

# Periimplant diseases: where are we now? – Consensus of the Seventh European Workshop on Periodontology

Niklaus P. Lang<sup>1</sup> & Tord Berglundh<sup>2</sup>,  
on Behalf of Working Group 4 of the  
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Periodontology

<sup>1</sup>Prince Phillip Dental Hospital, The University of Hong Kong, Hong Kong SAR, China; <sup>2</sup>Department of Periodontology, Göteborg University, Göteborg, Sweden

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## Abstract

**Background:** Peri-implant diseases present in two forms – peri-implant mucositis and peri-implantitis.

**Materials and Methods:** The literature was systematically searched and critically reviewed. Four manuscripts were produced in specific topics identified as key areas to understand the microbial aetiology and the pathogenesis of peri-implant diseases and how the implant surface structure may affect pathogenesis.

**Results:** While peri-implant mucositis represents the host response of the peri-implant tissues to the bacterial challenge that is not fundamentally different from gingivitis representing the host response to the bacterial challenge in the gingiva, peri-implantitis may differ from periodontitis both in the extent and the composition of cells in the lesion as well as the progression rate. A self-limiting process with a "protective" connective tissue capsule developing appears to dominate the periodontitis lesion while such a process may occasionally be lacking in peri-implantitis lesions. Bacterial biofilm formation on implant surfaces does not differ from that on tooth surfaces, but may be influenced by surface roughness. Nevertheless there is no evidence that such differences may influence the development of peri-implantitis.

**Conclusion:** It was agreed that clinical and radiographic data should routinely be obtained after prosthesis installation on implants in order to establish a baseline for the diagnosis of peri-implantitis during maintenance of implant patients.

Working Group 4: Ingemar Abrahamsson (Göteborg, Sweden), Noel Claffey (Dublin, Ireland), Hugo De Bruyn (Ghent, Belgium), Nikolaos Donos (London, UK), Marco Esposito (Milano, Italy), Lisa J.A. Heitz-Mayfield (Perth, WA, Australia), Björn Klinge (Stockholm, Sweden), Andrea Mombelli (Geneva, Switzerland), Richard M. Palmer (London, UK), Marc Quirynen (Leuven, Belgium), Stefan Renvert (Kristianstad, Sweden), Giovanni E. Salvi (Berne, Switzerland), Ignacio Sanz (Madrid, Spain), Frank Schwarz (Düsseldorf, Germany), Anton Sculean (Berne, Switzerland), Fabio Vignoletti (Madrid, Spain), Nicola U. Zitzmann (Basel, Switzerland)



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The Sixth European Workshop on Periodontology (2008) has confirmed that “peri-implant diseases are infectious in nature. *Peri-implant mucositis* describes an inflammatory lesion that resides in the mucosa, while *peri-implantitis* also affects the supporting bone” (Lindhe & Meyle 2008).

While these definitions are considered adequate, the diagnostic criteria for them are less clear.

Based on longitudinal clinical studies and in agreement with the consensus of the Sixth EWP (Heitz-Mayfield 2008), the *time of prosthesis installation* should be chosen to establish baseline criteria representing homeostasis following implant installation with or without subsequent abutment connection. It is realized that, e.g. with immediate loading protocols, such baseline documentation may be premature.

To establish baseline, a radiograph should be obtained to determine alveolar bone levels after physiologic remodeling, and peri-implant probing assessments performed.

It is evident that recorded baseline data will be the reference from which the development of peri-implant disease can be recognized and followed in subsequent examinations.

Therefore, when changes in the clinical parameters indicate disease (bleeding on probing, increased probing depth), the clinician is encouraged to take a radiograph to evaluate possible bone loss. In relation to clinical studies evaluating periodontal patients over time, probing depth increases beyond the 5 mm level in combination with bleeding on probing indicated a higher risk for disease progression and hence, radiographic evaluation is required.

It is assumed that bone loss occurring after initial remodelling is mainly due to bacterial infection.

The key parameter for the diagnosis of *peri-implant mucositis* is bleeding on gentle probing (<0.25 N).

