Dental Research Journal

Original Article

The impact of obesity on the outcome of periodontal disease treatment: Systematic review and meta-analysis

Klenise Paranhos[1], Simone Oliveira[2], Rafael Bonato[3], Neda Niknami[1], Shalin Vinayak[4], Peter Loomer[5]

[1]Department of Cariology and Comprehensive Care, New York University College of Dentistry, New York, NY, [5]Dental Science Department at UT Health San Antonio, San Antonio, Texas, USA, [2]Fluminense Federal University, [3]School of Dentistry, Rio de Janeiro, Brazil, [4]Smile Africa, Nairobi, Kenya

ABSTRACT

Background: Obesity and periodontitis are two commonly occurring disorders that affect a considerable amount of the world's population. Several studies have mentioned that there may be a link between the two. The purpose of this systematic review was to determine whether there was a difference in response to nonsurgical periodontal therapies (NSPTs) between obese and nonobese individuals.

Materials and Methods: An online search was assembled with a combination of Medical Subject Headings terms and free-text words of the literature published up to December 2020, to identify interventional studies limited to an adult human population. Titles, abstracts, and finally full texts were scrutinized for possible inclusion by two independent investigators. Reduction in periodontal pocket depth was the primary parameter used to assess the outcome of NSPT.

Results: The primary search yielded 639 significant titles and abstracts. After filtering, data extraction, and quality assessment, 34 full-text studies were selected. All studies matching inclusion criteria, suggest a positive association between obesity and periodontal disease.

Conclusion: Although a possible correlation exists between periodontitis and obesity, as with other oral-systemic disease implications, some controversy exists. While some studies have reported a distinct correlation between periodontitis and obesity, other papers have suggested only moderate or no association between the two conditions at all. These results advise of a difference between response to NSPT amid obese and nonobese individuals. However, with few quality studies and variable reported findings, there is limited evidence of any significant difference in clinical practice. However, it can be a positive warning that obesity is a risk factor toward the outcome of periodontal disease treatment.

Key Words: Overweight, periodontitis, scaling and root planing

Received: 31-Mar-2022
Revised: 25-Jul-2022
Accepted: 31-Jul-2023
Published: 26-Oct-2023

Address for correspondence:
Dr. Neda Niknami,
345, 24th Street, New York, NY 10010, USA.
E-mail: nn1130@nyu.edu

INTRODUCTION

Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health and as a health-care provider we must inform patients about oral health and specific periodontal health concerns.[1,2] The prevalence of obesity is increasing worldwide and the number of obese individuals has more than doubled since 1980.[3] In 2014, more than

Access this article online

Website: www.drj.ir
www.drjjournal.net
www.ncbi.nlm.nih.gov/pmc/journals/1480

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: WKHLRPMedknow_reprints@wolterskluwer.com

How to cite this article: Paranhos K, Oliveira S, Bonato R, Niknami N, Vinayak S, Loomer P. The impact of obesity on the outcome of periodontal disease treatment: Systematic review and meta-analysis. Dent Res J 2023;20:108.

1.9 billion adults were overweight. Of these, over 600 million were obese. For adults, WHO (World Health Organization) calculates BMI by dividing the person's weight in kilograms by the square of his height in meters (kg/m^2). Based on this calculation, BMI greater than or equal to 25 is categorized as overweight and a BMI greater than or equal to 30[2-4] is categorized as obesity.

Adipose tissue has been thoroughly studied in the recent years, and contrary to the assumption of it solely being a fat storage medium, it has been identified as a tissue responsible for metabolism and energy conversion.[5] This endocrine tissue is also associated with significant activity related to chemical substance secretion and inflammatory responses, much like an endocrine organ. Studies have also identified other organs besides adipose tissue which act as storage media of fat, such as the liver, and this could have important implications in measuring total body fat and composition, as these are means of determining weight imbalances and obesity.[6] By realizing the fact that obesity and weight imbalances stem from mechanisms which render energy intake and energy expenditure imbalances, many world health agencies have regarded obesity as a chronic disease.[7]

