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# Prevalence of peri-implant disease and risk indicators in a Japanese population with at least 3 years in function—A multicentre retrospective study

Masahiro Wada[1] | Tomoaki Mameno[1] | Yoshinobu Onodera[2] | Hirofumi Matsuda[3] | Koji Daimon[4] | Kazunori Ikebe[1]

[1]Department of Prosthodontics, Gerodontology and Oral Rehabilitation, Osaka University Graduate School of Dentistry, Suita, Japan

[2]Private Dental Office, Aichi, Japan

[3]Private Dental Office, Nara, Japan

[4]Private Dental Office, Mie, Japan

**Correspondence**
Masahiro Wada, Department of Prosthodontics, Gerodontology and Oral Rehabilitation, Osaka University Graduate School of Dentistry, Suita, Japan.
Email: masahiro@dent.osaka-u.ac.jp

## Abstract

**Objectives:** The aim of this study was to evaluate the prevalence of peri-implant disease and analyze risk indicators in Japanese subjects with ≥3 years of implant function.

**Material and methods:** Five hundred and forty-three subjects treated with 1,613 implants were evaluated. Information was collected about the patients' physical and dental history, as well as implant details. Peri-implant evaluation included probing depth, bleeding on probing (BoP), suppuration (Sup), and keratinized tissue width. Bone loss was calculated from intra-oral radiographs taken after 1 year and more than 3 years of function. Implants were classified into three groups: healthy, peri-implant mucositis (BoP without bone loss), and peri-implantitis (BoP and/or Sup with bone loss >1 mm). These data were analyzed by multivariable multinomial logistic regression.

**Results:** The prevalence of peri-implant mucositis and peri-implantitis at the subject level was 23.9% and 15.8%, respectively. An association was found between peri-implant mucositis and plaque control record (PCR) >20% and keratinized tissue width <2 mm. Peri-implantitis was associated with PCR >20%, smoking, insertion in the maxilla, and keratinized tissue width <2 mm.

**Conclusions:** Within the limitations of this study, the prevalence of peri-implant diseases was elucidated in a Japanese population. Peri-implant mucositis was associated with poor oral hygiene and less keratinized tissue. Poor oral hygiene, smoking, insertion in the maxilla, and less keratinized tissue were risk indicators for peri-implantitis.

KEYWORDS

peri-implantitis, peri-implant mucositis, multicentre research

## 1 | INTRODUCTION

Dental implants are used to replace missing teeth and to help patients recover lost oral function and improve esthetics. Implants are designed to function over the long term (Jung et al., 2008; Ravald, Dahlgren, Teiwik, & Gröndahl, 2013); however, many studies of implants suggest that the prevalence of peri-implant disease is higher than expected (Fransson, Lekholm, Jemt, & Berglundh, 2005). Derks et al. (2016) revealed in their large-scale cross-sectional study that peri-implant mucositis was present in 32% of cases at the subject level and 35.1% of cases at the implant level, while peri-implantitis occurred in 45% of cases at the

subject level and 24.9% of cases at the implant level (bleeding on probing [BoP]/suppuration [Sup] and bone loss >0.5 mm) (Derks et al., 2016). If a different case definition (BoP/Sup and bone loss >2 mm) had been employed, the prevalence of peri-implantitis would have been 14.5% at the subject level and 8.0% at the implant level. Because case definition varies among the published cross-sectional studies, there is inconsistency in terms of the prevalence of peri-implantitis. One study investigated the prevalence of peri-implant disease in Japan (Ogata et al., 2017). Although they used a strict case definition (BoP/Sup and any bone loss), the prevalence of peri-implant mucositis and peri-implantitis was relatively low (33.3% and 9.7%, respectively) compared with other reports. These findings may be related to the quality of periodontal treatment and supportive periodontal therapy managed by the periodontal specialists. Because the implants were treated and maintained by periodontists, the reported disease prevalence may reflect the efficacy of implant treatment rather than the effectiveness. Additionally, a potential risk indicator in the Japanese population was not reported.

