

How do implant surface characteristics influence peri-implant disease?

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Abstract

Objectives: To review the literature on how implant surface characteristics influence peri-implant disease.

Material and Methods: A search of PubMed and The Cochrane Library of the Cochrane Collaboration (CENTRAL) as well as a hand search of articles were conducted. Publications and articles accepted for publication up to March 2010 were included.

Results: Thirteen studies were selected for the review. *Human studies:* To date, few studies have investigated if such differences occur. Limited data suggest that smooth surfaces may be less affected by peri-implantitis than rough surface implants. *Animal studies:* In ligature-induced peri-implantitis studies, no difference between surfaces has been reported. In a spontaneous progression model of peri-implantitis, there was a suggestion that the progression was more pronounced at implants with a porous anodized surface.

Conclusion: The current review revealed that only a few studies provided data on how implant surfaces influence peri-implant disease. Based on the limited data available, there is no evidence that implant surface characteristics can have a significant effect on the initiation of peri-implantitis.

Key words: disease progression; peri-implantitis; surface characteristics

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Implant therapy is a commonly used method of replacing missing teeth. There is an ongoing effort to improve the interface between bone and implant in order to speed up the process of osseointegration and improve its quality. These efforts have been concentrating in improving this interface chemically (by incorporating inorganic phases on or into the titanium oxide layer) or physically (by increasing the level of roughness) (Ehrenfest et al. 2010). Some engineering processes such as electrochemical anodization of

the titanium surface can combine both techniques (Ehrenfest et al. 2010). Human and animal histomorphometric evaluations have shown significantly greater bone-to-implant contact at rough surface implants compared with machined surface implants (Lazzara et al. 1999). In a consensus report that was published in 2009, it was concluded that “moderately rough and rough surfaces provided enhanced bone integration compared with smooth and minimally rough surfaces” (Lang & Jepsen 2009).

It can occur that the coronal portion of the implant, which was initially designed to facilitate osseointegration, becomes exposed to the oral environment as a result of peri-implantitis (Zetterqvist et al. 2010). Some types of rough-surfaced implants, for instance those coated with hydroxyapatite (HA), were reported to have a higher

incidence of complications (Piattelli et al. 1995). Although the initiation of bone loss around such implants was more than likely associated with the delamination of the HA coating, these failures have contributed to the suspicion that an exposed rough surface, especially in patients with poor oral hygiene, can lead to increased bacterial plaque accumulation and eventually peri-implantitis (Zetterqvist et al. 2010). However, evidence for the influence of the implant surface characteristics as a risk indicator for peri-implantitis is very limited (Heitz-Mayfield 2008).

As is known, the presence of microorganisms is fundamental for the development of peri-implant disease. Within weeks after the installation of titanium implants, a sub-gingival microflora associated with periodontitis is established (van Winkelhoff et al. 2000, Quirynen

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et al. 2006). Although the characteristics of the biofilm in peri-implant disease is covered in another review, we should mention that peri-implant diseases have been associated with a predominantly Gram-negative anaerobic microflora and the microbial flora associated with failing implants has been identified as being identical or very similar to that of advanced periodontitis around natural teeth (Becker et al. 1990, Mombelli & Lang 1998, Leonhardt et al. 1999, Renvert et al. 2007). According to Teughels et al. (2006), roughness of the implant surface as well as its chemical composition has a significant impact on the amount and quality of plaque formation. Additionally, contamination is known to affect the titanium oxide layer, which may lead to the pathological loss of osseointegration through peri-implantitis (Ehrenfest et al. 2010).

During the 6th European Workshop on Periodontology in 2008, it was proposed that the term “peri-implant disease” is a “collective term for inflammatory reactions in the tissues surrounding an implant” and that peri-implant mucositis will be used to describe “the presence of inflammation in the mucosa at an implant with no signs of loss of supporting bone” (Zitzmann & Berglundh 2008). Additionally, it was proposed that the term peri-implantitis is an “inflammatory process around an implant, characterized by soft tissue inflammation and loss of supporting bone” (Zitzmann & Berglundh 2008). Clinically, peri-implantitis is often accompanied by a crater-like bone defect surrounding the implant. The aim of this review was to search the literature for the existing evidence on the effect of different implant surfaces on peri-implant disease.

