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Review Article

Impact of supportive periodontal therapy and implant surface roughness on implant outcome in patients with a history of periodontitis

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Abstract

Objective: This review searched for a relationship between susceptibility to periodontitis and peri-implantitis, with implant outcome as the primary outcome variable and supportive periodontal therapy (SPT) and implant surface roughness as confounding factors.

Material and Methods: It is based on a MEDLINE search up to June 2006. Only 16 fulfilled the selection criteria. The heterogeneity of the studies (e.g. periodontal status, SPT, prosthetic design, . . .) rendered a meta-analysis impossible.

The impact of a history of periodontitis on early implant loss was negligible. Only five papers reported sub-data for patients with different degrees of periodontitis. Four out of five papers indicate a higher incidence of late implant loss and/or marginal bone loss in patients with a history of periodontitis. This difference was most obvious for very rough implants (three papers), and/or when SPT was not organized (one paper). Other confounding factors were often neglected. Another 10 papers only reported the outcome of implants in patients with a history of periodontitis. In case of SPT and when avoiding roughened surfaces, late implant loss remained below 3%, and marginal bone loss remained low.

Conclusions: These results seem to indicate that periodontally compromised patients can be successfully treated with minimally/moderately rough implants, in the presence of SPT.

Key words: attachment loss; bone loss; implants; peri-implantitis; periodontitis; plaque; smoking; susceptibility

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Several review papers clearly indicated that partially edentulous patients can predictably be rehabilitated by means of oral implants (e.g. van Steenberghe et al. 1990, Berglundh et al. 2002, Pjetursson et al. 2004, Esposito et al. 2005a, b). The question remains, however, whether the outcome of oral implants is influenced by a history of periodontitis. Malmstrom et al. (1990) opened this debate by reporting on a single partially edentulous patient who was rehabilitated by implants after an unsuccessful treatment of a rapidly progressing, early-onset periodontitis. The anamnesis of the patient included smoking and a chemotactic defect in the patient's neutrophils. Within the first 2 months of subgingival healing, three maxillary and one mandibular implant had to be removed due to recurrent abscesses. A comparable case was

presented by Fardal et al. (1999). Both studies are often misquoted to support the idea that the survival/success of oral implants might be jeopardized in patients with a history of periodontitis. Recently, a number of clinical long-term studies reported significant bone loss around implants in some patients, and this for all the major implant systems (Karoussis et al. 2004a, b, Naert et al. 2004, Fransson et al. 2005, Rasmusson et al. 2005, Schwartz-Arad et al. 2005, Roos-Jansaker et al. 2006a, b, c, Telleman et al. 2006). Implant loss as well as marginal bone loss around implants seem to cluster in a small group of patients (Weyant & Burt 1993, Jemt 1994, Hutton et al. 1995, Friberg et al. 1997, Chuang et al. 2001, 2002a, b, 2005, Roos-Jansaker et al. 2006a). The question arises as to whether this might be linked to a history of periodontitis or whether other factors might be involved.

Predisposition/confounding factors for periodontitis and peri-implantitis

Besides a direct link between tooth loss and implant loss (e.g. microbial load, oral hygiene), several mutual confounding factors have been identified. Such confounding factors have to be taken into consideration when comparing data from different studies in partially edentulous patients rehabilitated with implants. Important confounding factors are smoking (for a review, see Chuang et al. 2002a, b, 2005, Klinge et al. 2005, Nitzan et al. 2005, Roos-Jansaker et al. 2006a, b), uncontrolled diabetes (for a review, see Beikler & Flemmig 2003) and genetic pre-disposition (Gruica et al. 2004, Jansson et al. 2005).

Early *versus* late implant loss

It is of course essential to make, from the start, a clear distinction between early and late implant losses, because their aetiopathogenesis, and thus also their relationship/mode of interaction with periodontitis, is different (Esposito et al. 1998a, b, Quirynen et al. 2002, Roos-Jansaker et al. 2006a).

An early implant loss or impaired healing corresponds to the inability to establish osseointegration defined as a ''direct structural and functional connection between ordered living bone and the surface of a load-carrying implant'' (Brånemark 1985). Besides a number of patient-related factors such as smoking (Bain & Moy 1993, Chuang et al. 2002a, b, Klinge et al. 2005), bone quality (Jaffin & Berman 1991, Hutton et al. 1995), osteoporosis (for a review, see Beikler & Flemmig 2003), systemic diseases or chemotherapy (for a review, see van Steenberghe et al. 2000, 2002, Moy et al. 2005), surgical trauma, and bacterial contamination during implant insertion seem to be the most important causes of early implant loss (for a review, see Esposito et al. 1998a, b, 1999, Quirynen et al. 2002). An early loss due to infection can be explained by: (i) a pre-existing, undiagnosed, infection/inflammatory process within the recipient site or in the immediate vicinity of an integrating implant (e.g. Quirynen et al. 2005), (ii) a direct bacterial contamination during implant insertion (infection of the implant or the bony socket, e.g. Piattelli et al. 1995, Esposito et al. 1998a, b), (iii) an early contamination of the blood clot along the integrating part of the implant via the oral cavity in the case of a one-stage procedure (indeed the ''pristine'' peri-implant pocket is colonized within days e.g. Quirynen et al. 2006) or (iv) an indirect bacterial contamination of this blood clot from infections in the surrounding area (e.g. gingivitis, periodontitis) via the blood supply (van Steenberghe et al. 1990). The incidence of the last three modes of infection may be different in patients with a healthy periodontium *versus* patients with gingivitis/periodontitis.