*Peri-implantitis* is characterized by changes in the level of the crestal bone

in conjunction with bleeding on probing with or without concomitant deepening of peri-implant pockets. Pus is a common finding in peri-implantitis sites.

It has to be realized, however, that peri-implantitis may be initiated and/or maintained by iatrogenic factors (e.g. excess cement remnants, inadequate restoration-abutments seating, over-countouring of restorations, implant mal-positioning, technical complications). Moreover, bone loss induced at the time of implant placement by traumatizing the pristine bone beyond its adaptive capacity may also persist.

There are, however, implant (e.g. platform switching) and/or abutment designs at which probing may be difficult, and probing depth may underestimate the extent of the lesion.

*Do mucositis lesions around implants differ from gingivitis lesions around teeth?* (Lang et al. 2011)

1. *Can structural differences between peri-implant tissues and periodontal tissues influence onset and progression of mucositis/gingivitis?*

Although there are developmental differences of junctional epithelia at teeth and implants, there is no evidence for structural or functional diversity and hence, the “epithelial sealing” around implants is considered to be identical to that of teeth.

The structural differences mentioned consider the supracrestal connective tissue compartment in the gingiva compared with that of the peri-implant mucosa. Animal experiments have revealed differences in the fibroblast to collagen ratio and in the arrangement of the vasculature. The increased collagen to fibroblast ratio in the interfacial connective tissue together with a decreased vascularity of that region may have an impact on the onset and progression of mucositis. Nevertheless, the detailed molecular aspects of the adhesion of the connective tissue are not known. In the peri-implant mucosa, connective tissue fibers are predominantly arranged in a circular mode and in the long axis of the implant proper. Hence, the resistance to clinical probing of this region differs from that encountered around teeth.

Nevertheless, in the onset of mucositis/gingivitis, there is no evidence that the host response to the bacterial challenge is different at tooth and implant sites.

2. *Are mucositis lesions reversible?*

In agreement with the conclusions of the Sixth European Workshop on Perio-

dentology (2008) regarding the efficacy of mucositis therapy, it has to be reiterated that mucositis is reversible. While this statement is based on biopsies from animal and human studies, a recent human clinical experiment using the experimental gingivitis/mucositis model, revealed reversibility of mucositis at the biomarker level (MMP 8, IL-1 $\beta$ ) demonstrated in crevicular fluid samples after 3 weeks (Salvi et al. 2011).

Therefore, these findings represent the basis for the prevention and therapy of peri-implant mucositis.

3. *Is host response to microbial challenge different at implants and teeth?*

Since a host response is reacting to a bacterial challenge, there is no reason to assume that it will be different in the peri-implant mucosa when compared to the gingiva. Indeed, initial biofilm formation has led to lesions similar in size and composition after 3 weeks. However, owing to the various histories of such sites before the experimental induction of biofilm formation, it has to be realized that the extent of the host response at implant sites may be different than at tooth sites when biofilms persisted for prolonged periods of time.

4. *How well does experimental mucositis/gingivitis mimic the natural long-standing counterparts?*

Human studies have indicated that the composition of the inflammatory infiltrate and the pattern of cytokine expression (Trombelli et al. 2010) that develops as a result of an experimental bacterial challenge may vary substantially from the composition of a long-standing gingivitis infiltrate and cytokine expression.

Clinically however, the signs and symptoms of experimental and long-standing gingivitis may not be distinguishable from each other.

Similar biopsy studies for peri-implant mucositis are lacking. Hence, definite conclusions regarding this issue cannot be made, and it may be assumed that, similar to the situation in gingivitis, differences may exist. The clinical impact of the known differences in gingivitis, however, is yet to be established.

5. *Is the conversion from mucositis to peri-implantitis similar to that of gingivitis to periodontitis?*

On the basis of the prevalence of peri-implantitis in cross-sectional studies and comparing it with the prevalence and progression rate of periodontitis from longitudinal studies, it may be assumed that mucositis lesions

may progress to peri-implantitis earlier than their counterparts around teeth. However, this concept has to be validated in appropriately sized longitudinal studies.

