Tropical Medicine and International Health doi:10.1111/tmi.12027

VOLUME 18 NO 2 PP 222–229 FEBRUARY 2013

Prevalence and aetiology of juvenile skeletal fluorosis in the south-west of the Hai district, Tanzania – a community-based prevalence and case–control study

Helen G. Jarvis[1,2], Peta Heslop[1], John Kisima[3], William K. Gray[1], Godwin Ndossi[4], Anne Maguire[5] and Richard W. Walker[1]

1 *Northumbria Healthcare NHS Foundation Trust, North Tyneside General Hospital, North Shields, UK*
2 *London School of Hygiene and Tropical Medicine, London, UK*
3 *Hai District Hospital, Kilimanjaro, Tanzania*
4 *Tanzanian Food and Nutrition Centre, Dar-es-Salaam, Tanzania*
5 *Centre for Oral Health Research, Newcastle University, Newcastle upon Tyne, UK*

Abstract

INTRODUCTION Fluorosis is endemic throughout the East African Rift valley, including parts of Tanzania. The aim of the study was to identify all cases of deforming juvenile skeletal fluorosis (JSF) in a northern Tanzanian village and to document the extent of dental fluorosis (DF).

METHODS Door-to-door prevalence survey of all residents of the village. Residents were assessed for the presence of DF and JSF. Those with JSF and randomly selected controls from the same age range were further assessed for possible JSF risk factors.

RESULTS The village had a population of 1435. DF was endemic within the population, being present in 911 (75.5%; 95% CI, 73.0–77.9) of dentate individuals who were examined (n = 1207). JSF was present in 56 of 1263 people examined, giving a prevalence of 4.4% (95% CI, 3.3–5.6) and was more common in males. Low body mass index, drinking predominantly well water 3 years previously, not being weaned on bananas, the use of fluoride salts in cooking during childhood and drinking more cups of tea per day were independent predictors of JSF.

CONCLUSIONS Juvenile skeletal fluorosis is a common and preventable public health problem. Providing clean, low-fluoride, piped water to affected communities is of obvious health benefit.

keywords fluorosis, prevalence, Tanzania, Africa

Introduction

Skeletal fluorosis (SF) is caused by ingestion of high levels of fluoride ion and has been shown to occur in areas with excessively high-fluoride concentrations in the drinking water predominantly sourced from boreholes (Christie 1980). WHO guidelines on drinking water quality advise that fluoride be ingested at a maximum concentration of 1.5 mg of fluoride/l where possible, to prevent skeletal changes as well as most cases of dental fluorosis (DF) (World Health Organization 2004).

In adult onset SF, the density of the affected bone is increased (osteosclerosis), and the fluoride encourages the growth of new abnormal bone at ligament and tendon insertion sites, causing ossification (Gupta *et al.* 1993). This normally manifests as joint pain and stiffness in adult life with associated neurological complications and is endemic in many areas with high-fluoride ingestion. Juvenile onset skeletal fluorosis (JSF) (Kenhardt bone disease) generally affects children during times of bone growth and manifests as deformities, particularly of the weight-bearing bones. This form of SF is much less common and less well researched. Evidence from India has suggested that the more severe, deforming JSF only occurs when there is inadequate calcium in the diet and that protein consumption levels are also important (Krishnamachari 1986; Chakma *et al.* 1997; Teotia *et al.* 1998). Where the calcium supply is insufficient, the absorbed fluoride may exacerbate this low calcium intake by chelating with the calcium, leading to secondary hyperparathyroidism and subsequent areas of both porosis and sclerosis of the bone (Mithal *et al.* 1993; Teotia *et al.* 1998).

