

Parameters to Define Peri-Implantitis: A Review and a Proposed Multi-Domain Scale

Guo-Hao Lin, DDS, MS^{1,2*}

Yvonne Kapila, BDS, MS, PhD²

Hom-Lay Wang, DDS, MSD, PhD³

Peri-implant diseases have received much attention since dental implants are generally used in contemporary dentistry. Several contributing factors associated with the development of peri-implant diseases have also been investigated. The prevalence of peri-implantitis has been reported but with great heterogeneity because of a lack of a universally accepted classification system that could define the extent and severity of peri-implantitis. Several parameters—including radiographic bone loss, probing depth, bleeding on probing, and suppuration—have been introduced in these reports to assist with clinical diagnosis. This article provides an objective evaluation of these parameters based on currently available evidence, offers further recommendations, and proposes a multidomain scale for diagnosis of peri-implantitis. Future investigations and modifications may be needed to develop a comprehensive, evidence-based classification system that addresses the multifactorial etiology of peri-implant diseases.

Key Words: dental implants, peri-implantitis, periodontal pocket, implant-supported dental prosthesis, risk factors

INTRODUCTION

Peri-implant diseases have received much attention since dental implants began extensive use in contemporary dentistry.^{1–3} The American Academy of Periodontology Academy Statement³ defines *peri-implant mucositis* as the presence of inflammation confined to the soft tissues surrounding a dental implant with no signs of bone loss following initial bone remodeling. *Peri-implantitis* is defined as an inflammatory process around an implant, which includes both soft tissue inflammation and progressive bone loss following initial bone remodeling.³ The etiology of peri-implant diseases is multifactorial. Similar to the etiology of gingivitis and periodontitis, the primary etiology of peri-implant diseases is reported to be bacterial in nature with subsequent activation of the host immune response.^{3–5}

Several contributing factors associated with the development of peri-implant diseases have also been investigated, including but not limited to a history of periodontitis,^{6,7} smoking,^{8,9} and residual cement.¹⁰ In addition, occlusal overload is often as a significant factor when bone loss is observed proximal to dental implants.¹¹ Other factors that have been linked to peri-implant bone loss are implant malposition,¹² compression necrosis,¹³ and even foreign body reaction.¹⁴ However, these remain controversial with regards to

their true association with peri-implant bone loss or even peri-implantitis.

The prevalence of peri-implantitis has been widely reported but with great heterogeneity.^{3,15} Since there is no universally accepted classification system to define the extent and severity of peri-implantitis, most clinical publications define a disease status based on their own criteria (Table 1).^{16–31} According to these publications, the prevalence of peri-implantitis ranges from 6.2% to 39.3% at the implant level and 10.5% to 47.8% at the patient level. Several parameters have been introduced in these reports to assist with clinical diagnosis. Among the most widely used parameters are radiographic bone loss (RBL), probing depth (PD), bleeding on probing (BOP), and suppuration. This article's goal is to provide an objective evaluation of these parameters based on currently available evidence, provide further recommendations, and propose a multidomain scale for future diagnosis of peri-implantitis.

RADIOGRAPHIC BONE LOSS

Peri-implant bone loss is the major criterion that differentiates peri-implantitis from peri-implant mucositis. As a result, radiographic bone loss (RBL) is introduced to determine the peri-implant bone level due to its convenience and lack of invasiveness. However, there is no consensus on the ideal threshold of RBL that defines disease status. In 1986, Albrektsson et al³² proposed that a successful implant must present no mobility, no peri-implant radiolucency, bone loss of less than 0.2 mm per year after the first year of loading, and no persistent pain, discomfort, or infection. In their study, the authors reported 1 mm of RBL during the first year after abutment connection. It is important to note that the authors proposed these criteria based on observations made on pure

¹ Department of Surgical Sciences, Marquette University School of Dentistry, Milwaukee, Wis.

² Department of Orofacial Sciences, University of California San Francisco School of Dentistry, San Francisco, Calif.

³ Department of Periodontics and Oral Medicine, University of Michigan School of Dentistry, Ann Arbor, Mich.

