Received: 25 July 2016 | Revised: 1 August 2017 | Accepted: 8 September 2017

DOI: 10.1111/jcpe.12953

2017 WORLD WORKSHOP

WILEY Journal of Clinical Periodontology

Peri-implant mucositis

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The proceedings of the workshop were jointly and simultaneously published in the *Journal of Periodontology* and *Journal of Clinical Periodontology*.

Abstract

Objectives: This narrative review was prepared for the 2017 World Workshop of the American Academy of Periodontology and European Federation of Periodontology to address key questions related to the clinical condition of peri-implant mucositis, including: 1) the definition of peri-implant mucositis, 2) conversion of peri-implant health to the biofilm-induced peri-implant mucositis lesion, 3) reversibility of peri-implant mucositis, 4) the long-standing peri-implant mucositis lesion, 5) similarities and differences between peri-implant mucositis at implants and gingivitis at teeth, and 6) risk indicators/factors for peri-implant mucositis.

Methods: A literature search of MEDLINE (PubMed) and The Cochrane Library up to and including July 31, 2016, was carried out using the search strategy (peri-implant[All Fields] AND ("mucositis"[MeSH Terms] OR "mucositis"[All Fields])) OR (periimplant[All Fields] AND mucosits[All Fields]). Prospective, retrospective, and cross-sectional studies and review papers that focused on risk factors/indicators for peri-implant mucositis as well as experimental peri-implant mucositis studies in animals and humans were included.

Findings: Peri-implant mucositis is an inflammatory lesion of the soft tissues surrounding an endosseous implant in the absence of loss of supporting bone or continuing marginal bone loss. A cause-and-effect relationship between experimental accumulation of bacterial biofilms around titanium dental implants and the development of an inflammatory response has been demonstrated. The experimental peri-implant mucositis lesion is characterized by an inflammatory cell infiltrate present within the connective tissue lateral to the barrier epithelium. In long-standing peri-implant mucositis, the inflammatory cell infiltrate is larger in size than in the early (3-week) experimental peri-implant mucositis lesion. Biofilm-induced peri-implant mucositis is reversible at the host biomarker level once biofilm control is reinstituted. Reversal of the clinical signs of inflammation may take longer than 3 weeks. Factors identified as risk indicators for peri-implant mucositis include biofilm accumulation, smoking, and radiation. Further evidence is required for potential risk factors, including diabetes, lack of keratinized mucosa, and presence of excess luting cement.

Conclusions: Peri-implant mucositis is caused by biofilm accumulation which disrupts the host–microbe homeostasis at the implant–mucosa interface, resulting in an inflammatory lesion. Peri-implant mucositis is a reversible condition at the host

biomarker level. Therefore, the clinical implication is that optimal biofilm removal is a prerequisite for the prevention and management of peri-implant mucositis. An understanding of peri-implant mucositis is important because it is considered a precursor for peri-implantitis.

KEYWORDS
peri-implant disease, peri-implant mucositis, peri-implantitis, risk factor, risk indicator

Peri-implant diseases, including peri-implant mucositis and peri-implantitis, were first defined and described at the First European Workshop on Periodontology in Ittingen in 1993.[1] Following this, there have been numerous workshops addressing the definition, prevalence, and treatment of these diseases.[2,3] Peri-implant mucositis is considered to be the precursor of peri-implantitis. The objective of this narrative review was to address key questions related to peri-implant mucositis, including: 1) the definition of peri-implant mucositis, 2) conversion of peri-implant health to the biofilm-induced peri-implant mucositis lesion, 3) reversibility of peri-implant mucosits, 4) the long-standing peri-implant mucositis lesion, 5) similarities and differences between peri-implant mucositis at implants and gingivitis at teeth, and 6) risk indicators/factors for peri-implant mucositis.

MATERIALS AND METHODS

A literature search of MEDLINE (PubMed) and The Cochrane Library up to and including July 31, 2016, was carried out using the search strategy (peri-implant[All Fields] AND ("mucositis"[MeSH Terms] OR "mucositis"[All Fields])) OR (periimplant[All Fields] AND mucositis[All Fields]), resulting in 224 papers. Prospective, retrospective, and cross-sectional studies and review papers focused on risk factors/indicators for peri-implant mucositis as well as experimental peri-implant mucositis studies in animals and humans were included. Following discussion, the current authors agreed on the studies to be included in this narrative review based on their relevance to the questions, outlined above, addressing the topic of peri-implant mucositis.

