Influence of smoking on the survival rate of various dental implant systems - an 18-year report

[<u>Deutsche Version</u>: Einfluβ des Rauchens auf die Verweildauerwahrscheinlichkeit dentaler Implantate unterschiedlicher Systeme unter Berücksichtigung der Dosisabhängigkeit]

G. Gomez-Roman¹, E.L. Maier², D. Lukas³

¹ Priv.-Doz. Dr. med. dent, oral surgeon, senior physician in the Department of Prosthodontics and Medical Matherials (medical director: Univ.-Prof. Dr. med.-dent. H. Weber), Center for Dentristry and Oral Medicine, University Hospital at Eberhard Karls University Tübingen, Germany

² Dr. med. dent, practicing in Leinfelden-Echterdingen, Germany

³ M. Sc. Physics, formerly scientific associate in the of Oral and Maxillofacial Surgery, (medical director: Univ.-Prof. Dr. med. Dr. med. dent. S. Reinert), Center for Dentristry and Oral Medicine, University Hospital at Eberhard Karls University Tübingen, Germany

This paper is dedicated to our teacher Prof. Willi Schulte on the occasion of his 75th birthday

Mailing address:

Priv.-Doz. Dr. G. Gomez-Roman, Poliklinik für Zahnärztliche Prothetik und Propädeutik, Universitätsklinikum Tübingen, Osianderstrasse 2-8, D-72076 Tübingen, Germany, Fax: +49-7071-293 982, e-mail: german.gomez-roman@med.uni-tuebingen.de

Abstract

Influence of smoking on the survival rate of various dental implant systems – an 18– year report

Purpose: This inquiry was done to reveal possible negative effects of smoking on the probability of survival of implants. *Patients and Methods:* The influence of smoking on the success of dental implants was studied using 934 implant patients who had been treated with various implant systems. 712 patients (76.1%) identified themselves as non-smokers, 223 (23.9%) as smokers. A total of 1776 implants including Bonefit (212), Brånemark system (42), Frialit-2 (555), IMZ (179), TPS (21), ITI (1) and Tübingen (761) types were placed in these 934 patients. 1379 implants (77.7%) were placed in non-smokers, 379 (22.3%) in smokers. *Results:* The smokers showed higher failure rates and significantly reduced implant survival rates (Kaplan-Meier). The negative consequences of smoking were evident even at a very low rate of tobacco consumption per day. The degree of influence resulting from smoking depended on the location of the implant. This was most clearly noticed with a reduction of the implant prognosis in the maxilla. *Conclusions:* The particularly marked reduction in the probability of success in the maxilla over the entire survival period of the implant led to the hypothesis that the primary causes for the reduced implant prognosis was long-term local effects. These effects seemed to be the result of two causes: the toxic chemical effect of components of tobacco smoke on the peri-implant tissue and the damage caused by forces below the threshold of perception which affect the implants. The forces result from soft tissue movements, which are increased by smoking.

Key words: smoking, dental implants, clinical study

Introduction

Restoration with implant-supported prostheses meets the patient's desire for the closest possible substitute for the original dentition¹⁻⁵.

To reduce the risk of complications as much as possible, a number of prerequisites must be met for any planned dental implant procedure. The prerequisites have been developed in consensus conferences for implant dentistry⁶.

Gomez-Roman, Maier, Lukas

Smoking as a possible risk factor has been discussed in the literature⁷⁻¹³ when investigating the reasons for implant losses that cannot be explained by generally accepted causes. Many of the effects of smoking have long been known and have been thoroughly researched¹⁴⁻²¹. The effects of smoking on the periodontal tissues have also been confirmed in numerous studies²²⁻²⁷. In comparison to non-smokers with the same standard of oral hygiene, the periodontal parameters of probing depth, furcation involvement and odontosis in smokers generally show significantly poorer values²², and the alveolar bone is often more atrophied^{26,28-30}. In contrast, the gingiva seems less likely to bleed in spite of greater plaque deposition resulting from smoking^{27,31,32}. Clarke, Shepard and Hirsch pointed to an ischemia of the gingiva caused by nicotine as the reason for the reduced tendency to bleed^{27,31-33}. Kraal and Chancellor and also MacFarlane and Herzberg pointed to an impediment to migration and a nicotine-induced functional defect of the polymorphonuclear leukocytes in the area of the marginal gingiva that could result in an inhibition of the clinical inflammatory symptoms^{27,31-35}.

In addition to the known influence on healthy tissue, the negative effects on wound healing of very different types caused by smoking have also been confirmed³⁶⁻³⁹. After oral surgical procedures, problems with the healing of wounds occur far more frequently in smokers than in non-smokers⁴⁰. Sweet and Butler⁴¹ and Siebert, Sonner and Reichart⁴² observed an increased occurrence of problems with wound healing after removal of mandibular third molars.

