COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

COT STATEMENT ON FLUORINE IN THE 1997 TOTAL DIET STUDY

Introduction

1. In 2000 the COT considered the results of a study conducted by the Food Standards Agency in which samples collected in the 1997 Total Diet Study (TDS) were analysed for the presence of fluorine, bromine and iodine. The Committee concluded that the total dietary intakes of bromine and iodine estimated from the survey were unlikely to pose a risk to health[1]. However, consideration of fluorine was deferred as the toxicity of this trace element was due to be considered by the *ad hoc* Expert Group on Vitamins and Minerals (EVM). The final report of the EVM was published in May 2003. The EVM concluded that fluoride was not within its remit as food fortification with fluoride is carried out as a public health measure. Determination of maximum levels of supplementation therefore has to take place within the context of local exposure and involves a consideration of risks and benefits, which was not in the terms of reference of the EVM. The EVM report is available at http://www.food.gov.uk/science/ouradvisors/vitandmin/.

Background

2. Fluorine is a trace element which is ubiquitous in the environment and is present at low levels in all plants and animals. The analytical method used in this survey did not distinguish between different forms of fluorine. Elemental fluorine is a highly reactive gas, and the ionic form fluoride is present in food. Therefore this statement considers the toxicity of fluoride, and uses the term fluoride throughout for ease of clarity.

3. Based on studies in animals, fluoride was considered by a WHO expert committee to be necessary for animal life [2]. However, although low intakes of fluoride in humans are associated with increased incidences of dental caries and general weaknesses of bones and teeth, a true fluoride deficiency state has not been documented. Human requirements have therefore not been determined.

4. Fluoride has a well-documented beneficial effect in protecting against dental caries [3]. Systemic exposure to fluoride during the pre-eruptive development of teeth results in its incorporation into the enamel matrix of the tooth, forming an enamel which is more resistant to acid decay. Post-eruption,

fluoride has a beneficial topical effect, apparently by reducing enamel demineralisation and promoting remineralisation, and by inhibiting plaque acid-producing bacteria. For these reasons many dental products and some public water supplies are artificially fluoridated.

Toxicity of fluoride

Dental fluorosis

5. The most sensitive effect of excessive fluoride exposure in humans is considered to be dental fluorosis, a developmental defect of the tooth enamel. Dental fluorosis is caused by the over-incorporation of fluoride into the dental enamel to the effect that the composition and structure of the enamel are altered.

6. Dental fluorosis may be classified, using Dean's classification, as very mild, mild, moderate or severe. Pictures of the various forms of dental fluorosis can be viewed on pages 32-34 of the book 'Health Effects of Ingested Fluoride', by the US National Research Council [4], which is available to view electronically at http://books.nap.edu/books/030904975X/html. In its mildest forms, dental fluorosis presents as a barely visible white mottling of the teeth, which may not be apparent to the affected individual and is not considered to be aesthetically significant. The dental integrity of mild to moderately fluorosed teeth is not affected, and they may be more resistant to acid decay than non-fluorosed teeth [5]. Moderate and severe effects of dental fluorosis include more noticeable white mottling, yellow/brown staining and pitting of the enamel [6].

7. Data from epidemiological studies suggest that the enamel tissue is most susceptible to fluoride-induced changes during the third or fourth years of life for the permanent anterior teeth and at 22-26 months for the maxillary central incisors, which is when the enamel is in the transitional or early maturation stages [7]. The maxillary central incisors are the two teeth most visible when smiling and therefore fluoride-induced changes in these teeth would be of most concern. The pre-eruptive maturation of the permanent teeth is completed by the age of 8 years, and children over the age of 8 years and adults are not susceptible to dental fluorosis.

8. Epidemiological studies by Dean (1942)[8] showed that in populations with drinking water containing about 2 mg/L fluoride, less than 5% of the population had moderate dental fluorosis. According to the US Food and Nutrition Board, total fluoride intakes from food and water in these populations were estimated as 0.08-0.12 mg/kg bw/day[5]. Fluoride-containing dental products and supplements were not available at the time. In populations with water containing close to 1 mg/L fluoride, a low prevalence of very mild to mild dental fluorosis (10-12%) and no cases of moderate dental fluorosis were observed[8] (estimated average total fluoride intake, 0.05 mg/kg bw/day[5]). An

intake of 0.05 mg/kg bw/day is therefore assumed to be a no observed adverse effect level (NOAEL) for moderate dental fluorosis.