*Are peri-implantitis lesions different from periodontitis lesions?* (Berglundh et al. 2011)

1. *How well do the lesions produced in experimental peri-implantitis/periodontitis resemble the natural counterparts?*

Animal experiments have demonstrated that placement of ligatures around the neck of an implant/tooth in a submucosal/subgingival position together with plaque formation results in the development of peri-implantitis/periodontitis lesions that have many features in common (e.g. size and composition) with natural lesions as presented in human biopsies.

2. *What is the relevance of experimental peri-implantitis/periodontitis in regards to disease progression?*

While ligature-induced breakdown does not mimic natural disease progression, progression of peri-implantitis with additional loss of supporting tissues was demonstrated in an experimental peri-implantitis model including an extended period of plaque formation following ligature removal. A similar model has not been described for experimental periodontitis.

3. *Is the mechanism of bone loss during disease progression similar in peri-implantitis and periodontitis?*

Bone loss during disease progression is mediated by inflammatory reactions resulting in osteoclastic resorption in both diseases. Factors influencing the recruitment and activation of osteoclasts in both diseases have not been compared.

4. *Can structural differences between peri-implant tissues and periodontal tissues influence onset and progression of peri-implantitis/periodontitis?*

Structural differences between peri-implant and periodontal tissues may influence host response in peri-implantitis and periodontitis. Results from analysis of human biopsy material and animal experiments have revealed that histopathological differences exist between peri-implantitis and periodontitis lesions. The following features were observed:

The apical extension of the lesion was more pronounced in peri-implantitis than in periodontitis.

The lesion in peri-implantitis sites consistently extended apical of the

pocket epithelium, and the apical portion of the lesion was in direct contact with the biofilm residing on the implant surface.

While plasma cells and lymphocytes dominated in both types of lesions, neutrophil granulocytes and macrophages occurred in larger proportions in peri-implantitis than in periodontitis.

A ‘self-limiting’ process existed in the tissues around teeth that resulted in a protective connective tissue capsule that separated the lesion from the alveolar bone. Such a ‘self-limiting’ process did not occur in peri-implant tissues, and the lesion extended to the bony crest.

Peri-implantitis lesions, in contrast to periodontitis lesions, exhibited signs of acute inflammation and large amounts of osteoclasts that lined the surface of the crestal bone.

5. *Are there differences in the morphology of periodontitis lesions and peri-implantitis lesions? If so, do we understand the reasons for it?*

While the localized nature of periodontitis lesions may be related to, e.g. anatomical features, the reason for the commonly observed circumferential nature of peri-implantitis lesions is not known. It may be speculated that characteristics, such as the absence of a periodontal ligament or lateral spread of the infection on the implant surface may determine the morphology of the lesion.

*The characteristics of biofilms in peri-implant disease* (Mombelli & Décaillet 2011)

1. *Are there differences in biofilm formation between teeth and implants?*

Because the ecological environment is the same, the basic principles of biofilm formation are similar. However, in vitro and in vivo studies on abutment surfaces suggest that there may be differences related to chemical and physical properties of the surface on which the biofilm establishes (materials, roughness, surface energy).

2. *What is the role of supra-mucosal biofilm formation in the pathogenesis of peri-implant disease?*

From experimental mucositis/gingivitis studies there is evidence for a cause and effect relationship between biofilm formation on implants/teeth and the development of mucositis/gingivitis.

The continuous presence of a biofilm on implants during 6 months induced an inflammatory lesion in the connective tissue of the peri-implant mucosa dominated by plasma cells and lymphocytes.

Under the assumption that mucositis is the precursor of peri-implantitis, supramucosal plaque formation should be considered as an initial event in the development of peri-implantitis. The lack of marked microbiological differences between mucositis and peri-implantitis may reflect the fact that, in most cases, the disease develops from mucositis to peri-implantitis.