Poor oral hygiene, a history of periodontitis, and cigarette smoking have been identified as substantial risk indicators for peri-implant disease (Heitz-Mayfield, 2008). Other reported potential risk indicators with limited or conflicting evidence include cement residue, genetic factors, diabetes, and occlusal overload (Peri-implant mucositis and peri-implantitis: a current understanding of their diagnoses and clinical implications, 2013). Researchers have debated whether keratinized peri-implant mucosa is necessary to maintain healthy peri-implant soft and hard tissue, and whether inadequate keratinized tissue is problematic and a risk indicator for peri-implant disease. Wennström and Derks (2012) found in their review that there was limited need for keratinized mucosa around implants to maintain peri-implant soft and hard tissue stability. In contrast, a systematic review by Lin, Chan, and Wang (2013) demonstrated that the presence of at least 1- to 2-mm-wide keratinized mucosa might be crucial for the decreasing the plaque score, modified gingival index score, mucosal recession, and loss of clinical attachment, but did not affect bleeding on probing [BoP], probing pocket depth, or the stability of the marginal radiographic bone.

To clarify the prevalence of and risk indicators for peri-implant disease, an adequate sample size should be used. According to a consensus report, a limited convenience sample may not be representative of the target population (Sanz, Chapple, & Working Group 4 of the VIII European Workshop on Periodontology, 2012).

The aim of the present study was to evaluate the prevalence of peri-implant disease and to analyze potential risk indicators in a large Japanese population with at least 3 years of implant function.

## 2 | MATERIALS AND METHODS

### 2.1 | Subjects

In this retrospective study, patients treated at a dental university hospital and seven general dental offices between November 1996 and December 2013 were evaluated. Patients having at least one rough surface titanium implant in function for more than 4 years were evaluated. All the implant treatment, including the surgery, was performed by dentists who had at least 10 years' experience of implant treatment. Patients, who had uncontrolled systemic diseases, who did not attend a regular maintenance program, who took antibiotics within 3 months of the examination, and whose final prosthesis had been in function for <3 years, were excluded from the study. All patients who fulfilled the inclusion criteria were informed about this study and completed a written consent form.

Details were collected about the patients' age, sex, presence of systemic diseases, number of teeth, history or presence of periodontitis, plaque control record (PCR; O'Leary score), smoking habit (more than one cigarette per day), alcohol intake (daily drinking or not), parafunctional activity such as bruxism, and frequency of maintenance. Periodontitis was defined as the existence of periodontal pockets more than 6 mm deep and attachment loss of 2 mm according to Derks et al. (2016).

### 2.2 | Implant and peri-implant examination

Information was recorded about implant location, implant size, surgical procedure used (immediate, one-stage, two-stage, with or without bone graft), and fixation type (screw/cement or other). The following parameters were recorded by the attending dentists for the peri-implant evaluation: minimum keratinized tissue width around implant, peri-implant probing depths at four aspects of each implant (mesial, distal, lingual/buccal, and palatal/buccal), peri-implant BoP, and peri-implant suppuration. Probing procedure was performed under a light pressure (0.25 N) with a manual periodontal probe (PCP15; Hu-Friedy Inc., Leimen, Germany).

### 2.3 | Radiographic examination

Intra-oral radiographs were taken of each implant at baseline (after 1 year of function) and at follow-up (after more than 3 years of function).

Before measuring the bone loss on the intra-oral radiographs, intra-observer error and inter-observer error were confirmed by using intra-class correlation case 1 and case 2 analyses. There was no significant difference in intra-observer error (correlation coefficient = 0.996; 95% confidence interval [CI]: 0.982–1.000) or inter-observer error (correlation coefficient = 0.994; 95% CI: 0.985–0.998). Therefore, one examiner assessed the radiographs in this study. The bone level was defined as the distance between the platform of the implant and the bone crest. The implant length (a) and the bone crest level (b) from the apex of the implant on the intra-oral radiograph were measured.

The radiographic bone level was then corrected to the actual bone level using the ratio of the implant length on the intra-oral radiograph and the actual implant length ($a'$) in the formula $([a - b] \times a')/a$. (Figure 1) The bone loss around the implant was obtained from the difference in the bone levels between the baseline and the follow-up