An early failure should thus not be confused with peri-implantitis being an ''inflammatory process'' affecting the tissues around an osseointegrated implant in function, resulting in loss of supporting bone and eventually in late implant loss (Albrektsson & Isidor 1994). An implant fracture can also be considered as a late failure, as well as the loss of an implant due to occlusal overload (situations in which the functional load applied to the implants exceeds the capacity of the bone–implant anchoring) (for a review, see Quirynen et al. 2002). Factors associated with peri-implantitis are less well understood and seem to be related to peri-implant environmental factors and host parameters (for a review, see Mombelli & Lang 1998, Tonetti 1998, Mombelli 1999, Quirynen et al. 2002). A large portion of late implant losses have been assigned to peri-implantitis (for a review, see Esposito et al. 1998a, b, 1999). The microbiota involved in the peri-implantitis process resembles the flora associated with periodontitis (for reviews, see Mombelli & Lang 1998, Mombelli 1999, Quirynen & Teughels 2003, Sbordone & Bortolaia 2003).

Supportive periodontal therapy (SPT)

SPT (identified as regular visits to the therapist for periodontal control and maintenance in a well-organized scheme, the number of appointments per year following a pre-designed subject-tooth/implant-site risk assessment method; Lang & Tonetti 2003) forms the basis of long-term success after periodontal surgery (for a review, see Renvert & Persson 2004). Overall, SPT seems to be effective in preventing recurrence of periodontitis. The risk assessment for disease recurrence includes smoking habits, the presence of the remaining deep pockets following periodontal therapy, the proportion of sites with bleeding on probing, the number of missing teeth, the degree of bone loss in relation to patients' age (Gilbert et al. 2002), interleukin-1 (IL-1) gene polymorphism, and other genetic factors (for a review, see Lang & Tonetti 2003, Renvert & Persson 2004).

Implant design

Albrektsson & Wennerberg (2004) identified three distinctive different types of surface roughness among the available oral implants: minimally rough ($S_a \pm 0.5\,\mu$m, which is the majority of previously marketed implants, also called the machined implants), moderately rough (S_a between 1.0 and 2.0 μm, presently most marketed implants such as Osseotite, TiUnite and SLA) and rough ($S_a > 2.0\,\mu$m, like some plasma-sprayed or HA-coated implants). Within the oral cavity, surface roughness has a dominant impact on the biofilm formation (for a review, see Quirynen & Bollen 1995, Teughels et al. 2006). All intra-oral hard surfaces (teeth, dentures, restorative materials and implant surfaces) attract more bacteria (supra- as well as subgingivally) when increasing their surface roughness (for a review, see Teughels et al. 2006). As such, it might be reasonable to consider the implant surface roughness as a co-factor in the analysis of their longevity. Becker et al. (2000) compared minimally rough implants placed in one and two stages with plasma-sprayed implants, over a period up to 3 years, and observed

significantly more marginal bone loss around the latter. Åstrand et al. (2004) illustrated in an randomized-controlled trial (RCT) trial with a split-mouth design that an implant with a rough surface developed significantly more peri-implantitis than minimally rough implants. The latter was confirmed via a systematic review (Esposito et al. 2005a, b).

Besides the surface, the macro-design of the implant might also play a significant role. As such, several large variations have been reported within implant systems, depending on the implant design (e.g. Karoussis et al. 2004a, b, Nowzari et al. 2006).

Teeth as the reservoir for periopathogens

Several studies indicate that, at least in partially edentulous patients, teeth act as a reservoir for the colonization of the subgingival area around implants (Lekholm et al. 1986, Apse et al. 1989, Quirynen & Listgarten 1990, Koka et al. 1993, Leonhardt et al. 1993, Mombelli et al. 1995, Mengel et al. 1996, Papaioannou et al. 1996, Gouvoussis et al. 1997, Sbordone et al. 1999, Hultin et al. 2000, 2002). Two recent studies (De Boever & De Boever 2006, Quirynen et al. 2006) explored the ''early'' colonization of the pristine peri-implant pocket (after placement of a one-stage implant or connection of abutment to a two-stage implant) in partially edentulous patients. Both studies indicated a rapid colonization. Within 2 weeks, the subgingival area around implants was colonized by similar numbers of bacteria (including significant proportions of periopathogens) as observed along the neighbouring teeth. The quick colonization of the peri-implant pocket is in agreement with previous observations by Mombelli et al. (1988), who followed the initial colonization of implants in fully edentulous patients and also reported a nearly complete maturation already 1 week after insertion. It is therefore not surprising that many clinicians make an association between a susceptibility for periodontitis and peri-implantitis, especially in partially edentulous patients.

This paper aims to review studies on the relationship between periodontitis and the incidence of implant loss and/or peri-implantitis. The review will make a distinction between early and late implant losses. Finally, it will take into consideration both the implant surface roughness and the inclusion of an SPT programme as possible confounding factors.

