Tooth Sensitivity in Fluorotic Teeth

Mine Ozturk Tonguc^a Yener Ozat^a Tugba Sert^b Yonca Sonmez^c F. Yesim Kirzioqlu^a

ABSTRACT

Objectives: The aim of this study was to compare the demographic and clinical features of tooth sensitivity (TS) in subjects with and without fluorosis.

Methods: A total of 2249 subjects (378 subjects with fluorosis and 1871 subjects without fluorosis) were examined for TS during a study period of one year and TS was determined in 122 subjects. The level of TS was evaluated on a visual analogue scale (VAS). The sensitivity evaluation was made by applying tactile and cold air stimuli. In teeth sensitive to any stimuli, the plaque index (PI), gingival index (GI), gingival recession (GR) and periodontal pocket depth (PPD) were recorded. Fluorosis was assessed using the Dean Index.

Results: One hundred and twenty-two participants were found to have TS (5.42%). The frequency of TS in subjects with fluorosis was 9.26%, while the frequency of TS in subjects without fluorosis was 4.65%. There was a statistically significant difference between the groups in terms of TS frequency (P=0.0003). In contrast, there were no significant differences between the groups for periodontal parameters except PI.

Conclusions: The results of the study showed that the subjects with fluorosis may have been suffering from TS more than the subjects with normal dentition. Further studies are necessary to determine the factors that contribute to sensitivity of teeth with fluorosis. (Eur J Dent 2011;5:273-280)

Key words: Fluorosis; Tooth sensitivity; Visual analogue scale; Epidemiology.

 Suleyman Demirel University, Faculty of Dentistry, Department of Periodontology, Isparta, Turkey.

- ^b Private practice, Antalya, Turkey.
- ^c Suleyman Demirel University Faculty of Medicine Department of Public Health, Isparta, Turkey.
- Corresponding author: Mine Ozturk Tonguc Suleyman Demirel University, Faculty of Dentistry, Department of Periodontology, 32260, Cunur, Isparta, Turkey.
 Phone: +90 246 2118799
 Fax: +90 246 2370607
 E-mail: mineperio@gmail.com

INTRODUCTION

Tooth sensitivity (TS) is an exaggerated response to a sensory stimulus that usually causes no response in a normal, healthy tooth. It is a source of chronic irritation that affects eating, drinking and breathing. Increased hypersensitivity hinders the ability to control dental plaque and compromises oral health.¹ The chief symptom of tooth sensitivity is rapid, sharp pain against tactile (i.e. tooth brushing), thermal (hot or cold) and chemical (acids and sweet) stimuli, as well as exposure to air.¹

July 2011 - Vol.5

Tooth quality relates to the tooth's ability to fulfil its function and is evaluated by measuring mechanical and structural properties of tooth material.² It was demonstrated in studies conducted on teeth with molar incisor hypomineralization that the mechanical and structural properties of tooth material are also related to other tooth characteristics such as TS.^{3,4} Different properties of teeth and the effects of intrinsic and extrinsic factors on tooth quality have been investigated in several studies.⁵⁻¹⁰ Despite the caries preventive effectiveness of fluoride, it was found to have some negative effects on tooth quality.²

Dental fluorosis is a common disorder of teeth associated with high fluoride intake, especially from drinking water containing high concentrations of fluoride. The adverse effect of excessive exposure to fluoride is dental fluorosis, which is a permanent hypomineralization in the subsurface of enamel, characterized in its mildest form by small, clearly visible, white flecks found on the cusp tips and on facial surfaces of permanent dentition. Fluorosis is mostly found on permanent teeth surfaces ranging from obvious white opaque areas (moderate form) to darkly stained and pitted enamel (severe form).¹¹

It is frequently claimed that it is very difficult to discriminate between dental fluorosis and other enamel disturbances. The generalized nature of dental fluorosis within the dentition and over entire tooth surfaces makes it easy to distinguish fluoride-induced enamel changes from other defects.¹²

The teeth affected by severe dental fluorosis suffer from post-eruptive enamel breakdown. The effect of fluoride on forming enamel results in a number of changes. These changes in the structure of enamel involve increased porosity, higher protein levels, and lower amounts of minerals and, in severe cases, the formation of a pitted surface.¹³ With increasing severity, the surface and subsurface of enamel become more hypomineralized and the tooth becomes increasingly porous. The most severe change described is a subsurface hypomineralization lesion which extends towards the inner enamel and enamel-dentin junction.¹⁴ In a related study, a positive correlation between dentin fluoride concentration and dentin tubule size was shown, demonstrating wider dentin tubules in teeth with higher levels of fluoride concentration in the dentin. It was also shown that the high fluoride content of the tooth decreased the mineralization rate in the tooth's structure.²

Hypomineralized teeth frequently have ex-

The aim of this study was to compare the demographic and clinical features of tooth sensitivity between subjects with and without fluorosis.