9. The prevalence of aesthetically significant fluorosis (moderate or severe) is estimated to be 3-4% in areas of the UK where the drinking water is artificially fluoridated and 0.5-1% where it is not artificially fluoridated[3].

Skeletal fluorosis

10. Symptomatic or clinical skeletal fluorosis is a condition characterised by skeletal abnormalities and joint pain. It is caused by pathological bone formation due to the mitogenic action of fluoride on osteoblasts. In its more severe forms, skeletal fluorosis causes kyphosis, crippling and invalidism. Secondary neurological complications in the form of myelopathy, with or without radiculopathy, may also occur.

11. Clinical skeletal fluorosis is endemic in regions of the world which have high fluoride levels in the water (up to 18 mg/L in 15 states of India) and hot, dry climates. In such climates clinical skeletal fluorosis has been associated with consumption of water containing fluoride levels as low as 1.5 mg/L [9]. However, studies conducted in the US in the 1950s indicate that in more temperate climates, no cases of clinical skeletal fluorosis were associated with fluoride levels up to 4 mg/L in drinking water [10]. The reason for the difference is uncertain, but it is likely to be largely due to the increased consumption of water in hot, dry climates. Dietary differences and fluoride exposures from other sources may also have contributed to the difference. There is no evidence of clinical skeletal fluorosis arising from exposures in the UK.

Other skeletal effects

12. A number of studies have investigated possible links of fluoride exposure, primarily through fluoridation of water, with fracture risk. Some studies reported a protective effect of increased fluoride exposure and others an increase in fracture incidence. In a recent meta-analysis of studies on bone fracture frequency and water fluoridation, no significant associations were found, except for studies of 10 years or longer, which showed a protective effect of water fluoridation on fracture risk [11].

13. A number of studies in humans and animals have investigated a possible relationship between fluoride intake and incidence of osteosarcoma because fluoride accumulates in bone and has a mitogenic action on osteoblasts.

14. The Committee on Mutagenicity of Chemicals in Food, Consumer Products and the Environment (COM) reviewed the mutagenicity of fluoride in 1990. The COM concluded that although *in-vitro* mutagenic effects were seen at relatively high concentrations, the activity was considered to be indirect and unlikely to occur at low concentrations. All well-conducted *in-vivo* mutagenicity tests were negative. The COM therefore concluded that the consumption of

fluoridated water would not constitute a mutagenic hazard to man. This view was endorsed in 1995 when some additional studies were considered [12].

15. The Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (COC) considered the available epidemiology and animal bioassay data in 1990, including the US National Toxicology Program (NTP) 2 year carcinogenicity study in F344/N rats and B6C3F1 mice, with drinking water concentrations up to 175 mg/L sodium fluoride (79 mg/L fluoride ion) [13]. The COC concluded that there was no evidence to indicate any carcinogenic risk to humans from exposure to fluoride [14]. There have been no data published since then to warrant seeking a further view from the COC.

Renal toxicity

16. Fluoride nephrotoxicity has been investigated in a number of animal species but the observed effects have generally been subtle, e.g. dilatation of the renal tubules and increased diuresis. No renal effects were observed in the 2-year NTP studies in rats and mice given drinking water containing 79 mg/L fluoride, providing intakes of approximately 7.9 and 15.8 mg/kg bw/day, respectively [13].

17. No renal disorders have been identified in humans in areas of endemic dental and skeletal fluorosis. One report exists of a single case of renal failure occurring in an individual who consumed, over a long period of time, large amounts of a mineral water containing 8.5 mg/L fluoride. Osteosclerosis was also diagnosed and the effects were attributed to the fluoride. However, it was not possible to calculate the fluoride intake of this individual[15].

Cardiac effects

18. Research conducted in China has shown that the percentage of patients with abnormal electrocardiograms (ECG) increased with increasing severity of skeletal fluorosis symptoms [16]. The relevance of this to fluoride exposure in the UK is unclear.

Reproductive effects

19. Fluoride has been shown to be toxic to the male reproductive system following administration to rats at 9 mg/kg bw/day for 29 days (as sodium fluoride, given by oral gavage). Effects on testis, prostate and seminal vesicle weights, reduced plasma testosterone levels, reduced epididymal sperm counts and reduced testicular Ä5,3β-hydroxysteroid dehydrogenase and 17β-hydroxysteroid dehydrogenase activities have been reported. Histological findings included dilatation of seminiferous tubules and reduced numbers of mature luminal spermatozoa[17]. However, other studies in rats and rabbits have shown no adverse effects at higher doses (8.1-9.5 and 27 mg/kg bw/day in rats and 18 mg/kg bw/day in rabbits) given in the drinking water[18,19].

