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Periodontal conditions and subgingival microflora in Down syndrome patients

A case–control study

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Abstract

Objectives: The periodontal conditions and the subgingival microflora of children, adolescents and young adults (8–28 years old) with Down syndrome were investigated in the present cross-sectional study and compared with those of healthy individuals and subjects with cerebral palsy.

Material and Methods: Seventy Down syndrome patients, 121 age-matched healthy individuals and 76 patients with cerebral palsy participated in the present study. Full-mouth recordings of clinical parameters (probing depth, probing attachment level, bleeding on probing, hygiene index) and the community periodontal index of treatment needs were assessed and subgingival plaque samples were taken from the Ramfjord teeth and analysed for 14 species using ''checkerboard'' DNA–DNA hybridization.

Results: Clinical indices of periodontal inflammation and treatment needs were statistically significant higher among Down syndrome patients compared with the other two groups (ANOVA, $p = 0.000$). Important periodontal pathogens colonize these subjects earlier and at higher levels (chi-squared test, $p = 0.000$).

Discussion: Down syndrome patients display more severe periodontal destruction earlier, and heavier colonization with periodontal pathogens compared with age-matched healthy individuals and patients with cerebral palsy.

Key words: Down syndrome; periodontal conditions; subgingival microbiota

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Several differences in the oral conditions of Down syndrome patients compared with healthy individuals, have been described in the literature. Various studies have reported that periodontal disease appears with increased prevalence and higher severity among individuals with Down syndrome compared with that of age-matched healthy individuals or other groups of special needs patients (Reuland-Bosma & van Dijk 1986, Modeer et al. 1990, Genco & Loe 1993, Desai 1997, Hennequin et al. 1999, Gabre 2000). It is noteworthy that more recent studies report that severity of periodontal disease in individuals with the syndrome is milder compared with that described in older studies. This fact is attributed to better dental care of these individuals, both at home and at the dental surgery, compared with the past (Stabholz et al. 1991a, Desai 1997, Agholme et al. 1999, Hennequin et al. 1999, Amano et al. 2001, Gabre et al. 2001).

No definite conclusions have been drawn concerning the pathogenesis of periodontal destruction observed among persons with the syndrome. One line of investigation has focused on differences in the subgingival microflora while immune disorders related to the syndrome have been also proposed as a major pathogenic factor (Izumi et al. 1989, Barr-Agholme et al. 1992, Yavuzyilmaz et al. 1993, Sreedevi & Munshi 1998, Amano et al. 2000, Chaushu et al. 2002). A higher prevalence of certain periodontal pathogens, especially *Actinobacillus actinomycetemcomitans*, has been reported in certain studies but findings in the literature are not unanimous (Santos et al. 1996, Barr-Agholme et al. 1992, Amano et al. 2000, Reuland-Bosma et al. 2001). Other consensus pathogens such as members of the ''red complex'' and *Capnocytophaga* spp. have been detected subgingivally, even in young age groups (2–5 years old) (Hanookai et al. 2000). The aim of the present study was to investigate the periodontal conditions and subgingival microflora of Down syndrome patients compared with

age-matched healthy individuals and cerebral palsy patients.

Material and Methods

Subject sample

This study was designed as a cross-sectional case–control study. A total of 267 subjects aged 8–28 years, living in Thessaloniki, Greece, participated in the present study. 70 non-institutionalized Down syndrome patients (diagnosed by chromosomal examination) represent the study group (Group A), These subjects were recruited through the Down Syndrome Association of Greece. Seventy-six patients with cerebral palsy comprised the first control group (Group B). These patients were recruited through the Hellenic Society for Disabled Children, a non-profit organization. In addition, 121 healthy subjects, attending elementary and high schools of the area, as well as being patients of the Department of Preventive Dentistry, Periodontology and Implant Biology, constituted the second control group (Group C). Informed consent was obtained from all parents of participants. All three groups were divided into age-based subgroups according to the following classification: children = 8 years and 1 month–13 years old, adolescents = 13 years and 1 month–19 years old and young adults = 19 years and 1 month–28 years old.

Periodontal examination

Clinical data were recorded concerning all teeth present in the dentition. A total of 5794 teeth were examined (1609 in the children subgroup, 2126 in the adolescent subgroup and 2059 in the young adult subgroup). The following indices were recorded at four sites for each tooth (distal, buccal, mesial and lingual)

(a) Probing depth (PD)
(b) Probing attachment level (PAL)
(c) Bleeding on probing (BOP)
(d) Hygiene index (presence or absence of plaque)

All measurements were performed by one calibrated examiner (K. A.), using a manual probe (Hu-Friedy, Chicago, IL, USA). Based on clinical recordings, the Community Periodontal Index of Treatment Needs (CPITN) was calculated for all participants.

Microbiological examination

Plaque samples were taken from the mesio-buccal surface of the six Ramfjord teeth (16, 21, 24, 36, 41, 44).