Obesity has several negative implications on a person's systemic condition including hypertension, type II diabetes, coronary heart disease, stroke, osteoarthritis, and its presence significantly lowers a person's life expectancy.[8] Several studies have shown that it is also linked to reduced periodontal health.[9] The exact mechanism by which obesity predisposes to periodontitis is not fully understood; however, obese individuals have higher levels of circulating tumor necrosis factor-α (TNF-α) and interleukin (IL)-6, secreted by the adipose tissue, compared to nonobese individuals, and these have been implicated in the pathogenesis of periodontal disease.[10-12]

Nonsurgical periodontal therapy (NSPT) is the first step in the clinical management of chronic periodontitis.[7-9] It involves the mechanical removal of bacterial biofilm and deposits with scaling and root planing (SRP), creating a local environment favorable to better periodontal health. The efficacy of NSPT in the management of periodontitis is established, and clinical trials have shown a reduction of inflammation, pocket depth reduction, and clinical attachment gains following therapy. However, it is not clear if obesity affects the outcome of nonsurgical periodontal therapy.[10-13]

The purpose of this systematic review was to determine whether there was a difference in response to NSPT between obese and nonobese individuals.

MATERIALS AND METHODS

The literature search was structured according to the Population, Interventions, Comparisons, and Outcomes format [Table 1]. The population was composed of health subjects (>18 years old) obese and nonobese. The intervention was nonsurgical periodontal therapy. The comparator/control was nonobese subjects with nonsurgical periodontal therapy. The outcome is the response to NSPT between obese and nonobese individuals.

A search was conducted in the Medline, Embase, and Scopus databases using the keywords periodontal disease/periodontitis, overweight/obesity/obese, and treatment/outcome of therapy. Results in English language only up to December 31, 2020, were included. Results from all databases were combined and duplicates were removed. Studies were then subsequently excluded based on title, abstract, and finally full-text review for a final inclusion of 8 studies [Figure 1].

Inclusion criteria

1. Interventional studies assessing the effects of periodontal therapy on obese individuals and/or comparing the effects of periodontal therapy on obese and nonobese subjects
2. Minimum age 18 years
3. Obesity/overweight assessment (e.g., body mass index, waist/hip ratio, and waist circumference)
4. Diagnosis of periodontal disease
5. Subjects underwent periodontal therapy nonsurgical periodontal therapy: supra, subgingival SRP.

Table 1: Population, Interventions, Comparisons, and Outcomes

PICO	Population, Interventions, Comparisons, and Outcomes
P: Population	Health subjects (>18 years old) obese and nonobese
I: Intervention	Nonsurgical periodontal therapy
C: Comparisons/ Control	Nonobese subjects with nonsurgical periodontal therapy
O: Outcome	Response to nonsurgical periodontal therapy between obese and nonobese individuals

