

# A cross-sectional study of dentine hypersensitivity

J. S. Rees and M. Addy

Division of Restorative Dentistry, Department of Oral & Dental Science, University of Bristol Dental School, Lower Maudlin St., Bristol BS1 2LY, U.K

Rees JS, Addy M. A cross-sectional study of dentine hypersensitivity. *J Clin Periodontol* 2002; 29: 997–1003. © Blackwell Munksgaard, 2002.

## Abstract

**Aim:** The aim of this study was to establish the prevalence of dentine hypersensitivity in a cross-sectional study of patients visiting general dental practitioners in the United Kingdom over a period of one calendar month.

**Methods:** Nineteen dental practitioners examined 4841 patients over a period of one calendar month and patients that had dentine hypersensitivity diagnosed were questioned further about their occupation and smoking habits. The amount of buccal gingival recession associated with the sensitive teeth was also recorded using a study form.

**Results:** 201 patients were diagnosed as having dentine hypersensitivity, giving a prevalence figure of 4.1%. The commonest teeth affected were the upper premolar teeth and the commonest initiating factor was cold drinks. A tendency for a greater number of sensitive teeth was also found for patients with periodontal disease who also smoked. There was also a tendency for the patients with sensitive teeth to come from higher social groups.

Key words: dentine; dentine hypersensitivity; epidemiology; gingival recession; smoking

Accepted for publication 30 October 2001

## Introduction

Dentine hypersensitivity may be defined as pain arising from exposed dentine, typically in response to chemical, thermal or osmotic stimuli that cannot be explained as arising from any other form of dental defect or pathology (Addy & Urquart 1995). Dentine hypersensitivity is a common problem found in many adult populations with prevalence figures ranging from 4 to 74% (Table 1) (Jensen 1964, Graf & Galasse 1977, Flynn *et al.* 1985, Orchardson & Collins 1987, Fischer *et al.* 1992, Murray & Roberts 1994, Chabanski *et al.* 1997, Irwin & McCusker 1997, Liu *et al.* 1998, Rees 2000). This wide variation in prevalence may be due to a number of factors, including different methods used to diagnose the condition and variation in the consumption of erosive foods and drinks. Dentine hy-

persensitivity is also commonly found in patients with chronic periodontal disease, as the root surface may become exposed as part of the disease process. Prevalence figures for dentine hypersensitivity are higher in this group of patients, with reported figures of between 72.5% and 98% (Chabanski *et al.* 1997). This led Dababneh *et al.* (1999) to suggest that the dentine hypersensitivity associated with periodontal disease may have a different aetiology, possibly related to bacterial penetration of the dentinal tubules (Adriaens *et al.* 1988).

Most of the previous investigations of dentine hypersensitivity (Table 1) have examined a sample of patients referred to a university hospital and these results are therefore likely to be based on a biased sample (Jensen 1964, Flynn *et al.* 1985, Orchardson & Collins 1987, Fischer *et al.* 1992, Murray & Roberts

1994, Chabanski *et al.* 1997, Liu *et al.* 1998). The only studies carried out in a dental practice setting appear to be those of Graf & Galasse (1977), Irwin & McCusker (1997) and Rees (2000). Graf & Galasse (1977) reported a prevalence of 14.5%, while Irwin & McCusker (1997) reported a prevalence of 57% and Rees reported a lower value of 3.8%. The study of Irwin & McCusker (1997) was carried out using a patient questionnaire with no subsequent clinical examination, so that it is likely to be an overestimation due to the inclusion of other causes of sensitivity.

The aim of the present study was to carry out a cross-sectional study of a group of patients treated in general dental practice in the United Kingdom to estimate the prevalence of dentine hypersensitivity and to investigate the potential effect of smoking and social

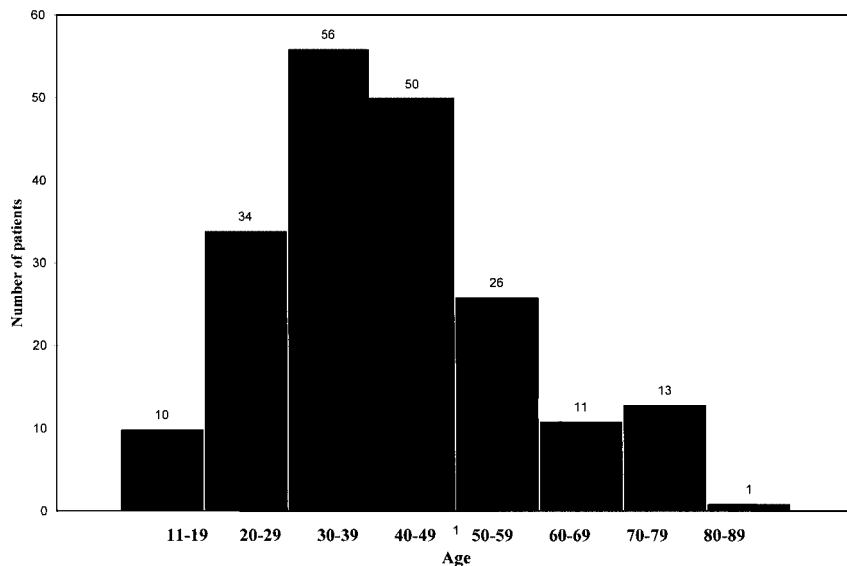


Fig. 1. The age distribution of the patients with sensitive teeth.