The success rate of periodontal therapy in non-smokers is often better than that of smokers both with conservative and with surgical procedures, as measured by the reduction of probing depth^{32,43-48}.

Negative effects on the post-operative wound healing have also been observed in connection with implant dentistry. Jones and Triplett⁴⁰ found extended healing processes and increased occurrence of problems with wound healing in smokers after intraoral bone grafting with simultaneous implantation. Small et al.⁴⁹ reported complications with pre-implant surgical procedures.

Publications on the possible effects of smoking on the probability of success for dental implants are available for Brånemark system implants only. Bain and Moy studied 2,194 Brånemark system implants in 545 patients and found a total loss rate of 5.92% ⁷. In their study, the implant had been in place between 1 and 81 months. When sorted by smokers and non-smokers, the loss rate for smokers was significantly higher at 11.28% compared to 4.76% for non-smokers. The survival rate of the implants until loss was not considered in this study, so it is not possible to distinguish between early losses in the healing phase and late losses in the functional phase.

De Bruyn and Collaert in their retrospective study¹⁰ also examined early losses of Brånemark system implants (i.e. losses before restoration with the prosthetic superstructure) and found significantly higher loss figures in smokers (9%) compared to non-smokers (1%). Gorman et al.⁵⁰ published an interim evaluation of a more comprehensive dental implant study, which also confirmed an increased loss rate in smokers. Smoking is considered to have a causal effect in connection with the occurrence of peri-implant changes seen as a possible precursor of implant loss^{51,52}. In addition, Bain has noted that the implant prognosis improves if the patient stops smoking⁹.

The purpose of this article was to investigate possible negative effects of smoking on the probability of survival of implants in patients of the Tübingen implant research register^{53,54}. The results could have an immediate influence on counseling implant patients on the dangers of smoking in connection with dental implant treatment and also on planning dental implant treatment in general.

Material and Methods

The data derived from a patient survey were linked to the data of the Tübingen implant register in an anonymous form. The patients' smoking habits and their possible effects on the probability of survival of the implants they received were determined in the course of a subsequent study.

There was no possibility of finding 353 patients. All of the other 1 352 patients who regularly participated actively in post implant treatment (cf. <u>Table 1</u>) and who had

attended the post-implant examinations were contacted. They had received a total of 2 542 implants representing various systems.

A questionnaire and a letter was sent to these patients⁵⁵. They were prepared in cooperation with the Psychological Institute of the University of Tübingen in a similar form to comparable studies⁵⁶⁻⁵⁸. The questions regarding smoking habits were integrated into a complex of questions to prevent manipulation of answers. The most important questions were formulated as casually as possible to ensure a spontaneous answer from the patient without tactical considerations.

A subject was assessed *as a smoker* for an implant as follows: 1. if the question "do you smoke?" is answered with "yes" and no times are given, 2. if the patient only stopped smoking after placement of the implant. *Non-smokers* are the following: 1. if the question "do you smoke?" is answered with "no" and no times are given, 2. if the patient stopped smoking at least three years before placement of the implant. It had to be assumed that in patients that had stopped smoking just shortly before implant placement (less than three years before) the side effects of smoking had not disappeared completely. In addition, the number of such patients was so small that they could ignored without distorting the results.

Data pertaining to other than the patients' smoking habits were taken exclusively from the Tübingen implant register. This was because the register data are more reliable regarding number of implants, time of operation and implant data than information given by the patient on the survey form. The data were given in anonymous form for this article

The data were statistically evaluated with the commercial statistics program SAS⁵⁹. A distinction was made between losses during the healing phase and losses during functional loading.

The Kaplan-Meier method⁶⁰⁻⁶³ was used to calculate an estimated value for all times observed during the entire period of observation of the implants. This gives the probability that an implant represented by a spot test is still *in situ*. The graph of the estimated value

curve is the Kaplan-Meier curve (see <u>Fig 1</u>). A 95% confidence interval is shown for every estimate.

To compare the Kaplan-Meier curves between different implant groups (e.g., implants in smokers or non-smokers, implants in the maxilla or mandible), the log-rank test for equality of the two groups was run⁶⁴. This means that the probability of survival for the two implant groups in question are equal was tested.

It can be assumed that the survival time of multiple implants in the patient's mouth is interdependent (e.g. a traumatic influence). In practice such simultaneous losses occur rather scarcely ⁶⁵.