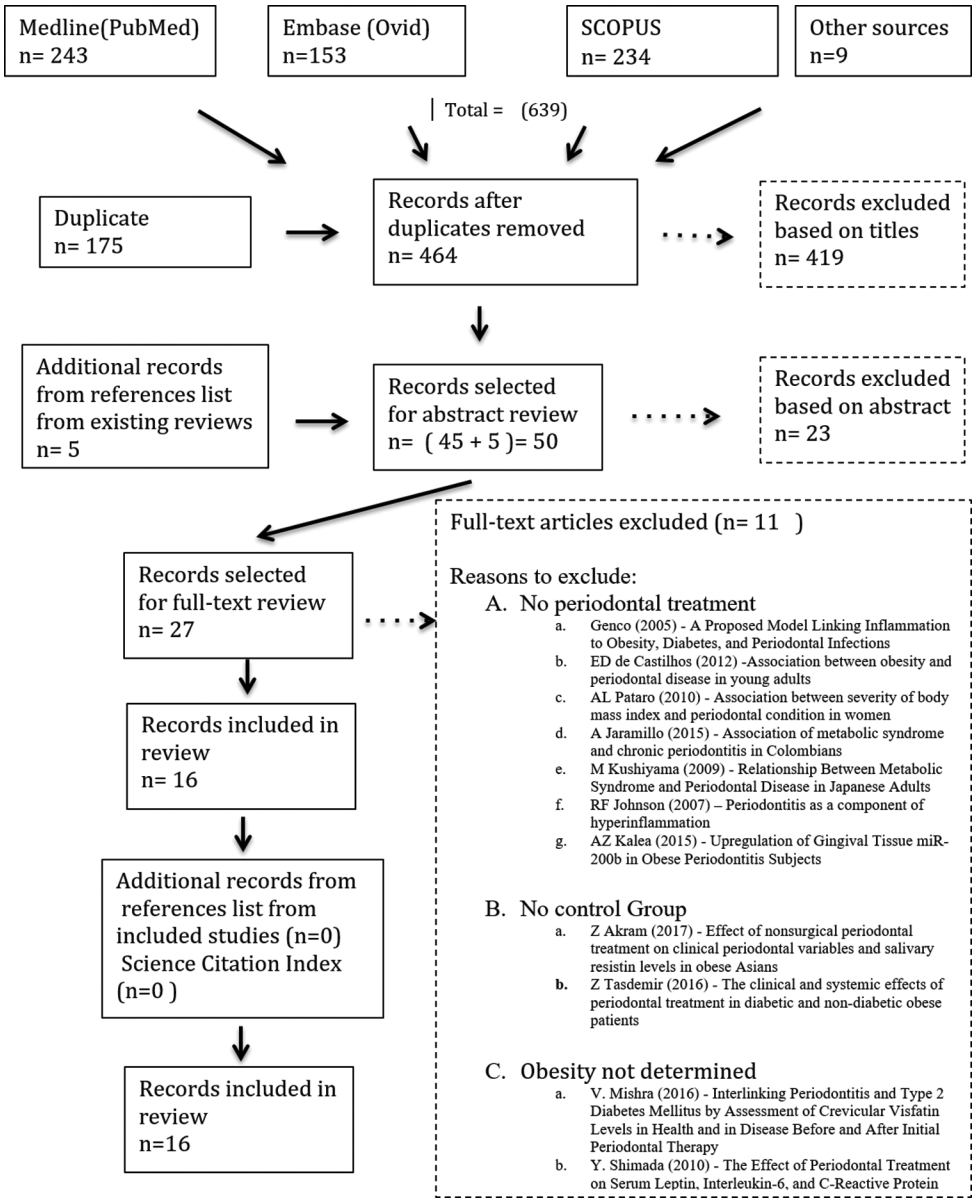

Figure 1: Search strategy studies.

Exclusion criteria

1. Uncontrolled or poorly controlled systemic diseases
2. Observational studies, case–control, cross-sectional studies, longitudinal retrospective studies, animal studies, and *in vitro* studies
3. Letters to the editor, reviews, and conference abstracts.

Analysis and forest plots were created with Open Meta (analyst). A random effects model was employed with 95% confidence intervals. For baseline analyses, group means were summarized by subtracting the BMI+ (overweight and obese) mean from BMI− (normal) mean. For the analysis of change between baseline and 1st follow-up, change data were computed by subtracting the follow-up mean from the baseline mean so that higher scores indicate more improvement, such as greater reduction in pocket depth and clinical attachment loss (CAL). To compare groups on levels of improvement, the mean summary effect was then computed by subtracting the BMI+ group score from the BMI− group scores so that higher scores indicate greater treatment effects in the control group [Figures 2 and 3].

After a systematic review,[10,11,14-27] 8 articles, published between 2012 and 2020, were selected for the meta-analysis.[14-24] The other 9 had insufficient data for inclusion. To estimate the mean of probing depth

