Comparative biology of chronic and aggressive periodontitis vs. peri-implantitis

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Peri-implant disease following successful integration of an endosseous implant is the result of an imbalance between the bacterial challenge and the host response. Peri-implant diseases may affect the peri-implant mucosa only (peri-implant mucositis) or also involve the supporting bone (peri-implantitis) (112).

In the 6th European Workshop on Periodontology in 2008, the definitions of peri-implant diseases were revised as follows: peri-implant mucositis is the presence of inflammation in the mucosa at an implant with no signs of loss of supporting bone; and peri-implantitis, in addition to inflammation in the mucosa, is characterized by the loss of supporting bone (57).

For the purpose of this review, these two diseases are compared with their counterparts around teeth (i.e. gingivitis and periodontitis) without any consideration of the classification of periodontitis (chronic and aggressive). The question therefore arises: Are periodontitis and peri-implantitis fundamentally different from the perspectives of etiological aspects, pathogenesis aspects, risk assessment and therapy?

Etiological aspects (human studies)

Colonization (dynamics and surfaces)

In the late 1980s, the colonization of oral implants in edentulous patients was studied using culture techniques. It was demonstrated that 1 week after implant placement, a microbiota characterized predominantly by gram-positive, facultative organisms was established and maintained throughout the duration of the study (6 months), with the exception of one implant that showed suppuration after 3 months. At this particular site the subgingival microbial community changed continuously within the preceding months from a gram-positive facultative microbiota to one rich in anaerobic gram-negative bacteria (71).

In recent years, studies have focused on the colonization of implants in partially edentulous individuals (Figs. 1A–C). It was shown that bacterial colonization occurred immediately following transmucosal implant placement (30 min) (29) and was stable after 2 weeks (29, 82, 83). Moreover, the composition of the microbiota present after 3 months was shown to be predictive for the colonization after 1 year (91). In addition, it was recognized that the composition of the biofilms established on the implant surfaces corresponded closely to those identified from teeth surrounded by healthy tissues. Hence, it can be anticipated that the microbiota present in the oral cavity may have a substantial impact on biofilm formation on newly placed implants.

Microbiota associated with healthy peri-implant tissues

The microbiota associated with healthy peri-implant tissues has been identified in many cross-sectional studies that have generally characterized the composition as being dominated by gram-positive facultative cocci and rods (20, 29, 55). However, gram-negative anaerobic rods may also be found in small numbers and in low proportions at some implants.
Microbiota associated with peri-implant infections

Association studies have identified a microbiota characterized by high counts and proportions of gram-negative anaerobic bacteria around implants with clinical signs of peri-implantitis. These studies have found a high prevalence of pathogens associated with periodontitis (2, 9, 15, 16, 52, 66, 69, 71, 101, 104), which included members of the red complex species (*Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythia*) and orange complex species (*Fusobacterium* sp. and *Prevotella intermedia*), as defined by Socransky et al. (102). The presence of *Aggregatibacter actinomycetemcomitans* has also been reported at peri-implantitis sites (39, 110). A number of studies have also reported an association between the presence of *Staphylococcus aureus*, enteric rods and *Candida albicans*, and peri-implantitis (7, 15, 47, 55, 84, 90).

Intra-individual transmission of pathogens

The similarity between the composition of the microbiota around teeth and implants within the same subject has clearly been demonstrated (3, 46, 53, 70, 75, 80, 81, 83, 103, 105). Furthermore, longitudinal studies have investigated the transmission of putative periodontal pathogens from periodontal sites to implant sites. In a group of patients treated for periodontitis and undergoing a maintenance-care program, the deepest periodontal site in each quadrant was sampled before, and 3 and 6 months after, implant placement. The same putative periodontal pathogens were identified in residual periodontal pockets and colonizing the implants after 3 and 6 months (70). The transmission of bacteria from tooth to implant sites was confirmed in studies investigating the dynamics of colonization (20, 109). De Boever & De Boever (20) identified the presence of periodontal pathogens around implants 1, 3 and 6 months after implant placement in partially dentate patients who were successfully treated for aggressive periodontitis and yielded plaque and bleeding scores of <20%. Only 5 of the 22 patients were colonized with putative periodontal pathogens, which had already occurred by 14 days. This microbiota remained unchanged at the 6-month examination. Likewise, the remainder of the patients displayed a microbiota at 6 months that had a composition very similar to that encountered after 10 days. This underlines the importance of eliminating potential reservoirs of periodontal pathogens before implant placement and...
the importance of maintaining periodontal health in partially dentate patients with oral implants (70, 109).

