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Original Article

An 8-Year Cohort Study of Obesity and Morbidity of Periodontal Disease in Large Factory Workers

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Abstract

Purpose: To demonstrate that obesity is an independent risk factor for periodontal diseases, a cohort study was conducted on the employees of a large firm.

Methods: The subjects were about 2,000 employees working for a large firm. With BMI and CPI as indices, cohorts I and II were followed up for 4 and 8 years, respectively.

Results and discussion: In both cohorts I and II, the groups with high CPI levels were found to exhibit a high mean BMI. In the same cohorts, the percentage of obese individuals was noted to be higher with statistical significance in the CPI 4 group in comparison with the CPI 0, 1, 2 and 3 groups. In cohort II, the probability of contracting periodontal diseases among obese individuals in the non-smoking group was 2.85 times greater than that of those with a normal body weight (95% confidence interval: 1.061 − 7.709). The same probability was 2.41 times greater among smokers (95% CI: 1.136 − 5.119). It was epidemiologically indicated that obese subjects, when compared to those of normal body weight, have a higher probability to develop periodontal diseases, establishing that obesity is an independent risk factor for periodontal diseases.

Key words : Cohort Study, Obesity, Morbidity of Periodontal Disease, Large Factory Workers, Community Periodontal Index

Ⅰ. Introduction

Obesity is a risk factor for various life-style-related diseases such as ischemic heart disease, diabetes mellitus, cerebrovascular disorders and hypertension [1,2]. It was reported recently that obesity is also a factor responsible for the development of periodontal diseases, which are known to cause one to lose his teeth and in turn affect systemic diseases, such as diabetes mellitus, ischemic heart diseases, and cerebrovascular disorders. Thus these have been cited in "Healthy Japan 21" as an important disease category to be prevented [3-5]. To prevent periodontal diseases, many studies have been conducted on the etiological factors. Smoking habits, aging, diabetes mellitus, and the subgingival presence of bacteria pathogenic to periodontal diseases have been incriminated [6,7] Recently, obesity was added to this growing list of risk factors for periodontal diseases [8].

Recently, studies have indicated that obesity is not a simple phenomenon in which excess energy is stored in the adipose tissue [9-11] : instead, it was recognized that the adipose tissue is important in that it is composed of adipocytes, which manufacture and secrete various physiologically active substances, such as leptin, tumor necrosis factor-α (TNF-α), free fatty acids, PAI-1, adipsin and resistin [12]. Among these, the importance of TNF-α has been singled out for its action to promote bone absorption [13], in particular for the possibility of acting as the cause for periodontal diseases that are characterized by the absorption of the alveolar bone [14].

The mechanism involving obesity and the development of periodontal diseases has been elaborated mainly in animal experiments [15] ; but studies on the relationship between these two in man − employing highly reliable immunological

techniques − are scarce. Most are cross-sectional studies using the body mass index (BMI), body weight and waist-hip ratio as evaluation indices [16], while only a few were epidemiological studies with research designs suitable to prove a cause-effect relationship. Thus the current 8-year prospective cohort study was conducted on employees of a large business establishment to prove that obesity does indeed constitute a true independent risk factor for periodontal diseases.

Ⅱ. Subject and Method

A cohort study on the incidences of periodontal diseases using obesity and the community periodontal index (CPI) as indices was conducted on employees of a large business concern. The study was composed of cohort I (a follow-up lasting 4 years) and cohort II lasting 8 years.

1. Subjects

The subjects of the present survey were approximately 2,000 employees of a chemical factory located in Osaka-Fu. They included those assigned to the production line on 3 shifts. Most were involved in operating machinery through the use of computers: none was involved in potentially risky operations (such as exposure to toxic substances).

To remove the effects of complex factors related to the development of periodontal diseases, these subjects were limited at the start of the survey to men between 20 and 59 years who met the following conditions ① through ③ during the duration of Cohort I or II.

① Those who responded to a list of questions concerning their life style;

② Those who were judged by physicians to be free of diseases (such as diabetes mellitus and cerebrovascualr disorders) that may affect the performance of their work;

③ Those who had examinations for periodontal diseases.

2. Method for conducting the survey on the subjects

The subjects (who had fasted on the day of the examination) reported to a room designated for examination within the company, submitted to a blood specimen collection, a standard health examination and for periodontal diseases, and responded to a survey on their life style (such as smoking habits). A self-responding questionnaire format was adopted for the survey on life-style.