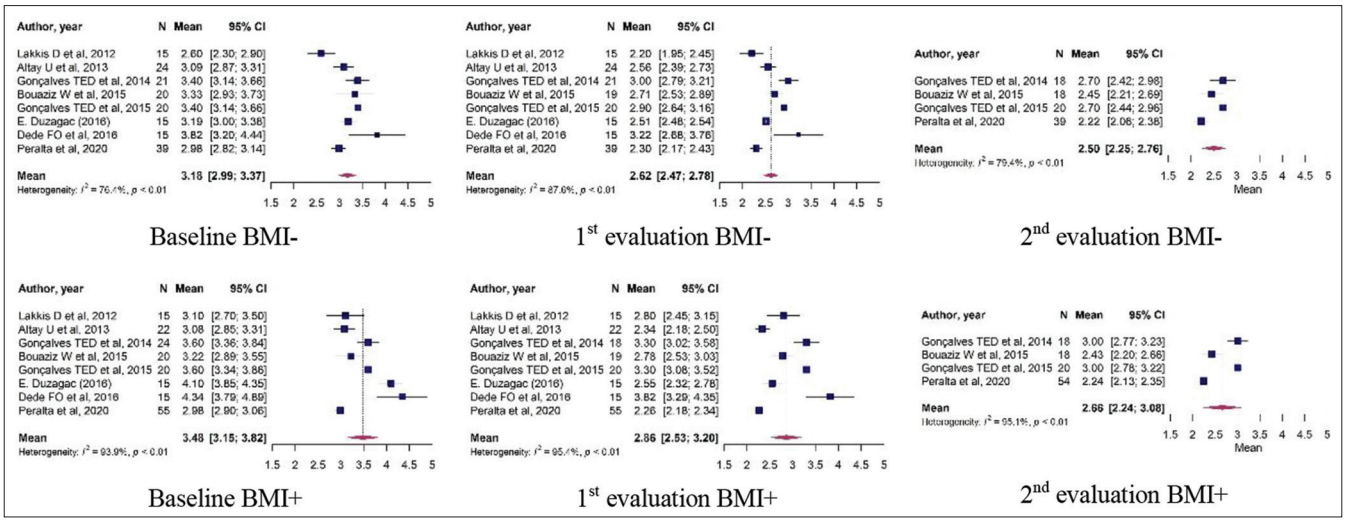

Figure 2: Forrest plot for estimating the mean of each group and evaluation time – Probing Depth BMI: Body mass index; CI: Confidence interval.

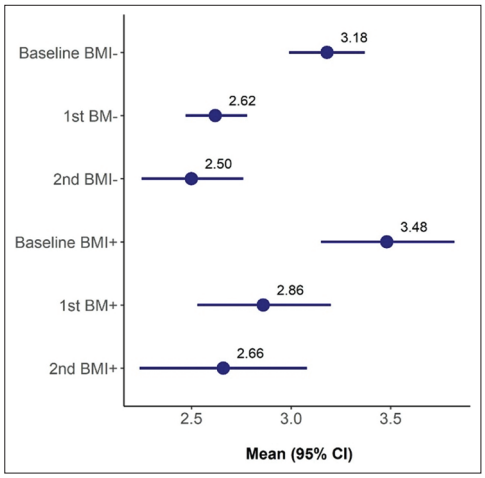

Figure 3: Mean and 95% confidence interval, estimated by meta-analysis using the restricted maximum likelihood – Probing Depth method. BMI: Body mass index; CI: Confidence interval.

and clinical attainment loss (CAL), random models were used using the restricted maximum likelihood method.

To identify heterogeneity between studies, the Q test was used, and to quantify heterogeneity, the I^2 statistic was used. This statistic estimates the proportion of heterogeneity observed in the studies, which can vary from 0% to 100%, the higher the value, the greater the difference between the selected studies.

The presence of outliers was evaluated through externally standardized residuals. Outlier studies are not necessarily influential, that is, they significantly modify the parameter estimate. To detect influential studies, the leave-one-out technique was used. None of the studies presented itself as influential.

It was not possible to perform a bias analysis; for this analysis, at least 10 articles are required.

The significance level adopted was 5%.

RESULTS

The selected articles present different patient follow-up times, all eight articles present an evaluation after the baseline, four articles present a second evaluation, and only two articles present a third evaluation. As there were only two articles with the third evaluation, this result was not considered in this report.

