Dental implant complications have grown rapidly over the past decades. Peri-implant diseases, like periodontal diseases, have been regarded as a complex interaction between bacteria and host-defense. Thus, maintenance care could be one efficient method for better plaque control and for the prevention of subsequent disease progression. The etiologic factors of plaque-induced peri-implant mucositis and peri-implantitis are primarily correlated with bacterial infection, and plaque accumulation after implant placement remains the main cause of the disease. To prevent bacterial-associated biologic complications, supportive therapy for dental implants should be instituted soon after restoration delivery to lower the risk.

A cumulative interceptive supportive therapy was proposed as one of the tools to ensure the stability of implant health over a long-term period. This therapy was developed according to the diagnosis at recall visits with guidelines of different supportive therapies.
It is generally recommended that the interval of cumulative interceptive supportive therapy should be individually tailored based on patients’ needs as well as clinical diagnosis and risk profiles.\(^6\) However, at least a 5- to 6-month interval was recommended in several studies.\(^{10-13}\) No matter which supportive therapies were followed, the consensus supported that professional intervention is mandatory for implant maintenance.\(^6,8\)

History of periodontitis (HP) has been considered to be one of the most significant risk indicators of peri-implant mucositis and peri-implantitis.\(^{12,14,15}\) HP patients often correlate with poor compliance, higher host susceptibility, and poor plaque control.\(^{12,14,15}\)

Under supportive peri-implant treatment (SPT) coverage, some have shown that HP could trigger lower implant survival rates and poor clinical parameters due to residual pockets.\(^8,16\) Nonetheless, there is a lack of evidence if HP remains a risk indicator under SPT. Moreover, despite the negative impacts of HP on peri-implant health, SPT could be the answer to long-term stability of implants. Therefore, by comparing all the clinical outcomes during the maintenance period between patients with and without HP, the purpose of this review was to evaluate whether HP remains a negative indicator even under regular SPT.

In the present review, the primary purpose was focused on implant survival rate, and all clinical parameters were assessed as the secondary purpose to evaluate whether HP possesses a negative impact on peri-implant health under regular SPT coverage.

**MATERIALS AND METHODS**

The present systematic review and meta-analysis was conducted and reported following the checklist of PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement.\(^17\)

**Focused Question**

In the present literature, the question of this search was illustrated according to the Population, Intervention, Comparison, and Outcome (PICO) criteria\(^18\):

- **P:** Patients who received one or more dental implants with a past history of periodontitis were the population.
- **I:** After implant loading, all patients were followed up under either regular or tailored SPT coverage.
- **C:** The differences in implant survival rate and peri-implant clinical parameters (eg, radiographic marginal bone loss, pocket depth, bleeding on probing, and plaque accumulation index) were evaluated between patients with and without HP.
- **O:** The primary outcome was the differences of survival rates at the implant level between HP and non-HP patients.

In addition, secondary outcomes considered differences in related clinical parameters (radiographic marginal bone loss, pocket depth, bleeding on probing, and plaque accumulation index).

Hence, the focused question for this review was “whether history of periodontitis has negative impacts on peri-implant health under regular SPT coverage.”

**Selection Criteria**

Potentially qualified studies that met the following criteria were included: (1) any human studies (ie, prospective or retrospective, randomized or controlled clinical trials) with SPT application; (2) the details of SPT should be provided in studies for implant maintenance; (3) the article should contain and compare the outcomes of implants from both patients with and without HP; and (4) peri-implant conditions (either survival rate, radiographic marginal bone loss, plaque and bleeding status, or prevalence of peri-implant mucositis and/or peri-implantitis) are required for data extraction.

**Search Strategy**

Three electronic databases—MEDLINE, EMBASE, and Cochrane Central—were used to conduct the search process for articles published up to May 2018 in English. The search strategy used in PubMed was as follows: ( (((supportive treatment[Title/Abstract]) OR maintenance[Title/Abstract])) AND (((dental implant [MeSH Terms]) OR dental implantation[MeSH Terms])) OR implant[Title/Abstract])))) AND periodontitis. For the EMBASE library, the search terms were as follows: (‘tooth implantation’/de OR ‘tooth implant’/de OR ‘implant’/de) AND (‘maintenance therapy’/de OR ‘supportive therapy’/de OR ‘maintenance’/exp OR maintenance) AND (‘periodontal disease’/de). For the Cochrane Library, implant AND maintenance AND periodontitis was applied in Title, Abstract, and Keywords in Trials.

maintenance therapy on the prevention of peri-implant diseases were also screened for article identification.

