

Relationship between the prognosis of periodontitis and occlusal force during the maintenance phase – a cohort study

**N. Takeuchi¹, D. Ekuni¹,
T. Yamamoto², M. Morita¹**

¹Department of Preventive Dentistry, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Shikata-cho, Kita-ku, Okayama, Japan and ²Division of Sociological Approach in Dentistry, Department of Dental Sociology, Kanagawa Dental College, Inaoka-cho, Yokosuka, Japan

Takeuchi N, Ekuni D, Yamamoto T, Morita M. Relationship between the prognosis of periodontitis and occlusal force during the maintenance phase – a cohort study. J Periodont Res 2010; 45: 612–617. © 2010 John Wiley & Sons A/S

Background and Objective: Few studies have longitudinally investigated the relationship between periodontal disease progression and occlusal factors in individual subjects during the maintenance phase of periodontal therapy. The aim of this cohort study was to investigate the relationship between biting ability and the progression of periodontal disease in the maintenance phase.

Material and Methods: A total of 194 patients were monitored for 3 years during the maintenance phase of periodontal therapy. The subjects with disease progression (Progress group) were defined based on the presence of ≥ 2 teeth demonstrating a longitudinal loss of proximal attachment of ≥ 3 mm or tooth-loss experience as a result of periodontal disease during the study period. The subjects with high occlusal force were diagnosed as men who showed an occlusal force of more than 500 N and women who showed an occlusal force of more than 370 N. The association between biting ability and the progression of periodontitis was investigated using logistic regression analysis.

Results: There were 83 subjects in the Progress group and 111 subjects in the Non-progress group. A backward, stepwise logistic regression model showed that the progression of periodontal disease was significantly associated with the presence of one or more teeth with a high clinical attachment level (CAL) of ≥ 7 mm (odds ratio: 2.397; 95% confidence interval: 1.306–4.399) ($p = 0.005$) and low occlusal force (odds ratio: 2.352; 95% confidence interval: 1.273–4.346) ($p = 0.006$).

Conclusion: The presence of one or more teeth with a high CAL of ≥ 7 mm and low occlusal force might be possible risk factors for periodontal progression in the maintenance phase of periodontal therapy.

Daisuke Ekuni, DDS, PhD, Department of Preventive Dentistry, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, 2-5-1 Shikata-cho, Kita-ku, Okayama 700-8558, Japan
Tel: +81 86 235 6712
Fax: +81 86 235 6714
e-mail: dekuni7@md.okayama-u.ac.jp

Key words: occlusal force; risk factors; periodontal progression; maintenance phase

Accepted for publication January 6, 2010

Many reports have discussed risk factors for the progression of periodontal disease. Age, gender, socioeconomic status, education, ethnicity, smoking, stress, obesity, diabetes, osteoporosis, specific bacteria in subgingival plaque, genetic factors (interleukin-1 geno-

type), crowding and bruxism have been identified as risk factors for periodontal disease progression (1–3).

The maintenance phase of periodontal therapy is necessary to keep periodontal tissue stable after initial preparation therapy, periodontal sur-

gical therapy or therapy for recovering oral function. However, it is possible that the risk of periodontal disease progression and the risk of recurrence of periodontal disease increase during the maintenance phase of periodontal therapy (4–6). Therefore, it is impor-

tant to determine the risk factors for the progression of periodontal disease, even in the maintenance phase of periodontal therapy.

Several studies have reported on risk factors for disease progression in the maintenance phase of periodontal therapy. Pretzl *et al.* (7) identified tooth-related factors, such as baseline bone loss, furcation involvement and use as an abutment tooth, to be risk factors. According to Matuliene *et al.* (8), a residual probing pocket depth of ≥ 6 mm after active periodontal therapy represented a risk factor for both the progression of periodontitis and tooth loss during the maintenance phase of periodontal therapy at the patient, tooth and site levels. They also mentioned that multiple sites (nine or more) with residual probing pocket depths of ≥ 5 mm represented a risk for further progression of periodontitis in patients. Kamma *et al.* (9) proposed that factors related to periodontal disease progression included high bacterial counts of *Porphyromonas gingivalis*, *Treponema denticola* and total bacterial load, number of acute episodes, number of teeth lost, smoking and stress.