The forest plot [Figures 2 and 4, 5] is a graph that allows the visualization of the estimated measures and their confidence intervals. Each study is plotted on the graph and its representation has two elements, a box that represents the estimate for each study and a horizontal line, which represents the confidence interval for that estimate. Small horizontal lines indicate better accuracies of the study results.

Figure 2 is a summary of the graphs in Figure 1, and it presents the means and 95% confidence intervals for each time and group, a form of descriptive comparison between times and groups.

Table 1 presents the result of the meta-regression that compares the times and groups for PD. This table presents two models, one considering only the baseline and 1st evaluation (8 articles) and another considering the baseline, 1st evaluation, and 2nd evaluation (4 articles). In both models, the interaction between group and time was tested and

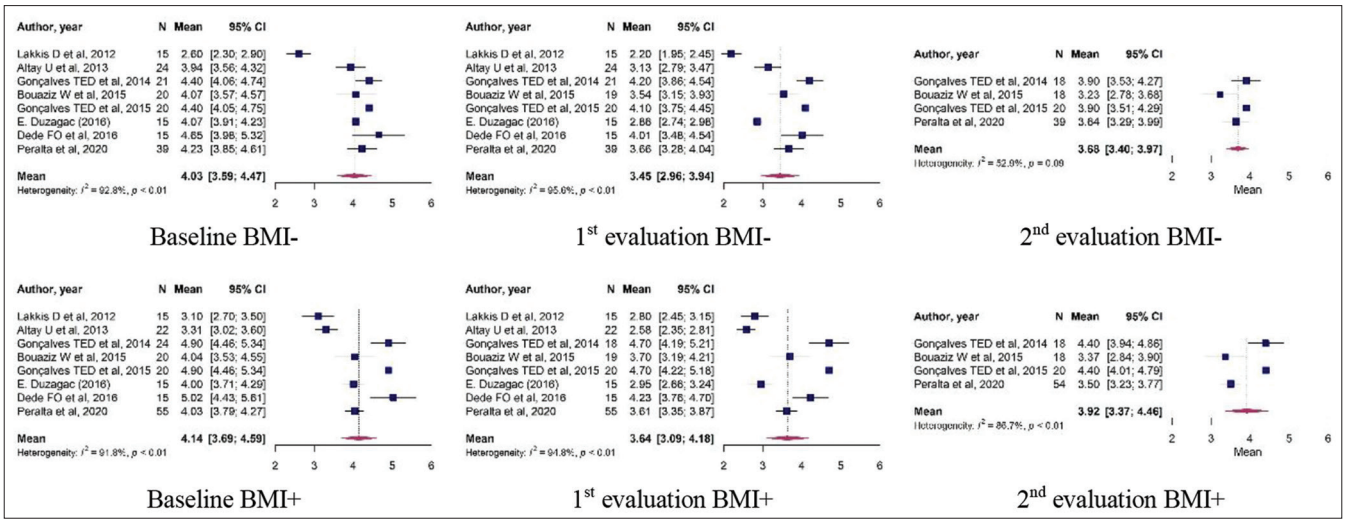

Figure 4: Forrest plot for estimating the mean of each group and evaluation time – Clinical Attachment Loss. BMI: Body mass index; CI: Confidence interval.

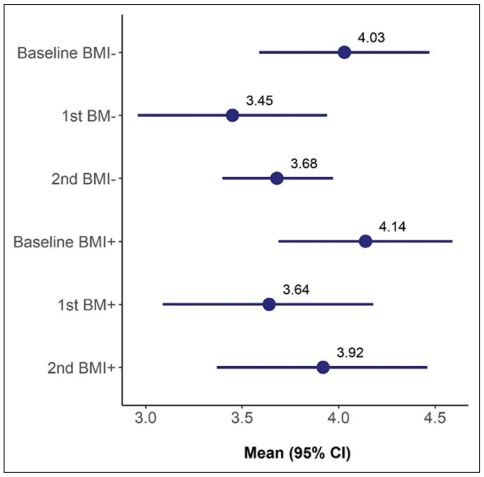

Figure 5: Mean and 95% confidence interval, estimated by meta-analysis using the restricted maximum likelihood – Clinical Attachment Loss method. BMI: Body mass index; CI: Confidence interval.