Fluorosis is endemic in at least 25 countries across the world owing to naturally high concentrations of fluoride in the groundwater, which generally occur in volcanic areas such as the East African Rift Valley (UNICEF 1999; Chernet *et al.* 2001; Ayoob & Gupta 2006). Parts

of Tanzania are within this volcanic system, and problems owing to high-fluoride exposure in Tanzania are well established (Fawell *et al.* 2006). Much of the previous work in relation to DF in Northern Tanzania has focused on the use of the fluoride containing salt, 'magadi' or 'trona', which is used extensively in Tanzania as a tenderiser, and for adding flavour, in cooking. There is good evidence that this and/or other dietary sources of fluoride contribute to DF in villages throughout Tanzania, though there are no previous studies of the effect of such dietary factors on the development of JSF there (Mabelya *et al.* 1992, 1997). There is also evidence that when tea is grown in endemic areas, fluoride concentrates within the tea plant and can contribute to the development and severity of DF (Gulati *et al.* 1993).

A previously reported study by our group in Tanzania identified two villages in the Hai district of Northern Tanzania with very high groundwater fluoride levels of up to 25 mg fluoride/l (Gulati *et al.* 1993). This pilot survey in two village schools confirmed endemic DF as well as large numbers of school children with skeletal deformities presumably secondary to JSF. The survey raised questions regarding prevalence of JSF amongst the remainder of the population, as well as possible causal factors (Shorter *et al.* 2010).

There are no published population prevalence estimates for deforming JSF in an endemic area in Sub-Saharan Africa. Although there are a few published studies looking at the physical and social effects of JSF in communities, there are no previous studies looking at risk factors for JSF within a prevalent population (Haimanot *et al.* 1987; Bo *et al.* 2003).

This study had two main aims: to conduct a door-to-door prevalence survey to identify all the cases of DF and deforming JSF in a defined population, in an area of Tanzania with known high groundwater fluoride concentrations and to conduct a case–control study, nested within the prevalence study, to help identify possible aetiological factors important in the development of this form of JSF.

Materials and methods

Design

The study design was a door-to-door prevalence survey with a nested case–control study.

Setting

The study was conducted in the village of Tindigani in the Hai district of Northern Tanzania. Tindigani is situated on the dry flatlands, immediately south of the fertile areas on the slopes of Mount Kilimanjaro and Mount Meru. This area of Tanzania forms a sector of the East African Rift Valley running north into Kenya and Ethiopia. The main source of drinking water in this area for the last 30 years has been groundwater from hand-dug wells and boreholes that have often been provided by non-governmental organisations or charities. Before this, the population collected surface water from the nearby rivers. However, in the last 2 years, some of the larger settlements in the area have received a low-fluoride piped water supply.

Cases

Hai was one of three project areas set up by the Adult Morbidity and Mortality Project (2004) and has been retained as a disease surveillance site (DSS) comprising 52 demarcated villages. Each household and individual within the household has a unique identifying number. Since 1992, trained and experienced enumerators, who are often nurses or clinical officers, have carried out a regular census of the population. From the most recent census, conducted at the beginning of 2009, 1435 people were identified as living in Tindigani. Cases were identified from within this population.

Controls

Controls from within the village of Tindigani were selected by a random number generator from the census database, after the exclusion of cases already identified. The controls were age restricted to between 2 and 30 years of age to reflect the age range of the cases identified. No other matching was carried out. Only those members of the population born and raised in the area were included in the study, to ensure an accurate reflection of prevalence, within an area of high-fluoride exposure.

Assessment

All data were collected between April and July 2009. Translators and interpreters were involved during the study, as appropriate. The information and consent sheets regarding the study were translated into Swahili and then back-translated to ensure accuracy. The assessment questionnaires were translated into Swahili/Maasai by an experienced local translator helped by a local health worker.

Prevalence

Each member of the population underwent a brief dental and musculoskeletal examination (HJ and PH), for which

training was received from AM. The prevalence and severity of DF were graded using the Thylstrup and Fejerskov Index (TFI) (Fejerskov *et al.* 1988). The criteria for diagnosis of deforming JSF were based on that suggested by Krishnamachari (1986). This consisted of any of genu valgum/varum, sabre tibia (anterior bending and thickening of the tibia), kyphosis/scoliosis (with no other obvious explanation) with or without neurological signs of sensory loss and muscle wasting.