MATERIALS AND METHODS

The subjects of this study were selected from patients referred to the Department of Periodontology of the Faculty of Dentistry at Suleyman Demirel University, in Isparta. The city of Isparta is one of the endemic fluorosis areas of Turkey and the city is situated on a volcanic region. According to Local Health Organization data, the mean fluoride level in drinking water was 2.74 ± 0.64 ppm in Isparta in the year 2000.¹⁵

The study was conducted between May 1, 2006 and May 1, 2007. Approval of the Ethical Investigation Committee from the Faculty of Medicine at Suleyman Demirel University was received prior to the beginning of the study.

Inclusion criteria for subjects were:

1. informed consent for participation in the study,

2. no systemic diseases,

3. no orthodontic appliances,

4. no history of any disease requiring drugs such as analgesics, tranquilizers or mood altering medication,

5. no history of periodontal treatment in last six months,

6. no usage of desensitizing tooth paste or mouth rinse in the last six weeks,

7. no history of any restorative dental treatment in the last month,

8. no presence of acute toothache.

The patients who fitted the inclusion criteria and who gave written consent were recruited for a preliminary screening. A total of 5130 subjects applied to the periodontology clinic in the one year study period and 2884 of them were excluded from the preliminary screening because they did not match the inclusion criteria.

The dentitions of 2249 subjects who fitted the inclusion criteria were examined for fluorosis by the Dean Index (DI).¹⁶ Subjects with dental fluorosis were included in the fluorosis group and subjects with normal dentition were included in the non-fluorosis group.

Afterwards, each subject recruited to the preliminary screening was questioned: 'Have you any sensitivity to hot and/or cold foods, cold air, brushing, or sweet and/or sour foods in your teeth in your daily life?' Six hundred and forty-five subjects answered this question positively. These subjects were asked to quantify their sensitivity levels by making a mark on the visual analogue scale (VAS). A 10 cm horizontal line, equally divided into 10 levels, was used for the VAS. On this scale, the starting point (0) represented no pain whilst the end point (10) stood for unbearable pain.¹⁷ The distance between 0 and the point marked by subjects was measured with a ruler and recorded for the tooth sensitivity that was sensed by the subjects in their daily lives.

After the subjects quantified their daily life tooth sensitivity using the VAS, their teeth were clinically examined. All teeth present in the mouth except those showing any exclusion criteria were assessed for fluorosis and tooth sensitivity. The subjects who had at least one tooth that responded positively to a cold air or tactile stimulus test were identified as having TS and these subjects were recruited to the sensitivity study.

Information such as age, gender and smoking habits of the subjects included in the study were recorded. The subjects were also questioned about the initiating factors (hot/cold foods or drinks, sour/sweet foods, cold air, brushing or a combination of these factors) of TS.

The exclusion criteria for teeth were:

- 1. having had a crown restoration,
- 2. having cracks or fractures in the enamel,
- 3. having caries,
- 4. having had any type of restoration,

5. having endodontic problems (sensitive to vertical percussion),

6. having non-carious cervical lesions in the enamel,

7. abutment teeth for dentures,

8. having clinical attachment loss of more than 3 mm

9. third molars.

A total of 854 teeth (35%) were excluded from the study because they had one or more of the exclusion criteria; 1586 were evaluated for sensitivity.

The sensitivity levels of the teeth were evaluated utilizing tactile and cold air stimuli. Tactile sensitivity was assessed using a William's periodontal probe, which was applied perpendicular to the cervical surface of each tooth and the tip of the probe was used to scratch the surface in a horizontal direction. Ten minutes after the tactile stimulus, the patient's response to a cold air stimulus was assessed using a blast of cold air from a triple syringe applied approximately 5 mm from and perpendicular to the tooth's surface, whilst isolated from neighbouring teeth, for 3 seconds.^{18,19} Immediately after each application, the patients were asked to qualify their sensitivity using the VAS for each tooth. The points marked by the subjects were measured with a ruler and recorded as tactile stimulus VAS and cold air stimulus VAS, respectively, for each tooth.

