recently gained increasing attention [148, 149]. Literature indicates a direct causal association of peri-implantitis with subgingival excess cement resulting from incomplete removal during cementation [150–154]. Contrarily, literature also appears to suggest that both cement and screw-retained restorations may yield comparable alveolar bone stability and implant loss in the absence of excess cement [146, 155]. Clinicians should carefully consider the type and specific design of the retention mechanism depending on various factors, comprising the extension, type, location of the cementation margin and any requirements for potential future maintenance and removal [152].

Lastly, the type of prosthesis and its contouring are critical determinants of oral hygiene accessibility and plaque retention, thereby influencing the risk of developing

peri-implantitis. Full-arch restorations, for example, have been associated with a 16.1-fold increased risk of peri-implantitis compared to single crowns, possibly due to, for example, reduced inter-implant spacing compromising cleanability [131, 156]. Prosthetic designs that promote plaque accumulation and limit access for hygiene with convex emergence profiles and bulky contours in splinted restorations have been shown to elevate the risk of peri-implant disease significantly [126, 157, 158]. In some cases, over-contoured or bulky restorations may result from suboptimal implant positioning, thus representing iatrogenic factors [29, 130]. Clinicians are therefore encouraged to implement appropriate measures in anatomically or prosthetically challenging cases to prevent the delivery of restorations that may increase the risk for future biological complications.

Author details

Alexandre Perez[1*] and Tommaso Lombardi[2]

1 Unit of Oral Surgery and Implantology, Division of Oral and Maxillofacial Surgery, Department of Surgery, University of Geneva and University Hospitals of Geneva, Faculty of Medicine, Geneva, Switzerland

2 Unit of Oral Medicine and Oral Maxillofacial Pathology, Division of Oral and Maxillofacial Surgery, Department of Surgery, Geneva University Hospitals, Faculty of Medicine, University of Geneva, Geneva, Switzerland

*Address all correspondence to: alexandre.perez@hug.ch

References

[1] Buser D, Janner SFM, Wittneben J-G, Brägger U, Ramseier CA, Salvi GE. 10-year survival and success rates of 511 titanium implants with a sandblasted and acid-etched surface: A retrospective study in 303 partially edentulous patients. Clinical Implant Dentistry and Related Research. 2012;**14**:839-851

[2] Francetti L, Cavalli N, Taschieri S, Corbella S. Ten years follow-up retrospective study on implant survival rates and prevalence of peri-implantitis in implant-supported full-arch rehabilitations. Clinical Oral Implants Research. 2019;**30**:252-260

[3] Adell R, Lekholm U, Rockler B. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. International Journal of Oral Surgery. 1981;**10**:387-416

[4] Jung RE, Pjetursson BE, Glauser R, Zembic A, Zwahlen M, Lang NP. A systematic review of the 5-year survival and complication rates of implant-supported single crowns. Clinical Oral Implants Research. 2008;**19**:119-130

[5] Pjetursson BE, Thoma D, Jung R, Zwahlen M, Zembic A. A systematic review of the survival and complication rates of implant-supported fixed dental prostheses (FDP s) after a mean observation period of at least 5 years. Clinical Oral Implants Research. 2012;**23**:22-38

[6] Howe M-S, Keys W, Richards D. Long-term (10-year) dental implant survival: A systematic review and sensitivity meta-analysis. Journal of Dentistry. 2019;**84**:9-21

[7] Hamilton A, Gonzaga L, Amorim K, Wittneben J, Martig L, Morton D, et al. Selection criteria for immediate implant placement and immediate loading for single tooth replacement in the maxillary esthetic zone: A systematic review and meta-analysis. Clinical Oral Implants Research. 2023;**34**:304-348

[8] Buser D, Chappuis V, Belser UC, Chen S. Implant placement post extraction in esthetic single tooth sites: When immediate, when early, when late? Periodontology 2000. 2017;**73**:84-102

[9] Patel R, Ucer C, Wright S, Khan RS. Differences in dental implant survival between immediate vs. delayed placement: A systematic review and meta-analysis. Dentistry Journal. 2023;**11**:218

[10] Bentour E, Papamanoli E, Karoussis IK. The decision between tooth retention or replacement with implants: A continuing dilemma. Dentistry Journal. 2025;**13**:99

[11] Elani HW, Starr JR, Da Silva JD, Gallucci GO. Trends in dental implant use in the U.S., 1999-2016, and projections to 2026. Journal of Dental Research. 2018;**97**:1424-1430

[12] Albrektsson T, Zarb G, Worthington P, Eriksson AR. The long-term efficacy of currently used dental implants: A review and proposed criteria of success. The International Journal of Oral & Maxillofacial Implants. 1986;**1**:11-25

