

The association between selected risk indicators and severity of peri-implantitis using mixed model analyses

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Abstract

Aim: The aim of the study was to assess possible risk indicators for peri-implantitis at different levels of severity using multi-level analyses.

Material and Methods: One hundred and nine subjects attended the examination, 69 females and 40 males. Mean time of implants in function was 8.4 years (standard deviation 4.6) (subject level). The participants were examined clinically and radiographically. Information regarding general health and habits was gathered, with special emphasis on smoking, oral hygiene and susceptibility to periodontitis.

The relation between possible risk indicators and the following features were assessed:

- Detectable peri-implantitis: detectable radiographic bone loss (>0.4 mm) and inflammation
- Overt peri-implantitis: radiographic peri-implant bone loss ≥ 2.0 mm and bleeding on probing /suppuration at pocket probing depth ≥ 4 mm.

Results: Multi-level statistical analyses identified location in the maxilla as risk indicator for detectable peri-implantitis. Regarding overt peri-implantitis, gender (male) and history of periodontitis were identified as risk indicators.

Conclusion: Individuals with a history of periodontitis were prone to peri-implantitis, peri-implant bone loss ≥ 2.0 mm and overt in the present study. No association was found between smoking and peri-implant disease in the present study population.

Key words: dental implants; peri-implant bone loss; peri-implantitis; periodontitis

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During the last decade, several studies have reported complications related to dental implants, either in terms of implant loss or presence of inflammation in combination with loss of bone surrounding the implant, peri-implantitis (Berglundh et al. 2002, Schou 2008).

Conflict of interest and source of funding statement

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The definition and diagnosis of peri-implant disease and health have recently been debated. A definition of peri-implantitis represents the theoretical basis of the term. In contrast, the clinical application of the definition is determined by diagnostic criteria describing the severity of the disease (Heitz-Mayfield 2008, Zitzmann & Berglundh 2008). The significance of various factors related to implant treatment outcome is currently being discussed; surgical techniques, operator skills, implant features such as length, width or surface as well as within-patient

factors such as health and habits (Lindhe & Meyle 2008). However, there is clearly a need for further studies to better understand the relative importance of the many factors involved in peri-implantitis. A risk factor may be defined as “an environmental, behavioural, or biological factor that, if present directly increases the probability of a disease (or adverse event) occurring and, if absent or removed, reduces that probability. Risk factors are part of the causal chain, or expose the host to the causal chain” (Genco et al. 1996). A risk indicator may be defined as a

“probable risk factor that has not been confirmed by carefully conducted longitudinal studies” (Genco et al. 1996). Cause and effect can only be determined by observing subjects over a time period. In order to identify true risk factors for peri-implant disease, prospective longitudinal studies are required. Retrospective and cross-sectional studies can only identify risk indicators for disease (Heitz-Mayfield 2008).

A previously published study discussed how assessment of peri-implantitis at different levels of severity yielded a substantial difference in prevalence. The severities assessed included peri-implantitis with bone loss from very small changes in bone level (>0.4 mm) to ≥ 3.0 mm. Depending on the threshold used, 47–11% of subjects in the study population were recorded with peri-implantitis at one or more implant (Koldsland et al. 2010).

In the consensus report of the Sixth European Workshop on Periodontology, Lindhe & Meyle (2008) concluded that the following indicators were associated with peri-implant diseases: Poor oral hygiene, history of periodontitis and cigarette smoking, but these variables may be assessed in a variety of ways (Heitz-Mayfield 2008, Renvert & Persson 2009). Thus, these possible risk indicators may be subdivided and may be assessed by means of different surrogate parameters.

Smoking and a history of periodontitis have been correlated previously with implant loss (Koldsland et al. 2009) in the present study population. It would therefore be interesting to evaluate if these and other selected variables were associated with peri-implantitis. Furthermore, as the definition of peri-implantitis is based on two features (bone loss and inflammation), it was interesting to assess these features separately.

Previously published studies assessing risk factors and risk indicators for peri-implantitis have mainly based their conclusions on single-level logistic regression analyses (Brocard et al. 2000, Hardt et al. 2002, Karoussis et al. 2003, Baelum & Ellegaard 2004, Evian et al. 2004, Rosenberg et al. 2004, Roos-Jansåker et al. 2006a, b, Fransson et al. 2008). However, in order to account for clustering of implants in subjects, the use of multi-level statistical models may be more appropriate.

The aim of the present study was to assess possible risk indicators for peri-

implantitis at different levels of severity using multi-level statistical models.