TABLE 1 Similarities and differences between biofilm-induced gingivitis and peri-implant mucositis

	Gingivitis	Peri-implant mucositis
Definition	Gingival inflammation without periodontal attachment loss	Peri-implant mucosal inflammation in absence of continuous marginal peri-implant bone loss
Clinical signs	Redness, swelling, and bleeding on gentle probing	Redness, swelling, bleeding on gentle probing, and suppuration
Experimental inflammation in humans	Increase in bleeding sites during experimental gingivitis[12,13]	Experimental peri-implant mucositis leads to greater increase in bleeding sites compared with experimental gingivitis.[12,13]
Reversibility in humans	Experimental gingivitis clinically reversible after reinstitution of biofilm control[14] Resolution of host biomarkers in gingival crevicular fluid following 21 days of reinstituted biofilm control[12,13]	Experimental peri-implant mucositis may take longer than 3 weeks for clinical reversibility.[12,13] Resolution of host biomarkers in peri-implant crevicular fluid following 21 days of reinstituted biofilm control[12,13]
Analysis of human biopsies	Experimental biofilm accumulation results in increased proportions of inflammatory cells in connective tissue[11]	Increased proportions of inflammatory cells in connective tissue similar to those found in experimental gingivitis[11]
Short- vs. long-standing inflammation	3-week and 3-month experimental biofilm accumulation results in similar intensity of inflammatory responses in gingiva of dogs[17,72]	3-month experimental biofilm accumulation in dogs results in a more pronounced inflammatory response in peri-implant mucosa compared with inflammatory response in the gingiva[17] Inflammatory lesions from long-standing mucositis in humans[20] considerably larger compared with those of short-term (3-week) experimental mucositis lesions[11]
Variability in humans	High and low responders to experimental biofilm accumulation[73]	High and low responders to experimental biofilm accumulation not yet identified

DEFINITION OF PERI-IMPLANT MUCOSITIS

Peri-implant mucositis has been defined in previous workshops as an inflammatory lesion of the mucosa surrounding an endosseous implant without loss of supporting peri-implant bone.[1–3] The important criteria for the definition of peri-implant mucositis are inflammation in the peri-implant mucosa and the absence of continuing marginal peri-implant bone loss. The clinical sign of inflammation is bleeding on probing, while additional signs may include erythema, swelling, and suppuration (Table 1). The clinical case definition of peri-implant mucositis has been addressed in another review prepared for this workshop.

CONVERSION FROM HEALTHY PERI-IMPLANT MUCOSA TO PERI-IMPLANT MUCOSITIS

Healthy peri-implant mucosa is characterized by the presence of an oral epithelium extending into a non-keratinized barrier epithelium with basal lamina and hemidesmosomes facing the implant or abutment surface.[4] In the connective tissue adjacent to the epithelial barrier, inflammatory cell infiltrates representing the host's defense against the bacterial challenge are present. In healthy peri-implant mucosal conditions, the barrier epithelium and the presence of scattered inflammatory cells constitute the soft tissue seal separating the peri-implant attachment from the oral cavity.[5–9]

Peri-implant mucositis develops from healthy peri-implant mucosa following accumulation of bacterial biofilms around osseointegrated dental implants. A cause-and-effect relationship between experimental accumulation of bacterial biofilms around titanium dental implants and the development of an inflammatory response (i.e., experimental peri-implant mucositis) has been demonstrated in humans.[10–13]

In an early study by Pontoriero et al.,[10] twenty partially edentulous patients received dental implants following successful completion of periodontal therapy. After 6 months of supervised oral hygiene, the peri-implant mucosa was characterized by the absence of obvious signs of clinical inflammation. Following this period, the patients were asked to abolish oral hygiene practices for 3 weeks. At the end of this period, optimal biofilm control was reinstituted. At all examinations the following clinical parameters were assessed around the implants: plaque index (PI), gingival index (GI), sulcus bleeding index (SBI), probing depths (PD), and marginal recession (REC). The 3-week period of abolished oral hygiene practices revealed the development of visible signs of mucosal inflammation, such as swelling, redness, and bleeding. This cause-and-effect relationship between the accumulation of bacterial biofilms and the development of peri-implant mucositis is consistent with the results obtained in the experimental gingivitis model by Löe et al.[14] In another study by Zitzmann et al.[11] involving 12 partially edentulous patients the inflammatory. The inflammatory response to the experimental bacterial challenge was characterized by the enumeration of the proportions of T- and B-cells in peri-implant tissues. Biopsies harvested around implants in a clinically healthy situation and after 21 days of experimental biofilm accumulation indicated that the connective tissue surrounding the implants displayed an increased volume of T- and B-lymphocytes as a consequence of abolished oral hygiene practices.[11] It was also noted that the size of the inflammatory cell infiltrate and the number of several immune cell populations was not significantly different when comparing biopsies from gingiva at teeth and biopsies from peri-implant mucosa.[11]