[13] Wittneben J, Wismeijer D, Brägger U, Joda T, Abou-Ayash S. Patient-reported outcome measures focusing on aesthetics of implant- and tooth-supported fixed dental prostheses: A systematic review and meta-analysis. Clinical Oral Implants Research. 2018;**29**:224-240

[14] Berglundh T, Armitage G, Araujo MG, Avila-Ortiz G, Blanco J, Camargo PM, et al. 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 Periodontology. 2018;**89**:S313-S318

[15] Misch CE, Perel ML, Wang H-L, Sammartino G, Galindo-Moreno P, Trisi P, et al. Implant success, survival, and failure: The International Congress of Oral Implantologists (ICOI) Pisa Consensus conference. Implant Dentistry. 2008;**17**:5-15

[16] Papaspyridakos P, Chen C-J, Singh M, Weber H-P, Gallucci GO. Success criteria in implant dentistry: A systematic review. Journal of Dental Research. 2012;**91**:242-248

[17] Pjetursson BE, Heimisdottir K. Dental implants – Are they better than natural teeth? European Journal of Oral Sciences. 2018;**126**:81-87

[18] Chackartchi T, Romanos GE, Sculean A. Soft tissue-related complications and management around dental implants. Periodontology 2000. 2019;**81**:124-138

[19] Al-Sabbagh M, Bhavsar I. Key local and surgical factors related to implant failure. Dental Clinics of North America. 2015;**59**:1-23

[20] Sakka S, Baroudi K, Nassani MZ. Factors associated with early and late failure of dental implants. Journal of Investigative and Clinical Dentistry. 2012;**3**:258-261

[21] Safi Y, Amid R, Zadbin F, Ghazizadeh Ahsaie M, Mortazavi H. The occurrence of dental implant malpositioning and related factors: A cross-sectional cone-beam computed tomography survey. Imaging Science in Dentistry. 2021;**51**:251

[22] Chen ST, Buser D, Sculean A, Belser UC. Complications and treatment errors in implant positioning in the aesthetic zone: Diagnosis and possible solutions. Periodontology 2000. 2023;**92**:220-234

[23] Canullo L, Bignozzi I, Cocchetto R, Cristalli MP, Iannello G. Immediate positioning of a definitive abutment versus repeated abutment replacements in post-extractive implants: 3-year follow-up of a randomised multicentre clinical trial. European Journal of Oral Implantology. 2010;**3**:285-296

[24] Tomasi C, Derks J. Etiology, occurrence, and consequences of implant loss. Periodontology 2000. 2022;**88**:13-35

[25] Ting M, Suzuki JB. Peri-Implantitis. Dentistry Journal. 2024;**12**:251

[26] Scarano A, Khater AGA, Gehrke SA, Serra P, Francesco I, Di Carmine M, et al. Current status of peri-implant diseases: A clinical review for evidence-based decision making. Journal of Periodontal & Implant Science. 2023;**14**:210

[27] Romandini M, Lima C, Pedrinaci I, Araoz A, Costanza Soldini M, Sanz M. Clinical signs, symptoms, perceptions, and impact on quality of life in patients suffering from peri-implant diseases: A university-representative cross-sectional study. Clinical Oral Implants Research. 2021;**32**:100-111

[28] Insua A, Monje A, Wang H, Inglehart M. Patient-Centered perspectives and understanding of Peri-Implantitis. Journal of Periodontology. 2017;**88**:1153-1162

[29] Schwarz F, Derks J, Monje A, Wang H-L. Peri-implantitis. Journal of Periodontology. 2018;**89**(Suppl. 1):S267-S290

[30] Albrekstsson T, Isidor F. Consensus report of session IV. In: Proceedings of the 1st European Workshop on Periodontology. Quintessence Publishing Co Ltd; 1994. pp. 365-369

[31] Lindhe J, Meyle J, On behalf of Group D of the European Workshop on Periodontology. Peri-implant diseases: Consensus report of the sixth European workshop on periodontology. Journal of Clinical Periodontology. 2008;**35**:282-285

[32] Zitzmann NU, Berglundh T. Definition and prevalence of peri-implant diseases. Journal of Clinical Periodontology. 2008;**35**:286-291

[33] Lang NP, Berglundh T. On behalf of working group 4 of the seventh European workshop on periodontology. Periimplant diseases: Where are we now? – Consensus of the seventh European workshop on periodontology. Journal of Clinical Periodontology. 2011;**38**:178-181

[34] Renvert S, Persson GR, Pirih FQ, Camargo PM. Peri-implant health, peri-implant mucositis, and peri-implantitis: Case definitions and diagnostic considerations: Diagnostic criteria of peri-implant health and diseases. Journal of Periodontology. 2018;**89**:S304-S312