The head of each household was then asked questions to elucidate drinking water habits over time. This was carried out after case ascertainment to ensure blinding to drinking water source when identifying cases. Water sources were tested for current concentrations of fluoride using standard methods. Fluoride analysis of the water samples was determined using an ion-specific electrode (3221 Fluoride ISE; Thermo Orion research Inc.).

Case–control study

A nested case–control design was used during the second part of the study. During this phase, cases and controls were assessed in more detail. For cases, the extent of their deformities was assessed. The coronal tibio-femoral (CTF) angle and knee flexion were measured on both lower limbs using a 12 Inch Protractor Goniometer (HJ and PH). CTF angle is the angle between the anatomical femoral axis and the anatomical tibial axis. Genu varum (bow-legs) was defined as a CTF angle of more than 7° and genu valgum (knock-knees) when the angle is negative (Sass & Hassan 2003).

For both cases and controls, nutritional status [height (cm), weight (kg) and triceps skin fold thickness (mm)] were measured. From these, body mass index (BMI) was calculated. Where available, the mother of the subject was asked a series of detailed questions about the case or control, to attempt to estimate fluoride exposure over time. This included drinking water consumption history, breast-feeding history and toothpaste use. Specific questions were asked regarding tea drinking (as well as intrinsically elevated fluoride in tea, exposure is also dependent on the concentration, brewing time and quantities drunk) and magadi exposure, as known possible aetiological factors. The possible protective effects of good nutrition including calcium and protein intake were assessed by asking about weaning and childhood diet.

Statistical analysis

Data were analysed as for an unmatched case–control study using standard statistical software, SPSS (version 18; SPSS, Chicago, IL, USA) and SAS (version 9.2; SAS Institute, Cary, NC, USA). Confidence intervals (CI) were calculated for odds ratios (OR; categorical data), with a 95% CI not containing 1.0 signifying significance. For differences between means, 95% CI not crossing zero signified significance. Logistic regression modelling was used to estimate univariate ORs for continuous variables and to identify significant predictors of caseness by multivariate modelling. The model was constructed using a combination of stepwise methods and validated by consideration of eigenvalues, variance inflation factors and residual analysis. For continuous variables, the OR indicates the change in the odds of caseness for a unit change in the predictor variable.

Ethics

The Tanzanian National Institute of Medical Research (NIMR) and the London School of Hygiene and Tropical Medicine granted ethical approval for the study.

Results

Prevalence

The population of Tindigani at the time of the door–door survey was 1435 (762 females, 53.1%) living in 281 households (average five persons per household). The age of the population was skewed markedly towards the younger age groups, showing a typical pre-demographic transition population structure, with a median age of 13 years (inter-quartile range 6–28 years).

Dental fluorosis

Dental fluorosis was endemic in the village with 911 (75.5%; 95% CI, 73.0–77.9) of dentate 1207 people examined having some evidence of DF according to the TFI scoring system; the prevalence of DF by age group is shown in Table 1. The prevalence of DF was 73.6% in males (380/516; 95% CI, 69.8–77.4) and 76.8% in females (531/691; 95% CI, 73.7–78.0).

Dental fluorosis did not tend to be severe, and was often not evident at all in the primary dentition. Of 255 children aged 0–4 years who were dentally examined and had teeth, 47 (18.4%) had evidence of DF; a relatively low prevalence compared to other age groups. If this age group was excluded, the prevalence of DF in the remainder of the population (i.e. those in late primary, mixed or permanent dentition) was 90.8% (864 of the 952 examined). The mean TFI score for each age group by gender is shown in Figure 1.