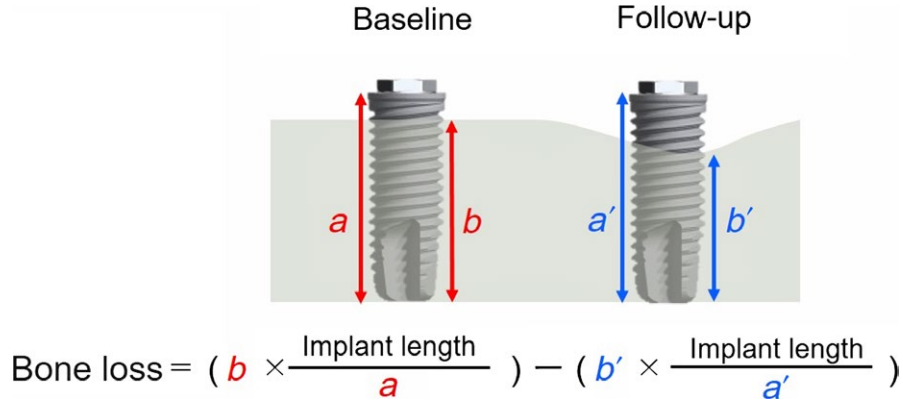


**FIGURE 1** Measurement method of bone loss

examination. All the measurements were performed by an image analysis software (ImageJ 1.49v; Wayne Rasband, National Institutes of Health, Bethesda, MD).

### 2.4 | Definition of peri-implant diseases

Peri-implant mucositis was defined by the presence of BoP without bone loss around the implants, while peri-implantitis was defined by the presence of BoP and/or Sup with bone loss >1 mm. According to this definition, implants were classified into three groups: healthy, peri-implant mucositis, and peri-implantitis.

### 2.5 | Statistical analysis

All data were analyzed using Stata 14.2 (StataCorp LLC, College Station, TX). The collected data were used for the descriptive analysis including an overview of the subjects, implants, and peri-implant tissue. The mean, standard deviation, and percentage were calculated for each variable. To consider correlations among implants in the same subject, the identification of subjects was used as a multilevel latent variable in generalized structural equation modeling (GSEM). Additionally, because there were three objective variables in this study (healthy, peri-implant mucositis, and peri-implantitis), a multinomial logistic regression using GSEM was performed to determine risk indicators. Initially, a univariate multinomial logistic regression was performed to confirm the correlations between each parameter and the dependent variables (peri-implant mucositis and peri-implantitis) after adjusting for sex and age. Then, a multivariable multinomial regression analysis was performed to explain peri-implant disease based on the independent variables which showed significant differences in the univariate analyses. Additionally, we performed interaction analyses by adding interaction terms to clarify relationships among the variables. The results of the multinomial logistic regression were presented as the odds ratio (OR) and the 95% confidence interval. Statistical significance was set at a $p$-values <0.05 for all analyses. This study is in accordance with the STROBE guidelines for observational studies. This study was also approved by the Ethics Committee of the University Graduate School of Dentistry (H28-E24).

## 3 | RESULTS

### 3.1 | Subject data

Of the 571 subjects recruited for this study, 26 subjects were excluded. The reasons for exclusion were as follows: uncontrolled systemic disease (10 subjects), took antibiotics within 3 months (3 subjects), no radiograph at baseline (13 subjects), and did not attend regular maintenance (2 subjects). Finally, 543 subjects who received implant treatment were analyzed in this study. Three hundred and fifty subjects were females, and 193 were males. The mean age at baseline was 63.0 ± 11.9 years. In the medical history, 9.2% of subjects were smokers, 27.1% had a drinking habit, 5.3% had diabetes, 13.8% had hypertension, 5.7% had hyperlipidemia, and 2.4% had osteoporosis. Half of the subjects (52.5%) had parafunctional problems. The average PCR was 23.1 ± 17.4%. At the time of evaluation, 27.8% of subjects had periodontitis and 44.6% had a history of periodontitis before implant treatment. The distribution of subject variables is summarized in Table 1.

### 3.2 | Implant data

A total of 1,613 implants were examined in this study: 43.1% in the maxilla and 56.9% in the mandible. The mean observation period was 5.8 ± 2.5 years. The surgical procedures used were one-stage (42.7%) and two-stage (57.3%). The presence of bleeding was detected in approximately one-third of implants, and 3.8% of implants had suppuration at the time of examination. The average minimal keratinized tissue width around the implants was 2.53 ± 1.61 mm. Other variables, including implant brand, bone augmentation, pocket depth, fixation type, and superstructure material, are described in Table 2.