Outcomes of a comparative study in humans by Salvi et al.[12] indicated that 3 weeks of experimental biofilm accumulation resulted in a higher proportion of bleeding sites in the peri-implant mucosa when compared with that in the gingiva. In that study, the PI at tooth sites was significantly elevated when compared with that at implant sites after 3 weeks of abolished oral hygiene.[12] However, the increase of the GI at tooth sites was significantly lower compared with that at implant sites, indicating that a comparable bacterial challenge yielded a more severe inflammatory response at implant sites.

A recent study, by Meyer et al.,[13] compared clinical and biologic responses during experimental gingivitis and peri-implant mucositis in subjects aged ≥70 years. Although less biofilm accumulation was observed at implant sites, the peri-implant mucosa yielded a higher proportion of bleeding sites compared with that observed in the gingiva,[13] thus confirming the results by Salvi et al.[12] obtained in a younger patient sample.

IS BIOFILM-INDUCED PERI-IMPLANT MUCOSITIS A REVERSIBLE DISEASE?

Although a cause–effect relationship between experimental biofilm accumulation and the development of experimental peri-implant mucositis was claimed in the two studies mentioned previously,[10,11] the case for a true cause–effect relationship would be strengthened by the proof of reversibility to pre-experimental levels of mucosal health. In the study by Salvi et al.,[12] the GI at implant sites dropped significantly less compared with that at tooth sites following 3 weeks of reinstituted oral hygiene practices. Moreover, pre-experimental levels of GI were not reached at implant sites 21 days after reinstitution of self-performed biofilm control.[12] This indicated that resolution of experimental peri-implant mucositis in humans may take longer than 3 weeks (Table 1). In contrast to the study by Salvi et al.,[12] all clinical parameters assessed in an elderly patient sample (i.e., ≥70 years) returned to pre-experimental levels after 3 weeks of reinstituted biofilm control, thus documenting reversibility of experimentally induced peri-implant mucositis.[13]

Resolution of experimental peri-implant mucositis was achieved in both studies at the host biomarker level, as identified by the decrease to pre-experimental values of crevicular fluid pro-inflammatory biomarkers.[12,13] These outcomes[12,13] corroborated the findings of a study in which levels of interleukin (IL)-1β, tumor necrosis factor-alpha (TNF-α), and transforming growth factor-beta2 (TGF-β2) were determined in crevicular fluid samples of 25 subjects before and

after a 3-week period of abolished oral hygiene and after 69 days of re-established oral hygiene practices.[15] While TNF-α and TGF-β2 levels did not change during the experimental period, IL-1β yielded a significant increase after 3 weeks of abolished oral hygiene and was reversed to pre-experimental levels after 69 days.[15] Although the period of reinstituted oral hygiene was shorter at 3 weeks in the studies by Salvi et al.[12] and Meyer et al.,[13] IL-1β crevicular fluid levels returned to pre-experimental values, thus confirming the outcomes obtained by Schierano et al.[15]

EXPERIMENTAL PERI-IMPLANT MUCOSITIS MODELS VERSUS LONG-STANDING PERI-IMPLANT MUCOSITIS LESIONS

Experimental studies in humans and animals have demonstrated that *de novo* biofilm accumulation results in an inflammatory lesion within the peri-implant mucosa with migration of leukocytes through the barrier epithelium and the establishment of an inflammatory infiltrate with an increased proportion of T- and B-cells in the connective tissue adjacent to the barrier epithelium.[6,8,10,16]