Table 1 Age-specific prevalence rates of dental fluorosis (Thylstrup and Fejerskov Index score > 0) in those dentate individuals who were dentally examined

Age range (years)	Cases (*n*)	Study population (*n*)	Prevalence (%, 95% CI)
0–4	47	255	18.4 (13.7–23.2)
5–9	228	263	86.7 (82.6–90.8)
10–14	172	174	98.9 (97.3–100.4)
15–19	98	99	99.0 (97.0–100.9)
20–24	76	76	100.0 (–)
25–29	56	57	98.2 (94.8–101.7)
30–34	48	51	94.1 (87.7–100.6)
35 and over	186	232	80.2 (75.0–85.3)
Total	911	1207	75.5 (73.0–77.9)

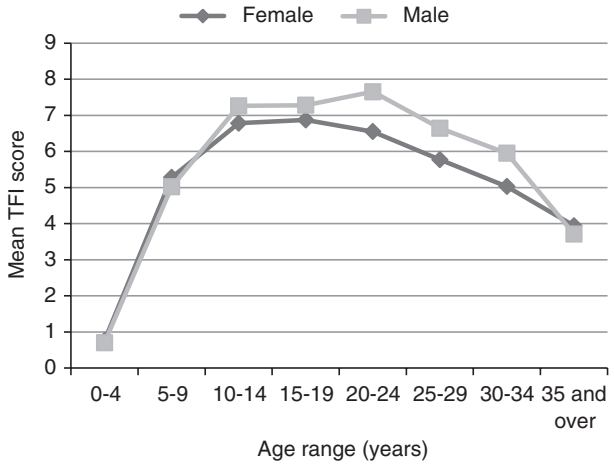

Figure 1 Mean Thylstrup and Fejerskov Index score for the dentate population of the village according to age group and gender.

Juvenile skeletal fluorosis

Fifty-six cases (34 males, 60.7%) of severe, deforming JSF were identified from the 1263 [691 females (54.7%) 88.0% of the entire village population] that were available for musculoskeletal examination. This represented a prevalence of 4.4% (95% CI, 3.3–5.6). Twenty-four cases of JSF (contained within 10 families) had at least one sibling who was also affected. The prevalence in males was 6.2% (95% CI, 4.2–8.2) and in females 3.1% (95% CI, 1.8–4.4). The age of the cases of JSF identified ranged from 2 to 30 years; their mean age was 12.0 years (95% CI, 10.1–13.9). Age specific prevalence rates in Table 2 show that JSF was most common in 10–14 year olds.

Table 2 Age-specific prevalence rates of juvenile skeletal fluorosis

Age range (years)	Cases (*n*)	Study population (*n*)	Prevalence (%, 95% CI)
0–4	8 (6 males)	287	2.8 (0.8–4.7)
5–9	15 (8 males)	265	5.7 (2.9–8.4)
10–14	17 (11 males)	174	9.8 (5.4–14.2)
15–19	9 (5 males)	103	8.7 (3.3–14.2)
20–34*	7 (4 males)	196	3.6 (1.0–6.2)
35 years and over	0	238	0
Total	56 (34 males)	1263	4.4 (3.3–5.6)
Total in those aged 0–34 years	56 (34 males)	1025	5.5 (4.1–6.9)

*Age bands combined owing to low numbers of cases in each category.

Juvenile skeletal fluorosis case–control study

Sixty control subjects (32 males; 53.3%) were recruited with a mean age of 12.4 years (95% CI, 10.8–13.9). There was no significant difference between cases and controls in terms of gender distribution ($\chi^2_1 = 0.643$, $P = 0.422$). Although controls were slightly older than cases, the difference was not significant, based on 95% CIs. Ages in both groups were broadly normally distributed, with a small tail of older people in the cases sample.

The profile of potential risk factors for JSF for cases and controls is compared in Table 3. All of the variables presented in Table 3 were considered for inclusion in a logistic regression model; the model parameter estimates are given in Table 4. Five independent predictors of caseness were identified; lower BMI, bananas not used in weaning, drinking more cups of tea per day, drinking mainly well water 3 years ago and using magadi in cooking in childhood. Overall, the model predicted 71.9% of the variability in outcome (Nagelkerke's R^2).

There was no significant difference in mean TFI score between cases (6.0; 95% CI, 5.3–6.7) and controls (5.9; 95% CI, 5.3–6.5).