### 3.3 | Peri-implant mucositis

The prevalence of peri-implant mucositis at the subject level and the implant level was 23.9% and 27.4%, respectively. The median bone loss in the peri-implant mucositis group is shown in Table 3. A significant correlation was found between peri-implant mucositis and smoking, PCR (>20%), presence of periodontitis, implant position (maxilla), surgical procedure (two-stage), and keratinized tissue width (<2 mm) by

**TABLE 1** Description of subject ($n$ = 543)

| Variable | n | % |
|---|---|---|
| Gender | | |
| Female | 350 | 64.5 |
| Male | 193 | 35.5 |
| Age (years) | | |
| ≦49 | 74 | 13.6 |
| 50–59 | 107 | 19.7 |
| 60–69 | 194 | 35.7 |
| 70–79 | 141 | 26.0 |
| 80< | 27 | 5.0 |
| Treatment place | | |
| University hospital | 139 | 25.6 |
| Private office | 404 | 74.4 |
| Smoking | | |
| Yes | 50 | 9.2 |
| No | 493 | 90.8 |
| Drink | | |
| Yes | 147 | 27.1 |
| No | 396 | 72.9 |
| Plaque control record (%) | | |
| >20 | 244 | 44.9 |
| ≦20 | 299 | 55.1 |
| Presence of periodontitis | | |
| Yes | 151 | 27.8 |
| No | 392 | 72.2 |
| History of periodontitis | | |
| Yes | 242 | 44.6 |
| No | 301 | 55.4 |
| Systemic disease | | |
| Diabetes | 29 | 5.3 |
| Hypertension | 75 | 13.8 |
| Hyperlipidemia | 31 | 5.7 |
| Osteoporosis | 13 | 2.4 |
| Other | 16 | 2.9 |
| Parafunction | | |
| Yes | 285 | 52.5 |
| No | 258 | 47.5 |

**TABLE 2** Description of implant ($n$ = 1,613)

| Variable | n | % |
|---|---|---|
| Implant brand | | |
| Nobel biocare | 644 | 39.9 |
| Dentsply | 602 | 37.3 |
| Zimmer biomet | 164 | 10.2 |
| GC | 109 | 6.8 |
| Straumann | 66 | 4.1 |
| Other | 28 | 1.7 |
| Implant position | | |
| Maxilla | 695 | 43.1 |
| Mandible | 918 | 56.9 |
| Surgical procedure | | |
| One-stage | 688 | 42.7 |
| Two-stage | 925 | 57.3 |
| Immediate | 0 | 0 |
| Bone augmentation | | |
| GBR | 111 | 6.9 |
| Sinus lift | 67 | 4.2 |
| Socket lift | 39 | 2.4 |
| Fixation type | | |
| Cement | 1,144 | 70.9 |
| Screw | 431 | 26.7 |
| Removable | 38 | 2.4 |
| Material of superstructure | | |
| Ceramic | 959 | 59.5 |
| Metal | 251 | 15.6 |
| Other | 403 | 25.0 |
| Keratinized tissue width (mm) | | |
| <2 | 583 | 36.1 |
| ≧2 | 1,030 | 63.9 |
| Probing depth (mm) | | |
| <3 | 289 | 17.9 |
| 3–6 | 1,301 | 80.7 |
| >6 | 23 | 1.4 |
| Bleeding on probing | | |
| Yes | 556 | 34.5 |
| No | 1,057 | 65.5 |
| Suppuration | | |
| Yes | 62 | 3.8 |
| No | 1,551 | 96.2 |

univariate analysis (Table 4). In a multivariable multinomial logistic regression using these significant variables, PCR > 20% (OR = 8.66; 95% CI: 4.91–15.26) had a significant association with peri-implant mucositis development (Table 5). The results of interaction analyses showed that there were no significant interaction effects among variables.

## 3.4 | Peri-implantitis

The prevalence of peri-implantitis at the subject level and the implant level was 15.8% and 9.2%, respectively. The median bone

**TABLE 3** Bone resorption in each group (mm)

| Group | $n$ | Median | Interquartile range |
|---|---|---|---|
| Healthy | 1,023 | 0.10 | 0.28 |
| Peri-implant mucositis | 442 | 0.14 | 0.39 |
| Peri-implantitis | 148 | 1.60 | 0.92 |
| Total | 1,613 | 0.14 | 0.36 |