Animal models

Experimental peri-implant mucositis models have evaluated the response of the peri-implant mucosa to both early (3 weeks) and long-standing (90 days) periods of undisturbed biofilm accumulation.[16,17] In these dog studies, comparisons were made between the response of the gingiva at teeth and the peri-implant mucosa at implants. Clinical examinations, biofilm sampling, and biopsies were obtained at both the early and long-standing inflammatory lesions. At 3 weeks there was abundant biofilm accumulation, and both the gingiva and the peri-implant mucosa showed clinical signs of inflammation. Histology showed an inflammatory cell infiltrate within the connective tissue which was found in the marginal portion of the soft tissues, immediately adjacent to the barrier epithelium at implants and the junctional epithelium at teeth.[16] In contrast, after a longer period (90 days) of undisturbed biofilm accumulation, the peri-implant mucositis lesions contained a smaller number of fibroblasts than the gingival counterparts, and the area occupied by the inflammatory infiltrate was greater in the peri-implant mucositis lesions than the gingivitis lesions, although it did not extend beyond the barrier epithelium.[17]

Ericsson et al.,[18] in an experimental dog study, obtained biopsies of peri-implant mucosa after 9 months of biofilm accumulation and showed an inflammatory infiltrate located within the marginal portion of the peri-implant mucosa. In another experimental study in the dog model, long-standing biofilm-associated lesions of 5 months duration were established in the peri-implant mucosa adjacent to three different implant systems.[19] The findings of this study confirmed that the size and apical extension of the inflammatory infiltrate did not extend beyond the barrier epithelium for all three implant systems used.

Human studies

Experimental studies in humans have evaluated the response to 3 weeks of biofilm accumulation, corresponding to the time frame of the experimental gingivitis study by Löe et al.,[14] where reversibility of the inflammatory lesion around teeth was demonstrated after reinstitution of biofilm control after 3 weeks. There are studies reporting on human biopsies of peri-implant tissues where long-standing peri-implant mucositis lesions were evaluated.[20,21] Gualini et al.[20] described the immunohistochemical features of peri-implant mucositis lesions obtained from 10 partially edentulous subjects with implants in function between 2 and 5 years. Clinically, the degree of redness and swelling of the inflamed tissues varied; however, all sites bled on gentle probing. In all biopsies the histologic sections showed a small and well-defined inflammatory infiltrate in the connective tissue lateral to the barrier epithelium. The lesions included 7.3% T-cells (CD3 positive) and 4.1% B-cells (CD19 positive). Elastase-positive polymorphonuclear neutrophils (PMN) occured within the barrier epithelium and in the connective tissue compartment immediately lateral to the barrier epithelium. The area of the inflammatory lesions corresponded to 0.36 mm^2, considerably larger than the size of the lesions observed in the experimental short-term (3 week) peri-implant mucositis study by Zitzmann et al.[11] and histologic samples taken mainly from clinically healthy sites.[6,8] These studies confirmed the findings of Seymour et al.[21] who also evaluated biospies of nine subjects with long-standing peri-implant mucositis and found an increase in size of the inflammatory lesion compared to clinically healthy sites.[21]

Peri-implant mucositis may be present for extensive periods of time without progression to peri-implantitis. Conversion of the peri-implant mucositis lesion to peri-implantitis in humans is difficult to study in an experimental design for obvious ethical reasons. However, in a longitudinal study of patients diagnosed with peri-implant mucositis, those with a lack of adherence to supportive peri-implant therapy had a higher incidence of peri-implantitis at 5 years.[22] Hence, sites with peri-implant mucositis should be considered at increased risk for the development of peri-implantitis.

RISK INDICATORS/FACTORS FOR PERI-IMPLANT MUCOSITIS

At a previous World Workshop on Periodontology the definition of a risk factor was agreed as, "an environmental, behavioral or biologic factor confirmed by temporal sequence, usually in longitudinal studies, which if present, directly increases the probability of a disease occurring and, if absent or removed reduces that probability."[23] To identify a true risk factor, prospective studies are required.[24–26] The majority of studies available are cross-sectional or retrospective in design and, therefore, in this review paper the term "risk" refers to a factor which is associated with peri-implant mucositis or a risk indicator.

