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Effect of Smoking and Locations of Dental Implants on Peri-Implant Parameters: 3-Year Follow-Up

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Background: Smoking may be a risk factor for marginal bone loss (MBL) and oral mucosal inflammation surrounding dental implants. This retrospective study evaluated the effects of smoking on dental implants in patients with fixed implant-supported prostheses over a period of 36 months following loading.





Material/Methods: We assessed 120 patients (68 women, 52 men, ages 19–74 years (mean age: 55.10 years) with 315 implants. Implants were classified according to location in the upper and lower jaws and anterior (placed between canines) or posterior (placed between pre-molars and molars) as follows: 1=maxilla anterior, 2=maxilla posterior, 3=mandible anterior, 4=mandible posterior. We also measured MBL, plaque index (PI), sulcus bleeding index (SBI), and probing depth (PD). P-values less than 0.05 were considered statistically significant.

Results: MBL was statistically greater in smokers ($P<0.001$) as compared to non-smokers in both jaws. MBL did not vary significantly by location in either group (smokers: $p=0.415$; non-smokers: $p=0.175$). Mean PI and PD scores were significantly higher in smokers as compared to non-smokers ($P<0.001$). A positive correlation was found between PI and PD scores in both groups. No statistically significant difference in SBI was observed between the 2 groups ($P>0.05$).

Conclusions: Smoking was associated with increases in marginal bone loss around implants, independent of their location in the jaws. Also, both plaque indices and probing depths were greater in smokers than in non-smokers.

MeSH Keywords: **Alveolar Bone Loss • Dental Implants • Smoking • Soft Tissue Infections**

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Background

Implant-supported restorations offer extremely effective and predictable treatment of complete and partial edentulism. However, while implants enjoy high success and survival rates [1], the incidence of peri-implant disease has been gradually increasing [2]. An important factor in implant failure, peri-implant disease occurs as a result of a disruption in the balance between bacteria and host-response following osseointegration [3]. Any efforts at prevention and treatment of peri-implant disease must clearly address the contributing factors [4], which include poor oral hygiene, smoking, a history of periodontitis, diabetes mellitus, genetic factors, alcohol consumption, and implant surface characteristics, all of which have been mentioned as possible risk factors in the development of peri-implant disease [5,6].

Smoking has been highlighted as a predisposing factor for implant failure in a number of previous studies [7,8]. The first study to identify smoking as a major factor in implant failure was conducted by Bain and Moy [9], who reported significantly higher implant failure rates in smokers (11.28%) as compared to non-smokers (4.76%), with failures of 44 out of 390 implants in smokers and only 86 out of 1804 placed in non-smokers. Nicotine, the chemical component responsible for tobacco dependence, mediates the hemodynamic effects of smoking and is thought to play a role in the pathogenesis of numerous diseases [10]. The specific effect of smoking on the bone-implant interface reflects a number of direct and indirect systemic and local effects on bone metabolism. For example, Eick et al. [11] reported larger numbers of pathogenic bacteria surrounding implants in smokers as compared to non-smokers; the authors suggested that these bacteria play an important role in peri-implant inflammation and that both smoking and periodontal disease are risk factors in pathogenic colonization of implants.

By examining MBL and periodontal markers, this retrospective study evaluated the effects of smoking on dental implants in patients with fixed implant-supported prostheses over a period of 36 months following loading. The first null hypothesis was that peri-implant probing depths, plaque index scores, sulcus bleeding index scores, and marginal bone losses would not differ significantly between habitual smokers and individuals who had never used any tobacco product. The second null hypothesis was that marginal bone loss around implants placed in smokers and non-smokers would not vary according to the location of the implant in the jaws.

Material and Methods

This retrospective study was conducted using records from 120 patients (68 women, 52 men) ages 19–74 years (mean

age: 55.10 years) with 315 implants placed between 2012 and 2019. The study protocol was approved by the local ethics committee of Eskisehir Osmangazi University's Faculty of Medicine (Protocol decision No: 21/29.01.2019).

Inclusion criteria were: systemically healthy individuals aged >18 capable of performing oral-health self-care; either no previous use of any tobacco product ('non-smoker') or habitual smoking of more than 10 cigarettes/day for at least 2 years [12] ('smoker'); availability of a completed anamnesis form with data on sex, age, and tobacco use, as well as a digital panoramic radiograph from the time of loading; and presence of an implant-supported fixed prosthesis with masticatory-functional implant loading for at least 36 months. Exclusion criteria were: any systemic disease (e.g., prediabetes, xerostomia, diabetes mellitus, psychomotor disturbances, cardiovascular and hepatocellular disorders, kidney-related diseases); pregnancy; concomitant surgical procedures (e.g., sinus/bone augmentation); and use of anti-inflammatory or antibiotic medication within the previous 2 years.