[35] Zheng Z, Ao X, Xie P, Jiang F, Chen W. The biological width around implant. Journal of Prosthodontic Research. 2021;**65**:11-18

[36] Smith DE, Zarb GA. Criteria for success of osseointegrated endosseous implants. The Journal of Prosthetic Dentistry. 1989;**62**:567-572

[37] Araujo MG, Lindhe J. Peri-implant health. Journal of Periodontology. 2018;**89**:S249-S256

[38] Wehner C, Bertl K, Durstberger G, Arnhart C, Rausch-Fan X, Stavropoulos A. Characteristics and frequency distribution of bone defect configurations in peri-implantitis lesions—A series of 193 cases. Clinical Implant Dentistry and Related Research. 2021;**23**:178-188

[39] Monje A, Pons R, Insua A, Nart J, Wang H, Schwarz F. Morphology and severity of peri-implantitis bone defects. Clinical Implant Dentistry and Related Research. Aug 2019;**21**(4):635-643

[40] Schwarz F, Sahm N, Schwarz K, Becker J. Impact of defect configuration on the clinical outcome following surgical regenerative therapy of peri-implantitis. Journal of Clinical Periodontology. 2010;**37**:449-455

[41] Papathanasiou E, Finkelman M, Hanley J, Parashis AO. Prevalence, etiology and treatment of peri-implant mucositis and peri-implantitis: A survey of periodontists in the United States. Journal of Periodontology. 2016;**87**:493-501

[42] Papapanou PN, Sanz M, Buduneli N, Dietrich T, Feres M, Fine DH, et al. Periodontitis: Consensus report of workgroup 2 of the 2017 world workshop on the classification of periodontal and peri-implant diseases and conditions. Journal of Periodontology. Jun 2018;**89**(Suppl. 1):S173-S182. DOI: 10.1002/JPER.17-0721

[43] Caton JG, Armitage G, Berglundh T, Chapple ILC, Jepsen S, Kornman KS, et al. A new classification scheme for periodontal and peri-implant diseases and conditions - Introduction and key changes from the 1999 classification. Journal of Clinical Periodontology. 2018;**45**:S1-S8

[44] Derks J, Tomasi C. Peri-implant health and disease. A systematic review of current epidemiology. Journal of Clinical Periodontology. 2015;**42**:S158-S171

[45] Lee C-T, Huang Y-W, Zhu L, Weltman R. Prevalences of peri-implantitis and peri-implant mucositis: Systematic review and meta-analysis. Journal of Dentistry. 2017;**62**:1-12

[46] Tomasi C, Derks J. Clinical research of peri-implant diseases - quality of reporting, case definitions and methods to study incidence, prevalence and risk factors of peri-implant diseases. Journal of Clinical Periodontology. 2012;**39**:207-223

[47] Mombelli A, Müller N, Cionca N. The epidemiology of peri-implantitis. Clinical Oral Implants Research. 2012;**23**:67-76

[48] Roos-Jansåker A, Lindahl C, Renvert H, Renvert S. Nine- to fourteen-year follow-up of implant treatment. Part II: Presence of peri-implant lesions. Journal of Clinical Periodontology. 2006;**33**:290-295

[49] Koldsland OC, Scheie AA, Aass AM. Prevalence of Peri-Implantitis related to severity of the disease with different degrees of bone loss. Journal of Periodontology. 2010;**81**:231-238

[50] Derks J, Schaller D, Håkansson J, Wennström JL, Tomasi C, Berglundh T. Effectiveness of implant therapy analyzed in a Swedish population: Prevalence of Peri-implantitis. Journal of Dental Research. 2016;**95**:43-49

[51] Roccuzzo M, Layton DM, Roccuzzo A, Heitz-Mayfield LJ. Clinical outcomes of peri-implantitis treatment and supportive care: A systematic review. Clinical Oral Implants Research. 2018;**29**:331-350

[52] Kordbacheh Changi K, Finkelstein J, Papapanou PN. Peri-implantitis prevalence, incidence rate, and risk factors: A study of electronic health records at a U.S. dental school. Clinical Oral Implants Research. 2019;**30**:306-314

[53] Salvi GE, Aglietta M, Eick S, Sculean A, Lang NP, Ramseier CA. Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. Clinical Oral Implants Research. 2012;**23**:182-190

[54] Ferreira SD, Silva GLM, Cortelli JR, Costa JE, Costa FO. Prevalence and risk variables for peri-implant disease in Brazilian subjects. Journal of Clinical Periodontology. 2006;**33**:929-935

[55] Zitzmann NU, Berglundh T, Marinello CP, Lindhe J. Experimental peri-implant mucositis in man. Journal of Clinical Periodontology. 2001;**28**:517-523

[56] Pontoriero R, Tonelli MP, Carnevale G, Mombelli A, Nyman SR, Lang NP. Experimentally induced peri-implant mucositis. A clinical study in humans. Clinical Oral Implants Research. 1994;**5**:254-259

[57] Aguirre-Zorzano LA, Estefanía-Fresco R, Telletxea O, Bravo M. Prevalence of peri-implant inflammatory disease in patients with a history of periodontal disease who receive supportive periodontal therapy. Clinical Oral Implants Research. 2015;**26**:1338-1344

[58] Berglundh T, Wennström JL, Lindhe J. Long-term outcome of surgical treatment of peri-implantitis. A 2-11-year retrospective study. Clinical Oral Implants Research. 2018;**29**:404-410

[59] Schwarz F, Sculean A, Engebretson SP, Becker J, Sager M.

