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Impact of IL-1 genotype and smoking status on the prognosis of osseointegrated implants

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Abstract

Aim: This study evaluated the impact of the IL-1 genotype and smoking status on the prognosis and development of complications of osseointegrated implants.

Material and methods: The clinical charts of 180 consecutively admitted patients were analyzed with respect to the occurrence of biological complications in conjunction with oral implants. Biologic complications were defined as clinical conditions with suppuration from the peri-implant sulcus, development of a fistula or peri-implantitis with radiologic bone loss. All patients had received one or more ITI[®] dental implants, which had been in function for at least 8 (range: 8–15) years. This patient population had received 292 implants. From these, 51 implants in 34 patients showed late (infectious) biologic complications, and 241 implants had survived without any biologic complications at all.

Results: Of the 180 patients, 53 were smokers, who were subdivided in a series of classes according to their intensity of smoking and 127 were never smokers. Sixty-four of 180 (36%) patients tested positive for the IL-1 genotype polymorphism. This prevalence corresponds to previous reports for the prevalence of European descent populations. The results for the non-smoking group indicated no significant correlation between implant complications and a positive IL-1 genotype. However, there was a clear association for heavy smokers between a positive IL-1 genotype and implant complications. 6 of 12 or half of the heavy smokers and IL-1 genotype-positive patients had either an implant failure, i.e. loss of implant, or a biologic complication during the follow-up period.

Conclusions: These findings have led to the conclusion that there is a synergistic effect between a positive IL-1 genotype and smoking that puts dental implants at a significantly higher risk of developing biologic complications during function.

Although some oral implants such as the ITI[®] system have high survival and success rates, failures and biological and technical complications do occur (Buser et al. 1997; Brägger 1999). Biological complications are generally characterized by signs of peri-implant mucositis and peri-implantitis, which include the development of fistulas, suppuration in the peri-implant sulcus and radiographic signs of marginal bone loss (Mombelli et al. 1987).

Specific bacteria may be an essential factor for the onset of periodontitis. They trigger inflammation in the periodontal tissues. However, the severity of periodontitis is the result of multiple factors that influence the inflammatory response. This, in turn, may be altered by host factors such as the presence/absence of genetic polymorphisms and behavioral factors such as smoking (Genco & Löe 1993). Interleukin-1 (IL-1), prostaglandin E₂ (PGE₂)

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and matrix metalloproteinases (MMP) (Stashenko et al. 1991; Offenbacher 1996) have been associated as inflammatory mediators for periodontal disease severity or activity of progressing disease. It is also known that lipopolysaccharides (LPS) of Gram-negative bacteria stimulate monocytes and macrophages to produce IL-1 (Santamaria et al. 1989). Furthermore, IL-1 plays a central role in bone resorption and destruction of extracellular matrices upregulating MMP activity.

A specific composite genotype of the polymorphic IL-1 gene cluster has been identified (Komman et al. 1997), which appeared to identify adults who, in the presence of a bacterial challenge, were more likely to develop severe periodontitis than age-matched subjects without the respective genotype. Also, the risk for tooth loss in IL-1 genotype positive compared with IL-1 genotype negative patients appeared to be 2.7 times higher (McGuire & Nunn 1999). Smoking and a positive IL-1 genotype influenced tooth loss after periodontal therapy both independently and combined (7.7 times greater likelihood). A retrospective analysis of changes in bleeding on probing (BOP) during periodontal maintenance indicated that BOP was significantly affected by the IL-1 genotype status, with IL-1 genotype-positive patients having a significantly higher BOP percentages (Lang et al. 2000). There is also an association between this specific genotype and increased levels of IL-1 in monocytes *in vitro* (Pociot et al. 1992) and in the gingival crevicular fluid (Engelbreton et al. 1999).

Controversy exists in answering the question of whether or not patients positive for this composite IL-1 polymorphism are also at elevated risk for peri-implant infections. One single provisional report addressing this hypothesis failed to establish a relationship between implant failures and the IL-1 gene polymorphisms (Wilson & Nunn 1999). In this study, more than 50% of the failures were early failures, i.e. occurred during the first year after placement of the implants. Since early failures may result from a variety of factors ranging from surgical trauma to other clinical management aspects, such cases may not appropriately represent failures due to immuno-inflammatory responses that may be influenced by the IL-1 genetic factor.