Two independent reviewers (C.L. and Z.C.) screened the articles in the search process. Among all articles, inclusion criteria, titles, and abstracts of search results were evaluated, and potential articles were then assessed in full text. The level of interreviewer agreement for study inclusion was calculated by κ value. Following thorough discussion and consultation, a final decision would be made by with a senior reviewer (H.L.W.) if there was a disagreement on selected articles.

**Risk of Bias Assessment**

The quality assessment of included nonrandomized studies was judged using the Newcastle-Ottawa Scale (NOS). Each selected article was evaluated on eight items, among which four items consider the selection of cohorts/case and controls; one item is related to the comparability of the cohorts/case and controls; and three items are based on the ascertainment of the outcome/exposure. In the “selection” and “outcome” sections, one star is awarded for each item if the criteria are fulfilled. The final scores ranged from 0 to 8, considering 0 to 3 as low, 4 to 6 as moderate, and 7 to 8 as high quality, respectively.

**Data Extraction and Statistical Analysis**

Screening all eligible articles, two independent reviewers (C.L. and Z.C.) extracted the data for further evaluation. Any interreviewer disagreement was resolved by discussion. Additionally, corresponding authors of potential articles were contacted in cases of unclear or missing data.

Statistical software program (Stata software, v14.0, StataCorp) was applied for conducting statistical analyses. To standardize the reporting of results, risk ratios (RRs) and 95% confidence interval (CI) were calculated from the absolute number of events reported in survival rate, and they were analyzed at the implant level. Weighted mean difference (WMD) and 95% CI were used to compare clinical parameters (radiographic marginal bone loss, pocket depth, and bleeding on probing) between patients with and without a history of periodontitis. A random-effects model was used if heterogeneity among trials tested with the Q test ($P < .1$) and I² statistics $\geq 75\%$ proved to be high. To avoid bias from combining differently designed trials, meta-analysis with the same implant surface (machined or rough) was performed in subgroups.

The probability of publication bias was assessed with Harbord plot for dichotomous data and Egger plot for continuous data. A significant publication bias was considered if $P < .05$.

**RESULTS**

**Study Selection**

The screening process is shown in Fig 1. A total of 319 records from different electronic databases (PubMed: 172, EMBASE: 136, Cochrane Database: 11) and 10 records from manual searching were found in the initial screening. After assessing the titles and abstracts of potential studies, 18 articles were further selected for full-text screening, and 5 of them were excluded with reasons (Table 1). Finally, 13 articles were included for the quantitative synthesis and meta-analyzed to evaluate the impact of HP on survival rate, radiographic marginal bone loss, pocket depth, and bleeding on probing around implants. Regarding the interreviewer agreement for potentially relevant studies, the k value was 0.85 in title/abstract screening and 0.92 in full-text screening, indicating a consistent agreement between the two reviewers.

**Description of Studies**

The main characteristics of included studies were summarized with details, and articles either with or without HP were presented in terms of clinical parameters (Table 2). First, 7 of 13 were prospective studies. Although the variation of follow-up duration in the present review was shown to be 4 to 16 years, the mean duration of implant maintenance was more than 8 years in most studies, not including two articles with 5 and 7.99 years. Regarding the type of periodontitis, included subjects were diagnosed as generalized aggressive periodontitis in three articles, while some articles completely excluded the patients with generalized aggressive periodontitis. In addition, different implant surfaces were mentioned in different articles: Three articles used a turned/machined surface, and the remaining articles included rough-surface implants with different systems. With regard to smoking habit, one study only focused on heavy smokers, two articles did place emphasis on nonsmokers independently as a different group, and 10 studies included both smokers and nonsmokers. Additionally, most cases did not reveal the percentage of peri-implant mucositis or peri-implantitis under SPT, while two studies mentioned the differences between HP and non-HP groups: 15.4% to 26% versus 0% to 10% occurrence of peri-implantitis at the implant level; 16.2% to 42.8% versus 0% to 11.1% at the patient level, respectively.

