The mystery of declining tooth decay

from Mark Diesendorf

Large temporal reductions in tooth decay, which cannot be attributed to fluoridation, have been observed in both unfluoridated and fluoridated areas of at least eight developed countries over the past thirty years. It is now time for a scientific re-examination of the alleged enormous benefits of fluoridation.

FLUORIDATION consists of raising the concentration of the fluoride ion F^- in water supplies to about 1 part per million (p.p.m.) with the aim of reducing dental caries (tooth decay) in children. In fluoridated areas, there are now many longitudinal (temporal) studies which record large reductions in the incidence of caries[1]. The results of these and of fixed time surveys have led to the 'fluoridation hypothesis', namely that the principal cause of these reductions is fluoridation.

Until the early 1980s, there had been comparatively few longitudinal studies of caries in unfluoridated communities. Only a small minority of the studies in fluoridated areas had regularly examined control populations, and there seemed to be little motivation to study other unfluoridated communities. But during the period 1979–81, especially in western Europe where there is little fluoridation, a number of dental examinations were made and compared with surveys carried out a decade or so before. It soon became clear that large reductions in caries had been occurring in unfluoridated areas (see below). The magnitudes of these reductions are generally comparable with those observed in fluoridated areas over similar periods of time.

In this article, these reductions are reviewed and attention is also drawn to a second category of caries reduction which cannot be explained by fluoridation. This category is observed in children described by proponents of fluoridation as having been 'optimally exposed', that is, children who have received water fluoridated at about 1 p.p.m. from birth. The observation is that caries is declining with time in 'optimally exposed' children of a given age. In some cases, the magnitudes of these reductions are much greater in percentage terms than the earlier reductions in the same area which had been attributed to fluoridation.

The problem of explaining the two categories of reduction goes well beyond the field of dentistry: contributions from nutritionists, immunologists, bacteriologists, epidemiologists and mathematical statisticians, amongst others, may be required.

Table 1 Studies reporting large reductions in dental caries in unfluoridated areas

Location		Years surveyed	References
Australia	Brisbane	1954, '77	2, 3
	Sydney	1961, '63, '67	4
Denmark	Various towns	1972, '79	53
Holland	The Hague	1969, '72, '75, '78	38
	Various towns	1965, '80	11
New Zealand	Auckland (parts)	1966, '74, '81	12
Norway	Various towns	1970, '80	54
Sweden	Various towns	1973, '78, '81	39
	North Sweden	1967, '77	55
United Kingdom	Bristol	1970, '79	56
	Bristol	1973, '79	56
	Devon	1971, '81	37
	Gloucestershire	Annually from 1964	37*
	Isle of Wight	1971, '80	57
	North-West England	1969, '80	58
	Scotland	1970, '80	59
	Shropshire	1970, '80	10
	Somerset	1975–79 annually	60
	Somerset	1963–79	61
United States	Dedham, Mass.	1958, '74	40
	Norwood, Mass.	1958, '72, '78	40
	Massachusetts: sample of schools	1951, '81	41
	Ohio	1972, '78	62

* Unpublished communication from J. Tee (1980), Area Dental Officer, Gloucestershire, to R. J. Anderson *et al.*[37]

Caries in unfluoridated areas

Table 1 lists over 20 studies which report substantial temporal reductions in caries in children's permanent teeth in unfluoridated areas of the developed world. In many of these cases, the magnitudes of these reductions are comparable with those observed in fluoridated areas and attributed to fluoridation.

Several of these studies give clues as to factors which are unlikely to be the main causes of the reductions. A comparison of the 1954 and 1977 dental health surveys in Brisbane[2,3] indicates to a reduction of about 50% in caries, as measured by the number of decayed, missing and filled permanent teeth (DMFT) per child and averaged over the age groups, in the 23-year period. The 1977 survey distinguished between children who took fluoride tablets regularly, irregularly or not at all. Although there were differences in caries incidences between the three categories (which could reflect factors unrelated to fluoride levels), even the "no tablet" group had on average 40% less caries experience than that recorded in 1954. So fluoride tablets were not the principal cause of the reductions observed in Brisbane.