Material and Methods

This study was a cross-sectional, clinical study approved by the Regional Committee for Research Ethics, Oslo, Norway (S-06413a) and Norwegian Social Science Data Services, Bergen, Norway (15585).

All participants signed an informed consent.

Definitions applied

- Detectable peri-implant bone loss: radiographic bone loss exceeding the standard deviation (SD) of the measurement errors (>0.4 mm) (Koldsland et al. 2010).
- Peri-implant inflammation: mucosal bleeding index score >0 (Mombelli et al. 1987) and/or bleeding on probing (BOP)/suppuration.
- Detectable peri-implantitis: detectable peri-implant bone loss combined with inflammation.
- Overt peri-implantitis: radiographic peri-implant bone loss ≥ 2.0 mm and BOP/suppuration at pocket probing depth (PPD) ≥ 4 mm.

The definitions are based on thresholds. Hence, all subjects/implants registered with overt peri-implantitis are also registered with detectable peri-implantitis.

Subjects

All 109 subjects included in the present study were registered with implants inserted and suprastructures made at the Institute of Clinical Dentistry, Dental Faculty, University of Oslo, between 1990 and 2005.

Fifteen subjects were edentulous at the present examination. The mean number of teeth in the study population was 17.1 (SD 10.7). The study population comprised 69 females and 40 males, with a mean age of 43.8 years at the time of implant insertion (range: 18–80). The mean time in function was 8.4 years (SD 4.6) (subject level). After initial adjustments of the suprastructures, the participants had not been recalled or maintained by the institute as part of a clinical routine, but the maintenance was to be performed by the referring dentist.

The study population, prevalence of peri-implant disease and time in function have been further described in previous publications (Koldsland et al. 2009, 2010). In short 47.1% of the subjects were registered with detectable peri-implantitis (Koldsland et al. 2010). When the clinical and radiographic diagnostic thresholds of BOP at PPD ≥ 4 mm and peri-implant bone loss ≥ 2.0 mm were combined in order to describe the severity of peri-implantitis, 20.4% of the subjects were diagnosed accordingly at one or more implants (Koldsland et al. 2010).

Implants

A total of 374 solid screw implants had been inserted and the number of implants still in function was 354 at the time of examination (Koldsland et al. 2009). The implants were of different brands (Brånemark System, Nobel Biocare AB, Göteborg, Sweden; Astra Tech, Mölndal, Sweden; Straumann, Basel, Switzerland; Biomet 3i, Palm Beach Gardens, FL, USA). These brands constitute the most commonly inserted implants in Norway. The results implied that 36.6% of the implants were registered with detectable peri-implantitis according to the definition applied (Koldsland et al. 2010).

When the clinical diagnostic thresholds of BOP at PPD ≥ 4 mm and peri-implant radiographic bone loss ≥ 2.0 mm were combined in order to describe the severity of peri-implantitis, 11.4% of the implants in the study population were diagnosed accordingly. Detailed results and time in function are described in a previous publication (Koldsland et al. 2010).

Radiographic and clinical examination

Radiographic assessment was based on full-mouth status (intra-oral analogue pictures) (Promax, Planmeca, Helsinki, Finland) (70 kV, 8 mA). Analogue radiographs from the patient charts and from the present examination were scanned (Epson Perfection V750 PRO, Epson America Inc., Long Beach, CA, USA) before measurements were performed (Image J software, Research Services Branch, NIH, Bethesda, MD, USA). A suitable reference point (fixture–abutment connection or abutment–crown connection) was determined for each implant. The distance from the reference point to the first bone-to-

implant contact was measured at the baseline radiographs and the radiographs from the present examination in order to calculate changes in bone level (Koldsland et al. 2010). Clinical recordings were: presence of plaque and mucosal bleeding at four sites (mesial, buccal, distal and lingual) per tooth/implant, measurements of PPD were performed at four sites per tooth/implant and registered if ≥ 4 mm, BOP/suppuraction and the presence or absence of supraocclusion. Keratinized mucosa was registered as present or absent at the buccal aspect of the implants (Koldsland et al. 2010). Whenever radiographs from time of implant loading were unreliable; the first radiograph meeting the inclusion criteria was used as baseline. Implants with suprastructure of shape, contour and proximity to the mucosa allowing reliable probing and radiographs showing threads at both mesial and distal aspects of the implant sharply projected, were included in the analysis. Criteria for inclusion and exclusion of data into the analyses have been described in detail previously (Koldsland et al. 2010). The clinical examination was performed by one examiner (O. C. K.) after having been calibrated (repeated measurements) by an experienced periodontist (A. M. A.).