3. Evaluation of the extent of obesity and periodontal diseases

1) Extent of obesity

The extent of obesity was determined by using body mass index (BMI), which was computed from "body weight (kg) / body height (m)^2. In accordance with the standards set by WHO and the Japan Society for the Study of Obesity, low body weight, normal weight and obesity were defined as BMI < 18.5, 18.5 ≦ BMI < 25.0 and BMI ≧ 25.0, respectively [17].

2) Examination for periodontal diseases

To examine for periodontal diseases, dentists and dental hygienists reported to the business office and carried out the test as part of a standard physical examination that is conducted by the company. Those in charge of this test first ran preliminary tests repeatedly to set the test standard among the examiners. Simple chairs designed for dental patients were used to stabilize the patient's head and the test was conducted under proper lighting. The procedure required about 5 minutes for each patient. To evaluate periodontal diseases, the Community Periodontal Index (CPI) [18] was applied, using the periodontal probe (by Yamaura, Inc.) specified by WHO and based on the method of Ainamo et al. Specifically, the oral cavity was divided into 6 areas; and by using the periodontal probe, the periphery of the teeth for each area was rated for the presence (or absence) of gingival hemorrhage, dental calculi and gingival pockets. The findings were displayed by using a numerical score. The presence of periodontal diseases was defined as a maximum CPI of 6 areas being 3.0 or more.

4. Outline of the cohort study

The subjects are those who met conditions ① through ③. These patients continued to satisfy the conditions continuously for 4 years in Cohort I and 8 years in Cohort II. The subjects numbered 1,747 in the first year. Cohorts I and II included 1,102 and 379 subjects, respectively.

5. Statistical analysis

Those with low body weight, normal weight and excessive weight were grouped according to their BMI. Using those subjects with normal body weight as the reference, the development of periodontal diseases was analyzed by logistic regression analysis [19]. Furthermore, each group was sorted by CPI and one-way analysis of variance was conducted on BMI.

The Macintosh StatView 5.0 computer program was used as the software for statistical analysis. The significance level was set at $p < 0.05$.

Ⅲ. Results

1. Extent of obesity of the subjects

The BMI distribution at the conclusion of Cohort I is shown in Fig. 1-a. The mean BMI was 22.9 ± 2.7. The percentages for the "thin", "normal" and "obese" people

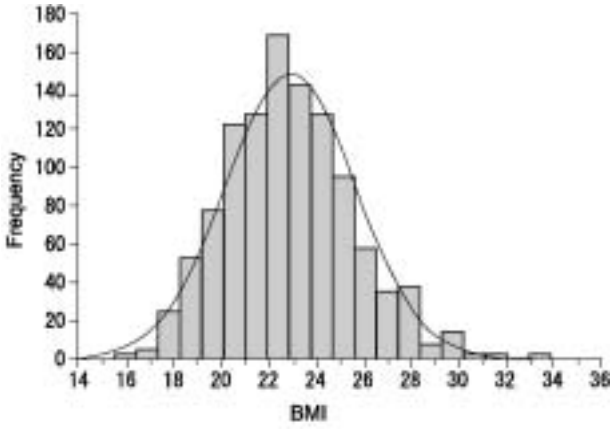

Fig.1-a The BMI distribution at the conclusion of Cohort Ⅰ

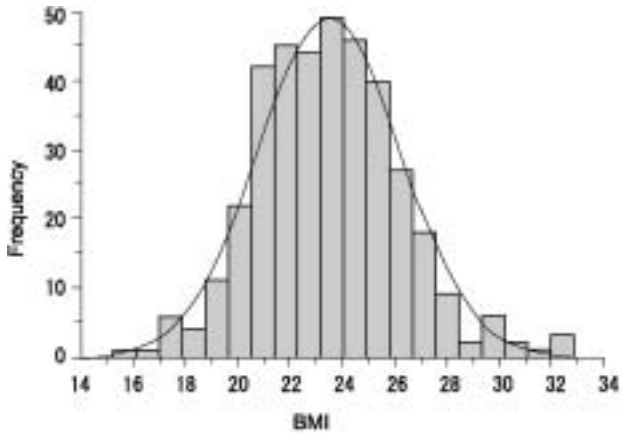

Fig.1-b The BMI distribution at the conclusion of Cohort Ⅱ

were 4.4, 75.3 and 20.3%, respectively. Fig. 1-b shows the distribution of BMI at the conclusion of Cohort II. The mean BMI was 23.5 ± 2.7. The percentages for the "thin", "normal" and "obese" individuals were 2.6, 68.9 and 28.5%, respectively.