20. In humans, high fluoride intakes and symptoms of skeletal fluorosis have been associated with decreased serum testosterone levels[20]. The relevance of this observation to fluoride exposure in the UK is also unclear.

Neurotoxicity and neurobehavioural effects

21. Nine adult male Long-Evans rats were exposed for up to 52 weeks to drinking-water reported to contain 2.1 mg/L sodium fluoride (0.95 mg/L fluoride, or about 0.06 mg fluoride/kg bw/day)[21]. Although the administered fluoride is likely to have been insignificant in comparison to the fluoride content of the normal rat diet (which was not assessed), the authors reported statistically significant differences from a control group for brain aluminium, neuronal cell injury, and cerebral IgM and immunoreactivity for beta-amyloid. This study was seriously flawed by a high incidence of intercurrent infections[22] and mortality, which may have contributed to the findings.

22. The behavioural effects of pre-natal exposure to sodium fluoride have been studied in two experiments in Sprague-Dawley rats[23]. Pregnant dams were administered sodium fluoride by 9 subcutaneous injections of 0.13 mg/kg bw (0.06 mg fluoride per kg bw) in order to produce high peak plasma fluoride levels. In the first experiment, 1 or 2 injections per day were given to 7 dams on gestation days 14-18 inclusive. In the second experiment, 3 injections per day were given to 9 dams on gestation days 17-19 inclusive. Plasma fluoride levels were measured in pups at 3 and 9 weeks of age, and did not differ from levels in controls. Behavioural testing on the pups was undertaken at 9 weeks of age, leading to three measures of spontaneous behaviour (initiations, total time, and time structure). The only statistically significant difference from matched controls was for behavioural time structure, in males, in the GD 17-19 study.

23. These authors applied the same behavioural tests in a study of Sprague-Dawley rats provided with drinking-water containing fluoride at 0, 75, 100 or 125 mg/L from weaning at age 3 weeks, for 6-20 weeks[23]. At 75 mg/L (corresponding to a daily fluoride intake of about 3.8 mg/kg body weight), there was no statistically significant difference from controls. At 100 mg/L (corresponding to a daily fluoride intake of about 5 mg/kg body weight), differences in behaviour were found in females; no data on males at this dose were presented. The concentration of 125 mg/L (corresponding to a daily fluoride intake of about 6.3 mg/kg bw) was associated with reduced weight gain (8-17% reduction) and differences in behaviour, in both males and females. In a further study[23], adult male and female Sprague-Dawley rats were given drinking-water containing 100 mg/L fluoride for 6 weeks, from age 12 weeks; differences in behaviour were found in females but not in males.

24. Dose-related reductions in cell size and number of neurons in the hippocampus and dentate gyrus were noted in a study of adult female Swiss albino mice given drinking-water containing sodium fluoride at 0, 30, 60 and 120 mg/L for 30 days (5 animals per dose)[24]. It is not clear whether the concentrations were stated as fluoride or sodium fluoride; if the latter, they

correspond to fluoride concentrations of 0, 13.6, 27 and 54.3 mg/L, and estimated fluoride intakes of approximately 0, 2.7, 5.4 and 10.9 mg/kg bw per day). Adverse effects on motor co-ordination, swim endurance and maze skill were noted at the top dose but not at the lower doses.

25. No abnormalities were seen in the brain (frontal cortex and basal ganglia, parietal cortex and thalamus, cerebellum and pons) pituitary gland, and spinal cord, in F344N rats given up to 300 mg/L sodium fluoride or in B6C3F1 mice given up to 600 mg/l sodium fluoride in drinking-water for 6 months[13]. No abnormalities were seen in these tissues, or in sciatic nerve, in these strains of rat and mouse given up to 175 mg/L sodium fluoride in drinking-water for 2 years[13]. These studies did not include routine examination of the hippocampus, or specific neurobehavioural tests.

The 1997 Total Diet Study

26. The Total Diet Study (TDS) forms part of the Food Standards Agency's surveillance programme for chemicals in food. Analyses for metals and other elements are generally carried out every three years. However, 1997 was the first year since 1980 in which fluoride had been considered.