On the other hand, a recent analysis of 182 implants in 90 consecutively admitted

patients after an average of 5.6 years of function has clearly established that IL-1 genotype-positive heavy smokers demonstrated a significantly increased risk for peri-implant bone loss following prosthetic reconstruction and during the supportive periodontal care phase of the treatment when compared with the IL-1 genotype negative smokers (Feloutzis et al. 2003).

The aim of the present study was (i) to examine the relation between the IL-1 polymorphism and biological complications of osseointegrated ITI® dental implants and (ii) to explore the association between these allelic variants of the IL-1 gene complex and peri-implant complications in both smokers and non-smokers.

Material and methods

Patients and implants

The patients evaluated in this study were recruited as part of a long-term oral implant maintenance population of the Department of Oral Surgery and Stomatology, University of Berne, Switzerland. Of the original 223 patients in the cohort, 180 were available for re-evaluation. The remainder could not be recalled because of moving away from the area or having passed away. All of them were Caucasian (87 males and 93 females) and between the ages of 25 and 90. They were all in good general health. All patients had at least one ITI® implant (Straumann, Waldenburg, Switzerland) in function for at least 8 years, with a range of 8–15 years. All patients were enrolled in a maintenance care program at the Department of Oral Surgery and Stomatology, University of Berne, Switzerland, or in private dental offices.

A total of 292 ITI® dental implants had been installed in different areas of the jaws between 1986 and 1993. Out of these, 67 were of the hollow cylinder design, 137 hollow screws and 88 were solid screws. All implants were placed in a non-submerged modality (one-stage concept) and supported by either single crowns or short-span fixed partial dentures.

All patients had been recalled annually for both a clinical and radiographic examination.

Clinical evaluation

The clinical examinations included the evaluation of the parameters described

previously (Buser et al. 1990), such as suppuration, modified plaque index (mPLI) (Mombelli et al. 1987), modified bleeding index (mBII) (Mombelli et al. 1987), probing depth (PD), the distance between the implant shoulder and the coronal margin of the peri-implant mucosa (DIM) and finally, the attachment level (AL) calculated as the sum of PD + DIM. The 8-year AL values were compared with the 1-year values to evaluate the AL changes around the implants over the period between both examinations (ΔAL_{1y-8y}).

Radiographic analysis

Standardized intra-oral radiographs were obtained applying the paralleling technique at annual intervals following implant installation. Only the 1- and 8-year radiographs were used for analysis. For each implant, the distance between the implant shoulder and the first visible bone-implant contact (DIB) was measured (in mm) by the same blinded examiner (B. G.) at the mesial and distal aspect of each implant (Buser et al. 1990; Weber et al. 1992). The individual DIB value was calculated based on the average of the mesial and distal readings. The 8-year DIB values were deducted from the 1-year DIB values to evaluate the crestal bone changes encountered around the implants over the 7-year period between examinations (ΔDIB_{1y-8y}).

Interleukin-genotype and smoking status

For this study, each patient was recalled for a check-up in addition to the routine maintenance visit of the longitudinal evaluation. On this occasion, a smoking history was assessed and the IL-1 genotype was determined using the PST™ (PST™ Genetic Susceptibility Test: Hain Diagnostika GmbH, Nehren, Germany). The DNA was collected from a swab sample with cheek cells. The PST test identified two polymorphisms by polymerase chain reaction: one located at +4845 bp in the IL-1 α region and one at +3954 bp in the IL-1 β region. The result reported the presence of allele '1' or '2'. A homozygous type was considered if there was an allele 2 in both positions on the IL-1A and IL-1B genes. A heterozygous type was considered if there was only one allele 2 in both positions. Both homo- and heterozygous combinations were considered as IL-1 genotype positive.

Based on a smoking history questionnaire (Ramseier 2003), each patient was classified as either:

- Non-smokers (NS): The patient had never smoked.
- Former light smokers (FLS): The patient had smoked <20 cigarettes/day, but had quit smoking at least 5 years ago.
- Former heavy smokers (FHS): The patient had smoked ≥20 cigarettes/day, but had quit smoking at least 5 years ago. Former smokers (heavy or light) who have quit smoking less than 5 years ago were still considered current smokers.
- Light smokers (LS): These patients were current smokers and smoked <20 cigarettes/day.
- Heavy smokers (HS): These patients were current smokers and smoked ≥20 cigarettes/day.