For each tooth that was sensitive to either of the stimuli given, the plaque index (PI),²⁰ gingival index (GI),²¹ gingival recession (GR) and periodontal pocket depth (PPD) were recorded from the mid-buccal surface. The fluorosis status of the teeth was assessed using the Dean Index (DI),¹⁶ which is a recommended method of evaluation in prevalence studies regarding fluorosis,^{22,23} and it remains the gold standard index in the public health armamentarium.²³

The same experienced investigator (MOT) performed all VAS evaluations and clinical examinations of all subjects on their first visit. Prior to the actual data collection, 10 subjects were randomly selected and used to calibrate the investigator. The investigator evaluated these subjects on two separate occasions, 48 h apart. Calibration was accepted if the PPD measurements at baseline and at 48 h were similar to the millimeter level for > 90% of the evaluations.

Statistical analysis

Descriptive statistics including means, standard deviations and frequency distributions were constructed using a statistical package software program (SPSS 13.0, SPSS Inc. IL, USA). Each subject was assessed as an experimental unit. The mean values of the VAS, PI, GI, PPD and GR measurements of the teeth were calculated for each subject. Since DI is a categorical classification, the median DI score of all teeth was used for each subject and the frequency data were determined.

The chi-square test (χ^2) was used to compare the presence of tooth sensitivity, gender distribution and smoking status of the groups. Comparisons of all of the other parameters were made between the groups using the independent samples t test; P<.05 was accepted as the level of statistical significance.

Pearson's correlation coefficient (r) was used to determine correlations between the VAS parameters.

RESULTS

In the preliminary screening, a total of 2249 subjects fitted the inclusion criteria and 378 of these were determined as having dental fluorosis (fluorosis group), while 1871 of them had a normal dentition (non-fluorosis group).

The comparisons of the fluorosis and non-fluorosis groups regarding the number of subjects and the number of sensitive teeth and the frequencies of TS are presented in Table 1. There was a statistically significant difference between the groups according to the frequency of TS (P<.001 χ^2 =13.05). When compared to the non-fluorosis group, the percentage of subjects with TS was significantly higher in the fluorosis group (P<.001).

However, although 645 subjects (28.7%) answered the question 'Have you any sensitivity to hot and/or cold foods, cold air, brushing, or sweet and/or sour foods in your teeth in your daily life?' positively, TS was only found in 122 subjects (5.42%) in the clinical examination, and these were subjected to the sensitivity study.

The number of sensitive teeth per subject was higher in the fluorosis group and this difference was statistically significant (P<.001).

There was no statistically significant difference between the groups concerning mean VAS values

(P>.05). The mean VAS values of the groups are given in Table 2.

There were positive correlations between the mean VAS scores of tactile and cold air stimuli in both the fluorosis group (r=0.424 and P=0.000) and in the non-fluorosis group (r=0.502, P=0.000).

The characteristics of the subjects in each of the groups are given in Table 3. Out of all of the subjects, tooth sensitivity was most prevalent in those aged between 30-39 years old (41 subjects, 33.6%). The most frequently affected teeth were premolars and the most common initiating factor was hot and/or cold food. Out of all of the participants, 9% had at least two or more initiating factors for TS. The ratios of the initiating factors of TS in the participants were hot and/or cold food (34.4%), sweet and/or sour foods (25.4%), cold air (21.4%), and brushing (9.8%).

Because Isparta is an endemic fluorosis region, the subjects who were in the fluorosis group had dental fluorosis in all of the teeth in their mouths, at various levels of severity. The median and the most prevalent (79 teeth, 41.6%) DI score for the fluorosis group was 3 (min 1, max 4).

There were no statistically significant differences between the groups concerning the means of age or gender, whereas a smoking habit was

Table 1. The number of subjects, the number of sensitive teeth, the mean number of sensitive teeth and the frequency of tooth sensitivity in the groups.

	Subject N (%)	Subject with TS N (%)	Examined teeth n (%)	Sensitive teeth n (%)	Sensitive teeth (mean± sd)	TS (%)
Total	2249 (100)	122 (100)	1586 (100)	660 (41.61)	5.38±2.27	5.42
Fluorosis group	378 (16.8)	35 (28.68)	296 (18.66)	190 (64.18)	5.40±2.45	9.26
Non-fluorosis group	1871 (83.2)	87 (71.31)	1290 (81.34)	470 (36.43)	5.37±1.75	4.65
Р	N/A	N/A	N/A	0.0008***	NS	0.0003***

N: number of subjects, n: number of teeth, sd: standard deviation, TS: tooth sensitivity, ***: P<.001 N/A: not applicable, NS: Not significant.