PPD was measured using a 0.2 N (20 g) defined force periodontal probe (DB 764 R, University of North Carolina, NC, USA; AESCULAP[®], B Braun, Tuttlingen, Germany).

The suprastructures were not removed before probing.

All examinations were performed from February 2007 to February 2008 at the Institute of Clinical Odontology, University of Oslo, Oslo, Norway.

Information from patient charts

Based on the patient charts at the time of implant insertion, the following anamnestic information was retrieved:

- Subject age.
- Gender.
- Information regarding the subjects' medical conditions and use of medications.
- Periodontal status.
- Smoking habits.
- Reason for tooth loss.
- Brand of implant.
- Suprastructure.
- Complications related to implant treatment.

- Time from implant loading to present examination.

Information from interview

The medical and dental history were supplemented in an interview when the subjects attended the clinical examination:

- Systemic diseases with particular focus on cardiovascular diseases, lung-/respiratory diseases, diabetes, rheumatic diseases, osteoporosis, allergies and immune system deficiencies.
- Medications (including use of antibiotics).
- Oral hygiene habits.
- Visits to dentist/hygienist.
- History of periodontitis and reason for tooth loss.
- Use of tobacco.
- Use of alcohol.
- Complications related to implants; mechanical and biological.

Different modes of stratification regarding oral hygiene, smoking and periodontal status were assessed:

Oral hygiene

- Plaque at $\geq 30\%$ of teeth/implant surfaces or $< 30\%$ of teeth/implant surfaces.
- Reported daily use of inter-dental cleaning tools.
- Reported visits to a dentist/hygienist.
- Presence of plaque *versus* no plaque at implant.

Smoking habits

- Previous and present smokers *versus* subjects who never smoked on a daily basis (Spiekerman et al. 2003).
- Tobacco load; heavy *versus* light smokers according to pack years (< 10 *versus* ≥ 10) (Genco et al. 2005).
- Current daily smoker or not current smoker.

Periodontal status

- History of periodontitis.
- Experience of tooth loss due to periodontitis.

- Radiographic bone level at the remaining teeth.

All subjects registered with periodontitis had received periodontal treatment before implant insertion. The extent of radiographic bone loss at the mesial and/or distal aspects of the remaining teeth was measured from the cemento-enamel junction (CEJ) to the alveolar crest (AC) and registered if the distance CEJ-AC ≥ 4 mm. Subjects were divided into three categories according to periodontal bone loss ≥ 4 mm: 0–30%, 31–50% or 51–100% of teeth with bone loss (Roos-Jans  ker et al. 2006a). Current periodontitis was diagnosed on the basis of the present clinical evaluation if two or more teeth were registered with PPD ≥ 5 mm, BOP and radiographic bone loss ≥ 6 mm (CEJ-AC) (Koldsland et al. 2009).

Data analyses

Statistical analyses included descriptive statistics for clinical parameters at implant and subject level (SPSS for Windows, version 14.0, SPSS Inc., Chicago, IL, USA).

In addition, a multi-level logistic regression model for binary response was performed with the variables inflammation, detectable bone loss, bone loss ≥ 2.0 mm, detectable peri-implantitis and overt peri-implantitis, with random intercepts for patients and implants. The function *xtmelogit* in Stata 11, which fits mixed-effects models for binary/binomial responses, was used. The estimation method implemented in Stata was maximum likelihood (ML) using adaptive quadrature with five integration points; confidence intervals were estimated for all variance components.

The following independent variables were analysed using multi-level analyses:

- Time from loading of implants to current examination (time in function).
- Presence of keratinized mucosa.
- Presence of plaque at implant.
- Location of implant in maxilla.
- Subject gender.
- History of periodontitis.
- Previous or present smoker.

Results

The distribution of different features at subject and implant level in the study

population is summarized in Table 1. The distribution of detectable and overt peri-implantitis is presented in Tables 2 and 3.

The results of the multi-level analyses are presented in Tables 4 and 5. Regarding

the dependent variable "Detectable bone loss", no significant association was found. The dependent variable "Bone loss ≥ 2.0 mm" was significantly associated with the independent variables "Gender (male)" and "His-

tory of periodontitis". The dependent variable "Inflammation" was statistically significant associated with the presence of plaque at the implant, location in the maxilla and gender (male) (Table 4). Regarding "Detectable peri-implantitis", a statistical significant association was found with location of the implant in the maxilla as opposed to the mandible (Table 5). Statistical significant associations were found between the dependent variable "Overt peri-implantitis" and the independent variables "Gender (male)" and "History of periodontitis" when multi-level statistical analyses were performed.