* Corresponding author, e-mail: ghlin@umich.edu

DOI: 10.1563/aaid-joi-d-17-00035

TABLE 1
Studies reporting the prevalence and criteria for peri-implantitis*

Author(s)	Prevalence (Implant Level)	Prevalence (Patient Level)	Thresholds					
			Baseline Bone Level	Follow-up (years)	RBL	Probing Depth (mm)	BOP	Suppuration
Fransson et al ¹⁶	12.4%	27.8%	Radiographs taken at first year after restoration	5	≥3 threads	Not used	Not used	Not used
Ferreira et al ³¹	7.4%	8.9%	Smooth parts and threads	3.54 ± 1.43	Vertical RBL	≥5 mm	Yes	Yes
Roos-Jansaker et al ¹⁷	6.6%	16%	Radiographs taken at first year after restoration	9–14	≥3 threads or ≥1.8 mm	Not used	Yes	Yes
Koldslund et al ¹⁸	36.6%	47.1%	Abutment level	7.4 ± 4.7	≥2 mm	≥4 mm	Yes	Yes
Simonis et al ¹⁹	16.94%	10.53%–37.93%	Implant shoulder	10–16	≥2.5mm or ≥3 threads	≥5 mm	Yes	Yes
Atieh et al ²⁰	9.6%	18.8%	NA	NA	≥2 mm or ≥3 threads	≥5 mm	Yes	Yes
Meijer et al ³⁰	11.5%	16.9%	Fixed reference point	5	≥2 mm	Not used	Yes	Yes
Schuldt Filho et al ²²	27.95%	29.63%	Implant platform	>5	>2mm	>4 mm	Yes	Yes
Renvert et al ²¹	39.3%	47.8%	Implant platform	11.8 ± 3.3	≥2 mm	≥4 mm	Yes	Yes
Aguirre-Zorzano et al ²³	9.8%	15.1%	Radiographs taken at 6 months after restoration	5.25 ± 3.42	≥1.5 mm	Increased	Yes	Yes
Daubert et al ²⁴	16%	26%	2 mm from the expected level	10.9 ± 1.5	≥2 mm	≥4 mm	Yes	Yes
Konstantinidis et al ²⁵	6.2%	12.9%	Implant platform or apical termination of polished collar	5.5 ± 3.8	>2 mm	>5 mm	Yes	Not used
Derks et al ²⁸	NA	14%–30%	Radiographs taken at up to 1 year after restoration	9	≥0.5 mm	Not used	Yes	Yes
Schwarz et al ²⁶	7.6%	13.9%	Radiographs taken at the time of restoration	2.20 ± 1.38	With changes	Increased	Yes	Yes
Dalago et al ²⁷	7.3%	16.4%	Abutment level	1–14	>2 mm	>5 mm	Yes	Yes
Rokn et al ²⁹	8.8%	20%	Implant shoulder	4.43 ± 2.25	>2 mm	Not used	Yes	Yes

*RBL indicates radiographic bone loss; BOP, bleeding on probing.

titanium implants, which are different from those often used today (namely, rough or coated surface implants). Similarly, Adell et al³³ reported an average of 1.5 mm of RBL during the first year of healing when a pure titanium implant was used and only 0.1 mm of RBL annually thereafter. This “first-year bone loss” after connection of the restoration was then further investigated and is now considered part of “physiologic bone remodeling.”¹

Since physiologic bone remodeling is an inevitable process, four different patterns have been proposed to describe this phenomenon.³⁴ Although bone remodeling occurs primarily within the first year after placement of the abutment connection,³² the reasons for its occurrence and the unpredictability of the amount of RBL are still not fully understood. Several factors—including the level of implant placement, tissue biotype, types of abutment connections, implant macro- and microdesigns, and implant surface topography—have all been analyzed.^{2,3,15,21,28,35} A recent animal study reported that different types of implant designs and depth of implant placement have significant impact on this remodeling process.³⁵ These findings underscore that using a certain level of RBL (ie, 2 mm) from a fixed reference point (ie, implant shoulder) to define “acceptable” bone remodeling is challenging.^{2,25} Consequently, the prevalence of peri-implantitis report-

ed throughout the literature is inconsistent because of the diverse cut-off values for RBL defined in individual studies.