Search Strategy and Results

In order to obtain available data of interest, the PubMed database of the US: National Library of medicine and

The Cochrane Library of the Cochrane Collaboration (CENTRAL) were used as electronic databases. A literature search was carried out on articles published up to and including March 2010.

The key words used in this search were;

(Periimplantitis OR peri-implantitis OR peri implantitis OR peri implant OR periimplant mucositis OR peri-implant mucositis OR peri implant mucositis) AND (surface characteristics OR surface roughness OR material characteristics OR titanium surface OR surface decontamination OR implant types OR implant surfaces OR surface topography OR surface analysis).

During the search in PubMed database, the following limits were applied: Language; English language.

Titles and abstracts were searched in order to find papers eligible for the review.

Only studies using some or all the indicators identified by the existing literature as correct for identifying peri-implantitis and peri-mucositis were included (Heitz-Mayfield 2008). No retrospective studies were included in this review because subjects were not selected randomly. Out of 57 selected papers, three prospective controlled clinical studies and 10 experimental studies (nine animal and one human biopsy) were included.

Human studies

Wennerberg et al. (2003) investigated the early inflammatory response to mucosa-penetrating abutments with different surface topography. At the end of the 4-week test period, clinical and histological evaluations failed to demonstrate any relation between surface roughness and peri-implant mucositis (Table 1).

A 3 year follow-up report of a comparative study of ITI[®] dental implants

(Waldenburg, Switzerland) (titanium plasma sprayed surface) and Brånemark[®] (Nobel Biocare AB, Gothenborg, Sweden) system implants (turned surface) in the treatment of the partially edentulous maxilla was published in 2004 (Åstrand et al. 2004) (Table 2). The authors attempted to compare the outcome of fixed partial prostheses supported by these implants in terms of survival rates, changes in marginal bone level, aesthetic results and frequency of peri-implantitis. Statistically significant differences between the implant systems were found with the rough surface implants having more peri-implantitis. Peri-implantitis was seen in seven ITI[®] implants, one of which subsequently failed completely at 12 months and another at 3 years (Åstrand et al. 2004).

Wennström et al. (2004), in a 5-year prospective randomized controlled clinical trial, studied the results of oral rehabilitation of periodontitis susceptible subjects with implant-supported fixed partial dentures. Each patient received a minimum of two implants (Astra Tech[®], Mölndal, Sweden) and every second implant installed had a machined surface and the remaining had a roughened surface. No signs of peri-implantitis were seen in any of the implants at the end of the 5-year follow up. (Table 2).

Several studies over the years have confirmed the superiority of dual acid etched (DAE) surfaced implants with respect to greater bone-to-implant contact, in comparison with the turned surfaced implants (Lazzara et al. 1999, Stach & Kohles 2003, Feldman et al. 2004). Recently, a prospective, multicentre, randomized, controlled 5-year clinical trial, intended to determine the incidence of peri-implantitis for fully etched implants with a DAE surface extending to the implant platform. In this study, participants had implant sites randomly assigned to receive one hybrid control (coronal part of the implant had

Table 1. Human studies (peri-implant mucositis)

Study	Number of patients/ implants	Implant type	Study	Evaluation period	Results	Comments
Wennerberg et al. (2003) Peri-implant mucositis	10 patients 50 Nobel Biocare implants	Nobel Biocare	An experimental study in humans	4 weeks	No relation was found between peri-implant soft tissue inflammatory response and abutment surface topography	It is possible that longer observation period is needed in order to detect differences

Table 2. Human studies (peri-implantitis)