Statistical analysis

Implants showing a fistula, peri-implant supuration or peri-implantitis with radiographic signs of bone loss in the crestal region were considered as having a biologic complication. The changes of DIB and AL over the follow-up period were obtained from the differences of peri-implant bone measurements and clinical AL between years 1 and 8. The mean peri-implant bone loss/gain and the mean clinical attachment loss/gain for each patient were calculated. Ordinal scores were assigned to patients' smoking status. Former smokers (light and heavy) and never smokers were assigned a score of 0, the current light smokers a score of 1 and the current heavy smokers a score of 2.

The associations of IL-1 genotypes with implant failures/complications, with the presence of bone loss or the presence of clinical attachment loss were determined using χ^2 tests. Student's *t*-tests were used to determine the differences in age, years of smoking and numbers of implants in each patient, between implants with failures/complications and the controls, between the presence and absence of bone loss or between the presence and absence of attachment loss. The non-parametric Wilcoxon test was used to determine the difference in smoking status between the two genotype groups.

Data were analyzed for each implant and each patient according to implant failures/complications, mean DIB and AL. The multivariate linear regression was utilized to assess the associations of mean DIB and mean AL, with risk factors adjusting for confounding factors. The multivariate logistic regression model was used to determine the associations between implant failures/complications, presence of bone loss or presence of attachment loss and risk factors adjusting for confounding variables. Given that treatment responses for implants placed in different subjects are assumed to be statistically independent, and treatment responses within subjects are assumed to be correlated, a generalized estimating equation (GEE) model was used to determine the association between the risk of having radiographic bone loss or having clinical attachment loss and IL-1 genotype and smoking status while adjusting for possible confounding variables. The statistically significant *P*-value was adjusted to $\alpha = 0.01$, according to the Bonferroni effect due to multiple comparisons in the analysis. The statistical analyses were performed using SAS software (Statview[®], SAS Institute, Cary, NC, USA).

Results

Clinical observation

Of the 180 patients, 53 were smokers (including light and heavy smokers) and 127 were non-smokers (including permanent non-smokers and former light and heavy smokers). Sixty-four of 180 (36%) subjects tested positive for the IL-1 genotype. In total, 51 implants with biologic

complications were encountered in 34 patients leaving 241 implants in 146 patients without any complications.

Implants with biologic complications

The associations between biological complications of implants and the IL-1 genotype on the one hand and biological complications and smoking status on the other were determined first, unadjusted for confounding variables. The smoking status was significantly associated with biological implant complications ($P = 0.0012$) (Table 1). However, there was no association between biological implant complications and the IL-1 genotype alone. The possible confounding variables included age, gender, years of service of the implant, numbers of implants placed in each patient and number of years of cigarette smoking. Patients with biological implant complications had smoked significantly longer ($P = 0.01$) and had higher numbers of implants placed in their mouth ($P = 0.0075$) than the patients without biological implant complications (Table 2).

For the non-smoking patients, there was no significant correlation between the IL-1 genotype and biological implant complications. Strikingly, 50% (6/12) of IL-1 genotype-positive heavy smokers had implants that exhibited biological complications during the follow-up period (Fig. 1).

The multivariate logistic regression model showed that, after adjusting for all the confounding variables, only the number of implants placed in each patient ($P = 0.016$) and the interaction of the IL-1 genotype and the smoking status ($P = 0.0079$) were significantly associated with biological

Table 1. Distribution of implant complications by smoking status

Smoking status	Biologic complications	%	No complications	%	Total	
Never and former smokers	17	13	110	87	127	
Light current smokers	3	17	15	83	18	
Heavy current smokers	14	40	21	60	35	
Total	34	19	146	81	180	$P = 0.0012$

Table 2. Comparisons of number of years smoked and number of implants placed between implants with and without failure/complication

	Biologic complications		No complications		<i>P</i>
	Mean	SD	Mean	SD	
Number of years smoked	24.5	13.7	17.2	10.2	0.0101
Number of implants placed	2.00	1.07	1.54	0.85	0.0075