Microbial plaque samples were taken prior the clinical measurements. After isolating with cotton rolls, drying and removal of supragingival plaque, subgingival samples were taken by means of a sterile Gracey curette, placed in 100 μl of TE buffer (Tris HCL 10 mM, EDTA 1 mM, pH 7.5) and stored after treatment with an alkali solution (0.5 M NaOH) at -20°C. A total of 1602 samples were processed for 14 bacterial species, using the ''checkerboard'' DNA–DNA hybridization technique as described in detail by Socransky et al. (1994) and elsewhere (Socransky et al. 1994, Sakellari et al. 2001) Subgingival species used for development of digoxigenin-labelled whole genomic probes were *Porphyromonas gingivalis* (FDC 381), *A. actinomycetemcomitans* serotype b (FDC Y4), *Bacteroides forsythus* (*Tannerella forsythensis*) (FDC 338), *Prevotella intermedia* (FDC 581), *Prevotella nigrescens* (VPI 8944), *Fusobacterium nucleatum* ss. *vincentii* (FDC 364), *Campylobacter rectus* (FDC 371), *Veillonella parvula* (ATCC 10790), *Peptostreptococcus micros* (FDC JH20), *Eikenella corrodens* (ATCC 23834), *Capnocytophaga sputigena* (ATCC 33612), *Streptococcus sanguis* (FDC SSI), *Streptococcus oralis* (FDC SSII) and *Actinomyces naeslundii* genospecies ll (FDC T14).

Cell numbers were quantified by comparing the signal intensities of unknowns to those of standard suspensions of 10^5 and 10^6 bacterial cells, according to the following scale: 0 = absence of signal, 1 = signal corresponding to $<10^5$ bacterial cells, 2 = signal corresponding to 10^5 bacterial cells, 3 = signal corresponding to $>10^5$ bacterial cells but $<10^6$, 4 = signal corresponding to 10^6 bacterial cells and 5 = signal corresponding to $>10^6$ bacterial cells.

Statistical analysis

The statistical analysis of the data was carried out with the statistic package SPSS, 10th version. Indicators of Descriptive Statistics were used, such as frequencies, percentage, average, variance, typical divergences, cohesive factors and relating factors. To check the hypothesis, controlling-tests of the Inductive Statistic, such as the chi-squared test and the analysis of variance (ANOVA) procedure were applied. For the comparison of percentages, the z-test was applied and the adjusted standardized residuals like the result from the chi-squared controls. The homogeneity of the variance was controlled by the Levene test. Reproducibility of clinical and microbiological assessments was tested in 15% of the subject sample.

Results

According to data analysis, the clinical and microbiological assessments were reproducible (Pearson's test, $r = 0.971$ for PD, $r = 0.989$ for PAL, κ test =0.927–0.984 for microbiological assessments, depending on the bacterial species).

The results of the present study concerning clinical data are presented in Tables 1–3. Table 1 displays descriptive statistics (means and standard deviation, with the subject as the observational unit) and comparisons for all groups and subgroups concerning clinical indices of periodontal conditions. At all age subgroups, Down syndrome patients exhibited higher indices of periodontal inflammation (mean BOP, PD and PAL) and worse levels of oral hygiene compared with healthy individuals (Table 1, ANOVA, $p = 0.000$). Statistically significant differences concerning these parameters were observed between Down syndrome and cerebral palsy patients only in the 19–28 years old subgroup. Furthermore, these differences concerned only BOP and PAL (Table 1, ANOVA, $p = 0.000$).

Additional differences in periodontal destruction were sought, with the site instead of the subject as the observational unit (Table 2). According to the present findings, Down syndrome patients, at all age subgroups, displayed lower percentages of shallow pockets (0–3 mm), and higher percentages of pockets 4–6 mm and >6 mm than healthy individuals or cerebral palsy patients (Table 2, z-test, $p<0.05$). In general, cerebral palsy patients also displayed lower percentages of shallow pockets (0–3 mm), and higher percentages of pockets 4–6 mm and >6 mm compared with healthy individuals (Table 2, z-test, $p<0.05$).

The same pattern of differences was observed for PAL, where Down syndrome patients belonging to the 13–19 and the 19–28 years old subgroups displayed statistically significant lower percentages of PAL 0–3 mm and higher percentages of