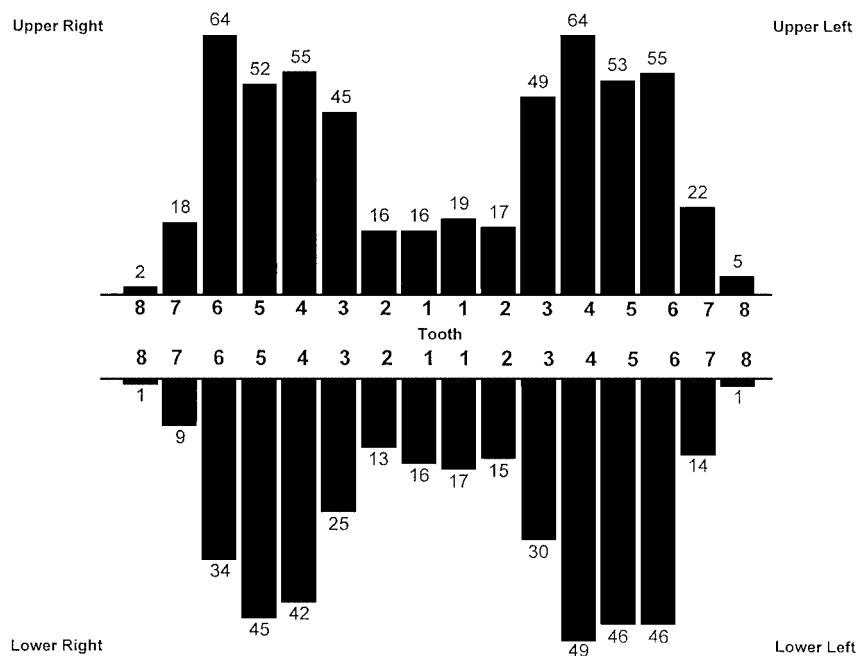


Fig. 2. Dentine hypersensitivity by tooth type.

class on dentine hypersensitivity. It was postulated that, since smokers often have more severe periodontal disease (Salvi *et al.* 2000), it was possible that they would also have more teeth with dentine hypersensitivity.

**Materials and methods**

All 19 General Dental Practitioners who were undertaking a postgraduate distance-learning course on toothwear

run by the University of Bristol Dental Postgraduate Department were recruited to participate in this study and all the participants completed the study. Prior to the start of the study, the practitioners met with the authors to finalise details of the study protocol. In addition to this, they were also asked to study a module that gave an overview of the topic of dentine hypersensitivity (Addy 2000). This module included a number of review articles on the topic

(Dowell *et al.* 1985, Addy & Urquart 1992, Addy & Pearce. 1994). Throughout the study protocol meeting and in the module, it was emphasised that, in order to make the diagnosis of dentine hypersensitivity, other pathology, such as caries, must be ruled out.

The study ran from May 1st – May 31st 2000 and all of the patients seen by each dentist during the trial period were screened for sensitive teeth. If the dentist received a positive response, the diagnosis was confirmed using a blast of air from a triple syringe and by ruling out other causes of sensitivity, such as caries. Tactile sensitivity using a probe applied to the cervical region was not assessed as Chabanski *et al.* (1997) found no difference in the subjective response to tactile and evaporative stimuli. Where a diagnosis of dentine hypersensitivity was made, a study form was completed. This included details of the patient’s age, gender, occupation, smoking habits, teeth affected and any factors that initiated the sensitivity. In addition to this, each participant was asked to measure any buccal gingival recession associated with these sensitive teeth. Measurements were made using a 1 mm graduated periodontal probe from the amelocemental junction to the free gingival margin. They were also asked to record the total number of patients seen during the trial period and the various methods they employed to manage the sensitivity.

**Results**

The total number of patients seen by the 19 dental practitioners involved in the study was 4841. A total of 782 teeth were diagnosed as having dentine hypersensitivity in 201 patients, giving an overall prevalence figure for dentine hypersensitivity of 4.1%. The individual prevalence figures for each of the 19 practices involved in the study are given in Table 2. As this demonstrates, individual prevalence figures range from 1.2 to 18.3%. The average age of these patients was 41.4 years, with a range of 16 years to 82 years. 58 patients were male and 143 were female, giving an overall male:female ratio of 1:2.5. A histogram showing the age distribution of the patients with hypersensitive dentine is given in Fig. 1. By far, the highest numbers of patients with dentine hypersensitivity belonged to the 30–50 years age group.