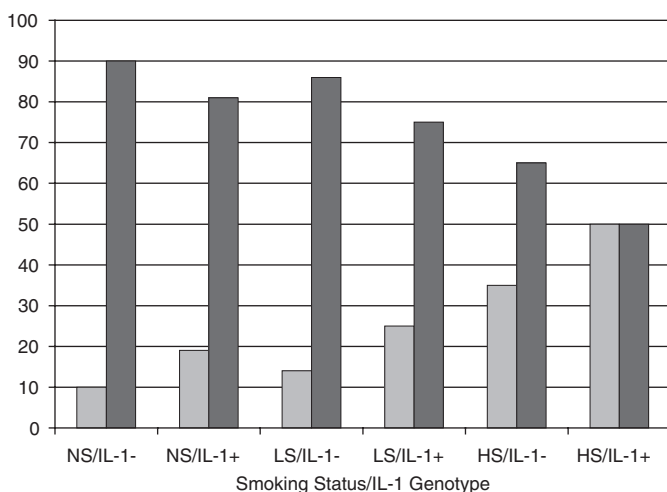


Fig. 1. Complications by IL-1 genotype and smoking status.

Table 3. Association of implant complications and risk factors using multivariate logistic regression model

Variables	Estimate	OR	99% CL	P
Interaction between smoking and IL-1 genotype	0.84	2.32	1.0–35.23	0.0079
Number of implants	0.46	1.59	0.97–2.60	0.0158

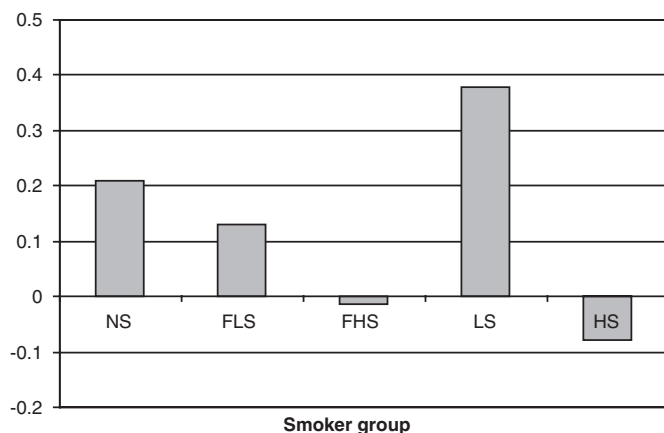


Fig. 2. Change of bone-implant contact (DIB) by smoking status: NS, non-smokers; FLS, former light smokers (< 20 cigarettes/day); FHS, former heavy smokers (≥ 20 cigarettes/day); LS, light smokers (< 20 cigarettes/day); HS, heavy smokers (≥ 20 cigarettes/day).

implant complications (Table 3). Thus, for the IL-1 genotype negative patients, smoking was not associated with biological implant complications, and for the non-smoking patients (including former smokers), IL-1 genotypes had no effects on biological complications. However, for IL-1 genotype positive current light smokers, the odds ratio of having biological implant complications was 2.35 (95% CL: 1.376–4.021); for the IL-1 genotype-positive heavy smokers, the odds ratio for biological implant complications was 4.15 (95% CL: 2.089–8.243).

Radiological measurement of bone loss

If the radiographic bone changes between years 1 and 8 (ΔDIB_{1y-8y}) were positive, actual bone gain around the implant over the 7-year period may be assumed. Conversely, if the ΔDIB_{1y-8y} levels were negative the patient had obviously lost bone around the implant.

The associations of ΔDIB_{1y-8y} and all the risk factors have been evaluated in two different modes. First, each implant ($n = 271$) was treated as an individual unit. However, by using the GEE model, all the implants within each individual were con-

sidered to be related. Secondly, each patient was treated as an individual unit by using the mean ΔDIB_{1y-8y} of all implants in the mouth.

Implants in patients with a positive IL-1 genotype ($n = 90$) had statistically more peri-implant bone loss (mean: -0.06 , SD 0.57 mm) than those of patients with a negative IL-1 genotype ($n = 181$) (mean: 0.22 , SD 0.61 mm) ($P = 0.0004$). Smoking habits were also associated with ΔDIB_{1y-8y} ($P = 0.0397$) (Fig. 2, Table 4).