Occlusion is also considered to relate to periodontal disease. For example, Burgett *et al.* (10) reported a significantly greater gain of clinical periodontal attachment in patients who received an occlusal adjustment compared with those who did not. Nunn *et al.* (11) found a strong association between initial occlusal trauma and various clinical parameters indicative of periodontal disease. Teeth with untreated occlusal trauma showed a significantly greater increase in probing depth per year than either teeth without initial occlusal trauma or teeth with treated initial occlusal trauma (12). By contrast, patients with occlusal trauma did not have more severe periodontal destruction than patients without occlusal trauma (13,14). Therefore, the relationship between the progression of periodontal destruction and occlusion has been controversial. In addition, most of these studies have focused on tooth-level, not individual-level, traumatic occlusion.

In a previous cross-sectional study (15) we found that periodontal

destruction was significantly associated with low individual-level biting ability. Therefore, it was hypothesized that individual-level biting ability (occlusal force, occlusal pressure, or occlusal contact area) can be used as a risk factor for the progression of periodontal disease in the maintenance phase. Few previous studies have longitudinally investigated the relationship between periodontal disease progression and occlusal factor in individual subjects. The aim of this cohort study was to investigate any relationship between biting ability and the progression of periodontal disease in the maintenance phase.

Material and methods

Study population

This cohort study was performed from April 2006 to March 2009. The study protocol was approved by the Ethics Committee of the Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences. Written informed consent was obtained from all subjects who agreed to participate. At baseline, 223 subjects (65 men and 158 women, 63.8 ± 10.2 years of age) who had received periodontal maintenance care at the Clinic of Preventive Dentistry of Okayama University Hospital, were selected for the study.

The subjects in the periodontal maintenance phase were identified as those who had been diagnosed with chronic periodontitis; those who had been receiving comprehensive dental care, including nonsurgical periodontal therapy consisting of oral hygiene instructions, supra/subgingival debridement, and scaling and root planing of all pockets (≥ 4 mm); those who had $< 20\%$ sites showing bleeding on probing (BOP); and those who had shown no features of acute periodontal inflammation or gingival abscess within the previous 6 mo (15).

Smokers, diabetic patients and patients with systemic diseases requiring antibiotic therapy were excluded from the analysis. Subjects were also excluded if they had no occlusal contacts between maxillary and mandibular

teeth, and if they had temporomandibular dysfunction (15).

Clinical examination

Three dentists who were familiar with the clinical examination procedure and produced closely correlated results examined the whole teeth present. The clinical measurements included probing pocket depth, clinical attachment level (CAL), BOP, tooth mobility and the presence of a prosthesis. Probing pocket depth and CAL, rounded to the nearest 1 mm, were measured at six sites in each tooth (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual and disto-lingual) using a colour-coded probe (CP-8; Hu-Friedy, Chicago, IL, USA). The CAL was determined as the distance from the cemento–enamel junction to the bottom of the periodontal pocket. BOP was defined as the presence of bleeding after gentle probing with 25 g of probing force.

Measurement of biting ability

Biting ability, including occlusal force, occlusal contact area and occlusal pressure, was measured using a pressure-sensitive sheet (Dental Prescale, Type-R 50H; Fuji Film, Tokyo, Japan) and analyzed using an image scanner (Occluzer, FPD-707; Fuji Film). The pressure-sensitive sheet consisted of two paper sheets and numerous microcapsules containing a colour-forming material between them (16). The microcapsules are broken to react with a colour-developing material when the sheet is bitten on. According to the magnitude of the pressure applied, different densities of colour are formed. The occlusal force and the occlusal contact area were calculated after scanning the sheet using an image scanner (Occluzer), taking into consideration the area and different densities of colour. The occlusal force (N) was determined as the sum of the degree of coloration and the area at each contact point. The area discoloured by biting was recorded as the occlusal contact area (mm^2). The occlusal pressure (N/mm^2) is the occlusal force per 1 mm^2 of the occlusal contact area (17).

The subjects were seated with their heads upright and in an unsupported natural head position. After several trial attempts to maintain an intercuspal bite, the sheet was placed in the patient's mouth so that the midline of the arch coincided with the midline of the sheet. Care was taken to include all of the teeth present. The buccal mucosa was retracted so as not to deform the sheet. Each patient was instructed to bite as forcefully as possible for about 3 s. Subjects who were wearing removable partial dentures were asked to remove their denture before their biting ability was measured (15).