**Similarities and dissimilarities with periodontal infections**

Whilst the majority of studies show that the composition of the subgingival microbiota associated with health and disease is similar around implants and teeth (3, 46, 53, 70, 75, 80, 81, 83, 103, 105), there is emerging evidence that differences may be present in some of the peri-implant infections (87).

Biofilm formation is influenced by the properties of the surface to be colonized, including chemical composition, surface roughness and surface free-energy (107). *In vitro* studies have demonstrated an affinity of *S. aureus* for titanium surfaces (34). A number of clinical studies have identified high levels of *S. aureus* at deep peri-implant pockets with the presence of suppuration and bleeding on probing (85, 87). *S. aureus* is not strongly associated with chronic periodontitis. However, it is well documented that *S. aureus* can be associated with therapy-resistant (refractory) cases of periodontitis (19, 27, 37, 84).

**Pathogenesis aspects**

**Host response to biofilm formation**

The host response to biofilm formation has been studied using various animal models. Berglundh et al. (13), in a dog model, evaluated clinical and histological characteristics following *de novo* plaque formation around teeth and implants. The size and extent of the subepithelial connective tissue infiltrate was assessed after 3 weeks of undisturbed plaque accumulation. Both of these parameters, as well as the composition of the cellular infiltrate, were identical in the gingiva around teeth and in the mucosa around osseointegrated implants. This suggested that the early host response to the bacterial challenge around the implanto-mucosal unit is of a similar magnitude and intensity as the one that occurs at the dentogingival unit.

The effect of biofilm formation on the development of the inflammatory response was later studied experimentally in humans (77) (Fig. 2). Twenty partially dentate patients received oral implants following successful completion of periodontal therapy. After 6 months of closely supervised periodontal conditions the patients were asked to refrain from oral hygiene practices for a period of 3 weeks. At the end of this period optimal plaque-control measures were re-instituted. Comparing the accumulation of biofilm and the host response expressed at gingival and peri-implant mucosal tissues revealed no difference in the development of gingivitis and mucositis, respectively. Hence, there is a similar cause-and-effect relationship between the accumulation of biofilm and the development of experimental gingivitis and peri-implant mucositis (60). This cause-and-effect relationship has since been confirmed in another group of partially dentate human volunteers (114). In addition to confirming the development of clinical parameters, the inflammatory response was also characterized by the enumeration of the proportions of T and B cells in both gingiva and peri-implant mucosa. No statistically significant differences in the host response of these two tissues could be demonstrated after 3 weeks.

**Host response with established biofilms**

In an experimental dog study, 3 months of biofilm accumulation with respect to the host response was analyzed using both clinical and histological parameters, comparing gingival tissues with peri-implant mucosal tissues (21). After 90 days of undisturbed biofilm formation, the dogs had accumulated large amounts of biofilm and the soft tissues around implants and teeth bled on gentle probing. The histological examination of the two types of inflamed soft tissues revealed that (i) both gingiva and peri-implant mucosa contained an inflammatory cell infiltrate within the apical extension of the junctional epithelium and (ii) the composition of these infiltrates was
similar in both gingiva and peri-implant mucosa, showing a substantial loss of collagen and a significant increase in the numbers of inflammatory cells. However, the apical extension of the inflammatory infiltrate in the mucosa, as well as the size of the lesion, was significantly greater (almost threefold higher) than that in the gingiva. This, in turn, meant that the host response to the bacterial challenge after a period of 3 months was more pronounced in the peri-implant mucosa than in the gingiva. It is, however, unknown whether this fact will render the peri-implant mucosal tissue more prone to the loss of supporting bone (i.e. peri-implantitis). The host response to the bacterial challenge has been demonstrated to develop irrespective of the implant system used (1) (Figs. 4A and B).