Table 2: Meta-regression results – pocket depth

Model	Variable	Estimation	SE	*P*
Baseline e 1st	BMI+	0.2661	0.1490	0.0849
	1st	−0.6141	0.1490	0.0003
Baseline, 1st e 2nd	BMI+	0.2398	0.1260	0.0653
	1st	−0.6137	0.1420	0.0001
	2nd	−0.7498	0.1695	<0.0001

BMI: Body mass index; SE: Standard error

Table 3: Meta-regression results - clinical attainment loss

Model	Variable	Estimation	SE	*P*
Baseline e 1st	BMI+	0.1846	0.2583	0.4808
	1st	−0.5827	0.2583	0.0321
Baseline, 1st e 2nd	BMI+	0.1962	0.2147	0.3671
	1st	−0.5837	0.2406	0.0205
	2nd	−0.2920	0.2912	0.3229

BMI: Body mass index; SE: Standard error

was not significant. In the first model [Table 2], it is noted that there is no significant difference between the groups ($P = 0.0849$) and that the 1st evaluation is significantly lower than the baseline ($P = 0.0003$). In the second model, it is noted that there is no significant difference between the groups ($P = 0.0653$) and that the 1st evaluation ($P = 0.0001$) and 2nd evaluation ($P < 0.0001$) are significantly lower than the baseline [Table 3]. Considering the significance level of 5%, it is concluded that the groups are not different, and both reduce the mean PD over time.

Table 2 presents the result of the meta-regression that compares the times and groups for CAL. This table presents two models, one considering only the baseline and 1st evaluation (8 articles) and another considering the baseline, 1st evaluation, and 2nd evaluation (4 articles). In both models, the interaction between group and time was tested and was not significant. In the first model, it is noted that there is no significant difference between the groups ($P = 0.4808$) and that the 1st evaluation is significantly lower than the baseline ($P = 0.0321$). In the second model, it is noted that there is no significant difference between the groups ($P = 0.3671$) and that the 1st evaluation ($P = 0.0205$) is significantly lower than the baseline; no significant difference was observed between baseline and 2nd evaluation ($P = 0.3229$). Considering the significance level of 5%, it is concluded that the groups are not different, and

both reduce the mean CAL from the baseline to the 1st evaluation.[28,29]

DISCUSSION

Despite decades of research and treatment, periodontitis remains one of the most common inflammatory conditions in humans. Often associated with systemic diseases such as cardiovascular disease and diabetes mellitus, there is mounting evidence that obesity may play a role in the immune response related to periodontal disease.[3,9-14] Considering that obesity affected 39.8% of adults in the U. S. in 2015–2016 (CDC NCHS data brief, No. 288, October 2017), the global health implications of periodontitis in obese individuals could be significant.[4,7]

The relationship between periodontitis and obesity has been reported by several studies focusing on periodontal pathogens dissemination from epithelial through the circulation and consequently the immune system.[11]

Obesity as a chronic disease has been reported by recent studies to be associated with changes in many inflammatory mediators such as IL-6, TNF-α, monocyte chemoattractant protein-1 (MCP-1), and IL receptor agonists. Obesity has also been shown to be linked to changes in some adipose-related proteins such as leptin, adiponectin, resistin, and visfatin, suggesting systemic and immunological mechanisms by which obesity may be associated with other inflammatory or immunological-related disorders.[26,30-33]

Furthermore, it has been shown by experimental studies done on obese rats that periodontal disease markers, such as alveolar bone loss and periodontal inflammatory mediators linked to periodontitis, are significantly higher in these rats.[31] These results point toward a possible correlation between periodontitis and obesity.

Although a possible correlation exists between periodontitis and obesity, as with other oral-systemic disease implications, some controversy exists. While some studies have reported a distinct correlation between periodontitis and obesity, other papers have suggested only moderate or no association between the two conditions at all.