**Risk of Bias and Quality Assessment**

A total of 13 selected clinical trials that fulfilled the inclusion criteria were assessed with the risk of bias (Appendix Table 1; see online version of this article at quintpub.com). The Newcastle-Ottawa Scale (NOS)
was applied for quality assessment of cohort studies and case-control studies. There were four articles (30.8%) comprising six stars after evaluation based on the inadequate representativeness of the exposed cohort, unclear selection process of controls, and insufficient comparability. On the other hand, four articles (30.8%) received seven stars according to insufficient comparability in SPT and non-SPT. Four studies (30.8%) met all criteria and received eight stars with high quality after assessment.

Primary Outcomes (History of Periodontitis to Implant Survival Rate)
Eleven articles reported the value of implant survival rate during SPT observation, among which nine studies used rough-surface implants and three studies used machine-surface implants. As for the primary outcome, the present review extracted the data of the implant survival rate in both patients with and without HP from included articles (Fig 2).

The results showed a significantly higher implant survival rate in the non-HP group compared with the HP group (RR: 0.96, 95% CI: 0.94 to 0.99, \( P < .001 \)). Subgroup analysis was performed to examine the effect of implant surface and showed that HP combined with a rough surface could contribute to a significantly lower implant survival rate (RR: 0.96, 95% CI: 0.94 to 0.98, \( P < .001 \)) compared with a machined surface. Interestingly, in implants with a machined surface, no statistical significance was found between HP and non-HP patients (RR: 0.98, 95% CI: 0.92 to 1.04, \( P = .895 \)). Low heterogeneity was found among these studies in both subgroups (machined-surface group: \( I^2 = 0.0\% \), \( P = .907 \); rough-surface group: \( I^2 = 22.0\% \), \( P = .227 \)).

Secondary Outcomes (History of Periodontitis to Clinical Parameters)
Radiographic marginal bone loss was significantly higher in the HP group than in the non-HP group.

Table 1  Excluded Articles

<table>
<thead>
<tr>
<th>Study (year)</th>
<th>Reasons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ferreira (2006)</td>
<td>No exact numbers of clinical parameters for both test and control groups</td>
</tr>
<tr>
<td>Rinke (2011)</td>
<td>No exact numbers for post-implant outcomes</td>
</tr>
<tr>
<td>Frisch (2014)</td>
<td>No exact numbers for post-implant outcomes</td>
</tr>
<tr>
<td>Monje (2017)</td>
<td>No exact numbers of clinical parameters for both test and control groups</td>
</tr>
<tr>
<td>Gallego (2018)</td>
<td>No control group for comparison</td>
</tr>
</tbody>
</table>
(WMD: 0.49 mm, 95% CI: 0.37 to 0.61, P < .001) (Fig 3). Among all the studies, five used rough-surface implants,¹⁶,²⁵,²⁸,³⁰,³³ and the other two studies used machined-surface implants.²⁵,³⁰ Subgroup analysis based on implant surface design was then conducted. Both groups showed that HP could significantly increase radiographic marginal bone loss, and there was low heterogeneity among selected studies (machined-surface group: 0.88 mm [WMD], 0.65 to 1.11 [95% CI], P < .001; I² = 0.0%, P = .854; rough-surface group: 0.34 mm [WMD], 0.20 to 0.48 [95% CI], P < .001; I² = 12.8%, P = .333).
### Table 2  Included Articles with Outcome Variables with Control Group

<table>
<thead>
<tr>
<th>Study</th>
<th>Period</th>
<th>Type</th>
<th>Treatment</th>
<th>Follow-up</th>
<th>Subgroup</th>
<th>Outcome Variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lee et al36</td>
<td>2014</td>
<td>Cohort</td>
<td>None</td>
<td>3M</td>
<td>None</td>
<td>Survival, N (%)</td>
</tr>
<tr>
<td>Swierkot et al35</td>
<td>2010, 2012</td>
<td>Pros</td>
<td>None Tailored 10Y</td>
<td>P:29</td>
<td>None NR 10Y</td>
<td>Survival, N (%)</td>
</tr>
<tr>
<td>Rasperini et al30</td>
<td>2014</td>
<td>Cohort</td>
<td>None</td>
<td>3M</td>
<td>None</td>
<td>Survival, N (%)</td>
</tr>
<tr>
<td>Aglietta et al 25</td>
<td>2011</td>
<td>Retro</td>
<td>None</td>
<td>6M</td>
<td>None</td>
<td>Survival, N (%)</td>
</tr>
<tr>
<td>De Boever et al26</td>
<td>2010</td>
<td>Pros</td>
<td>None NR 10Y</td>
<td>P:25</td>
<td>None NR 10Y</td>
<td>Survival, N (%)</td>
</tr>
<tr>
<td>Mengel et al29</td>
<td>2014</td>
<td>Cohort</td>
<td>None</td>
<td>3M</td>
<td>None</td>
<td>Survival, N (%)</td>
</tr>
<tr>
<td>Roccuzzo et al31,33</td>
<td>2017</td>
<td>Pros</td>
<td>None NR 10Y</td>
<td>P:20</td>
<td>None NR 10Y</td>
<td>Survival, N (%)</td>
</tr>
<tr>
<td>Roccuzzo et al32</td>
<td>2017</td>
<td>Pros</td>
<td>None NR 10Y</td>
<td>P:20</td>
<td>None NR 10Y</td>
<td>Survival, N (%)</td>
</tr>
<tr>
<td>Tan et al32 (2017)</td>
<td>2011</td>
<td>Retro</td>
<td>None</td>
<td>3M</td>
<td>None</td>
<td>Survival, N (%)</td>
</tr>
</tbody>
</table>