The multivariate linear regression model showed that only the IL-1 genotype ($P = 0.0023$) and smoking status ($P = 0.0168$) were statistically significantly associated with the amount of ΔDIB_{1y-8y} for each implant. Other variables, such as age, gender, years of service of the implants or years of smoking were not associated with ΔDIB_{1y-8y} . The GEE model was used to determine the effect of the IL-1 genotype and smoking on the presence of peri-implant bone loss during the maintenance period of 7 years, by considering that all the implants within each subject were related. The IL-1 genotype ($P = 0.0022$) and smoking ($P = 0.0205$) were significantly associated with the peri-implant bone loss adjusting for all other variables (Table 5).

Figure 3 demonstrates the distribution of bone loss among implants of patients with different IL-1 genotypes and smoking status (Table 6).

When each patient was treated as an individual unit, the mean values for ΔDIB_{1y-8y} from all the implants in each patient ($n = 174$) were calculated. In six patients, this could not be performed because of missing implants as a consequence of biological complications. The results were similar to the findings described above. The IL-1 genotype positive patients ($n = 61$, mean ΔDIB_{1y-8y} : -0.050 , SD 0.60 mm) had significantly more bone loss than the IL-1 genotype negative population ($n = 113$, mean ΔDIB_{1y-8y} : 0.22 , SD 0.57 mm) ($P = 0.0036$). Again, the multivariate logistic regression model was used to determine the association between the presence of bone loss in patients and the risk factors. Only the IL-1 genotype ($P < 0.0001$) was statistically associated with the peri-implant bone loss after adjusting for all other variables. Smoking was not associated with peri-implant bone loss ($P = 0.09$). The odds ratio for the IL-1 genotype positive patients

Table 4. Change of DIB by smoking status

	Mean changes DIB (y 1–y 8)	SD	n
NS	0.21	0.64	63
FLS	0.13	0.35	25
FHS	−0.014	0.66	39
LS	0.38	0.64	18
HS	−0.08	0.54	35

DIB, bone–implant contact, NS: non-smokers, FLS, former light smokers; FHS: former heavy smokers; LS, light smokers; HS, heavy smokers.

Table 5. Association of peri-implant bone loss and risk factors using the GEE model

Variables	Parameter estimates	Standard error	P
IL-1 genotype	0.85	0.28	0.0022
Smoking	0.38	0.17	0.0205

GEE, generalized estimating equation.

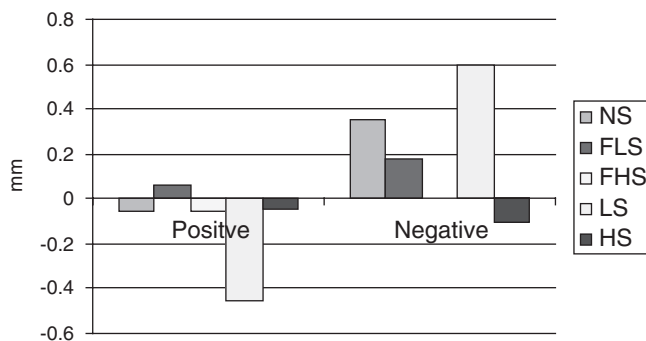


Fig. 3. Change of bone–implant contact (DIB) in mm by IL-1 genotype and smoking status: NS, non-smokers; FLS, former light smokers (<20 cigarettes/day); FHS, former heavy smokers (≥20 cigarettes/day); LS, light smokers (<20 cigarettes/day); HS, heavy smokers (≥20 cigarettes/day).

Table 6. Change of DIB by genotype and smoking status

	Smoking status				
	Genotype				
	NS	FLS	FHS	LS	HS
Mean					
Positive	−0.06	0.063	−0.06	−0.45	−0.041
Negative	0.35	0.18	0.004	0.6	−0.1
SD					
Positive	0.68	0.37	0.56	0.43	0.44
Negative	0.57	0.34	0.71	0.49	0.59
N					
Positive	22	11	11	4	12
Negative	41	14	28	14	23

DIB, bone–implant contact, NS: non-smokers, FLS, former light smokers; FHS: former heavy smokers; LS, light smokers; HS, heavy smokers.

to have bone loss over the 7-year maintenance period was 3.75 (99% CI 1.57–8.95) after adjusting for the smoking status.