27. A total of 400 samples were collected from retail outlets in 20 locations throughout the UK. Each of the samples was prepared or cooked according to normal domestic practice at a central location. The samples were combined into 20 composite food groups, the proportion of each food in a food group reflecting its importance in the average UK diet (largely based on an average of three years previous consumption data from the National Food Survey). The fluoride present in each sample was diffused as hydrogen fluoride at room temperature in the presence of perchloric acid saturated with hexamethyldisiloxane. The released fluoride was absorbed into a trapping layer of sodium hydroxide, which was then dried, dissolved in water and the fluoride content determined by ion exchange chromatography[25].

Results of the Total Diet Study

28. The full results of the survey were published in a Food Surveillance Information Sheet[25]. The highest mean fluoride concentrations were found in fish (1.9 mg/kg) and beverages (1.1 mg/kg). The fluoride content of beverages largely reflects the fluoride content of the water used in their preparation. However, tea contains higher amounts as fluoride is selectively taken up from the soil by the tea plant. The high fluoride levels in fish are thought to originate mainly from the skeleton, as fluoride accumulates in the bones of fish and some canned fish contains small bones.

Dietary exposure to fluoride

29. The TDS data were used to estimate dietary exposure to fluoride. Using food consumption data from the National Food Survey, which is

updated every year based on household food purchases and so reflects changes in consumption patterns, the mean population intake of fluoride was estimated to be 1.2 mg/person/day (0.02 mg/kg bw/day for an average 60 kg person). Dietary exposure to fluoride was last estimated in 1984, when the mean population intake, calculated from concentrations of fluoride determined in selected food samples from the 1978, 1979 and 1980 Total Diet Studies, was estimated to be 1.8 mg/person/day. However, due to changes in the TDS design since 1981 and the limited number of samples that were used to estimate the intake in 1984, a direct comparison between the 1997 TDS and this earlier estimate cannot be made.

30. Mean and high level consumer intakes of fluoride for adults and children were estimated using consumption data from the National Diet and Nutrition Survey (NDNS) and are shown in Table 1. The highest intakes were for the 4 to 6 years age group, for whom high level dietary exposure was 0.06 mg/kg bw/day.

Table 1: Estimated dietary exposure to fluoride by children and adult consumers

Population group	Dietary exposure (mg/kg bw/day)	
	Mean	97.5th percentile
1½ to 4½ years[a]	0.023	0.053
4 to 6 years[b]	0.031	0.060
7 to 10 years[b]	0.024	0.047
11 to 14 years[b]	0.017	0.037
15 to 18 years[b]	0.015	0.034
Adults (19+ years)[c]	0.016	0.033

Notes

a. Food consumption data from the NDNS: children aged 1½ to 4½ years [26]
b. Food consumption data from the NDNS: young people aged 4 to 18 years [27]
c. Food consumption data from the 1986/87 Dietary and Nutritional Survey of British Adults [28]

Other sources of exposure

31. Drinking water is a notable source of fluoride. The regulatory limit for fluoride in the UK public water supply, defined by The Water Supply (Water Quality) Regulations 1989, is 1.5 mg/L. Most public water supplies contain less than 0.7 mg/L fluoride. However, 10% of the UK water supply is artificially fluoridated to a level of 1.0 mg/L as a public health measure to protect against dental decay.

32. The regulatory limit for fluoride in spring water and bottled drinking water, defined by the Natural Mineral Water, Spring Water and Bottled Drinking Water Regulations 1999, is 1.5 mg/L, the same as for tap water.

There is currently no regulatory limit on the amount of fluoride that natural mineral water may contain; however, an EC directive specifying a limit of 5 mg/L has been proposed, to apply from 1 July 2004 onwards. In a recent survey of 25 brands of bottled waters purchased in the UK, the maximum fluoride concentration identified was 0.37 mg/L [29].

33. Estimated fluoride intakes from water are 0.062, 0.033, 0.023 and 0.021 mg/kg bw/day for children aged 7 months to 4 years, 5 to 11 years, above the age of 12 years and adults, respectively. This assumes consumption of 0.8, 0.9, 1.3 and 1.5 L/day [30, 31] of water containing 1 mg/L fluoride and average body weights of 13, 27, 57 and 70 kg [30, 31], respectively for these age groups. Table 2 indicates total possible intakes from the diet and drinking water combined.