Statistical analysis

The subjects diagnosed as those with disease progression (Progress group) were defined based on the presence of ≥ 2 teeth demonstrating a longitudinal loss of proximal attachment of ≥ 3 mm during the 3-year study period (18). The subjects who had undergone at least one tooth extraction as a result of periodontal disease (19) were also included in the Progress group. The other subjects were categorized into the Non-progress group. The subjects with high occlusal force were diagnosed as men who showed an occlusal force of more than 500 N and women who showed an occlusal force of more than 370 N, according to a study by Kono (20).

A Mann–Whitney *U*-test and a chi-square test were used to assess significant differences ($p < 0.05$) of clinical variables between the Progress group and the Non-progress group. A backward, stepwise logistic regression analysis was performed to determine the optimal model for the prediction of periodontal disease progression using the following dependent variables: the presence of one or more teeth with probing pocket depth of ≥ 6 mm (8); the presence of one or more teeth with CAL of ≥ 7 mm (21); the percentage of mobile teeth; and biting ability. The odds ratio (OR) and 95% confidence interval (CI) were calculated. The logistic regression models were reviewed for goodness of fit and validated by means of the Hosmer–Lemeshow sta-

tistic (22,23). All analyses were performed using a software program (SPSS 15.0 J for Windows; SPSS Japan, Tokyo, Japan). The level of significance was set at $p < 0.05$.

Results

During the study period, eight subjects had one or more teeth extracted because of periodontal disease and 75 subjects had two or more teeth demonstrating a longitudinal loss of proximal attachment of ≥ 3 mm. Finally, data from 194 subjects (51 men and 143 women; 63.7 ± 10.0 years of age) were available. There were 83 subjects in the Progress group and 111 in the Non-progress group. The age and

gender distribution of the subjects are shown in Table 1.

The mean probing pocket depth and mean CAL in the Non-progress group were 1.8 ± 0.4 mm and 2.4 ± 0.7 mm, respectively. The mean probing pocket depth and mean CAL in the Progress group were 1.9 ± 0.5 mm and 2.9 ± 0.8 mm, respectively. Table 2 shows the basic characteristics at baseline of the Progress group and the Non-progress group. The means and standard deviations of occlusal force, occlusal contact area and occlusal pressure in the Non-progress group were 564.0 ± 321.3 N, 16.5 ± 9.3 mm² and 34.8 ± 7.6 N/mm², respectively. The mean occlusal force, occlusal contact area and occlusal pressure in the Progress

Table 1. Age and gender distribution of the subjects in the Progress and Non-progress group

Age group (years)	Non-progress group (<i>n</i> = 111)			Progress group (<i>n</i> = 83)		
	Male	Female	Subtotal	Male	Female	Subtotal
≤ 49	2	10	12	1	0	1
50–59	6	22	28	8	18	26
60–69	12	34	46	7	18	25
70–79	7	13	20	5	21	26
80–89	1	4	5	2	3	5
Total	28	83	111	23	60	83
Mean \pm SD	62.2 ± 10.5			65.7 ± 9.0		

SD, standard deviation.

Table 2. Clinical parameters and biting ability at baseline (mean \pm standard deviation)

	Non-progress group <i>n</i> = 111	Progress group <i>n</i> = 83	Total <i>n</i> = 194
Number of teeth present	25.0 ± 4.2	$23.6 \pm 4.4^\ddagger$	24.4 ± 4.3
Percentage of teeth with restorations ^a	63.6 ± 32.0	63.6 ± 30.6	63.6 ± 31.3
Percentage of removable denture uses	17.1	19.3	18.0
Percentage of subjects with at least one tooth [highest PPD (≥ 6 mm)]	34.2	55.4^\dagger	43.3
Percentage of subjects with at least one tooth [highest CAL (≥ 7 mm)]	36.0	61.4^\dagger	46.9
Percentage of sites with BOP	6.4 ± 5.7	7.3 ± 5.1	6.8 ± 5.4
Percentage of mobile teeth	8.2 ± 14.5	$13.9 \pm 18.1^\ddagger$	10.6 ± 16.3
Occlusal force (N)	564.0 ± 321.3	$412.7 \pm 275.3^\ddagger$	499.3 ± 310.9
Percentage of subjects with low occlusal force (N)	29.7	54.2^\dagger	40.2
Occlusal contact area (mm ²)	16.5 ± 9.3	$12.0 \pm 8.2^\ddagger$	14.6 ± 9.1
Occlusal pressure (N/mm ²)	34.8 ± 7.6	35.8 ± 8.7	35.2 ± 8.1