3. *What is the role of the oral environment (e.g. periodontal health/disease) for the initiation and progression of peri-implant disease?*

It has been shown in multiple papers that the microbiota in the oral environment determines to a large extent the composition of the microbiota developing around implants.

Pathologic conditions in the oral environment (e.g. the persistence of untreated periodontal disease) may induce changes in the ecosystem that may favour the colonization of pathogenic microorganisms in implant sites.

4. *Are current assessment techniques adequate in identifying potential differences in the composition in the biofilm at peri-implantitis and periodontitis sites?*

The research on this subject has initially been driven by the microbiological investigations carried out in the periodontal field; therefore, the main focus has been on periodontal pathogens (PG, AA). However, this approach may, indeed, have led to neglecting or underestimating potential pathogens that are not considered to be important in periodontal diseases, but are important in extra-oral infections. The issue of non-cultivable bacteria has gained considerable attention in the study of the aetiology of periodontal diseases. So far, similar data in relation to peri-implantitis are lacking.

In addition, classical microbiology has been based to a large extent on the investigation of microorganisms grown under laboratory conditions that are not representative of how microorganisms live in biofilms.

5. *Is the formation of submucosal calculus in peri-implantitis a common feature?*

Although research data are lacking, clinical observation suggests that submucosal calculus formation on implants is less common than sub-gingival calculus formation on teeth. This phenomenon may be related to the observation that peri-implant disease, in general, progresses faster than periodontitis. Because adequate debridement is con-

sidered an important part in the treatment of peri-implant infections, the issue of removal of mineralized deposits must be further explored. Macroscopic or microscopic differences in the design of implants and/or other components may require different methods for mechanical debridement.

6. *Is there evidence of bacterial invasion in peri-implantitis?*

Histological data from experimental peri-implantitis studies show the presence of epithelial ulceration in conjunction with a disruption of the connective tissue adhesion. This suggests that the soft tissue seal around implants is less resistant to the bacterial challenge and hence, bacterial invasion may be likely in peri-implantitis lesions. However, no evidence from naturally occurring peri-implant disease is available to substantiate this hypothesis.

*How do implant surface characteristics influence peri-implant disease?* (Renvert et al. 2011)

1. *Is there evidence for a specific implant surface/design (intra-osseous/transmucosal parts) being at increased risk for onset and/or progression peri-implant disease?*

Based on the surface roughness, implants have been proposed to be categorized as (Albrektsson & Wennerberg 2004):

- smooth  $S_a < 0.5 \mu\text{m}$ ,
- minimally rough  $S_a: 0.5\text{--}1.0 \mu\text{m}$ ,
- moderately rough  $S_a: 1.1\text{--}2.0 \mu\text{m}$  and
- rough  $S_a: > 2.0 \mu\text{m}$ .

However, for the purpose of discussion, the  $S_a$  values have to be interpreted with care. Moreover,  $S_a$  values are not the exclusive parameter characterizing the implant surface.

Rough surface implants [titanium plasma sprayed (TPS)] are more likely to develop peri-implantitis than minimally rough implants once exposed to the oral environment. Furthermore, based on two animal studies, some moderately rough implant surfaces may be more susceptible to *disease progression* than others.

1. *Is there evidence for a specific implant surface/design influencing biofilm formation?*

Biofilms form on all implant surfaces. However, surface characteristics may influence the amount and composition of biofilm formation. So far, there is not enough evidence to make definitive conclusions on the clinical implications.

2. *Does degradation of implant surface coatings influence onset/progression of peri-implant disease?*

Case reports have indicated that thick HA (hydroxyapatite) coating failure and subsequent infection can lead to implant loss when the coating detaches from the underlying titanium surface. However, the outcome of the use of thin HA coatings and other types of coatings has not been adequately evaluated as yet (Overgaard et al. 1996).

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Address:

Niklaus P. Lang  
 Prince Phillip Dental Hospital  
 The University of Hong Kong  
 Hong Kong SAR  
 China  
 E-mail: nplang@dialed.unet.ch