Compendium

July/August 2013, Volume 34, Issue 8
Published by AEGIS Communications

Peri-Implant and Periodontal Tissues: A Review of Differences and Similarities

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ABSTRACT

The health and vitality of an osseointegrated implant depends on the surrounding supporting tissues, which not only anchor the implant to the bone but also have the important function of providing a protective seal. The aim of this article is to provide a basic understanding of differences and similarities between the periodontal and peri-implant tissues at the histologic, clinical, and immunologic levels; it is essential to know these differences and similarities during the clinical handling of these similar-looking tissues. The comparative features are of clinical relevance because it is critical to understand the behavior of the soft tissue found around the tooth and implant. This knowledge is vital from the preliminary stage of treatment planning through prosthetic rehabilitation.

The functional usefulness of oral implants lies in the fact that piercing the gingival or oral mucosa of the oral cavity establishes a transmucosal connection between the external environment and the inner dental implant. The term *osseointegration* refers to the direct bone contact to the alloplastic metallic implant. Titanium, which has excellent biocompatibility properties in physiologic conditions, has been the most commonly used biometal for implants.[1] The soft tissue around the implant, ie, peri-implant mucosa, is similar in many ways to the gingiva found around the tooth. The aim of this review article is to highlight the clinical, biochemical, histologic, and other features of the two similar-looking tissues—the gingival and peri-implant mucosa—which also exhibit several areas of dissimilarity.

Peri-Implant Mucosa

Peri-implant mucosa is composed of well-keratinized oral epithelium, sulcular epithelium, and junctional epithelium, as well as underlying connective tissue. Between the implant surface and the epithelial cells are hemidesmosomes and basal lamina.[2] The soft-tissue interface is made up of the epithelium and the underlying connective tissue, which includes the biologic zone known

as the “biologic width,” referring to the height of the dentogingival attachment apparatus encircling the tooth. The term “biologic width” is based on the work of Gargiulo et al, who described the dimensions of the dentogingival junction in human cadavers.[3] The average dimension of 2.04 mm (1.07 mm + 0.97 mm) is comprised of supra-alveolar connective tissue and junctional epithelial attachment. Functionally, in a clinical sense, there must not be any encroachment within 2 mm of the bone that surrounds the tooth. That a similar relationship of bone to overlying soft tissues exists around implants and changes in this relationship may be one of the reasons for the early crestal bone loss.[4]

The proliferative capacity of the junctional epithelium leads to the rapid migration of the epithelial cells as soon as the fibrin clot/granulation tissue starts forming at the implant installation. Once the cells reach the implant surface, their attachment occurs rapidly through the basal lamina and the hemidesmosomes.[5] Another possible attachment modality hypothesized is an indirect epithelium-to-implant contact.[6] The peri-implant sulcus shares the structural, ultrastructural, and functional characteristics with the gingival tissues. Human studies have demonstrated that epithelium surrounding dental implants possesses similar patterns of differentiation and function to gingival tissues.[7] The presence of granulation tissue adhering to the surface of the transmucosal components is considered the principal factor that stops the epithelium from migrating down apically.[8] Berglundh speculated that the reason the epithelium does not migrate down apically is likely due to the interaction between the titanium and the soft tissue.[9]

Connective tissue adhesion of the healing wounds involves the following: formation and adhesion of the fibrin clot to the implant surface; adsorption of the fibrin clot to the implant surface; adsorption of the extracellular matrix (ECM) proteins and connective tissue cells to the implant surface; transformation of the clot into granulation tissue; and migration of epithelial cells on top of the fibrin clot/granulation tissue.[10] The connective tissue zone next to the implant surface is primarily divided into two parts. The first is a 50-µm inner zone that is rich in fibers; resembling scar tissue, several scattered fibroblasts in close contact with the titanium surface maintain the seal between the peri-implant bone and the oral environment.[11,12] The remaining part of the connective tissue comprises fibers running in different directions, along with the cellular elements and blood vessels.[11] Connective tissue cells and the collagen fiber bundles are separated from the TiO2 surface with a 20-nm-wide proteoglycan layer.[13] Table 1 presents the salient features of similarity and dissimilarity between the peri-implant and periodontal tissues.[9,11,14-37]