The number of sensitive teeth classi-

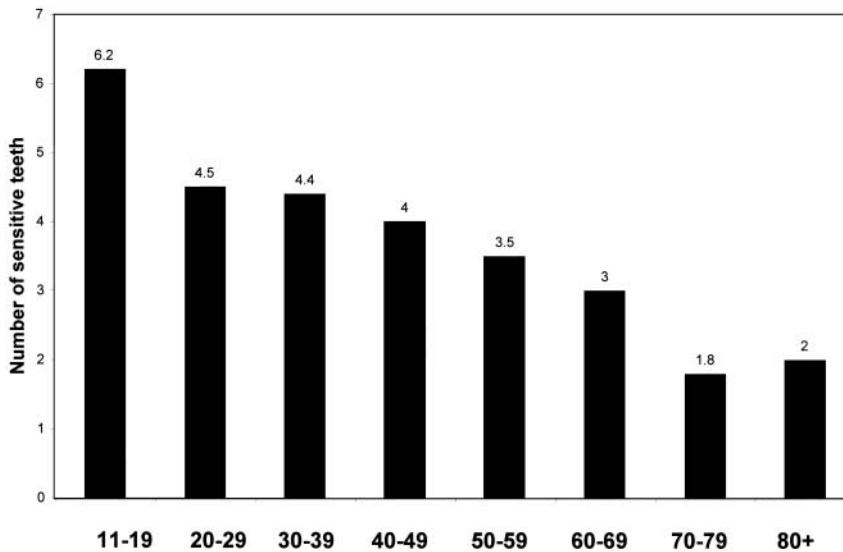


Fig. 3. The mean number of sensitive teeth per patient.

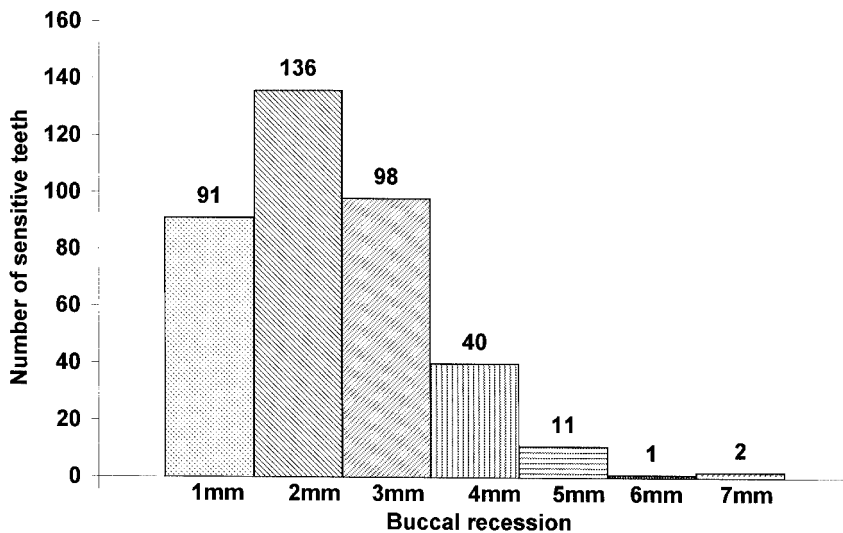


Fig. 4. Gingival recession associated with sensitive teeth.

fied by tooth type (Fig. 3) shows that the tipper premolars and first molars were the most commonly affected.

The mean number of sensitive teeth per patient by age group (Fig. 4) shows a peak of 6.2 sensitive teeth for the 11–19 years age group, with the number of sensitive teeth per patient then gradually diminishing with each subsequent decade.

The amount of gingival recession associated with the sensitive teeth (Fig. 4) shows that, overall, 708 of the 782 sensitive teeth (91%) had some associated buccal gingival recession, the majority (87%) in the range of 1–3mm.

The various initiating factors were re-

corded and found that cold drinks were the major stimulus for dentine hypersensitivity in 55% of the sample.

The mean amount of gingival recession per patient that was associated with the sensitive teeth was recorded (Fig. 6). This was calculated by summing the total amount of gingival recession associated with the sensitive teeth in individual patients and dividing by the number of sensitive teeth per patient. This data was classified according to whether the patients were smokers or non-smokers and whether they had periodontal disease or not. The data is also presented in Table 3. As this shows, patients who smoked and had peri-

odontal disease had a greater number of sensitive teeth (5.9) and more gingival recession in comparison to the other groups. Analysis of variance also showed that the amount of mean gingival recession found in the group who smoked and had periodontal disease was statistically significant ( $P < 0.001$ ) from the other groups.