2. The BMI stratified by CPI

Tables 1-a and 1-b show the mean BMIs stratified by smoking status and CPI in Cohorts I and II. In Cohort I, both "non-smokers" and "smokers" showed a generally high mean BMI with statistical significance, in descending order of CPI-4, CPI-3, CPI-0, −1 and −2 groups.

In Cohort II, both "smokers" and "non-smokers" showed high mean BMI in the order of CPI-4, CPI-3, CPI-0, -1 and -2. However, no statistically significant difference was noted.

3. Percentages of obese individuals among those with

Table1-a The mean BMI stratified by smoking status and CPI : Cohort Ⅰ

	Nonsmoker			Quit Smoker			Smoker		
Year/CPI	N	Mean ± S.D.	p value	N	Mean ± S.D.	p value	N	Mean ± S.D.	p value
Base line									
CPI 0,1,2	205	22.8 ± 2.7	0.041	45	22.4 ± 2.2	0.005	302	22.2 ± 2.8	0.016
CPI3	113	23.2 ± 2.3		53	24.0 ± 2.6		268	22.6 ± 2.7	
CPI4	32	24.4 ± 2.8		12	23.6 ± 2.0		72	23.3 ± 2.9	
2 nd year									
CPI 0,1,2	205	22.8 ± 2.8	0.003	45	22.7 ± 2.4	0.185	302	22.3 ± 2.9	0.002
CPI3	113	23.0 ± 2.4		53	23.6 ± 2.9		268	22.7 ± 2.6	
CPI4	32	24.5 ± 2.5		12	23.7 ± 2.5		72	23.6 ± 3.0	
3 rd year									
CPI 0,1,2	205	22.8 ± 2.8	0.013	45	22.6 ± 2.3	0.146	302	22.4 ± 2.9	0.016
CPI3	113	23.0 ± 2.4		53	23.5 ± 2.8		268	22.7 ± 2.5	
CPI4	32	24.3 ± 2.6		12	23.9 ± 2.4		72	23.4 ± 2.8	
4 th year									
CPI 0,1,2	205	23.0 ± 2.9	0.057	45	22.8 ± 2.4	0.267	302	22.6 ± 2.8	0.014
CPI3	113	23.1 ± 2.4		53	23.4 ± 2.5		268	22.9 ± 2.6	
CPI4	32	24.3 ± 2.6		12	23.9 ± 2.6		72	23.6 ± 2.9	
5 th year									
CPI 0,1,2	205	22.9 ± 2.8	0.042	45	22.8 ± 2.5	0.389	302	22.6 ± 2.8	0.083
CPI3	113	23.1 ± 2.6		53	23.2 ± 2.6		268	22.7 ± 2.5	
CPI4	32	24.2 ± 2.4		12	23.9 ± 2.3		72	23.4 ± 3.0	

periodontal diseases

Tables 2-a and 2-b show percentages of obese individuals classified by their smoking status and CPI values in Cohorts I and II. In Cohort I, the percentage in the CPI4 group (both smokers and non-smokers) was significantly higher than those of CPI3 and CPI, 1, 2. The means for 4 years for CPI4, CPI3, and CPI, 1, 2 were 32.9%, 20.8% and 17.6%, respectively, showing evident differences between the groups. The trend in Cohort II was similar to that in Cohort I but perhaps due to the small number of subjects, fluctuations were noted in the figures. For both "smokers" and "non-smokers", the percentage of obese individuals in the CPI4 group was higher, with statistical significance in comparison with those for the CPI3 or CPI0, 1, 2 groups. The mean for 8 years was 27.1%, 24.4%, and 16.6% for the CPI4, CPI3 and CPI0, 1, 2 groups. Inter-group differences were evident.