Study	Number of patients/implants	Implant type	Study	Evaluation period	Results	Comments
Åstrand et al. (2004) Peri-implantitis	28 patients 73 turned Brånemark System [®] implants 77 ITI [®] (TPS) implants	Turned Brånemark System [®] ITI [®] (TPS)	Prospective, randomized-controlled trial All patients had anterior residual dentition in the maxilla. They were provided with implants on each side of the dentition randomly allocated to test and control	3 years	2 ITI [®] (TPS) implants were lost due to peri-implantitis. Peri implantitis was observed in a further seven of the ITI [®] (TPS) implants but with none of the control	Increased risk of peri-implantitis for TPS implants in comparison to turned implants
Wennström et al. (2004) Peri-implantitis	51 patients 73 Astra Tech [®] turned implants 75 Astra Tech [®] Tioblast [®] implants	Astra Tech [®] turned Astra Tech [®] Tioblast [®]	Prospective, randomized-controlled trial For every short span fixed partial denture the patients received a minimum of two implants and every second implant had a turned surface	5 years	2.7% of the implants were lost at the implant level but none due to peri-implantitis	No increased risk of peri-implantitis for fully etched implants in comparison to turned implants
Zetterqvist et al. (2010) Peri-implantitis	112 patients 139 Biomet 3i [®] hybrid dual acid-etched implants 165 Biomet 3i [®] fully Dual acid-etched implants	Biomet 3i [®] hybrid dual acid-etched Biomet 3i [®] fully dual acid-etched	Prospective, randomized-controlled trial Randomly selected sites to receive one Biomet 3i [®] hybrid dual acid-etched and at least one Biomet 3i [®] Fully Dual acid-etched implant in support of a short span fixed restoration	5 years	There was one declaration of peri-implantitis (Biomet 3i [®] hybrid dual acid-etched)	No increased risk of peri-implantitis for fully etched implants in comparison to hybrid-designed implants

Biomet 3i[®], Palm Beach Gardens, FL, USA.

TPS, titanium plasma sprayed.

Tioblast[®], Astra Tech[®], Mölndal, Sweden.

a machined surface) and at least one fully etched implant (Zetterqvist et al. 2010) (Table 1). At the follow-up visits, patients were interviewed for any symptoms that could indicate infection and clinically assessed for bleeding on probing and supuration. The overall oral hygiene was also evaluated with recording of plaque and gingival inflammation. Additionally, standardized periapical X-rays were taken at every follow-up visit in order to identify progressive bone loss. The results did not show any increased risk of peri-implantitis for the fully etched implants (Zetterqvist et al. 2010) (Table 2).

Animal studies (peri-implant mucositis)

Zitzmann et al. (2002) used the mandibular pre-molar areas of five beagle dogs to examine soft tissue reactions of the peri-implant mucosa to plaque formation on implant abutments with different surface topography. No significant differences in plaque formation on different surface characteristics were observed. In a similar study, Pongnarisorn et al. (2007) used the mandibular pre-molar areas of eight greyhound dogs to determine the nature of the inflammatory infiltrate associated with transmucosal abutments with

different surface topography. Again, no significant differences were observed (Table 3).

Animal studies (ligature-induced peri-implantitis)

A number of studies have over the last 20 years compared different implant designs and surfaces in order to evaluate their performance regarding the peri-implant bone loss. In the majority of them, it was reported that no major differences were found (Table 4). All existing animal studies used the ligature-induced peri-implantitis model in dogs. This model has been suggested to be a valid representation of naturally occurring defects in humans and it has extensively been used in experimental studies investigating the treatment of peri-implantitis. This model takes into consideration factors like peri-implant soft and hard tissue break down, the presence of a bone defect (Schwarz et al. 2006). There were some concerns although about the validity of studies designed to compare types and surfaces of implants, based only on the progression of the peri-implant tissue destruction during the “active breakdown

phase”. According to Albouy et al. (2008), this destruction, is attributable not to the surface characteristics of the implants but to the presence and position of the ligature. Accordingly, these studies in Table 4 will not be discussed in detail. Recently, the design of these studies has changed and although the experimental peri-implantitis is still ligature induced, the hard tissue destruction that occurs during the “active breakdown phase” is considered the starting point of the observation period. The “spontaneous progression” of peri-implantitis is now considered as a more valid representation of the naturally occurring peri-implantitis.