CONCLUSION

These results advise of a difference between response to NSPT in obese and nonobese individuals. However, with few quality studies and variable reported findings, there is limited evidence of any significant difference in clinical practice. However, it can serve as a positive warning that obesity is a risk factor toward the outcome of periodontal disease treatment.

Financial support and sponsorship
Nil.

Conflicts of interest
The authors of this manuscript declare that they have no conflicts of interest, real or perceived, financial or non-financial in this article.

REFERENCES

1. Natto ZS, Hameedaddin A. Methodological quality assessment of meta-analyses and systematic reviews of the relationship between periodontal and systemic diseases. J Evid Based Dent Pract 2019;19:131-9.
2. World Health Organization (WHO). Obesity and Overweight. Fact Sheet. June; 2016. Available from: https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight. [Last accessed on 2020 Mar 16].
3. Danaei G, Finucane MM, Lin JK, Singh GM, Paciorek CJ, Cowan MJ, *et al.* National, regional, and global trends in systolic blood pressure since 1980: Systematic analysis of health examination surveys and epidemiological studies with 786 country-years and 5·4 million participants. Lancet 2011;377:568-77.
4. WHO Consultation on Obesity. Obesity: Preventing and Managing the Global Epidemic: Report of a Who Consultation. Geneva, Switzerland: World Health Organization; 2000. Available from: https://apps.who.int/iris/handle/10665/42330. [Last accessed on 2020 Mar 18].
5. Bray GA. Obesity is a chronic, relapsing neurochemical disease. Int J Obes Relat Metab Disord 2004;28:34-8.
6. Lyon CJ, Law RE, Hsueh WA. Minireview: Adiposity, inflammation, and atherogenesis. Endocrinology 2003;144:2195-200.
7. World Health Organization. Regional Office for Europe. Nutrition, Overweight and Obesity: Factsheet on Sustainable Development Goals (SDGs): Health Targets. World Health Organization. Regional Office for Europe; 2021. Available from: https://apps.who.int/iris/handle/10665/341982. [Last accessed on 2020 Feb 20].
8. Kim S, Popkin BM. Commentary: Understanding the epidemiology of overweight and obesity – A real global public health concern. Int J Epidemiol 2006;35:60-7.
9. Perlstein MI, Bissada NF. Influence of obesity and hypertension on the severity of periodontitis in rats. Oral Surg Oral Med Oral Pathol 1977;43:707-19.
10. Zuza EP, Barroso EM, Carrareto AL, Pires JR, Carlos IZ, Theodoro LH, *et al.* The role of obesity as a modifying factor in patients undergoing non-surgical periodontal therapy. J Periodontol 2011;82:676-82.
11. Suvan J, D'Aiuto F, Moles DR, Petrie A, Donos N. Association between overweight/obesity and periodontitis in adults. A systematic review. Obes Rev 2011;12:e381-404.