Animal models for peri-implant mucositis and peri-implantitis. Periodontology 2000. 2015;(68):168-181

[60] Subramani K, Jung RE, Molenberg A, Hammerle CHF. Biofilm on dental implants: A review of the literature. The International Journal of Oral & Maxillofacial Implants. 2009;**24**:616-626

[61] Atsuta I, Ayukawa Y, Kondo R, Oshiro W, Matsuura Y, Furuhashi A, et al. Soft tissue sealing around dental implants based on histological interpretation. Journal of Prosthodontic Research. 2016;**60**:3-11

[62] Schwarz F, Mihatovic I, Golubovic V, Eick S, Iglhaut T, Becker J. Experimental peri-implant mucositis at different implant surfaces. Journal of Clinical Periodontology. 2014;**41**:513-520

[63] Salvi GE, Stähli A, Imber J, Sculean A, Roccuzzo A. Physiopathology of peri-implant diseases. Clinical Implant Dentistry and Related Research. 2023;**25**:629-639

[64] Berglundh T, Lindhe J, Jonsson K, Ericsson I. The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. Journal of Clinical Periodontology. 1994;**21**:189-193

[65] Butera A, Pascadopoli M, Pellegrini M, Gallo S, Zampetti P, Scribante A. Oral microbiota in patients with Peri-implant disease: A narrative review. Applied Sciences. 2022;**12**:3250

[66] Hall-Stoodley L, Costerton JW, Stoodley P. Bacterial biofilms: From the natural environment to infectious diseases. Nature Reviews. Microbiology. 2004;**2**:95-108

[67] Kashani H, Hilon J, Rasoul MH, Friberg B. Influence of a single preoperative dose of antibiotics on the early implant failure rate. A randomized clinical trial. Clinical Implant Dentistry and Related Research. 2019;**21**:278-283

[68] Shirazi S, Ravindran S, Cooper LF. Topography-mediated immunomodulation in osseointegration; ally or enemy. Biomaterials. 2022;**291**:121903

[69] Albrektsson T, Becker W, Coli P, Jemt T, Mölne J, Sennerby L. Bone loss around oral and orthopedic implants: An immunologically based condition. Clinical Implant Dentistry and Related Research. 2019;**21**:786-795

[70] Baseri M, Radmand F, Hamedi R, Yousefi M, Kafil HS. Immunological aspects of dental implant rejection. BioMed Research International. 2020;**2020**:7279509

[71] Ratner BD, Bryant SJ. Biomaterials: Where we have been and where we are going. Annual Review of Biomedical Engineering. 2004;**6**:41-75

[72] Murray PJ. Macrophage polarization. Annual Review of Physiology. 2017;**79**:541-566

[73] Pitchai M, Ipe D, Tadakamadla S, Hamlet S. Titanium implant surface effects on adherent macrophage phenotype: A systematic review. Materials. 2022;**15**:7314

[74] Fretwurst T, Buzanich G, Nahles S, Woelber JP, Riesemeier H, Nelson K. Metal elements in tissue with dental peri-implantitis: A pilot study. Clinical Oral Implants Research. 2016;**27**:1178-1186

[75] Jacobi-Gresser E, Huesker K, Schütt S. Genetic and immunological markers predict titanium implant failure: A retrospective study. International Journal of Oral and Maxillofacial Surgery. 2013;**42**:537-543

[76] Jepsen S, Berglundh T, Genco R, Aass AM, Demirel K, Derks J, et al. Primary prevention of peri-implantitis: Managing peri-implant mucositis. Journal of Clinical Periodontology. 2015;**42**(Suppl. 16):S152-S157

[77] Karoussis IK, Muller S, Salvi GE, Heitz-mayfield LJA, Bragger U, Lang NP. Association between periodontal and peri-implant conditions: A 10-year prospective study. Clinical Oral Implants Research. 2004;**15**:1-7

[78] Roccuzzo M, De Angelis N, Bonino L, Aglietta M. 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. 2010;**21**:490-496