Table 1. Clinical data (mean ± SD) and comparisons of the three groups

Age	Group	*N*	HI (mean ± SD)	BI (mean ± SD)	PD (mean ± SD)	PAL (mean ± SD)
8–13	A	21	84.81 ± 17.67 (a)	62.33 ± 21.23 (a)	1.81 ± 0.26 (a)	1.78 ± 0.24 (a)
	B	27	78.73 ± 22.13 (a)	53.24 ± 24.73 (a)	1.65 ± 0.29 (a)	1.66 ± 0.29 (a)
	C	42	48.21 ± 14.10 (b)	29.52 ± 9.92 (b)	1.31 ± 0.20 (b)	1.31 ± 0.18 (b)
			$p = 0.000$ (*)	$p = 0.000$ (*)	$p = 0.000$ (*)	$p = 0.000$ (*)
13–19	A	21	88.29 ± 12.41 (a)	66.91 ± 24.82 (a)	1.86 ± 0.36 (a)	1.86 ± 0.34 (a)
	B	27	88.31 ± 13.92 (a)	55.14 ± 23.31 (a)	1.74 ± 0.25 (a)	1.79 ± 0.26 (a)
	C	44	47.84 ± 18.17 (b)	33.26 ± 17.42 (b)	1.32 ± 0.18 (b)	1.33 ± 0.17 (b)
			$p = 0.000$ (*)	$p = 0.000$ (*)	$p = 0.000$ (*)	$p = 0.000$ (*)
19–28	A	28	84.23 ± 16.72 (a)	67.94 ± 20.84 (a)	2.09 ± 0.58 (a)	2.33 ± 0.89 (a)
	B	23	92.55 ± 6.96 (a)	46.47 ± 25.66 (b)	1.73 ± 0.38 (a)	1.75 ± 0.30 (b)
	C	35	42.87 ± 18.95 (b)	26.14 ± 12.32 (c)	1.30 ± 0.15 (b)	1.30 ± 0.15 (c)
			$p = 0.000$ (*)	$p = 0.000$ (*)	$p = 0.000$ (*)	$p = 0.000$ (*)

A statistically significant difference ($p < 0.05$) was found between groups followed by different letter while no statistically significant difference ($p > 0.05$) was found between groups followed by the same letter (ANOVA) Group A, Down syndrome patients; HI, hygiene index; Group B, cerebral palsy patients; BI, bleeding index; Group C, controls; PD, probing depth; PAL, probing attachment level.

Table 2. Distribution and comparisons of different categories of PD and PAL

Age	Group	*N*	PD = 0–3 mm (%)	PAL = 0–3 mm (%)	PD = 4–6 mm (%)	PAL = 4–6 mm (%)	PD > 6 mm (%)	PAL > 6 mm (%)
8–13	A	1328	98.1 (a)	98.9 (a)	1.9 (a)	1.1 (a)	0	0
	B	1504	99.4 (b)	99.3 (a)	0.6 (b)	0.7 (a)	0	0
	C	3604	99.92 (c)	99.95 (b)	0.08 (c)	0.05 (b)	0	0
13–19	A	1376	97.4 (a)	96.6 (a)	2.6 (a)	3.3 (a)	0	0.1 (a)
	B	2296	98.5 (b)	98.35 (b)	1.4 (b)	1.61 (b)	0.1	0.04 (ab)
	C	4832	99.9 (b)	99.9 (c)	0.1 (c)	0.1 (c)	0	0 (b)
19–28	A	2377	92.8 (a)	87.1 (a)	6.8 (a)	9.2 (a)	0.4 (a)	3.7 (a)
	B	2016	97.4 (b)	97.6 (b)	2.6 (b)	2.4 (b)	0 (b)	0 (b)
	C	3844	100 (c)	100 (c)	0 (c)	0 (c)	0 (b)	0 (b)

Statistically significant differences ($p < 0.05$) between groups are indicated by different letters in parentheses while no statistical significant differences ($p > 0.05$) between groups are indicated by the same letter (*z*-test). Group A, Down syndrome patients; Group B, cerebral palsy patients; Group C, controls; PD, probing depth; PAL, probing attachment level.

Table 3. Comparisons of the CPITN between the three groups

Age	Group	*N*	CPITN					
			0 (%)	1 (%)	2 (%)	3 (%)	4 (%)	
8–13	A	21	0	66.7	20.0	13.3	0	$\chi^2 = 5.997$ $p = 0.184$ (NS)
	B	27	0	81.3	12.5	6.3	0	
	C	42	0	89.5	10.5	0	0	
13–19	A	21	7.7*	7.7*	38.5	46.2*	0	$\chi^2 = 31.075$ $p = 0.000$ (*)
	B	27	0	40.9	31.8	22.7	4.5	
	C	44	0	72.7*	25.0	2.3*	0	
19–28	A	28	0	16.7*	20.8	37.5*	25.0*	$\chi^2 = 41.752$ $p = 0.000$ (*)
	B	23	0	30.0	45.0*	25.0	0	
	C	35	2.9	74.3*	22.9	0*	0*	

*Statistically significant difference (χ^2-test, $p < 0.05$); Group A, Down syndrome patients; Group B, cerebral palsy patients; Group C, controls; CPITN, Community Periodontal Index of Treatment Needs; NS, not significant.

PAL, 4–6 mm and > 6 mm, than healthy individuals or cerebral palsy patients (Table 2, *z*-test, $p < 0.05$). These differences were not observed for the subgroup 8–13 year olds, between Down syndrome and cerebral palsy patients, which both differed from age-matched healthy individuals (Table 2, *z*-test $p < 0.05$).

The data regarding the CPITN are presented in Table 3. According to the findings, differences of treatment needs among the three groups appear in the 13–19 and 19–28 years old subgroups (Table 3, chi-squared test, $p = 0.000$). Adolescents and young adults with Down syndrome display statistically significant greater needs for intensive treatment (grades 3 and 4 of the CPITN) compared with the other two groups (Table 3, chi-squared test, $p = 0.000$).