Source of fluoride

Of the 275 households reporting a drinking water source, 62.2% were currently walking 3 km to collect low-fluoride piped water from a neighbouring village, 22.5% were drinking surface/river water and 15.3 were drinking well water. Three years before this study started, the number of households drinking from a low-fluoride piped water source was only 12 (4.4%), whilst 117 (42.5%)

Table 3 Comparison of risk factors for juvenile skeletal fluorosis in cases and controls

Risk factor	Cases (n = 56)	Controls (n = 60)	Odds ratio (95% CI)
Anthropomorphic measurements			
Triceps skin fold thickness (mm)	Median, 5.7; IQR, 4.8–7.0	Median, 6.0; IQR, 5.0–8.0	0.96 (0.85–1.08)
Body mass index	Median 15.1; IQR, 13.8–16.6	Median, 16.6; IQR, 14.7–19.6	0.83 (0.73–0.94)
Exposure to fluoride			
Main source of drinking water 3 years ago*	48 (87.3%) well water, 7 piped or surface water, 1 MV	36 (60%) well water, 24 piped or surface water	4.57 (1.77–11.78)
Current main source of drinking water*	16 (29.1%) well water, 39 piped or surface water, 1 MV	5 (8.3%) well water, 55 piped or surface water	4.51 (1.53–13.35)
Weaning			
Age of weaning from breast milk (months)	Median, 36.0; IQR, 24.0–36.0	Median, 28.0; IQR, 24.0–33.5	1.14 (1.06–1.22)
Bananas used in weaning*	9 (17.6%) yes, 42 no, 5 MV	24 (40%) yes, 36 no	0.32 (0.13–0.78)
Cows' milk used in weaning*	35 (68.6%) yes, 16 no, 5 MV	34 (56.7%) yes, 26 no	1.67 (0.77–3.65)
Diet			
Magadi used in cooking throughout childhood*	21 (41.2%) yes, 30 no, 5 MV	8 (13.8%) yes, 52 no	4.55 (1.80–11.53)
Magadi used in cooking now*	38 (73.1%) yes, 14 no, 4 MV	29 (48.3%) yes, 31 no	2.91 (1.31–6.42)
Ate beans throughout childhood*	39 (75.0%) yes, 13 no, 4 MV	55 (91.7%) yes, 5 no	0.27 (0.09–0.83)
Ate spinach throughout childhood*	42 (80.8%) yes, 10 no, 4 MV	56 (93.3%) yes, 4 no	0.30 (0.09–1.02)
Age started drinking tea (years)	Median 5.0, IQR 3.5–12.0, 11 MV	Median 12.0, IQR 6.0–12.0, 1 MV	0.96 (0.91–1.00)
Number of cups of tea per day†	6 (12.8%) none, 36 (76.6%) one to two cups, 5 (10.6%) three to four cups, 9 MV	37 (62.7%) none, 21 (35.6%) one to two cups, 1 (1.7%) three to four cups, 1 MV	8.68 (3.47–21.68)

IQR, interquartile range; MV, missing value.
*Yes = 1, no = 0.
†0 = less than one cup per day, 1 = 1–2 cups per day, 2 = 3–4 cups per day.

Table 4 Logistic regression model for juvenile skeletal fluorosis from the case–control study

	Model coefficient	Standard error	Significance	Odds ratio (OR)	95% CI for OR	
					Lower	Upper
Body mass index	−0.538	0.165	0.001	0.58	0.42	0.81
Cups of tea (coded)*	3.990	0.913	<0.001	54.08	9.03	323.71
Drank mainly well water 3 years ago†	3.234	0.965	0.001	25.39	3.83	168.22
Bananas used in weaning†	−1.674	0.781	0.032	0.19	0.04	0.87
Magadi used in cooking as a child†	2.635	0.847	0.002	13.95	2.65	73.34
Constant	3.060	2.301	0.184	21.32		

*0 = less than one cup per day, 1 = 1–2 cups per day, 2 = 3–4 cups per day.
†1 = yes, 0 = no.

were drinking surface water and 146 (53.1%) were drinking from at least one of the 13 wells in the village which had been dug 11 and 19 years earlier (in 1990 and 1998). The fluoride levels at the time of analysis ranged from 9.3 to 35.0 mg/l from well sources, 2.1–9.5 mg/l from surface water sources and the piped water contained 0.2 mg/l of fluoride.

