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ORIGINAL ARTICLE

INTER RELATIONSHIP BETWEEN THE IMPLANT AND BIOFILM BASED ON THEIR MICROBIAL LINKAGE

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ABSTRACT:
Mouth provides a congenial environment for the growth of the microorganisms as compared to any other part of the human body by exhibiting an ideal nonshedding surface. Dental plaque happens to be a diverse community of the microorganisms found on the tooth surface. Periodontal disease and the peri-implant disease are specific infections that are originating from these resident microbial species when the balance between the host and the microbial pathogenicity gets disrupted. This review discusses the biofilms in relation to the peri-implant region, factors affecting its presence, and the associated treatment to manage this complex microbial colony. Search Methodology: Electronic search of the medline was done with the search words: Implants and biofilms/dental biofilm formation/microbiology at implant abutment interface/surface free energy/roughness and implant, periimplantitis/local drug delivery and dental implant. Hand search across the journals - clinical oral implant research, implant dentistry, journal of dental research, international journal of oral implantology, journal of prosthetic dentistry, periodntology 2000, journal of periodontology were performed. The articles included in the review comprised of in vivo studies, in vivo (animal and human) studies, abstracts, review articles.
Key Words: Biofilms, dental implants, peri-implantitis, plaque microbiology

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INTRODUCTION

Biofilm is described as relatively undefinable microbial community associated with tooth surface or any hard nonshedding material. [1] Biofilms are ubiquitous and they form on virtually all surfaces immersed in natural aqueous environment, e.g., water pipes, living tissue, tooth surface, implanted medical devices, dental implants, etc., Biofilm adhesion-mediated infections most commonly seen are on the implanted heart valves, venous catheters, vascular prosthesis, fracture fixation devices, breast implants, intraocular lenses, and dental implants. [2] Biofilms consist of one or more communities of microorganisms nonrandomly distributed in a glycocalyx. These biofilms allow the microorganisms to stick and multiply on the surfaces. The interactions among the various bacterial species residing and growing in the biofilm takes place by metabolic exchange, physical contact, exchange of genetic information, signaling molecule-mediated information. [3]

Biofilms formed on the tooth surface is called as dental plaque. Bacteria proliferating in the dental plaque form the main etiologic factors for the majority of the dental ailments, e.g., caries, gingivitis, periodontitis, and periimplantitis. Microbial attack has been cited as the main cause of the dental implant failure. [4] Biofilms are responsible for their association of about 65% of diseases including peri-implantitis and periodontitis. [5]The review addresses the pathogenesis, factors affecting implant biofilm, and the treatment associated.

BIOFILM AND TOOTH

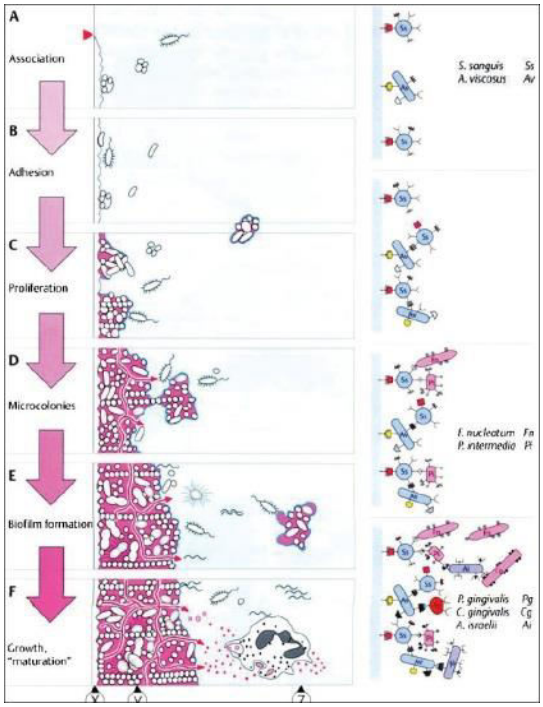
The formation of the microbial complex called biofilm in the oral cavity is a multistage journey. [6] Saliva provides the major source of nutrients to the bacteria. The thin film covering the tooth called as acquired pellicle is derived from the salivary proteins and covers the enamel within seconds after brushing. Proteins and the glycoproteins are the molecules binding to the tooth surface, implants, restorations, etc., These molecules primarily act to promote the adhesion and coaggregation of the oral bacteria. The bacterial adherence to the pellicle is facilitated by the special surface molecules (adhesins) chiefly lectins present