The first Sydney study[4] showed that children with "naturally sound" teeth increased from 3.8% in 1961 to 20.2% in 1967 and 28% in 1972. The paper, which was titled enthusiastically "The Dental Health Revolution", was originally used widely to promote fluoridation in Australia. The authors stated that: "Almost certainly, the availability of fluoride both in tablet form and delivered through town water supplies has been the predominant factor. . . . These very large reductions represent a modern triumph of preventive health care"[4]. Yet the major proportion of the reported improvement had already occurred before Sydney was fluoridated in 1968. Moreover, no evidence was presented that fluoride tablets were widely used in the 1960s. Fluoride toothpaste was only introduced into Australia in 1967[3]. Although the index "naturally sound" teeth is unsuitable for more detailed

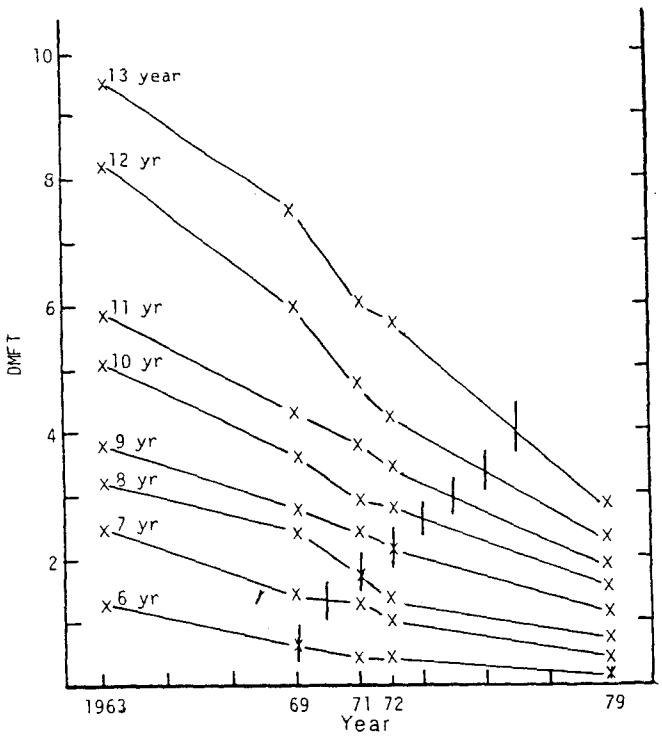

Fig. 1 Decline in caries, as measured by DMFT, in Tamworth, Australia, for children in age groups 6 years to 13 years. Data compiled from refs 14, 15. The vertical line cutting graph for each age group denotes year at which maximum possible benefit from fluoridation was reached. Tamworth was fluoridated in 1963.

studies which distinguish decayed, missing and filled teeth, the populations examined were very large (over 9,000 children at each examination) and the results clear-cut.

A second Sydney study[5] used the DMFT index, but was irrelevant for establishing any link with fluoridation, since it reported only on examinations in 1963 and 1982, but not around 1968 when Sydney was fluoridated. As in several other fluoridation studies, the key data were either not collected or not reported[6]. Although the two Sydney papers have an author in common (James S. Lawson, a senior officer of the New South Wales Health Commission), the second paper does not even cite the first. This suggests that, once it became clear that the first Sydney study contained evidence unfavourable to fluoridation, it was a source of embarrassment to some fluoridation proponents who are apparently trying to denigrate it.

However, independent confirmation of the large reductions in caries before fluoridation reported in the first Sydney study[4] is readily obtained by comparing the results of two surveys[7,8] separated by 20 years by Barnard. These surveys showed that the mean DIMF index ('I' denotes a permanent tooth which cannot be restored) for school children aged 13 and 14 declined from 11.0 in 1954–55 to 6.0 in 1972. The four years from 1968, when fluoridation commenced in Sydney, to 1972, would not have contributed significantly to the decline in caries prevalence in this age group[9].

The authors of one of the British studies[10] cited in Table 1 point out that sales of fluoride toothpaste in the United Kingdom were less than 5% of total sales in 1970, but rose to more than 95% of sales in 1977. They quote unpublished annual data from unfluoridated parts of Gloucestershire, collected from 1964 onwards, which show substantial improvements in children's teeth before the use of fluoride toothpaste became significant.