Discussion

Multi-level statistical analyses identified location in the maxilla as a risk indicator for the dependent variable detectable peri-implantitis. Regarding overt peri-implantitis, gender (male) and history of periodontitis were identified as risk indicators.

Multi-level regression analysis was chosen as this model allowed analysis even if dependency may have existed between implant and subject data. However, variables not reported as risk indicators in the present study might have been identified as risk indicators in a larger population as this model is best suited for large populations. Just like any other estimation procedure, this is an approximate method, but by default, xtlogit uses adaptive quadrature with five integration points. However, estimation with higher numbers of integration points may lead to more accurate estimates in some cases. The ML method produces models that are comparable and selection of the best models was based on the likelihood ratio test.

The present study assessed different parameters regarding periodontitis as a possible risk indicator. The statistical analyses indicated that the results were strongly influenced by the selection of surrogate parameters identifying subjects susceptible to periodontitis. Different ways of segregating the population identified many, but not all, of the same subjects (data not shown). The variable "History of periodontitis" included all subjects registered with experience of tooth loss due to periodontitis and subjects registered with bone loss ≥ 4 mm bone at $\geq 30\%$ of the remaining teeth. Hence, the two latter variables comprised fewer subjects than the former.

Table 1. Distribution of selected possible risk indicators at subject and implant level in the study population

Variables	N	%
<i>Subject variables</i>		
Overall	109	
Mean number of teeth present at current examination	17.1 (SD 10.7)	
Edentulous at time of insertion	12	11.0
Edentulous at time of current examination	15	13.8
Female	69	63.3
Male	40	
History of periodontitis	28	25.7
No history of periodontitis	78	
Not recordable	3	
Having lost tooth/teeth due to periodontitis	23	21.1
Lost due to other reasons	82	
Not recordable	4	
Bone loss ≥ 4 mm at $\geq 30\%$ of teeth	23	21.1
Bone loss ≥ 4 mm at $<30\%$ of teeth	71	
Not recordable, edentulous at examination	15	
Current periodontitis	7	6.4
No current periodontitis	102	
Heavy smoker (≥ 10 packyears)	41	37.6
No/light smokers (<10 packyears)	63	
Not recordable	5	
Current daily smoker	18	16.5
Former smoker	41	37.6
Never smoker	50	
Plaque at $\geq 30\%$ of teeth/implant surfaces	11	10.1
Plaque at $<30\%$ of teeth/implant surfaces	98	
Daily inter-dental cleaning	60	55.0
No daily inter-dental cleaning	49	
Recalled by dentist/dental hygienist	82	75.2
Not maintained	27	
Age >45 at time of insertion	55	50.5
Age ≤ 45 at time of insertion	54	
Age >60 at time of insertion	27	24.7
Age ≤ 60 at time of insertion	82	
Cardiovascular disease	17	15.6
No such condition reported	92	
Diabetes	5	4.6
No such condition reported	104	
<i>Implant variables</i>		
Overall	354	
Keratinized mucosa	322	91.0
No keratinized mucosa	30	
Not recordable	2	
Presence of plaque	77	21.8
No plaque	277	
Maxilla	227	64.1
Mandibula	127	
Single crown	96	27.1
Fixed partial prosthesis	99	28.0
Fixed total prosthesis	130	36.7
Removable partial prosthesis	1	0.3
Removable total prosthesis	28	7.9
0–5 years of functional loading	128	36.2
5–10 years of functional loading	106	29.9
>10 years of loading	114	32.2
Missing data	6	1.7

Table 2. Prevalence of detectable and overt peri-implantitis segregated according to possible subject level risk indicators

Feature assessed	Detectable peri-implantitis			Overt peri-implantitis		
	N*	n	%	N*	n	%
Overall	104	49	47.1	103	21	20.4
Female	67	28	41.8	66	9	13.6
Male	37	21	56.8	37	12	32.4
History of periodontitis	25	17	68.0	24	12	50.0
No history of periodontitis	77	32	41.6	77	9	11.7
Experience of tooth loss due to periodontitis	23	14	60.9	22	9	40.9
Tooth lost for other reasons	78	34	43.6	78	11	14.1
Bone loss ≥ 4 mm at $\geq 30\%$ of teeth	20	11	55.0	19	9	47.4
Bone loss ≥ 4 mm at $<30\%$ of teeth	71	32	45.1	71	7	9.9
Current periodontitis	6	4	66.7	6	4	66.7
No current periodontitis	98	45	45.9	97	17	17.5
Smoker/former smoker	55	28	50.9	55	15	27.3
No history of smoking	49	21	42.9	48	6	12.5
≥ 10 packyears	38	20	52.6	38	12	31.6
<10 packyears	62	28	45.2	61	8	13.1
Current daily smoker	17	8	47.1	16	5	31.3
Not current smoker	87	41	47.1	87	16	18.4
Plaque at $\geq 30\%$ of surfaces	9	5	55.6	10	2	20.0
Plaque at $<30\%$ of surfaces	95	44	46.3	93	19	20.4
Daily inter-dental cleaning	58	28	48.3	58	15	25.9
No daily inter-dental cleaning	46	21	45.7	45	6	13.3
No recall by dentist/hygienist	26	11	42.3	25	4	16.0
Regular recall	78	38	48.7	78	17	21.8