Microbiological data from the present study are presented in Figs 1–3, each one representing a separate age subgroup. Only data of comparisons between Down syndrome patients and the two other groups are presented in the present paper. In the 8–13 years old group, statistical analysis has shown that Down syndrome patients displayed higher frequencies and levels of *T. forsythensis* and *A. naeslundii* (Fig. 1, chi-squared test, $p < 0.05$). In the 13–19 years old group, statistical analysis has shown that Down syndrome patients displayed higher frequencies and levels of: *P. gingivalis*, *A. actinomycetemcomitans*, *T. forsythensis*, *C. rectus*, *P. intermedia*, *C. sputigena* and *A. naeslundii* (Fig. 2, chi-squared test, $p < 0.05$).

Finally in the 19–28 years old Group, statistical analysis has shown that Down syndrome patients displayed higher frequencies and levels of *P. gingivalis*, *A. actinomycetemcomitans*, *T. forsythensis*, *C. rectus*, *E. corrodens*, *P. intermedia*, *P. nigrescens*, *C. sputigena*, *P. micros* and *A. naeslundii* (Fig. 3, chi-squared test, $p < 0.05$)

Discussion

The findings of the present study corroborate previous reports in the literature which conclude that Down syndrome patients display, among other things, more extensive and severe periodontal destruction compared with age-matched healthy individuals. In specific, at all age groups examined, subjects with the syndrome exhibited statistically significantly higher indices of periodontal inflammation, expressed as mean BOP, mean PD and mean PAL (Table 1, ANOVA, $p = 0.000$). Statistically significant differences were also observed when comparing the levels of oral hygiene, at all age groups, between Down syndrome patients and healthy controls (Table 1, ANOVA, $p = 0.000$). The findings of the present study definitely confirm the need to enforce oral hygiene procedures with subjects of any age group not achieving acceptable ratios of plaque-free surfaces. At this point it is worth mentioning that