BOP, bleeding on probing; CAL, clinical attachment level; PPD, probing pocket depth.

^aRestorations on the occlusal surfaces of premolars and molars.

[†] $p < 0.05$, significantly different from the Non-progress group as determined using the chi-square test.

[‡] $p < 0.05$, significantly different from the Non-progress group as determined by the Mann–Whitney *U*-test.

group were 412.7 ± 275.3 N, 12.0 ± 8.2 mm² and 35.8 ± 8.7 N/mm², respectively. The mean values of occlusal force and occlusal contact area in the Progress group were significantly lower than those in the Non-progress group. The proportion of subjects with low occlusal force (54.2%) in the Progress group was significantly higher than the proportion of subjects with low occlusal force (29.7%) in the Non-progress group ($p = 0.001$). The proportion of subjects with one or more teeth with a high probing pocket depth (of ≥ 6 mm) and the proportion of subjects with one or more teeth with a high CAL (of ≥ 7 mm) was also significantly higher in the Progress group than in the Non-progress group ($p = 0.004$, $p = 0.001$, respectively).

A backward, stepwise logistic regression model showed that the progression of periodontal disease was significantly associated with the presence of one or more teeth with a high CAL of ≥ 7 mm (OR: 2.397; 95% CI: 1.306–4.399) ($p = 0.005$) and a low occlusal force (OR: 2.352; 95% CI: 1.273–4.346) ($p = 0.006$). (Table 3).

Discussion

As few studies (12) have longitudinally investigated the relationship between periodontal disease progression and the initial occlusal factor, we performed this 3-year cohort study focusing on biting ability. We found that the group with a high occlusal force had a better prognosis of periodontitis than the group with a low occlusal force. The result also confirmed the findings of our previous cross-sectional study, in which peri-

odontal destruction was significantly associated with decreased biting ability of individual subjects (15). It is possible that the occlusal force can be used to determine the prognosis of periodontal disease during maintenance care.

The relationship between occlusion and periodontal health has been reported in animals (24–27) and in humans (11,12,14). As previously mentioned, most of these studies were concerned with the effect of tooth-level traumatic occlusion, not the occlusal factor in individual subjects. To understand the relationship between occlusion and periodontal health, the occlusal factor in individual subjects could be considered in addition to smoking, gender or diabetes.

Previous *in vitro* studies support our results. The reaction to mechanical force is an essential biological reaction (28). The periodontal ligament (PDL) is a highly specialized connective tissue that lies between the cementum and the alveolar bone, maintaining their homeostasis while being subjected continuously to the mechanical stress caused by occlusion and mastication (29,30). When a tooth has lost occlusion, atrophy of periodontal tissue from disuse occurs, and alveolar bone starts to resorb (31,32). In other words, PDL cells respond to a moderate cyclical occlusal load, and suppression of osteoclastic bone resorption occurs in alveolar bone (33). Based on different responses of PDL cells to tensional stress, mechanical stress plays an important role in the remodeling and functional regulation of the PDL (34).

The cyclic force that activates PDL cell function is dose-dependent (35–37). Previous studies (29,33–37) were mostly in the field of orthodontics. The

mechanical force examined in these studies was a tension force, not occlusal load. The effects of tension force and occlusal load on periodontal tissue might be different. However, a practical mechanical stress, such as occlusal force (and not tension force) might be required for PDL cell function.

Makino *et al.* (38) examined whether initial periodontal treatment yielded functional improvement of the occlusal force and periodontal pocket. They reported that occlusal force significantly increased after periodontal treatment. They mentioned that an increase in the occlusal force might have been induced because of improvements in the periodontium by restoration of the PDL fibers and because the number of inflammatory cells had decreased in the PDL. An increase in occlusal force following initial therapy can contribute to a good prognosis of periodontal disease in maintenance therapy.