Most recent studies introduced the concept of 2 mm of bone loss from a fixed point (such as the implant shoulder, the platform level, or the most apical extension of the polished surface) as the reference point from which to measure the RBL after the first year of loading (Table 2). However, this definition often makes clinical diagnosis difficult since the amount of physiologic bone remodeling cannot be predicted from the reference point due to its multifactorial nature. To date, the only consensus report aiming to define this threshold is the VIII European Workshop on Periodontology.² In this consensus report, *peri-implantitis* was defined as “changes in the level of the crestal bone in conjunction with BOP with or without concomitant deepening of peri-implant pockets”³⁶ if a baseline radiograph is available. However, in the absence of previous radiographic records, a threshold of 2 mm of vertical bone loss from the expected marginal bone level was suggested. Although this report provides a clear diagnostic guideline for clinicians, other issues such as “expected marginal bone level” and “acceptable amount of annual bone loss” after the first year of loading are subjective. One study suggested that “RBL >2 mm from the implant platform for bone-level implants or >2 mm from the apical termination of the polished collar for tissue-level implants” could be used as the threshold indicating

Authors		RBL	PD	BOP and/or Suppuration
Froum and Rosen ³⁷	Severity	Early: <25% of the implant length Moderate: 25% to 50% of the implant length Advanced: >50% of the implant length	≥4 mm ≥6 mm ≥8 mm	BOP and/or suppuration BOP and/or suppuration BOP and/or suppuration
Sanz and Chapple ²	With baseline Without baseline	Changes in the level of crestal bone Bone loss >2 mm from expected bone level	With or without concomitant deepening PD With or without concomitant deepening PD	BOP and/or suppuration BOP and/or suppuration
Decker et al ³⁸	Severity	Early: <25% of the implant length Moderate: 25% to 50% of the implant length Advanced: >50% of the implant length	≥4 mm ≥6 mm ≥8 mm	BOP and/or suppuration BOP and/or suppuration BOP and/or suppuration
Derks et al ²⁸	Severity	Slight/Mild: >0.5 mm Moderate/Severe >2 mm	Not specified	BOP and/or suppuration BOP and/or suppuration

*RBL indicates radiographic bone loss; PD, probing depth; BOP, bleeding on probing.

peri-implantitis.²⁵ In addition, an annual bone loss of less than 0.2 mm after physiologic remodeling could be considered acceptable.¹ Therefore, a modified scale that incorporates the type of implant design and years of service would be beneficial in deriving a more uniform diagnosis.

In our proposed multidomain scale (Table 3), clinicians should evaluate whether or not a baseline radiograph taken at least 1 year after definite crown delivery is available. If this baseline radiograph is available, the acceptable RBL will be no more than 0.2 mm annually compared to the baseline radiograph. On the contrary, if a baseline radiograph is not available, a threshold of 2 mm RBL from a fixed point (eg, implant platform for bone-level and termination of polished collar for tissue-level implants) should be used to determine the disease status. It is worth taking a baseline radiograph whenever possible since it provides valuable information in terms of bone level after the physiologic bone remodeling process. The threshold of "2 mm RBL from a fixed point" should be applied only when a baseline radiograph is not retrievable. A clinical case with a baseline radiograph is demonstrated in Figure 1 as an example.

In terms of the severity of peri-implantitis, several parameters have been proposed.^{28,37,38} Froum and Rosen³⁷ and Decker et al³⁸ used the percentage of RBL relative to the total implant length to define the severity of peri-implantitis. Derks et al²⁸ proposed the amount of RBL (>0.5 mm as mild/

slight and >2 mm as moderate/severe) to address the severity. A comparison of these systems and a proposed scale is listed in Table 2. Since there is no available consensus report to elaborate on this issue, we suggest using the percentage of RBL relative to the total implant length, proposed by the aforementioned articles,^{37,38} to define severity since it is relatively objective and easy to determine radiographically.