**TABLE 4** Risk factor analysis using the univariate multinomial logistic regression after adjusting for gender and age

| | Peri-implant mucositis | | | Peri-implantitis | | |
|---|---|---|---|---|---|---|
| **Variable** | **Odds ratio** | **95% CI** | ***p* value** | **Odds ratio** | **95% CI** | ***p* value** |
| Smoking | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 2.73 | 1.14–6.55 | 0.02 | 7.23 | 2.29–22.8 | <0.01 |
| Drink | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 1.60 | 0.86–2.96 | 0.14 | 1.80 | 0.75–4.28 | 0.19 |
| Plaque control record | | | | | | |
| ≦20 | 1 | | | 1 | | |
| >20 | 11.08 | 6.32–19.4 | <0.01 | 8.87 | 3.96–19.9 | <0.01 |
| Presence of periodontitis | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 2.53 | 1.42–4.52 | <0.01 | 3.09 | 1.38–6.90 | <0.01 |
| History of periodontitis | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 1.16 | 0.69–1.95 | 0.58 | 1.48 | 0.70–3.12 | 0.30 |
| Diabetes | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 1.03 | 0.34–3.11 | 0.96 | 2.75 | 0.69–10.9 | 0.15 |
| Hypertension | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 0.66 | 0.31–1.39 | 0.27 | 0.42 | 0.14–1.22 | 0.11 |
| Hyperlipidemia | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 1.54 | 0.56–4.23 | 0.40 | 1.70 | 0.44–6.61 | 0.44 |
| Osteoporosis | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 0.53 | 0.10–2.95 | 0.47 | 1.60 | 0.20–13.0 | 0.66 |
| Parafunction | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 0.63 | 0.38–1.04 | 0.07 | 1.10 | 0.53–2.30 | 0.80 |
| Maintenance interval (month) | 0.90 | 0.79–1.03 | 0.14 | 0.63 | 0.48–0.81 | <0.01 |
| Number of implants | 1.10 | 0.99–1.21 | 0.06 | 1.14 | 0.99–1.30 | 0.07 |
| Implant diameter (mm) | 1.11 | 0.77–1.60 | 0.58 | 1.09 | 0.64–1.86 | 0.74 |
| Implant length (mm) | 0.96 | 0.87–1.06 | 0.40 | 1.06 | 0.92–1.21 | 0.45 |
| Implant position | | | | | | |
| Mandible | 1 | | | 1 | | |
| Maxilla | 1.44 | 1.00–2.08 | 0.048 | 1.98 | 1.16–3.38 | 0.01 |
| Surgical procedure | | | | | | |
| One-stage | 1 | | | 1 | | |
| Two-stage | 1.59 | 1.07–2.35 | 0.02 | 1.55 | 0.89–2.70 | 0.12 |
| GBR | | | | | | |
| No | 1 | | | 1 | | |

*(Continues)*

**TABLE 4** (Continued)

| Variable | Peri-implant mucositis | | | Peri-implantitis | | |
|---|---|---|---|---|---|---|
| | Odds ratio | 95% CI | *p* value | Odds ratio | 95% CI | *p* value |
| Yes | 0.50 | 0.23–1.11 | 0.09 | 1.12 | 0.42–3.01 | 0.82 |
| Sinus lift | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 1.17 | 0.39–3.48 | 0.78 | 2.55 | 0.63–10.3 | 0.19 |
| Socket lift | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 2.41 | 0.80–7.27 | 0.12 | 1.66 | 0.32–8.49 | 0.54 |
| Fixation type | | | | | | |
| Screw | 1 | | | 1 | | |
| Cement | 1.09 | 0.62–1.91 | 0.76 | 1.40 | 0.65–3.02 | 0.40 |
| Removable | 2.54 | 0.58–11.1 | 0.22 | 3.84 | 0.55–26.8 | 0.17 |
| Material of superstructure | | | | | | |
| Ceramic | 1 | | | | | |
| Metal | 0.95 | 0.57–1.56 | 0.83 | 1.84 | 0.96–3.52 | 0.07 |
| Other | 1.48 | 0.94–2.34 | 0.09 | 1.09 | 0.54–2.18 | 0.81 |
| Keratinized tissue width (mm) | | | | | | |
| ≧2 | 1 | | | 1 | | |
| <2 | 1.54 | 1.03–2.30 | 0.04 | 2.59 | 1.47–4.55 | <0.01 |
| Implant brand | | | | | | |
| Nobel biocare | 1 | | | 1 | | |
| Dentsply | 0.56 | 0.32–1.00 | 0.051 | 0.47 | 0.21–1.07 | 0.07 |
| Zimmer biomet | 1.36 | 0.62–3.02 | 0.44 | 1.37 | 0.47–4.03 | 0.56 |
| GC | 2.11 | 0.83–5.36 | 0.12 | 0.66 | 0.16–2.77 | 0.57 |
| Straumann | 2.09 | 0.65–6.67 | 0.21 | 0.39 | 0.05–3.14 | 0.38 |
| Other | 0.55 | 0.11–2.61 | 0.45 | 0.20 | 0.01–3.56 | 0.27 |