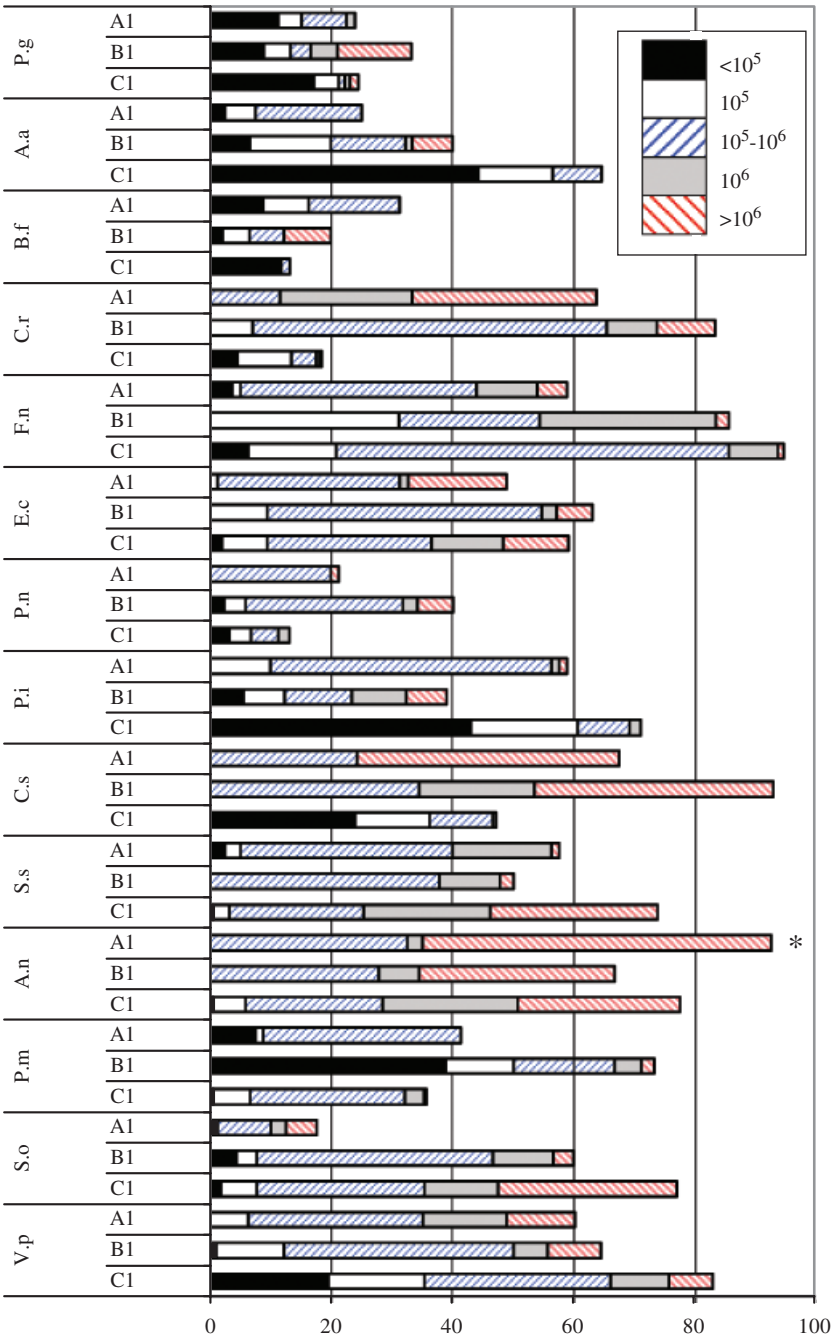

Fig. 1. Prevalence and levels of subgingival species in 8–13 year olds (children). *statistically significant higher prevalence of subgingival species in Down syndrome patients compared with the two other groups (χ^2 $p<0.05$). A1 = Down syndrome patients (8–13 years old), B1, Cerebral palsy patients (8–13 years old), C1, Controls (8–13 years old). P.g., *P. gingivalis*, A.a., *A. actinomycetemcomitans*, B.f, *B. forsythus (T. forsythensis)*, C.r, *C. rectus*, F.n., *F. nucleatum*, E.c, *E. corrodens*, P.n., *P. nigrescens*, P.i., *P. intermedia*, C.s, *C. sputigena*, S.s, *S. sanguis*, A.n., *A. naeslundii*, P.m, *P. micros*, S.o., *S. oralis*, V.p., *V. parvula*.

the subject sample in this study derived from non-institutionalized Down syndrome patients, who were not participating in any oral health program, their parents being responsible for the proper application of oral hygiene.

A similar statement applies to the cerebral palsy patients included as a second control group in the present study. These subjects did not display statistically significant differences in relation to means of hygiene index, BOP, PD and PAL compared with Down syndrome patients in the age groups 8–13 and 13–19 years old (Table 1, ANOVA, $p = 0.000$). Although no differences were observed in oral hygiene between young adults (19–28) in this category and those with Down syndrome, interestingly, subjects with the syndrome displayed worse periodontal conditions concerning BOP and PAL (Table 1, ANOVA, $p = 0.000$).

The differences in periodontal destruction, as expressed by PD and PAL among the three groups were investigated further using a second approach. Statistical analysis was performed using the site instead of the patient as the observational unit. The differences in the percentages of pockets 0–3, 4–6 and >6 mm at all age groups were thus investigated (Table 2). This approach revealed differences that could be masked by comparing mean values for patients. Specifically, periodontal destruction as indicated by the presence of pockets 4–6 mm was more pronounced among Down syndrome patients at all age-groups (Table 2, *z*-test, $p<0.05$), and this appeared to increase with age. A similar pattern applied to PAL 4–6 mm. Severe periodontal destruction as expressed by PD and PAL >6 mm was rarely observed in our subject sample, but it mainly occurred in 19–28 years old Down syndrome patients. Differences between percentages of PD and PAL >6 mm suggest the presence of recession at a substantial number of sites in this age group.

Data concerning the CPITN recorded in the present study is open to two interpretations. Firstly, it indicates that older patients with the syndrome (13–29 years old) require more intensive treatment for their entire dentition. Secondly, patients with the syndrome display statistically significant differences compared with age-matched controls concerning grades 3 and 4 of the CPITN (Table 3, $p<0.05$). Although the CPITN of patients with cerebral palsy displayed numerical differences with the other two groups, these differences did not reach statistically significant levels. Collectively, clinical data from the present study, in accordance with several previous studies, have shown that patients with Down syndrome are in dire need of having their periodontal condition monitored. Periodontal destruction increases with age and it increasingly appears as total attachment loss rather than increased pocket depth.

In the present study, further differences in the subgingival microflora of the subject sample were sought that might account for differences in perio-