Discussion

Prevalence of juvenile skeletal and dental fluorosis

Our population-based study reveals the effect on health that water containing very high fluoride can have in northern Tanzania. Deforming JSF was present in 4.4% of study participants, particularly in adolescent males. DF

in the permanent dentition was endemic in this population, suggesting a universally high-fluoride exposure over time. Primary-dentition DF was less prevalent; the primary teeth are formed *in utero* and early life, before there has been significant exposure to fluoride [breast milk is known to contain low concentrations of fluoride (Mosha & Jorgen 1983)]. The variation in the prevalence and severity of DF with age (with the highest TFI scores and highest prevalence seen in those aged 10–25 years) and the restriction of JSF cases to those under 30 years of age may be related to crucial periods of high-fluoride exposure during tooth and skeletal development in those age groups. The wells in the village were dug between 1990 and 1998, and those older than 30 years would have completed dental and skeletal development before this sudden increase in exposure to very high levels of fluoride. The population results appear to show a loose correlation between the ages most at risk of deforming JSF and higher severity of DF, with the TFI score highest in those aged 10–25 years, the age group where the majority of JSF cases were also found.

The case–control data do not support a more direct correlation, with no difference being found between the cases and controls in average TFI score. The TFI scoring system was developed in populations exposed to much lower levels of fluoride (Thylstrup & Fejerskov 1978). It may be that with water concentrations of fluoride as high as 30 mg/l, this scoring system is not sensitive enough to distinguish subtle differences between people who have severe DF. Our study suggests that JSF may be more common in males than females; this phenomenon has been noted by other authors (Christie 1980; Krishnamachari 1986; Chakma *et al.* 1997). It may be due to the protective effect of oestrogen on the developing skeleton, although the exact reasons remain unclear. A significant proportion of the cases had at least one sibling who was also classified as a case. This raises the possibility that genetic factors may play a role in the likelihood of developing deforming JSF. This has not previously been described.

Sources of fluoride

Water consumption from sources with excessively high-fluoride concentrations (principally well and bore-hole water) remains common in many parts of the world, including this region of Northern Tanzania. As a result of the scale of the problem, the Tanzanian government decided in 1974 on 8 mg/l as an acceptable safe fluoride level to mitigate the most severe health effects of high-fluoride exposure (Ministry of Water, Energy & Minerals (MAJI) 1974). Lack of infrastructure, resources and awareness means that many areas have no clean, piped, low-fluoride water. Nevertheless, as a result of the previous work by our group in Tindigani, local awareness of the problem with drinking from a non-piped water supply is now relatively high. This is reflected in the large increase in those using low-fluoride piped water now compared to 3 years ago (Table 3). This may also have contributed to the lack of DF seen in the younger ages. The situation has been further improved as this study took place with a low-fluoride piped supply now being provided within the village. This was as a direct result of discussions with village elders and the forming of a committee to liaise with the district water authority.

Case–control study

In the multivariate analysis, a number of risk factors for JSF emerged. As expected, predominantly drinking well water (rather than low-fluoride piped or ground water) was a significant independent risk factor, which is modifiable. As has been previously reported in relation to DF, magadi consumption, particularly consumption regularly throughout childhood, seems to be an important independent risk factor contributing to the development of JSF. Other dietary factors may be of importance in the development of deforming disease, either because they increase the load of fluoride that the body is exposed to, or provide a protective effect. When taking dietary histories, it became clear that the staple diet in Tindigani was 'ugali', a maize-based porridge that is used from weaning onwards. In some families, this was served with bananas (particularly during weaning), spinach or beans but only on an infrequent basis.