loss in the peri-implantitis group was 1.93 (±0.95) mm (Table 3). Smoking, PCR (>20%), presence of periodontitis, maintenance interval, implant position (maxilla), and keratinized tissue width (<2 mm) each had a significant correlation with peri-implantitis in the univariate analysis (Table 4). A significant association was found between peri-implantitis development and PCR >20% (OR = 6.12; 95% CI: 2.75–13.7), smoking (OR = 3.51; 95% CI: 1.20–10.3), implant insertion in the maxilla (OR = 1.85; 95% CI: 1.09–3.14), and keratinized tissue width <2 mm (OR = 2.32; 95% CI: 1.29–4.16) using multivariable multinomial logistic regression analysis. The maintenance interval (OR = 0.63; 95% CI: 0.46–0.86) was negatively associated with peri-implantitis development (Table 5). There were also significant interaction effects between smoking and keratinized tissue width ($p < 0.01$), and PCR and keratinized tissue width ($p < 0.01$). Therefore, subgroup analyses were conducted to compare the effect of smoking and PCR in subjects with keratinized tissue width ≧2 mm and those with keratinized tissue width <2 mm. The subgroup analyses revealed that smoking and PCR >20% had a significant association with a high OR for peri-implantitis development in subjects with keratinized tissue width <2 mm. However, in the presence of keratinized tissue (≧2 mm), smoking was not associated with peri-implantitis development (Table 6).

## 4 | DISCUSSION

Implant therapy has been commonly used over recent decades for rehabilitation in partially and fully edentulous patients, and implants have a long-term survival rate (Berglundh, Persson, & Klinge, 2002; Horikawa et al., 2017; Lang et al., 2004; Roos-Jansåker, Lindahl, Renvert, & Renvert, 2006). At the same time, peri-implant disease has become a common complication (Costa et al., 2012; Derks & Tomasi, 2015; Zitzmann & Berglundh, 2008). Several recent studies have reported a higher than expected prevalence of peri-implant disease (Atieh, Alsabeeha, Faggion, & Duncan, 2013; Fransson et al., 2005; Lee, Huang, Zhu, & Weltman, 2017; Mir-Mari, Mir-Orfila, Figueiredo, Valmaseda-Castellón, & Gay-Escoda, 2012). Many factors, such as systemic diseases, smoking habits, periodontal status, oral hygiene, implant surface characteristics, location, and prosthetic design, have been proposed as risk indicators (Aguirre-Zorzano, Estefanía-Fresco, Telletxea, & Bravo, 2015; Daubert, Weinstein, Bordin, Leroux, & Flemming, 2015;

**TABLE 5** Association between the variables and peri-implant diseases using a multivariate multinomial logistic regression after adjusting confounding variables (gender, age, and dentists)

| | Peri-implant mucositis | | | Peri-implantitis | | |
|---|---|---|---|---|---|---|
| **Variable** | **Odds ratio** | **95% CI** | ***p* value** | **Odds ratio** | **95% CI** | ***p* value** |
| Smoking | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 1.36 | 0.61–3.03 | 0.45 | 3.51 | 1.20–10.3 | 0.02 |
| Plaque control record (%) | | | | | | |
| ≦20 | 1 | | | 1 | | |
| >20 | 8.66 | 4.91–15.26 | <0.01 | 6.12 | 2.75–13.7 | <0.01 |
| Presence of periodontitis | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 1.60 | 0.90–2.83 | 0.11 | 1.69 | 0.76–3.78 | 0.20 |
| Maintenance interval (month) | – | – | – | 0.63 | 0.46–0.86 | <0.01 |
| Implant position | | | | | | |
| Mandible | 1 | | | 1 | | |
| Maxilla | 1.36 | 0.94–1.98 | 0.10 | 1.85 | 1.09–3.14 | 0.02 |
| Surgical procedure | | | | | | |
| One-stage | 1 | | | – | – | – |
| Two-stage | 1.24 | 0.83–1.86 | 0.29 | – | – | – |
| Keratinized tissue width (mm) | | | | | | |
| ≧2 | 1 | | | 1 | | |
| <2 | 1.34 | 0.89–2.02 | 0.17 | 2.32 | 1.29–4.16 | <0.01 |