on the bacterial cell surface. Further intercellular bacterial adhesion and secretion of the extracellular polysaccharides, e.g., levans, dextrans, form the multilayered bacterial colonies suspended in the polymer matrix. Slow aggregation of the bacterial colonies leads to the formation of the multilayered cell clusters in the polymer matrix. [7] The microbial load in the saliva constitutes about 10 [7] bacteria per milliliter. [8] The bacterial cells colonize on the tooth surface within 4 hours of the pellicle formation. The initial colonizers being the *Streptococci* (*S. viridens, S. mitis, S. oralis*). The planktonik bacteria that are unable to bind directly to the tooth surface take the aid of their receptors to bind to the cell surfaces of the initial colonizing bacteria and finally on to the tooth surface. This bacterial cell to cell reaction that occurs or the coaggregation happens to be an important mechanism leading to the bacterial colonization and dental biofilm formation. Secondary colonizers bind to the bacteria predominantly comprising of the *Actinomyces* species, *S. mutans, S. sobrinus*. The bacteria multiply and co aggregate with the partner species. *Fusobacterium nucleatum* has the property to co-aggregate with multiple bacteria hence this species is an important link in the dental biofilms bridging the early and the late colonizers. [9] The oral bacteria thrive for their nutrient supply from saliva, gingival crevicular fluid, sugar rich food metabolic products of other bacteria, and food debris. The mature plaque has an inherent "circulatory system." The plaque begins to behave as a complex microorganism. Metabolic products and evulsed cell wall constituents (lipopolysaccharides, vesicles) activate the host response [Figure 1]. Specialized cell-cell communication is exhibited by the bacteria that coordinate the gene expression. This communication is passed on as signals. Bacteria sense the changes in the local environment (cues) and receive the information of the adjacent population. Specific interspecies communication within the biofilms is mediated through the metabolic exchange, genetic exchange, and the quorom sensing. [10] Quorum sensing is genetically governed chemical communication among bacteria in response to cell density and influence several functions of the bacteria, e.g., virulence, acid tolerance, and the biofilm formation. Two specific signaling molecules have been produced by the oral bacteria. Gram-positive bacteria communicate via small diffusible pepitide channel called as "Competence Stimulating Peptides (CST) and AI-2." AI-2 (autoinducer-2) is a popular signaling molecule exhibited by both gram-positive and gram-negative bacteria responsible for the quorum sensing. [9],[11] The biofilm acts as a barrier for the bacteria against host immunity and the antimicrobial agents. The anaerobic microflora succeeds to occupy the subgingival environment gradually as the plaque starts maturing. Supragingival plaque sets up the stage for the disease process of gingivitis and the subgingival microbial colonies advance the gingivitis to an established form of periodontitis.

Figure 1: Stages of biofilm formation (ref Wolfe HF. Biofilm plaque formation on tooth and root surfaces. In: Wolfe, H.F. Rateitschak, K.H. (eds). Periodontology, ed 3. Stuttgart: Thieme 2005; 24)

