J Clin Periodontol 2005; 32: 556–560 doi: 10.1111/j.1600-051X.2005.00712.x

Journal of Clinical Periodontology

Effect of periodic preventive care on the progression of periodontal disease in young adults with Down's syndrome

Toshihiro Yoshihara, Takanobu Morinushi, Sachiko Kinjyo and Youichi Yamasaki

Department of Pediatric Dentistry, School of Dentistry, Kagoshima University, Kagoshima, Japan

Yoshihara T, Morinushi T, Kinjyo S, Yamasaki Y. Effect of periodic preventive care on the progression of periodontal disease in young adults with Down's syndrome. J Clin Periodontol 2005; 32: 556–560. doi: 10.1111/j.1600-051X.2005.00712.x. © Blackwell Munksgaard 2005.

Abstract

Objectives: To examine the effect of periodic preventive care on the progression of periodontal disease in 24 young adults with Down's syndrome (DS) (mean age ± SD: 20.8 ± 5.6 years)

Material and Methods: Subjects were divided into two groups: 13 subjects who had frequently visited our clinic (mean interval between visits: 3.7 ± 1.3 months; managed group) and 11 subjects who had not visited our clinic for more than 1 year (mean duration of no visits: 27.5 ± 10.1 months; interrupted group). The progression of periodontal disease in the subjects was evaluated clinically, microbiologically and roentgenologically.

Results: Clinical parameters (probing depth, frequency of the presence of a pathological periodontal pocket, alveolar bone loss, frequency of the incidence of pathological bone loss, and modified total PMA (M-PMA)) and benzoyl-DL-arginine-naphthylamide (BANA) scores in the interrupted group were significantly higher than those in the managed group. Subject age showed significant positive correlations with probing depth and alveolar bone loss. BANA scores showed significant positive correlations with probing depth, alveolar bone loss and M-PMA in the interrupted group.

Conclusions: The results suggest that periodic preventive care is effective for suppressing the progression of periodontal disease in young adults with DS.

Key words: Down's syndrome; periodic dental care; periodontal disease; progression

Accepted for publication 29 September 2004

A high prevalence of advanced periodontal disease has been reported in subjects with Down's syndrome (DS), with rates varying between 60% and 100% for those under 30 years of age (Sznajder et al. 1968, Barnett et al. 1986). Increased prevalence and severity of periodontal disease has been found in DS subjects compared with those of other intellectually impaired subjects, despite the fact that the oral hygiene levels in both subject groups were similar because of the same degrees of intellectual impairment and the same environmental conditions (Saxen & Aula 1982, Shaw & Saxby 1986). The high prevalence of periodontal disease in DS subjects may be because of impaired host defense associated with reduced neutrophil and monocyte chemotaxis, impaired neutrophil phagocytoxis, reduced T lymphocyte counts and immature T lymphocytes (Hanookai et al. 2000).

Although several studies have shown that periodontal treatment is not able to suppress the progression of periodontal disease (Saxén & Aula 1982, Hanookai et al. 2000), the effectiveness of frequent preventive care in DS subjects has recently been reported (Sakellari et al. 2001). However, there have been few studies on the relationship between preventive care for periodontal disease and progression of periodontal disease in young adult subjects with DS. The purpose of this study was to determine the effectiveness of frequent preventive care for periodontal disease in young adults with DS by evaluating the progression of diseases clinically, microbiologically and roentgenologically.

Materials and Methods

Subjects

Twenty-four young adults with DS (18 males and six females; mean age ± SD,

20.8 ± 5.6 years) attending the Pediatric Dental Clinic of Kagoshima University Dental Hospital participated in this study (Table 1). The criteria for inclusion were (1) no antibiotic treatment within 3 months prior to the start of the study and (2) similar levels of cooperation regarding the frequency of examinations and preventive care. Preventive care consisted of professional tooth cleaning, various combinations of scaling, and counseling for caregivers regarding caries and periodontal disease.