**Other Clinical Parameters (Pocket Depth, Bleeding on Probing, Plaque Accumulation Index)**

All involved articles relating other clinical parameters only contained rough-surface implants, as a result, no subgroup analysis was performed. In view of pocket depth and bleeding on probing around implants, larger values were observed in the HP group (WMD: 0.47 mm, 95% CI: 0.19 to 0.74, P < .001; WMD: 0.08 mm, 95% CI: 0.04 to 0.11, P < .001) compared with the non-HP group (Figs 4 and 5). Regarding conditions of HP patients under regular SPT, the range of pocket depth and bleeding on probing around implants, larger values were observed in the HP group (WMD: 0.47 mm, 95% CI: 0.19 to 0.74, P < .001; WMD: 0.08 mm, 95% CI: 0.04 to 0.11, P < .001) compared with the non-HP group (Figs 4 and 5).
### Study ID Survival rate RR (95% CI) Events, treatment Events, control % weight

**R**
- Karoussis (P+) (2003) 1.01 (0.88, 1.14) 11/11 70/73 2.45
- Mengel (2007) 0.88 (0.70, 1.11) 30/36 7/7 1.54
- De Boever GaGP (2009) 0.87 (0.78, 0.98) 50/59 253/261 11.62
- De Boever CAP (2009) 0.99 (0.96, 1.03) 186/193 253/261 26.79
- Aglietta (2011) 0.90 (0.69, 1.18) 9/10 10/10 1.31
- Rocuzzo (ModP) (2012) 0.96 (0.89, 1.03) 88/95 59/61 8.95
- Rocuzzo (SevP) (2012) 0.93 (0.86, 1.01) 81/90 59/61 8.76
- Rasperini (2014) 0.89 (0.73, 1.10) 17/20 19/20 2.37
- Rocuzzo (ModP) (2014) 0.95 (0.88, 1.03) 52/55 32/32 5.09
- Rocuzzo (SevP) (2014) 0.97 (0.91, 1.03) 71/74 32/32 5.62
- Tan (2017) 1.02 (0.96, 1.08) 59/60 54/56 6.96
- Graetz (2018) 1.00 (0.94, 1.05) 67/69 74/76 8.77
- Subtotal (I² = 22.0%, P = .227) 0.96 (0.94, 0.98) 721/772 922/950 90.21

**T**
- Aglietta (2011) 1.00 (0.75, 1.34) 9/10 9/10 1.12
- Swierkot GaGP (2012) 0.97 (0.92, 1.03) 144/150 30/30 6.30
- Rasperini (2014) 1.00 (0.87, 1.15) 19/20 19/20 2.37
- Subtotal (I² = 0.0%, P = .907) 0.98 (0.92, 1.04) 172/180 58/60 9.79

**Overall (I² = 0.0%, P = .457)** 0.96 (0.94, 0.99) 893/952 980/1010 100.00

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**Fig 2** Meta-analysis was performed in assessment of implant survival rate in patients with and without history of periodontitis (HP), and subgroups were also assessed based on different implant surfaces: rough surface (R) and turned surface (T). GaGP = generalized aggressive periodontitis; SevP = severe periodontitis; ModP = moderate periodontitis; CAP = chronic adult periodontitis.