On the other hand, the random selection of a single implant may be the source for distortion. The selection of a failed implant or the selection of a successful one is of strong significance if only very few losses could be observed, as in case of the implant systems used by us.⁶⁵

Consequently, the estimated survival time analysis according to Kaplan-Meier were performed in this study without selecting one implant per patient.

Patients with an average daily consumption of up to 10 cigarettes, "light smokers", were distinguished from smokers with an average consumption of more than 10 cigarettes, who were classified as "heavy smokers".

The connection between implant location and effect of smoking was evaluated using separate studies for the maxilla and mandible.

The implants were, of course, also examined considering other factors: implant type, length, diameter, type of prosthetic restoration, other dental measures if necessary, diseases etc. These factors can not be excluded or influenced in a treatment-related study. However, in a preliminary examination it had been assessed that relevant factors (implant type, prosthetics) were equally present in the control groups and sub-groups.

Results

1 000 of the 1 352 questionnaires sent out were returned within two months. This corresponds to a return rate of 74%. A total of 934 (93%) of the returned questionnaires could be evaluated (see <u>Table 2</u>). Of the 66 returned questionnaires that could not be evaluated, 19 were not completed, 16 were incompletely filled out and 31 had clearly implausible information.

Description of patients and placed implants

The 934 patients whose information could be evaluated consisted of 473 female and 461 male. <u>Table 3</u> shows the number of patients in the study divided by *gender* and shows the division into smokers and non-smokers. There were 346 non-smoking male patients and 115 male smokers. There were 365 female non-smokers and 108 female smokers. Fig 2 shows only the 180 patients who supplied information on the type and quantity of their tobacco consumption. The amount of tobacco consumed ranged from one to 40 cigarettes, pipes or cigars per day. There were four pipe-smokers and six cigar smokers among the smokers. These two groups were not evaluated separately from the cigarette smokers because of the small number in each group.

Implants: a total of 1,776 implants could be evaluated for the 934 patients who returned completed questionnaires; 1,379 implants (77.7%) were placed into non-smokers and 397 implants (22.3%) were placed into smokers.

Of the total of 1,776 placed implants 916 were placed into female (52%) and 860 into male (48%). <u>Table 4</u> shows the number of implants placed by gender and the distribution of smokers and non-smokers.

All implants in the patients involved were considered for the study of the *age distribution* of the patients at the time of the operation. The result of this is that patients who received multiple implants appear several times in the study with their age at the time of the implant in question.

Based on the number of all implants, the average age at the time of implant placement was 34.8 years. The youngest patient was 9 years old at the time of implantation, the oldest 81 years. Fig 3 shows the patients classified into five age groups separated by smokers and non-smokers. The number of smokers among the implant patients decreased with increasing age.

The patients included in the study received implants of the following types: Bonefit, Brånemark system, Frialit-2, IMZ, ITI-Extensions, TPS and Tübingen (<u>Fig 4</u>)

The *prosthetic indication* that resulted in the placement of an implant was documented preoperatively in the implant register. Five indications defined by the clinical situation were defined: single-tooth replacement, distal-extension situation, larger tooth-bounded gap (n>1) and edentulous maxilla and mandible.

Most implants that could be evaluated were placed to replace a single tooth (862), as shown in <u>Fig 5</u>. Next was distal-extension restoration with 295 implants and then restoration of edentulous mandible with 291 implants. Wide gaps were restored using 210 implants and 118 implants were placed to restore the edentulous maxilla.

The individual implant *locations* are summarized in four jaw regions (Fig 6): anterior and posterior teeth in the maxilla and mandible. No distinction was made between the right and left sides. The anterior teeth include the incisors and the canines. Most implants (821) were placed in the anterior maxilla for both smokers and non-smokers. Next was the posterior mandible (434 implants) and the anterior mandible (286 implants). 235 implants were placed into the posterior maxillary region.

Probability of success and implant loss

Early losses: 60 implants were lost before prosthetic restoration. The percentage of implants lost prior to prosthetic restoration is 4.3% for smokers⁶⁶ and 3.1% for non-smokers⁶⁷. The slightly higher number (by a factor of 0.4) of implants lost before prosthetic restoration in smokers is <u>not</u> significant, as shown by the chi-squared test.

Total observation period: the loss rate among smokers over the entire observation period was 17% compared to 10% among non-smokers. This corresponds to an increased implant loss rate among smokers compared to non-smokers at the level p=0.05 significant by the factor 1.7:1.