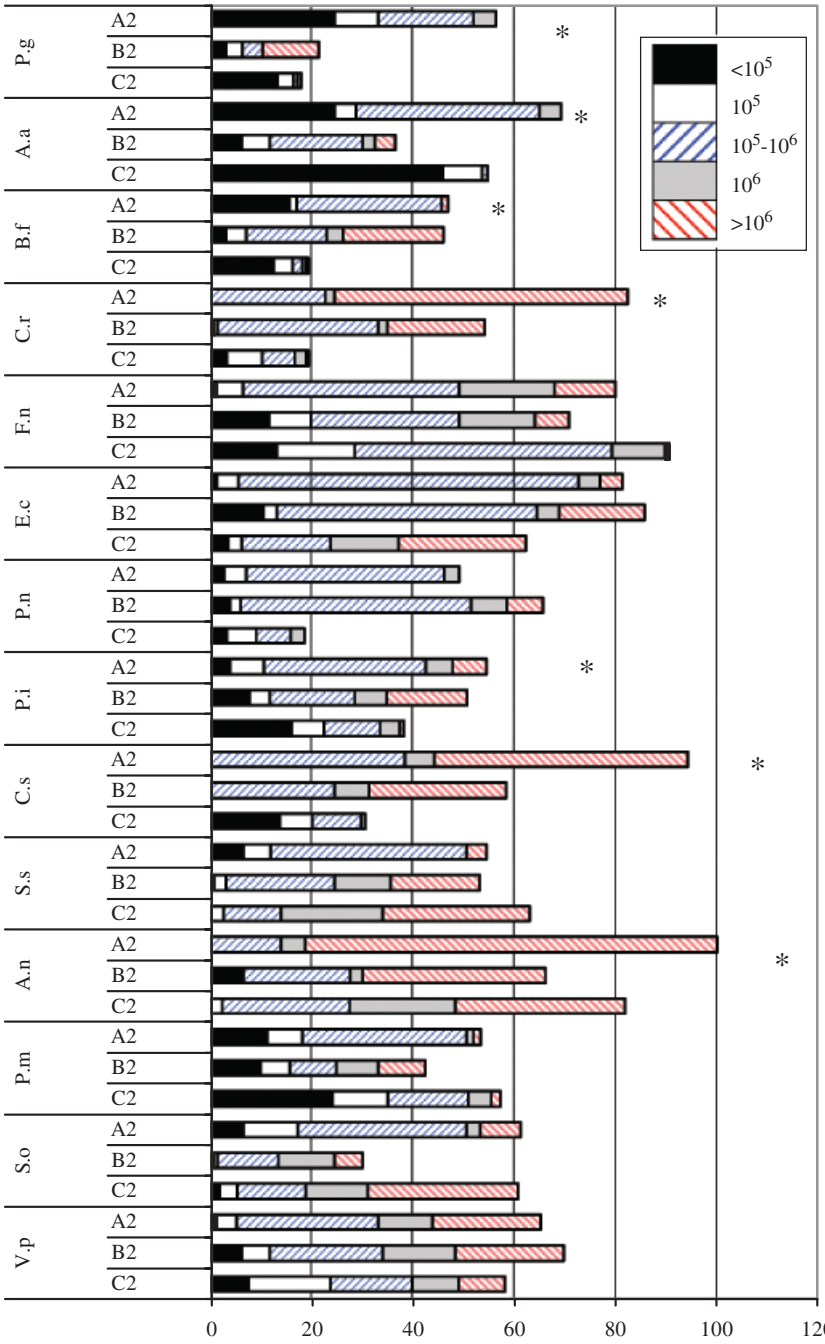

Fig. 2. Prevalence and levels of subgingival species in 13–19 year olds (adolescents). ✻statistically significant higher prevalence of subgingival species in Down syndrome patients compared with the two other groups (χ^2 $p<0.05$). A2, Down syndrome patients (13–19 years old), B2, Cerebral palsy patients (13–19 years old), C2, Controls (13–19 years old). P.g, *P. gingivalis*, A.a., *A. actinomycetemcomitans*, B.f, *B. forsythus (T. forsythensis)*, C.r, *C. rectus*, F.n., *F. nucleatum*, E.c, *E. corrodens*, P.n., *P. nigrescens*, P.i., *P. intermedia*C.s, *C. sputigena*, S.s, *S. sanguis*, A.n., *A. naeslundii*, P.m, *P. micros*, S.o., *S. oralis*, V.p., *V. parvula*.

dontal conditions. Subgingival samples were taken from the mesial surfaces of Ramfjord teeth, as indicative for the dentition generally, and analysed for presence and levels of 14 subgingival species. The vast majority of sites sampled exhibited pocket depth 0–3 mm and therefore the microbiological data shown refer only to shallow sites of the subject sample which were further analysed for statistical differences among the three groups. Since the microbiological findings of the present study refer to shallow pockets, the analysis does not allow for direct comparison with studies where differences were sought in the subgingival microflora of deep pockets of Down syndrome patients. A limited number of studies in the literature have examined the composition of microbial plaque in Down syndrome patients presenting pocket depths less than 3 mm; furthermore, these studies have only included adolescents (Barr-Agholme et al. 1992) and children (Sreedevi & Munshi 1998, Amano et al. 2000). Despite the limitations described above, in the present study direct comparisons of the microflora can be made between the three groups and all age–subgroups, as well as with increasing age.

In this study, statistically significant differences were found in the composition of subgingival dental plaque, especially between adolescents and young adults of the three groups, although no statistically significant differences were observed between individuals with the syndrome and individuals with cerebral palsy regarding hygiene levels and gingival bleeding.

Healthy individuals from all age subgroups displayed statistically significantly higher percentages of detection of *F. nucleatum*, *S. sanguis* and *S. oralis* (data not shown). These findings are in agreement with periodontal health or minor gingival inflammation mainly observed in this group of participants (Table 1).

Overall, subjects with Down syndrome, in all age subgroups, displayed statistically significantly higher percentages of detection of *T. forsythensis* and *A. naeslundii* II (Figs. 1–3, chi-squared test, $p<0.05$). In particular, in adolescents with the syndrome, statistically significantly higher prevalence was observed for *P. gingivalis*, *A. actinomycetemcomitans*, *T. forsythensis*, *C. rectus*, *P. intermedia*, *C. sputigena* and *A. naeslundii II* (Fig. 2, chi-squared test, $p<0.05$). This finding suggests that colonization with important periodontal pathogens occurs frequently in these subjects, even in adolescence. These differences persisted in young adults with the syndrome, with the addition of statistically significant differences for *E. corrodens*, *P. nigrescens* and *P. micros* (Fig. 3, chi-squared test, $p<0.05$). Taken collectively, the above data suggest that individuals with the syndrome do display differences in subgingival microflora towards more pathogenetic species, particularly with increasing age.