Clinical AL

If AL at year 8 was greater than AL at year 1, the implant was considered having lost attachment over the maintenance period. Implants of heavy smokers showed signifi-

cantly more attachment loss (ΔAL_{1y-8y}) than implants of patients with lighter smoking habits or non-smokers over the 7-year period. ($P < 0.0001$) (Table 7).

No significant differences in clinical attachment changes (ΔAL_{1y-8y}) were noted between patients with a positive and those with a negative IL-1 genotype. The multivariate linear regression analysis showed

that the clinical attachment changes were only barely, but significantly correlated with the patients' age ($P = 0.03$) and smoking status ($P = 0.02$). A GEE model was used to determine the associations between the presence of attachment loss and all the risk factors. Only the smoking status was highly significantly associated with the mean clinical attachment loss ($P = 0.0006$). In each patient, changes of mean clinical AL were only associated with the patient's smoking habits ($P = 0.0074$).

Discussion

In the present study, patients recruited from an ongoing prospective cohort study who underwent implant therapy 8 years before were analyzed for biological complications and peri-implant bone loss in relation to their IL-1 genotype status and smoking habits. All patients included yielded an observation period of exactly 7 years. Consequently, no adjustments had to be made of any of the parameters regarding various times of implant service. In this cohort, 36% of the patients were of the IL-1-positive genotype. This proportion is in agreement with previous reports of Caucasian populations (Korhonen et al. 1997; Gore et al. 1998; Lang et al. 2000; McDevitt et al. 2000; Cullinan et al. 2001; Feloutzis et al. 2003).

The study population consisted of 29% heavy and light smokers, thus representing the national average of smoking patients in Switzerland (Janin-Jacquat & François 1999). Most of the patients sought implant therapy because of tooth loss due to dental decay, trauma or agenesis, not because of chronic periodontitis. This, in turn, means that, from the aspect of biological complications, the patient cohort represented a relatively low-risk population (Karoussis et al. 2003). Hence, less than one-fifth (19%) of all the patients had a biological complication during the observation period of 8 years. As biological complication, all inflammatory lesions such as mucositis, suppuration, development of a fistula or peri-implantitis with radiographic bone loss were included irrespective of the etiologic factors.

The present study has clearly demonstrated that IL-1-positive heavy smokers were at high risk for both the development

Table 7. Mean of AL change (in mm) among different smoking status

	Change of AL	
	Mean	SD
Non-smokers (never, former light and heavy)	- 0.49	0.9
Current light smokers	- 0.42	0.84
Current heavy smokers	- 0.97	0.73

AL, attachment level.

of biological complications and increased peri-implant marginal bone loss with odds ratios of 5.35 and 3.75, respectively. In IL-1 genotype negative patients, smoking did not have a significant impact on either the development of biological complications or peri-implant bone loss in the present patient cohort. Nor did IL-1-positive non-smokers reveal an increased risk for biological complications or bone loss. These results are in agreement with recent studies on the occurrence of peri-implantitis (Feloutzis et al. 2003) or periodontitis (Axelsson 2002), which also points to the significant impact of the combination of smoking and IL-1-positive genotypes in the pathogenesis of chronic periodontitis and peri-implantitis. They, however, do not support the conclusions presented in earlier studies (Wilson & Nunn 1999), in which no association was reported between the IL-1 genotype and implant failures. The reason for the discrepancy between the results of the present and this previous preliminary study (Wilson & Nunn 1999) certainly lies within the selection of the patients. While the present study incorporated only patients with an 8-year follow-up and evaluated a radiographic documentation of 7 years prospectively, the former study (Wilson & Nunn 1999) encompassed patients with a short observation period and hence, reported predominantly on early implant failures. In this context, it must be assumed that the influence of surgical technique could not be assessed.

In the present study, IL-1 genotype negative smokers did not present with a significantly increased risk for biological complications and/or peri-implant bone loss. In this respect, the results of the present study, again, corroborated those of a recently published report on implant patients (Feloutzis et al. 2003). Elevated rates of implant failures have also been associated with heavy smoking in studies in which the IL-1 genotypes of the patients

were unknown (Bain & Moy 1993). The authors of these studies recommended smoking cessation programs to be performed prior to implant placement (Bain 1996).