The relationship between dentine hypersensitivity and social class was examined (Fig. 7) using the Registrar General's Classification of Occupations as used by Bradnock *et al.* (2001) in the UK Adult Dental Health Survey. This divides occupations into a series of six groups using the following classification:

- I Professional (e.g. doctor, dentist, lawyer)
- II Managerial and lower professional (e.g. manager, nurse, school teacher)
- IIIN Skilled, non-manual (e.g. clerk, cashier)
- IIIM Skilled, manual (e.g. carpenter, bricklayer, coal face worker)
- IV Semi-skilled, manual (e.g. postman, agricultural worker)
- V Unskilled, manual (e.g. porter, ticket collector, general labourer).

It was found that the majority of the patients with sensitivity (74%) fell into the first three groups (I, II and IIIN).

## Discussion

The overall prevalence figure for dentine hypersensitivity reported in this study was 4.1%, lower than many of the prevalence figures reported previously (Table 1). There could be a number of reasons for this. Firstly, many previous studies only used a patient questionnaire with no subsequent clinical examination. This approach is likely to overestimate the prevalence value, as the sensitivity recorded could be due to several other pathologies (Dowell *et al.* 1985). Secondly, only three previous studies have attempted to estimate the prevalence of dentine hypersensitivity within a general practice population (Graf & Galasse 1977, Irwin & McCusker 1997, Rees 2000). The study of Graf & Galasse (1977) examined a fairly small population ( $n = 351$ ), while the study of Irwin & McCusker (1997) used a questionnaire design with no clinical examination. The previous study by one of the authors (Rees 2000) used the same methodology but a to-

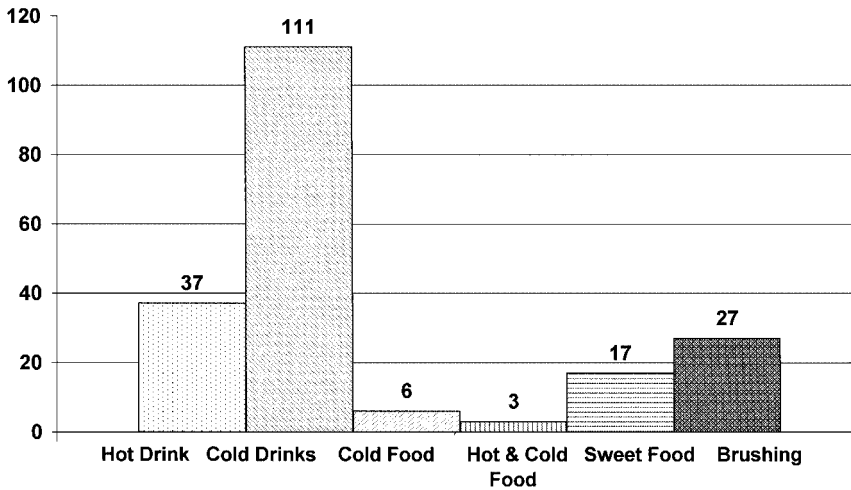


Fig. 5. Initiating factors.