BIOFILM AND IMPLANT

Microbiologic evidence of the first human biofilm-related peri-implant infection comes from the study on plaque samples collected from apical most part of 17 diseased



implants. Implants with deeper probing pockets showed a presence of lesser number of coccoid and more levels of the spirochetes. [12] Biofilm formation on dental implants and the teeth follow the similar pattern of microbial colonization. [13] Biofilm formation around natural teeth occurs in minutes and the specific species start colonizing as early as 2-6 hours. The reason attributed possibly lies in the fact that the clean tooth surfaces are likely to have remnants of unattached microbiota that can immediately multiply and provide a favorable surface for the attachment of the late colonizers. [14] The pristine surfaces of the implants lack the desired indigenous microbiota and demand the early colonizers to set the stage for the complex communities to develop. [15] The pellicle starts forming on the implant surface as early as 30 minutes after the implant is exposed in the oral cavity. [16] The acquired pellicle on the dental implants owing to their lower albumin absorption capacity causes a low plaque formation around implants. Early colonizers are predominantly the gram-positive cocci, rods, and actinomyces species. [17] The periodontal pathogens colonizing on the *Streptococci* (*P. gingivalis, P. intermedia,* etc) are the causative microorganisms responsible for peri-implantitis and periodontitis [Table 1]. [25]

Author and Reference	Study design	Type of surface	Surface parameter	Studied results
Quireynen *et al.*,[18] abutments FEP abutments	*In vivo*	Ti and FEP coated abutments	SFE of the abutment material	CFU count of titanium abutments 5 times higher than
Rimondini *et al.*,[19]	*In vivo*	Ti (Ra 0.088-1.02)	Surface roughness	Ra<0.08 inhibits biofilm formation
Grossner *et al.*,[20]	*In vitro*	Ti surface treated with	Surface modified Ti	Surface coating of TiN/ZrN reduces the bacterial adhesion versus the polished Ti
Scarano *et al.*,[21]	*In vivo*	Ti surface (control) Zirconia surface (test)	Extent of bacterial adhesion on the surfaces with similar reduction of adhesion *P*=0.00001 as Ra (roughness average)	Compared to Ti surface
Burgers *et al.*,[22]	*In vivo* & *in vitro*	Machined (smooth) Surface roughness (Ra)	High bacterial adhesion around acid etched (rough) and surface free energy (SFE)	Treated surface (rough) than machined
Bollen *et al.*,[23]	*In vivo*	Ti machined (Ra=0.2)	Surface roughness reduced surface roughness (<0.2) and ceramic abutment has no impact on the supra/Ra=0.06)(highly polished)	Subgingival microbial colony
Quireynen *et al.*,[24]	*In vivo*	Ti abutments of varying	Surface roughness Decreased Ra (<0.02) has no surface roughness qualitative/quantitative	Effect on the microbial colonization

TiN – Titanium nitride; Ra – Roughness average; SFE – Surface free energy; Zr – Zirconia nitride

Table 1: Study data on the effect of dental implant surface properties on biofilm formation

The osseointegration around the dental implant is largely influenced by its surface roughness. [30] However, greater is the surface roughness, higher is the rate of the biofilm formation around the implant. [27] The attachment of the microorganisms to the hard surfaces, i.e., teeth and implants, besides their interactions with the surface components (roughness) also require certain specific characteristics of these interacting surfaces in terms of their wettability/hydrophobicity and surface free energy (SFE). In an *in vivo,* study a smooth titanium abutment and a sandblasted titanium surface was evaluated for the biofilm accumulation. The results revealed that surface roughening harbored lower percentage of the coccoid cells (64.2%) as compared to the smooth abutments (81%) [28] In yet another previous study, Quireynen in a 96 hour supragingival plaque formation reported a positive relationship between the surface roughness and the plaque growth rate and pathogenicity. [29] These studies highlight an important fact that the surface roughness has a significant contribution for the increased plaque buildup. The bacterial adhesion initially has a weak and reversible binding to the surface before the final irreversible attachment occurs. The possible explanation of the initial weak reversible attachment of the bacteria on the rough surface is attributed to the fact that the bacteria indirectly get a protection against the mechanical shear. An SEM results for an *in vitro* study on the attachment of the microbes to the different surface morphologies of the titanium discs (grooved, smooth, and rough) revealed a significant bacterial attachment to the rough titanium surface [Table 2]. [34] SFE/wettability of the surface influences the formation of the biofilm. SFE is defined as the interaction between the forces of adhesion and the forces of cohesion that determine the property of wetting, i.e., spreading of the liquid over the surface. [35] An *in vivo* study was undertaken on the supra and subgingival microbial plaque samples in patients with implant-supported fixed prosthesis.