Being weaned on bananas came out as an independent protective factor. Although the reasons for this are not entirely clear, it may reflect the relatively high calcium levels found in bananas. It is also possible that children who were weaned on bananas consequently consumed less 'ugali' during early life. As 'ugali' is made with boiling water, it is likely to have a relatively high-fluoride content.

In univariable analysis, eating both beans and spinach on a regular basis (more than once a week) was protective. This may reflect beans being one of the few regular sources of protein (meat was eaten on special occasions only) and also may represent a marker of better nutritional status overall. Owing to the difficulty, recalling exact diet over a long period of time, it was only possible to obtain a rough estimation of calcium and protein intake, but many families (particularly amongst the cases) were essentially only eating 'ugali' and, therefore,

likely to be calcium and protein deficient. We do not think that vegetables themselves would have been a major source of fluoride. The people in the village were of predominantly Maasai tribal origin. They raised animals and did not generally grow their own vegetables. Vegetables were bought locally on a very irregular basis, and we cannot be sure whether the water used to irrigate these vegetables was from high-fluoride containing wells.

Low BMI also emerged as a significant predictor of outcome, although whether a low BMI represents a marker of poor nutritional status that is a causative factor in developing JSF or whether low BMI is a consequence of having SF remains unclear. Those who drank more tea per day had an increased risk of JSF. This is most likely to be due to increased concentration of fluoride ion, which occurs during the boiling of water and also the possibility of high concentrations of fluoride in the tea leaves used (Gulati *et al.* 1993). The tea consumed was regionally grown black tea. Some of these factors may help explain why there is variation in the penetrance of skeletal changes seen in exposed populations.

The study had one main limitation. As with any case–control study, much of the data collected rely on subjective recall of previous exposures to risk factors and this can be subject to recall bias. Cases and their relatives are often more likely to recall exposure to a known risk factor than controls. However, we feel that the effect of this is minimal in our study because both cases and controls were relatively young and were asked to recall events only a few years previously. Furthermore, in the absence of specialised knowledge, many of the risk factors considered will have had no apparent association with fluorosis.

Conclusions

Within this population exposed to very high-fluoride levels, JSF is a common and preventable public health problem. DF is also an endemic problem. Providing clean, low-fluoride, piped water to affected communities is of obvious health benefit and the ideal solution. However, this is not always realistic. A number of factors were identified as being associated with greater odds of developing JSF. Most of these risk factors are modifiable with small changes in behaviour and diet and may be important to other remote villages with little prospect of a low-fluoride piped water supply. Of particular importance is the finding that weaning using bananas may have a preventative role. This is likely to be a cost effective and easily implementable change even in remote communities. Community leaders, politicians and healthcare workers need to be aware of the causes of JSF. Initiatives to educate residents regarding risk factors for JSF and how to avoid them should be encouraged. In areas with a piped water supply, fluoride levels should be monitored regularly.

Acknowledgements

We would like to thank all subjects, and their relatives, who participated in this study. We would also like to thank Jane Rogathi for carrying out the translation of the questionnaires. We would also like to thank Professor Sandy Cairncross of the London School of Hygiene and Tropical Medicine for supervision of HJ during the work described. This study was part funded by a grant for HJ from the Denis Burkitt Foundation.

References

Adult Morbidity and Mortality Project (AMMP) (2004) Policy Implications of Adult Morbidity and Mortality; final report. Available from: http://research.ncl.ac.uk/ammp/finrep/. Accessed 3rd April 2010.

Ayoob S & Gupta AK (2006) Fluoride in drinking water: a review on the status and stress effects. *Critical Reviews in Environment Science and Technology* **36**, 433–487.

Bo Z, Mei H, Yongsheng Z, Xueyu L, Xuelin Z & Jun D (2003) Distribution and risk assessment of fluoride in drinking water in the west plain region of Jilin province, China. *Environmental Geochemistry and Health* **25**, 421–431.

Chakma T, Singh SB, Godbole S & Tiwary RS (1997) Endemic fluorosis with genu valgum syndrome in a village of district Mandla, Madhya Pradesh. *Indian Journal of Pediatrics* **34**, 232–236.