[79] Chrcanovic BR, Albrektsson T, Wennerberg A. Periodontally compromised vs. periodontally healthy patients and dental implants: A systematic review and meta-analysis. Journal of Dentistry. 2014;**42**:1509-1527

[80] Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ. Prevalence of periodontitis in adults in the United States: 2009 and 2010. Journal of Dental Research. 2012;**91**:914-920

[81] Eke PI, Borgnakke WS, Genco RJ. Recent epidemiologic trends in periodontitis in the USA. Periodontology 2000. 2000;**2020**(82):257-267

[82] Nazir M, Al-Ansari A, Al-Khalifa K, Alhareky M, Gaffar B, Almas K. Global prevalence of periodontal disease and lack of its surveillance. The Scientific World Journal. 2020;**2020**:1-8

[83] Kaldahl WB, Kalkwarf KL, Patil KD, Dyer JK, Bates RE. Evaluation of four modalities of periodontal therapy. Mean probing depth, probing attachment level and recession changes. Journal of Periodontology. 1988;**59**:783-793

[84] Cho-Yan Lee J, Mattheos N, Nixon KC, Ivanovski S. Residual periodontal pockets are a risk indicator for peri-implantitis in patients treated for periodontitis. Clinical Oral Implants Research. 2012;**23**:325-333

[85] Chrcanovic BR, Martins MD, Wennerberg A. Immediate placement of implants into infected sites: A systematic review: Immediate implants in infected sites. Clinical Implant Dentistry and Related Research. 2015;**17**:e1-e16

[86] Chrcanovic BR, Albrektsson T, Wennerberg A. Smoking and dental implants: A systematic review and meta-analysis. Journal of Dentistry. 2015;**43**:487-498

[87] Guney Z, Altingoz SM, Has H, Serdar MA, Kurgan S. The impact of electronic cigarettes on peri-implant health: A systematic review and meta-analysis. Journal of Dentistry. 2024;**143**:104883

[88] Heitz-Mayfield LJA, Huynh-Ba G. History of treated periodontitis and smoking as risks for implant therapy. The International Journal of Oral & Maxillofacial Implants. 2009;**24**(Suppl.):39-68

[89] Ma L, Zheng LW, Sham MH, Cheung LK. Uncoupled angiogenesis and osteogenesis in nicotine-compromised bone healing. Journal of Bone and Mineral Research. 2010;**25**:1305-1313

[90] Ma L, Zwahlen RA, Zheng LW, Sham MH. Influence of nicotine on the biological activity of rabbit osteoblasts: Influence of nicotine on gene expression in osteoblasts. Clinical Oral Implants Research. 2011;**22**:338-342

[91] Rinke S, Ohl S, Ziebolz D, Lange K, Eickholz P. Prevalence of periimplant disease in partially edentulous patients: A practice-based cross-sectional study: Cross-sectional study on the prevalence of periimplant disease. Clinical Oral Implants Research. 2011;**22**:826-833

[92] Roos-Jansåker AM, Lindahl C, Renvert H, Renvert S. Nine-to fourteen-year follow-up of implant treatment. Part I: Implant loss and associations to various factors. Journal of Clinical Periodontology. 2006;**33**:283-289

[93] Romandini M, Lima C, Pedrinaci I, Araoz A, Soldini MC, Sanz M. Prevalence and risk/protective indicators of peri-implant diseases: A university-representative cross-sectional study. Clinical Oral Implants Research. 2021;**32**:112-122

[94] Chrcanovic BR, Albrektsson T, Wennerberg A. Diabetes and Oral implant failure: A systematic review. Journal of Dental Research. 2014;**93**:859-867

[95] Daubert DM, Weinstein BF, Bordin S, Leroux BG, Flemmig TF. Prevalence and predictive factors for Peri-implant disease and implant failure: A cross-sectional analysis. Journal of Periodontology. 2015;**86**:337-347

[96] Moy PK, Medina D, Shetty V. Dental implant failure rates and associated risk factors. The International Journal of Oral & Maxillofacial Implants. 2005;**20**:569-577

[97] Monje A, Catena A, Borgnakke WS. Association between diabetes mellitus/hyperglycaemia and peri-implant diseases: Systematic review and meta-analysis. Journal of Clinical Periodontology. 2017;**44**:636-648

[98] Aguilar-Salvatierra A, Calvo-Guirado JL, González-Jaranay M, Moreu G, Delgado-Ruiz RA, Gómez-Moreno G. Peri-implant evaluation of immediately loaded implants placed in esthetic zone in patients with diabetes mellitus type 2: A two-year study. Clinical Oral Implants Research. 2016;**27**:156-161

[99] Zhang Z, Ji C, Wang D, Wang M, Song D, Xu X, et al. The burden of diabetes on the soft tissue seal surrounding the dental implants. Frontiers in Physiology. 2023;**14**:1136973