PROBING DEPTH

Probing depth is another parameter that has been used to determine peri-implant tissue health. Although this parameter is reproducible and repeatable within 1 mm of accuracy at periodontal sites,³⁹ the accuracy of PD around dental implants remains challenging. Since the reproducibility of PD depends on several factors—such as probing force, probing angulation, probe tip diameter, and tissue inflammatory status—implant-supported restorations might hinder the ability to attain accurate measurements. It has been reported that implant and abutment designs might increase difficulty in obtaining accurate PD measurements, further underestimating the extent of the peri-implant lesion.³⁶

Due to the absence of periodontal ligament fibers, supra-crestal connective tissue fibers are arranged in a circular pattern^{40,41} around the peri-implant tissues, thus decreasing the resistance to clinical probing compared to natural

Proposed Multidomain Scale	RBL	PD	BOP and/or Suppuration
With baseline (1 year after abutment connection)	Progressive bone loss >0.2 mm annually compared to the baseline	Deepening PD and with progressive RBL	Suppuration and/or with clinical signs of inflammation
Without baseline	Bone level implant: RBL >2 mm from the implant platform Tissue-level implant: RBL >2 mm from the apical termination of the polished collar	PD consistent with concomitant RBL	Suppuration and/or with clinical signs of inflammation
Severity	Slight/Mild: <25% of the implant length Moderate: 25%–50% of the implant length Advanced: >50% of the implant length		

*RBL indicates radiographic bone loss; PD, probing depth; BOP, bleeding on probing.

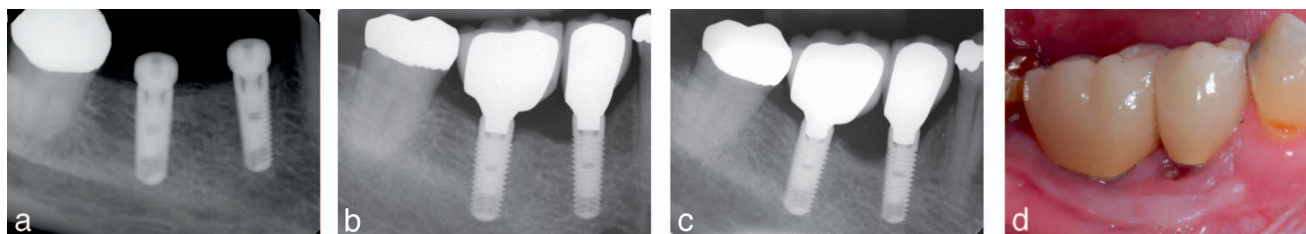


FIGURE A 67-year-old Caucasian female received dental implant placement to replace the missing teeth #29 and #30. (a) Periapical radiograph was taken at the time of implant placement. (b) Another periapical radiograph was taken 1 year after definite restoration. This periapical radiograph is served as a baseline radiograph to represent radiographic bone level. (c) Periapical radiograph taken 2 years after crown delivery showed progressive peri-implant bone loss at distal aspect of #29. No apparent peri-implant bone loss was seen at #30. (d) A clinical picture taken 2 years after crown delivery showed inflamed peri-implant tissue with suppuration at #29 and #30. Based on the proposed multi-domain scale, #29 was determined with peri-implantitis and #30 was determined with peri-implant mucositis.

teeth.^{36,41} In an animal model, Schou et al⁴¹ reported that the probe tip was closer to the bone around implants than around teeth in diseased sites, and even mild inflammation was associated with deeper probe penetration around implants. Histologically, the probe tip stopped at the apical extent of the connective tissue attachment in the mucositis sites, and close to the crestal bone level in the peri-implantitis sites. Therefore, using an absolute cut-off number for PD (ie, ≥ 4 mm) to define disease status is challenging and difficult for monitoring disease progression. Additionally, a deeper PD has been reported for implants placed subcrestally compared to those placed crestally,⁴² which indicates that a baseline PD should be established as a basis for comparison before applying PD as a diagnostic criterion.