In terms of implant location, implants were classified by region, as either anterior implants, i.e., implants placed between canines (1=anterior maxilla, 3=anterior mandible), or posterior implants, i.e., implants placed between pre-molars and molars (2=posterior maxilla, 4=posterior mandible).

Clinical parameters [13] were assessed and recorded, as follows:

Modified plaque index (PI): PI [14] was assessed at 6 sites (midlingual, mesiolingual, distolingual, midbuccal, distobuccal, and mesio Buccal) per implant. Each site was scored individually (0=no plaque detected; 1=plaque recognizable by running a probe across the smooth marginal surface of the implant; 2=plaque visible to the naked eye; and 3=an abundance of soft matter), and the average of these 6 scores was recorded as the PI score for that particular implant.

Sulcus bleeding index (SBI): SBI [15] was assessed at the above-mentioned 6 sites, each of which was scored individually (0=no bleeding when periodontal probe passes along the sulcus adjacent to the implant; 1=visible, isolated spots of bleeding; 2= blood forming a red line in the sulcus; and 3=heavy or profuse bleeding from the sulcus), the average of which was recorded as the final SBI value for that implant.

Probing depth (PD): PD [16] was measured (mm) at the above-mentioned 6 sites, and the average of the 6 measured values was recorded as the PD score for that implant.

Digital panoramic radiographs (Morita Veraview IC5, J. Morita Mfg. Corp., Kyoto, Japan) taken at the time of loading and again 1 year and 3 years later were analyzed at 20× magnification

Table 1. Plaque index scores in the maxillary and mandibular anterior and posterior regions in smokers and non-smokers.

PI		Non-smokers	Smokers	P value
Maxilla	Anterior zone	1.06 (\pm 0.98)	2.18 (\pm 0.88)	0.001
	Posterior zone	1.44(\pm 0.90)	2.36 (\pm 0.75)	0.003
Mandible	Anterior zone	0.80 (\pm 0.64)	1.61 (\pm 0.76)	0.007
	Posterior zone	1.17 (\pm 0.78)	2.21 (\pm 0.58)	0.001

Table 2. Probing depth scores in the maxillary and mandibular anterior and posterior regions in smokers and non-smokers.

PD		Non-smokers	Smokers	P value
Maxilla	Anterior zone	3.21 (\pm 2.62)	4.65 (\pm 3.72)	0.001
	Posterior zone	3.36 (\pm 2.84)	4.80 (\pm 3.20)	0.001
Mandible	Anterior zone	3.05 (\pm 2.73)	4.38 (\pm 3.03)	0.019
	Posterior zone	3.29 (\pm 2.67)	4.75 (\pm 3.87)	0.001

Table 3. Sulcus bleeding index scores in the maxillary and mandibular anterior and posterior regions in smokers and non-smokers.

SBI		Non-smokers	Smokers	P value
Maxilla	Anterior zone	0.49(\pm 0.64)	0.53(\pm 0.60)	0.511
	Posterior zone	0.53(\pm 0.72)	0.69(\pm .74)	0.122
Mandible	Anterior zone	0.72 (\pm 0.98)	0.76 (\pm 0.83)	0.631
	Posterior zone	0.48 (\pm 0.76)	0.89 (\pm 0.65)	0.09

using the software program CorelDraw 11.0 (Corel Corp and Coral Ltd, Ottawa, Canada). Measurements were performed by 2 examiners blinded to the study protocol. The distance from the widest part of the implant to the crestal bone level was measured on the magnified images. Radiographic distortion was corrected for by determining the ratio of the measured width to the manufacturer's reported width (using the reported diameter at the collar region as a reference point) and using this ratio to identify actual bone height from measured bone height. Marginal bone attachment at the distal and mesial surfaces of all implants was visually assessed, the average of their measurements was calculated, and the difference in marginal bone over time was recorded as the MBL of each implant. Statistical analysis was performed using Statistical Package for Social Sciences (SPSS) for Windows software (IBM Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY, USA). The Shapiro-Wilk test was used to determine if the parameters measured met the assumptions of normal distribution. Three-way ANOVA was used to determine if there is an interaction effect between 3 independent variables (sex, location, smoking) on the peri-implant parameters. Pearson's correlation coefficient tests were used for correlation analysis. The significance level was set at $P < 0.05$.