Material and Methods

Search strategy

A thorough MEDLINE search of the English literature had been carried out in June 2006 applying the following search terms: ''implants'' and ''periodontitis'', or ''periodontal'', or ''peri-implantitis''. All retrieved abstracts/titles were analysed by two independent reviewers (M. A., N. V. A.), who selected all studies with potentially useful data (e.g. human study, clinical data, 1-year follow-up, ...) for the following PICO questions (Patient, Intervention, Comparison and Outcome): ''Is the outcome of implants in patients with a history of periodontitis similar as for periodontitis free patients, and are SPT and implant surface roughness confounding variables''. Finally, manual searches were performed based on bibliographies of previous reviews in the following journals: *Clinical Implant Dentistry & Related Research*, *Clinical Oral Implants Research*, *International Journal of Oral & Maxillofacial Implants*, *International Journal of Periodontics & Restorative Dentistry*, *Journal of Clinical Periodontology* and *Journal of Periodontology*.

Study inclusion criteria

For this review, only conventional root-form endosseous implants were considered, not mini implants. Only studies with a clear definition on the periodontal condition of the included patients were selected. Prospective and retrospective studies (randomized and non-randomized clinical trials, cohort studies, case–control studies, or case reports) were considered if a follow-up (under loading) of at least 1 year was respected for at least 80% of the implants. If it was not evident from the paper that it was a prospective study, the paper was classified as retrospective. Case reports were only included if $\geqslant$eight patients or $\geqslant 10$ implants were enrolled. Two types of studies were included: (i) papers with a direct comparison between patients with and without a history periodontitis and (ii) papers reporting on outcome variables for only patients with a history of periodontitis.

Outcome variables

Even though the impact of the implant-based rehabilitation on the quality of patients life should be the primary outcome variable tested, this review could only retrieve data on an implant/prosthesis level. The following variables have been included in the review process:

- *Implant loss*. For this parameter, the criteria of each paper have been respected. This means that an evaluation of an implant immobility (as assessed on individual implants), or an absence of peri-implant radiolucency (assessed on radiographs) – standard criteria of proper osseointegration – was not always available. A distinction was made between implants lost or removed before the prosthetic reconstruction (regarded as early loss) and those lost or removed afterwards (called late failures), and of fractured implants.
- *Marginal bone*. The degree of marginal bone loss during implant loading was also considered. Studies without radiographic examinations were indicated as ND (No Data). Data from radiographic examinations, presented as frequency distributions, received priority. The data are presented as millimetre per year, after the first year of bone remodelling, or as a proportion reaching a certain threshold bone loss (depending on the specific paper).
- *Attachment level/probing depth*. Results from attachment level and probing depth assessments were also analysed. Data are presented as frequency distributions, received priority. The data on attachment loss are again presented as millimetre per year. Data on probing depth measurements are presented as proportions reaching a certain threshold depth (depending on the data available in each specific paper).
- *Bleeding upon probing*. This parameter is presented as a proportion of sites with bleeding upon probing.
- *Peri-implantitis*. For this parameter, the criteria of each paper have been respected.

Implant outcome in relation to the periodontal health in the natural dentition was thus the primary outcome variable for this review. However, during the analysis of the included papers,

special attention has been paid to the impact of SPT and implant surface roughness as possible confounding factors.

Results

Paper selection and validity assessment

From the 1852 initially retrieved abstracts (first screening), 1798 were excluded because they were not relevant for this PICO question. Two independent reviewers (M. A. and N. V. A.) performed a full-text analysis of the 54 selected studies with possible relevance against the inclusion criteria. The inter-examiner agreement for study in/exclusion was high (κ score of >0.93 for abstracts, 1.00 for full papers).

The data were stored in an Excel file (data abstraction form) to allow optimal comparison and to perform simple analysis (calculation of means and standard deviations). Thirty-eight papers were excluded following full-text analysis. Most papers were excluded because of a lack of significant clinical data ($n=37$) or the inability to break down the data per periodontal disease status ($n=1$).

The 16 remaining papers were included without taking into consideration further quality assessment on aspects such as: inclusion of general outcome confounders [e.g. smokers, bone quality, … (e.g. see Chuang et al. 2002a, b), proper statistical analysis, presentation of inclusion/exclusion criteria, inclusion of objective outcome variables for implant success, inclusion of ''all'' consecutive patients, unbiased patient assignment, blind data analysis, ...]. The 16 selected papers, 11 prospective and five retrospective studies, are presented in Tables 1 and 2. Most of the studies reported on partially edentulous patients.

Implants in partially edentulous patients with different degrees of periodontitis: comparative studies

Only five studies (Table 1) compared the implant outcome variables of patients with a healthy periodontium with those of patients with a history of periodontitis. Unfortunately, nearly no attention has been given to confounding factors such as smoking (4/5), oral hygiene (5/5), and genetic predisposition (5/5). From one large-scale study (Roos-Jansaker et al. 2006a–c) on

Table 1. Outcome variables for implants in partially edentulous patients comparing different types of periodontal status: prospective and retrospective studies grouped and in alphabetic order