According to a study by Okiyama *et al.* (39), a large maximal occlusal force was associated with a high masticatory performance, especially with harder foods. Kono (20) reported that after adjustment for age and the number of residual teeth, occlusal force had a positive partial correlation with handgrip strength in men, and positive partial correlations with usual walking speed and body muscle mass in women. Maintenance of a high occlusal force in the maintenance phase of periodontal therapy could therefore improve the quality of life.

In the present study, the risk of periodontitis progression was increased in subjects having one or more teeth with a high CAL of ≥ 7 mm at baseline. Ogawa *et al.* (40) reported that smoking and a high CAL of ≥ 6 may be considered as risk factors for further periodontal disease progression among healthy elderly people of ≥ 70 years of age. Hirotsu *et al.* (21) reported that teeth severely affected by periodontitis (a CAL of ≥ 7 mm) at baseline are risk factors associated with periodontal disease progression. Therefore, the results of this study agree with those of previous reports.

We defined periodontal progression as the presence of at least two teeth demonstrating a longitudinal loss of

Table 3. Backward stepwise logistic regression analysis for the progression of periodontal disease

Independent variables	Adjusted OR	95% CI	<i>p</i> -value
Occlusal force (N)			
> 500 for men/> 370 for women	1.000		0.006
≤ 500 for men/ ≤ 370 for women	2.352	1.273–4.346	
Percentage of subjects with at least one tooth (highest CAL ≥ 7 mm)			
Highest CAL < 7 mm	1.000		0.005
Highest CAL ≥ 7 mm	2.397	1.306–4.399	

CAL, clinical attachment level; CI, confidence interval; OR, odds ratio.

proximal attachment of ≥ 3 mm, or tooth loss because of periodontal disease, during the 3-year study. During the study period, 42.8% of the subjects were assigned to the Progress group. Matuliene *et al.* (8) used the same definition and reported a similar prevalence of progression of periodontitis in the subjects in their study: 43.3% of subjects of 14–69 years of age were classified as having progressive periodontitis. Therefore, the subject population monitored in the present study was not a special subgroup. Fardal *et al.* (19) used the definition of periodontal progression as tooth loss. In their study, 21.6% of nonsmoker subjects were classified as having progressive periodontitis. In our study, eight patients (4.1%) lost at least one tooth for periodontal reasons. Fardal *et al.* monitored patients for 10 years, whereas the follow-up period in our study was 3 years. Therefore, the results cannot be easily compared.

In this study, we used Dental Prescale® to measure occlusal force. Another method used to measure occlusal force is a strain-gauge force transducer, which can measure a single pair of opposing upper and lower teeth (41). However, it is not easy to measure the occlusal force of all pairs of teeth. In addition, this device is thick and does not measure the occlusal force in the centric occlusion. Because the thickness of the pressure-sensitive sheet used in this study was 98 ± 5 μ m, we were able to measure the full-mouth occlusal force in the position that is extremely close to centric occlusion.

There have been some previous reports on periodontal risk factors in supportive periodontal therapy (7–9), such as smoking, stress, alveolar bone loss, furcation involvement, use as an abutment tooth and high bacterial counts of *P. gingivalis* and *T. denticola*. We included only nonsmokers; however, we did not record stress, furcation involvement and specific bacteria in subgingival plaque. It is possible that these factors also affect the progression of periodontal disease. A further comprehensive study is required to determine the effect of each risk factor.

There are other limitations of this study. A previous report used radiographic alveolar bone loss to define the progression of periodontal disease (42). In our study, full-mouth radiographs at baseline were not available in some patients. Thus, we could not use bone loss as the definition for disease progression. However, it might be reasonable to compare the results between subjects who received tooth extraction because of periodontal disease and those who did not. However, because the number of subjects who received tooth extraction because of periodontal disease was too small ($n = 8$), we considered that the comparison would not be appropriate.

In conclusion, the presence of one or more teeth with a high CAL of ≥ 7 mm and a low occlusal force (men, ≤ 500 N; women, ≤ 370 N) might be possible risk factors for periodontal progression during the maintenance phase of periodontal therapy.