### Study ID RMBL WMD (95% CI) N, mean (SD) treatment N, mean (SD) control % weight

**R**
- Karoussis (2003) 0.48 (–0.00, 0.96) 21, .97 (1) 91, .49 (1.09) 6.29
- Rocuzzo (ModP) (2010) 0.39 (0.07, 0.71) 88, 1.14 (1.11) 61, 0.75 (1.09) 14.31
- Rocuzzo (SevP) (2010) 0.23 (–0.12, 0.58) 81, 0.98 (1.22) 61, 0.75 (.88) 12.29
- Lee PCP (2012) 0.19 (–0.12, 0.50) 56, 0.45 (.94) 61, 0.26 (.72) 15.73
- Aglietta (2011) 0.37 (0.11, 0.63) 20, 2.32 (.41) 20, 1.95 (.42) 22.17
- Rasperini (2014) 0.34 (0.20, 0.48) 276 304 72.58
- Subtotal (I² = 12.8%, P = .333)

**T**
- Aglietta (2011) 0.82 (0.12, 1.52) 10, 3.47 (1.09) 10, 2.65 (.31) 2.97
- Rasperini (2014) 0.89 (0.65, 1.13) 20, 2.32 (.41) 20, 1.43 (.38) 24.44
- Subtotal (I² = 0.0%, P = .854) 0.88 (0.65, 1.11) 30 30 27.42

**Heterogeneity between groups: P = .000**

**Overall (I² = 6.66%, P = .004)**

---

**Fig 3** Meta-analysis was performed in assessment of radiologic marginal bone loss (RMBL) in patients with history of periodontitis (HP) or without HP. SevP = severe periodontitis; ModP = moderate periodontitis; T = turned surface; R = rough surface.
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depth was 2.5 to 4.2 mm, and the range of bleeding on probing (%) was 13% to 35% (Table 2). Moreover, different indices were used to record plaque accumulation index in five articles (Table 3): Plaque Index in one study,\(^31\) full-mouth Plaque Index in two studies,\(^{16,32}\) and modified Plaque Index\(^39\) in two studies.\(^{28,34}\) Hence, meta-analysis could not be conducted in plaque accumulation index due to various record indices being used.

**DISCUSSION**

**Patient with History of Periodontitis Under Regular SPT**

It is generally agreed that plaque is the primary etiologic factor for peri-implant mucositis and peri-implantitis; hence, it is often believed that individuals with HP may be prone to having peri-implant mucositis and peri-implantitis.\(^3^5\) This is because natural dentition may be deemed as a bacteria reservoir that contributes bacterial pathogens for later infection around implants, especially in patients with HP.\(^4^0\) Research has demonstrated a cause-effect relationship between plaque and inflammation during non-SPT,\(^4^1\) and one review and meta-analysis also proposed that non-SPT maintenance could be correlated with peri-implant disease.\(^2\) Although HP has been regarded as one of most significant risk indicators of peri-implantitis,\(^3,5,14,42,43\) no article has actually examined if this effect remains true when HP patients are under regular SPT care.

**Primary Outcome (Effect of History of Periodontitis to Implant Survival Rate)**

Data from the present study indicated that the existence of HP could significantly affect implant survival rate in patients under regular SPT.\(^16,25,26,29,33,35,36\) Nonetheless, some studies did show an equally favorable implant survival rate compared with non-HP patients.\(^16,26,27,30,32,37\) This could be explained by some well-known negative indicators being concomitantly included in the study. First, three articles included generalized aggressive periodontitis patients,\(^26,29,35\) who are often associated with an impaired host immune response.

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**Table 2** Meta-analysis was conducted to compare pocket depth (PD) in patients with history of periodontitis (HP) or without HP. SevP = severe periodontitis; ModP = moderate periodontitis.