Periodontal Probing

Periodontal probing is one of the basic diagnostic tools used to measure clinical attachment level (CAL), pocket depth, and width of the attached gingiva.[38] The probing depth is the distance between the gingival margin and the depth of the probe tip penetration into the pocket.[39] Increased probing depth and loss of clinical attachment are pathognomonic of periodontal disease.[40] Peri-implant probing provides an assessment of different parameters such as bleeding on probing, suppuration, and exudation from the sulcus and peri-implant tissues.[37] Studies have shown that, when used, probe pressure of 0.5 N penetrates an average of 0.7 mm deeper at implant sites.[41] Clinical probing depth is higher around implants versus teeth, as the probe tip

ends apically to the junctional epithelium into the connective tissue close to the bone crest.[42] Bleeding on probing is a more reliable sign of inflammation around a tooth but is less reliable around implants. Probing depth penetration around teeth has been found to be < 3 mm as opposed to 2.5 mm to 4 mm around implants.

Fiber Arrangement

In natural teeth, the nonkeratinized junctional epithelium attaches to the enamel surface via the internal basal lamina and desmosomes along the entire length of the junctional epithelium; however, the attachment of the peri-implant epithelium to the implant surface is confined to the apical region only. Fibers run a parallel course to the implant surface, as observed in human subjects.[17] Yet several other authors have found fibers to be running in different directions. A perpendicular direction was also found with implants harboring porous surfaces.[37,43] The orientation seems to be dependent on the quality of the mucosa, which tend to be parallel in alveolar mucosa and perpendicularly directed in keratinized mucosa. Fibers are perpendicularly inserted into the cementum around the tooth. Apart from the orientation of the fibers, there exists a significant difference between the connective tissue around the tooth and abutment. The dentogingival collagen fibers are firmly inserted into the cementum and the bone and in a perpendicular or oblique direction, thus serving as a barrier to the epithelial migration and the impending bacterial invasion.[44] The connective tissue adhesion with implants has a poor mechanical resistance as compared to the natural tooth.[25]

There appears to be a resilient connection between bone, periodontal ligament, and cementum around a tooth. However, a rigid connection appears in the form of functional ankylosis/osseointegration due to the lack of periodontal ligament around the implant. Absence of resiliency somewhat leads to the direct transmission of the loads to the bone–implant interface, and no compensatory tooth movements can accommodate the occlusal disharmony. The lack of periodontal ligament also precludes the use of implants in growing individuals. The highly sensitive receptors present within the periodontal ligament are responsible for the propioceptive and tactile sensitivity around the tooth. Absence of the periodontal ligament leads to reduced tactile sensation and reflex function around implants.[15] The adaptive capacity of the periodontal ligament allows the tooth movements, but the orthodontic movements cannot be undertaken with implants.

Inflammatory Response

Diagnostic criteria for detection of peri-implant health and for monitoring the progression of disease are similar to that for periodontal disease. The gingival/mucosal tissues constitute the primary defense mechanism against microbial infections. The conversion of the junctional epithelium to the pocket epithelium is considered to be the key to the progression of gingivitis/peri-mucositis to periodontitis/peri-implantitis. When performing visual inspection of peri-implant soft tissue, signs of disease include color alteration, swelling, thickness, and bleeding on probing, all clinical indices used for the evaluation of gingival disease. Inflammatory lesions may be present in the absence of the visual signs of inflammation. The peri-implant crevice is surgically created and is not developed as it is for natural teeth. Pocket depth is

determined by many factors such as abutment height, depth of fixture countersinking at stage 1 surgery, and the amount of tissue thinning during stage 2 surgery.[11] Structural differences between the peri-implant and periodontal tissue dictate the probing pattern around the implants as well. The parallel disposition of the collagen fibers to the implant surface and the absence of the connective tissue insertion cause the probe to go beyond the epithelial seal and results in injury to the connective tissue.[45]

Sulcular fluid around the gingiva is called gingival crevicular fluid (GCF), and that which is around the implant is known as peri-implant sulcus fluid (PISF). Gingival crevicular fluid is a healthy serum transudate in a healthy free gingiva, and during inflammation GCF is converted into an inflammatory exudate originating from the vessels of the gingival plexus and is recognized as apart of the gingival defense system. GCF is rich in leucocytes, especially polymorphonuclear leukocytes (PMN), and is attracted by a chemotactic gradient of bacterial or host origin. It is also rich in host-derived molecules from blood as well as substances from microorganisms of the dental plaque. The GCF flow requires permeability-induced initiators of inflammation. About 65 different infection-induced enzymes and their inhibitors and regulators have been found.[32]

PISF is an inflammatory exudate originating from the vessels of the gingival plexus and is similar to GCF. It contains the host-derived enzymes and their inhibitors and host-response modifiers and some tissue breakdown products. PISF volume, along with the increased enzymatic activity, has been suggested to be elevated during inflammation, which confirms the diagnostic potential of PISF in peri-implant inflammation. Table 2 summarizes the gingival crevicular fluid and peri-implant sulcular fluid components.[32]

GCF functions to continuously flush the dentogingival crevice and release antimicrobial components of serum such as antibodies and complement enzymes. In disease, the crevicular fluid flow increases 30 times more than in health.

