

## Comparison between Obesity and Periodontitis Status

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### Abstract

Obesity is a disorder involving an excessive amount of body fat. People with obesity are at high risk for diabetes mellitus, hypertension, stroke, heart attack, and cancer. The objective of this study was to analyze the relationship between obesity and periodontitis status in patients who did not smoke. Participants were 36 people aged 35 to 55 years, 23 of whom were obese (intima-media thickness [IMT] >25) and 13 of whom were not (control subjects; IMT ≤ 25). The periodontitis status was reflected by pocket depth, gingival recession, and tooth loss. The Mann-Whitney U test, used to analyze the relationship between obesity and periodontitis status, revealed greater pocket depth and loss of more teeth among the non-obese participants, but there was no significant relationship between obesity and depth of the pocket or between obesity and loss of the teeth. The amount of recession was higher among obese patients than among nonobese patients. The relationship between obesity and periodontitis status was not significant. This result could have been affected by local factors such as plaque and oral trauma. Our tool for measuring obesity was body mass index modified for Indonesia. Further study with other obesity measurements are needed to clarify our results.

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### Introduction

Obesity is the presence of excessive body fat. The causes of obesity include genetic, environmental, medical, and physiological factors, as well as low amounts of activity.<sup>1</sup> Obesity can be determined by measure the body mass index (**BMI**), a measure of body fat that is based on the body weight and height of a person. The body weight in kilograms and the height in meters are used to calculate BMI<sup>2</sup>: BMI = body weight (kg)/height<sup>2</sup> (m). According to Indonesia's Department of Health, BMI represents weight status as follows<sup>3,4</sup>: Extremely underweight, <17; Somewhat underweight, 17 to 18.4; Normal, 18.5 to 25; Somewhat obese, 25.1 to 27; Extremely obese, >27. The BMI classification by the World Health Organization has been modified for Indonesia according to clinical experiences and trials in this economically developing country.

This BMI classification has been used to guide health recommendations. People with obesity are at high risk for diabetes mellitus, hypertension, stroke, heart attack, cancer, and other serious disorders. Obesity also has been known to aggravate periodontitis. Periodontitis is a chronic inflammation of the periodontal tissue that can lead to loss of teeth. Several investigators have found that the prevalence of periodontitis among people with obesity is 76%.<sup>5</sup> This prevalence is higher than that among people with normal weight. Other studies by Genco showed that there is a positive correlation between obesity and depth of the pocket.<sup>6</sup> In 2008, study from Amin showed that the rate of clinical attachment loss is 10 times higher in women with obesity than in women without obesity.<sup>7</sup> Obesity can affect the periodontal tissue because the adipose tissue is a large reservoir of biologically active mediators, such as tumor necrosis factor  $\alpha$  and other adipokines. Adipokines such leptin, resistin, and adiponectin are known to be involved in inflammatory processes.

Periodontal diseases are infections of the structures around the teeth: the gingiva, cementum, periodontal ligament, and alveolar

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bone.<sup>8</sup> Periodontal disease is caused by the bacteria in the dental plaque. Plaque is a thin layer formed by microorganisms around the teeth. Right after the teeth are brushed, a thin layer of microorganisms surrounds the surface of the enamel. At first, this thin layer does not contain bacteria, but the bacteria soon start to proliferate, transforming the layer into plaque. The earliest stage of tooth decay, gingivitis, is an infection of the gingiva. If the infection spreads, it affects other structures and can ultimately result in the loss of alveolar bone.<sup>6</sup>

Many factors contribute to the severity of periodontal disease<sup>9,10</sup>, genes (some people are more likely than others to get periodontal disease because of their genetic makeup), smoking (many people who smoke have deeper periodontal pockets and are likely to lose more bone), misalignment of the teeth (malpositioning of the teeth, especially crowding, makes it harder to brush or floss the teeth and enhances the formation of the plaque), bruxism (grinding, clenching, or gnashing, which exert excess force on the teeth and speed up the breakdown of the periodontal ligament and bone), stress hormones (which weaken the immune system), certain medicines (some can cause dry mouth and gingival enlargement), systemic diseases (e.g., diabetes mellitus, human immunodeficiency [HIV] infection, and osteoporosis), nutrition disorders (poor nutrition or overnutrition can affect the immune system).

The main focus of periodontal therapy is the elimination of the cause and other factors that aggravate periodontal disease. Periodontal therapy has four phases.<sup>10</sup> In phase 1, initial therapy, the purpose is to remove the cause and predisposing factors; it includes dental health education, diet modification, scaling and root planning, correction of poor restorations, elimination of caries, antimicrobial therapy, occlusal therapy, minor orthodontic movement, splinting, and temporary prostheses. In phase 2, surgical therapy, the purpose is to control and eliminate the periodontal disease. This phase includes therapy for the periodontal pocket, curettage, gingivectomy, open-flap debridement, resection of necrotic gingiva, and regenerative therapy. Phase 2 also includes the correction of anatomic conditions that can cause periodontal disease or aesthetic correction. Phase 3, reconstructive therapy, includes placement of the final restoration and prosthesis. In phase 4,

maintenance therapy, the purpose is to maintain the health of the oral cavity after therapy. It includes regular control and evaluation of plaque and calculus, depth of the pocket, gingival inflammation, and occlusion.