[100] Preshaw PM, Alba AL, Herrera D, Jepsen S, Konstantinidis A, Makrilakis K, et al. Periodontitis and diabetes: A two-way relationship. Diabetologia. 2012;**55**:21-31

[101] Tawil G, Od D, Younan R. Conventional and advanced implant treatment in the type II diabetic patient: Surgical protocol and long-term clinical results. The International Journal of Oral & Maxillofacial Implants. 2007;**23**:744-752

[102] Eskow CC, Oates TW. Dental implant survival and complication rate over 2 years for individuals with poorly controlled type 2 diabetes mellitus. Clinical Implant Dentistry and Related Research. 2017;**19**:423-431

[103] Javed F, Romanos GE. Impact of diabetes mellitus and Glycemic control on the Osseointegration of dental implants: A systematic literature review. Journal of Periodontology. 2009;**80**:1719-1730

[104] Naujokat H, Kunzendorf B, Wiltfang J. Dental implants and diabetes mellitus—A systematic review. International Journal of Implant Dentistry. 2016;**2**:5

[105] Sannino G, Montemezzi P, Pantaleo G, Agliardi E. Dental implants

survival rate in controlled type I diabetic patients: A prospective longitudinal study with a 2-year follow-up. Journal of Biological Regulators and Homeostatic Agents. 2020;**34**:37-45

[106] Alrabiah M, Al-Aali KA, Al-Sowygh ZH, Binmahfooz AM, Mokeem SA, Abduljabbar T. Association of advanced glycation end products with peri-implant inflammation in prediabetes and type 2 diabetes mellitus patients. Clinical Implant Dentistry and Related Research. 2018;**20**:535-540

[107] Mokeem S, Alfadda SA, Al-Shibani N, Alrabiah M, Al-Hamdan RS, Vohra F, et al. Clinical and radiographic peri-implant variables around short dental implants in type 2 diabetic, prediabetic, and non-diabetic patients. Clinical Implant Dentistry and Related Research. 2019;**21**:60-65

[108] Dreyer H, Grischke J, Tiede C, Eberhard J, Schweitzer A, Toikkanen SE, et al. Epidemiology and risk factors of peri-implantitis: A systematic review. Journal of Periodontal Research. 2018;**53**:657-681

[109] Máximo MB, De Mendonça AC, Alves JF, Cortelli SC, Peruzzo DC, Duarte PM. Peri-implant diseases may Be associated with increased time loading and generalized periodontal bone loss: Preliminary results. Journal of Oral Implantology. 2008;**34**:268-273

[110] Dvorak G, Arnhart C, Heuberer S, Huber CD, Watzek G, Gruber R. Peri-implantitis and late implant failures in postmenopausal women: A cross-sectional study: Peri-implantitis in postmenopausal women. Journal of Clinical Periodontology. 2011;**38**:950-955

[111] Renvert S, Aghazadeh A, Hallström H, Persson GR. Factors related to peri-implantitis - a retrospective study. Clinical Oral Implants Research. 2014;**25**:522-529

[112] De Medeiros FCFL, Kudo GAH, Leme BG, Saraiva PP, Verri FR, Honório HM, et al. Dental implants in patients with osteoporosis: A systematic review with meta-analysis. International Journal of Oral and Maxillofacial Surgery. 2018;**47**:480-491

[113] Lafuente-Ibáñez De Mendoza I, Setien-Olarra A, García-De La Fuente AM, Aguirre-Urizar JM, Marichalar-Mendia X. Role of proinflammatory mutations in peri-implantitis: Systematic review and meta-analysis. International Journal of Implant Dentistry. 2022;**8**:2

[114] Wennström JL, Derks J. Is there a need for keratinized mucosa around implants to maintain health and tissue stability? Clinical Oral Implants Research. 2012;**23**:136-146

[115] Gobbato L, Avila-Ortiz G, Sohrabi K, Wang C-W, Karimbux N. The effect of keratinized mucosa width on peri-implant health: A systematic review. The International Journal of Oral & Maxillofacial Implants. 2013;**28**:1536-1545

[116] Lin G, Chan H, Wang H. The significance of keratinized mucosa on implant health: A systematic review. Journal of Periodontology. 2013;**84**:1755-1767

[117] Roccuzzo M, Grasso G, Dalmasso P. Keratinized mucosa around implants in partially edentulous posterior mandible: 10-year results of a prospective comparative study. Clinical Oral Implants Research. 2016;**27**:491-496

[118] Souza AB, Tormena M, Matarazzo F, Araújo MG. The influence of peri-implant keratinized mucosa on

brushing discomfort and peri-implant tissue health. Clinical Oral Implants Research. 2016;**27**:650-655