Table1-b The mean BMI stratified by smoking status and CPI in Cohort: Cohort Ⅱ

	Nonsmoker			Quit smoker			Smoker		
Year/CPI	N	Mean ± S.D.	p value	N	Mean ± S.D.	p value	N	Mean ± S.D.	p value
Base line									
CPI 0,1,2	72	22.6 ± 2.1	0.113	13	23.5 ± 2.9	0.949	126	22.3 ± 2.6	0.697
CPI3	42	23.1 ± 2.6		17	23.2 ± 2.0		75	22.6 ± 2.6	
CPI4	4	24.9 ± 1.9		5	23.5 ± 2.0		25	22.7 ± 3.0	
2 nd year									
CPI 0,1,2	72	22.5 ± 2.3	0.067	13	22.7 ± 2.8	0.966	126	22.3 ± 2.6	0.474
CPI3	42	23.0 ± 2.9		17	22.9 ± 2.7		75	22.6 ± 2.6	
CPI4	4	25.3 ± 1.4		5	23.1 ± 2.7		25	22.9 ± 3.0	
3 rd year									
CPI 0,1,2	72	22.5 ± 2.4	0.164	13	22.5 ± 2.6	0.868	126	22.4 ± 2.5	0.533
CPI3	42	23.0 ± 2.8		17	22.9 ± 2.5		75	22.6 ± 2.6	
CPI4	4	24.7 ± 1.4		5	23.1 ± 2.3		25	23.0 ± 3.0	
4 th year									
CPI 0,1,2	72	22.6 ± 2.5	0.515	13	22.7 ± 2.7	0.865	126	22.6 ± 2.5	0.661
CPI3	42	23.0 ± 2.7		17	23.2 ± 2.9		75	22.8 ± 2.6	
CPI4	4	23.9 ± 0.7		5	22.9 ± 2.2		25	23.1 ± 3.1	
5 th year									
CPI 0,1,2	72	22.6 ± 2.5	0.491	13	22.8 ± 2.7	0.972	126	22.5 ± 2.4	0.491
CPI3	42	22.8 ± 2.7		17	23.0 ± 2.8		75	22.8 ± 2.5	
CPI4	4	24.0 ± 0.7		5	23.1 ± 2.6		25	23.0 ± 3.0	
6 th year									
CPI 0,1,2	72	23.0 ± 2.6	0.451	13	23.4 ± 2.9	0.947	126	23.1 ± 2.6	0.740
CPI3	42	23.4 ± 2.7		17	23.7 ± 2.9		75	23.3 ± 2.5	
CPI4	4	24.4 ± 1.1		5	23.9 ± 2.7		25	23.6 ± 3.0	
7 th year									
CPI 0,1,2	72	23.0 ± 2.5	0.311	13	23.4 ± 2.9	0.946	126	23.2 ± 2.6	0.84
CPI3	42	23.5 ± 2.8		17	23.6 ± 3.0		75	23.4 ± 2.5	
CPI4	4	24.7 ± 0.4		5	23.2 ± 3.3		25	23.4 ± 3.3	
8 th year									
CPI 0,1,2	72	23.0 ± 2.6	0.291	13	23.4 ± 2.8	0.961	126	23.3 ± 2.7	0.962
CPI3	42	23.4 ± 3.0		17	23.7 ± 2.8		75	23.3 ± 2.4	
CPI4	4	25.0 ± 0.7		5	23.7 ± 3.0		25	23.4 ± 3.0	
9 th year									
CPI 0,1,2	72	23.2 ± 2.6	0.191	13	23.8 ± 3.0	0.952	126	23.5 ± 2.7	0.741
CPI3	42	23.7 ± 3.2		17	23.9 ± 2.7		75	23.3 ± 2.6	
CPI4	4	25.6 ± 0.8		5	23.5 ± 3.4		25	23.6 ± 2.9	

Table 2-a Percentages of obese individuals classified by their smoking status and CPI values：Cohort Ⅰ

Smoking status	Base line (%)	2 nd year (%)	3 rd year (%)	4 th year (%)	5 th year (%)
Nonsmoker					
CPI 0,1,2	18.0	19.1	19.1	21.5	18.5
CPI 3	21.2	21.1	19.3	21.1	20.2
CPI 4	37.5	34.4	31.3	34.4	34.4
Quit smoker					
CPI 0,1,2	11.1	20.0	17.8	22.2	22.2
CPI 3	32.1	34.0	34	30.2	30.2
CPI 4	33.3	33.3	33.3	33.3	33.3
Smoker					
CPI 0,1,2	14.6	16.8	16.8	17.5	16.8
CPI 3	16.4	18.4	19	20.5	18.3
CPI 4	26.4	33.3	33.3	37.5	30.6

Table 2-b Percentages of obese individuals classified by their smoking status and CPI values：Cohort Ⅱ