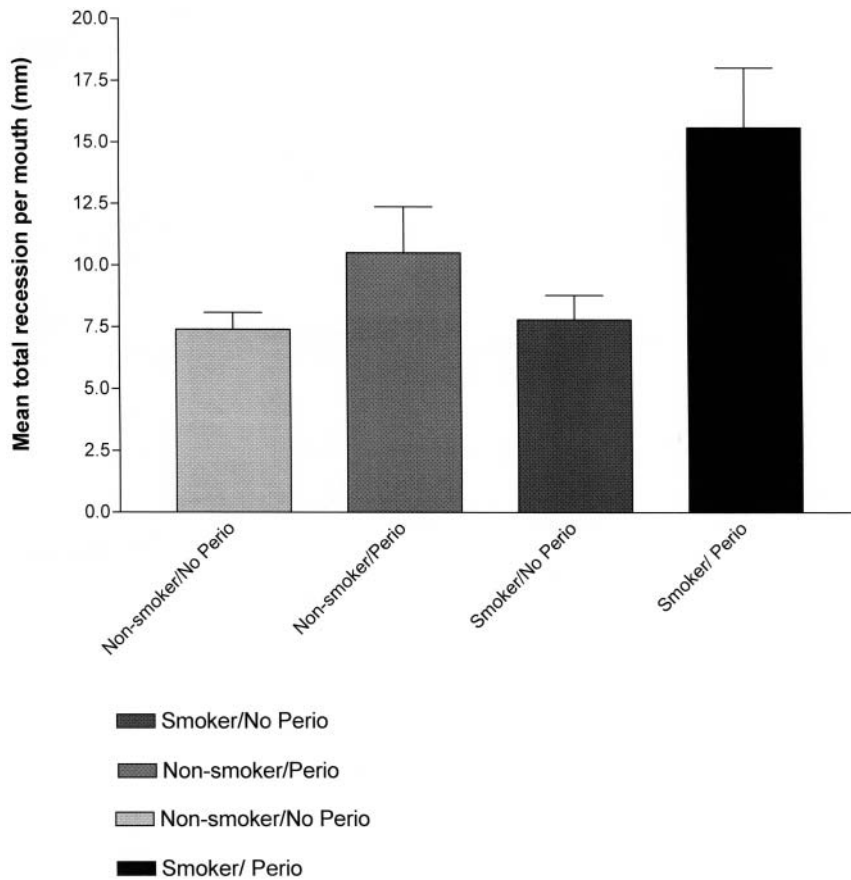


Fig. 6. Mean total gingival recession per mouth related to smoking and periodontal disease.

tally different group of dentists and patients. It is therefore interesting to note that this study produced an overall prevalence figure of 4.1% (Rees 2000) that is similar to the figure of 3.8% reported here.

However, the prevalence figure re-

ported here must be interpreted with a certain amount of caution, as there was a wide variation around this figure of between 1.2% and 18.3% for the individual practices involved in the study. Firstly, the practices involved in the study were not selected randomly, but

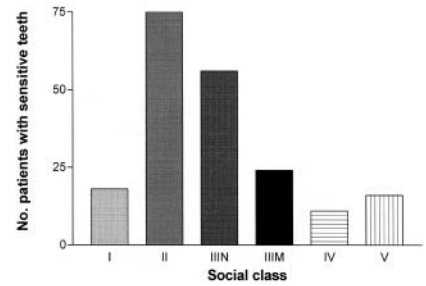


Fig. 7. Patients with dentine hypersensitivity classified by social class.

were self-selected due to their dentist's participation in a postgraduate programme. Secondly, it is difficult to assess whether or not the patients attending these practices could be considered to be representative of all patients in general practice. However, the wide range of individual prevalence figures may suggest that this was due to differences related to the individual examiners rather than their patients. Furthermore, the individual prevalence figures for each practice will be strongly influenced by the composition of the patients attending each individual practice. Table 2 shows the geographical location of the various practices involved in the study and shows that the practices cover most areas of the UK. They also serve a mixture of inner-city and rural areas. Therefore, it is likely that the social class profile of the patients attending these individual practices differs and this may also contribute to the variation in the individual prevalence data recorded. Finally, since a binary index was used for the diagnosis of sensitivity, the chances of an alpha error due to examiner bias is highly unlikely.

It is well established that dentine hypersensitivity occurs more commonly in females. Table 2, which outlines the individual prevalence data, together with the average age, age range and male:female ratio, supports this finding, with 16 out of the 19 practices reporting a female bias. However, this bias towards a higher prevalence in females may also represent a stronger need to seek treatment.

In this study, dentine hypersensitivity was greatest in the 30–49 years age group, with a peak in the 30–39 years age group ( $n=41$ ) and slightly less in the 40–49 years age group ( $n=38$ ). Several investigators have reported the age

Table 1. Summary of prevalence studies on dentine hypersensitivity;

Authors	Country	Setting	Study type	n	Prevalence
Jensen (1964)	USA	University	Clinical	3000	30%
Graf & Glase (1977)	Switzerland	Practice	Clinical	351	15%
Flynn et al. (1985)	UK	University	Clinical	369	18%
Orchardson & Collins (1987)	UK	University	Clinical	109	74%
Fischer et al. (1992)	Brazil	University	Clinical	635	17%
Murray & Roberts (1994)	Indonesia	Not stated	Questionnaire	1000	27%
Murray & Roberts (1994)	USA	Not stated	Questionnaire	1000	18%
Murray & Roberts (1994)	Japan	Not stated	Questionnaire	1000	16%
Murray & Roberts (1994)	France	Not stated	Questionnaire	1000	14%
Murray & Roberts (1994)	Germany	Not stated	Questionnaire	1000	13%
Murray & Roberts (1994)	Australia	Not stated	Questionnaire	1000	131%
Chabanski et al. (1997)	UK	University	Clinical	51	73%
Irwin & McCusker (1997)	UK	Practice	Questionnaire	250	57%
Liu, Lan & Hsieh (1998)	Taiwan	University	Clinical	780	32%
Rees (2000)	UK	Practice	Clinical	3593	4%

Table 2. The individual prevalence figures, average age, age range and male:female ratio for each of the practices participating in the study