Chernet T, Travi Y & Valles V (2001) Mechanism of degradation of the quality of natural water in the Lakes Region of the Ethiopian rift valley. *Water Research* **35**, 2819–2832.

Christie DP (1980) The spectrum of radiographic bone changes in children with fluorosis. *Radiology* **136**, 85–90.

Fawell J, Bailey K, Chilton J, Dahi E, Fewtrell L & Magara Y (2006) Fluoride in Drinking-Water: WHO Drinking-water Quality Series World Health Organization, Geneva, Switzerland.

Fejerskov O, Manji F & Baelum V (1988) Dental Fluorosis – A Handbook for Health Workers. Munksgaard, Copenhagen.

Gulati P, Singh V, Gupta MK, Vaidya V, Dass S & Prakash S (1993) Studies on the leaching of fluoride in tea infusions. *Science of the Total Environment* **138**, 213–221.

Gupta SK, Gambhir S, Mithal A & Das BK (1993) Skeletal scintigraphic findings in endemic skeletal fluorosis. *Nuclear Medicine Communications* **14**, 384–390.

Haimanot RT, Fekadu A & Bushra B (1987) Endemic fluorosis in the Ethiopian Rift Valley. *Tropical and Geographical Medicine* **39**, 209–217.

Krishnamachari KA (1986) Skeletal fluorosis in humans: a review of recent progress in the understanding of the disease. *Progress in Food & Nutrition Science* **10**, 279–314.

Mabelya L, Konig KG & van Palenstein Helderman WH (1992) Dental fluorosis, altitude, and associated dietary factors (short communication). *Caries Research* **26**, 65–67.

Mabelya L, van Palenstein Helderman WH, van't Hof MA & Konig KG (1997) Dental fluorosis and the use of a high fluoride-containing trona tenderizer (magadi). *Community Dentistry and Oral Epidemiology* **25**, 170–176.

Ministry of Water, Energy and Minerals (MAJI) (1974) Temporary Standards of Quality of Domestic Water Supply in Tanzania. Rural Water Supply Health Standards Committee, Dar es Salaam, Tanzania.

Mithal A, Trivedi N, Gupta SK, Kumar S & Gupta RK (1993) Radiological spectrum of endemic fluorosis: relationship with calcium intake. *Skeletal Radiology* **22**, 257–261.

Mosha HJ & Jorgen L (1983) Dental caries, oral hygiene, periodontal disease and dental fluorosis among school children in northern Tanzania. Oral health surveys. *Odonto-Stomatologie Tropicale* **6**, 149–156.

Sass P & Hassan G (2003) Lower extremity abnormalities in children. *American Family Physician* **68**, 461–468.

Shorter JP, Massawe J, Parry N & Walker RW (2010) Comparison of two village primary schools in northern Tanzania affected by fluorosis. *International Health* **2**, 269–274.

Teotia M, Teotia SP & Singh KP (1998) Endemic chronic fluoride toxicity and dietary calcium deficiency interaction syndromes of metabolic bone disease and deformities in India: year 2000. *Indian Journal of Pediatrics* **65**, 371–381.

Thylstrup A & Fejerskov O (1978) Clinical appearance of dental fluorosis in permanent teeth in relation to histologic changes. *Community Dentistry and Oral Epidemiology* **6**, 315–328.

UNICEF (1999) A future global agenda for children: the links with sanitation, hygiene, water and environment. *Waterfront* **13**, 1–28. http://www.unicef.org/wash/files/wf13e.pdf (Accessed 5th January 2012).

World Health Organization (2004) Guidelines for Drinking-Water Quality, 3rd edn. World Health Organization, Geneva, Switzerland.

Corresponding Author **Helen G. Jarvis**, Department of Medicine, North Tyneside General Hospital, Rake Lane, North Shields, Tyne and Wear NE29 8NH, UK. Telefax: +44 191 293 2709; E-mail: hgjarvis77@hotmail.com