The *estimated values for the probable survival rate* calculated by the Kaplan and Meier method for smokers and non-smokers are compared in Fig 7. The lines show the estimated values and the circular series mark the limits of the 95% confidence intervals. The confidence limits of the values for smoker implants are shown by light circles while the confidence limits of the values for non-smoker implants are shown by dark circles. The logrank test for smokers compared to non-smokers resulted in the p-value of 0.001, marking the two estimated value curves shown in Fig 7 as significantly different. This confirms that the probable survival rate for implants in smokers is significantly lower than that for nonsmokers over the observation period. This is also shown by the confidence intervals, which with sufficient case numbers do not overlap in the first years.

Amount of tobacco: the loss rate among heavy smokers (over 10 cigarettes a day) was 20% compared to 16% among patient who smoke less. However, this difference is not significant (chi-squared test at 5% levels). Fig 8 shows the estimated value curves for implant success in connection with the amount of tobacco smoked for the smokers who provided information on the amounts smoked. The p-value of 0.94, result of the log-rank test, characterizes the two estimated value curves for heavy smokers and light smokers as not significantly different. This is also confirmed by the strong overlap of the two curves in Fig 8.

Implant site: the connection between implant location and implant success was studied with a comparison between maxilla and mandible. The loss rate for *maxillary implants* in smokers was 21.3% compared to 11.6% for non-smokers. This difference was significant at the 5% level. Fig 9 shows the probable survival rates and confidence intervals for maxillary implants in comparison between smokers and non-smokers. The p-value of 0.0002, result of the log-rank test, characterizes the two curves as significantly different, i.e. the probable survival rates of maxillary implants for smokers was significantly reduced.

The loss rate for *mandibular implants* in smokers was 8.1% and for non-smokers 7.0%, i.e. only a coincidental difference. Fig 10 shows probable survival rates and associated confidence intervals for non-smokers and smokers with mandibular implants. The p-value of 0.66, result of the log-rank test, characterizes the two curves as not significantly different.

Discussion

The high return rate of 69.1% indicates that the sample under study is a representative cross section of the Tübingen implant patients and leads to the conclusion that the patients have a close relationship with the Tübingen clinic. This assumption is supported by the comparative age and gender distribution of the sample patient group and the frequency of the occurrence of the various implant types used, the proportions of which correspond to total proportions of the implant types used in Tübingen⁶⁸. The connection between the age distribution of the patients and implant indication shows that the data material is representative: a high proportion of patients in the age groups under 40 with an equally high proportion of implants, which were placed for restoration of interdental spaces with an emphasis on the anterior maxillary region. Restoration of patients with single-tooth implants in the anterior region is a particularly frequent implant indication in Tübingen^{60,63}.

The high number of implants evaluated (N = 1776) makes the absolute number of implants placed in smokers (N =397) sufficiently high, in spite of the relatively low percentage, to allow reliable evaluation of possible effects of smoking. The study by Bain and Moy⁷ examined 390 implants in smokers, while De Bruyn and Collaert¹⁰ examined 114.

It is notable that the proportion of implants placed in smokers in this study was only about one fifth of all implants. The proportion of smokers in the population over 16 years was calculated at 34.6% in 1992⁶⁹. The smaller number of smokers among the implant patients is possibly the result of a higher health consciousness in this group.

The failure rate during the healing phase was not significantly different between smokers and non-smokers (4.3% loss in smokers compared to 3.1% for non-smokers). This result is comparable with that of De Bruyn and Collaert¹⁰ and Lambert, Morris, et al. ⁷⁰.

Comparison of implant loss rates for smokers and non-smokers for the entire observation period yielded a loss rate higher by a factor 1:1.7 for smokers compared to non-smokers. This result suggests that smoking significantly increases the danger of implant loss.

The comparison of the probable survival rate values as per Kaplan and Meier very clearly shows how much the implant prognosis is adversely affected by smoking with significantly reduced values (p=0.0006) over the entire observation period for implants placed in smokers. Any *dosage dependency* for the effects of smoking could not be found in this study (see Fig 8). The proven negative effects of smoking on the prognosis of success of implants were clearly shown even with relatively low daily consumption. Comparison with other implant studies is not possible in this case, because none of the comparable studies addressed the question of whether the amount smoked affects the probable survival rate. A dosage dependency has been confirmed with natural teeth for periodontal bone atrophy²¹ and the success of periodontal therapies^{45,46}.

The marked detrimental effect of smoking on maxillary implants, which has also been observed by other authors^{7,9,10,45,46,51}, allows conclusions on the effects of tobacco smoke to be drawn. Possibly, the implants placed in the maxilla are affected in their success rate because they are more strongly subjected to the local effect of tobacco smoke and its ingredients than the mandibular implants, which are protected by the tongue. This may lead to the conclusion that damages of the osseointegration are due especially to the local effect of residual tobacco smoke on the peri-implant tissues, even if experimental results are still missing.