Table 2: Total possible fluoride intakes (mg/kg bw/day) from the diet and drinking water combined assuming water fluoride concentrations of 0.7 and 1.0 mg/L and mean or 97.5th percentile dietary intake

Population group	**Fluoride concentration of drinking water**			
	0.7 mg/L		1 mg/L	
	Mean intake (mg/kg bw/day)	97.5th %ile intake (mg/kg bw/day)	Mean intake (mg/kg bw/day)	97.5th %ile intake (mg/kg bw/day)
1½ to 4½ years	0.066	0.096	0.085	0.115
4 to 6 years	0.054	0.083	0.064	0.093
7 to 10 years	0.047	0.070	0.057	0.080
11 to 14 years	0.033	0.053	0.040	0.060
15 to 18 years	0.031	0.050	0.038	0.057
Adults	0.031	0.048	0.037	0.054

34. Few data are available on dietary exposure of infants to fluoride. This is likely to vary greatly between breast-fed and formula-fed infants, particularly where the formula is reconstituted using water with high fluoride content. Breast milk contains only trace amounts of fluoride and has been reported to provide less than 0.01 mg/day. In a survey conducted in Australia, fluoride contents of infant formulae reconstituted using deionised water ranged from 0.031 mg/L to 0.532 mg/L [32]. Assuming an average infant weight of 7 kg and consumption of formula reconstituted with 750 mL of water containing 1 mg/L fluoride, the intake of fluoride would range from 0.11 to 0.16 mg/kg bw/day. It is not known how relevant these data are to infant formulae on the UK market, but the water would provide the major contribution.

35. Dental products such as toothpaste and mouthwash generally contain added fluoride. Most toothpaste brands contain approximately 1000 mg/kg fluoride. Low-fluoride toothpastes for children contain 400-526 mg/kg fluoride.

Fluoridated mouthwashes typically contain 230 mg/kg fluoride; they are not recommended for use by children under the age of 6 years. Some of the toothpaste and mouthwash used will be ingested, especially by young children. The amount of toothpaste used varies considerably, as does the amount swallowed, but it has been suggested that children under the age of 4 use 0.2 – 0.5 g/day of toothpaste and swallow 50% on average [33]. If toothpaste containing 1000 mg/kg fluoride is used, fluoride intakes would be 0.1 to 0.25 mg/day (equivalent to 0.008 to 0.019 mg/kg bw/day, assuming an average body weight of 13 kg).

36. The use of fluoride supplements is recommended by the British Dental Association for infants and young children in areas where the water supply contains less than 0.7 mg/L water. The dosage recommended varies depending on age and the level of fluoride in the public water supply.

COT evaluation

37. The Committee considered that a study of pre-natal exposure to injected sodium fluoride in rats did not provide persuasive evidence of an effect on postnatal behaviour. Exposure of weanling and adult rats to fluoride in drinking-water at concentrations equivalent to doses of about 5 mg/kg bw/day was associated, in females only, with abnormalities in behaviour (not found at the lower dose of about 3.8 mg/kg bw/day); this dose was close to that which caused evident systemic toxicity (reduced weight gain) at about 6.3 mg/kg bw/day.

38. A study which found structural abnormalities in the brain of rats exposed to fluoride in drinking-water at concentrations equivalent to doses of about 0.06 mg/kg bw/day was seriously flawed by a high incidence of intercurrent infections and mortality. A brief account of a small short-term study in mice reported dose-related abnormalities in the hippocampus, at fluoride concentrations in drinking-water presumed to be equivalent to daily fluoride doses of about 2.7, 5.4 and 10.9 mg/kg bw, but behavioural abnormalities were found at the top dose only.

39. Neurotoxicity was not detected in rats and mice in well-conducted long-term studies which included fluoride concentrations in drinking-water higher than those used in the behavioural and neurotoxicity studies described above, but which did not include routine examination of the hippocampus, or specific neurobehavioural tests.

40. The Committee noted that the most sensitive effect in humans is dental fluorosis, which occurs in children under the age of 8 years. Mild and very mild forms of dental fluorosis are generally not considered to be aesthetically significant. Moderate and severe forms of dental fluorosis are characterised by more noticeable white mottling, yellow/brown staining and pitting of the enamel. The integrity of teeth with mild to moderate dental fluorosis is not affected, and the teeth may be more resistant to dental decay than non-fluorosed teeth. Research is needed to determine the impact of the cosmetic

effect of dental fluorosis on the affected individual, in order to determine whether the effects should be considered to be adverse.