In terms of the correlation between PD and peri-implant bone loss, Serino et al⁴³ reported a high linear and statistically significant correlation between PD recorded after removal of a prosthesis and the bone level; however, this correlation was not seen if the PD was recorded without removal of the prosthesis. Additionally, the authors also reported that only 37% of sites presented similar measurements between PD recorded with and without implant-supported prostheses, whereas 39% of sites presented a difference of 1 mm, and in 25% of sites, a 2-mm discrepancy was detected. Thus, PD measurements at sites with peri-implantitis with concomitant prosthetic reconstruction should be interpreted cautiously, since the accuracy and correlation with peri-implant bone loss is not consistent.

Based on the current evidence,^{42,43} PD may not be a reliable indicator for peri-implant disease status. Two studies that explored this topic^{2,28} suggested that there should be no cut-off value or specific PD for defining peri-implantitis. In our multidomain scale of diagnostic criteria (Table 3), we propose using a deepening PD and with progressive RBL when a baseline PD is available; if a baseline PD is not available, use a PD consistent with concomitant RBL to diagnose peri-implantitis. In a situation with a deepening PD but without signs of progressive RBL, a diagnosis of peri-implant mucositis should be made. However, if progressive RBL is detected, a diagnosis of peri-implantitis is indicated. In any case, we do not recommend using a PD cut-off number only to define disease status; RBL should always be used to define the progression of peri-implantitis.

BLEEDING ON PROBING

The presence of BOP or suppuration is generally used as one of the criteria to define peri-implant tissue inflammation. The clinical value of BOP has been studied extensively in the periodontal literature. Lang et al⁴⁴ reported that BOP is a good indicator for predicting future attachment level loss in natural dentition, with a 30% chance if BOP is present at the same site on four consecutive recall appointments. In addition, although BOP has a low positive predictive value, its negative predictive value was almost 100%,^{45,46} indicating that the absence of BOP is a good measure of periodontal health. It has been presumed that the same correlations apply to peri-implant tissues; however, this has not been clinically validated.⁴⁶

Since peri-implant tissue is less resistant to probing forces than is the periodontium,⁴⁷ simply using the presence of BOP may not adequately characterize the inflammatory status of the peri-implant tissues. Furthermore, previous studies have reported that BOP around dental implants was significant despite reduced signs of tissue inflammation.^{48,49} Therefore, because BOP does not accurately reflect the status of the peri-implant tissues, other measures (ie, redness, swelling, suppuration) are needed to evaluate the inflammatory condition around implants.

SUPPURATION

Suppuration is the most common parameter used with BOP to confirm the status of peri-implant tissue inflammation. Interestingly, though the value of this parameter has been investigated at periodontal sites,⁵⁰ there is currently no standardized method to measure this parameter at peri-implant sites. In an early study,⁵⁰ Kaldahl et al introduced the use of an egg ball burnisher to exert lateral pressure against the gingival margin to detect suppuration. A positive suppuration value was recorded if visible nonclear exudate was seen at the gingival crevice. Their study showed that gingival suppuration was a better prognosticator of future attachment loss than gingival bleeding or supragingival plaque. The authors further concluded that suppuration is an indicator of active attachment loss; however, lack of suppuration did not ensure health or the absence of breakdown.⁵⁰

With regards to peri-implant tissues, Derks et al²⁸ reported a positive value if an observation of suppuration was noted within 15 seconds following pocket probing. Other parameters—such as the plaque index, bleeding index, or gingival index—have also been used^{27,29} as adjunctive criteria to determine peri-implant tissue health. Based on the current evidence,^{46,48–50} the presence of suppuration presumably provides a higher sensitivity for peri-implant tissue breakdown than the presence of BOP. However, further clinical trials are needed to analyze the predictive values of these parameters at peri-implant sites.