References

1. Cronin AJ, Claffey N, Stassen LF. Who is at risk? Periodontal disease risk analysis made accessible for the general dental practitioner. *Br Dent J* 2008;**205**:131–137.
2. Van Dyke TE, Sheilesh D. Risk factors for periodontitis. *J Int Acad Periodontol* 2005; **7**:3–7.
3. Pihlstrom BL, Michalowicz BS, Johnson NW. Periodontal diseases. *Lancet* 2005;**366**:1809–1820.
4. Axelsson P, Lindhe J. The significance of maintenance care in the treatment of periodontal disease. *J Clin Periodontol* 1981; **8**:281–294.
5. Bostanci HS, Arpak MN. Long-term evaluation of surgical periodontal treatment with and without maintenance care. *J Nihon Univ Sch Dent* 1991;**33**:152–159.
6. Renvert S, Persson GR. Supportive periodontal therapy. *Periodontol* 2000 2004; **36**:179–195.
7. Pretzl B, Kaltschmitt J, Kim TS, Reitmeir P, Eickholz P. Tooth loss after active periodontal therapy. 2: tooth-related factors. *J Clin Periodontol* 2008;**35**:175–182.
8. Matuliene G, Pjetursson BE, Salvi GE *et al.* Influence of residual pockets on progression of periodontitis and tooth loss: results after 11 years of maintenance. *J Clin Periodontol* 2008;**35**:685–695.
9. Kamma JJ, Baehni PC. Five-year maintenance follow-up of early-onset periodontitis patients. *J Clin Periodontol* 2003;**30**:562–572.
10. Burgett FG, Ramfjord SP, Nissle RR, Morrison EC, Charbeneau TD, Caffesse RG. A randomized trial of occlusal adjustment in the treatment of periodontitis patients. *J Clin Periodontol* 1992; **19**:381–387.
11. Nunn ME, Harrel SK. The effect of occlusal discrepancies on periodontitis. I. Relationship of initial occlusal discrepancies to initial clinical parameters. *J Periodontol* 2001;**72**:485–494.
12. Harrel SK, Nunn ME. The effect of occlusal discrepancies on periodontitis. II. Relationship of occlusal treatment to the progression of periodontal disease. *J Periodontol* 2001;**72**:495–505.
13. Shefter GJ, McFall WT Jr. Occlusal relations and periodontal status in human adults. *J Periodontol* 1984;**55**:368–374.
14. Jin LJ, Cao CF. Clinical diagnosis of trauma from occlusion and its relation with severity of periodontitis. *J Clin Periodontol* 1992;**19**:92–97.
15. Takeuchi N, Yamamoto T. Correlation between periodontal status and biting force in patients with chronic periodontitis during the maintenance phase of therapy. *J Clin Periodontol* 2008;**35**: 215–220.
16. Suzuki T, Kumagai H, Watanabe T, Uchida T, Nagao M. Evaluation of complete denture occlusal contacts using pressure-sensitive sheets. *Int J Prosthodont* 1997;**10**:386–391.
17. Miyaura K, Matsuka Y, Morita M, Yamashita A, Watanabe T. Comparison of biting forces in different age and sex groups: a study of biting efficiency with mobile and non-mobile teeth. *J Oral Rehabil* 1999;**26**:223–227.
18. Tonetti MS, Claffey N, European Workshop in Periodontology group C. Advances in the progression of periodontitis and proposal of definitions of a periodontitis case and disease progression for use in risk factor research. Group C consensus report of the 5th European Workshop in Periodontology. *J Clin Periodontol* 2005;**32**(suppl 6):210–213.
19. Fardal Ø, Johannessen AC, Linden GJ. Tooth loss during maintenance following periodontal treatment in a periodontal practice in Norway. *J Clin Periodontol* 2004;**31**:550–555.
20. Kono R. Relationship between occlusal force and preventive factors for disability among community-dwelling elderly persons [in Japanese]. *Nippon Ronen Igakkai Zasshi* 2009;**46**:55–62.
21. Hirotsu T, Yoshihara A, Yano M, Ando Y, Miyazaki H. Longitudinal study on periodontal conditions in healthy elderly people in Japan. *Community Dent Oral Epidemiol* 2002;**30**:409–417.
22. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between