Practice number	Total number of patients seen	Prevalence	Mean age	Age range	M:F	County
1	305	3.3%	41.7	29–58	2.5:1	Somerset
2	483	1.6%	42.9	34–56	1:2	N. Ireland
3	121	5.8%	48.6	42–72	1:1	Cornwall
4	103	6.8%	38.7	27–52	1:2.2	Hampshire
5	133	9.0%	35.6	22–74	1:1.5	Greater London
6	280	3.8%	44.7	29–64	1:5.5	Cheshire
7	196	10.2%	36.3	16–55	1:7	N. Ireland
8	136	4.4%	39.5	25–57	1:4	Sussex
9	258	7.0%	37.9	17–75	1:2.3	Avon
10	440	3.0%	51.0	30–73	1:3.5	Sussex
11	65	7.7%	46.0	25–82	1:6	Wiltshire
12	188	9.0%	51.5	29–72	1:1.6	Avon
13	200	9.0%	34.2	16–75	1:1.5	W. Midlands
14	438	2.3%	41.8	23–65	1:1	Surrey
15	71	18.3%	39.1	18–75	1:2.5	Norfolk
16	261	3.1%	44.8	32–65	1:3.1	Perthshire
17	507	1.6%	47.5	37–72	1:3	Essex
18	250	1.2%	52.7	42–66	1:4.5	Devon
19	406	1.7%	35.9	26–56	1:4.3	W. Midlands

distribution of dentine hypersensitivity. Orchardson & Collins (1987) showed a peak prevalence between 20 and 25 years, Graf & Galasse (1977) between 25 and 29 years, Addy (1992) between 20 and 40 years and Fischer et al. (1992) between 40 and 49 years.

The teeth most often affected by dentine hypersensitivity were the upper premolars, followed by the upper first molars with the incisors being the least sensitive ones. This distribution is similar to earlier studies, with many dental workers reporting that the canine/premolar regions were the commonest sites for sensitivity. (Flynn *et al.* 1985, Orchardson & Collins 1987, Addy *et al.* 1987a, Fischer *et al.* 1992, Liu *et al.* 1998, Rees 2000). Chabanski *et al.*

(1997) found that molar teeth were the commonest teeth exhibiting sensitivity in a group of patients that were diagnosed with periodontal disease. It is interesting to note that, within our sample, 27% of the patients had periodontal disease and the second commonest tooth displaying sensitivity were first molars.

The mean number of sensitive teeth per patient for the sample was 3.0, with a range of 1–19. This is similar to the mean figure of 4 with a range of 1–16 reported by Orchardson & Collins (1987). These data were broken down further into age cohorts (Fig. 3). The mean number of sensitive teeth per patient reached a peak at 6.2 in the 11–19 years age group and then reduced

slowly in the older cohorts. This finding was a little surprising and may well be anomalous as only two patients were included in the 11–19 year-old-cohort.

Many of the sensitive teeth included in this study also had some degree of gingival recession (Fig. 4). Most teeth had at least 1–3 mm of gingival recession that is similar to the average recession of 2.5 mm reported by Addy *et al.* (1987b) in their sample of sensitive teeth.

The major stimulus that caused dentine sensitivity (Fig. 6) was cold drinks (55%) and, to a lesser extent, hot drinks (18%) and tooth brushing (13%). Many other studies have reported that cold stimuli, either an evaporative stimulus as applied in this study or as a direct thermal stimulus, has been reported as the most prevalent stimulus (Flynn *et al.* 1985, Orchardson & Collins 1987, Fischer *et al.* 1992, Irwin & McCusker 1997, Rees 2000).

It is now well established that smoking is a major risk factor for periodontal disease and that that exposure of root surfaces is a common sequelae of periodontal disease (Harber *et al.* 1993). Therefore, it was interesting to analyse whether there was any difference in the number of sensitive teeth per patient when the patient was a smoker or non-smoker and if they also did or did not have periodontal disease (Table 3). There seemed to be little difference in the number of sensitive teeth per patient when the patient was a non-smoker with and without periodontal disease (3.8 vs. 2.9). However, when patients with periodontal disease who also smoked were examined, the number of sensitive teeth per patient was approximately double that of a smoker with no periodontal disease (5.9 vs. 3.7). Presumably, the increased gingival recession and subsequent sensitivity present in patients with periodontal disease was due to the cumulative effects of periodontal disease and the effects of treatment.

It was therefore interesting to examine the amount of gingival recession associated with the sensitive teeth. The patients with sensitive teeth were divided into four groups, depending on whether they had periodontal disease or not and if they were smokers or non-smokers (Fig. 8). There was little difference in the amount of buccal gingival recession in the three groups which were classified as non-smoker/no periodontal disease, non-smoker/periodontal dis-

Table 3 Relationship between sensitive teeth, smoking and periodontal disease;

Group	n	Mean number sensitive teeth per patient	Mean total gingival recession per patient (mm)
Non-smoker/no periodontal disease	103	3.8	7.4
Non-smoker/periodontal disease	42	2.9	10.5
Smoker/no periodontal disease	27	3.7	7.8
Smoker/periodontal disease	29	5.9	15.6

ease and smoker/no periodontal disease. However, in the patients who smoked and also had periodontal disease, the amount of gingival recession was approximately one third greater than the other three groups. This difference was also found to be statistically significant at the  $p < 0.001$  level, using analysis of variance.