Animal studies (ligature-induced peri-implantitis and spontaneous progression)

Albouy et al. (2008, 2009) used the mandibular pre-molar areas of six labradors in order to clinically, radiographically and histologically examine the progression of ligature-induced peri-implantitis at implants with different geometry and surface characteristics. Four different implant systems were used and it was observed that spontaneous progression of peri-

Table 3. Animal studies (peri-mucositis)

Study	Number of animals and implants	Implant type	Study	Observation period	Results	Comments
Zitzmann et al. (2002)	Five beagle dogs 20 implants	Biomet3i [®] Osseotite [®] 3.75 × 8.5	An experimental study in dogs Reactions of the peri-implant mucosa to plaque accumulation at implant abutments with different surface topography	6 months	“Soft tissue reaction to plaque formation was similar at implants with rough and smooth abutment surfaces”	Histometric and morphometric measurements failed to show any differences in plaque formation and inflammation in the peri-implant mucosa
Pongnarisorn et al. (2007)	Eight greyhound dogs 64 implants	Nobel Biocare [®] One-piece designed implants Ti-Unite [®] surface 3.75 × 7	An experimental study in dogs. To determine the nature of the inflammatory infiltrate associated with different transmucosal implant surfaces in dogs	6 months	“Development of inflammation associated with implants is independent of surface type. Furthermore, different surfaces had no influence on the nature of the infiltrate”	Plaque control was carried out twice weekly for 6 months. Despite cleaning the abutments regularly, inflammatory lesions were detected and analysed

Table 4. Animal studies (ligature-induced peri-implantitis)

Study	Number of animals and implants	Implant type	Study	Observation period	Results	Comments
Tillmanns et al. (1997)	14 Beagle dogs 84 implants	28 Calcitek [®] (HA coated) 28 Calcitek [®] (turned Ti-A) 28 APS aterials (TPS)	An experimental study in dogs. Ligatureinduced peri-implantitis. (Clinical evaluation)	3 months for dogs 6 months for dogs	No significant differences for any factors studied	All surfaces were equally susceptible to ligatureinduced peri-implantitis
Tillmanns et al. (1998)	14 Beagle dogs 84 implants	28 Calcitek [®] (HA coated) 28 Calcitek [®] (turned Ti-A) 28 APS materials (TPS)	An experimental study in dogs. Ligature-induced peri-implantitis. (Histologic and microbiologic evaluation)	3 months for six dogs 6 months for eight dogs	No significant differences for any factors studied	All surfaces were equally susceptible to ligature-induced peri-implantitis
Shibli et al. (2003)	Six mongrel dogs 36 implants	9 ITI [®] (TPS) 9 Calcitek [®] (HA coated) 9 3i [®] (Hybrid) 9 3i [®] (turned CPTi)	An experimental study in dogs. Ligature-induced peri-implantitis. (Radiographic and microbiologic observations)	60 days	No significant differences for any factors studied	All surfaces were equally susceptible to ligature-induced peri-implantitis
Martins et al. (2004)	Six mongrel Dogs 36 implants	9 ITI [®] (TPS) 9 Calcitek [®] (HA coated) 9 3i [®] (Hybrid) 9 3i [®] (turned CPTi)	An experimental study in dogs. Ligature-induced peri-implantitis. (Radiographic and clinical observations)	60 days	No significant differences for any factors studied	All surfaces were equally susceptible to ligature-induced peri-implantitis

CPTi, commercially pure titanium; HA, hydroxyapatite; Ti-A, titanium alloy.

implantitis was associated with severe inflammation and tissue destruction around all implants but it was more pronounced around implants with the TiUnite[®] (Nobel Biocare

Nordic AB, Gotenburg, Sweden) surface. TiUnite[®] is an anodized surface and is considered to be a moderately roughened surface (Sul et al. 2006) (Table 5).