**TABLE 6** Association of smoking and plaque control record with peri-implantitis with/without keratinized tissue using a multivariate multinomial logistic regression

| | Keratinized tissue width ≧2 group | | | Keratinized tissue width <2 group | | |
|---|---|---|---|---|---|---|
| **Variable** | **Odds ratio** | **95% CI** | ***p* value** | **Odds ratio** | **95% CI** | ***p* value** |
| Smoking | | | | | | |
| No | 1 | | | 1 | | |
| Yes | 2.52 | 0.74–8.58 | 0.14 | 6.37 | 1.17–34.62 | 0.03 |
| Plaque control record (%) | | | | | | |
| ≦20 | 1 | | | 1 | | |
| >20 | 4.59 | 1.86–11.34 | <0.01 | 18.38 | 4.85–69.69 | <0.01 |

Gurgel et al., 2017; Marrone, Lasserre, Bercy, & Brecx, 2013; Pjetursson et al., 2012; Staubli, Walter, Schmidt, Weiger, & Zitzmann, 2017; Turri, Rossetti, Canullo, Grusovin, & Dahlin, 2016). However, many of these studies have not included all these factors in their statistical analysis. Therefore, the aim of this study was to evaluate the prevalence of peri-implant disease and to analyze these potential risk indicators in a large Japanese population of subjects with at least 3 years of implant function.

This study included a total of 543 subjects, with 1,613 implants inserted at a dental university hospital and seven general dental offices. The large sample size allowed multivariable analysis to be performed, and the data were collected from multiple centers thought to be meaningful.

Peri-implant mucositis was defined as the presence of BoP without bone loss around the implants, according to the classifications used by Ferreira, Silva, Cortelli, Costa, and Costa (2006) and Casado, Villas-Boas, Mello, Duarte, and Granjeiro (2013). The definition of peri-implantitis is less straightforward. Some studies assessed implantitis according to the bone level on the implant threads (Marrone et al., 2013; Mir-Mari et al., 2012), while others assessed it according to the thresholds for marginal bone loss (Cecchinato, Parpaiola, & Lindhe, 2014; Fransson, Wennström, & Berglundh, 2008; Koldsland, Scheie, & Aass, 2010; Roos-Jansåker et al., 2006) ranging from >0.4 to >5 mm. The baseline for the assessment of bone loss also differed between these studies. Some studies used prosthetic loading periods as the baseline (Koldsland et al., 2010; Zetterqvist et al., 2010) and others used post-implant insertion (Casado et al., 2013) or post-1-year loading (Cecchinato et al., 2014; Fransson et al., 2005; Roos-Jansåker et al., 2006). In this study, peri-implantitis was defined as the presence of BoP and/or suppuration with bone loss >1 mm. The

baseline was set at post-1-year loading to take bone remodeling into account (Albrektsson & Zarb, 1998).

Our study found that the prevalence of peri-implant mucositis was 23.9% at the subject level and 27.4% at the implant level. This rate is similar to some studies (Casado et al., 2013; Marrone et al., 2013), but relatively low compared with other previous studies (Cecchinato et al., 2014; Ferreira et al., 2006), in which the prevalence of mucositis ranged from 32.0% to 69.8%. This variance could be explained by differences in the subjects studied. In our study, evaluated data were collected from subjects who attended a regular maintenance recall program. The prevalence of peri-implantitis at the subject and implant levels was 15.8% and 9.2%, respectively, which is similar to previous studies (Cecchinato et al., 2014; Dvorak et al., 2012; Mir-Mari et al., 2012). These comparable values may be accounted for by the fact that there are many potential risk indicators for peri-implantitis apart from poor oral hygiene and regular maintenance.

The results of a multivariable multinomial logistic regression analysis performed in this study indicated that poor oral hygiene (defined as PCR > 20%) was associated with peri-implant mucositis development with a high OR (8.66). The finding of poor oral hygiene as a risk factor for peri-implant mucositis corresponds with the findings of previous studies (Gurgel et al., 2017; Salvi, Cosgarea, & Sculean, 2017).