Host response with advancing infections

For obvious ethical reasons, sequential studies on the pathogenesis of periodontitis and peri-implantitis can only be performed in animals. The ligature model for biofilm formation has been used both in dogs (56) and in monkeys (49, 96) to study the transition from peri-implant mucositis to peri-implantitis and to make comparisons with the pathogenesis of ligature-induced periodontitis.

In the monkey model, plaque and gingival indices, increasing probing depths and loss of attachment were identical at ligated teeth and ligated implants during the course of an 8-month plaque-accumulation study. Nonligated implants, however, only developed mucositis and minimal bone loss within the same period of time. It was concluded that massive plaque accumulation generated by ligature placement would result in similar tissue responses for gingival and peri-implant tissues (49). In the dog study, the progression of the inflammatory lesion was found to extend into the bone marrow at peri-implantitis sites and hence to progress to a greater extent than the periodontitis lesion (56).

Another study, in monkeys (96), compared the inflammatory response and the loss of supporting bone at ligature-induced peri-implantitis and ligature-induced periodontitis for both ankylosed teeth and normal control teeth. The histologic observations supported a greater extent of bone loss and inflammatory infiltrate around implants and ankylosed teeth compared with control teeth. It may therefore be speculated that the absence of the periodontal ligament could have a lesion-promoting effect in the pathogenesis of peri-implantitis (96).

Following ligature removal, some of the lesions will go into remission, while the majority of the sites will experience further bone loss. In about 25% of the cases it was demonstrated that rapid progressive
bone loss occurred within a period of 1 year after removal of the ligatures (113). This, in turn, means that peri-implant infections, once established, will probably continue to progress, even though arrest of the progression may be encountered in about 20% of cases. The spontaneous progression of peri-implantitis may be influenced by exposed roughness and topography of implant surfaces (5, 6, 12) favoring the colonization with specific pathogens. There is conflicting evidence, however, regarding the effect of the implant surface in the pathogenesis of peri-implantitis in humans (10, 111).

Similarities and dissimilarities with periodontal infections

Human case series reporting histopathological data at sites with peri-implantitis have described inflammatory lesions with high proportions of B cells and plasma cells, suggesting that the peri-implantitis lesion has features similar to those of both aggressive periodontitis and chronic periodontitis (11, 22, 32). The peri-implantitis lesion may progress from an existing mucositis with a greater infiltrate predominated by plasma cells that extends apically to the position of the pocket epithelium (11).

From overwhelming evidence in the literature, it appears that the development of periodontitis and peri-implantitis lesions follows a similar sequence of events. However, the dynamics of these pathological processes may not be identical at all times. One difference may be that the periodontitis lesion is always walled off by an intact supracrestal connective tissue fibre compartment (100). Therefore, the inflammatory cell infiltrates generally do not penetrate into the alveolar bone marrow. By contrast, a peri-implantitis lesion may progress without the presence of a healthy connective tissue fibre compartment walling off the lesion from the alveolar bone. Consequently, it appears that the infection may progress into the bone marrow in some instances (5, 6, 56, 61). Hence, from a clinical point of view, some peri-implantitis lesions can be expected to progress rapidly. This suggests that a peri-implant site diagnosed with peri-implantitis should be treated without delay.

Risk evaluation (human studies)

Identification of true risk factors for disease requires longitudinal studies with a prospective design to be carried out in humans. A risk indicator can be determined following cross-sectional analyses to identify factors associated with disease.

The known risk factors for periodontitis development and progression are as follows (35):
- poor oral hygiene;
- gingivitis;
- tobacco consumption; and
- diabetes mellitus.

By far the most important risk factor for both the development and the progression of periodontitis is poor oral hygiene practices (72). This, in turn, results in persistent gingivitis that has been identified as a risk factor both for disease progression and for tooth loss (93).

Tobacco consumption, in a dose-dependent manner, represents the third risk factor for the progression of periodontal disease. Heavy smokers should be considered as individuals at high risk for disease progression (14).