It can be hypothesized that the mechanisms of the effects of tobacco smoke discussed in connection with dental surgery and periodontology operate similarly in implant dentistry and that nicotine-induced effects such as gingival ischemia³³ and functional disturbances of the polymorphous cellular nucleus leukocytes^{34,35} also contribute to the deterioration of the implant prognosis.

In addition to these chemical-toxicologic effects, which are induced by components of tobacco smoke, mechanical factors could play a role in connection with possible implant losses caused by smoking. It has long been known that touch sensitivity and pressure

reception is reduced by a factor of approximately 10 in implants compared to natural teeth^{72,73}. Because there are no tissue-binding peri-implant structures with implants, pressure forces acting on the implant are transferred directly to the surrounding bone without transformation. The higher threshold of perception for effective forces has the result that forces that the tongue and soft tissues may exert on implants⁷⁴ cannot be perceived, although they can cause pressure loading on the bone and can adversely affect integration of the implant into the bone. As a result, such loads are more dangerous to the implant than strong chewing loads, which can be perceived and result in corresponding protective reactions. The imperceptible loads also occur more often.

Forces that might damage the implant because they are below the threshold of perception also occur in connection with smoking. During suction on a cigarette, lip pressure and tongue movements increase. This can lead to imperceptible but damaging forces acting on the implants. The finding that the probable survival rate for maxillary implants is particularly strongly reduced supports this hypothesis, as the anterior maxilla is particularly exposed to soft tissue movements⁷⁴.

Conclusions

This study supports the hypothesis that smoking must be considered a risk factor in implant dentistry. This result is not surprising since the negative effects of smoking are well known. Numerous studies that show smoking as a cause of damage to intraoral tissue^{22,27-29} and as a negative effect on the healing of wounds⁴⁰⁻⁴² also point to the negative effects of smoking on the probable survival rate of implants.

The exact mechanisms of influence on implants resulting from smoking ultimately cannot be explained by this study. However, the results do support the hypothesis that the local effects of smoking also play a role, particularly if their effects are extended over a longer period, similar to those discussed in connection with the effects of smoking on the intraoral tissue and wound healing in that area³³⁻³⁵. In addition, a negative influence on implants caused by pressure loading below the threshold of perception is likely from the increased movements of tongue and soft tissue caused by smoking.

The significantly higher risk of implant loss caused by smoking should lead to the, unfortunately utopian, demand that implant patients stop smoking. Because the risk of loss for smokers was higher than for non-smokers over the entire observation period, regardless of the amount smoked, the placement of an implant should be the cue for general abstinence from nicotine not only during the healing phase. Patients who smoke should be intensively counseled on the potential negative effects on the success of the dental implant rehabilitation.

Acknowledgement

We would like to thank Mrs. Keller for translating this paper, Mr. M. Kruppenbacher for his assistance with the statistical analysis.

This study was supported by the German Research Council (DFG).

References

- 1. Günay H, Veltmaat A, Schneller T, Neukam FW. Psychologische Aspekte bei Patienten nach Implantatversorgung. Dtsch Zahnärztl Z, 1991; 46: 698-701.
- 2. Harle TJ, Anderson JD. Patient satisfaction with implant-supported prostheses. Int J Prosthodont, 1993; 6: 153-162.
- 3. Wismeijer D, Vermeeren JI, van Waas MA. Patient satisfaction with overdentures supported by one-stage TPS implants. Int J Oral Maxillofac Implants, 1992; 7: 51-55.
- 4. Zimmer CM, Zimmer WM, Williams J, Liesener J. Public awareness and acceptance of dental implants. Int J Oral Maxillofac Implants, 1992; 7: 228–232.
- Hultin M, Gustafsson A, Klinge B. Long-term evaluation of osseointegrated dental irnplants in the treatment of partly edentulous patients. J Clin Periodontol, 2000; 27: 128-133.
- 6. Schnitman PA, Shulman LB. Recommendations of the consensus development conference on dental implants. J Am Dent Assoc, 1979; 98: 373-377.
- 7. Bain CA, Moy PK. The association between the failure of dental implants and cigarette smoking. Int J Oral Maxillofac Implants, 1993; 8: 609-615.
- 8. Bain CA, Moy PK. The influence of smoking on bone quality and implant failure. Int J Oral Maxillofac Implants, 1994; 9: 123-123.
- 9. Bain CA. Smoking and implant failure benefits of a smoking-cessation protocol. Int J Oral Maxillofac Implants, 1996; 11: 756-759.