Table 1. Distribution of ages and sexes of subjects

	Total ($n = 24$)	Managed group ($n = 13$)	Interrupted group ($n = 11$)
Age (years)			
Mean age ± SD	20.8 ± 5.6	20.2 ± 5.2	21.6 ± 6.2
Median	18.6	18.7	18.6
Range	14.4 – 36.8	14.4 – 31.0	16.6 – 36.8
Age at the initial visit to our clinic	7.8 ± 7.7	9.2 ± 9.1	5.2 ± 2.4
Sex			
Males:females	18:6	10:3	8:3

Study design

Subjects were divided into two groups: 13 subjects who had frequently visited our clinic (mean interval between visits: 3.7 ± 1.3 months; managed group) and 11 subjects who had not visited our clinic for more than 1 year (mean duration of no visits: 27.5 ± 10.1 months; interrupted group) and whose caregivers were asked by telephone to visit our clinic. Levels of intellectual impairment appeared to be almost equal for the two groups based on their ability to sufficiently accept the clinical examination and their daily life ability, as determined by interview with their caregivers. Clinical examinations and microbiological examinations for periodontal disease were carried out in both groups.

The scientific and ethical aspects of the protocol of this study were reviewed and approved by the Ethics Committee of the School of Dentistry, Kagoshima University and informed consent was obtained from the subjects' guardians prior to participation in the study.

Clinical examinations

The following clinical examinations were performed for all teeth by the same dentist.

Gingival condition

The degree of gingival inflammation was scored using a modified total PMA (M-PMA) based on the PMA Index (Morinushi et al. 1997). The presence or absence of gingivitis was noted in the following three areas of the buccal or labial gingiva: gingival papillae (P), gingival margin (M), and attached gingiva (A). Each of these areas was scored according to the presence (1) or absence (0) of inflammation, with the M-PMA for each subject defined as follows:

$$\text{M-PMA} = \frac{\text{summation of the PMA index}}{\text{total number of examined teeth}} \times 100\ (\%)$$

Probing depth

Probing depth was measured on six sites around the tooth using a periodontal probe. A tooth with a probing depth of 4.00 mm or more was diagnosed as having a pathological periodontal pocket. The frequency of the presence of a pathological periodontal pocket was presented as the percentage of the number of the subjects that had one or more teeth with a pathological periodontal pocket out of the total number of subjects.

Alveolar bone loss

A panoramic radiograph of each subject was taken at our hospital. Alveolar bone loss was estimated by the method described by Saxén et al. (1977) from the cemento-enamel junction to the alveolar bone margin on mesial and distal surfaces of a tooth using an electronic caliper accurate to 0.01 mm. Alveolar bone loss of teeth was taken as the mean bone loss on the mesial and distal surfaces of the tooth. The methodological error (S), calculated using the formula $S = \sqrt{d^2/2N}$ (where d is the individual differences between two measurements and N the number of repeated determinations) (Gabre et al. 2001), was determined as 0.12. Measurements were performed only in teeth in which the cemento-enamel junction and alveolar bone margin could be identified. Instances in which a patient's instability rendered radiographs unsuitable for measurements were excluded. Out of 601 teeth from the total number of subjects ($n = 24$), 479 teeth were judged readable (80%). A tooth with bone loss of 5.00 mm or more on the radiograph were diagnosed as having pathological bone loss. The frequency of the incidence of pathological bone loss was presented as the percentage of the number of the subjects that had one or more teeth with pathological bone loss out of the total number of subjects.

Microbiological examinations

Subgingival plaque samples were analysed for the presence of the anaerobic periodontal pathogens, *Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythensis*, using the benzoyl-DL-arginine-naphthylamide (BANA) test (Perioscan™, Oral-B Laboratories Inc., Redwood City, CA, USA) (Loesche et al. 1990b). Subgingival plaque samples, removed from mesiobuccal surfaces of four first molars and four mesiolabial surfaces of the central incisors with a curette, were wiped onto the BANA-impregnated lower strip on the bottom of the BANA reagent card. In the case of a missing tooth, a plaque sample from the adjacent teeth was used. The reaction was activated immediately by applying water to the upper strip containing the fast black dye. The lower strip was folded onto the upper, and both strips were held in place with a metallic clip. The card was placed in an incubator at 55°C for 15 min. Results were recorded as strong dark blue-spots (score of 2); weak light-blue spots (score of 1); or no colour change (score of 0). The BANA score for each subject was defined as the mean BANA score for eight teeth that had been examined.