Finally, studies indicate that diabetes patients with poor glycemic control have an increased risk for periodontal disease and disease progression (73). Regarding the risk evaluation of patients to develop peri-implantitis, the literature is less conclusive. Therefore, only proposed risk indicators can be addressed.

A recent review evaluated risk indicators for peri-implantitis (36). Cross-sectional analyses of the studies in that review included the following patient-based risk indicators:
- poor oral hygiene;
- history of periodontitis;
- tobacco consumption;
- diabetes mellitus;
- alcohol consumption; and
- genetic traits.

Poor oral hygiene

There is substantial evidence that poor oral hygiene, as expressed by median full-mouth plaque scores of ≥2, was highly associated with peri-implantitis with an odds ratio of 14.3 and a 95% confidence interval of 9.1–28.7 (26). Furthermore, a prospective study reported an association between poor oral hygiene and peri-implant bone loss after 10 years (59). A recent study identified inaccessibility for oral hygiene measures around implants in partially dentate patients as a risk for peri-implantitis (99).

History of periodontitis

As in cases of periodontitis, peri-implant infections may take years to develop. It is logical to assume that
the susceptibility to periodontitis, characterized by loss of attachment and alveolar bone loss, may translate to susceptibility to peri-implantitis. In recent years, eight systematic reviews have addressed a history of treated periodontitis as a risk indicator for implant outcomes (4, 43, 45, 74, 79, 94, 98, 108). Three studies reporting on the occurrence of peri-implantitis found a statistically significantly greater risk of peri-implantitis in patients with a history of treated periodontitis compared to those without a history of periodontitis (26, 44, 88). Reported odds ratios ranged from 3.1 to 4.7.

**Tobacco consumption**

Besides poor oral hygiene, tobacco consumption has been propagated as the second most important risk factor for disease progression for both teeth and oral implants. An association between smoking and peri-implantitis has been found in five cohort studies (31, 33, 48, 63, 88). A higher risk was found for smokers compared with nonsmokers, with odds ratios ranging from 3.6 to 4.6. Smoking has also been identified as an aggravating factor to poor oral hygiene (59); hence, all the studies mentioned above have adjusted for poor oral hygiene practices when evaluating the contribution of tobacco consumption.

**Diabetes mellitus and alcohol consumption**

There is limited evidence that diabetes mellitus and alcohol consumption are associated with peri-implant diseases, although diabetes mellitus has been associated with increased implant loss. Only one study in nonsmoking individuals with poor metabolic control has shown an increased risk for peri-implantitis (26). Similarly, only one study suggested that alcohol consumption of >10 g daily resulted in significantly greater marginal bone loss, even exceeding the amount identified in smokers (30).

**Genetic traits**

As identified in a systematic review on the role of the composite interleukin-1alpha and interleukin-1beta polymorphisms for the development of peri-implantitis has identified two retrospective cohort studies (25, 31) with conflicting and limited evidence (42). Hence, from a clinical point of view, the identification of genetic traits does not appear to be justified at this time.

**Similarities and dissimilarities with periodontal infections**

As both periodontitis and peri-implantitis are opportunistic infections with a similar etiology and similar clinical outcomes it is reasonable to assume that risk evaluation should follow similar pathways. In fact, the three major risk factors for periodontitis have also been shown to represent risk indicators for peri-implantitis. In addition, a history of periodontitis indicating susceptibility to this infection may be the most important risk factor for peri-implantitis.

**Diagnosis**

In determining the severity and extent of periodontitis, the diagnostic process encompasses at least (i) an assessment of the inflammatory changes and (ii) an assessment of the damage to the periodontal tissues, as expressed by probing depth and loss of clinical attachment. These probing measurements are highly sensitive, while a radiographic documentation of bone loss represents a specific test with which to document the sequelae of periodontitis. This disease may only be diagnosed properly if inflammation occurs concomitantly with pocket development (increasing probing depth). In the absence of inflammation and deepened pockets, loss of attachment represents a previous history of periodontitis or recession.