The biologic inflammatory response of the tissues around the tooth and implant depend largely on their histologic framework. Implants contain a dense network of collagen fibers that originate from the alveolar bone crest to the gingival margin in a parallel fashion, in contrast to teeth, where the fibers are perpendicular. The fibers in implants appear very large and follow a circular arrangement around the implant neck. Fiber-to-metal surface contact has generally not been observed. There are studies, however, that have observed direct fibrous attachment to the titanium surface.[25] The length of the supra-alveolar connective tissue in implants is also significantly larger than that of teeth. Teeth have multiple collagen bundle fibers that run in various directions to various adjacent structures. Studies based on the response of the tooth and implants to experimental breakdown have revealed the differences in the nature of the breakdown. Ligature-assisted subgingival plaque formation inducing the periodontal and peri-implant lesion formation in beagle dogs revealed more pronounced clinical and radiographic signs of destruction around implants than teeth. Furthermore, the size of the soft-tissue lesion was found to be larger around the implants than teeth and extended into the bone marrow.[33] Yet another study described the host-response results of longstanding plaque and gingivitis and revealed an inflammatory cell infiltrate that extended more apical into the peri-implant mucosa (~ 1.5 mm) than the gingiva (~ 0.9 mm).[24]

Histomorphometric studies have revealed that implants and teeth have a comparable ratio of collagen, vessels, and plasma cells, whereas implants have lower proportions of lymphocytes, macrophages, and PMNs. Hence, implants render a weak biologic barrier to prevent the apical migration of inflammatory cells compared to teeth.[21] Revealing results were demonstrated in a study to show the effects of the microbial response on the inflammatory markers myeloperoxidase (MPO) and nitrite in healthy and diseased teeth and implant sites. MPO and nitrite levels were stable in the GCF at healthy and diseased sites, whereas the PISF levels were found to be raised with MPO and nitrite levels.[46]

Biofilm

When exposed in the oral cavity through the transmucosal abutment, an osseointegrated implant provides a favorable surface for bacterial colonization. This further leads to the selective adsorption of the salivary proteins, peptides, etc, and rapid formation of the pellicle.[24] Biofilm formation around the implant is similar to that formed around teeth.[47] The composition of the pellicle formed around implants lacks the low molecular mucins commonly found on the enamel in natural teeth, which may lead to the qualitative difference in the early biofilm formation around the implant. This possibly contributes to the low plaque formation around implants.[48] However, these differences do not seem to influence the bacterial composition of the early biofilms formed on the implant surface. Biofilm formation on the implant is influenced by the properties of the surface to be colonized, including chemical composition, surface roughness, and surface free energy.[49]

Many studies have pointed out the comparative rates and the composition of the microbiota associated with health and disease as associated with teeth and implants.[50-52] Classic differences in the microbial profile of the peri-implant flora in certain in-vitro studies reveal an affinity of *Staphylococcus aureus* for the titanium surface, but it is not a common microflora around teeth.[30] A host response to the bacterial challenge is known to develop irrespective of the implant system,[53] while the initial host response to the bacterial challenge in the peri-implant mucosa is similar to that found in the gingiva. However, the longstanding inflammation does have a more pronounced response in the peri-implant tissues than in gingiva, leading to the significant apical extension of the inflammatory infiltrate in the mucosa and increased size of the lesion as compared to the gingival.[47] The Sixth European Workshop on Periodontology 2008 defines peri-implant diseases as inflammatory lesions that develop in the tissues around the implant and consisting of two entities: peri-implant mucositis and peri-implantitis. Peri-implant mucositis is the reversible inflammatory reaction in the soft tissues surrounding the functioning implant, whereas peri-implantitis is defined as the inflammation in the mucosa characterized by loss of supporting bone around an implant in function.[54]