SUMMARY

Based upon the currently available evidence, we propose a multidomain diagnostic scale for peri-implantitis (Table 3) that incorporates the use of RBL, PD, and BOP/suppuration. In terms of RBL, if a baseline radiograph taken at the time of abutment connection is available, we recommend less than 0.2 mm annual bone loss after physiologic bone remodeling as the acceptable threshold. If a baseline radiograph is not present, a threshold of 2 mm from the expected bone level (based on bone-level or tissue-level implants) is proposed. With respect to PD, since this parameter might not reliably reflect disease status, a threshold PD is not used as a criterion. Instead, a deepening PD with progressive RBL (if a baseline PD is available) or a PD consistent with concomitant RBL (if a baseline PD is not available) is proposed as an adjunctive parameter to define peri-implantitis. The presence of suppuration remains one of the criteria to define peri-implant tissue inflammation in our scale since this parameter tends to be more sensitive than BOP for detecting tissue breakdown. However, we suggest the inclusion of other clinical signs of tissue inflammation (ie, BOP, redness, swelling) to confirm the inflammatory status around implants. A severity scale of peri-implantitis based on the percentage of RBL relative to total implant length is also included in our proposed multidomain scale.

Since there is a high prevalence of peri-implant diseases in contemporary dentistry, a generally accepted diagnostic scale is crucial to achieve consistent diagnosis and further guide clinical practice. Although our proposed scale is based on the available evidence, future investigations and modifications may be warranted; namely, a comprehensive, evidence-based classification system is needed to address the multifactorial etiology of peri-implant diseases.

ABBREVIATIONS

BOP: bleeding on probing
PD: probing depth
RBL: radiographic bone loss

NOTE

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

REFERENCES

- Iacono VJ; Committee on Research S, Therapy tAAoP. Dental implants in periodontal therapy. *J Periodontol*. 2000;71:1934–1942.
- Sanz M, Chapple IL; Working Group 4 of the VEWoP. Clinical research on peri-implant diseases: consensus report of Working Group 4. *J Clin Periodontol*. 2012;39(suppl 12):202–206.
- American Academy of Periodontology Academy Statement. Peri-implant mucositis and peri-implantitis: a current understanding of their diagnoses and clinical implications. *J Periodontol*. 2013;84:436–443.
- Heitz-Mayfield LJ, Lang NP. Comparative biology of chronic and aggressive periodontitis vs. peri-implantitis. *Periodontol* 2000. 2010;53:167–181.
- Salvi GE, Aglietta M, Eick S, Sculean A, Lang NP, Ramseier CA. Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. *Clin Oral Implants Res*. 2012;23:182–190.
- Karoussis IK, Salvi GE, Heitz-Mayfield LJ, Bragger U, Hammerle CH, Lang NP. Long-term implant prognosis in patients with and without a history of chronic periodontitis: a 10-year prospective cohort study of the ITI Dental Implant System. *Clin Oral Implants Res*. 2003;14:329–339.
- Van der Weijden GA, van Bommel KM, Renvert S. Implant therapy in partially edentulous, periodontally compromised patients: a review. *J Clin Periodontol*. 2005;32:506–511.
- Klokkevold PR, Han TJ. How do smoking, diabetes, and periodontitis affect outcomes of implant treatment? *Int J Oral Maxillofac Implants*. 2007;22(suppl):173–202.
- Strietzel FP, Reichart PA, Kale A, Kulkarni M, Wegner B, Kuchler I. Smoking interferes with the prognosis of dental implant treatment: a systematic review and meta-analysis. *J Clin Periodontol*. 2007;34:523–544.
- Wilson TG Jr. The positive relationship between excess cement and peri-implant disease: a prospective clinical endoscopic study. *J Periodontol*. 2009;80:1388–1392.
- Miyata T, Kobayashi Y, Araki H, Ohto T, Shin K. The influence of controlled occlusal overload on peri-implant tissue. Part 3: A histologic study in monkeys. *Int J Oral Maxillofac Implants*. 2000;15:425–431.
- Monje A, Galindo-Moreno P, Canullo L, Greenwell H, Wang HL. Editorial: from early physiological marginal bone loss to peri-implant disease: on the unknown local contributing factors. *Int J Periodontics Restorative Dent*. 2015;35:764–765.
- Bashutski JD, D'Silva NJ, Wang HL. Implant compression necrosis: current understanding and case report. *J Periodontol*. 2009;80:700–704.
- Wilson TG Jr, Valderrama P, Burbano M, et al. Foreign bodies associated with peri-implantitis human biopsies. *J Periodontol*. 2015;86:9–15.
- Salvi GE, Cosgarea R, Sculean A. Prevalence and mechanisms of peri-implant diseases. *J Dent Res*. 2017;96:31–37.
- Fransson C, Lekholm U, Jemt T, Berglundh T. Prevalence of subjects with progressive bone loss at implants. *Clin Oral Implants Res*. 2005;16:440–446.
- Roos-Jansaker AM, Lindahl C, Renvert H, Renvert S. Nine- to fourteen-year follow-up of implant treatment. Part II: presence of peri-implant lesions. *J Clin Periodontol*. 2006;33:290–295.
- Koldsland OC, Scheie AA, Aass AM. Prevalence of peri-implantitis related to severity of the disease with different degrees of bone loss. *J Periodontol*. 2010;81:231–238.
- Simonis P, Dufour T, Tenenbaum H. Long-term implant survival and success: a 10–16-year follow-up of non-submerged dental implants. *Clin Oral Implants Res*. 2010;21:772–777.
- Atieh MA, Alsabeeha NH, Faggion CM Jr, Duncan WJ. The frequency of peri-implant diseases: a systematic review and meta-analysis. *J Periodontol*. 2013;84:1586–1598.
- Renvert S, Aghazadeh A, Hallstrom H, Persson GR. Factors related to peri-implantitis – a retrospective study. *Clin Oral Implants Res*. 2014;25:522–529.
- Schuldt Filho G, Dalago HR, Oliveira de Souza JG, Stanley K, Jovanovic S, Bianchini MA. Prevalence of peri-implantitis in patients with implant-supported fixed prostheses. *Quintessence Int*. 2014;45:861–868.
- Aguirre-Zorzano LA, Estefania-Fresco R, Telletxea O, Bravo M. Prevalence of peri-implant inflammatory disease in patients with a history of periodontal disease who receive supportive periodontal therapy. *Clin Oral Implants Res*. 2015;26:1338–1344.
- Daubert DM, Weinstein BF, Bordin S, Leroux BG, Flemming TF. Prevalence and predictive factors for peri-implant disease and implant failure: a cross-sectional analysis. *J Periodontol*. 2015;86:337–347.
- Konstantinidis IK, Kotsakis GA, Gerdes S, Walter MH. Cross-sectional