Because the pathogenesis of peri-implantitis closely resembles that of periodontitis, it is imperative to use the same diagnostic criteria for the detection of peri-implantitis and for monitoring the progression of lesions over time (50).

It has clearly been demonstrated that any disruption in the soft tissue–implant interface caused by probing the peri-implant sulcus will be followed by the formation of a new epithelial attachment within 5 days (23). This is also the case when probing the periodontal sulcus (106) (Figs. 5A–C). The application of a light probing force (0.2–0.3 N) will also provide reliable assessments of probing depth around implants (51, 97). In peri-implantitis, however, the
probe may penetrate into the connective tissue and only be stopped in its penetration by the alveolar bone (Figs. 6A–C).

In a maintenance program for the long-term care of implants, probing-depth measurements should be compared with baseline assessments of probing depth obtained after the placement of the reconstruction. In the light of the desirability of more apical placements of implants allowing for an optimal emergence profile of the crown, initial probing depth measurements may exceed the 3–4 mm depth usually encountered. Consequently, the development of disease will be associated with increasing probing depth from the baseline value. Nevertheless, a 6 mm peri-implant pocket has been found to be indicative of peri-implantitis (28).

In cases where there has been an increase in probing depth with the presence of bleeding on probing, supplemental radiographs should be obtained to further evaluate the progression of peri-implantitis. These will probably reveal an implant-specific saucer-shaped intraosseous lesion.
as opposed to the diagnostic site-specific characteristics, confirming probing measurements in periodontitis.

A very specific characteristic of implant failure, representing the total loss of osseointegration, is implant mobility. This highly specific parameter, however, is completely useless for the diagnosis of peri-implant mucositis or peri-implantitis.

A dynamic assessment of bone-to-implant contact reflecting implant stability would be desirable to assess the events during healing. Recently, noninvasive diagnostic methods have been advocated to specifically monitor the implant stability (8). Resonance frequency analysis is believed to yield the degree of bone-to-implant contact during the early phases of healing (64, 65). Consequently, resonance frequency analysis has been evaluated in relation to jaw bone characteristics and during early healing following implant installation (40). However, there was no evidence that resonance frequency analysis would reliably reveal loss of stability in advance of the clinically evident implant failure. When resonance frequency analysis records implant stability quotient values within the range of 57–70, implant stability can be assumed. No predictive value for losing implant stability could be attributed to resonance frequency analysis and hence the routine use of this diagnostic methodology may be questioned (40).

In conclusion, probing peri-implant sulci or pockets is an essential criterion for the diagnosis of peri-implant infections as is also true in cases of periodontitis. It is important that only light probing forces, preferably between 0.2 and 0.3 N, should be used. To monitor the inflammatory component for peri-implant mucositis and peri-implantitis, bleeding on probing, again using a light force, should be evaluated. Obviously the presence of pus represents the sign of ongoing infection that often accompanies the progression of peri-implantitis (89). As found in cases of periodontitis, loss of supporting bone, as revealed by radiographs, is a confirmatory test for the diagnosis of peri-implantitis.