*Total number may vary due to missing/uncertain information.

Table 3. Prevalence of detectable and overt peri-implantitis segregated according to possible implant level risk indicators

Feature assessed	Detectable peri-implantitis			Overt peri-implantitis		
	N*	n	%	N*	n	%
Overall	295	108	36.6	333	38	11.4
Keratinized mucosa	273	95	34.8	303	27	8.9
No keratinized mucosa	20	13	65.0	23	6	26.1
Presence of plaque	57	34	59.6	70	9	12.9
No presence of plaque	238	74	31.1	258	24	9.3
Maxilla	198	84	42.4	215	26	12.1
Mandibula	97	24	24.7	118	12	10.2

*Total number may vary due to missing/uncertain information.

Edentulous subjects were obviously not assessed regarding bone loss at the remaining teeth, further decreasing the number of subjects in the analysis. It may be speculated that the sample size affected the outcome, rendering "History of periodontitis" the most conclusive variable regarding association with peri-implantitis in this study. Thus, this variable was further evaluated in the multi-level analyses. In the present study, "Current periodontitis" was defined as two or more teeth registered with PPD ≥ 5 mm, BOP and radiographic bone loss ≥ 6 mm. A similar definition was proposed by Machtei et al. (1992), defining "established periodontitis" as the presence of CAL ≥ 6 mm in two or more teeth and one or more sites with PPD ≥ 5 mm.

Several studies have reported significantly higher incidences of biological complications related to dental implants in subjects susceptible to periodontitis than in periodontally healthy subjects (Hardt et al. 2002, Karoussis et al. 2003, Ferreira et al. 2006, Roos-Jansåker et al. 2006b). On the other hand, some studies report small or statistically insignificant differences regarding implant outcome between periodontitis susceptible subjects and subjects in need of implant therapy for other reasons (Baelum & Ellegaard 2004, Rosenberg et al. 2004). This might be due to the outcome variable assessed. A previous study, reporting from the same study population as the present, concluded that small changes in the diagnostic criteria for peri-implantitis yielded a large difference in the prevalence of disease (Koldslund et al. 2010).

Discrepancies between studies evaluating periodontitis as a risk indicator

Table 4. Extent of bone loss and inflammation related to selected variables in multi-level analyses

Parameter	Detectable bone loss		Bone loss ≥ 2.0 mm		Inflammation	
	random intercept		random intercept		random intercept	
	estimated	95% CI	estimated	95% CI	estimated	95% CI
<i>Fixed part: odds ratio</i>						
Time in function	1.03	0.99–1.07	1.01	1.0–1.02	1.01	1.00–1.02
Keratinized mucosa	0.11	0.00–9.55	0.27	0.06–1.29	0.58	0.14–2.42
Plaque at implant	7.70	0.23–259.16	0.76	0.24–2.43	7.28*	2.28–23.22
Maxilla	3.95	0.21–72.70	1.60	0.52–4.88	3.78*	1.66–8.60
Gender (male)	26.01	0.38–1801.49	4.98*	1.64–15.12	3.34*	1.18–9.45
History of periodontitis	39.70	0.26–6026.88	3.95*	1.18–13.18	1.23	0.36–4.20
Smoker/former smoker	1.07	0.05–22.88	1.71	0.50–5.89	1.54	0.52–4.52
<i>Random part</i>						
Subject	4.96		1.19		1.64	

*The result is statistically significant.