Results

Mean PI scores were significantly higher in smokers as compared to non-smokers ($P < 0.001$, Table 1). Mean PI scores of smokers ranged from 1.61 (\pm 0.76) in the anterior mandible to 2.36 (\pm 0.75) in the posterior maxilla, whereas mean PI scores of non-smokers ranged from 0.80 (\pm 0.64) in the anterior mandible and 1.44 (\pm 0.90) in the posterior maxilla. Mean PD scores were also significantly higher in smokers as compared to non-smokers ($P < 0.001$, Table 2), ranging from 4.38 (\pm 3.03) in the anterior mandible to 4.80 (\pm 3.20) in the posterior maxilla in smokers and from 3.05 (\pm 2.73) in the anterior mandible to 3.36 (\pm 2.84) in the posterior maxilla in non-smokers. A positive correlation was found between PI and PD scores in both groups ($r = 0.344$, $P < 0.001$). No statistically significant difference in SBI was observed between the 2 groups (Table 3).

MBL was significantly greater in smokers ($P < 0.001$) as compared to non-smokers in both jaws (Table 4). MBL did not vary significantly by location in either group (smokers: $p = 0.415$; non-smokers: $p = 0.175$). No significant sex differences were found in either group ($p = 0.257$).

Table 4. Marginal bone loss values in the maxillary and mandibular anterior and posterior regions in smokers and non-smokers.

MBL		Non-smokers	Smokers	p Value
Maxilla	Anterior zone	0.88 (±0.15)	2.3 (±1.5)	0.001
	Posterior zone	0.91 (±0.14)	2.5 (±0.9)	0.001
Mandible	Anterior zone	0.85 (±0.18)	2.2 (±0.5)	0.001
	Posterior zone	0.83 (±0.15)	2.3 (±0.7)	0.001
p Value		0.175	0.415	

Discussion

This study measured peri-implant probing depths, plaque index and sulcus bleeding index scores, and marginal bone loss surrounding dental implants in different regions of the jaws of smokers and non-smokers over a 36 month period following loading of fixed, implant-supported prostheses. PD and PI scores and MBL values differed significantly between non-smokers and smokers, whereas no statistically significant difference was observed in the SBI values of the 2 groups; therefore, the study's first null hypothesis was partially rejected. Moreover, despite slightly greater marginal bone loss in the maxilla compared to the mandible at the end of 3 years of follow-up, implant location (maxilla/mandible, anterior/posterior) had no significant effect on marginal bone loss around implants in either smokers or non-smokers; therefore, the second null hypothesis was accepted.

Radiography plays an essential role in routine clinical practice and in studies assessing MBL around implants. Periapical and panoramic radiography are the most common imaging methods used in clinical practice. Previous studies have reported that both these methods can be used to reliably evaluate MBL around implants [17,18]. Moreover, Gutmacher et al. [19] reported a strong, direct correlation between measurements of peri-implant MBL from periapical and panoramic radiographs, and they suggested that given their similar reliability and reproducibility, the choice of imaging treatment should be left to the discretion of the clinician. The greatest disadvantage of two-dimensional modalities like periapical panoramic radiography is that they are only capable of providing data on mesial and distal bone resorption, not on buccal bone resorption. Notwithstanding this limitation, this retrospective study measured MBL from panoramic radiographs taken during routine annual recall examinations.

This study found no statistically significant differences between MBL in males and females in either smokers or non-smokers. These findings are in line with previous studies that showed neither sex [12,20] nor age had a significant effect on MBL in either smokers or non-smokers [12,21].

Systematic reviews and meta-analyses of the effects of smoking on implant success rates concluded that smokers had significantly less osseointegration and greater MBL following implant placement [22,23]. A meta-analysis by Clementini et al. [23] reported that smoking increases MBL around implants by 0.16 mm per year, and a long-term retrospective study by Levin et al. [24] concluded that MBL was more severe in smokers compared to non-smokers at all assessed time periods. DeLuca et al. [25] demonstrated more MBL in smokers than non-smokers over a 10 year follow-up period, and concluded that localized exposure of peri-implant tissue to cigarette smoke is the main factor causing the higher implant failure rates observed in smokers as compared to non-smokers. Furthermore, based on their systematic review and meta-analysis of smoking and dental implants, Chrcanovic et al. [26] suggested that smoking affects the rate of implant failure as well as the incidence of postoperative infection and amount of MBL following implant insertion.