study on the prevalence and risk indicators of peri-implant diseases. *Eur J Oral Implantol.* 2015;8:75–88.

26. Schwarz F, Becker K, Sahn N, Horstkemper T, Rousi K, Becker J. The prevalence of peri-implant diseases for two-piece implants with an internal tube-in-tube connection: a cross-sectional analysis of 512 implants. *Clin Oral Implants Res.* 2017;28:24–28.

27. Dalago HR, Schuldt Filho G, Rodrigues MA, Renvert S, Bianchini MA. Risk indicators for peri-implantitis. A cross-sectional study with 916 implants. *Clin Oral Implants Res.* 2017;28:144–150.

28. Derks J, Schaller D, Hakansson J, Wennstrom JL, Tomasi C, Berglundh T. Effectiveness of implant therapy analyzed in a Swedish population: prevalence of peri-implantitis. *J Dent Res.* 2016;95:43–49.

29. Rokn A, Aslroosta H, Akbari S, Najafi H, Zayeri F, Hashemi K. Prevalence of peri-implantitis in patients not participating in well-designed supportive periodontal treatments: a cross-sectional study. *Clin Oral Implants Res.* 2017;28:314–319.

30. Meijer HJ, Raghoobar GM, de Waal YC, Vissink A. Incidence of peri-implant mucositis and peri-implantitis in edentulous patients with an implant-retained mandibular overdenture during a 10-year follow-up period. *J Clin Periodontol.* 2014;41:1178–1183.

31. Ferreira SD, Silva GL, Cortelli JR, Costa JE, Costa FO. Prevalence and risk variables for peri-implant disease in Brazilian subjects. *J Clin Periodontol.* 2006;33:929–935.