Many of the studies in the Netherlands, reviewed by Kalsbeek[11], were carried out to evaluate the effectiveness of the school dental health programme. Temporal reductions in DMFT of about 50% occurred between 1970 and 1980, whether or not the children had taken part in the dental health education program. Kalsbeek also reviewed the use of fluoride tablets and toothpaste and concluded from the data that "factors other than the effects of different fluoride programmes must play a role."

The study in the partly fluoridated city of Auckland, New Zealand[12], examined the influence of social class (which reflects environmental and lifestyle factors, such as diet) as well as fluoridation on dental health as measured by the levels of dental treatment received by children. The paper showed that treatment levels have continued to decline in both fluoridated and unfluoridated parts of the city and that these reductions are related strongly to social class, there being less caries in the "above average social rank" group than in other children. Thus the main ethical argument for fluoridation, that it should assist the disadvantaged, is not borne out by this study.

Fluoridation's benefits

On 15 December 1980, the Dental Health Education and Research Foundation, one of the main fluoridation promoting bodies in New South Wales (NSW), issued a press release entitled, "Fluoridation dramatically cuts tooth decay in Tamworth"[13]. This document, which highlighted results of a study conducted by the Department of Preventive Dentistry, Sydney University, and the Health Commission of NSW, stated in part:

> Tamworth's water supply was fluoridated in 1963, and the last survey in the area was conducted in August 1979. It shows decay reductions ranging from 71% in 15-year-olds to 95% in 6-year-olds.... All those surveyed were continuous residents using town water.

The "95%" reduction actually corresponded to a reduction in DMFT from 1.3 in 1963 to 0.1 in 1979[14], which is 92%. The press release implied incorrectly that all this reduction was due to fluoridation. However, it has been claimed ever since

Table 2 Extent of fluoridation in Australia, 1977 and 1983

State or territory	Capital city	Year city fluoridated*	% Of state fluoridated† in 1977	% Of state fluoridated† in 1983
ACT	Canberra	1964	100	100
Tasmania	Hobart	1964	74	77
NSW	Sydney	1968	81	81
WA	Perth	1968	83	83
SA	Adelaide	1971	71	70
Victoria	Melbourne	1977	0.7 then 73	71
Queensland	Brisbane	Not fluoridated	10	5

* Each capital city has the majority of the population of its state or territory.

† That is, the percentage of population of state/territory which drinks fluoridated water. Data from Annual Reports of Director-General of Health, for example ref. 17.

the commencement of fluoridation that the maximum possible benefits from fluoridation are obtained in children who have drunk fluoridated water from birth. Six-year-olds would have done this by 1969, when, according to the published data[15], they had a DMFT index of 0.6. The further reduction in caries in optimally exposed 6-year-olds, observed in years following 1969, cannot be due to fluoridation.

Thus, one can say that at best fluoridation could have approximately halved the DMFT rate in 6-year-olds between 1963 and 1969. (Since there was no control population, one could also say that at worst fluoridation might have had no effect in that period.) But from 1969 to 1979, caries in 6-year-olds was reduced a further 83%, by some other factor(s) than fluoridation.

Figure 1 shows that the unknown factors caused in children of each age from 6 years to 9 years similar large reductions in caries. Unfortunately, there are no published data for Tamworth beyond 1979 or in the years between 1972 and 1979, and so it cannot be confirmed whether the large reductions observed[14,15] from 1972 to 1979 in children aged 10 to 15 were also due to these unknown factors.

A similar reduction beyond the maximum possible for fluoridation is observed for children of each age from 6 to 9 in the published data from Canberra[16], which cover the period from 1964, the stated year of fluoridation, to 1974. In particular, DMFT rates declined by 50% in 6-year-olds from 1970 to 1974 and by 54% in 7-year-olds from 1971 to 1974. These reductions in optimally exposed children cannot be due to fluoridation. Published post-1974 data are needed to check on further reductions in optimally exposed children aged over 9 years.

From 1977 onwards, data have been systematically collected from the school dental services in each Australian state and territory[9,17]. Table 2 shows the degree of fluoridation in each of these states/territories in 1977 and 1983 and also the dates of fluoridation of the capital cities of these regions. Each of these cities dominates the population of the state or territory in which it lies. The evidence presented in Fig. 2 and Table 2 suggests that states and territories which had been extensively fluoridated for at least 9 years before 1977 (Tasmania, Western Australia and New South Wales) had qualitatively similar large reductions in caries from 1977 to 1983 as a state which was only extensively fluoridated in 1977 (Victoria) and a state which had a small and declining fraction of fluoridation (Queensland). Although the results of the school dental health survey are recorded by age and state, the data have only been published[9,17,18] so far for ages 6-13 averaged in each state, or for each age for the whole of Australia. There is evidence that the use of fluoride toothpaste in Australia reached a high plateau around 1978, so these observed reductions in caries can be due neither to fluoride toothpaste[9] nor to fluoridated water.