Author and reference	Study design	Results	Microbial flora studied
Do Nascimento *et al.*,[30]	*In vitro*	Presence of the bacterial growth irrespective of the type of abutment (cast/ premachined	*Fusobacterium nucleatum*
Keller *et al.*,[31]	*In vitro*	Cement retained abutments exhibit decreased bacterial permeability, Spirochetes as compared to screw retained	*Prevotella intermedia, P gingivalis, Fusobacterium*
Piatelli *et al.*,[32]	*In vitro*	Cement retained abutments exhibit decreased bacterial permeability as compared to screw retained	-
Teixeira *et al.*,[33]	*In vitro*	Microbial penetration of the *S. aureus* in both the morse taper and internal hexagon implant abutment connection	*Staphylococcus aureus*

Table 2: Studies (*in vitro* and *in vivo* data) on the microbiology in relation to the dental implants

Two-stage abutments titanium versus coated abutment (Flouroethylene propylene abutment) were studied. A 3 month microbial analysis (phase contrast microscopy, DNA analysis, and colony forming unit (CFU), respectively) for the supragingival and subgingival plaque revealed increased microbial count around FEP-coated abutments. A predominant population of cocci was evident around FEP abutment, whereas the titanium abutments harbored more of spirochetes. CFU count was higher on the titanium abutments than the FEP abutments. The results of the study revealed that SFE of the implant and the abutment material have a vital role in the colonization of the bacteria. [18] An *in vivo* study done on the titanium discs for evaluation of the effect of the surface roughness and the microbial colonization concluded that a titanium surface with a roughness average Ra <0.088 inhibits the colonization and maturation of the plaque. [19] *In vitro* study comparing the Ti discs coated with TiN/ZrN (test) with the polished Ti (control) showed decreased bacterial adhesion in the test group. [20] Convincing results were also seen in an *in vivo* study that aimed at investigating the extent of

bacterial adhesion on the two surfaces with similar surface roughness. The conclusion drawn was that zirconium oxide surface has a low bacterial colonization potential than the titanium oxide surface. [21] Burgers *et al*. reported a twofold *in vivo* and *in vitro* study that aimed at bacterial adhesion on the different textured implant surfaces. In this study, machined and acid-etched titanium specimens (Ra 0.15 and 0.95, respectively) were worn for 12 hours by the subjects. The study aimed to investigate the bacterial growth of *S. sanguinis* after being incubated in the microbial suspension. The microbial growth was observed with fluorescent techniques. Results revealed a higher bacterial adhesion on the acid etched surface. [22] Surface roughness Ra >0.2 μm leads to increased rate of the biofilm formation and hence acts as the main etiology behind the peri-implant breakdown. [23] However, Ra <0.2 μm has no impact on the supra and subgingival plaque formation. [24] It has further been explored and reported that Ra <0.02 μm has further no quantitative or qualitative effect on the nature of the microflora [36] [Table 3].

Ra	Effect on biofilm
= 0.2 μm	Threshold value
>0.2 μm	Increased biofilm
< 0.2 μm	No qualitative/quantitative change in biofilm

Ra – Surface roughness

Table 3: Effect of Ra of implant and the biofilm formed[23],[24],[36]

MICROBIOLOGY OF THE BIOFILM AROUND IMPLANTS

A majority of the studies have pointed out the comparative rates and the composition of the microbiota associated with teeth and implants in health and disease. The microbiota in healthy peri-implant tissues is dominated with gram positive facultative cocci and rods. [37] A classic difference in the microbial profile of the peri-implant microflora in certain *in vitro* studies reveals affinity of the *Staphylococcus aureus* for the titanium surface but it isn't a common microflora around the teeth.