Periodontal disease thus results from the interaction between the host and microbial factors. Periodontal treatment should focus mainly on microbial factors and host immune response factors. In recent years, the focus on elimination of the microbial factors has expanded to include the host immune response. To maximize the treatment response, inflammation must be reduced, and destructive processes in the periodontal tissues must be inhibited.<sup>11</sup> The association between obesity and other chronic disease has been noted by several researchers, but the studies of obesity and periodontal disease are scarce. The hypothesis of this study is that there is a correlation between obesity and periodontitis status.

## Materials and methods

This study has performed in a private clinic in Bekasi, Indonesia. A total of 36 subjects aged 35 to 55 years participated, of whom 18 were male and 18 female. We measured the body weight (in kilograms) and height (in meters) and then calculated BMI according to the equation presented in the Introduction section.<sup>2</sup> We used the Indonesian modification of the classification of BMI, as described in the Introduction section.

The periodontal status was composed of periodontal pocket depth, gingival recession, and tooth loss. We excluded subjects who were pregnant, those with a history of diabetes mellitus, and those who smoked. Each subject went through a full-mouth periodontal status examination by two calibrated operators.

All subjects agreed to participate in this research and signed written informed consent forms. The study protocol was approved by the Ethical Committee of Faculty of Dentistry, Universitas Indonesia.

## Results

The characteristics of the subjects are presented in Table 1. The mean age of the whole sample was 42.47 years; the mean age of obese subjects was 43 years, and that of nonobese subjects was 41.54 years.

Patient Characteristics	Obese (BMI > 25)	Nonobese (BMI ≤ 25)
No. of subjects	23	13
Age (years)	43 (± 0.53)	41.54 (± 0.93)
Sex		
Male	12 (52%)	6 (47%)
Female	11 (48%)	7 (52%)

**Table 1.** Distribution of obese and nonobese subjects.

BMI, body mass index.

Not all the data about pocket depth, gingival recession, and other factors in the two groups (Table 2) were normally distributed, so we used the Mann-Whitney *U* test to determine the correlation between obesity and periodontal status (depth of pocket, gingival recession, and tooth loss). We found that subjects who were obese exhibited more recession but lesser pocket depth and loss of fewer teeth than did subjects who were not obese. We found no statistically significant relationship between obesity and depth of the pocket, obesity and recession, or obesity and loss of teeth (all *P*s > 0.05).

Variable	<i>N</i>	Mean	Range
Depth of pocket			
Obese subjects	23	1.197 mm	3–8 mm
Nonobese subjects	13	2.27 mm	3–10 mm
Gingival recession			
Obese subjects	23	2.21 mm	1–5 mm
Nonobese subjects	13	1.68 mm	1–7 mm
Loss of teeth			
Obese subjects	23	1.86 teeth	1–7 teeth
Nonobese subjects	13	2.89 teeth	1–7 teeth

**Table 2.** Distribution of periodontal variables among obese and nonobese subjects.

### Discussion

The possible relationship between obesity and periodontal disease was first reported in 1997 by Perlstein et al., who found alveolar bone resorption to be greater in obese rats than in nonobese rats. A more recent 4-year follow-up of

396 Finnish adults revealed no significant association between BMI and severe periodontal disease.<sup>12</sup>

Khader et al. showed that the incidence of periodontitis among individuals with excess weight was double that among individuals of normal weight, and the incidence among individuals with severe obesity was triple that among individuals of normal weight.<sup>2</sup>

One limitation of this study was that we measured only the BMIs of the subjects to determine whether they were obese or nonobese. Several studies have indicated that the amount of abdominal fat (central adiposity) is more strongly associated with periodontitis than is subcutaneous fat. Thus it is important to account for other measurements of obesity, such as waist circumference. Many researchers suggest that waist circumference is related to obesity-related health risk.

We also must consider that periodontal disease has multiple causes and results from the interaction between the host and microbial factors. Other factors such as age, plaque index, and oral trauma can affect severity of periodontitis. Only 36 subjects participated in this study, and this small number might have influenced the normality of the data and the outcomes of the statistical analysis. The timing of the measurement might be another possible cause of the insignificance of the differences. In addition, we defined obesity according to a standard of BMI that was modified for use in Indonesia.

### Conclusions

To minimize the influence of systemic diseases (such as diabetes mellitus) on periodontal status, we excluded patients with systemic diseases from this study in order to focus only on subjects' weight. We found no significant relationship between obesity and periodontitis status in patients who did not smoke. The depth of the pocket, gingival recession, and the loss of the teeth were not affected by subjects' weight.

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### Declaration of Interest

The authors report no conflict of interest.

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