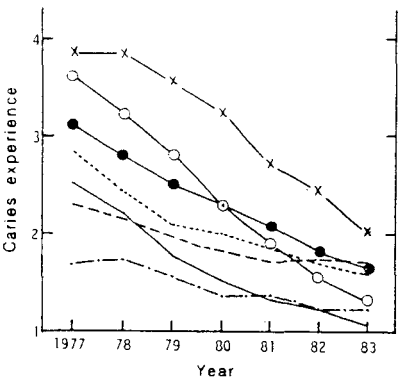

Fig. 2 Decline in the average number of (permanent) teeth per child with caries experience in each Australian state and the Australian Capital Territory as observed in school dental services[17]. 'Caries experience' can be one or more decayed, missing or filled teeth, and consists of an average for children aged 6-13 years. See Table 2 for information on the extent of fluoridation in each state/territory in 1977 and 1983 and the year when the main population centre of each state/territory was fluoridated. ×, Victoria; ○, Tasmania; ●, Queensland; - - - -, SA; ———, NSW; ——, WA; ·—·—·, ACT.

It is to be hoped that similar data on caries reductions in "optimally exposed" children will be sought in other fluoridated countries. In a region of Gloucestershire, United Kingdom where the main water supply was naturally fluoridated with 0.9 p.p.m. fluoride until 1972, reductions in caries of 51% were observed in 12-year-old children between 1964 and 1979[19]. Factors other than fluoridated water must have caused these reductions. After 1972, the main water supply was drawn from a bore with less than 0.2 p.p.m. fluoride, so a recent survey of caries there would be of great interest.

Benefits overestimated?

In some fluoridated areas (for example Tamworth, Australia), temporal reductions in caries have been wrongly credited to fluoridation. The magnitude of these reductions is similar in both fluoridated and unfluoridated areas, and is also generally comparable with that traditionally attributed to fluoridation. Can it be concluded that communities which prefer not to fluoridate, either because of concern about potential health hazards[20-25] or for ethical reasons (for example compulsory medication; medication with an uncontrolled dose), do not necessarily face higher levels of tooth decay than fluoridated communities? In other words, is it reasonable to ask whether it could be generally true that a major part of the benefits currently attributed to fluoridation is really due to other causes?

Such a hypothesis would seem to be possible in principle because it is well known that fluoridation is neither 'necessary' nor 'sufficient' (the words between inverted commas being used in the formal logic sense) for sound teeth; that is, some children can have sound teeth without fluoridation, and some children can have very decayed teeth even though they consume fluoridated water[25].

To confirm or refute the hypothesis, it is necessary (but not 'sufficient') to examine the absolute values of caries prevalence in fluoridated and unfluoridated areas. If it is true that the absolute values of caries prevalence in some unfluoridated areas are comparable with those in some unfluoridated areas of the same country, then the hypothesis is supported (but not proven), and there would be a strong case for the scientific re-examination of the epidemiological studies which appear to demonstrate large benefits from fluoridation.

The earliest set of studies comparing caries in fluoridated and unfluoridated areas were time-independent surveys of caries prevalence in areas with 'high' natural levels of fluoride in water supplies, conducted by H. T. Dean and others in the United States[26]. The surveys purported to show that there is an "inverse relationship" between caries and fluoride concentration. From the viewpoint of modern epidemiology, these early studies were rather primitive. They could be criticized for the virtual absence of quantitative, statistical methods, their nonrandom method of selecting data and the high sensitivity of the results to the way in which the study populations were grouped[25].

Results running counter to the alleged inverse relationship have been reported from time-independent surveys in naturally fluoridated locations in India[27], Sweden[28], Japan[29], the United States[30] and New Zealand[31,63]. The Japanese survey[29] found a minimum in caries prevalence in communities with water F-concentrations in the range 0.3-0.4 p.p.m.; above and below this range, caries prevalence increased rapidly.