TABLE 2 Evidence for factors as risk indicators for peri-implant mucositis

Risk indicator	Publication	Summary	Odds ratio (95% CI), multivariate analysis	Significance
Plaque biofilm presence	Roos-Jansaker et al.[28]	218 subjects, 9- to 14-year follow-up, multivariate analysis	1.9 (1.2 –2.9)	$P = 0.004$
Plaque score: poor = median plaque score > 1 and < 2	Ferreira et al.[30]	212 subjects all non-smokers, 6-month to 5-year follow-up, multinomial regression analysis	1.9 (1.2 – 2.3)	$P = 0.0021$
Plaque score: very poor = median plaque score ≥2	Ferreira et al.[30]	212 subjects all non-smokers, 6-month to 5-year follow-up, multinomial regression analysis	2.9 (2.0 – 4.1)	$P = 0.0027$
Full-mouth plaque score 0.30 – 0.43	Konstandinitis et al.[36]	186 subjects, minimum 5-year follow-up, multilevel analysis	1.15 (1.01 – 1.33)	$P < 0.04$
Full-mouth plaque score > 0.43	Konstandinitis et al.[36]	186 subjects, minimum 5-year follow-up, multilevel analysis	1.36 (1.18 – 1.58)	$P < 0.001$
Periodontal BOP > 30% sites affected	Ferreira et al.[30]	212 subjects all non-smokers, 6-month to 5-year follow-up, multinomial regression analysis	3.2 (2.0 – 3.3)	$P = 0.0025$
Presence of keratinized peri-implant mucosa	Roos-Jansaker et al.[28]	218 subjects, 9- to 14-year follow-up, multivariate analysis	1.6 (1.1 – 2.3)	$P = 0.008$
Smoking	Roos-Jansaker et al.[28]	218 subjects, 9- to 14-year follow-up, multivariate analysis	2.8 (1.2 – 6.2)	$P = 0.02$
Smoking	Karbach et al.[27]	100 subjects, 1- to 19-year follow-up, cancer patients, multivariate logistic regression analysis	3.0 (1.14 – 7.92)	$P = 0.26$
Smoking	Rinke et al.[29]	89 subjects, mean observation period 68.2 ± 24.8 months, multiple logistic regression analysis	3.77 (1.2 – 11.86)	$P = 0.023$
Radiation therapy	Karbach et al.[27]	100 subjects, 1- to 19-year follow-up, cancer patients, multivariate logistic regression analysis	2.9 (1.08 – 7.83)	$P = 0.035$
Male gender	Ferreira et al.[30]	212 subjects all non-smokers, 6-month to 5-year follow-up, multinomial regression analysis	1.7 (1.5 – 2.9)	$P = 0.0027$
Diabetes	Ferreira et al.[30]	212 subjects all non-smokers, 6-month to 5-year follow-up, significant association in univariate analysis but not in multinomial regression analysis	NA	NS
Time in function	Ferreira et al.[30]	212 subjects all non-smokers, 6-month to 5-year follow-up, significant association in univariate analysis but not in multinomial regression analysis	NA	NS
Time in function	Máximo et al.[33]	113 subjects, mean follow-up 3.4 years, weak correlation Pearson correlation coefficient (r = 0.44, $P = 0.0058$)	NA	NS

NA, not applicable; NS, not significant; CI, confidence interval

General risk indicators/factors

Factors which may affect host susceptibility to biofilm-induced peri-implant mucositis have been investigated. Cigarette smoking has been identified as a risk indicator for peri-implant mucositis in three studies (Table 2).[27–29] There is also evidence for radiation therapy as a risk indicator for peri-implant mucositis.[27] There is some evidence for diabetes mellitus as a risk indicator for peri-implant mucositis.[28,30] Poorly controlled diabetes mellitus (HbA1c levels > 10.1) was shown to be associated with increased bleeding on probing at implants.[31] While a history of cardiovascular disaease has been associated with an increased risk of peri-implantitis, there is no evidence for an association with peri-implant mucositis.[32] Máximo et al.[33] reported a significant but weak correlation (r = 0.44, Pearson χ^2 test) between peri-implant mucositis and increased time of loading of the implant. However, this study did not account for confounding factors, and the reported association may have been due to the increased time in function without regular removal of the biofilm.

Similarly, in a recent cross-sectional study conducted in 193 patients with implants in function for at least 12 months (range, 1 to 9 years), an association between peri-implant mucositis and age and time of prosthesis in function was reported.[34] However, a clear distinction between peri-implant mucositis and peri-implantitis was not described. Ferreira et al.[34] also reported an association with peri-implant mucositis and systemic disease. However, the systemic diseases described included "diabetes mellitus, hormonal changes, menopause, chemotherapy, thyroid alterations, cardiac problems, and alcohol use," and thus the results of the study are difficult to interpret.