Statistical analysis

Median values between the two groups were compared using the Mann–Whitney U-test. Distributions between the

Table 2. Clinical parameters and BANA score for subjects

	Total ($n = 24$)	Managed group ($n = 13$)	Interrupted group ($n = 10$)	*P*-value (managed group *versus* interrupted group)
Probing depth (mean ± SD) (mm)	2.8 ± 0.4	2.5 ± 0.3	3.1 ± 0.3	<0.001*
Frequency of the presence of a pathological periodontal pocket (⩾4 mm) (%)	67 (16/24)	46 (6/13)	91 (10/11)	0.034†
Alveolar bone loss (mean ± SD) (mm)	3.54 ± 0.62	3.88 ± 0.75	4.56 ± 0.51	0.031*
Frequency of the incidence of pathological bone loss (⩾5 mm) (%)	79 (19/24)	62 (8/13)	100 (11/11)	0.041†
M-PMA (mean ± SD) (%)	24.8 ± 16.2	18.8 ± 14.2	31.9 ± 16.0	0.045*
BANA score (mean ± SD)	0.4 ± 0.4	0.2 ± 0.4	0.7 ± 0.4	0.012*

*Mann–Whitney *U*-test.
†Fisher's exact test.
BANA, benzoyl-DL-arginine-naphthylamide; M-PMA, modified total PMA.

two groups were compared using Fisher's exact test. Correlation was estimated by using Spearman's rank correlation and a $p<0.05$ was considered to be statistically significant.

Results

Clinical parameters and BANA score (Table 2)

The mean values ± SD for probing depths in the managed and interrupted groups were 2.5 ± 0.3 and 3.1 ± 0.3 mm, respectively. Frequency of the presence of a pathological periodontal pocket in the managed and interrupted groups was 46% and 91%, respectively. Probing depth and frequency of the presence of a pathological periodontal pocket were significantly higher in the interrupted group than in the managed group ($p<0.001$, Mann–Whitney *U*-test; $p=0.034$, Fisher's exact test).

The mean values ± SD of alveolar bone loss in the managed and interrupted groups were 3.88 ± 0.75 and 4.56 ± 0.51 mm, respectively. Frequency of the incidence of pathological alveolar bone loss in the managed and interrupted groups was 62% and 100%, respectively. The alveolar bone loss and frequency of the incidence of pathological bone loss in the interrupted group were significantly larger than those observed in the managed group ($p=0.031$, Mann–Whitney *U*-test; $p=0.041$, Fisher's exact test).

The mean values ± SD of M-PMA in the managed and interrupted groups were 18.8 ± 14.2% and 31.9 ± 16.0%, respectively. M-PMA in the interrupted group was significantly higher than that in the managed group ($p=0.045$, Mann–Whitney *U*-test).

The mean values ± SD of the BANA score in the managed and interrupted groups were 0.2 ± 0.4 and 0.7 ± 0.4 mm, respectively. The BANA score in the interrupted group was significantly higher than that in the managed group ($p=0.012$, Mann–Whitney *U*-test).

Table 3. Correlations between clinical and microbiological parameters

	Subject age	Probing depth	Alveolar bone loss	M-PMA	BANA score
Total ($n = 24$)					
Subject age		0.480 ($p = 0.021$)	0.610 ($p = 0.004$)	0.254 (NS)	0.384 (NS)
Probing depth			0.891 ($p<0.001$)	0.494 ($p = 0.018$)	0.038 (NS)
Alveolar bone loss				0.480 ($p = 0.021$)	0.142 (NS)
M-PMA					0.038 (NS)
BANA score					
Managed group ($n = 13$)					
Subject age		0.600 ($p = 0.038$)	0.352 (NS)	0.598 ($p = 0.042$)	0.254 (NS)
Probing depth			0.806 ($p = 0.005$)	0.402 (NS)	−0.024 (NS)
Alveolar bone loss				0.349 (NS)	−0.091 (NS)
M-PMA					−0.024 (NS)
BANA score					
Interrupted group ($n = 11$)					
Subject age		0.521 (NS)	0.506 (NS)	0.032 (NS)	−0.310 (NS)
Probing depth			0.638 ($p = 0.049$)	0.642 ($p = 0.044$)	0.835 ($p = 0.008$)
Alveolar bone loss				0.445 (NS)	0.623 ($p = 0.050$)
M-PMA					0.670 ($p = 0.034$)
BANA score					

NS, not significant; BANA, benzoyl-DL-arginine-naphthylamide; M-PMA, modified total PMA.

Correlations between clinical and microbiological parameters (Table 3)

Subject age was significantly and positively correlated with probing depth ($r=0.480$, $p=0.021$) and alveolar bone loss ($r=0.610$, $p=0.004$). Probing depth showed significant positive correlations with alveolar bone loss ($r=0.891$, $p<0.001$) and M-PMA ($r=0.494$, $p=0.018$). Alveolar bone loss showed a significant positive correlation with M-PMA ($r=0.480$, $p=0.021$).