**Therapy**

**Similarities with periodontal therapy**

Because the etiologies of periodontitis and peri-implant infections are almost identical it is obvious that all therapeutic approaches should be anti-infective. In the treatment of periodontitis, this consists of detailed instruction to improve oral hygiene practices concomitant with a thorough mechanical debridement of the contaminated root surfaces. This will result in a healing of the debrided wound area with a junctional epithelium closely adhering to the previously exposed root surface. The patient’s supragingival plaque control subsequently slows re-infection of the subgingival environment with pathogens. In conjunction with appropriate, professionally administered periodontal maintenance therapy, the clinical outcome of such treatment has been demonstrated to be excellent in the long-term (38, 58). In deep periodontal defects, nonsurgical mechanical debridement may result in both inadequate biofilm removal and pocket reduction. In such situations it may be necessary to obtain access to the contaminated root surface by reflecting mucoperiosteal flaps. Such access flaps may also be used in specific cases to attempt regeneration of periodontal tissues that have been destroyed by the periodontal infection. Similar therapeutic principles govern the treatment of peri-implantitis because poor oral hygiene is the most pertinent risk factor for the development of infection in the oral cavity. Instruction in oral hygiene practices represents a prerequisite for successful treatment outcomes. Owing to the fact that existing periodontitis lesions may act as reservoirs of pathogens to colonize implant surfaces, it is imperative that periodontitis be successfully treated and controlled, preferably before implant placement. However, if peri-implantitis is found in patients with untreated periodontitis, treatment of periodontitis must be given concurrently with the treatment of peri-implantitis. If satisfactory treatment outcomes are to be expected, mechanical debridement of contaminated root and implant surfaces must be performed. However, mechanical debridement is difficult to perform on implant surfaces because the use of steel curettes should be avoided in this situation (62). Furthermore, implant and supported reconstruction designs may often hinder access to the contaminated implant surfaces for thorough mechanical debridement. It may therefore be desirable to apply antimicrobial agents with proven efficacy, either to the peri-implant sulci and / or as a mouthrinse, to deplete the supragingival and supramucosal plaque reservoirs. However, only three controlled clinical trials have evaluated the efficacy of such combined mechanical and antiseptic debridement protocols (18, 24, 78). These measures of mechanical and adjunctive antiseptic debridement are, in most cases, sufficient to treat peri-implant mucositis.

Cases of peri-implantitis characterized by deepened probing depths, concomitant bone loss and
bleeding on probing cannot usually be treated by nonsurgical mechanical and antiseptic debridement alone. However, plaque control and submucosal debridement are a prerequisite before further steps are taken in the treatment of peri-implantitis (e.g., administration of systemic or local antibiotics and/or surgical debridement). In studies dealing with the treatment of peri-implantitis, desirable outcomes have included resolution of inflammation, reduction in probing depths and a substantial reduction in the number of putative pathogens. These satisfactory outcomes have been reported when systemic or local antibiotics are adjunctively used for the management of sites with deepened pockets of ≥6 mm (17, 67, 68, 86, 92) (Figs. 7A–E).

Owing to the lack of randomized controlled clinical trials, the benefits of antibiotics adjunctive to debridement, before or concomitant with surgical debridement, may be debated. However, the often rapid progression of peri-implantitis and the proposed differences observed in advancing peri-implantitis lesions compared with periodontitis lesions may justify the use of antibiotics directed against the recognized putative pathogens.

In a variety of situations, surgical access to the contaminated implant surface may be required. Because the main goal of the open-flap procedure is access to the contaminated surface, the typical saucer-shaped and deep peri-implantitis lesions can only be decontaminated effectively by surgical access (Figs. 8A–E). However, to date, only one study has evaluated the long-term (5 years) outcome of peri-implantitis treated with surgical access including the use of antibiotics (54). Out of the 27 implants with peri-implantitis placed in nine patients at baseline, 25% of the implants were lost in four patients during the follow-up period despite a significant reduction in the presence of plaque and mucosal bleeding. Four implants continued to lose bone, while in six sites bone formation was observed. Therefore, it appears that surgical treatment does not guarantee the long-term stability without re-infection and hence the outcomes should be considered unpredictable.
Surgical procedures are often advocated to gain access for regenerative procedures. However, there is no evidence to date that such procedures reveal additional benefits or secure treatment outcomes in the long term.

**Exception to the rule**

While periodontal treatment involves the debridement of contaminated tooth surfaces, treatment of peri-implantitis focuses on the decontamination of implant surfaces. As indicated above, the inability to access microbial habitats in the subgingival/submucosal region may often result in less than optimal treatment outcomes. Irrespective of surface roughness and configuration, decontamination of the titanium surface poses inherent problems and can probably not be achieved by mechanical debridement alone. It can be speculated that irrigation with antiseptic solutions and/or physiologic saline might dilute the bacterial load, thereby allowing innate and adaptive host responses to control the infection.