Table 2. Visual Analog Scale (VAS) values of the groups.

	VAS	VAS	VAS
	(mean±sd)	(mean±sd)	(mean±sd)
Total	5.49±1.5	4.66±1.52	4.78±1.48
Fluorosis	5.37±1.53	4.75±1.03	4.71±1.05
Non-Fluorosis	5.54±1.56	4.63±1.69	4.81±1.63
Р	NS	NS	NS

NS: not statistically significant

 Table 3. The characteristics of the subjects with TS in the groups.

Group	Subject with TS (n)	Age (mean±sd)	Gender (F / M)	Smokers n (%)
Fluorosis	35	36.5±13.03	24 / 11	15 (42.9)
Non-flourosis	87	35.4±8.17	55 / 32	17 (19.5)
Total	122	36.2±11.8	79 / 43	32 (26.2)
Ρ	N/A	NS	NS	0.009*

n: number of subjects, sd: standard deviation, TS: tooth sensitivity, F: Female, M: Male, N/A: not applicable, NS: Not significant, *: P<.05

European Journal of Dentistry

significantly higher in the fluorosis group (P<.05). The comparisons of the mean values of the clinical periodontal parameters of the groups are presented in Table 4.

There was no statistically significant difference between the groups for the clinical periodontal parameters, except for PI. The mean PI value in the non-fluorosis group was significantly higher compared to that in the fluorosis group (P=0.017) (Table 4).

DISCUSSION

To the best of our knowledge, this was the first study to be conducted on the presence and clinical features of TS in subjects with fluorosis.

The principal findings of this study were the higher TS frequency and number of sensitive teeth per subject in the fluorosis group compared to the non-fluorosis group. These results may be related to the effects of fluorosis on the structure of teeth. Mild to moderate enamel fluorosis makes the enamel more resistant to dental caries. However, recent research revealed that a systemic fluoride intake could have the opposite effect on dentin, making dentin more susceptible to dental caries and other defects such as tooth fractures. Dentin fluorosis has been found to distort the intertubular collagen network in dentin, thereby causing detrimental hypermineralization of dentin, resulting in a higher susceptibility to acid degradation.²⁴ In addition, Rochas-Sanchez et al²⁵ reported that the dentin in fluorotic teeth was characterized by a highly mineralized sclerotic pattern when compared to healthy teeth or fluorotic enamel lesions. In response to the effects of severe fluorosis in the enamel, the dentin showed hypermineralization, as seen in other enamel disorders. Furthermore, it was also shown that there was a positive correlation between the dentin fluoride concentration and the dentin tubule size, demonstrating wider dentin tubules in teeth with higher levels of fluoride in the dentin.²

The cause of the higher prevalence of TS in the fluorosis group may be associated with the changes in dentin tubule size and the both the enamel and dentin. But histological alterations in the teeth related to fluorosis were not investigated in this study.

Jälevik and Klingberg³ reported hypersensitivity in teeth with molar incisor hypomineralization. In another study, inflammatory changes were observed in the pulp of hypomineralized teeth exhibiting enamel loss and it was hypothesized that subclinical pulpal inflammation could lead to hypersensitivity.⁴ In addition, it was suggested that a bacterial invasion of the dentine tubules, causing an inflammatory response in the pulp, could contribute to the hypersensitivity of hypomineralized teeth.²⁶ This situation could apply to fluorotic teeth due to the hypomineralization and dentin tubule widening of these teeth. However, the presence of this kind of inflammation in fluorotic teeth has not yet been reported.

Studies using a questionnaire approach with the patients self-reporting their sensitivity levels without any subsequent clinical examination are likely to grossly overestimate the prevalence, as the sensitivity reported could be the result of a number of different pathologies.³ Actually, in our study, the number of subjects who positively answered the question 'Have you any sensitivity to hot and/or cold foods, cold air, brushing, or sweet and/or sour foods in your teeth in your daily life?' was 645 (28.7%). However, the number of subjects with clinically determined TS was only 122 (5.4%).