<table>
<thead>
<tr>
<th>Study ID</th>
<th>PD</th>
<th>WMD (95% CI)</th>
<th>% weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Karoussis (2003)</td>
<td>0.51 (-0.21, 1.23)</td>
<td>7.37</td>
<td></td>
</tr>
<tr>
<td>Roccuzzo (ModP) (2012)</td>
<td>0.40 (0.17, 0.63)</td>
<td>13.89</td>
<td></td>
</tr>
<tr>
<td>Roccuzzo (SevP) (2012)</td>
<td>0.80 (0.59, 1.01)</td>
<td>14.02</td>
<td></td>
</tr>
<tr>
<td>Lee PCP (2012)</td>
<td>0.02 (-0.18, 0.22)</td>
<td>14.21</td>
<td></td>
</tr>
<tr>
<td>Roccuzzo (ModP) (2014)</td>
<td>0.00 (-0.30, 0.30)</td>
<td>12.93</td>
<td></td>
</tr>
<tr>
<td>Roccuzzo (SevP) (2014)</td>
<td>0.40 (0.10, 0.70)</td>
<td>13.01</td>
<td></td>
</tr>
<tr>
<td>Seki (2017)</td>
<td>0.46 (0.19, 0.73)</td>
<td>13.39</td>
<td></td>
</tr>
<tr>
<td>Graetz (2018)</td>
<td>1.30 (0.88, 1.72)</td>
<td>11.19</td>
<td></td>
</tr>
<tr>
<td>Overall (I(^2) = 86.5%, (P = .000))</td>
<td>0.47 (0.19, 0.74)</td>
<td>100.00</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 4** Meta-analysis was conducted to compare pocket depth (PD) in patients with history of periodontitis (HP) or without HP. SevP = severe periodontitis; ModP = moderate periodontitis.

**Table 3** Meta-analysis was performed to compare bleeding on probing (BoP) in patients with history of periodontitis (HP) or without HP. SevP = severe periodontitis; ModP = moderate periodontitis.

<table>
<thead>
<tr>
<th>Study ID</th>
<th>BoP</th>
<th>WMD (95% CI)</th>
<th>% weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>Karoussis (2003)</td>
<td>-0.11 (-0.29, 0.07)</td>
<td>3.26</td>
<td></td>
</tr>
<tr>
<td>Roccuzzo (ModP) (2012)</td>
<td>0.10 (0.09, 0.11)</td>
<td>26.53</td>
<td></td>
</tr>
<tr>
<td>Roccuzzo (SevP) (2012)</td>
<td>0.14 (0.13, 0.15)</td>
<td>26.51</td>
<td></td>
</tr>
<tr>
<td>Roccuzzo (ModP) (2014)</td>
<td>-0.02 (-0.12, 0.08)</td>
<td>7.68</td>
<td></td>
</tr>
<tr>
<td>Roccuzzo (SevP) (2014)</td>
<td>0.08 (-0.02, 0.18)</td>
<td>7.73</td>
<td></td>
</tr>
<tr>
<td>Seki (2017)</td>
<td>-0.02 (-0.13, 0.09)</td>
<td>7.39</td>
<td></td>
</tr>
<tr>
<td>Tan (2017)</td>
<td>0.06 (0.02, 0.10)</td>
<td>20.91</td>
<td></td>
</tr>
<tr>
<td>Overall (I(^2) = 92.2%, (P = .000))</td>
<td>0.08 (0.04, 0.11)</td>
<td>100.00</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 5** Meta-analysis was performed to compare bleeding on probing (BoP) in patients with history of periodontitis (HP) or without HP. SevP = severe periodontitis; ModP = moderate periodontitis.
system and high susceptibility to periodontal diseases. To compare with non-HP patients, one study showed an equally high implant survival rate in the short-term follow-up, but it was significantly lower 5 years later. Therefore, generalized aggressive periodontitis might be considered as a strong negative influence factor other than HP in terms of implant survival rate. Second, both HP and smoking could be associated with higher risks for developing peri-implantitis and peri-implantitis, and the combination of both might have an even stronger negative effect on implant survival rate and radiographic marginal bone loss.

Different surface designs of implants could also be related to different progression of peri-implantitis. A rough surface has often been related to being more inclined to have peri-implantitis, bone loss, and implant loss. The results obtained from the present study are in agreement with the aforementioned findings. Basically, when patients have HP, individuals with rough-surface implants could have a lower implant survival rate compared to individuals without HP even if these individuals are under regular SPT. Among all included articles, some studies placed a strong emphasis on the effect of implant surface toward development of peri-implantitis. In contrast with rough-surface implants, machined-surface implants showed no significant difference between HP and non-HP groups despite smokers and patients with generalized aggressive periodontitis.