12. Akram Z, Safii SH, Vaithilingam RD, Baharuddin NA, Javed F, Vohra F. Efficacy of non-surgical periodontal therapy in the management of chronic periodontitis among obese and non-obese patients: A systematic review and meta-analysis. Clin Oral Investig 2016;20:903-14.
13. Gerber FA, Sahrmann P, Schmidlin OA, Heumann C, Beer JH, Schmidlin PR. Influence of obesity on the outcome of non-surgical periodontal therapy – A systematic review. BMC Oral Health 2016;16:90.
14. Acharya A, Bhavsar N, Jadav B, Parikh H. Cardioprotective effect of periodontal therapy in metabolic syndrome: A pilot study in Indian subjects. Metab Syndr Relat Disord 2010;8:335-41.
15. Al-Zahrani MS, Alghamdi HS. Effect of periodontal treatment on serum C-reactive protein level in obese and normal-weight women affected with chronic periodontitis. Saudi Med J 2012;33:309-14.
16. Lakkis D, Bissada NF, Saber A, Khaitan L, Palomo L, Narendran S, *et al.* Response to periodontal therapy in patients who had weight loss after bariatric surgery and obese counterparts: A pilot study. J Periodontol 2012;83:684-9.
17. Altay U, Gürgan CA, Ağbaht K. Changes in inflammatory and metabolic parameters after periodontal treatment in patients with and without obesity. J Periodontol 2013;84:13-23.
18. Eldin AM, Nasr SA, Hassan NE. Effect of non-surgical periodontal therapy on interleukin-8 (il-8) level in gingival crevicular fluid in overweight and obese subjects with chronic periodontitis. World J Med Sci 2013;9:173-9.
19. Suvan J, Petrie A, Moles DR, Nibali L, Patel K, Darbar U, *et al.* Body mass index as a predictive factor of periodontal therapy outcomes. J Dent Res 2014;93:49-54.
20. Bouaziz W, Davideau JL, Tenenbaum H, Huck O. Adiposity measurements and non-surgical periodontal therapy outcomes. J Periodontol 2015;86:1030-7.
21. Gonçalves TE, Zimmermann GS, Figueiredo LC, Souza Mde C, da Cruz DF, Bastos MF, *et al.* Local and serum levels of adipokines in patients with obesity after periodontal therapy: One-year follow-up. J Clin Periodontol 2015;42:431-9.
22. Duzagac E, Cifcibasi E, Erdem MG, Karabey V, Kasali K, Badur S, *et al.* Is obesity associated with healing after non-surgical periodontal therapy? A local versus systemic evaluation. J Periodontal Res 2016;51:604-12.
23. Öngöz Dede F, Bozkurt Doğan Ş, Ballı U, Avcı B, Durmuşlar MC. The effect of initial periodontal treatment on plasma, gingival crevicular fluid and salivary levels of 8-hydroxy-deoxyguanosine in obesity. Arch Oral Biol 2016;62:80-5.
24. Balli U, Ongoz Dede F, Bozkurt Dogan S, Gulsoy Z, Sertoglu E. Chemerin and interleukin-6 levels in obese individuals following periodontal treatment. Oral Dis 2016;22:673-80.
25. Zuza EP, Barroso EM, Fabricio M, Carrareto AL, Toledo BE, Pires JR. Lipid profile and high-sensitivity C-reactive protein levels in obese and non-obese subjects undergoing non-surgical periodontal therapy. J Oral Sci 2016;58:423-30.
26. Shimada Y, Komatsu Y, Ikezawa-Suzuki I, Tai H, Sugita N, Yoshie H. The effect of periodontal treatment on serum leptin, interleukin-6, and C-reactive protein. J Periodontol 2010;81:1118-23.
27. Sripradha S. To study the impact of nonsurgical periodontal therapy on BMI and hip-waist ratio in obese patients with and without periodontitis. Res J Pharm Technol 2016;9:1704-6.
28. Core Team R. A: A Language and Environment for Statistical Computing. Foundation for Statistical Computing, Vienna, Austria; 2021. Available from: https://www.R-project.org/. [Last accessed on 2020 Mar 29].
29. Sutton AJ, Abrams KR, Jones DR, Sheldon TA, Song F. Methods for Meta-Analysis in Medical Research. Chichester: John Wiley & Sons, Ltd; 2000.
30. Fantuzzi G. Adipose tissue, adipokines, and inflammation. J Allergy Clin Immunol 2005;115:911-9.
31. Zuza EP, Garcia VG, Theodoro LH, Ervolino E, Favero LF, Longo M, *et al.* Influence of obesity on experimental periodontitis in rats: Histopathological, histometric and immunohistochemical study. Clin Oral Investig 2018;22:1197-208.
32. Peralta FD, Cortelli SC, Rovai ES, Aquino DR, Miranda TB, Costa FO, *et al.* Clinical and microbiological evaluation of non-surgical periodontal therapy in obese and non-obese individuals with periodontitis: A 9-month prospective longitudinal study. J Appl Oral Sci 2020;28:e20190694.
33. Gonçalves TE, Feres M, Zimmermann GS, Faveri M, Figueiredo LC, Braga PG, *et al.* Effects of scaling and root planing on clinical response and serum levels of adipocytokines in patients with obesity and chronic periodontitis. J Periodontol 2015;86:53-61.