These surveys[27-31] also selected their study regions nonrandomly. But recently Ziegelbecker[32] attempted to make a selection close to a random sample by considering 'all' available published data on caries prevalence in naturally fluoridated areas. His large data set, which includes Dean's as a sub-set, comprises 48,000 children aged 12-14 years drawn from 136 community water supplies in seven countries. He found essentially no correlation between caries and log of fluoride concentration. The surveys[27-32] are generally omitted from lists[1] of studies on the role of fluoridation in caries prevention.

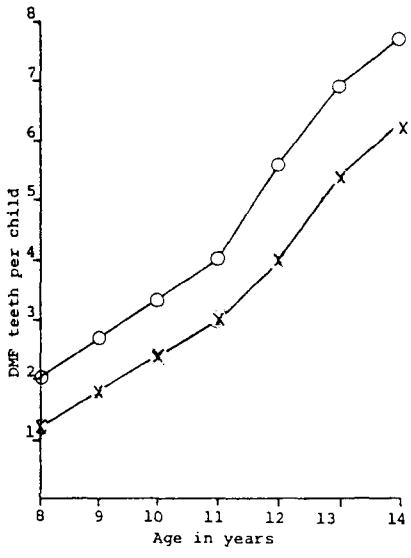

Fig. 3 The variation with age of decayed, missing and filled permanent teeth (DMFT) in fluoridated test towns (×) and unfluoridated control towns (○) in Britain, graphed from data published by the UK Department of Health[33]. Note that the rate of increase of DMFT is essentially the same in both groups. Children in the fluoridated areas have an average only one less cavity than children of the same age in the unfluoridated areas.

Further evidence can be drawn from Fig. 2. In 1983, the absolute value of caries prevalence in the Australian state of Queensland (which is only 5% fluoridated) was approximately equal to that in the states of Western Australia (83% fluoridated) and South Australia (70% fluoridated).

The classical British fluoridation trials at Watford and Gwalchmai were longitudinal controlled studies. In this regard they were better designed than the majority of other studies which have been conducted around the world. However, as in the case of almost all other surveys, the examinations were not 'blind'. The review of the British trials by the UK Department of Health after 11 years of fluoridation showed that children in fluoridated towns had approximately one less DMFT (that is, essentially one less cavity) than children of the same age in unfluoridated towns (see Fig. 3). The rate of increase in caries with age was the same in both populations[33].

Thus there are a number of counter-examples to the widely-held belief that "All studies show that communities where water contains about 1 p.p.m. fluoride have about 50% lower caries prevalence than communities where water has much less than 1 p.p.m. fluoride".

At this point the empirical data presented here may be summarized as follows. In the developed world:

(1) there have been large temporal reductions in caries in unfluoridated areas of at least eight countries;

(2) there have been large temporal reductions in several fluoridated areas which cannot be attributed to fluoridation;

(3) the absolute values of caries prevalence in several fluoridated areas are comparable with those in several unfluoridated regions of the same country.

Hence there is a case for scientific re-examination of the experimental design and statistical analysis of those studies which appear to prove or "demonstrate" that fluoridation causes large reductions in caries. Indeed the few re-examinations which have already been done confirm that there are grounds for concern.

The original justification for fluoridation in the United States, Britain, Canada, Australia, New Zealand and several other English-speaking countries was based almost entirely on the North American studies, which were of two kinds. The limitations of the first set, the time-independent surveys conducted in naturally fluoridated areas of the United States[26], have been referred to above.

The second set of North American studies consists of five longitudinal studies—carried out at Newburgh, Grand Rapids, Evanston and Brantford (two studies)—which commenced in the mid-1940s. Only three of them had controls for the full period of the study. These studies were criticized rigorously in a detailed monograph by Sutton[34], on the grounds of inadequate experimental design (for example, no 'blind' examinations and inadequate baseline measurement), poor or negligible statistical analysis and, in particular, failure to take account of large variations in caries prevalence observed in the control towns. The second edition of Sutton's monograph contains reprints of replies by authors of three of the North American studies and another author, together with Sutton's comments on these replies. It is difficult to avoid the conclusion that Sutton's critique still stands. Indeed, this was even the view of the pro-fluoridation Tasmanian Royal Commission[35]. Yet, in major, recent reviews of fluoridation, such as that by the British Royal College of Physicians[36], these North American studies are still referred to as providing the foundations for fluoridation, and Sutton's work[34] is not cited.