32. Albrektsson T, Zarb G, Worthington P, Eriksson AR. The long-term efficacy of currently used dental implants: a review and proposed criteria of success. *Int J Oral Maxillofac Implants.* 1986;1:11–25.

33. Adell R, Lekholm U, Rockler B, Branemark PI. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. *Int J Oral Surg.* 1981;10:387–416.

34. Schwartz-Arad D, Herzberg R, Levin L. Evaluation of long-term implant success. *J Periodontol.* 2005;76:1623–1628.

35. Hermann JS, Jones AA, Bakaeen LG, Buser D, Schoolfield JD, Cochran DL. Influence of a machined collar on crestal bone changes around titanium implants: a histometric study in the canine mandible. *J Periodontol.* 2011;82:1329–1338.

36. Lang NP, Berglundh T; Working Group 4 of the Seventh European Workshop on Periodontology. Periimplant diseases: where are we now? Consensus of the Seventh European Workshop on Periodontology. *J Clin Periodontol.* 2011;38(suppl 11):178–181.

37. Froum SJ, Rosen PS. A proposed classification for peri-implantitis. *Int J Periodontics Restorative Dent.* 2012;32:533–540.

38. Decker AM, Sheridan R, Lin GH, Sutthiboonyapan P, Carroll W, Wang HL. A prognosis system for periimplant diseases. *Implant Dent.* 2015;24:416–421.

39. Jeffcoat MK, Reddy MS. Advances in measurements of periodontal bone and attachment loss. *Monogr Oral Sci.* 2000;17:56–72.

40. Berglundh T, Lindhe J, Ericsson I, Marinello CP, Liljenberg B, Thomsen P. The soft tissue barrier at implants and teeth. *Clin Oral Implants Res.* 1991;2:81–90.

41. Schou S, Holmstrup P, Stoltze K, Hjorting-Hansen E, Fiehn NE, Skovgaard LT. Probing around implants and teeth with healthy or inflamed peri-implant mucosa/gingiva. A histologic comparison in cynomolgus monkeys (*Macaca fascicularis*). *Clin Oral Implants Res.* 2002;13:113–126.

42. Huang B, Meng H, Piao M, Xu L, Zhang L, Zhu W. Influence of placement depth on bone remodeling around tapered internal connection implant: a clinical and radiographic study in dogs. *J Periodontol.* 2012;83:1164–1171.

43. Serino G, Turri A, Lang NP. Probing at implants with peri-implantitis and its relation to clinical peri-implant bone loss. *Clin Oral Implants Res.* 2013;24:91–95.

44. Lang NP, Joss A, Orsanic T, Gusberti FA, Siegrist BE. Bleeding on probing. A predictor for the progression of periodontal disease? *J Clin Periodontol.* 1986;13:590–596.

45. Lang NP, Adler R, Joss A, Nyman S. Absence of bleeding on probing. An indicator of periodontal stability. *J Clin Periodontol.* 1990;17:714–721.

46. Lang NP, Wilson TG, Corbet EF. Biological complications with dental implants: their prevention, diagnosis and treatment. *Clin Oral Implants Res.* 2000;11(suppl 1):146–155.

47. Gerber JA, Tan WC, Balmer TE, Salvi GE, Lang NP. Bleeding on probing and pocket probing depth in relation to probing pressure and mucosal health around oral implants. *Clin Oral Implants Res.* 2009;20:75–78.

48. Bragger U, Burgin WB, Hammerle CH, Lang NP. Associations between clinical parameters assessed around implants and teeth. *Clin Oral Implants Res.* 1997;8:412–421.

49. Karoussis IK, Muller S, Salvi GE, Heitz-Mayfield LJ, Bragger U, Lang NP. Association between periodontal and peri-implant conditions: a 10-year prospective study. *Clin Oral Implants Res.* 2004;15:1–7.

50. Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP. Relationship of gingival bleeding, gingival suppuration, and supragingival plaque to attachment loss. *J Periodontol.* 1990;61:347–351.