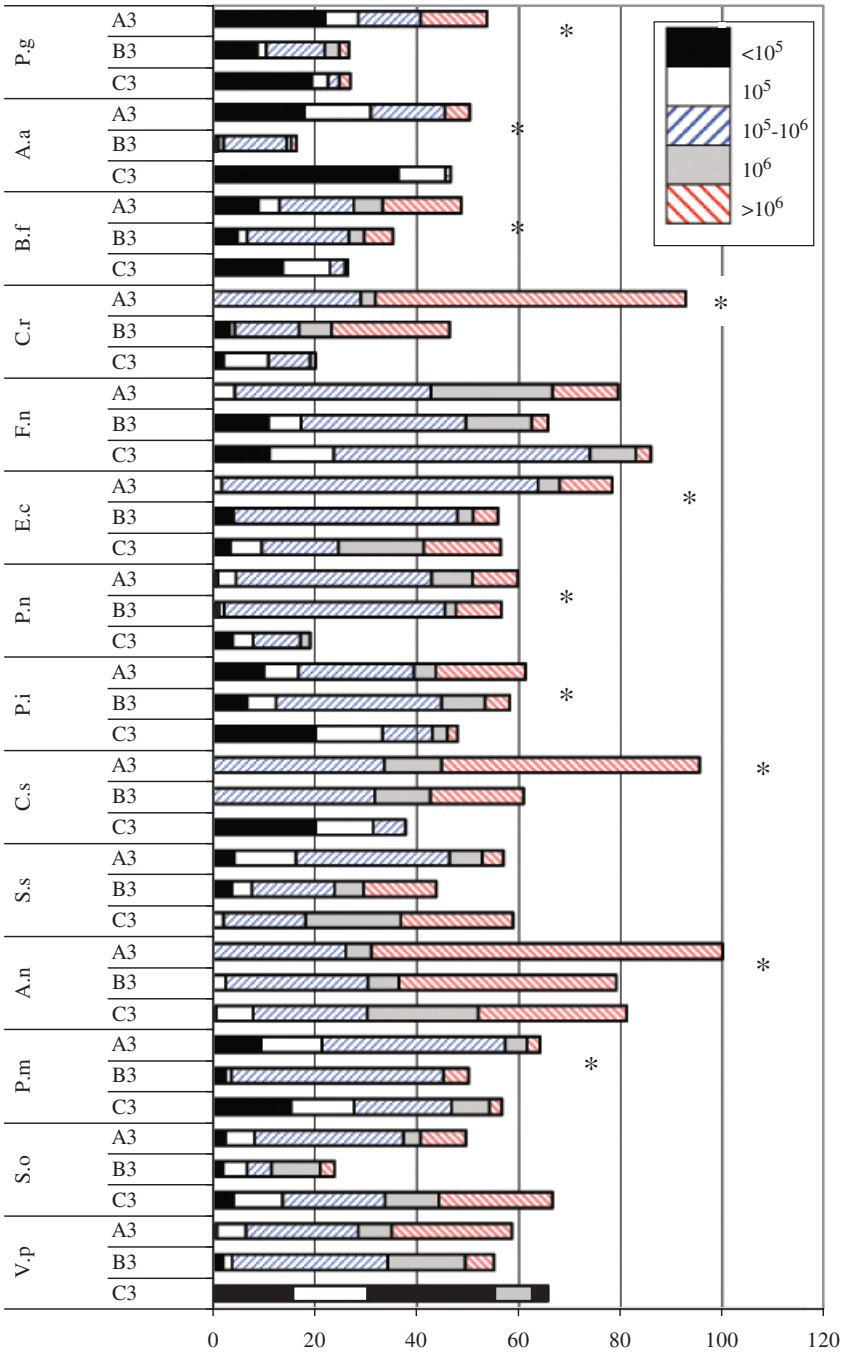

Fig. 3. Prevalence and levels of subgingival species in 19–28 year olds (young adults). ✻statistically significant higher prevalence of subgingival species in Down syndrome patients compared with the two other groups (χ^2-test, $p<0.05$). A3, Down syndrome patients (19–28 years old), B3, Cerebral palsy patients (19–28 years old), C3, Controls (19–28 years old). P.g, *P. gingivalis*, A.a., *A. actinomycetemcomitans*, B.f, *B. forsythus (T. forsythensis)*, C.r, *C. rectus*, F.n., *F. nucleatum*, E.c, *E. corrodens*, P.n., *P. nigrescens*, P.i., *P. intermedia*, C.s, *C. sputigena*, S.s, *S. sanguis*, A.n., *A. naeslundii*, P.m, *P. micros*, S.o., *S. oralis*, V.p., *V. parvula*.

A recent line of research suggests that severe periodontal destruction observed among persons with the syndrome is because of various factors mainly related to their chromosomal abnormality (Reuland-Bosma et al. 2001, Chaushu et al. 2002). In this group of patients, various abnormalities in host response have been reported, among which disorders concerning neutrophil and monocyte chemotaxis and reduced numbers and immature forms of T-lymphocytes are the most prominent (Yavuzyilmaz et al. 1993, Sreedevi & Munshi 1998). However, the issue of whether altered host response allows for easier colonization and proliferation of periodontal pathogens as reported in the present study, remains unclear.