These findings provide some support for the hypothesis of Dababneh *et al.* (1999) who suggests that the dentine hypersensitivity associated with periodontal disease may have a different aetiology, possibly related to bacterial penetration of the dentinal tubules (Adriaens *et al.* 1988). However, this data must be interpreted with a certain amount of caution, as there were only 29 patients (14%) in the group that smoked and had periodontal disease.

It is the authors' clinical impression, supported by some data (Absi *et al.* 1987), that dentine hypersensitivity is more prevalent in patients with clean mouths who have good oral hygiene practices, as tends to be the case in the higher social groups (Dummer *et al.* 1987). To investigate this further, the patients with dentine hypersensitivity were divided into social groups using the Registrar General's Classification of Occupations as used in the recent UK Adult Dental Health Survey (Bradnock *et al.* 2000). This demonstrated (Fig. 7) that dentine hypersensitivity was more prevalent in the higher social groups, with 74% of the sensitive teeth being found in the top three social groups (I, II and IIIN). However, this data must also be interpreted with a certain amount of caution, as this sample may be further biased by the fact that a number of regular attenders at dental surgeries are also known to belong to higher social groups.

In conclusion, this cross-sectional study found that the prevalence of dentine hypersensitivity in patients attending general dental practice in this study was 4.1%. There was a wide variation around this average value of 1.2–18.3% for the practices involved in this study,

which may be related to differences in the number of female patients attending each practice and the different social make up of the population served by each practice.

This study has also provided some preliminary data that suggests that dentine hypersensitivity may occur more commonly in patients who have periodontal disease and also smoke and in patients from higher social groups.

#### Acknowledgements

We would like to thank all the dentists undertaking the Bristol University Open Learning for Dentists (BUOLD) programme in tooth wear, who participated in the study.

#### Zusammenfassung

*Eine Querschnittstudie zur Hypersensitivität des Dentins*

**Ziel:** Das Ziel dieser Studie war die Erfassung der Dentinüberempfindlichkeit in einer Querschnittstudie bei Patienten, die eine allgemein-zahnärztliche Praxis im Vereinigten Königreich während eines Kalendermonats besuchten.

**Methoden:** 19 Zahnärzte überprüften 4841 Patienten während eines Kalendermonats. Patienten, die eine Dentinhypersensitivität hatten, wurden über ihre Beschäftigung und den Raucherstatus befragt. Die Größe der bukkalen Rezessionen, die in Verbindung mit den hypersensitiven Zähnen stand, wurde auch aufgezeichnet.

**Ergebnisse:** 201 Patienten hatten eine Dentinhypersensitivität, was eine Prävalenz von 4,1% ergibt. Die am meisten betroffenen Zähne waren die oberen Prämolaren, und der am häufigsten die Hypersensitivität auslösende Faktor waren kalte Getränke. Eine Tendenz für eine größere Anzahl sensibler Zähne wurde bei Patienten mit parodontalen Erkrankungen, die auch rauchten, gefunden. Es gab auch eine Tendenz bei den Patienten mit sensiblen Zähnen, dass sie von höheren sozialen Gruppen kamen.

#### Résumé

*Une étude croisée sur l'hypersensibilité dentinaire*

**But:** Le but de cette étude était d'établir la

prévalence de l'hypersensibilité dentinaire lors d'une étude croisée sur des patients suivis en pratique générale au Royaume uni, pendant une période d'un mois.

**Méthodes:** 19 praticiens ont examiné 4841 patients sur une période d'un mois et les patients pour lesquels fut diagnostiqué une hypersensibilité dentinaire furent interrogés sur leurs habitudes comportementales et tabagiques. L'importance des récessions vestibulaires associées avec les dents sensibles fut également enregistrée sur un formulaire d'étude.

**Résultats:** 201 patients furent diagnostiqués comme présentant une hypersensibilité ce qui représente une prévalence de 4.1%. Les dents les plus souvent atteintes étaient les prémolaires supérieures et le facteur déclenchant le plus souvent associé était une boisson froide. Une tendance vers un plus grand nombre de dents atteintes était trouvée chez les patients ayant une maladie parodontale et qui fumaient. Il y avait aussi une tendance à la sensibilité chez les patients issus de groupes sociaux supérieurs.