References	Confounding factors: prospective/retrospective	Confounding factors: general health	Confounding factors: infection	Confounding factors: % smokers	Confounding factors: perio health	Confounding factors: SPT x/year	Follow-up: months	Follow-up: range	Population: n pat	Population: age	Population: status	Implant characteristics: n impl	Implant characteristics: surface	Implant characteristics: stage 1 or 2	Implant characteristics: impl loss early %	Implant characteristics: impl loss late %	Implant characteristics: tooth loss	Marginal bone: impl (after year 1) mean mm/y	Marginal bone: impl (after year 1) frequency range	Marginal bone: impl (after year 1) %	Marginal bone: teeth mean mm/y	Marginal bone: teeth frequency range	Marginal bone: teeth %	Marginal bone: tooth vs impl	Attachment level/probing depth: impl mean mm/y	Attachment level/probing depth: impl frequency range	Attachment level/probing depth: impl %	Attachment level/probing depth: teeth mean mm/y	Attachment level/probing depth: teeth frequency range	Attachment level/probing depth: teeth %	implant BOP %	tooth BOP %	peri-impl %impl	fracture %impl
Evian et al. (2004)	Retro	Okay	Healthy	NR	y	Regular	31	1–134	72	NR	Part	72	r	2	0.0	8.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
			Chronic	NR	y	Regular	31	1–134	77	NR	Part	77	r	2	0.0	20.7	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Hardt et al. (2002)	Retro	Okay	< Perio	NR	NR	No	60		25	57.3	Part	92 ε	sm	2	0.0	3.3	ND	0.18	>2 mm ζ	24	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
			Perio			No	60		25	53.3	Part	100 ε	sm	2	0.0	8.0	ND	0.26	>2 mm ζ	64	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Karoussis et al. (2003)	Pros	NR	Healthy	19.8	y	Regular	120		45	NR	Part	91	r	1	0.0	3.5	ND	*0.05* α	NR	NR	ND	ND	ND	ND	NR	PPD ≥ 6	3.3	ND	ND	ND	ND	ND	5.8	ND
			Chronic	47.6	y	Regular			8	NR	Part	21	r	1	0.0	9.5	ND	*0.09* α	NR	NR	ND	ND	ND	ND	NR	PPD ≥ 6	19.0	ND	ND	ND	ND	ND	28.6	ND
Mengel & Flores-de-Jacoby (2005)	Pros		Healthy	NR	y	4/y, 3 y	36		12	31	Part	30	sm	2	0.0	0.0	ND	*0.06* α	NR	NR	*0.4%* δ	NR	NR	NS #	0.61	NR	NR	0.08	NR	NR	ND	ND	ND	ND
			Chronic		y	4/y, 3 y	36		12	34	Part	43	sm	2	0.0	0.0	ND	*0.09* α	NR	NR	*0.7%* δ	NR	NR	NS #	0.35	NR	NR	0.00	NR	NR	ND	ND	ND	ND
			Aggressive		y	4/y, 3 y	36		15	32	Part	77	sm	2	0.0	2.6	ND	*0.15* α	NR	NR	*0.9%* δ	NR	NR	NS #	0.71	NR	NR	0.40	NR	NR	ND	ND	ND	ND
Rosenberg et al. (2004)	Retro	Okay	Healthy	NR	y	4/y, 1 y	156		183	49.5	Part	399	sm	2	5.3	1.0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.0	ND
											Part	189	r		5.0	1.0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	1.0	ND
			Compromised condition		y	4/y, 1 y	156		151	61.8	Part	382	sm	2	8.1	2.1	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	2.1	ND
											Part	541	r		4.8	3.9	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	3.9	ND

General health: okay means no confounding systemic diseases, infection, type of periodontal disease (e.g. perio health, periodontal health in remaining dention at start study); SPT, supportive periodontal therapy; ε, in maxillary posterior segment only; surface: sm, minimally rough; m, moderately rough; r, rough; tooth *versus* implant, correlation between bone loss around teeth and implants in same patient; NR, not reported; ND, no data; data in italic, pay attention to applied parameter (α, scored from shoulder; δ, relative to root length; ζ, first year of remodelling included); bold, significantly different from healthy group; BOP, bleeding on probing; y, year.

Table 2. Outcome variables for implants in patients with history of periodontitis: prospective and retrospective studies grouped by year of publication