Dentin hypersensitivity (DH), which cannot be ascribed to any other form of dental defect or pathology, has been typically described as a 'short, sharp pain' arising from exposed dentin in response to thermal, evaporative, tactile, osmotic or chemical stimuli.¹⁷ Studies regarding patients of periodontology clinics indicated that the prevalence of DH was higher compared to the general dental population.^{17,18,27-29} The results of these studies showed that the prevalence of DH ranged from 60.3% to 98%. Periodontitis and periodontal treatment results in gingival recession and increases DH.^{17,30} In order to discriminate between TS and DH, the participants in our study were not periodontally treated before recording their clini-

Table 4. The comparisons of the mean values of clinical periodontal parameters of the groups.

Group	GI (mean±sd)	PI (mean±sd)	GR (mean±sd)	PPD (mean±sd)
Fluorosis	1.54±0.41	1.55±0.39	1.31±0.41	1.63±0.27
Non-flourosis	1.62±0.43	1.75±0.43	1.27±0.41	1.62±0.28
Total	1.59±0.43	1.69±0.43	1.29±0.41	1.63±0.28
Р	NS	0.017*	NS	NS

GI: Gingival index, PI: Plaque index, GR: Gingival recession, PPD: Probing pocket depth, NS: Not significant, *: P<.05

July 2011 - Vol.5

European Journal of Dentistry

cal measurements in order to eliminate a possible increase in sensitivity due to periodontal therapy. In addition, in order to eliminate the negative effects of a periodontal breakdown, the teeth with a clinical attachment loss of more than 3 mm were also excluded from our study.

The frequency of TS in the fluorosis group was 9.26%, which was significantly higher than the prevalence noted in the non-fluorosis group in this study (P=0.0003) and also higher than the DH prevalence of the general dental populations noted in other studies.^{17,31,32}

In the present study, the participants were asked what the initiating factor for their TS was and they were allowed to make a choice of one or more from; hot or cold foods, sour or sweet foods, cold air, and brushing. The most reported initiating factor was hot or cold food in both groups. Similarly, cold was the provoking factor that was most frequently cited in the literature.^{18,27,31-35} The second most prevalent provoking factor reported in the literature was heat.^{17,33,34,36}

The PI of the fluorosis group was found to be lower than those of the non-fluorosis group. This result is consistent with the results of our previous study.³⁷ It was found that plaque accumulation, gingival bleeding and inflammation were lower in subjects with fluorosis who were resident in Isparta compared to subjects with normal dentition who were resident in Konya, which is a non-fluorosis area in Turkey.³⁷ Similarly, it was reported that as the concentration of fluoride in drinking water increased, plaque accumulation on tooth surfaces decreased.³⁸ Moreover, it was shown that high level of fluoride in dentifrices reduces de novo plague formation on tooth surfaces³⁹ because high fluoride concentrations inhibit the metabolic and physiological pathways of biofilms.40,41 For this reason, although the enamel surfaces of fluorotic teeth have a high porosity, the amount of plaque deposited on these surfaces is lower than on nonfluorotic enamel surfaces.

In our study, the percentage of subjects in the fluorosis group who smoked was higher than in the non-fluorosis group (P=0.009). However, there was no difference between the groups regarding GR. Smoking is known to be a major risk factor for periodontal disease and attachment loss. There were conflicting results in the literature regarding the effect of smoking on DH. Some studies reported a higher frequency of gingival recession and dentin sensitivity in smokers.^{16,32} However, other studies did not support a relationship between smoking and DH.^{42,43} In the present study,

the higher number of smokers in the fluorosis group compared to the non-fluorosis group may be coincidental.

There were a number of limitations to our study. Since the study population was formed from subjects referred to the periodontology clinic, the sample in our study did not represent the general population. For this reason, the prevalence of TS in subjects with fluorosis was not determined; only the frequency of TS was determined in this population.

As tooth sensitivity is a subjective symptom that may vary between individuals, each subject was treated as an experimental unit in this study. However, in this kind of study, it would be better if each tooth was the experimental unit instead.

There were difficulties in discussing the results of this study. No data were found in the literature regarding TS in fluorotic or normal dentition. The dental literature contained data about dentin hypersensitivity (DH) in normal dentition and TS with molar incisor hypomineralization (MIH). For this reason, the results of the present study were discussed in light of the studies on DH and MIH.