- 10. De Bruyn H, Collaert B. The effect of smoking on early implant failure. Clin Oral Implants Res, 1994; 5: 260-264.
- 11. Lindquist LW, Carlsson GE, Jemt T. A prospective 15-year follow-up study of mandibular fixed prostheses supported by osseointegrated implants. Clinical results and marginal bone loss. Clin Oral Implants Res, 1996; 7: 329-336.
- 12. Minsk L, Polson AM, Weisgold A, Rose LF, Sanavi F, Baumgarten H, Listgarten MA. Outcome failures of endosseous implants from a clinical training center. Compend Contin Educ Dent, 1996; 17: 848-856.
- 13. Sennerby L, Roos J. Surgical determinants of clinical success of osseointegrated oral implants: A review of the literature. Int J Prosthodont, 1998; 11: 408-420.
- 14. Adler J, Hensel O. Über intravenöse Nikotineinspritzungen und deren Einwirkung auf die Kaninchen-Aorta. Dtsch Med Wchnschr, 1919; 32: 1826–1828.
- 15. Jousilahti P, Vartiainen E, Tuomilehto J, Puska P. Symptoms of chronic bronchitis and the risk of coronary disease. Lancet, 1996; 348: 567–572.
- 16. Krueger JK, Rohrich RJ. Clearing the smoke: the scientific rationale for tobacco abstention with plastic surgery. Plast Reconstr Surg, 2001; 108: 1063–1073.
- 17. Liloglou T, Ross H, Prime W, Donelly RJ, Spandidos DA, Gosney JR, Field JK. p53 gene aberrations in non-small-cell lung carcinomas from a smoking population. Br J Cancer, 1997; 75: 1119-1124.
- 18. Martin LM, Bouquot JE, Wingo PA, Heath CW. Cancer prevention in the dental practice: oral cancer screening and tobacco cessation advice. J Public Health Dent, 1996; 56: 336-340.
- 19. Mishima Y. Thromboangiitis obliterans (Buerger's disease). Int J Cardiol, 1996; 15: 185-187.
- 20. Scully C, Ward-Booth RP. Detection and treatment of early cancers of the oral cavity. Crit Rev Oncol Hematol, 1995; 21: 63-75.
- 21. Telivuo M, Kallio P, Berg MA, Korhonen HJ, Murtomaa H. Smoking and oral health: a population survey in Finland. J Public Health Dent, 1995; 55: 133-138.
- 22. Bergström J, Eliasson S. Noxious effect of cigarette smoking on periodontal health. J Periodontal Res, 1987; 22: 513-517.
- 23. Burt BA. Position Paper of the Direction of the Commitee on Research, Science and Therapy of the American Academy of Periodontology: Epidemiology of Periodontal Diseases. J Periodontol, 1996; 67: 935-945.
- 24. Demirel K, Gur H, Meric H, Sevuk C. Damping characteristics of teeth with periodontal breakdown: correlation of mobility meter values with bone and attachment loss. J Periodontol, 1997; 68: 166-171.
- 25. Genco RJ. Current view of risk factors for periodontal diseases. J Periodontol, 1996; 67: 1041-1049.