Major local risk indicators/factors

Oral hygiene

Outcomes of cross-sectional clinical studies have clearly indicated that biofilm accumulation is associated with the presence of peri-implant mucositis around osseointegrated dental implants.[30,35,36] Ferreira et al.[30] reported on 212 patients treated with three different implant systems and diagnosed with peri-implant mucositis. All implants had been in function for a period ranging from 6 months to 5 years. The modified plaque index[37] was recorded, and the full-mouth plaque scores were stratified as good (median score ≤1), poor (median score > 1 and < 2), and very poor (median score ≥2). The authors reported a significant dose-dependent association between plaque scores and peri-implant mucositis. The prevalence of peri-implant mucositis was reported as 64.6% at patient level and 62.6% at implant level.[30] Outcomes of another study involving 218 patients with 999 implants in function for a period of 9 to 14 years indicated that plaque scores were significantly associated with the presence of peri-implant mucositis.[35]

Mechanical biofilm control should be considered the standard of care for management of peri-implant mucositis administered either by the patient[38] or the oral healthcare professional.[39]

Compliance/lack of compliance with supportive implant therapy (SIT)

Among patients not adhering to regular supportive implant therapy (SIT), peri-implant mucositis was reported to be a common finding with a prevalence of 48% during an observation period of 9 to 14 years.[28,35,40] Conversely, outcomes of a prospective cohort study with a 5-year follow-up indicated that implants placed in patients with treated periodontal conditions and adhering to an SIT program yielded a 20% prevalence of peri-implant mucositis.[41] In that study, upon diagnosis of peri-implant mucositis, all implants with the exception of one were successfully treated according to a cumulative anti-infective protocol.[42] Findings from a 3-month randomized placebo-controlled clinical trial revealed that mechanical debridement with or without local application of chlorhexidine gel in conjunction with optimal self-performed biofilm control completely resolved bleeding on probing around 38% of implants diagnosed with peri-implant mucositis.[43]

In partially edentulous patients, pre-existing peri-implant mucositis in conjunction with lack of adherence to SIT was associated with a higher incidence of peri-implantitis during a 5-year follow-up period.[22] The outcomes of that study yielded a 5-year incidence of peri-implantitis of 18.0% in the group of patients with SIT and of 43.9% in the group without SIT, respectively.[22] The logistic regression analysis revealed that lack of adherence to SIT within the overall patient sample was significantly associated with the onset of peri-implantitis with an odds ratio of 5.92.[22] Hence, therapy of peri-implant mucositis should be considered a prerequisite for the prevention of peri-implantitis.

Materials and surface characteristics of implant components

Evidence for the influence of implant surface roughness on the incidence of peri-implant mucositis in humans is limited.[44] A 12-month comparative analysis in humans between machined titanium abutments (Ra = 0.2 μm) and highly polished ceramic abutments (Ra = 0.0 6 μm) indicated that further reduction in surface roughness had no impact on bleeding on probing (BOP) scores.[45] A study in humans investigated the association between abutment surfaces of varying roughness and the early inflammatory response of the peri-implant mucosa.[46] Although a statistically significant difference among patients was observed with respect to biofilm accumulation on the abutment surfaces and inflammatory cells, no association was observed between the inflammatory response and abutment surface roughness after an observation period of 4 weeks.[46]

Compared with implants and abutments made of titanium, more beneficial properties in terms of biocompatibility have recently been claimed for implants and abutments made of zirconium dioxide (ZrO_2). It has to be noted, however, that in clinical studies no significant differences in BOP scores[47,48] or slightly higher BOP scores[49,50] were reported around ZrO_2 compared with titanium abutments.

Design of implant-supported prostheses

Accessibility for biofilm removal around implant-supported prostheses plays an important role in the prevention and management of peri-implant diseases. Implants with supramucosal restoration margins yielded significantly greater reductions in probing depths following treatment of peri-implant mucositis compared with those with submucosal restoration margins.[43] This finding corroborates previous observations on the association between subgingival restoration margins at natural teeth and periodontal inflammation and attachment loss.[51–53]

Outcomes of a clinical retrospective study indicated that high proportions of implants diagnosed with peri-implantitis were associated with inadequate biofilm control or lack of accessibility for oral hygiene measures, while peri-implantitis was rarely detected at implants supporting cleansible prostheses or when proper biofilm control was performed.[54] Consequently, oral hygiene instructions should be individually adapted to patients treated with dental

implants because peri-implant mucositis may be considered a precursor for peri-implantitis. Furthermore, whenever possible, margins of implant-supported prostheses should be placed at or above the peri-implant mucosal margin to facilitate access for biofilm control. Implant-supported reconstructions impairing access for biofilm removal should be adjusted or replaced by cleansible prostheses.