Poor oral hygiene, smoking, implant insertion in the maxilla, keratinized tissue width <2 mm, and maintenance interval were associated with peri-implantitis development by multivariable multinomial logistic regression analysis. The association between poor oral hygiene and peri-implantitis is in agreement with many studies focusing on the indicators for peri-implant disease (Ferreira et al., 2006; Salvi & Lang, 2004; Serino & Ström, 2009). Smoking is also thought to be a risk indicator for peri-implantitis (Atieh et al., 2013; Heitz-Mayfield, 2008; Heitz-Mayfield & Huynh-Ba, 2009; Turri et al., 2016). In our study, smoking had a strong association with peri-implantitis development, which is consistent with these studies. Keratinized tissue width <2 mm was also associated with peri-implantitis development. Several studies (Chung, Oh, Shotwell, Misch, & Wang, 2006; Wennström & Derks, 2012; Zigdon & Machtei, 2008) reported that keratinized tissue is not necessary for the maintenance of peri-implant tissue and bone under proper oral hygiene conditions. These studies, none of which included multivariable analysis, conflict with our findings and those of other studies and systematic reviews (Buyukozdemir Askin et al., 2015; Lin et al., 2013; Pranskunas, Poskevicius, Juodzbalys, Kubilius, & Jimbo, 2016; Souza, Tormena, Matarazzo, & Araújo, 2016) that revealed the necessity to maintain keratinized tissue around implants to prevent plaque accumulation and peri-implant soft tissue inflammation. Additionally, keratinized tissue width had significant interaction effects with smoking and PCR. Subgroup analyses revealed that smoking and poor oral hygiene became higher risk indicators for peri-implantitis when the keratinized tissue width was insufficient. At the same time, if the keratinized tissue width was adequate, smoking was not considered as a risk indicator for peri-implantitis development. Therefore, it is thought that keratinized tissue around implants is an important factor in preventing the onset of peri-implantitis. The results of our study also showed a higher prevalence of peri-implantitis in implants inserted in the maxilla, which is consistent with previous studies (Schuldt Filho et al., 2014; Schwartz-Arad, Kidron, & Dolev, 2005). Paradoxically, maintenance interval was negatively associated with peri-implantitis in this study. Although it seems logical that a regular maintenance program and structured supportive implant therapy would play an important role in preventing peri-implant disease (Monje et al., 2016; Rokn et al., 2017), this contradictory finding in our study relates to the fact that we used a retrospective cohort study design, so that subjects with peri-implant disease needed frequent maintenance recalls to prevent progression of the disease. In other words, when tailoring the maintenance interval, the frequency should be decided on the basis of risk assessment. If a patient were at low risk of peri-implant disease, the maintenance interval would be longer. Therefore, the result of this study is understandable.

In this study, we did not find an association between the presence of periodontitis and peri-implant disease. Although many studies have cited a previous history of periodontitis and the presence of periodontitis as risk indicators for peri-implantitis (Heitz-Mayfield, 2008; Karoussis, Kotsovilis, & Fourmousis, 2007), in our multivariable multinomial logistic regression analysis, a history of periodontitis was excluded as a variable because of strong multicollinearity with the PCR. The presence of periodontitis also exhibited weak multicollinearity with the PCR, and therefore, the relationship could not be confirmed.

Diabetes and hypertension were not confirmed as risk indicators for peri-implantitis in our study. Monje, Catena, and Borgnakke (2017) concluded in their meta-analysis that the risk of peri-implantitis is greater in patients with diabetes. They also concluded that the association between diabetes and peri-implant mucositis did not reach statistical significance, which is in accordance with our results. Abuohashish, Ahmed, Sabry, Khattab, and Al-Rejaie (2017) reported that some antihypertensive drugs (renin–angiotensin system medicines) improve bone metabolism and the strength of bone. Additionally, Wu et al. (2016) reported that the implant survival rate is higher in patients taking antihypertensive drugs than in healthy patients. The reason for these differences from our findings is that the systemic diseases of the subjects participating in our study were relatively well controlled.

Questions remain about age, the retention type (screw/cement), the implant surface characteristics, and the prosthetic design and materials, all of which have been reported as risk indicators in several studies (Daubert et al., 2015; Fu, Hsu, & Wang, 2012; Schuldt Filho et al., 2014; Staubli et al., 2017), and which did not influence the peri-implant status in this study. One limitation of our study is that our data were analyzed at baseline and one evaluation period, so we could not consider the variable of time in function until peri-implant disease development. Marrone et al. (2013) reported that patients with implants in function for more than 10 years experienced a higher incidence of peri-implantitis than those with more recent implants. Therefore, future prospective studies are required to consider these factors.

## 5 | CONCLUSION

Within the limitations of the present large-scale study involving multiple centers, poor oral hygiene and less keratinized tissue were associated with peri-implant mucositis development, and poor oral hygiene, smoking, and implant insertion in the maxilla and less keratinized tissue were risk indicators for peri-implantitis development.

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## CONFLICT OF INTEREST

The authors report no conflicts of interest related to this study.

## ORCID

*Masahiro Wada* https://orcid.org/0000-0002-0718-8371

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## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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