- 26. Machtei EE, Dunford R, Hausmann E, Grossi SG, Powell J, Cummins D, Zambon J, Genco R. Longitudinal study of prognostic factors in established periodontitis patients. J Clin Periodontol, 1997; 24: 102–109.
- 27. Preber H, Kant T, Bergström J. Cigarette smoking, oral hygiene and periodontal health in Swedish army conscripts. J Clin Periodontol, 1980; 7: 106-113.
- 28. Bergström J, Eliasson S. Cigarette smoking and alveolar bone height in subjects with a high standard of oral hygiene. J Clin Periodontol, 1987; 14: 466-469.
- 29. Bergström J, Eliasson S, Preber H. Cigarette smoking and periodontal bone loss. J Periodontol, 1991; 62: 242-246.
- 30. Lindquist LW, Carlsson GE, Jemt T. Association between marginal bone loss around osseointegrated mandibular implants and smoking habits: A 10-year follow-up study. J dent Res, 1997; 76: 1667-1674.
- 31. Ismail AI, Burt BA, Eklund SA. Epidemiologic patterns of smoking and periodontal disease in the United States. J Am Dent Assoc, 1983; 106: 617-623.
- 32. Preber H, Bergström J. Occurrence of gingival bleeding in smoker and non-smoker patients. Acta Odontol Scand, 1985; 43: 315-320.
- 33. Clarke NG, Shepard BC, Hirsch RS. The effects of intra-arterial epinephrine and nicotine on gingival circulation. Oral Surg, 1981; 52: 577-582.
- 34. Kraal JH, Chancellor MB, Bridges RB, Bemis KG, Hawke JE. Variations in the gingival polymorphonuclear leukocyte migration rate in dogs induced by chemotactic autologous serum and migration inhibitor from tobacco smoke. J Periodontal Res, 1977; 12: 242-249.
- 35. MacFarlane GD, Herzberg MC, Wolff LF, Hardie NA. Refractory periodontitis associated with abnormal polymorphonuclear leukocyte phagocytosis and cigarette smoking. J Periodontol, 1992; 63: 908–913.
- 36. Lovich SF, Arnold PG. The effect of smoking on muscle transposition. Plast Reconstr Surg, 1994; 93: 825-828.
- 37. Mosely LH, Finseth F, Goody M. Nicotine and its effect on wound healing. Plast Reconstr Surg, 1978; 61: 570-575.
- 38. Rees TD, Liverett DM, Guy CL. The effect of cigarette smoking on skin-flap survival in the face lift patient. Plast Reconstr Surg, 1984; 73: 911-915.
- 39. Siana JE, Rex S, Gottrup F. The effect of cigarette smoking on wound healing. Scand J Plast Reconstr Surg, 1989; 23: 207-209.
- 40. Jones JK, Triplett RG. The relationship of cigarette smoking to impaired intraoral wound healing. J Oral Maxillofac Surg, 1992; 50: 237-239.
- 41. Sweet JB, Butler DP. The relationship of smoking to localized osteitis. J Oral Surgery, 1979; 37: 732-735.
- 42. Siebert O, Sonner S, Reichart PA. Prospektive Studie zu Wundheilungsstörungen nach operativer Weisheitszahnentfernung im Unterkiefer. Dtsch Zahnärztl Z, 1995; 50: 75-75.

- 43. Ah MK, Johnson GK, Kaldahl WB, Patil KD, Kalkwarf KL. The effect of smoking on the response to periodontal therapy. J Clin Periodontol, 1994; 21: 91–97.
- 44. Grossi SG, Skrepcinski FB, DeCaro T, Zambon J, Cummins D, Genco RJ. Response to periodontal therapy in diabetics and smokers. J Periodontol, 1996; 67: 1094–1102.
- 45. Grossi SG, Zambon J, Machtei EE, Schifferle R, Andreana S, Genco RJ, Cummins D, Harrap G. Effects of smoking and smoking cessation on healing after mechanical periodontal therapy. J Am Dent Assoc, 1997; 128: 599-607.
- 46. Kaldahl WB, Johnson GK, Kashinath DP, Kalkwarf KL. Levels of cigarette consumption and response to periodontal therapy. J Periodontol, 1996; 67: 675–681.
- 47. Preber H, Bergström J. Cigarette smoking in patients referred for periodontal treatment. Scand J Dent Res, 1986; 94: 102–108.
- 48. Preber H, Bergström J. Effect of cigarette smoking on periodontal healing following surgical therapy. J Clin Periodontol, 1990; 17: 324-328.
- 49. Small SA, Zinner ID, Panno FV, Shapiro HJ, Stein JI. Augmenting the maxillary sinus for implants: Report of 27 patients. Int J Oral Maxillofac Implants, 1993; 8: 523-528.
- 50. Gorman LM, Lambert PM, Morris HF, Ochi S, Winkler S. The effect of smoking on implant survival at second-stage surgery:DICRG Interim Report No. 5. Dental Implant Clinical Research Group. Implant Dent, 1994; Fall, 3: 165-168.
- 51. Haas R, Haimbock W, Mailath G, Watzek G. The relationship of smoking on peri-implant tissue: a retrospective study. J Prosthet Dent, 1996; 76: 592-596.
- 52. Weyant RJ. Characteristics associated with the loss and peri-implant tissue health of endosseous dental implants. Int J Oral Maxillofac Implants, 1994; 9: 95-102.
- 53. d'Hoedt B, Lukas D. Statistische Ergebnisse des Tübinger Implantates. Dtsch Zahnärztl Z, 1981; 36: 551-562.
- 54. Schulte W, Kleineikenscheidt H, Schareyka R, Heimke G. Konzept und Prüfung des Tübinger Sofortimplantates. Dtsch Zahnärztl Z, 1978; 33: 319–325.
- 55. Maier, E.-L.Hat das Rauchen einen Einfluss auf die Verweildauerwahrscheinlichkeit dentaler Implantate? 1999. Diss med dent Univ Tuebingen. 1999, Diss med dent Univ Tübingen.
- 56. Albrektsson T, Blomberg S, Branemark A, Carlsson GE. Edentulousness an oral handicap. Patient reactions to treatment with jawbone–anchored prostheses. J Oral Rehabil, 1987; 14: 503–511.
- 57. Grogono AL, Lancaster DM, Finger IM. Dental implants: a survey of patients` attitudes. J Prosthet Dent, 1989; 62: 573-576.
- 58. Hartmann HJ. Implantate aus der Sicht von Implantatträgern Eine Pilotstudie. Quintessenz, 1989; 9: 7217–1–7217/8.
- 59. SAS II. SAS Language: Reference, Version 6. Cary, NC, USA: SAS Institute Inc., 1990.