In the managed group, subject age was significantly and positively correlated with probing depth ($r=0.600$, $p=0.038$) and M-PMA ($r=0.598$, $p=0.042$), and the positive correlation

between probing depth and alveolar bone loss was also significant ($r = 0.806$, $p = 0.005$). In the interrupted group, probing depth was significantly and positively correlated with alveolar bone loss ($r = 0.638$, $p = 0.049$), M-PMA ($r = 0.642$, $p = 0.044$) and BANA score ($r = 0.835$, $p = 0.008$). Alveolar bone loss exhibited a significant positive correlation with BANA score ($r = 0.623$, $p = 0.050$) and M-PMA was significantly and positively correlated with BANA score ($r = 0.670$, $p = 0.034$).

Discussion

In the present study, subject age (mean age ± SD: 20.8 ± 5.6 years) was significantly and positively correlated with probing depth and alveolar bone loss. A correlation between age and prevalence of bone loss in DS subjects (mean age ± SD: 17.7 ± 7.1 years) has previously been reported by Izumi et al. (1989), suggesting an age-dependent prevalence in periodontal disease in young adults with DS. It therefore seems likely that, regardless of age, some progression of periodontal disease with age is unavoidable in DS subjects.

Conversely, the present study also suggests the usefulness of periodic preventive care for suppressing the progression of periodontal disease in DS subjects. Saxén & Aula (1982) reported that preventive programs had little effect on the progression of periodontal disease in DS subjects, since such disease in these subjects progressed rather rapidly and led inexorably to loss of dentition. Conversely, Sakellari et al. (2001) reported that close monitoring of oral hygiene habits and frequent professional cleaning of the teeth of five DS subjects aged 26–37 years old had a significant effect of the progression of periodontal disease. In the present study, all clinical parameters and BANA scores related to periodontal diseases in the interrupted group were significantly higher than those of the managed group. This difference in the rates of progression of periodontal disease between the two groups might be because of periodic preventive care. The results of the present study and those of Sakellari et al. (2001) suggest that continuous, systematic and individualized preventive dental care can suppress the progression of periodontal disease in DS subjects.

In the subjects of the present study, M-PMA showed significant positive correlations with probing depth and alveolar bone loss, supporting the notion that DS subjects should be included in a preventive oral hygiene program focused on diminishing gingival inflammation, which may diminish the risk for progression of periodontal disease (Agholme et al. 1999).

Studies using normal subjects have demonstrated that *P. gingivalis*, *T. denticola* and *T. forsythensis* were frequently associated with adult forms of periodontal disease (Moore et al. 1982, Loesche et al. 1990a, b, Amalfitano et al. 1993). These BANA-positive bacterial species may also be involved in periodontal disease seen in DS subjects, and a significant association between BANA score and probing depth has been reported for DS subjects (Figueiredo et al. 2000). The results obtained in the present study corroborate these findings as significant positive correlations between probing depth and BANA score, and between alveolar bone loss and BANA scores, were only observed in the interrupted group. The effect of frequent professional supragingival plaque control on subgingival microbiota and clinical variables in DS subjects have been reported elsewhere (Sakellari et al. 2001). Consequently, inadequate oral hygiene in DS subjects because of the interruption of professional preventive dental care might therefore affect supragingival and consequently subgingival plaque composition, and result in increased probing depth and alveolar bone loss.

It is most likely that young adults with DS generally need more assistance in many daily activities than their non-disabled counterparts (Randell et al. 1992). The most frequently neglected facet of health care for such intellectually impaired individuals, regardless of whether they are institutionalized or not, may be dental care (Nowak 1974). Furthermore, dental treatment of sufficient quality is not always possible because of poor cooperation. There is increasing concern for the quality of life of intellectually impaired people, and the results of the present study should contribute not only to an understanding of periodontal disease in general but also to an extension of preventive dental care for DS patients. The results of this study show that individualized preventive dental care, performed regularly using generally available methods and maintaining adequate oral hygiene, are effective for suppressing the severity and progression of periodontal diseases in DS patients.

References

Agholme, M. B., Dahllof, G. & Modeer, T. (1999) Changes of periodontal status in patients with Down syndrome during a 7-year period. *European Journal of Oral Sciences* **107**, 82–88.