CONCLUSIONS

The frequency of TS in subjects with fluorosis was higher than in subjects without fluorosis. The results of the study suggest that the subjects with fluorosis may have been suffering more from TS than the subjects with normal dentition. Further studies that use each tooth as an experimental unit, preferably considering the degree of pulpal inflammation, are needed in order to evaluate the effects of fluorosis severity on TS.

REFERENCES

- 1. Bissada NF. Symptomatology and clinical features of hypersensitive teeth. *Archs Oral Biol* 1994;39,Suppl:31S-32S.
- Vieira APGF, Hancock R, Dumitriu M, Limeback H, Grynpas MD. Fluoride's effect on human dentin ultrasound velocity (elastic modulus) and tubule size. *Eur J Oral Sci* 2006;114:83-88.
- Jälevik B, Klingberg GA. Dental treatment, dental fear, and behavior management problems in children with severe enamel hypomineralization of their first permanent molars. *Int J Pediatr Dent* 2002;12:24-32.
- Rodd HD, Boissonade FM, Day PF. Pulpal status of hypomineralized permanent molars. *Pediatr Dent* 2007;29:514-520.

Tonguc, Ozat, Sert, Sonmez, Kirzioglu

- Wang R, Weiner S. Human root dentin: structural anisotropy and Vickers microhardness isotropy. *Connect Tissue Res* 1998;39:269-279.
- Watts DC, El Mowafy OM, Grant AA. Temperature dependence of compressive properties of human dentin. J Dent Res 1987;66:29-32.
- Mahoney E, Holta A, Swain M, Kilpatrick N. The hardness and modulus of elasticity of primary molar teeth: an ultramicro-indentation study. *J Dent* 2000;28:589-594.
- Kinney JH, Gladden JR, Marshall GW, Marshall SJ, So JH, Maynard JD. Resonant ultrasound spectroscopy measurements of the elastic constants of human dentin. *J Biomech* 2004;37:437-441.
- Kruzic JJ, Nalla RK, Kinney JH, Ritchie RO. Crack blunting, crack bridging and resistance-curve fracture mechanics in dentin: effect of hydration. *Biomaterials* 2003;24:5209-5221.
- Arola D, Reprogel RK. Effects of aging on the mechanical behavior of human dentin. *Biomaterials* 2005;26:4051-4061.
- 11. Den Besten PK. Mechanism and timing of fluoride effects on developing enamel. *J Public Health Dent* 1999;59:247-251.
- Fejerskov O, Richards A, DenBesten P. The effect of fluoride on tooth mineralization. In Fejerskov O, Ekstrand J, Burt BA. (Ed.) Fluoride in Dentistry Munksgaard, Copenhagen, 1996, 2nd ed., pp.112-146.
- Lyaruu DM, Bervoets TJM, Bronckers ALJJ. Short exposure to high levels of fluoride induces stage-dependent structural changes in ameloblasts and enamel mineralization. *Eur J Oral Sci* 2006;114(Suppl. 1):111-115.
- Fejerskov O, Larsen MJ, Richards A, Baelum V. Dental tissue effects of fluoride. *Adv Dent Res* 1994;8:15-31.
- Yıldız M, Oral B. Effects of menopause on bone mineral density in women with endemic fluorosis. *Clin Nuc Med* 2003;28:308-311.
- Dean H. T. The investigation of physiological effects by the epidemiological method. In Moulton FR (ed). Fluorine and dental health, Washington: AAAS; 1942. p. 23
- Al-Wahadni A., Linden GJ. Dentine hypersensitivity in Jordanian dental attenders. A case control study. J Clin Periodontol 2002;29:688-693.
- 18. Rees JS. The prevalence of dentine hypersensitivity in general dental practice in the UK. *J Clin Periodontol* 2000;27:860-865.
- 19. Rees JS, Jin LJ, Lam S, Kudanowska I, Vowles R. The prevalence of dentin hypersensitivity in a hospital clinic population in Hong Kong. *J Dent* 2003;31:453-461.
- Silness J, Löe H. Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. *Acta Odontol Scand* 1964;22:121-135.