Histopathologic data of the human case series have described the inflammatory lesions as comprising the B cells and plasma cells, suggesting that peri-implantitis and periodontitis lesions are similar.[55,56] Despite the fact that peri-implantitis and periodontitis develop similarly, the dynamics of this process could be different. Because the periodontitis lesion is walled off by the intact supracrestal connective tissue fiber compartment, the penetration of the infiltrates into the bone marrow is generally not evident. However, because the peri-implantitis lesion progresses without the presence of the connective tissue compartment, it often progresses rapidly into the

marrow.[46,57] Periodontitis and peri-implantitis share common risk factors, such as poor oral hygiene, tobacco consumption, and diabetes mellitus. Cross-sectional analyses have evaluated the risk indicators for peri-implantitis to be poor oral hygiene, history of periodontitis, tobacco consumption, diabetes mellitus, alcohol consumption, and genetic traits.[58]

Microflora Around Implants

The microbiota on implants in edentulous and partially edentulous patients and in patients with a history of periodontal disease varies. Studies have stated that the microbiota obtained from colonizing clinically healthy implant fixtures in fully edentulous subjects are similar to the microbiota associated with healthy periodontal sites in periodontally healthy subjects.[59] It was suggested that extraction of all teeth results in elimination of the *Porphromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* from the oral microbiota.[60]

In partially edentulous subjects the developing microbiota around implants is similar to that of naturally occurring teeth.[61] This microflora—85% of which are identified as gram-positive cocci—inhabit immediately after installation of the implant. Microbial colonization and the ensuing inflammatory reaction in the peri-implant tissues might be analogous to the key events in the pathogenesis of periodontitis. The literature comparing the microbiota around implants in fully edentulous and partially edentulous mouths stated a higher percentage and frequency of the black pigment bacteroids, fewer coccoids and motile rods, and high frequency of the *P gingivalis* and *P intermedia* on implant surfaces in partially edentulous subjects.[62,63] The microbiota of the remaining teeth serve as the primary source of the putative pathogens. This reveals that the microbial state of the remaining teeth influences the fate of the newly incorporated implants.[64] Microbiota on implants in subjects with a history of periodontal disease are similar in nature to those found in the periodontal pockets around teeth.[65] It would seem likely that susceptibility to periodontitis may translate to peri-implantitis. Several reviews have reported a history of treated periodontitis as a risk indicator for implant outcomes with statistically significant results.[66-68]

Healing

The healing response of the tissues around implants varies from that of the natural tooth.[69] Implants exhibit a poor vascular supply compared to teeth. Following implant insertion, tissue repair requires development of the vasculature at the site of injury for the delivery of oxygen and nutrients and the removal of cell debris for a complete healing process.[70] Berglundh reported that implants placed following flap elevation resulted in a poor vascular supply between the junctional epithelium and marginal bone.[9] Ericcson explained that the poor vascular supply in the peri-implant mucosa may be the reason for the extensive progression of plaque-associated inflammation.[24]

Mucoperiosteal flap elevation results in postoperative bone resorption.[71] The amount of the tissue injury is known to influence the speed and quality of healing.[72] Small, clean, and closed wounds heal more quickly with little scar formation, whereas large wounds heal slowly and with significant scarring. Flapless surgery perhaps has the added benefit of providing a better vascular supply and retaining the vitality of the supracrestal connective tissue around the implant

compared to raised soft-tissue flaps, which may inadvertently lead to cutting and damaging the supraperiosteal vessels, which apparently are the sole source of blood supply around implants.[22] The surrounding mucosa after the flapless procedure has smaller, cleaner, and more closed wounds than the flap procedure. The flapless procedure preserves the connective tissue vascularization when no flaps are reflected. The supracrestal connective tissue lateral to the implant surface is supplied by the branches of the supraperiosteal vessels. When soft-tissue flaps are reflected, the branches of the supraperiosteal vessels can be cut or damaged.

Due to the similar etiologies for periodontitis and peri-implant infections, the therapeutic approach also appears to be similar—ie, anti-infective. Periodontal treatment and the long-term results are promising, as compared to implants.[73] Since existing periodontal lesions can become a reservoir of pathogens to colonize implant surface, it is imperative that the periodontitis be successfully treated and controlled before implant placement. Periodontal treatment involves the debridement of the contaminated root surfaces, and the treatment of peri-implantitis focuses on the decontamination of the implant surface. Despite the surface roughness and configuration, decontamination of the titanium surface poses inherent problems and can probably not be achieved by debridement alone. Animal studies have demonstrated that there is no superiority of one decontamination protocol versus the other.[74]