Berglundh et al. (2007) used the mandibular pre-molar areas of five beagle dogs to examine radiographically and histologically the progression of ligature-induced peri-implantitis at

Table 5. Animal studies (ligature-induced peri-implantitis and spontaneous progression)

Study	Number of animals and implants	Implant type	Study	Observation period	Results	Comments
Berglundh <i>et al.</i> (2007)	Five beagle dogs 30 implants	15 ITI [®] (turned) 15 ITI [®] (SLA)	An experimental study in dogs. Spontaneous progression of ligature-induced peri-implantitis. (Radiographic and histological observations)	24 weeks	“The radiographic examinations revealed that progression of bone loss was larger at SLA than at polished sites. Histological examination showed evidence of larger inflammatory cell infiltrates in the connective tissue around the SLA than around the turned implant sites”	Increased risk of peri-implantitis for SLA implants in comparison to turned implants
Albouy <i>et al.</i> (2008)	Six Labrador dogs 24 implants	6 Biomet 3i [®] ICE [®] (turned) 6 Astra Tech [®] TiOblast [®] 6 ITI [®] (SLA) 6 Brånemark System [®] TiUnite [®]	An experimental study in dogs. Spontaneous progression of ligature-induced peri-implantitis. (Clinical and radiographic observations)	24 weeks	“The spontaneous progression of peri-implantitis that occurred during the plaque accumulation period produced an average of 1.84 mm bone loss around the turned implants, 1.72 mm bone loss around the TiO blast implants, 1.55 mm bone loss around the SLA implants and 2.78 mm of bone loss around the Ti Unite implants. The difference between the TPS and the TiUnite was statistically significant”	Increased risk of peri-implantitis for TiUnite implants in comparison to a turned, SLA or TiOblast implants
Albouy <i>et al.</i> (2009)	Six Labrador dogs 24 implants	6 Biomet 3i [®] ICE [®] (turned) 6 Astra Tech [®] TiOblast [®] 6 ITI [®] (SLA) 6 Brånemark System [®] TiUnite [®]	An experimental study in dogs. Spontaneous progression of ligature-induced peri-implantitis. (Histological observations)	24 weeks	“The peri-implant tissues in all specimens showed evidence of large inflammatory cell infiltrates extending apical to the pocket epithelium. An increased number of osteoclasts was also observed, indicative of active tissue destruction. All the above were more pronounced at implants with a TiUnite surface”	Increased risk of peri-implantitis for TiUnite implants in comparison to a turned, SLA or TiOblast implants

Sulzer Calcitek[®], Carlsbad, CA, USA.

SLA, sand-blasted large grit acid etched; TPS, titanium plasma sprayed.

implants with identical geometry but different surface characteristics. Three implants with either a sand-blasted acid-etched surface (SLA) or a turned surface were installed bilaterally. At study termination, it was observed radiographically that the progression of bone loss was greater at SLA surfaces than at turned surfaces. Furthermore, histologically, both bone loss and the size of the connective tissue inflammatory lesion were more pronounced in SLA than in turned implant sites (Table 5).

Because clinical data are scarce, it could be of benefit to only mention a report by Baelum & Ellegaard (2004). This study reported on 57 (two-stage,

Astra Tech[®], TiO-blasted, moderately rough surface) and 201 (one-stage, ITI[®], hollow or solid screw, TPS, rough surface) implants that were placed in a private clinic from June 1988 to June 2002. All patients included had a history of periodontitis and after treatment they were able to maintain a good level of oral hygiene. These patients were followed with respect to the survival of their implants, as well as a number of other periodontal parameters. The study also aimed to evaluate, among others, the effect of factors such as smoking and implant length on implant survival. Most of the patients were smokers and they were closely monitored by the

clinicians with regular recalls (every 3 months) for the first few years after implant insertion. Taking into consideration the limitations of this study, the results showed that after 5 years of observation, the survival rates were 97% for the moderately rough surface implants and 94% for the rough surface implants. After 10 years though, the survival rates for the rough-surfaced implants had dropped to 78% but remained high for the moderately rough implants (Baelum & Ellegaard 2004). However, as the authors stress in there discussion, “one tentative explanation for the relatively decreased 10 year survival rate of the one stage implants

could relate to the fact that until 1995 these implants were hollow screw implants, which are virtually impossible to treat once peri-implantitis has developed". Accordingly, this may have overruled the difference in surface structure.