Table 5. Severities of peri-implantitis related to selected variables in multi-level analyses

Parameter	Detectable peri-implantitis		Overt peri-implantitis	
	random intercept		random intercept	
	estimated	95% CI	estimated	95% CI
<i>Fixed part: odds ratio</i>				
Time in function	1.05	1.00–1.09	1.01	1.00–1.02
Keratinized mucosa	0.05	0.00–2.93	0.41	0.08–2.15
Plaque at implant	56.00	0.89–3533.76	0.63	0.17–2.34
Maxilla	177.76*	1.29–24,438.17	1.29	0.37–4.44
Gender (male)	21.75	0.45–1047.99	4.62*	1.28–16.62
History of periodontitis	3.99	0.09–183.55	6.19*	1.40–27.50
Smoker/former smoker	0.34	0.01–9.95	0.86	0.20–3.57
<i>Random part</i>				
Subject	5.54		1.45	

*The result is statistically significant.

for peri-implantitis might be caused by diagnostic criteria regarding peri-implantitis. Such discrepancies might also be caused by the multitude of options related to the diagnosis of periodontitis. The present study compared some of these diagnostic criteria. The variable ‘‘History of periodontitis’’ resembled the variable in the study by Baelum & Ellegaard (2004), but the implant outcome variables differed somewhat. Baelum & Ellegaard reported no difference between subjects with or without a history of periodontitis when implant loss was the outcome variable, but reported a statistical difference when peri-implantitis (exceeding 1.5 mm bone loss) was the outcome variable. Roos-Jans aker et al. (2006b) assessed clinical signs of inflammation and peri-implantitis with bone loss exceeding 1.8 mm. This feature was associated with subjects registered with bone loss ≥ 4 mm at $\geq 30\%$ of the remaining teeth. Hardt et al. (2002) assessed peri-implant bone loss exceeding 2.0 mm from time of abutment connection, a baseline resembling the determined baseline in the present study. An association with history of periodontitis was reported in the study by Hardt et al. (2002) as well as in the present study. However, the criteria determining history of periodontitis were different.

Loss of teeth due to periodontitis has been assessed by Karoussis et al. (2003) and Rosenberg et al. (2004). Both studies reported an association with peri-implantitis. Ferreira et al. (2006) used similar criteria for peri-implantitis and reported similar conclusions as Karoussis et al. (2003), but assessed different criteria determining periodontitis. The

results indicate an association between susceptibility to periodontitis and peri-implantitis.

Having a history of smoking was not associated with peri-implantitis in the present study. This was in contrast to the results reported previously from the same study population regarding loss of dental implants (Koldslund et al. 2009). Interestingly, in the studies by Roos-Jans aker, the opposite results were presented; smoking was associated with peri-implantitis but not with implant loss (Roos-Jans aker et al. 2006a, b). As other studies also report an association between peri-implant disease and smoking habits (Ekelund et al. 2003, Fransson et al. 2008), the results in the present study might have been influenced by the number of participants and/or because registration of smoking habits was based on self report. Another explanation might be that the smoker/former smoker group included too many subjects who had stopped smoking before implant insertion, and/or current smokers may not have obtained signs and symptoms of disease yet.

Plaque at an implant was only associated with inflammation, not with bone loss or peri-implantitis in the present study. On a subject level, plaque present at $\geq 30\%$ of implant/teeth surfaces was relatively rare in the present population. Other studies have frequently reported an association between plaque/poor oral hygiene and peri-implant disease (Ekelund et al. 2003, Ferreira et al. 2006, Serino & Str om 2009). Daily use of inter-dental cleaning tools was frequently reported in the present study. Self-reporting might overestimate the actual compliance in a population. On

the other hand, the results might indicate a population capable of daily implant cleaning and awareness of the consequences of neglecting oral hygiene.

As the definition of peri-implantitis is based on the combination of two features, bone loss and inflammation, it was interesting to evaluate which of these features was the most influential at the different levels of severity described. A strong association was shown between the presence of plaque and inflammation, but no association was found between the presence of plaque and detectable bone loss. These results might illustrate the significance of presenting the influence of a risk indicator on both sub-elements of the definition of peri-implantitis, emphasizing the need of both clinical and radiographic evaluation of dental implants.

All subjects diagnosed with detectable bone loss and/or detectable peri-implantitis might not be in immediate need of interceptive treatment. Subjects with these diagnoses might be in need of closer supervision and maintenance, though. Studies of a prospective longitudinal design are needed in order to follow the progression of peri-implant bone loss. As this was a cross-sectional study, time as a possible risk indicator could only be assessed as a continuous variable related to different thresholds of disease. No association between implant time in function and peri-implantitis was found.

Implants inserted in the maxilla were more likely to be registered with inflammation and detectable peri-implantitis than implants in the mandible. It may be speculated that minor buccal bone loss due to anatomical features (e.g. thin or traumatized buccal bone wall at implants in the maxillary incisor region) might be explanatory rather than progressive bone loss due to peri-implantitis. Implants replacing incisors in the maxilla lost due to trauma often have long abutments, creating pseudo pock-ets. Hence, these implants might be more prone to inflammation according to the definition used in the present study. Fransson et al. (2009) reported significantly more peri-implantitis associated bone loss (exceeding 1.8 mm) at implants placed in the lower front region compared with implants placed in other regions.