Amalfitano, J., De Filippo, A. B., Bretz, W. A. & Loesche, W. J. (1993) The effects of incubation length and temperature on the specificity and sensitivity of the BANA (N-benzoyl-DL-arginine-naphthylamide) test. *Journal of Periodontology* **64**, 848–852.

Barnett, M. L., Press, K. P., Friedman, D. & Sonnenberg, E. M. (1986) The prevalence of periodontitis and dental caries in a Down's syndrome population. *Journal of Periodontology* **57**, 288–293.

Figueiredo, L. C., Toledo, B. E. & Salvador, S. L. (2000) The relationship between place BANA reactivity and clinical parameters in subjects with mental disabilities. *Special Care in Dentistry* **20**, 195–198.

Gabre, P., Martinsson, T. & Gahnberg, L. (2001) Longitudinal study of dental caries, tooth mortality and interproximal bone loss in adults with intellectual disability. *European Journal of Oral Sciences* **109**, 20–26.

Hanookai, D., Nowzari, H., Contreras, A., Morrison, J. L. & Slots, J. (2000) Herpesviruses and periodontopathic bacteria in Trisomy 21 periodontitis. *Journal of Periodontology* **71**, 376–384.

Izumi, Y., Sugiyama, S., Shinozuka, O., Yamazaki, T., Ohyama, T. & Ishikawa, I. (1989) Defective neutrophil chemotaxis in Down's syndrome patients and its relationship to periodontal destruction. *Journal of Periodontology* **60**, 238–242.

Loesche, W.J, Bretz, W. A., Kerschensteiner, D., Stoll, J., Socransky, S. S., Hujoel, P. & Lopatin, D. E. (1990a) Development of a diagnostic test for anaerobic periodontal infections based on plaque hydrolysis of benzoyl-DL-arginine-naphthylamide. *Journal of Clinical Microbiology* **28**, 1551–1559.

Loesche, W. J., Bretz, W. A., Lopatin, D., Stoll, J., Rau, C. F., Hillenburg, K. L., Killoy, W. J., Drisko, C. L., Williams, R. & Weber, H. P. (1990b) Multi-center clinical evaluation of a chairside method for detecting certain periodontopathic bacteria in periodontal disease. *Journal of Periodontology* **61**, 189–196.

Moore, W. E., Holdeman, L. V., Smibert, R. M., Hash, D. E., Burmeister, J. A. & Ranney, R. R. (1982) Bacteriology of severe periodontitis in young adult humans. *Infection and Immunity* **38**, 1137–1148.

Morinushi, T., Lopatin, D. E. & Poperin, N. V. (1997) The relationship between gingivitis and the serum antibodies to the microbiota associated with periodontal diseases in children with Down's syndrome. *Journal of Periodontology* **68**, 626–631.

Nowak, A. J. (1974) The role of dentistry in the normalisation of the mentally retarded person. *Journal of Dentistry for Children* **41**, 34–38.

Randell, D. M., Harth, S. & Seow, W. K. (1992) Preventive dental health practices of non-institutionalized Down syndrome children: a controlled study. *Journal of Clinical Pediatric Dentistry* **16**, 225–229.

Sakellari, D., Belibasakis, G., Chadjipadelis, T., Arapostathis, K. & Konstantinidis, A. (2001) Supragingival and subgingival microbiota of adult patients with Down's syndrome: changes after periodontal treatment. *Oral Microbiology and Immunology* **16**, 376–382.

Saxén, L. & Aula, S. (1982) Periodontal bone loss in patients with Down's syndrome: a follow-up study. *Journal of Periodontology* **53**, 158–162.

Saxén, L., Aula, S. & Westermarck, T. (1977) Periodontal disease associated with Down's syndrome: an orthopantomographic evaluation. *Journal of Periodontology* **48**, 337–340.

Shaw, L. & Saxby, M. S. (1986) Periodontal destruction in Down's syndrome and in juvenile periodontitis. How close a similarity? *Journal of Periodontology* **57**, 709–715.

Sznajder, N., Carraro, J. J., Otero, E. & Carranza, F. A. Jr (1968) Clinical periodontal findings in trisomy 21 (Mongolism). *Journal of Periodontal Research* **3**, 1–5.

Address:
Toshihiro Yoshihara
Department of Pediatric Dentistry
School of Dentistry
Kagoshima University
Sakuragaoka 8-35-1
Kagoshima 890-8544
Japan
E-mail: tyoshi@denta.hal.kagoshima-u.ac.jp