S. aureus has high adhesion for titanium surfaces [38] and has been associated with bleeding on probing and suppuration. [39],[40] Several specific adhesins are expressed on the surface of *S. aureus* that interact with a number of host proteins such as fibrinogen, fibronectin, collagen vironectin, and laminin. These surface adhesions have been referred to as microbial surface components recognizing adhesive matrix molecules (MSCRAMMS). After the placement of the implant, they are coated with the host plasma constituents including extracellular matrix (ECM). The fate of the implant/biomaterial surface may be conceptualized as "race for the surface" involving ECM, host cells and the bacteria. The adhesion mechanism of the *S. aureus* facilitates their adhesion to the biomaterials and the ECM deposited on the implant surface. [41] Transition from health to disease (peri-implantitis) causes a shift of the microflora from predominantly gram-positive to gram-negative microorganisms. Microflora of the implant in peri-implantitishave a high prevalence of the red and orange complex species as defined by Socransky. [42] This microflora is predominated with the red complex species as *P. gingivalis, T. forsythia, and T. denticiola*, the orange complex species as *F. nucleatum, P. intermedia. Candida albicans* has been found to have increased adhesion to titanium implants in certain *in vitro* studies. [43]

BIOFILM AT THE IMPLANT - ABUTMENT INTERFACE

The two-piece implant consists of an implant abutment junction (IAJ).There is a joint/gap between the implant and abutment referred as the "microgap." The histologic aspects of this microgap were studied by Ericsson *et al*., who identified two important microbiologic entities in the implant crestal region: (a) Plaque-associated inflammatory cell infiltrate (PaICT) and (b) implant-associated inflammatory cell infiltrate (IaICT). [44] The microgap has been reported to be as high as 40-60 μm [45] It allows micromovement during function [46] and permits microleakage of fluids congenial for bacterial growth. Several studies have reported the bacterial penetration across the implant abutment interface. [47],[48] An *in vitro* analysis for the possible microleakage at the implant abutment interface was carried out on the implant abutment assemblies in a blood serum media previously inoculated with microorganisms. After 7 days of anaerobic incubation of the partial or completely immersed implants in the medium, the microorganisms from the internal part of the implants were collected and incubated on the blood agar plates under anaerobic conditions. Microorganisms were found in both the assemblies indicating bacterial leakage at the implant abutment interface. [32]The conclusive remarks of the study reveal that the IAJ is a potential source of microbial contamination which affects the health and integrity of the biologic tissues (bone and soft tissue) around the osseointegrated implant.

MICROBIOTA IN EDENTULOUS/PARTIALLY EDENTULOUS/HISTORY OF PERIODONTITIS PATIENTS

Studies have stated that the microbiota colonizing the clinically healthy implant fixtures in fully edentulous subjects are similar to the microbiota associated with the healthy periodontal sites. [49] It was suggested that extraction of all the teeth results in elimination of the *P. gingivalis* and *A. actinomycetemcomitans* from the oral microbiota.[50] In partially edentulous subjects, the developing microbiota around the implants is similar to the naturally occurring teeth. This microflora inhabitate immediately after installation of the implant. 85% of the microflora is identified as gram-positive cocci. Microbial colonization and the subsequent inflammatory reaction in the peri-implant tissues might be analogous to the key events in the pathogenesis of the periodontitis. The

literature comparing the microbiota around implants in fully edentulous and partially edentulous mouths stated a high percentage of the black pigment bacteroids, fewer coccoids, and motile rods in a completely edentulous mouth, whereas a high frequency of *P. gingivalis* and *P. intermedia* on the implant surface was found in partially edentulous subjects. [51] Microbiota of the remaining teeth serve as the primary source of the putative pathogens and directly influence the fate of the newly incorporated implants. [52]