- 60. Gómez-Román G, Schulte W, d'Hoedt B, Axmann-Krcmar D. The Frialit-2 implant system: Five-year clinical experience in single-tooth and immediately postextraction applications. Int J Oral Maxillofac Implants, 1997; 12: 299-309.
- 61. Koch WL. Die zweiphasige enossale Implantation von intramobilen Zylinderimplantaten-IMZ. Quintessenz, 1976; 27: 21-27.
- 62. Mau J. Die Quantifizierung des Verlustrisikos dentaler Implantate. Z Zahnärztl Implantol, 1987; 3: 58-63.
- 63. Schulte W, d'Hoedt B, Axmann-Krcmar D, Gómez-Román G. 15 Jahre Tübinger Implantat und seine Weiterentwicklung zum Frialit-2-System. Z Zahnärztl Implantol, 1992; 8: 77–96.
- 64. Altman DG. Kaplan-Meier survival curve. In: AnonymousPractical Statistics for Medical Research. London, Glasgow, New York, Tokyo, Melbourne, Madras: Chapman & Hall, 1993: 368-371.
- 65. Gómez-Román, G. Eine vergleichende Untersuchung über Einzelzahnimplantationen unter besonderer Berücksichtigung möglicher prognostischer Faktoren. 2000, Habil med dent thesis Univ Tübingen.
- 66. d'Hoedt B. 10 Jahre Tübinger Implantat aus Frialit. Eine Zwischenauswertung der Implantatdatei. Z Zahnärztl Implantol, 1986; 2: 6-10.
- 67. Kaplan EL, Meier P. Nonparametric estimation from incomplete observations. J Am Statist Assoc, 1958; 53: 457-457.
- 68. d'Hoedt B, Schulte W. A comparative study of results with various endosseous implant systems. Int J Oral Maxillofac Implants, 1989; 4: 95–105.
- 69. Kunsch K. Der Mensch in Zahlen. Stuttgart: Gustav Fischer, 1997.
- 70. Lambert PM, Morris HF, Ochi S. The influence of smoking on 3-year clinical success of osseointegrated dental implants. Ann Periodontol, 2000; 5: 79-89.
- 71. Martinez-Canut P, Lorca A, Magan R. Smoking and periodontal disease severity. J Clin Periodontol, 1995; 22: 743-749.
- 72. Mühlbradt L, Meyle J, Lukas D, Schulte W. Die Tastsensibilität Tübinger Sofortimplantate. Dtsch Zahnärztl Z, 1980; 35: 334–338.
- 73. Mühlbradt L, Mattes S, Ulrich R, Möhlmann H. Zur zeitlichen Stabilität von Tastschwellen bei Tübinger Implantaten und natürlichen Zähnen. Z Zahnärztl Implantol, 1993; 9: 139–143.
- 74. Schulte W, Miller E. Druckbelastung des Alvolarfortsatzes durch Zunge und periorale Muskulatur. Dtsch Zahnärztl Z, 1962; 17: 416-416.

Table 1	Times how long all 934 patients were followed. Mean value: 5.47 years
---------	---

Follow-up time	Number of patients	
1	114	
2	126	
3	96	
4	113	
5	98	
б	83	
7	57	
8	52	
9	63	
10	42	
12	44	
14	6	
16	34	
18	6	
years		
		<u>Return to the text</u>

Result	Number	Rate
evaluation possible:	934	69.1%
not returned:	352	26.0%
clearly implausible information:	31	2.3%
not completed:	19	1.4%
incompletely filled out:	16	1.2%
Sum:	1352	100.0%
		<u>R</u> e

Table 2Return rate and usefulness of 1352 questionnaires sent out.

Result	Number	Rate
female smokers	108	11.6%
female non-smokers	365	39.1%
male smokers	115	12.3%
male non-smokers	346	37.0%
473 female and 461 male	934	100.0%

Table 3Gender distribution of patients

Table 4Distribution of implants by gender

Result	Number	Rate
female smokers	202	11.4%
female non-smokers	714	40.2%
male smokers	195	11.0%
male non-smokers	665	37.4%
916 placed in females; 860 in male	1776	100.0%

Return to the text