Discussion

In the paper by Åstrand et al. (2004), the TPS surface was reported to be more prone to peri-implantitis when compared with a turned surface. There have been several reports of peri-implantitis with implants with a TPS surface in the past but due to the fact that Straumann® (Waldenburg, Switzerland) ceased production of the rough TPS surface (which is an additive surface) implants and replaced them with the moderately rough SLA-surfaced (a subtractive surface) implants, makes it difficult to ascertain if the peri-implantitis problems were due to the increased surface roughness or the TPS surface itself (Esposito et al. 1997, Åstrand et al. 2000, Hellem et al. 2001). Another issue raised was that the greater prevalence of peri-implantitis among the TPS surface implants may have been caused by the complete denture covering the exposed implants; the Straumann® implants were one stage, whereas the Brånemark® implants were two-stage implants. As a result and unlike the turned surfaced implants, the Straumann® implants were exposed to the oral environment and covered by a denture during the initial 6 months of healing period and before abutment connection. Denture biofilm is comprised of 10^{11} microorganisms/g in wet weight and their metabolites and this colonization can serve as a significant reservoir for infections, perhaps leading to a greater propensity for peri-implantitis (Nikawa et al. 1998, 1999, Paranhos et al. 2009).

In the study by Zetterqvist et al. (2010) cited above, although results did not show any significant differences between in the incidence of peri-implantitis, it should be borne in mind that patients were closely monitored by the research team with annual recalls. Measures of overall hygiene conditions were also recorded annually. Furthermore, only patients that smoked <10 cigarettes per day and had no complicated medical history were included. Under these conditions, it can be speculated that 5 years may not be have been a long enough time for the development and

significant progression of peri-implantitis.

Interpretation of data in the literature today is hampered by a marked heterogeneity in the definitions of peri-implantitis. For example, in the study by Åstrand et al. (2000) peri-implantitis is described as "infection including purulent discharge and bone loss". There is no quantification of the amount of bone to be lost around the implants in order to include a site as a peri-implantitis site. On the other hand, Zetterqvist et al. (2010) defined peri-implantitis as "mucositis with a positive finding of bleeding and/or suppuration upon probing; a probing depth measuring >5 mm; and crestal bone loss which is progressive, >5 mm, and confirmed by radiography". Such definitions may exclude several areas that in other studies would have been diagnosed as peri-implantitis, possibly giving us a significant number of false negatives. The effects of using different definitions of peri-implantitis are also highlighted by the fact that the incidence of peri-implantitis has been reported in the range of 16–58% (Fransson et al. 2005, Roos-Jansåker et al. 2006, Zitzmann & Berglundh 2008, Koldslund et al. 2010). Consequently in future, it is important to formulate a definition of peri-implantitis that would be universally accepted and used in order to make meaningful comparisons based on numerous studies. Obviously, the angulations at which radiographs are made may influence the interpretation of bone crest level. It has been reported that bone levels around implants are associated with inter-examiner measurement error in the range of 0.4 mm (Pikner et al. 2009, Koldslund et al. 2010). This error should be taken into consideration in the formulation of such a definition. A comprehensive definition of peri-implantitis should also aim at avoiding the inclusion of normally expected bone loss shortly after placement. The difficulty in setting a common reference point for various implant designs is another potential source of error.