	Confounding factors						Follow-up		Population			Implant characteristics			Marginal bone							Attachment level/probing depth									
															impl loss		tooth	impl (after year 1)			teeth					impl			teeth			tooth impl			
References	pros/retro	general health	infection	% smokers	perio health	SPT	months	range	n pat	age mean	status	n impl	surface	stage 1 or 2	early %	late %	loss	mean mm/y	range	frequency %	mean mm/y	range	frequency %	tooth vs impl	mean mm/y	range	frequency %	mean mm/y	range	frequency %	BOP %	BOP %	perl-impl %impl	fracture %impl
Ellegaard et al. (1997a)	Pros	Okay	Progressive	64.0	y	4/y	30	12–40	19	59.9	Part	31	sm	2	0.0	0.0	ND	3 y > 3 mm α		0.0	ND	ND	ND	ND	y 3	PPD ⩾ 6	0.0	ND	ND	ND	ND	32.1	ND	ND
					y	4/y	33.3	3–84	56	59.8	Part	93	r	1	2.2	1.1	ND	3 y > 3 mm α		4.3	ND	ND	ND	ND	y 3		7.6	ND	ND	ND	ND	30.4	ND	ND
Ellegaard et al. (1997b) (Sinus region)	Pros	Okay	Progressive	62.5	y	4/y	30.4	3–40	17	42–71	Part	51	sm	2	0.0	2.0	ND	3 y > 3 mm α		0–5	ND	ND	ND	ND	y 3	PPD ⩾ 6	0.0	ND	ND	ND	ND	27–35	ND	ND
					y	4 y	27.4	3–59	13	42–71	Part	29	r	1	0.0	10.3	ND	3 y > 3 mm α		9.1–14.3	ND	ND	ND	ND	y 3		0.0	ND	ND	ND	ND	14–28	ND	ND
Brocard et al. (2000)	Pros	Okay	Chronic	NR	y	1/y	109	12–84	147	53	Part	375	r	1	0.0	5.1	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Yi et al. (2001a)	Pros	Okay	Advanced	NR	y	Custom	36	–	43	52	Part and full	125	sm	1	0.0	0.0	ND	3 y > 2 mm α		0.0	ND	ND	ND	ND	y 3	PPD ⩾ 4	2.0	ND	ND	ND	ND	2	0.0	ND
Yi et al. (2001b)	Pros	Okay	Compromised condition	NR	y	NR	18	13–35	35	49.5	Part	81	sm	2	0.0	0.0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Mengel et al. (2001)	Pros	Okay	Chronic	NR	y	4/y, 5 y	60		3	31–44	Part	12	sm	2	0.0	0.0	ND	0.03	ND	ND	*0.9%δ*	ND	ND	NS #	0.75	ND	ND	0.15	ND	ND	ND	ND	ND	ND
			Aggressive		y	4/y, 3 y	60		5		Part	36	sm	2	2.8	11.1	ND	0.07	ND	ND	*1.0%δ*	ND	ND	NS #	0.91	ND	ND	0.45	ND	ND	ND	ND	ND	ND
Leonhardt et al. (2002)	Pros	NR	Advanced	NR	y	Regular	120		15	21–71	Part	57	sm	2	0.0	5.3	13.0	*0.09* α	> 3 mm	12.3	0.08	ND	ND	No corr	ND	ND	ND	ND	⩾ 4 mm	15.7	61	35.3		
Karoussis et al. (2003)	Pros	NR	Chronic	47.6	y	Regular	120		8	NR		21	r	1	0.0	9.5	ND	*0.09* α	ND	ND	ND	ND	ND	ND	ND	PPD ⩾ 6	19.0	ND	ND	ND	ND	ND	28.6	ND
Baelum & Ellegaard (2004)	Pros	Okay	Chronic?	65.0	y	4/y	120	–	32	59.5	Part	57	sm	2	0.0	3.0	ND	10 y > 3.5 mm α		5.0	ND	ND	ND	ND	y 10	PPD ⩾ 6	23.3	ND	ND	ND	ND	90.5	ND	ND
					y	4/y	168	–	108	58.1	Part	201	r	1	0.0	22.0	ND	10 y > 3.5 mm α		13.6	ND	ND	ND	ND	y 10		24.6	ND	ND	ND	ND	69.5	ND	ND
Wennström et al. (2004)	Pros	Okay	Advanced	33.3	y	2–3/y	60	–	51	36–80	Part	74	sm	2	1.4	*0.0 β*	ND	0.01	> 3 mm	1.5	ND	ND	ND	ND	ND	PPD ⩾ 6	5.3	ND	ND	ND	5.0	ND	ND	2.7*
												75	m	2	0.0	*0.0 β*	ND	0.04	> 3 mm	1.4	ND	ND	ND	ND	ND		5.3	ND	ND	ND		ND	ND	1.3*
Mengel & Flores-de-Jacoby (2005)	Pros		Chronic	NR	y	4/y, 3 y	36		12	34	Part	43	sm	2	0.0	0.0	ND	*0.09* α	ND	ND	*0.7%δ*	ND	ND	NS #	0.35	ND	ND	0.00	ND	ND	ND	ND	ND	ND
			Aggressive		y	4/y, 3 y	36		15	32	Part	77	sm	2	0.0	2.6	ND	*0.15* α	ND	ND	*0.9%δ*	ND	ND	NS #	0.71	ND	ND	0.40	ND	ND	ND	ND	ND	ND
Ellegaard et al. (2006) (Only no grafted implant)	Pros	Okay	Chronic?	57	y	4/y	68	0–128	30	58.4	Part	50	sm	2	0.0	3.0	ND	10 y > 3.5 mm α		5.9	ND	ND	ND	ND	y 10	PPD ⩾ 6	26.8	ND	ND	ND	ND	100	ND	ND
				68	y	4/y	61	0–147	41	55.5	Part	81	r	1	0.0	41.0	ND	10 y > 3.5 mm α		12.2	ND	ND	ND	ND	y 10		34.5	ND	ND	ND	ND	83.1	ND	ND
Nevins & Langer (1995)	Retro	Okay	Chronic	NR	y	Regular	54	12–96	59	42–86	Part and full	309	sm	2	1.2	1.0	ND	1 y > 2 mm α		2.3	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Hardt et al. (2002)	Retro	NR	Perio	NR	NR	No	60		25	53.3	Part	100 ε	sm	2	0.0	8.0	ND	0.26	> 2 mm ζ	**64**	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Ricci et al. (2004)	Retro	NR	Periodontitis (no clear def)	41.2	y	2–4/y	60		51	47.7	Part	112	m	2	0.0	0.0	0.0	2.2	1.6–3.8	28.6	ND	ND	ND	ND	ND	PPD ⩾ 3	28.6	ND	ND	ND	ND	15.5	ND	ND
Evian et al. (2004)	Retro	Okay	Chronic	NR	y	Regular	31	1–134	77	NR	Part	77	r	2	0.0	20.7	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Rosenberg et al. (2004)	Retro	Okay	Compromised condition	NR	y	4/y, 1 y	156		151	61.8	Part	519	sm	2	8.1	0.5	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
													r		4.8	3.4	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND

General health, okay means no confounding systemic diseases; infection, type of periodontal disease (e.g. perio health, periodontal health in remaining dention at start study); SPT, supportive periodontal therapy; ε, in maxillary posterior segment only; surface: sm, minimally rough; m, moderately rough; r, rough; tooth vs implant, correlation between bone loss around teeth and implants in same patient; NR, not reported; ND, no data; data in italic, pay attention to applied parameter (α, scored from shoulder; δ, relative to root length; ζ, first year of remodelling included; £ ⩾ 5 threads); bold, significantly different from healthy group; Ø selected population, patients with at least one implant failure; BOP, bleeding on probing; y, year; PPD, probing pocket depth.

patients with different degrees of periodontal breakdown before implant insertion, data per subgroup could not be retrieved.

The incidence of early implant loss is 0.0% in four out of the five studies, for both the healthy group and the group of patients with a history of periodontitis. Only in the study of Rosenberg et al. (2004) high early failure rates were recorded, but similar for both subpopulations.

The proportion of late implant loss shows a large range (0–21%). The highest incidence of implant loss was reported for implants with a very rough surface (Karoussis et al. 2003, Evian et al. 2004, Rosenberg et al. 2004 for a subset), and in one study where SPT was not given to the patients (Hardt et al. 2002). Under these conditions, the number of late losses seems to be clearly higher for patients with a history of periodontitis (ca. 3 × higher). In the presence of SPT and using minimally rough implants (Rosenberg et al. 2004, Mengel & Flores-de-Jacoby 2005), the rate of late implant losses is not different between both subpopulations. Patients with a history of aggressive periodontitis are, however, more prone to late implant loss, even when minimally rough implants are used and SPT is given (Mengel & Flores-de-Jacoby 2005).

Bone-level measurements are only considered in three out of the five studies. In two of these studies, patients with a history of periodontitis show a higher level of marginal bone loss. This was the case when SPT is lacking (Hardt et al. 2002) or when a very rough implant surface (the old titanium plasma-sprayed TPS surface) is used (Karoussis et al. 2003, the latter, however, not statistically significant). In case of SPT and the use of minimally rough implants (Mengel & Flores-de-Jacoby 2005), a similar amount of bone loss is reported for patients with or without a history of periodontitis. Patients with a history of aggressive periodontitis show, however, double the amount of marginal bone loss when compared with subjects with a healthy periodontium (Mengel & Flores-de-Jacoby 2005).

Data on probing depth evaluations and/or on bleeding upon probing are sparse. Only two papers (rough implant) present data on the incidence of peri-implantitis, with a higher incidence for patients with a history of periodontal disease (Karoussis et al. 2003, Rosenberg et al. 2004). The studies of Roos-Jansaker et al. (2006a, b, c), reporting on 218 patients (1057 minimally rough implants), also found two potential explanatory variables for peri-implantitis (via a multivariate analysis): a history of periodontitis at implant insertion (more than 31% bone loss around their teeth, $p = 0.05$) and smoking ($p = 0.002$). The high incidence of peri-implantitis (6.6% or 16% of the patients) was explained by the long follow-up period (9–14 years) and a lack of a uniform SPT.

Implants in patients with a history of periodontitis

Table 2 summarizes the data from 12 prospective and five retrospective studies reporting on implants placed in patients with a history of periodontitis (including the five previous papers).

The percentage of early implant loss (overall mean with equal weight to each study: 0.8%, SD 1.9) remains in general very low (0% in 10/17 studies). Only in two studies (Mengel et al. 2001, aggressive periodontitis; Rosenberg et al. 2004, compromised periodontal conditions) an early implant loss rate of $>2.5\%$ has been reported.

The proportion of late implant losses in these patients with a history of periodontitis (overall mean with equal weight to each study: 6.0%, SD 9.5) is high and shows a wide variation ranging from 0% to 41%. The highest implant losses were reported for implants with a rough surface (eight studies: overall mean 14.1%, SD 13.3), *versus* 2.1% (14 studies: SD 3.2) for minimally or moderately rough implants. For studies in which both surfaces had been used (Ellegaard et al. 1997a, b, 2006, Baelum & Ellegaard 2004), implants with a very rough surface always showed higher rates of late implant loss when compared with minimally rough surfaces.