The present study found peri-implant MBL to be significantly higher in smokers than non-smokers, which confirms the results of previous studies [27,28]. In the early 1990s, bone loss of 1–1.5 mm in the first year following loading, followed by annual bone loss of 0.1–0.2 mm thereafter, was considered acceptable [29]. Although most studies still use these measurements in defining treatment success, the Pisa Consensus [30] agreed in 2008 that measured peri-implant bone loss of <2.0 should be used as the criteria for treatment success, since radiographic measurements of 0.1 mm were deemed operator-sensitive and thus unreliable. The present study found MBL values after 3 years to exceed the 2-mm threshold of acceptability (range: 2.5 mm ±0.9 to 2.2 mm ±0.5). In view of these findings, patients who smoke should be advised to quit and should be warned of the increased risk of implant failure and postoperative complications associated with smoking.

The present study found MBL to be significantly greater in smokers as compared to non-smokers, and also found significant differences in PI and PD scores of smokers and non-smokers. Probing depth is one of several important criteria used to evaluate the state of peri-implant tissue. Nicotine has been implicated in increases in dental plaque accumulation and reductions in cellular healing responses [31]. A previous

study demonstrated that the oral bio-film of smokers harbors a higher number of periodontal pathogens when compared to non-smokers, and that increases in PI are associated with increases in PD [32]. Furthermore, a study by Lopez-Piriz et al. [33] examining the health of peri-implant tissue reported a relationship between peri-implant bone loss and plaque indices and probing depths.

Despite the measured differences in PI and PD scores between smokers and non-smokers, the present study found no statistically significant differences in the SBI values of the 2 groups. Al-Aali et al. [27] reported similar findings, and suggested that the similarities in SBI values between smokers and non-smokers were due to the vasoconstrictive effects of nicotine. A study by Buduneli and Scott [34] also reported that smoking clearly suppresses bleeding in response to plaque and causes thermally-induced nerve damage in the oral cavity.

It is also possible that implant failure may vary with implant location in connection with the quality and quantity of alveolar bone in which the implant is placed, which varies in terms of mineral density, microarchitecture, and trabecular thickness [35]. A previous study reported the coronal-apical thickness of buccal bone is higher in mandibular as compared to maxillary dentition [36]. Truhlar et al. [37] investigated the distribution of bone quality in patients receiving endo-osseous dental implants, and concluded that the densest bone is located in the symphysis region, followed by the posterior mandible, anterior maxilla, and posterior maxilla. Bain and Moy [9] reported implant success rates were worst in the posterior maxilla and best in the anterior mandible, with failure rates in smokers significantly higher than in non-smokers in all areas except the posterior mandible.

According to the present study results, implant location (maxilla/mandible, anterior/posterior) has no significant effect on marginal bone loss around implants placed in either smokers or non-smokers. This finding is in line with previous studies by Peñarocha et al. [12] and Al-Aali et al. [27], who reported no significant differences in marginal bone loss surrounding implants in the posterior and anterior regions of the jaws, but it conflicts with a review by Tolstunov [38], which reported the anterior mandible is the most successful region and the

posterior maxilla is the least successful region for implant rehabilitation. It should be noted that advances in implant design and surface characteristics (e.g., an osteoconductive roughened surface topography), may reduce marginal bone loss around implants in any zone of the jaws. The present study was conducted with Osseospeed implants, which, according to their manufacturer, have been designed to meet the specific requirements of each individual region of the jaws. This may explain the lack of any significant differences in MBL values among implant locations. Moreover, all of the study participants received professional oral care during annual recall sessions, which may have had a positive effect on their attitude towards oral health maintenance, including maintenance of peri-implant tissue.

This study had a number of limitations. First, the strict eligibility criteria excluded individuals with diabetes. However, considering that oral soft-tissue inflammation and alveolar bone loss are known to be worse in patients with poorly controlled diabetes as compared to systemically healthy individuals, it is hypothesized that peri-implant soft-tissue inflammation and MBL are worse in smokers and non-smokers with poorly controlled diabetes as compared to systemically healthy non-smokers, regardless of jaw location. Another limitation is that smokers were not classified according to frequency and amount of nicotine consumption. The smokers in this study consumed an average of 10 cigarettes per day, so they could be considered to be light smokers; heavy smokers (i.e., individuals who consume at least 20 cigarettes per day) may have exhibited different results in terms of both periodontal markers and MBL values. Further investigations with larger sample sizes are needed to achieve more precise results.

Conclusions

Within the limitations of this study, it was concluded that smoking is associated with increases in marginal bone loss around implants, independent of their location in the jaws. Moreover, of the 3 peri-implant inflammatory parameters examined (plaque indices, sulcus indices, and probing depths), plaque indices and probing depths were observed to be greater in smokers than in non-smokers.

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