[119] Fons-Badal C, Agustín-Panadero R, Labaig-Rueda C, Solá-Ruiz M, García-Selva M, Fons FA. The influence of keratinized mucosa on the nonsurgical therapeutic treatment of peri-implantitis. Journal of Clinical Medicine. 2022;**11**:4118

[120] Mahardawi B, Jiaranuchart S, Damrongsirirat N, Arunjaroensuk S, Mattheos N, Somboonsavatdee A, et al. The lack of keratinized mucosa as a risk factor for peri-implantitis: A systematic review and meta-analysis. Scientific Reports. 2023;**13**:3778

[121] Schwarz F, Becker K, Sahm N, Horstkemper T, Rousi K, Becker J. The prevalence of peri-implant diseases for two-piece implants with an internal tube-in-tube connection: A cross-sectional analysis of 512 implants. Clinical Oral Implants Research. 2017;**28**:24-28

[122] Rokn A, Aslroosta H, Akbari S, Najafi H, Zayeri F, Hashemi K. Prevalence of peri-implantitis in patients not participating in well-designed supportive periodontal treatments: A cross-sectional study. Clinical Oral Implants Research. 2017;**28**:314-319

[123] Canullo L, Peñarrocha-Oltra D, Covani U, Botticelli D, Serino G, Penarrocha M. Clinical and microbiological findings in patients with peri-implantitis: A cross-sectional study. Clinical Oral Implants Research. 2016;**27**:376-382

[124] De Araújo NM, Mano Azul A, Rocha E, Maló P. Risk factors of peri-implant pathology. European Journal of Oral Sciences. 2015;**123**:131-139

[125] Heitz-Mayfield LJA, Heitz F, Lang NP. Implant disease risk assessment IDRA–a tool for preventing peri-implant disease. Clinical Oral Implants Research. 2020;**31**:397-403

[126] Serino G, Ström C. Peri-implantitis in partially edentulous patients: Association with inadequate plaque control. Clinical Oral Implants Research. 2009;**20**:169-174

[127] Tormena M, Matarazzo F, De Oliveira BM, Pereira O, Previdelli I, Araújo MG. The effect of inadequate access to peri-implant hygiene on marginal bone level. A 4-year cohort prospective study. Clinical Oral Implants Research. 2020;**31**:836-845

[128] Costa FO, Takenaka-Martinez S, Cota LOM, Ferreira SD, Silva GLM, Costa JE. Peri-implant disease in subjects with and without preventive maintenance: A 5-year follow-up. Journal of Clinical Periodontology. 2012;**39**:173-181

[129] Monje A, Wang H-L, Nart J. Association of Preventive Maintenance Therapy Compliance and Peri-Implant Diseases: A cross-sectional study. Journal of Periodontology. 2017;**88**:1030-1041

[130] Cortellini S, Favril C, De Nutte M, Teughels W, Quirynen M. Patient compliance as a risk factor for the outcome of implant treatment. Periodontology 2000. 2019;**81**:209-225

[131] Dalago HR, Schuldt Filho G, Rodrigues MAP, Renvert S, Bianchini MA. Risk indicators for Peri-implantitis. A cross-sectional study with 916 implants. Clinical Oral Implants Research. 2017;**28**:144-150

[132] Misch CE, Suzuki JB, Misch-Dietsh FM, Bidez MW. A positive correlation between occlusal trauma and Peri-implant bone loss: Literature support. Implant Dentistry. 2005;**14**:108-116

[133] Häggman-Henrikson B, Ali D, Aljamal M, Chrcanovic BR. Bruxism and dental implants: A systematic review and meta-analysis. Journal of Oral Rehabilitation. 2024;**51**:202-217

[134] Ionfrida JA, Stiller HL, Kämmerer PW, Walter C. Dental implant failure risk in patients with bruxism—A systematic review and meta-analysis of the literature. Dentistry Journal. 2024;**13**:11

[135] Isidor F. Influence of forces on peri-implant bone. Clinical Oral Implants Research. 2006;**17**:8-18

[136] Hermann JS, Schoolfield JD, Schenk RK, Buser D, Cochran DL. Influence of the size of the microgap on Crestal bone changes around titanium implants. A Histometric evaluation of unloaded non-submerged implants in the canine mandible. Journal of Periodontology. 2001;**72**:1372-1383

[137] Buser D, Mericske-Stern R, Dula K, Lang NP. Clinical experience with one-stage, non-submerged dental implants. Advances in Dental Research. 1999;**13**:153-161

[138] Cochran DL. The scientific basis for and clinical experiences with Straumann implants including the ITI dental implant system: A consensus report. Clinical Oral Implants Research. 2000;**11**(Suppl. 1):33-58