Microbiota on implants in subjects with the history of the periodontal disease is similar in nature to as found in the periodontal pockets around teeth. [52] It appears imperative to assume that susceptibility to periodontitis may translate to peri-implantitis. Several reviews have reported a history of treated periodontitis as a risk indicator for the implant outcomes with statistically significant results. [50],[51] Karousis in his study on the incidence of peri-implantitis in patients with a history of periodontitis reported a high incidence (28.6%) versus the subjects without previous history of periodontitis. [50]

The host response to the biofilms in relation to implants exhibit inflammatory cell infiltrate in the peri-implant mucosa with considerable loss of the collagen with high levels of the B cells and plasma cells. [53]

PREVENTION OF BIOFILM FORMATION

Biofilm formation is an inadvertent phenomenon to occur around the implant. Stringent maintenance therapy is the cornerstone of successful implantology. Systematic monitoring of the clinical and radiographic parameters, i.e., the presence of plaque and calculus, bleeding on probing, probing depths, presence/absence of suppuration, and radiographic evaluation, is important to assure the peri-implant health. Based on the periodic diagnosis, CIST protocol [54] was recommended. The recently introduced PIMI system [55] emphasizes the importance of prognosis while deciding the treatment plan.

Several pure metals, e.g., iron, titanium, nickel, exhibit bacteriostatic property. In an *in vitro* study done to check the antibacterial property of titanium with amalgam on the microbial strains of *S. sangius, S. mitis, A. naeslundi,* and *Fusobacterium* species revealed a weak antibacterial effects of titanium versus gold. [56] However, several orthopedic studies have documented the use of antibiotic coating on the implant surface in order to widen the antibacterial spectrum of titanium. [57] These studies give a new dimension in the possible role of the antibiotic coatings to provide an antibacterial barrier against microbial colony. Management of the biofilms has a multilevel approach: (1) Prevention of the microleakage at the IAJ thus limiting/eliminating the biofilm ingress; (2) treatment of the biofilm-related infections. Implant biofilm can lead to infection at two levels: Mucosal level (peri-implant mucositis) described as inflammatory lesion residing in the mucosa and bone level (peri-implantitis) which is explained as inflammatory lesion affecting the supporting tissues. [58] The management of peri-implant infections aim at reduction of inflammation, pathogenic bacterial load and the probing depths. Biofilms related to dental implants are best treated through debridement of the contaminated implant surface (mechanical/laser/photodynamics, etc.,) or the antimicrobial therapy with local or systemic antibiotics [Table 4]. Decontamination of the implant surface is challenging for a predictable treatment outcome. Nonsurgical mechanical therapy has been found effective in reducing the microbial load with enhanced results when combined with the antimicrobial rinse in the peri-implant mucositis lesions. [67] Various systemic local drugs, e.g., minocycline, tetracyclines, have shown promising results by decreasing the levels of the *P. gingivalis T. forsythia, A. actinomycetemcomitans.*[60],[61] Laser-assisted therapy for the management of the biofilm-related infection has also been documented in the literature with satisfactory results. [62] Photodynamic therapy, using low level lasers, has been used to decontaminate the infected implant surfaces. Hayek *et al*. in an animal study reported an effective reduction of the microbial count of Prevotella species, Strep hemolyticus on subjecting to the photodynamic therapy. [64] Photodynamic therapy and the regenerative periodontal treatment (autogenous bone graft) help in significant regeneration of the peri-implant bone defects. [65] An *in vivo* study examining the microbial profile of *A. actinomycetemcomitans, P. gingivalis, P. intermedia* before and after photodynamic therapy on the infected implant surfaces in 15 human subjects revealed a significant reduction in the bacterial species as observed from baseline. [66] IAJ is a vulnerable area for biofilm-related infections. Innovative implant abutment designs have helped reducing the microleakage at the IAJ with the sequential decrease in the microbial growth at the microgap. [68]Platform switch, use of tapered implants deceases or eliminates this probable microbial ingress. Any micro-structured part that is exposed to the oral cavity should be highly polished to generate a anti-plaque adhering surface. The principles of plaque maintenance around the implant are similar to those performed around the teeth with some basic differences. The oral antimicrobial rinse (e.g., chlorhexidine) can be advised as a daily regime for implant patients but fluoride mouth rinses should be avoided with the possible risk of surface damage to the titanium abutments. The plastic-coated scaling tips (ultrasonic and hand scaling) should be used to avoid the risk of surface scratches on the abutment as caused with metal instruments. Light intermittent forces should be applied to the abutment surface while polishing after scaling.