Smoking status	Base line (%)	2 nd year (%)	3 rd year (%)	4 th year (%)	5 th year (%)	6 th yea r(%)	7 th year (%)	8 th year (%)	9 th year (%)
Nonsmoker									
CPI 0,1,2	11.1	9.7	13.9	12.5	13.9	18.1	16.7	20.8	22.2
CPI 3	23.8	26.2	26.2	26.2	21.4	33.3	26.2	28.6	35.7
CPI 4	50	50	50	0	0	50	25	25	75
Quit smoker									
CPI 0,1,2	23.1	23.1	15.4	23.1	30.8	30.8	30.8	30.8	46.2
CPI 3	23.5	29.4	29.4	29.4	29.4	35.3	41.2	41.2	41.2
CPI 4	20	20	20	20	20	20	20	20	20
Smoker									
CPI 0,1,2	10.3	15.9	14.3	17.5	13.5	21.4	19	24.6	27.8
CPI 3	18.7	16	17.3	16	18.7	25.3	24	26.7	22.7
CPI 4	28	28	28	24	24	24	28	28	32

4. Logistic regression analysis related to obesity and the occurrence of periodontal diseases

Logistic regression analysis was conducted on the relationship between BMI and the development of periodontal diseases, based on BMI at the start of Cohort I and the CPI at the last year, as well as the BMI at the start of Cohort II and CPI of the last year (Table 3).

In Cohort I, the odds ratio to develop periodontal diseases of the obese individuals among the "non-smokers", in comparison with those of normal body weight, was 1.43 (95% confidence interval: 0.85-2.40). Among the "smokers", the odds ratio of the obese individuals to develop periodontal diseases, in comparison with those of normal body weight, was 1.31 (95% confidence interval: 0.86-2.00). No statistically significant difference was noted in these results.

Among the "non-smokers" of Cohort II, the odds ratio of obese individuals to develop periodontal diseases, in comparison with those of normal body weight, was 2.86 (95% confidence interval: 1.06-7.71). Among the "smokers", the odds ratio for developing periodontal diseases for obese individuals, in comparison with normal weight subjects, was 2.41 (95% confidence interval: 1.14-5.12). A statistically significant difference was noted in these results.

Ⅳ. Discussion

For obesity, one of the bases for metabolic syndrome, epidemiological and experimental studies have been continued to investigate its implication from diverse aspects, including mortality according to its complications, morbidity, factors related to life style and the mechanism of its development. As

Table3 Results of logistic regression analysis for BMI and the developement of periodontal disease

Smoking status BMI	Cohort Ⅰ			Cohort Ⅱ		
	Logistics coefficient	Standard error	Odds ratio (95%CI)	Logistics coefficient	Standard error	Odds ratio (95%CI)
Nonsmoker						
BMI 18.5-24.9			1.00			1.00
BMI ＜ 18.5	−1.321	0.779	0.27(0.06-1.23)	0.24	0.939	1.27(0.20-8.00)
BMI ≧ 25.0	0.356	0.266	1.43(0.85-2.40)	1.051	0.506	2.86(1.06-7.71)*
Quit smoker						
BMI 18.5-24.9			1.00			1.00
BMI ＜ 18.5	−0.817	1.245	0.44(0.04-5.07)	−	−	−
BMI ≧ 25.0	1.311	0.545	3.71(1.28-10.81)*	−0.125	−0.839	0.88(0.17-4.57)
Smoker						
BMI 18.5-24.9			1.00			1.00
BMI ＜ 18.5	−0.251	0.342	0.78(0.40-1.52)	0.806	0.663	2.24(0.61-8.21)
BMI ≧ 25.0	0.271	0.216	1.31(0.86-2.00)	0.880	0.384	2.41(1.14-5.12)*

*: $p < 0.05$

part of these ongoing investigations, the current project focused on a highly reliable 8-year cohort study on the onset and progression of periodontal diseases. Consequently, it came to be generally confirmed that obesity has an effect on the onset and progression of periodontal diseases.

As a pioneering study on the relationship between periodontal diseases and obesity, Perlstein et al. [15] conducted an animal experiment in 1977 and reported that the former exacerbated in obese rats suffering from hypertension. No studies on this subject were conducted for quite some time in the ensuing period; but in 1998, Saito et al. [20] conducted a study on 241 Japanese between the ages of 20 and 59 years and analyzed the relationship between BMI and CPI by employing logistic regression analysis that had been adjusted for age, gender, oral hygiene practices and smoking habits. They reported that the relative risk of developing periodontal diseases for those with a BMI of 25.0 to 29.9 was 3.4 times that of individuals with a BMI of less than 20.0. They also reported that the relative risk increased to 8.6 times when the BMI exceeded 30.0. They further investigated 643 subjects from the same geographic area by adding the waist-hip ratio as a risk factor. It was found that either BMI or the waist-hip ratio was proportional to the incidence of periodontal diseases.