- 21. Löe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontol Scand* 1963;21:533-551.
- 22. Medina-Solis CE, Pontigo- Loyola, Maupome G, Lamadrid-Figueroa H, Loyola-Rodriguez JP, Hernandez- Romano J, Villalobos- Rodelo JJ, Marquez-Corona ML. Dental fluorosis prevalence and severity using Dean's index based on six teeth and on 28 teeth. *Clin Oral Invest* 2008;12:197-202.
- 23. Fluorides and oral health. Geneva: World Health Organization; 1994. WHO Technical Report Series, No. 846.
- 24. Waidyasekera PG, Nikaido T, Weerasinghe DDS, Wettasinghe KA, Tagami J. Caries susceptibility of human fluorosed enamel and dentine. *J Dent* 2007;35:343-349.
- 25. Rojas-Sanchez F. Dentin in severe fluorosis: a quantitative histochemical study. *J Dent Res* 2007;86:857-861.
- 26. Fagrell TG, Lingström P, Olsson S, Steiniger F, Noren J. Bacterial invasion of dentinal tubules beneath apparently intact but hypomineralized enamel in molar teeth with molar incisor hypomineralization. *Int J Paediatr Dent* 2008;18:333-340.
- Dababneh RH, Khouri AT, Addy M. Dentine hypersensitivity an enigma? A review of terminology, epidemiology, mechanisms, aetiology and management. *Br Dent J* 1999;187:606-611.
- Chabanski MB, Gilliam DG, Bulman JS, Newman HN. The prevalence, distribution and severity of cervical dentine sensitivity (CDS) in a population of patients referred to a specialist periodontology department. J Clin Periodontol 1996;23:989-992.
- Taani Q, Awartani F. Clinical evaluation of cervical dentin sensitivity (CDS) in patients attending general dental clinics (GDC) and periodontal specialty clinics (PSC). J Clin Periodontol 2002;29:118-122.
- Addy M, Mostafa P, Newcombe RG. Dentine hypersensitivity: the distribution of recession, sensitivity and plaque. J Dent 1987;15:242-248.
- Bamise CT, Olusile AO, Oginni AO, Dosumu OO. The prevalence of dentin hypersensitivity among adult patient attending Nigerian teaching hospital. *Oral Health Prev Dent* 2007;5:49-53.
- 32. Rees JS, Addy M. A cross-sectional study of dentine hypersensitivity. *J Clin Periodontol* 2002;29:997-1003.
- Flynn J, Galloway R, Orchardson R. The incidence of 'hypersensitive' teeth in the West of Scotland. *J Den*t 1985;13:230-236.
- Fisher C, Fischer G, Wennberg A. Prevalence and distribution of cervical dentine hypersensitivity in a population in Rio de Janeiro, Brazil. J Dent 1992;20:272-276.
- Gillam DG, Seo HS, Newman HN, Bulman JS. Comparison of dentine hypersensitivity in selected occidental and oriental populations. *J Oral Rehabil* 2001;28:20-25.

```
European Journal of Dentistry
```

□ Tooth sensitivity in fluorotic teeth

- Kanapka JA, Colucci SV. Clinical evaluation of dentinal hypersensitivity: a comparison of methods. *Endod Dent Traumatol* 1986;2:157-164.
- Bozkurt FY, Gürsel M, Erdemir E, Fentoğlu Ö, Kıran M, Güngör İ. The clinical evaluation of the effects of fluorosis on periodontal condition. *AÜ Diş Hek Fak Derg* 2000;27:215-225. (Article in Turkish).
- Anuradha KP, Chadrashekar J, Ramesh N. Prevalence of periodontal disease in endemically fluorosed areas of Davangere Taluk, India. *Indian J Dent Res* 2002;13:15-19.
- Nordström A, Mystikos C, Ramberg P, Birkhed D. Effect on de novo plaque formation of rinsing with toothpaste slurries and water solutions with a high fluoride concentration (5,000 ppm). *Eur J Oral Sci* 2009;117:563-567.
- 40. Van Loveren C. Antimicrobial activity of fluoride and its in vivo importance: identification of research questions. *Caries Res* 2001;35 suppl 1:65-70.
- Aspiras M, Stoodley P, Nistico L, Longwell M, de Jager M. Clinical implications of power toothbrushing on fluoride delivery: Effects on biofilm plaque metabolism and physiology. *Int J Dent* 2010; Article ID 651869, doi:10.1155/2010/651869.
- Rees JS, Addy M. A cross-sectional study of buccal cervical sensitivity in UK general dental practice and a summary review of prevalence studies. *Int J Dent Hygiene* 2004;2:64-69.
- Que K, Ruan J, Fan X, Liang X, Hu DA. A multi-centre and cross-sectional study of dentine hypersensitivity in China. *J Clin Periodontol* 2010;37:631-637.