- upper body obesity and periodontitis. *J Dent Res* 2001;**80**:1631–1636.
23. Ekuni D, Yamamoto T, Koyama R, Tsuneishi M, Naito K, Tobe K. Relationship between body mass index and periodontitis in young Japanese adults. *J Periodontol Res* 2008;**43**:417–421.
 24. Ericsson I, Lindhe J. Lack of effect of trauma from occlusion on the recurrence of experimental periodontitis. *J Clin Periodontol* 1977;**4**:115–127.
 25. Ericsson I, Lindhe J. Effect of longstanding jiggling on experimental marginal periodontitis in the beagle dog. *J Clin Periodontol* 1982;**9**:497–503.
 26. Kantor M, Polson AM, Zander HA. Alveolar bone regeneration after removal of inflammatory and traumatic factors. *J Periodontol* 1976;**47**:687–695.
 27. Polson AM, Zander HA. Effect of periodontal trauma upon intrabony pockets. *J Periodontol* 1983;**54**:586–591.
 28. Pavlin D, Gluhak-Heinrich J. Effect of mechanical loading on periodontal cells. *Crit Rev Oral Biol Med* 2001;**12**:414–424.
 29. Davidovitch Z, Nicolay OF, Ngan PW, Shanfeld JL. Neurotransmitters, cytokines, and the control of alveolar bone remodeling in orthodontics. *Dent Clin North Am* 1988;**32**:411–435.
 30. Lekic P, McCulloch CA. Periodontal ligament cell population: the central role of fibroblasts in creating a unique tissue. *Anat Rec* 1996;**245**:327–441.
 31. Cohn SA. Disuse atrophy of the periodontium in mice. *Arch Oral Biol* 1965;**10**:909–919.
 32. Cohn SA. Disuse atrophy of the periodontium in mice following partial loss of function. *Arch Oral Biol* 1966;**11**:95–105.
 33. Kanzaki H, Chiba M, Sato A *et al*. Cyclical tensile force on periodontal ligament cells inhibits osteoclastogenesis through OPG induction. *J Dent Res* 2006;**85**:457–462.
 34. Ozaki S, Kaneko S, Podyma-Inoue KA, Yanagishita M, Soma K. Modulation of extracellular matrix synthesis and alkaline phosphatase activity of periodontal ligament cells by mechanical stress. *J Periodontol Res* 2005;**40**:110–117.
 35. Yamaguchi M, Shimizu N, Shibata Y, Abiko Y. Effects of different magnitudes of tension-force on alkaline phosphatase activity in periodontal ligament cells. *J Dent Res* 1996;**75**:889–894.
 36. Yamaguchi M, Ozawa Y, Nogimura A *et al*. Cathepsins B and L increased during response of periodontal ligament cells to mechanical stress in vitro. *Connect Tissue Res* 2004;**45**:181–189.
 37. Zhong W, Xu C, Zhang F, Jiang X, Zhang X, Ye D. Cyclic stretching force-induced early apoptosis in human periodontal ligament cells. *Oral Dis* 2008;**14**:270–276.
 38. Makino M, Murakami K, Yokota M. A study of alterations of the Occlusal force following non surgical treatment [in Japanese]. *J Jpn Soc Periodontol* 2007;**49**:37–46.
 39. Okiyama S, Ikebe K, Nokubi T. Association between masticatory performance and maximal occlusal force in young men. *J Oral Rehabil* 2003;**30**:278–282.
 40. Ogawa H, Yoshihara A, Hirotomi T, Ando Y, Miyazaki H. Risk factors for periodontal disease progression among elderly people. *J Clin Periodontol* 2002;**29**:592–597.
 41. Trulsson M, Gunne HS. Food-holding and -biting behavior in human subjects lacking periodontal receptors. *J Dent Res* 1998;**77**:574–582.
 42. Paulander J, Wennström JL, Axelsson P, Lindhe J. Some risk factors for periodontal bone loss in 50-year-old individuals. A 10-year cohort study. *J Clin Periodontol* 2004;**31**:489–496.

This document is a scanned copy of a printed document. No warranty is given about the accuracy of the copy. Users should refer to the original published version of the material.