From the clinical point of view, the results of the present and previous studies (Feloutzis et al. 2003) should be implicated in the decision-making process during treatment planning. It would be reasonable to assume that higher risks for biological complications and consequently, the development of peri-implantitis are to be assumed in heavy smokers of the IL-1-positive genotype seeking therapy. It is, therefore, recommended to analyze the risk profile of those patients and inform them accordingly.

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Résumé

Le but de cette étude a été d'évaluer l'impact du génotype IL-1 du tabagisme sur le pronostic et le développement de complication des implants buccaux ostéointégrés. Les dossiers cliniques de 180 patients ont été analysés en ce qui concerne l'arrivée de complications biologiques en association avec les implants buccaux. Des complications biologiques étaient définies en tant que conditions cliniques avec suppuration depuis le sillon paroiimplantaire, le développement d'une fistule ou d'une paroiimplantite avec perte osseuse radiologique. Tous les patients avaient reçu un ou plusieurs implants buccaux ITI qui avaient été en fonction pour au moins huit années (de 8 à 15 ans). Les patients avaient reçu 292 implants. Parmi ceux-ci 51 chez 34 patients ont montré des complications biologiques tardives (infectieuses) et 241 avaient survécu sans aucune complication biologique. Des 180 patients, 53

étaient fumeurs qui ont été divisés en une série de classes suivant l'intensité du tabagisme tandis que 127 étaient non-fumeurs. Soixante-quatre des 180 patients (36%) ont été testés positifs pour le polymorphisme du génotype IL-1. Cette fréquence globale correspond au rapport précédent pour la fréquence globale des populations européennes. Les résultats pour le groupe non-fumeur indiquait qu'il n'y avait aucune relation significative entre les complications implantaire et un génotype IL-1 positif. Cependant, il y avait une association évidente pour les gros fumeurs entre le génotype IL-1 positif et des complications implantaire. Six des douze soit la moitié des gros fumeurs et des patients au génotype IL-1 positif avaient soit un échec implantaire (c.-à-d. la perte de l'implant) soit une complication biologique durant le suivi. Ces résultats ont conduit à la conclusion qu'il y a un effet synergétique entre le génotype IL-1 positif et le tabagisme mettant les implants buccaux à un risque beaucoup plus important à développer des complications biologiques durant leur mise en fonction.

Zusammenfassung

Der Einfluss des IL-1 Genotyps und der Rauchgewohnheiten auf die Prognose von osseointegrierten Implantaten

Ziel: Diese Studie untersuchte den Einfluss des IL-1 Genotyps und der Rauchgewohnheiten auf die Prognose und auf die Entwicklung von Komplikationen bei osseointegrierten Implantaten.

Material und Methoden: Die Krankengeschichten von 180 aufgenommenen Patienten wurden auf biologische Komplikationen in Zusammenhang mit Zahni Implantaten untersucht. Biologische Komplikationen wurden definiert als klinische Zustände mit Suppuration aus dem periimplantären Sulcus, Entwicklung einer Fistel oder Periimplantitis mit radiologischem Knochenverlust. Alle Patienten waren mit einem oder mehreren ITI® Implantaten versorgt worden, welche für mindestens 8 Jahre (Bandbreite: 8–15 Jahre) in Funktion standen. Diese Patientenpopulation war mit 292 Implantaten versorgt worden. Von diesen 292 Implantaten zeigten 51 Implantate bei 34 Patienten biologische (infektiöse) Komplikationen und 241 Implantate hatten ohne biologische Komplikationen überlebt.

Resultate: Von den 180 Patienten waren 53 Raucher, welche in Untergruppen gemäss Schweregrad des Rauchens aufgeteilt wurden und 127 waren Nicht-Raucher. 64 der 180 Patienten (36%) zeigten ein positives Testergebnis bezüglich IL-1 Genotyp Polymorphismus. Diese Prävalenz entspricht früheren Berichten über die Prävalenz bei einer Population europäischer Abstammung. Die Resultate der Nicht-Raucher Gruppe zeigten keine signifikanten Korrelationen zwischen Implantatkomplikationen und einem positiven IL-1 Genotyp. Jedoch bestand bei den starken Rauchern eine klare Assoziation zwischen dem positiven IL-1 Genotyp und Implantatkomplikationen. 6 von 12 oder die Hälfte der starken Raucher und IL-1 Genotyp positiven Patienten zeigten entweder einen Implantat Misserfolg, z.B. Verlust des Implantats, oder eine

biologische Komplikation während der Beobachtungsperiode.