In the present study, a higher percentage of the male population was registered with overt peri-implantitis. Whether this was caused by true gen-

der-related genetic traits, or some risk indicator not assessed in the present project, was not evaluated. In contrast, Attard & Zarb (2004) reported that women experienced more peri-implant bone loss than men.

As discussed in previous publications regarding the same study population (Koldsland et al. 2010), strict inclusion criteria were preferred (to a larger sample size) in the present project. The attendance rate was 70%, which is an acceptable attendance rate (Tomasi et al. 2008). The recommendations from the Sixth European Workshop on Periodontology regarding sample size were fulfilled (Zitzmann & Berglundh 2008). Even so, in statistical terms, the present study population was small and this may have affected the results. In a larger population, more risk indicators should have reached statistical significance.

This study presents results regarding implants of different brands and surfaces, but these features have not been assessed. The population was not balanced for the purpose of comparing brands. Furthermore, the implants have been inserted from 1990 to 2005. The development in implant structure- and surface design has been substantial in this time period, both between and within each brand. These developmental changes might be more influential than the mere brand of the implant. Unfortunately, especially regarding the oldest implants in the material, information beyond the brand was scarce.

Longitudinal studies assessing different severities of peri-implant disease and consensus regarding diagnostic criteria identifying periodontitis and peri-implantitis in study populations are warranted.

Conclusion

Implants placed in the maxilla were statistically significantly more prone to be registered with detectable peri-implantitis in the present study. An association between overt peri-implantitis and the variables "history of periodontitis" and "gender (male)" was also observed. No association was found between smoking and peri-implant disease in the present study population.