Secondary Outcomes (Effect of History of Periodontitis on Clinical Parameters)

According to the guidelines of SPT, clinical parameter changes including radiographic marginal bone loss, pocket depth, bleeding on probing, and plaque accumulation index can represent the efficacy of SPT, the severity of peri-implant disease, and the outcomes of disease resolution after intervention. Articles have shown a positive correlation between higher bone loss and lower implant survival rate, and this is especially true in patients with generalized aggressive periodontitis, rough-surface implants, smoking, and residual pocket depth. In the present study, different implant surfaces were also investigated, and the results showed that radiographic marginal bone loss of both machined- and rough-surface implants was negatively impacted by HP. The results of marginal bone loss, unlike implant survival rate, had less benefit in machined-surface implants in patients with HP. The observation could be attributed to two related articles that were included in the machined-surface group, and one of them included only heavy smokers. Based on the observation of heavy smokers in that study, the amount of bone loss in non-HP patients was even more than that in the HP group in the other article in implants with either rough or machined surfaces. Therefore, along with implant surface, tobacco smoking could be another factor to consider in terms of radiographic marginal bone loss around implants. In other words, the impact of implant surface might be increased in smokers.

Lastly, these two studies may not take into consideration initial biologic bone remodeling, especially in the machined surface. The aforementioned three reasons may explain why there is slightly more impact of HP in the machined surface in terms of bone loss under regular SPT. Other factors may be considered to influence the relationship between HP and peri-implant disease, including but not limited to: implant positioning, residual cement, overcontouring or improper contouring of the prosthesis that made it difficult for the patient to clean, and many others. Due to the limitations of this study scope, this was not discussed in the present article.

When pocket depth and bleeding on probing were assessed, most of the included studies showed shallow pocket depth and acceptable bleeding on probing.
when patients, with or without HP, were placed under regular SPT coverage. It is generally understood that pocket depth can be influenced by position of implant, gingival thickness, presence of prosthesis, and the contour of the emergence profile; hence, the value could not be generally compared. Yet, residual pocket depth combined with bleeding on probing around both implants and adjacent teeth often indicate an early stage of disease, and they could be regarded as a negative factor to long-term success. Results obtained in the present review showed higher pocket depth in patients with HP; however, both groups presented a stable mean value of pocket depth less than 5 mm. This might suggest that regular SPT can help maintain implant health, either with or without HP. Interestingly, in the present study, most selected articles had < 20% of bleeding on probing in the non-HP group, except in one study, which found that bleeding on probing was even higher than the HP group. This finding might question the validity of using bleeding on probing alone as a diagnosis tool, particularly the tissue around dental implants.

Last but not least, plaque control can be the ultimate goal in SPT regimens, and the records of oral hygiene are mandatory during recall visits. However, in the present review, because three different plaque indices were used, the efficacy of SPT and the differences between the HP and non-HP groups could be assessed independently. Nonetheless, it was acknowledged that patients with HP, ending up with higher plaque indices, should go with a meticulous plaque control and a comprehensive SPT regimen for better long-term implant stability.

Limitations

This review had several limitations that should be mentioned. First, clinical trials but not randomized clinical trials were available, and limited articles were included in this meta-analysis, which could reduce the power of the results. Furthermore, different definitions and severity of history of periodontitis and peri-implantitis have been taken in selected papers, and these might have impacts on the results. Aside from the heterogeneity in diagnosis of peri-implantitis, smokers were included in most studies with uneven distribution, and the combination of different negative indicators could lead to synergistic effects in primary and secondary outcomes. Additionally, lack of baseline values of clinical parameters (pocket depth, bleeding on probing) made it difficult to investigate the change of peri-implant tissue in all aspects. As a result, it was not possible to eliminate individual bias. Consequently, all results should be interpreted with caution because of all the aforementioned limitations.

CONCLUSIONS

Despite the limitations of this study, history of periodontitis remains a negative indicator for implant survival rate, even under regular supportive post-implant treatment coverage, especially in rough-surfaced implants. To evaluate the role of history of periodontitis in peri-implant conditions under supportive post-implant treatment, more consistent and well-controlled studies with complete data are required in this field.

ACKNOWLEDGMENTS

University of Michigan Periodontal Graduate Student Research Fund partially supported this paper. No conflicts of interest were reported by the authors.

REFERENCES


### Appendix Table 1  
Newcastle-Ottawa Scale (NOS) for Included Clinical Trials

<table>
<thead>
<tr>
<th>Study (year)</th>
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<th>Comparability</th>
<th>Outcome</th>
</tr>
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<td>Representativeness of the cases</td>
<td>Selection of controls</td>
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