Conclusion

Periodontitis is the main cause of tooth loss in adults. Hence, it is assumed that a greater number of patients receiving implants have a history of periodontal disease. As in cases of periodontitis, peri-implant infections take several years to develop. The pathogenesis of the two diseases depends on the presence of biofilm-containing pathogens. The microbiota in periodontitis primarily comprises gram-negative bacteria; however, increasing evidence suggests that *S aureus* may be an important pathogen in the initiation of some cases of peri-implantitis.[53] The subgingival microflora is initially derived from the supragingival plaque and becomes more anaerobic as the oxygen pressure is reduced. Microbiota associated with both healthy and failing implants are similar to those observed in both healthy and diseased periodontium around teeth. The indigenous oral bacteria on the remaining teeth serve as the reservoir for colonization on the implant surface, which explains the similarity of the biofilm composition around the teeth and the implant in the same individual.[65]

A more pronounced inflammatory response is expressed in peri-implant mucosal tissues than in the dentogingival unit because of the structural differences of the tissues (vascularity and fibroblast-to-collagen ratios). Periodontal probing is one of the diagnostic tools used to measure pocket depth, clinical attachment level, gingival marginal location, and the width of gingiva. Peri-implant probing depth seems to be related to the thickness and type of the mucosa circumscribing the abutment. Alveolar mucosa is generally associated with deeper probing depths, whereas a keratinized collar around the abutment is usually accompanied by shallower depths.[75] The pocket depth around an implant is determined by several factors: abutment height, depth of the fixture countersinking at stage 1 surgery, and amount of the tissue thinning at the stage 2 surgical procedure.[11] The surface texture irregularities, shape of the implant, and configuration of the restoration may influence the path of the probe tip.[76] Peri-implant probing is

more sensitive to force variation than periodontal probing. Conventional probing of 0.25 N does not damage the peri-implant tissues.[58]

Decontamination of the implant surface located either in a submucosal or under surgical access represents the most pertinent challenge for the predictable treatment outcomes.[35] Substantial literature correlates the opportunistic infective nature of periodontitis and peri-implantitis and the possible similarities and dissimilarities.[77] Hence, future studies should aim at nonsurgical modalities for treatment of peri-implantitis, ie, decontamination, antibiotics, etc. The probable association between the risk indicators needs to be explored further.