Dimensions of keratinized peri-implant mucosa

The effect of the dimensions of peri-implant keratinized mucosa as a risk indicator for peri-implant mucositis was investigated in several studies in humans. While some studies reported higher rates of peri-implant mucositis at implants lacking or surrounded by an inadequate width (<2 mm) of keratinized mucosa,[55–60] other studies found no association[61–63] or a postive association.[28] Collectively, evidence for the presence or minimum width of keratinized mucosa around implants to maintain soft tissue health and stability remains controversial. In clinical situations of adequate self-performed biofilm control around implants, presence or grafting of keratinized mucosa to maintain peri-implant health does not seem to be essential.

Excess cement

Excess cement has been associated with clinical signs of peri-implant mucositis.[44,64–66] Patients restored with single-unit crowns with excess cement displayed more signs of peri-implant mucositis compared with those restored with single-unit crowns without excess cement.[64] In addition, peri-implant mucositis was more prevalent in patients with cemented prostheses compared with those with screw-retained prostheses.[65] Therefore, to avoid cement excess, restoration margins should be located at or above the peri-implant mucosal margin or restorations should be cemented on individualized abutments allowing proper cement removal.

SIMILARITIES AND DIFFERENCES BETWEEN RISK INDICATORS/FACTORS FOR PERIODONTAL DISEASES VERSUS PERI-IMPLANT MUCOSITIS

A recent systematic review summarized potential risk indicators for peri-implant mucositis and identified biofilm accumulation and smoking as risk indicators.[44] In addition, a cross-sectional study showed that plaque score was a risk indicator for peri-implant mucositis in a dose-dependent manner (Table 2).[36] Data from the 2009–2012 National Health and Nutrition Examination Survey (NHANES) identified cigarette smoking as a modifiable risk indicator for all levels of periodontitis severity.[67] Uncontrolled diabetes, male gender, and age were also identified as risk indicators for periodontal disease.[67] Thus, there are similarities in risk indicators for peri-implant mucositis and periodontal disease, although there is still limited information available regarding risk for peri-implant mucositis.

Non–biofilm-induced mucositis conditions

Mucosal diseases such as oral lichen planus (OLP) have been suggested to negatively affect the ability of the epithelium to attach to titanium surfaces. Hence, it may be postulated that peri-implant mucosa affected by such conditions would also respond differently than a healthy peri-implant mucosa to a bacterial challenge, resulting in a faster breakdown of the peri-implant soft tissue seal. The prevalence of peri-implant mucositis was assessed in patients diagnosed with oral lichen planus (OLP) and compared with that of control patients.[68] The results indicated that the presence of OLP was not associated with a higher prevalence of peri-implant mucositis.[68] These results were confirmed in a cross-sectional study failing to report significant differences in the prevalence of peri-implant mucositis in patients with dental implants and diagnosed with or without OLP.[69] However, in patients diagnosed with OLP and gingival desquamation, a significantly higher prevalence of peri-implant mucositis was observed.[68] This higher prevalence of peri-implant mucositis reported in the study by Hernandez et al.[68] may be associated with higher plaque scores, with the stomatologic condition per se or with both.

It has been suggested that susceptible patients may suffer from allergic/adverse reactions to materials such as titanium and titanium alloys;[70] however, the evidence remains very limited.[71]

CONCLUSIONS

Peri-implant mucositis is an inflammatory lesion of the peri-implant mucosa in the absence of continuing marginal bone loss. Peri-implant mucositis is primarily caused by a disruption of the host–microbe homeostasis at the implant–mucosa interface and is a reversible condition at the host biomarker level. Optimal biofilm control in experimental peri-implant mucositis studies may take longer than 3 weeks for complete resolution at the clinical level. Factors associated with peri-implant mucositis include biofilm accumulation, smoking, and radiation therapy. Regular supportive peri-implant therapy with biofilm removal is an important preventive strategy against the conversion of health to peri-implant mucositis and also against the progression of peri-implant mucositis to peri-implantitis.

ACKNOWLEDGMENTS AND DISCLOSURES

The authors report no conflicts of interest related to this review paper.

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How to cite this article: Heitz-Mayfield LJA, Salvi GE. Peri-implant mucositis. *J Clin Periodontol*. 2018;45 (Suppl 20):S237–S245. https://doi.org/10.1111/jcpe.12953