Author and reference	Type of study	Type of treatment	Study objectives	Results
Surface modifications				
Leonhardt *et al.*,[56]	*In vivo*	Surface studied: Ti, HA, Ag	Bacterial colonization	Ti has a weak response to the bacterial colonization
Antimicrobial therapy				
Systemic				
Mombelli[59]	*In vivo* (human)	SRP+systemic Abi (10 days)+CHX rinse 12 month f/u)	Antimicrobial effects of tetracycline fibers parameters	Significant improvement in clinical reduction in the microbial count (41-19%)
Local drug				
Mombelli[60]	*In vivo*	SRP+tetracycline fibers+XHX rinse (12 month f/u)	Antimicrobial effects of tetracycline fibers	Improved clinical parameters, 6% radiographic bone fill, reduced microflora
Salvi *et al.*[61]	*In vivo*	SRP+XHX (0.2% Gel)+Arestin (12 month f/u and P.D)	Effect on the clinical parameters	Statistical significant reduction in B.O.P
Lasers				
Deppe *et al.*[62]	*In vivo*	CO_2 laser	Decontamination of implant surface	Clinical improvement and increased bone to implant contact as compared to the control mechanical therapy group
Takasaki.[63]	*In vivo*	Er :YAG laser, decontamination and removal of granulation tissue	Decontamination and degranulation	Laser debridement has enhanced results as compared to manual (plastic curettes) histologic evidence of a more coronally positioned new bone on the laser treated surface
Photodynamic				
Hayek *et al.*[64]	*In vivo*	PDT specification: Diode laser (PS=azulene) CW, 40 Mw	Antimicrobial effects of PDT on the ligature induced peri-implantitis	Microbiologic reduction in the bacterial count on the implant surface
Haas *et al.*[65]	*In vivo*	PDT specification: diode laser (PS=TBO), CW	Decontamination of the infected implant surface	PDT decreases the bacterial load effective bone fill in periimplant defect with regenerative therapy
Dortbudak *et al.*[66]	*In vivo*	PDT specification: (human) diode laser (PS=TBO)	Antimicrobial effects on the infected microflora	TBO Independently reduced implant surface, microflora the bacterial load apart from the studied Aa, Pi, Pg combination effect with PDT

PDT – Photodynamic therapy; PS – Photosensitizer; SRP-Scaling and root planning; CHX-Chlorhexidine; Ti – Titanium; CFU – Colony forming units; BOP – Bleeding on probing; PD – Probing depth

Table 4: *In vitro* and *in vivo* (animal and human) study data on the treatment of biofilm/peri-implant infections

FUTURE PERSPECTIVE

Success of the dental implants lies on a successful osseointegration. The basic principles of biofilm formation are equally applicable in context to implants as they provide favorable grounds for the bacterial adhesion. Future research is required to design implant surfaces that inhibit or reduce the biofilm adhesion. The advent of the antimicrobial photodynamic therapy has added a new dimension in the treatment of the biofilm-related peri-implant infections. However, long-term randomized clinical and microbiological trials are required to fortify the beneficial effects of this therapy in combating the devastating infections caused by the biofilms. Recently, an *in vitro* study was performed on the principles of electrochemistry with the hypothesis if it can be used as a method to disinfect the implant surface. The conclusive findings for this study were in favor of reduction in the bacterial count. Research needs to be carried out in this dimension on the animal models of peri-implantitis. [69]

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