Ten out of the 12 prospective studies reported data on bone-level changes. The annual bone loss after the first year of bone remodelling (five papers: Mengel et al. 2001, Leonhardt et al. 2002, Karoussis et al. 2003, Wennström et al. 2004, Mengel & Flores-de-Jacoby 2005) remains below the 0.1 mm criterium for implant success (Albrektsson et al. 1994), except for patients with aggressive periodontitis (Mengel & Flores-de-Jacoby 2005). Seven studies presented a frequency distribution for the bone-level changes. The proportion of implants with $\geqslant 3$ mm bone loss after a loading period of 3–10 years ranges from 0.0 to 12.3 (but mostly $<6\%$) for minimally rough surfaces, and from 4.3 to 14.3 (but mostly $>10\%$) for rough implants, respectively. In studies with different surface characteristics, rougher implant surfaces always present more bone loss than minimally rough surfaces (Ellegaard et al. 1997a, b, 2006, Baelum & Ellegaard 2004). The probing depth measurements confirm these differences. In several studies, bleeding on probing has been reported. The data for this parameter show a wide variation (2–100%) without clear tendencies. Only two papers reported on the incidence of peri-implantitis. One study with minimally rough implants reported a 0% incidence after 3 years (Yi et al. 2001a, b); another paper on rough implants indicated an incidence of 28.6% after 10 years (Karoussis et al. 2003). Only one paper (Wennström et al. 2004) reported on implant fractures. In this study, involving patients with more than 50% loss of periodontal support in the natural dentition, a relatively high fracture rate of the implants was observed, probably due to the increased mobility of the neighbouring teeth.

Discussion

It is obvious from this review that a final answer to the question of whether there is a relationship between periodontitis and peri-implantitis cannot be provided. The low number of studies and a significant heterogeneity between the studies (SPT, implant surface, lack of data on bone levels) renders this task difficult. Especially the fact that most studies do not report on confounding factors (smoking, oral hygiene and genetic predisposition) makes a good analysis nearly impossible. These conclusions are in agreement with previous review papers on a similar topic (Van der Weijden et al. 2005, Schou et al. 2006).

From the five papers that compared patients with and without a history of periodontitis (Table 1), four clearly indicated a higher incidence of late implant loss/peri-implantitis for the former. The latter indicates either a direct link between tooth loss and implant loss (e.g. microbial load, oral hygiene), or an indirect link via mutual confounding

factors. Important confounding factors are: smoking (for a review, see Chuang et al. 2002a, b, 2005, Klinge et al. 2005, Nitzan et al. 2005, Roos-Jansaker et al. 2006a, b), uncontrolled diabetes (for a review, see Beikler & Flemmig 2003), and genetic predisposition (Gruica et al. 2004, Jansson et al. 2005). Four out of the five above-mentioned studies, however, did not correct their data for these confounding factors.

This review verified the impact of an SPT programme and of the implant surface roughness. In the presence of SPT and with minimally rough implants (Rosenberg et al. 2004, Mengel & Flores-de-Jacoby 2005), the rate of late implant losses/peri-implantitis did not differ between patients with and without a history of periodontitis. The failure rates for minimally and moderately rough implants in the partially edentulous patients with a history of periodontitis also correspond well to the survival data of a global population. Berglundh et al. (2002) reported in a systematic review on partially edentulous patients an implant loss of 2.7% before, and of 2.4% after loading (mostly 5 years). These observations seem to indicate that a history of periodontitis per se is not crucial, but probably the degree of plaque control. The latter is influenced by the SPT programme (Lang & Tonetti 2003, Renvert & Persson 2004), and smooth surfaces are known to reduce supra- and subgingival biofilm formation and maturation (for a review, see Teughels et al. 2006). Patients with a history of aggressive periodontitis, however, are more clearly prone to late failure rates, even when minimally rough implants are used and SPT is given (Mengel & Flores-de-Jacoby 2005).

A first analysis from the papers that only looked to patients with a history of periodontitis (Table 2, non-comparative studies and thus less significant since no control group) confirms the impression of a higher failure rate for implants in periodontitis patients. However, when looking to a subset of studies with either smooth or minimally rough implants in combination with an organized SPT programme, it becomes obvious that the incidence of implant loss remained low. If SPT is lacking (Hardt et al. 2002), significantly more marginal bone loss can be expected. The latter becomes obvious when comparing the Hardt et al. (2002) with the paper of Wennström et al. (2004), who followed a similar study design [similar implants and patient population ($>50\%$ bone loss around teeth)], but now with SPT. They recorded nearly no marginal bone loss around the implants. A lack of a proper SPT may explain the rather high incidence of implants with relevant bone loss in two studies analysing minimally rough implants (Fransson et al. 2005, Roos-Jansaker et al. 2006a, b, c). A low incidence of marginal bone loss was indeed reported in other studies using a similar implant but including SPT (Quirynen et al. 2001, Naert et al. 2004). The relationship between implant surface roughness, based on the classification of Albrektsson & Wennerberg (2004), and late implant loss/peri-implantitis/marginal bone loss is another topic of debate. In this paper, a clear tendency is seen towards more bone loss/higher incidence of implant loss/higher incidence of peri-implantitis around implants with a rough surface. Implants with a rough surface led to a six times higher rate of late implant losses when compared with minimally or moderately rough implants. The latter is in agreement with previous observations (Becker et al. 2000, Åstrand et al. 2004) and the outcome of a previous systematic review (Esposito et al. 2005a, b). The increased marginal bone loss along implants with a rough surface can be explained by the faster biofilm formation and maturation on rough intra-oral surfaces (for a review, see Quirynen & Bollen 1995, Teughels et al. 2006). This factor might thus be more important than the patients' susceptibility to periodontitis. It is of course obvious that the latter has to be confirmed by RCT studies, even though some papers already give a strong indication (Becker et al. 2000, Åstrand et al. 2004).