Wood et al. [21], all of the United States, also reported in their studies on periodontal diseases and obesity. Among these series of studies, Wood et al. quoted the results of the NHANES III investigation, a national health and nutrition survey on Americans, and presented a statistically significant correlation of attachment loss, an indicator for periodontal diseases, with various indicators for obesity. Furthermore, Al-Zahrani et al.[8] reported that among young adults ranging in age from 18 to 34 years, the relative risk for developing periodontal diseases in the obese subjects with a BMI exceeding 30.0 is 1.76 times greater than those of normal body weight (with a BMI ranging from 18.5 to 24.9).

These are the least reliable cross-sectional studies among epidemiological research. For epidemiological techniques, they present problems but do not prove a cause-effect relationship. They are the most reliable, randomized controlled, comparative studies but it is logically impossible to examine obesity and periodontal diseases with men as the subjects. Thus it is necessary to observe obesity and the development and progress of periodontal diseases through a cohort study to prove a cause-effect relationship from a long-term observation. In this study, the observation was continued for 4 years in a Cohort I and 8 years in Cohort II. Consequently, a similar tendency was found in these two series of studies. Specifically, the group with the highest CPI also indicated the highest percentage of obese individuals or mean BMI value. In Cohort II in which the observations lasted for 8 years, the results of a logistic regression analysis indicated that the odds ratio for developing periodontal diseases of obese individuals among "non-smokers" was 2.85 in comparison with those with normal body weight, while the ratio of the obese among "smokers" was 2.41. These results generally agreed with those of the earlier studies conducted mostly in the United States and Japan, proving that the probability for developing periodontal diseases is greater in obese individuals in comparison with those of normal body weight.

It was established recently that adipocytes produce not only leptin but also various other physiologically active substances, such as TNF-α, PAI-1 and adipsin. Thus a new

concept − adipose tissue is not just a site to store energy but it also serves as a huge endocrine organ − is being formed. For the mechanism by which obesity causes periodontal diseases, TNF-α, a type of cytokines that are secreted by adipocytes, is believed to serve as a major factor [22].

In addition to the anti-neoplastic action and defense against invasion into the body (such as activation of neutrophils), TNF-α plays an important role when the body is exposed to stressful conditions, such as inflammation, tissue destruction, fever and shock. Bone absorption is a form of stress brought about by TNF-α . Thus it is surmised that if a large amount of TNF-α is secreted from the adipose tissue of an obese individual, such an excess cytokine may have a significant effect on the development and progression of a periodontal disease (with its chief symptom being alveolar bone absorption). In periodontal diseases that affect an obese patient, TNF-α secreted by adipose tissue is added to that manufactured by macrophages of the local periodontal tissue. Thus absorption of the alveolar bone progresses, further aggravating the disease condition of the periodontium.

It is suspected that among the physiologically active substances secreted by adipocytes, PAI-1 in addition to TNF-α affects periodontal diseases. When the PAI-1 level in the blood rises, thrombi readily form in the microvasculature, adversely affecting the blood circulation in the periodontium, subsequently further aggravating the disease process. The significance of the formation of factors related to the complement system in the adipose tissue has not been thoroughly elucidated; but it has been suggested that these factors are also involved in the development of periodontal diseases in obese individuals [20].

It is currently believed that physiologically active substance(s) secreted by adipocytes have a role in the mechanism by which obesity is incriminated in the development of periodontal diseases. The results of the present study epidemiologically indicated that the probability of developing periodontal diseases is higher in obese individuals in comparison with those of normal body weight. The results confirmed that obesity is indeed a risk factor for these diseases.

In addition to the effects of physiologically active substances that are secreted by these adipocytes, it is necessary to consider the complex effects that diabetes mellitus exerts [12].

In the present study, diabetic patients were excluded to simplify the correlation between periodontal diseases and obesity. However, it is evident that diabetes mellitus exacerbates periodontal diseases. It is readily conceivable that certain metabolic anomalies provoked by obesity prior to the diabetic onset cause or exacerbate periodontal diseases.

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