Fig. 8. Treatment of advanced peri-implantitis with anti-infective therapy in a high-risk patient. (A) Radiograph of three osseointegrated implants (Nobel Biocare®, TiUnite surface) replacing three missing teeth. The patient had a history of treated periodontitis and was a heavy smoker (20 cigarettes per day for more than 20 years). (B) Photograph illustrating clinical signs of peri-implantitis: bleeding on probing, suppuration and deep pockets (7–9 mm) 5 years after implant placement. (C) Radiograph confirming peri-implantitis, illustrating advanced peri-implant bone loss at two implants and moderate bone loss at the implant in the premolar region. Following an anti-infective treatment protocol including nonsurgical mechanical debridement and oral hygiene instructions, supplemented by application of chlorhexidine gel (0.5%) for 4 weeks and smoking-cessation counselling, inflammation and deep pockets were still present at re-evaluation. Consequently, access surgery was scheduled. (D) Circumferential intraosseous (saucer shaped) peri-implant bone defects following surgical access and decontamination of the implant surface using physiologic sterile saline. The patient was prescribed adjunctive systemic antibiotics [metronidazole (400 mg) and amoxicillin (500 mg) three times daily, for 1 week]. After surgery the patient rinsed with chlorhexidine mouthrinse (0.2%) twice daily for a 4-week period. Maintenance care was then provided every 3 months. (E) Resolution of the inflammation 6 months after treatment. Decreased probing depths and mucosal recession, and no bleeding or suppuration on probing, were observed.
Animal studies have demonstrated that there is no superiority of one decontamination protocol vs. another (95). Even the application of carbon dioxide laser has not been shown to be superior to simple decontamination with saline, when applying the microbiological dilution principle (76). It is apparent that the decontamination of the implant surface located either in a submucosal region or under surgical access represents the most pertinent challenge for predictable treatment outcomes.

**Summary and conclusions**

This review was undertaken to address the similarities and dissimilarities between the two disease entities of periodontitis and peri-implantitis. The overall analysis of the literature on the etiology and pathogenesis of periodontitis and peri-implantitis provided an impression that these two diseases have more similarities than differences. First, the initiation of the two diseases is dependent on the presence of a biofilm containing pathogens. While the microbiota associated with periodontitis is rich in gram-negative bacteria, a similar composition has been identified in peri-implant diseases. However, increasing evidence suggests that *S. aureus* may be an important pathogen in the initiation of some cases of peri-implantitis. Further research into the role of this gram-positive facultative coccus, and other putative pathogens, in the development of peri-implantitis is indicated. While the initial host response to the bacterial challenge in peri-implant mucositis appears to be identical to that encountered in gingivitis, persistent biofilm accumulation may elicit a more pronounced inflammatory response in peri-implant mucosal tissues than in the dentogingival unit. This may be a result of structural differences (such as vascularity and fibroblast-to-collagen ratios).

When periodontitis and peri-implantitis were produced experimentally by applying plaque-retaining ligatures, the progression of mucositis to peri-implantitis followed a very similar sequence of events as the development of gingivitis to periodontitis. However, some of the peri-implantitis lesions appeared to have periods of rapid progression, in which the infective lesion reached the alveolar bone marrow. It is therefore reasonable to assume that peri-implantitis in humans may also display periods of accelerated destruction that are more pronounced than that observed in cases of chronic periodontitis.

From a clinical point of view the identified and confirmed risk factors for periodontitis may be considered as identical to those for peri-implantitis. In addition, patients susceptible to periodontitis appear to be more susceptible to peri-implantitis than patients without a history of periodontitis.

As both periodontitis and peri-implantitis are opportunistic infections, their therapy must be anti-infective in nature. The same clinical principles apply to debridement of the lesions and the maintenance of an infection-free oral cavity. However, in daily practice, such principles may occasionally be difficult to apply in peri-implantitis treatment. Owing to implant surface characteristics and limited access to the microbial habitats, surgical access may be required more frequently, and at an earlier stage, in peri-implantitis treatment than in periodontal therapy.

In conclusion, it is evident that periodontitis and peri-implantitis are not fundamentally different from the perspectives of etiology, pathogenesis, risk assessment, diagnosis and therapy. Nevertheless, some difference in the host response to these two infections may explain the occasional rapid progression of peri-implantitis lesions. Consequently, a diagnosed peri-implantitis should be treated without delay.

**References**


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