A systematic review by Heiz-Mayfield (2008) identified strong evidence in the literature that poor oral hygiene, history of periodontitis and cigarette smoking are strong indicators for peri-implant disease. Implant surface characteristics failed to be identified as one of them, mainly because there are only a few existing studies that can provide us with the information necessary to reach

to a safe conclusion. The results from the studies reviewed by Heiz-Mayfield (2008) can be viewed as surprising if a conclusion from Teughels et al. (2006) review is considered, which is that surface roughness and chemical composition of the implant surface have a significant impact on plaque formation. It has been established that rougher surfaces and surfaces with high surface free energy, as titanium has, accumulate and retain more plaque. Furthermore, initial adhesion of bacteria mainly starts at locations with high wettability (a characteristic of titanium) and where bacteria are protected (for example, in grooves and pits) from shear forces (Teughels et al. 2006). It may be important to recognize that in the Teughels et al. (2006) review, most of the articles included examined biofilm development on structures fitted on top of implants, such as abutments and restorations. This bacterial aggregation perhaps initiates the soft tissue inflammation. If this issue remains unresolved, this inflammation could perhaps spread eventually leading to the loss of the supporting bone, irrespective of the implant surface characteristics.

It is perhaps reasonable to postulate that rough surface implants are more difficult to clean than turned surfaces. However, the finding that previously contaminated rough surface implants demonstrated more osseointegration than turned implants after they were cleaned and placed in a disease-free site infers that smooth-surfaced implants may not be easier to decontaminate (Kolonidis et al. 2003, Alhag et al. 2007). Furthermore, when Persson et al. (2001) investigated the influence of surface roughness on the healing following treatment of peri-implantitis in the beagle dog, it was concluded that the amount of re-osseointegration was more pronounced in implants with a rough surface, possibly because the rough surface facilitated the stability of the clot during the early phase of healing. Finally, there is no existing evidence that implant surfaces exposed in the oral cavity show different biofilm compositions depending on the roughness of their surface (Groessner-Schreiber et al. 2004, 2009).

Although a number of studies have suggested that periodontal pathogens inside the peri-implant pockets contribute to peri-implant infections, there are also concerns about the interface between the abutment and the implant

body. It has been demonstrated that bacteria reside within the internal components of implants and this provides them with an environment sheltered from host defences (Persson et al. 1996). It has also been shown that bacteria find their way through the microgap (of approximately 10 µm) at the implant/abutment interface (Quirynen & van Steenberghe 1993, Quirynen et al. 1994, Jansen et al. 1997). Biological consequences of this contamination include soft tissue inflammation that can lead to bone loss (Hermann et al. 2001). According to Hermann et al. (2001), the dimensions of the peri-implant soft tissues are significantly influenced by the presence/absence of a microgap between the implant and the abutment and the location of this microgap in relation to the crest of the bone. The design of the implant/abutment interface determines the size of the microgap and therefore will influence the degree of microleakage (Tesmer et al. 2009). Another approach for reducing the contamination of peri-implant soft tissues was to try and move this gap away from the outer edge of the implant platform. This so-called "platform switching" often proved to be effective in maintaining more bone around the implant compared with the conventional approach. It is still unclear although if these results are due to decreased contamination of the peri-implant tissues or due to mechanical factors like moving of the stress concentration area away from bone (Canullo et al. 2009, 2010).

The bacteriostatic properties of pure metals were examined by Bundy et al. in 1980 as well as by Berry et al. in 1992. They both found an antibacterial activity of ions from titanium dental implants on various oral bacteria. More researchers investigated this possibility but the results have been somewhat contradictory (Leonhardt & Dahlén 1995, Joshi & Eley 1988).