Schlussfolgerung: Die Ergebnisse haben zur Schlussfolgerung geführt, dass ein synergistischer Effekt zwischen einem IL-1 positiven Genotyp und Rauchen besteht, welcher Zahni Implantate einem signifikant höheren Risiko für die Entwicklung von biologischen Misserfolgen während der Funktion aussetzt.

Resumen

Intención: Este estudio evaluó el impacto del genotipo IL-1 y el estatus de fumador en el pronóstico y desarrollo de complicaciones de implantes orales osteointegrados.

Material y métodos: Se analizaron las historias clínicas de 180 pacientes admitidos consecutivamente en lo referente a la ocurrencia de complicaciones biológicas en conjunción implantes orales. Se definieron las complicaciones orales como condiciones clínicas con supuración del surco periimplantario, desarrollo de una fístula o de periimplantitis con pérdida radiológica de hueso. Todos los pacientes recibieron uno o mas implantes orales ITI®, que levaban al menos 8 (rango 8–15) años en función. Esta población de pacientes recibió 292 implantes orales. De estos, 51 implantes de 34 pacientes mostraron (infecciones) complicaciones biológicas

cardías, y 241 implantes sobrevivieron sin ninguna complicación biológica.

Resultados: De los 180 pacientes, 53 eran fumadores que se subdividieron en una serie de categorías de acuerdo con su intensidad de consumo de tabaco y 127 nunca fueron fumadores. 64 de 180 (36%) dieron positivo al test del polimorfismo genético de la IL-1. Esta prevalencia corresponde con informes previos de prevalencia en poblaciones de origen europeo. Los resultados para el grupo de los no fumadores no indicó relación significativa entre complicaciones de los implantes y genotipo IL-1 positivo. Sin embargo, hubo una asociación clara para los fumadores intensos entre complicaciones de los implantes y un genotipo IL-1 positivo. 6 de 12 o la mitad de los fumadores intensos y los pacientes genotipo IL-1 positivo tuvieron o fracasó del implante i.e. pérdida del implante o una complicación biológica durante el periodo de seguimiento.

Conclusiones: Estos hallazgos nos han conducido a la conclusión de que hay un efecto sinérgico entre genotipo IL-1 positivo y tabaquismo que colocan a los implantes orales en un riesgo significativamente mas alto para desarrollar complicaciones biológicas durante la función.

要旨

目的: 本研究は骨性結合した口腔インプラントの合併症発症に対して、IL-1遺伝子型と喫煙が及ぼす影響を評価した。

材料と方法: 連続患者 180名の臨床カルテを、口腔インプラントに関連する生物学的合併症の発症について分析した。生物学的合併症は、インプラント周囲歯肉溝からの排膿、フィステルの形成あるいはレントゲン像上の骨喪失を伴うインプラント周囲炎と定義した。全ての患者には1本以上のITI®口腔インプラントが入っており、少なくとも8年間(8–15年)機能していた。この患者群に292本のインプラントが装着されていた。このうち、患者34名のインプラント51本が晚期(感染性)の生物学的合併症を示し、241本のインプラントには生物学的合併症は全くなく存続していた。

結果: 患者180名のうちの喫煙者35名は、喫煙の程度によってさらに分類した。127名は非喫煙者であった。180名(35%)のうち、64名はIL-1遺伝子型の多形性検査が陽性であった。この頻度は、過去の報告におけるヨーロッパ系母集団の頻度と一致している。非喫煙者群の結果は、インプラント合併症とIL-1遺伝子型の陽性の間に相関性を示さなかった。しかしヘビースモーカー群では、陽性のIL-1遺伝子型とインプラント合併症の間に明確な相関性が認められた。ヘビースモーカー群の半数、すなわち12名のうちの6名とIL-1遺伝子型陽性患者では、インプラントの失敗すなわちインプラントの喪失が生物学的合併症が追跡期間中におこっていた。

結論: これらの所見から、陽性のIL-1遺伝子型と喫煙の間には相乗作用があり、口腔インプラントの機能中に生物学的合併症が生じるリスクが有意に高くなると結論された。

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