Despite immunological defects, the important role of controlling microbial plaque in individuals with the syndrome is proven through the effectiveness of various preventive and therapeutic schemes, especially those including antiseptics (Stabholz et al. 1991b, Shapira & Stabholz 1996, Cichon et al. 1998, Hennequin et al. 1999, Sakellari et al. 2001).

Taken collectively, data from the present study suggest that periodontal disease and prevalence of important periodontal pathogens in Down syndrome patients differ from age-matched cerebral palsy patients and healthy controls. Individuals with the syndrome appear to be in dire need of having regular monitoring of periodontal conditions at any age.

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.

Amano, A., Kishima, T., Akiyama, S., Nakagawa, I., Hamada, S. & Morisaki, I. (2001) Relationship of periodontopathic bacteria with early-onset periodontitis in Down's syndrome. *Journal of Periodontology* **72**, 368–373.

Amano, A., Kishima, T., Kimura, S., Takiguchi, M., Ooshima, T., Hamada, S. & Morisaki, I. (2000) Periodontopathic bacteria in children with Down syndrome. *Journal of Periodontology* **71**, 249–255.

Barr-Agholme, M., Dahllof, G., Linder, L. & Modeer, T. (1992) *Actinobacillus actinomycetemcomitans*, *Capnocytophaga* and *Porphyromonas gingivalis* in subgingival plaque of adolescents with Down's syndrome. *Oral Microbiology and Immunology* **7**, 244–248.

Chaushu, S., Yefenof, E., Becker, A., Shapira, J. & Chaushu, G. (2002) Severe impairment of secretory Ig production in parotid saliva of Down syndrome individuals. *Journal of Dental Research* **81**, 308–312.

Cichon, P., Crawford, L. & Grimm, W. D. (1998) Early-onset periodontitis associated with Down's syndrome – clinical interventional study. *Annals of Periodontology* **3**, 370–380.

Desai, S. S. (1997) Down syndrome: a review of the literature. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics* **84**, 279–285.

Gabre, P. (2000) Studies on oral health in mentally retarded adults. *Swedish Dental Journal* **142** (Suppl.), 1–48.

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.

Genco, R. J. & Loe, H. (1993) The role of systemic conditions and disorders in periodontal disease. *Periodontology 2000* **2**, 98–116.

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.

Hennequin, M., Faulks, D., Veyrune, J. L. & Bourdiol, P. (1999) Significance of oral health in persons with Down syndrome: a literature review. *Developmental Medicine and Child Neurology* **41**, 275–283.

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.

Modeer, T., Barr, M. & Dahllof, G. (1990) Periodontal disease in children with Down's syndrome. *Scandinavian Journal of Dental Research* **98**, 228–234.

Reuland-Bosma, W., van der Reijden, W. A. & van Winkelhoff, A. J. (2001) Absence of a specific subgingival microflora in adults with Down's syndrome. *Journal of Clinical Periodontology* **28**, 1004–1009.

Reuland-Bosma, W. & van Dijk, J. (1986) Periodontal disease in Down's syndrome: a review. *Journal of Clinical Periodontology* **13**, 64–73.

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.

Santos, R., Shanfeld, J. & Casamassimo, P. (1996) Serum antibody response to *Actinobacillus actinomycetemcomitans* in Down's syndrome. *Special Care in Dentistry* **16**, 80–83.

Shapira, J. & Stabholz, A. (1996) A comprehensive 30-month preventive dental health program in a pre-adolescent population with Down's syndrome: a longitudinal study. *Special Care in Dentistry* **16**, 33–37.

Socransky, S. S., Smith, C., Martin, L., Paster, B. J., Dewhirst, F. E. & Levin, A. E. (1994) ''Checkerboard'' DNA–DNA hybridization. *BioTechniques* **17**, 788–792.

Sreedevi, H. & Munshi, A. K. (1998) Neutrophil chemotaxis in Down syndrome and normal children to *Actinobacillus actinomycetemcomitans*. *The Journal of Clinical Pediatric Dentistry* **22**, 141–146.

Stabholz, A., Mann, J., Sela, M., Schurr, D., Steinberg, D. & Shapira, J. (1991a) Caries experience, periodontal treatment needs, salivary pH, and *Streptococcus mutans* counts in a preadolescent Down syndrome population. *Special Care in Dentistry* **11**, 203–208.

Stabholz, A., Shapira, J., Shur, D., Friedman, M., Guberman, R. & Sela, M. N. (1991b) Local application of sustained-release delivery system of chlorhexidine in Down's syndrome population. *Clinical Preventive Dentistry* **13**, 9–14.

Yavuzyilmaz, E., Ersoy, F., Sanal, O., Tezcan, I. & Ercal, D. (1993) Neutrophil chemotaxis and periodontal status in Down's syndrome patients. *The Journal of Nihon University School of Dentistry* **35**, 91–95.

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