41. An intake of 0.05 mg/kg bw/day has been reported to be a NOAEL for moderate dental fluorosis. This intake level was associated with a low incidence (10-12%) of very mild to mild dental fluorosis, which is not usually considered to be aesthetically significant. The threshold dose at which fluoride causes moderate or aesthetically significant dental fluorosis is 0 .1 mg/kg bw/day, based on studies in which less than 5% of populations exposed to intakes of fluoride in the range 0.08-0.12 mg/kg bw/day had moderate dental fluorosis.

42. Information on total fluoride intakes is limited. The data in Table 1 derived from the 1997 TDS show that dietary exposure of high level consumers aged 1½ to 6 years exceeds the NOAEL of 0.05 mg/kg bw/day by up to 20%. Taking into account other sources of exposure such as water and dental products, it is likely that a significant proportion of children under the age of 8 years have a total fluoride exposure above the NOAEL. Some of these children may be at risk of mild to moderate dental fluorosis, particularly during the third and fourth years during formation of the permanent anterior teeth. Because of the imprecise information on total exposure, it is not possible to predict the proportion that would exceed the threshold of 0.1 mg/kg bw/day, at which 5% of the exposed population would be expected to develop moderate (aesthetically significant) dental fluorosis. However, data on the prevalence of dental fluorosis in the UK indicate it to be low.

43. Breast milk contains only trace amounts of fluoride and has been reported to provide less than 0.01 mg/day. Based on the results of an Australian survey of fluoride concentrations in infant formula, intake in formula-fed infants could exceed the threshold for aesthetically significant dental fluorosis. However, although dental fluorosis may occur in the primary teeth, this may not lead to dental fluorosis of the permanent teeth if fluoride intakes have decreased by the time of the development and maturation of the dental enamel of the permanent teeth. Therefore infants may be at lesser risk than children aged 3 to 4 years.

44. There is a lack of studies to follow up long-term health outcomes of children with dental fluorosis. However, on the basis of the available information, the most sensitive effect of fluoride in children above the age of 8 years and in adults is clinical skeletal fluorosis. In regions with temperate climates, clinical skeletal fluorosis is not seen in populations with water fluoride concentrations below 4 mg/L. Other possible adverse effects of fluoride are seen at doses higher than those required to cause clinical skeletal fluorosis. It therefore appears unlikely that clinical skeletal fluorosis or any other adverse effects would occur in the general population from typical total fluoride intakes in the UK.

Conclusions

45. We *note* that a small number of studies of sodium fluoride in rodents have variously suggested abnormalities in behaviour, and structural abnormalities in the brain. These findings cannot be fully assessed without confirmatory studies. We note that neurotoxicity was not observed in well-conducted long-term studies in rodents at higher doses (more than 50 times the NOAEL in humans).

46. We *note* that the most sensitive effect of fluoride in humans appears to be dental fluorosis, which occurs in children under the age of 8 years. A total fluoride intake of 0.05 mg/kg bw/day represents a NOAEL for moderate (aesthetically significant) dental fluorosis.

47. We *consider* the results of this survey indicate that during formation of the permanent teeth, a small proportion of children may be at risk of moderate dental fluorosis due to dietary exposure to fluoride. However, we *note* that the prevalence of moderate dental fluorosis in the UK appears to be low.

48. We *note* that fluoride intakes of formula-fed infants may exceed the NOAEL for dental fluorosis, but *consider* that infants are at lesser risk because the critical time for development of aesthetically significant dental fluorosis is during formation of the permanent teeth.

49. We *note* that the integrity of teeth with mild to moderate dental fluorosis is not affected, and that the teeth may be more resistant to dental decay than non-fluorosed teeth. However, we *recommend* that more research is needed to determine the impact of the cosmetic effect of dental fluorosis on the affected individual and on any possible long-term health outcomes in people affected by dental fluorosis.

50. We *note* that more information is needed on total fluoride exposure, including intakes from toothpastes and mouthwashes.

51. We *conclude* that, based on the current information available and the dietary intakes estimated from the 1997 TDS, no adverse effects other than mild to moderate dental fluorosis would be expected to be associated with fluoride intake from food, either in adults or in children, at the intake levels in the UK.

COT Statement 2003/03
September 2003

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