#### References

- Absi, E., Addy, M. & Adams, D. (1987) Dentine hypersensitivity. The patency of dentinal tubules in non-sensitive and sensitive dentine. *Journal of Oral Clinical Periodontology* **14**, 280–284.
- Addy, L. M. (2000) Dentine Hypersensitivity: Diagnosis and Clinical Management. *Bristol University Open Learning for Dentists Course, Dental Postgraduate Department*. University of Bristol: Bristol.
- Addy, M., Absi, E. D. & Adams, D. (1987b) Dentine hypersensitivity: the effects of acids and dietary compounds on burred and planed dentine. *Journal of Clinical Periodontology* **14**, 274–279.
- Addy, M., Mostafa, P. & Newcombe, R. G. (1987a) Dentine hypersensitivity: the distribution of recession, sensitivity and plaque. *Journal of Odontology* **15**, 242–248.
- Addy, M. & Pearce, N. X. (1994) Aetiological, predisposing and environmental factors in dentine hypersensitivity. *Archives of Oral Biology* **39** (Suppl. 3) (3 S–3), 8 S.
- Addy, M. & Urquhart, E. (1995) Dentine hypersensitivity: Its prevalence, aetiology and clinical management. *Dental Update* **22**, 407–412.
- Adriaens, P. A., de Boever, L. A. & Loesche, W. J. (1988) Bacterial invasion in root. cementum and radicular dentine of periodontally diseased teeth in humans – a res-

- ervoir of periodontopathic bacteria. *Journal of Periodontology* **59**, 222–230.
- Bradnock, G., White, D. A., Nutall, W. M., Morris, A. J., Treasure, E. T. & Pine, C. M. (2001) Dental attitudes and behaviour in 1998 and implications for the future. *British Dental Journal* **190**, 229–232.
- Chabanski, M. B., Gillam, D. G., Bulman, I. S. & Newman, H. N. (1997) Clinical evaluation of cervical dentine sensitivity in a population of patients referred to a specialist periodontology department: a pilot study. *Journal of Oral Rehabilitation* **24**, 666–672.
- Dababneh, R. H., Khouri, A. T. & Addy, M. (1999) Dentine hypersensitivity – an enigma? A review of terminology, epidemiology, mechanisms, aetiology and management. *British Dental Journal* **189**, 606–611.
- Dowell, P. D., Addy, M. & Dummer, P. M. H. (1985) Dentine hypersensitivity. Aetiology, differential diagnosis and management. *British Dental Journal* **158**, 92–96.
- Dummer, P. M. H., Addy, M., Hicks, R., Kingdon, A., Shaw, W. C. (1987) The effect of social class on the prevalence of caries plaque, gingivitis and pocketing. in 11–12 year-old children in South Wales. *Journal of Dentistry* **15**, 185–190.
- Fischer, C., Fischer, R. G. & Wennberg, A. (1992) Prevalence and distribution of cervical dentine hypersensitivity in a population in Rio de Janeiro, Brazil. *Journal of Dentistry* **20**, 272–276.
- Flynn, L., Galloway, R. & Orchardson, R. (1985) The incidence of 'hypersensitive' teeth in the West of Scotland. *Journal of Dentistry* **13**, 230–236.
- Graf, H. & Galasse, R. (1977) Morbidity, prevalence and intraoral distribution of hypersensitive teeth. *Journal of Dental Research* **AI 62**, Abstract no. 479.
- Harber, L., Wattles, L. & Crowley, P. (1993) Evidence for cigarette smoking as a major risk factor for periodontitis. *Journal of Periodontology* **64**, 16–23.
- Irwin, C. R. & McCusker, P. (1997) Prevalence of dentine hypersensitivity in a general dental population. *Journal of The Irish Dental Association* **43**, 7–9.
- Jensen, M. (1964) Hypersensitivity controlled by iontophoresis. Double blind clinical investigation. *Journal of The American Dental Association* **68**, 216–225.
- Kaldahl, W. B., Johnson, G. K., Patil, K. D. & Kalkwarf, K. L. (1996) Levels of cigarette consumption and response to periodontal therapy. *Journal of Clinical Periodontology* **67**, 675–681.
- Liu, H.-S., Lan, W.-H. & Hsieh, C.-C. (1998) Prevalence and distribution of cervical dentine hypersensitivity in a population in Taipei, Taiwan. *Journal of Endodontics* **24**, 45–47.
- Murray, L. E. & Roberts, A. L. (1994) The prevalence of reported hypersensitive teeth. *Archives of Oral Biology* **39** (Suppl.) 129S.
- Orchardson, R. & Collins, W. J. N. (1984) Orchardson, R. & Collins, W. J. N. (1987) Clinical features of hypersensitive teeth. *British Dental Journal* **162**, 253–256.
- Pitts et al.
- Rees J. S. (2000) The prevalence of dentine hypersensitivity in general dental practice in the UK. *Journal of Clinical Periodontology* **27**, 860–865.
- Salvi, G., Lawrence, H. P., Offenbacher, S. & Beck, J. D. (1997) Influence of risk factors on the pathogenesis of periodontitis. *Periodontology 2000* **14**, 73–201.