Also to be taken into consideration is that several chemical agents are commonly applied in everyday oral hygiene procedures and/or in the therapy of peri-implantitis. These agents, when used, may alter the morphology and chemical structure of the implant surface and this in turn might affect the fibroblast attachment to different implant surfaces. A study by Burchard et al. (1991) investigated the *in vitro* effects of chlorhexidine and stannous fluoride on the attachment of gingival fibroblasts to dental implants with different surface

characteristics (machined, TPS and HA) following their exposure to solutions of 0.12% CHX, 1.64% SF and normal saline. A more pronounced fibroblast attachment was observed to specimens treated with saline or chlorhexidine than to those treated with stannous fluoride. Additionally and perhaps more importantly, fibroblasts were more likely to attach to rough-surfaced than to smooth-surfaced implant specimens (Burchard et al. 1991).

It has been shown that the soft tissue interface around implants is comparable with teeth and in experimental animals is about 4 mm long (Klinge & Meyle 2006, Rompen et al. 2006). Both the epithelium and the connective tissue contribute to the biological width, which in itself is the main barrier against bacterial penetration (Berglundh et al. 1991, Cochran et al. 1997, Rompen et al. 2006). It has also been demonstrated that implant surface roughness has an impact on the quality of soft tissue sealing, and that the soft-tissue connection to the implant surface is of crucial importance as it relates to the prevention of peri-implant infection (Quirynen et al. 2002). A certain surface roughness may be needed for optimal soft tissue sealing, perhaps ensuing from the interaction between the surface texture and epithelial cell attachment and proliferation (Quirynen et al. 2002). It should also be borne in mind that the mucosal barrier is affected not only by the surface roughness of the transmucosal component but also by the choice of biomaterial used to make this abutment. Welander et al. (2008) suggested that the soft tissue healing to abutments made of titanium or zirconium oxide is superior to that at the abutments made of gold/platinum alloy.

A number of other factors should also be taken into consideration as the causative factors of the initiation and progression of peri-implantitis. Implant insertion in different bone qualities, the level of initial resorption of the marginal, buccal, lingual bone, existing dehiscences in the bone, the implant length or diameter, timing of placement and loading time all could have an effect. Additionally, in a study by Hultin et al. (2002), the microbiota and inflammatory host response around implants and teeth in patients with peri-implantitis were characterized. These authors concluded that there is a stronger inflammatory response around implants in partly edentulous patients than around

those in fully edentulous patients. Furthermore, their findings indicated a site-specific, bacterial-driven inflammatory reaction around implants with peri-implantitis rather than a patient-associated specific host response. Unless the importance of these findings can be further defined, it is perhaps impossible to ascertain which factors are important in the initiation and progression of peri-implantitis.

It seems that the advantages of rough-surfaced dental implants may be explained by the surface possibly providing support for the developing coagulum and thus facilitating greater bone healing and better quality of osseointegration. Perhaps hybrid implants should be considered only in individuals that are highly susceptible to periodontitis. In such patients, inflammation due to a rough or badly designed superstructure causes bone loss and exposed smooth threads. If in such patients, the smooth threads become exposed, these threads may be less plaque retentive, increasing the possibility of arresting the progression of peri-implantitis. It has now been recognized that the mucosal complications reported for the HA surfaces were initiated by mechanisms different to the traditional peri-mucositis – peri-implantitis pathway (Zetterqvist et al. 2010). Delamination or biodegradation of the coating may be partly responsible for the clinical failure in the implant/coating interface (Chang et al. 1999, Lee et al. 2000).

In reviewing the existing literature, it is possible to say that there is a lack of studies investigating the effect of implant surfaces on the initiation of peri-implantitis and available data cannot answer this question. There is limited evidence from experimental studies suggesting that surface characteristics may have an effect on the progression of established peri-implantitis. It would perhaps be interesting to further investigate this latter possibility in patients highly susceptible to periodontal/peri-implant infections.

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Clinical Relevance

Scientific rationale for the study: This review aimed to review articles investigating the effects of implant surface characteristics on peri-implant disease.

Principal findings: There is no evidence in the existing literature that implant surface characteristics have a significant effect on the initiation of peri-implantitis. However, there is some evidence that surface characteristics may influence the rate of progression of peri-implantitis.

Clinical implications: Not enough evidence exists to suggest significant changes in existing clinical protocols.

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