Several studies tried to correlate marginal bone loss between teeth and implants in the same patient. In none of the papers included in this review were the authors able to show that the marginal bone loss around implants was significantly different from the bone loss around teeth in the same patient (Mengel et al. 2001, Leonhardt et al. 2002, Mengel & Flores-de-Jacoby 2005). Quirynen et al. (2001) compared the marginal bone-level changes around machined surfaced implants and teeth in a group of 100 partially edentulous patients with different periodontal conditions, and were not able to find any correlation between the bone loss around teeth and implants within the same patient, not even over a period of 10 years. Whereas the teeth showed a wide variation in bone loss (0.5 ± 1.0 mm), the implants showed less bone loss with a smaller variation (0.1 ± 0.3 mm), independent of the rate of bone loss around the remaining teeth.

Clinicians are often confronted with the question on whether to keep teeth or to perform tooth extraction and implant placement. In order to answer this question, it is extremely important to compare populations with similar incidences of confounding factors (e.g. smoking, diabetes and genetic predisposition) and socio-economic conditions (including the social security system). Also, the local risk factors (oral hygiene, SPT, number of teeth lost, remaining deep pockets) should be comparable. In patients who lost teeth due to untreatable periodontitis, the starting condition for the inserted implants is already compromised, because tooth loss and bone loss/age are significant risk factors for further periodontal destruction (Lang & Tonetti 2003, Renvert & Persson 2004). Paulander et al. (2004) reported, for 50-year-old individuals, a 4.1% tooth loss over a 10-year period, and an annual marginal bone loss ranging from 0.04 mm (mandibular molars) to 0.08 mm (mandibular incisors). Jansson et al. (2002) observed, in an epidemiological study, a tooth loss of 11.8%, and a mean bone loss of 9–14% of the entire root length over a period of 20 years. Another prospective epidemiological study in Sweden (Hugoson & Laurell 2000, Laurell et al. 2003) reported on 574 dentate individuals, followed for 20 years, an average tooth loss ranging from 0.7% to 14.6%. The average annual marginal bone loss reached 0.1 mm, with a small subset (5%) showing a mean loss of $\geqslant 2$ mm over a 17-year period. When these data are considered, the outcome variables of oral implants, as presented in this review, for patients with a history of periodontitis, seem similar or even better. It might even be more reasonable to compare the implant outcome for this subpopulation with the periodontal parameters around teeth obtained in patients who received periodontal therapy. Fardal et al. (2004) followed 100 consecutive patients (2436 teeth) over a period of 9.8 years (9–11 years), with two to four SPT visits/year and reported a 1.5% tooth loss over this period. Checchi et al. (2002) followed 92 patients with adult periodontitis over

a period of 7 years, and observed a 2% tooth loss, but with patients complying erratically with SPT being at a 5.6 times greater risk. Goldman et al. (1986) examined, retrospectively, 211 patients who were treated for periodontitis in a private practice and maintained for 15–34 years on 3- to 6-month recall schedules. During a mean follow-up of 22 years, no $<13.4\%$ of the teeth were lost. Similar observations have been reported earlier for other populations under regular maintenance (0.23 teeth/patient/year by Tonetti et al. 1998, 0.29 teeth/patient/year by Nabers et al. 1988), whereas Hirschfeld & Wassermann (1978) reported a 0.09 teeth/patient/year. Compared with the data from oral implants, these success rates seem similar, especially in case of SPT.

Conclusion

Implants in patients with a history of periodontitis can function successfully for a long period of time, although slightly higher failure rates have been reported. The latter seems less obvious in the presence of a strict SPT programme. Patients with aggressive periodontitis and/or with very rough implants (S_a values $\geqslant$ of 3 μm) seem more susceptible to peri-implantitis/late implant loss. Longer-term studies, with follow-up periods of 10 years or more, are, however, needed before these statements can be generally accepted.

A clinician should, in partially edentulous patients treated by means of implants, be aware of the relevance of:

- the periodontal health of the remaining dentition, which can interfere with osseointegration,
- the intra-oral translocation of periodontopathogens, which can jeopardize the long-term success of implants because of the similarity in microflora between periodontitis and peri-implantitis, and
- the implant surface roughness.

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Clinical Relevance

Scientific rationale for the study: During periodontal treatment planning, a clinician often has to select between maintaining the natural dentition, or removing it to consider the use of implants. The longevity of implants in patients with a history of periodontitis remains a matter of debate. This review paper compares the outcome of implants in patients susceptible to periodontitis, with a special focus on SPT and on the impact of implant surface roughness.

Principal findings: Within the limitations of this review, one can conclude that both the survival rates, as well as the success rates, of oral implants with minimally and moderately rough surfaces are very high in patients with a history of periodontitis, when SPT is provided. Only in patients with aggressive periodontitis has more implant loss/peri-implant infection been noticed for these implant surfaces. When implants with a very roughened surface are used, the difference in the long-term outcome in patients with a history of periodontitis is clear-cut. These observations, however, still have to be confirmed by large-scale RCT studies.

Practical implications: Even in patients with a history of periodontitis, rehabilitation by means of implants can be advocated especially when combined with SPT and by avoiding implants with a very rough surface.