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TABLE 1

Similarities and Dissimilarities Between Peri-Implant and Periodontal Tissues

Tooth	Implant
In health	
Soft tissue around tooth called gingiva	Soft tissue called peri-implant mucosa
In disease	
Gingivitis = when limited to soft tissue	Peri-implant mucositis = when limited to soft tissue
Periodontitis = when extending to alveolar bone	Peri-implantitis = when extending to alveolar bone[14]
Sulcular fluid	
Gingival crevicular fluid (GCF)	Peri-implant sulcular fluid (PISF)
Histologic	
Soft-tissue interface	
1. Gingival fibers insert perpendicular into the cementum[7,15]	1. Gingival fibers run parallel to implant collar and are not inserted into implant surface[8,9]
2. Junctional epithelium adherent[16]; less permeable high, regenerative capacity[17]	2. Junctional epithelium poorly adherent[16]; more permeable, low regenerative capacity[17]
3. Periodontal ligament and cementum present	3. Periodontal ligament and cementum absent
4. Tooth not in direct contact with bone[17]	4. Implant directly in contact with bone[17]
5. Complete attachment of junctional epithelium to the enamel[16]	5. Junctional epithelium attached to the enamel only at apical region[18]
6. Junctional epithelium comprises basal lamina and hemidesmosomes and originates from the reduced enamel epithelium[19]	6. Junctional epithelium comprises basal lamina and hemidesmosomes and originates from the oral epithelium[19]
7. Gingival sulcus apically limited with junctional epithelium[17]	7. Gingival sulcus apically limited with junctional epithelium[17]
Hard-tissue interface	
Resilient connection; bone–periodontal ligament–cementum	Rigid connection; osseointegration; cementum and periodontal ligament absent
Cellular and molecular	
1. Collagen 1, 3, 4, 6, 7 and fibronectin present, collagen 5 decreased[20]	1. Collagen 1, 3, 4, 7 present, collagen 5 abundant, collagen 6 absent[20]
2. Low collagen and high fibroblast ratio[9]	2. High collagen and low fibroblast ratio[9]
Vascular response	
1. Vascular response increase[21]	1. Vascular response decrease[21]
2. Supraperiosteal, vascular plexus of the periodontal ligament[22]	2. Supraperiosteal only[22]
Biologic width formation	
Establishes after complete eruption of the tooth	Starts forming at time of abutment connection (stage 2),[9] at time of implant placement (stage 1)[23] Dimensions similar to tooth if implant abutment junction (IAJ) is supracrestal (one-stage implant/nonsubmerged), increased dimension if IAJ is subcrestal (two-stage/submerged)[23]
Response to biofilm	
Rate of plaque formation high, due to increased albumin adsorption capacity of pellicle	Rate of plaque formation low, possibly due to poor albumin adsorption capacity of pellicle around implants[24]
Response to healing and mechanical forces	
Structural composition enhances the reparative and healing capacity of the gingival tissue; good mechanical resistance[25]	Poor reparative and healing ability of the peri-implant tissues[9]; poor mechanical resistance[25]
Nature of healing	
Long junctional epithelial healing/regenerative procedures facilitated with GTR technique	Functional ankylosis/osseointegration at bone level and transmucosal seal formation at soft-tissue level (one-stage/two-stage)[26]; regenerative procedures facilitated with GBR
Microbiology and immunology	
1. Microbial species different in the supra and subgingival environment around teeth[27]	1. Supra and submucosal composition of the microflora similar around implant[28]
2. Spirochetes and *Staphylococcus aureus* not a usual associated microorganism[29]	2. Spirochetes and *Staphylococcus aureus* closely associated with peri-implantitis[30,31]
3. Pathways of destruction lead to a slow progression of the disease in the periodontal tissues[32]	3. Rapid progression of the disease[32]
4. Extent of pathologic lesion limited, within 1 mm of healthy connective tissue[33]	4. Extent of pathologic lesion extensive,[33] extending beyond connective tissue into bone marrow as far as 1.2 mm; neutrophil count high in peri-implant lesion, epithelial lining absent between the lesion and implant biofilm
Risk factors for periodontitis	***Risk factors for peri-implantitis***
Poor oral hygiene, gingivitis, tobacco consumption, diabetes[34]	Poor oral hygiene, history of periodontitis, tobacco smoking, diabetes mellitus, alcohol consumption[35]
Response to external stimuli	
1. Periodontal tactile sensation[32]	1. Reduced tactile sensitivity and reflex function[32]; phenomenon of osseoperception exhibited by implants; tactile sensitivity exhibited by endosseous implants stimulated by surrounding bone and periosteal neural endings
2. Periodontal ligament can allow tooth movements with its adaptive capacity	2. No adaptive capacity and no movements exhibited due to lack of periodontal ligament. Cannot be placed in growing individuals[36]
Clinical	
1. Gingival sulcus develops naturally as tooth erupts	1. Peri-implant sulcus surgically created and is dependent on multiple variables such as abutment height, depth of fixture countersinking at stage 1 surgery, amount of tissue thinning at stage 2 surgery[11]
2. Bleeding on probing is reliable indicator of inflammatory sign[26]	2. Bleeding on probing not reliable indicator[23]
3. Probing depth is shallow, < 3 mm[26]	3. Probing depth is deeper, 2.5 mm to 4 mm[37]
4. Biologic width is supracrestal, ~ 2 mm[37]	4. Biologic width is subcrestal, 2.5 mm to 4 mm[37]
Therapy	
Periodontal treatment deals with decontamination of tooth surface; comparatively promising results[38]	Implant treatment deals with decontamination of implant surface; challenging outcomes[38]

TABLE 2

Summary of GCF/PISF Components[32]

Host-Derived Enzymes and Their Inhibitors	
Elastase and elastase inhibitors	alfa 2-macroglobulin, alfa 1-proteinase inhibitor
Trypsin-like enzymes	
Collagenases	MMP-1, -8, -13
Gelatinases	MMP-2, -9
Tissue inhibitors of MMPs	TIMP-1
Stromyelysins	MMP-3, -10, -11
Myeloperoxidase	

Inflammatory Mediators and Host Modifiers	
Cytokines	Interleukins, tumor necrosis factor, interferon alfa
Antibacterial antibodies	IgGs, IgM, IgA
Substance P	
Prostaglandin E2	
Acute-phase proteins	Lactoferrin, transferrin, C-reactive protein
Leukotriene B4	

Tissue Breakdown Products	
Connective tissue and bone proteins	laminin, osteonectin, osteocalcin, type I collagen peptideses, haemoglobin beta-chain peptides
Glycosaminoglycans	Hyaluronic acid, chondroitin sulfates, dermatan sulphate, hydroxyproline, fibronectin fragments