[139] Kim S, Jung U-W, Cho K-S, Lee J-S. Retrospective radiographic observational study of 1692 Straumann tissue-level dental implants over 10 years: I. Implant survival and loss pattern. Clinical Implant Dentistry and Related Research. 2018;**20**:860-866

[140] Cosola S, Marconcini S, Boccuzzi M, Menchini Fabris GB, Covani U, Peñarrocha-Diago M, et al. Radiological outcomes of bone-level and tissue-level dental implants: Systematic review. International Journal of Environmental Research and Public Health. 2020;**17**:6920

[141] Atieh MA, Ibrahim HM, Atieh AH. Platform switching for marginal bone preservation around dental implants: A systematic review and meta-analysis. Journal of Periodontology. 2010;**81**:1350-1366

[142] Sasada Y, Cochran D. Implant-abutment connections: A review of biologic consequences and Peri-implantitis implications. The International Journal of Oral & Maxillofacial Implants. 2017;**32**:1296-1307

[143] Elkattan A, Torres-Velázquez N, Blázquez-Hinarejos M, Egido-Moreno S, Ayuso-Montero R, Roselló-Llabrés X, et al. Marginal bone loss around bone-level and tissue-level implants: A systematic review and meta-analysis. Applied Sciences. 2025;**15**:2286

[144] Meloni SM, Jovanovic SA, Lolli FM, Pisano M, Riu GD, Riu ND, et al. Platform switching vs regular platform implants: Nine-month post-loading results from a randomised controlled trial. European Journal of Oral Implantology;**7**:257-265

[145] Chrcanovic BR, Albrektsson T, Wennerberg A. Platform switch and dental implants: A meta-analysis. Journal of Dentistry. 2015;**43**:629-646

[146] Mattheos N, Janda M, Acharya A, Pekarski S, Larsson C. Impact of design elements of the implant supracrestal complex (ISC) on the risk of peri-implant mucositis and peri-implantitis: A critical review. Clinical Oral Implants Research. 2021;**32**:181-202

[147] Paolantoni G, Tatullo M, Miniello A, Sammartino G, Marenzi G. Influence of crestal and sub-crestal implant position on development of peri-implant diseases: A 5-year retrospective analysis. Clinical Oral Investigations. 2023;**28**:16

[148] Wittneben J, Joda T, Weber H, Brägger U. Screw retained vs. cement retained implant-supported fixed dental prosthesis. Periodontology 2000. 2017;**73**:141-151

[149] Wittneben J-G, Millen C, Brägger U. Clinical performance of screw-versus cement-retained fixed implant-supported reconstructions—A systematic review. The International Journal of Oral & Maxillofacial Implants. 2014;**29**:84-98

[150] Linkevicius T, Puisys A, Vindasiute E, Linkeviciene L, Apse P. Does residual cement around implant-supported restorations cause peri-implant disease? A retrospective case analysis. Clinical Oral Implants Research. 2013;**24**:1179-1184

[151] Gehrke P, Bleuel K, Fischer C, Sader R. Influence of margin location and luting material on the amount of undetected cement excess on CAD/CAM implant abutments and cement-retained zirconia crowns: An in-vitro study. BMC Oral Health. 2019;**19**:111

[152] Linkevicius T, Vindasiute E, Puisys A, Linkeviciene L, Maslova N, Puriene A. The influence of the cementation margin position on the amount of undetected cement. A prospective clinical study. Clinical Oral Implants Research. 2013;**24**:71-76

[153] Wilson TG Jr. The positive relationship between excess cement and peri-implant disease: A prospective clinical endoscopic study. Journal of Periodontology. 2009;**80**:1388-1392

[154] Agar JR, Cameron SM, Hughbanks JC, Parker MH. Cement removal from restorations luted to titanium abutments with simulated subgingival margins. The Journal of Prosthetic Dentistry. 1997;**78**:43-47

[155] Lemos CAA, De Souza Batista VE, Almeida DADF, Santiago Júnior JF, Verri FR, Pellizzer EP. Evaluation of cement-retained versus screw-retained implant-supported restorations for marginal bone loss. The Journal of Prosthetic Dentistry. 2016;**115**:419-427

[156] Schuldt Filho G, Dalago HR, Oliveira de Souza JG, Stanley K, Jovanovic S, Bianchini MA. Prevalence of peri-implantitis in patients with implant-supported fixed prostheses. Quintessence International. 2014;**45**:861-868

[157] Katafuchi M, Weinstein BF, Leroux BG, Chen Y, Daubert DM. Restoration contour is a risk indicator for peri-implantitis: A cross-sectional radiographic analysis. Journal of Clinical Periodontology. 2018;**45**:225-232

[158] Yi Y, Koo K, Schwarz F, Ben Amara H, Heo S. Association of prosthetic features and peri-implantitis: A cross-sectional study. Journal of Clinical Periodontology. 2020;**47**:392-403