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References

- Attard, N. J. & Zarb, G. A. (2004) Long-term treatment outcomes in edentulous patients with implant-fixed prostheses: the Toronto study. *International Journal of Prosthodontics* **17**, 417–424.
- Baelum, V. & Ellegaard, B. (2004) Implant survival in periodontally compromised patients. *Journal of Periodontology* **75**, 1404–1412.
- Berglundh, T., Persson, L. & Klinge, B. (2002) A systematic review of the incidence of biological and technical complications in implant dentistry reported in prospective longitudinal studies of at least 5 years. *Journal of Clinical Periodontology* **29**, 197–212.
- Brocard, D., Barthet, P., Bayse, E., Duffort, J. F., Eller, P., Justum, P., Marin, P., Oscaby, F., Simonet, T., Benque, E. & Brunel, G. (2000) A multicenter report on 1,022 consecutively placed ITI implants: a 7-year longitudinal study. *International Journal of Oral & Maxillofacial Implants* **15**, 691–700.
- Ekelund, J. A., Lindquist, L. W., Carlsson, G. E. & Jemt, T. (2003) Implant treatment in the edentulous mandible: a prospective study on Branemark system implants over more than 20 years. *International Journal of Prosthodontics* **16**, 602–608.
- Evian, C. I., Emling, R., Rosenberg, E. S., Waasdorp, J. A., Halpern, W., Shah, S. & Garcia, M. (2004) Retrospective analysis of implant survival and the influence of periodontal disease and immediate placement on long-term results. *International Journal of Oral & Maxillofacial Implants* **19**, 393–398.
- Ferreira, S. D., Silva, G. L., Cortelli, J. R., Costa, J. E. & Costa, F. O. (2006) Prevalence and risk variables for peri-implant disease in Brazilian subjects. *Journal of Clinical Periodontology* **33**, 929–935.
- Fransson, C., Wennström, J. L. & Berglundh, T. (2008) Clinical characteristics at implants with a history of progressive bone loss. *Clinical Oral Implants Research* **19**, 142–147.
- Fransson, C., Wennström, J. L., Tomasi, C. & Berglundh, T. (2009) Extent of peri-implantitis-associated bone loss. *Journal of Clinical Periodontology* **36**, 357–363.
- Genco, R. J., Grossi, S. G., Ho, A., Nishimura, F. & Murayama, Y. (2005) A proposed model linking inflammation to obesity, diabetes, and periodontal infections. *Journal of Periodontology* **76**, 2075–2084.
- Genco, R. J., Jeffcoat, M., Caton, J., Papapanou, P., Armitage, G. C., Grossi, S., Johnson, N., Lamster, I., Lang, N., Robertson, P. & Sanz, M. (1996) Consensus report periodontal diseases: epidemiology and diagnosis. *Annals of Periodontology* **1**, 216–222.
- Hardt, C. R., Gröndahl, K., Lekholm, U. & Wennström, J. L. (2002) Outcome of implant therapy in relation to experienced loss of periodontal bone support: a retrospective 5-year study. *Clinical Oral Implants Research* **13**, 488–494.
- Heitz-Mayfield, L. J. (2008) Peri-implant diseases: diagnosis and risk indicators. *Journal of Clinical Periodontology* **35** (Suppl.), 292–304.
- Karoussis, I. K., Salvi, G. E., Heitz-Mayfield, L. J., Bragger, U., Hammerle, C. H. & Lang, N. P. (2003) Long-term implant prognosis in patients with and without a history of chronic periodontitis: a 10-year prospective cohort study of the ITI Dental Implant System. *Clinical Oral Implants Research* **14**, 329–339.
- Koldsland, O. C., Scheie, A. A. & Aass, A. M. (2009) Prevalence of implant loss and the influence of associated factors. *Journal of Periodontology* **80**, 1069–1075.
- Koldsland, O. C., Scheie, A. A. & Aass, A. M. (2010) Prevalence of peri-implantitis-related to severity of the disease with different degrees of bone loss. *Journal of Periodontology* **81**, 231–238.
- Lindhe, J. & Meyle, J. (2008) Peri-implant diseases: consensus report of the Sixth European Workshop on Periodontology. *Journal of Clinical Periodontology* **35** (Suppl.), 282–285.
- Machtei, E. E., Christerson, L. A., Grossi, S. G., Dunford, R., Zambon, J. J. & Genco, R. J. (1992) Clinical criteria for the definition of "established periodontitis". *Journal of Periodontology* **63**, 206–214.
- Mombelli, A., van Oosten, M. A., Schurch, E. Jr. & Lang, N. P. (1987) The microbiota associated with successful or failing osseointegrated titanium implants. *Oral Microbiology & Immunology* **2**, 145–151.
- Renvert, S. & Persson, G. R. (2009) Periodontitis as a potential risk factor for peri-implantitis [review] [52 refs]. *Journal of Clinical Periodontology* **36** (Suppl.), 9–14.
- Roos-Jansäker, A. M., Lindahl, C., Renvert, H. & Renvert, S. (2006a) Nine-to fourteen-year follow-up of implant treatment. Part I: implant loss and associations to various factors. *Journal of Clinical Periodontology* **33**, 283–289.
- Roos-Jansäker, A. M., Renvert, H., Lindahl, C. & Renvert, S. (2006b) Nine-to fourteen-year follow-up of implant treatment. Part III: factors associated with peri-implant lesions. *Journal of Clinical Periodontology* **33**, 296–301.
- Rosenberg, E. S., Cho, S. C., Elian, N., Jalbout, Z. N., Froum, S. & Evian, C. I. (2004) A comparison of characteristics of implant failure and survival in periodontally compromised and periodontally healthy patients: a clinical report. *International Journal of Oral & Maxillofacial Implants* **19**, 873–879.
- Schou, S. (2008) Implant treatment in periodontitis-susceptible patients: a systematic review. *Journal of Oral Rehabilitation* **35** (Suppl.), 9–22.
- Serino, G. & Ström, C. (2009) Peri-implantitis in partially edentulous patients: association with inadequate plaque control. *Clinical Oral Implants Research* **20**, 169–174.
- Spiekerman, C. F., Hujoel, P. P. & DeRouen, T. A. (2003) Bias induced by self-reported smoking on periodontitis-systemic disease associations. *Journal of Dental Research* **82**, 345–349.
- Tomasi, C., Wennström, J. L. & Berglundh, T. (2008) Longevity of teeth and implants – a systematic review. *Journal of Oral Rehabilitation* **35** (Suppl.), 23–32.
- Zitzmann, N. U. & Berglundh, T. (2008) Definition and prevalence of peri-implant diseases. *Journal of Clinical Periodontology* **35** (Suppl.), 286–291.

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Clinical Relevance

Scientific rationale for the study: The literature describes variables believed to influence the development of peri-implantitis. Some variables are described in a multitude of ways, and their influence has been evaluated at different severities of

disease. It seems important to elucidate risk indicators from different perspectives, applied at the same study population regarding different severities of peri-implantitis using multi-level analyses.

Principal findings: Variables related to history of periodontitis seemed to

affect peri-implantitis at the levels of severity assessed.

Practical implications: Subjects susceptible to periodontitis with implants inserted, should be informed about possible biological complications and should be maintained frequently.

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