An examination has just been completed of the experimental design of all of the eight published fluoridation studies conducted in Australia. One (Tasmania) is a time-independent survey. Four (Townsville, Perth, Kalgoorlie and the second Sydney study) are longitudinal studies with only two examinations of the test group and either no control or only a single examination of a comparison group. The remaining three studies (Tamworth, Canberra and the first Sydney study) have several examinations of the test group, but no comparison group at all. Thus there has not been a single controlled longitudinal study in Australia. (M.D., to be published). Moreover, it has been shown above that three of the Australian studies (the first Sydney[4], Tamworth[14,15] and Canberra[16]) inadvertently provide evidence that some other factor(s) than fluoridation is/are playing an important role in the decline of caries prevalence.

Hence the hypothesis that fluoridation has very large benefits requires re-examination by epidemiologists, mathematical statisticians and others outside of the dental profession. The danger of failing to perform scientific research on the mechanisms underlying the large reductions in caries discussed in this paper is that the strong emphasis on fluoridation and fluorides may be distracting attention away from the real major factors. These factors could actually be driving a cyclical variation of caries with time[37]. It is possible that the condition of children's teeth could return to the poor state observed in the 1950s, even in the presence of a wide battery of F-treatments.

Causes of caries reductions

Many of the authors who reported the reductions in unfluoridated areas acknowledged that the explanation has not yet been determined scientifically[11,37-41]. It is after all much easier to perform a study which measures temporal changes in the prevalence of a multifactorial disease than to identify the causes of such changes.

Nevertheless, the authors of some of these studies have speculated that important causes of the reductions which they observe might be topical fluorides[38,53] (such as in toothpastes, rinses and gels), fluoride tablets[4,38], school dental health programmes[9], a lower *frequency* of sugar intake[39], the widespread use of antibiotics which may be suppressing *Streptococcus mutans* bacteria in the mouth[41], the increase in total fluoride intake from the environment[9,42], or a cyclical variation in time resulting from as yet unknown causes[37].

The present overview has revealed that several of the studies contain evidence against some of these proposed factors. We have seen that the Brisbane study[3] and

the Dutch review[11] suggest that fluoride tablets may not be important; the Sydney study[4], one of the British studies[10] and the Dutch review[11] each provides evidence against fluoride toothpaste; and the Dutch review[11] found no benefit in their school dental health education programmes.

Although there is evidence that fluoride toothpaste cannot be an important mechanism of caries reduction in some of the studies reported here, it must be stated that, unlike the case of fluoridation, there are also a few well-designed randomised controlled trials which demonstrate substantial reductions in caries from fluoride toothpaste[43]. Hence, the hypothesis can be made that topical fluorides sometimes improve children's teeth, although they are not necessary. So topical fluorides may comprise one of several factors contributing to the solution of the scientific problem of explaining the reduction in tooth decay.

Leverett[42] has speculated that the caries reductions in his smaller set of unfluoridated locations may be due to "an increase in fluoride in the food chain, especially from the use of fluoridated water in food processing, increased use of infant formulas with measurable fluoride content, and even unintentional ingestion of fluoride dentifrices." This hypothesis cannot explain the reductions in prefluoridation Sydney[4], or those in unfluoridated parts of Gloucestershire which started in the late 1960s[10]. The ingestion of fluoride toothpastes (and gels) by young children is well documented and could account for an intake of about 0.5 mg F^- per day in the very young[44]. But the food processing pathway is unlikely to be significant in western Europe where there is hardly any fluoridation, and infant formulas which are made up with unfloridated water will give only small contributions. Thus it appears that Leverett's hypothesis may at best be relevant to a minority of the studies listed in Table 1.

Here, the working hypothesis is presented that fluoridation and other systemic uses of fluoride, such as fluoride tablets, have at best a minor effect in reducing caries; that the main causes of the observed reductions in caries are changes in dietary patterns, possible changes in the immune status of populations and, under some circumstances, the use of topical fluorides. Indeed, a promising explanation is that the apparent benefit from fluorides is derived from their topical action. Then, since fluoridated water has a fluoride ion concentration 10^{-3} times that of fluoride toothpaste, its action in reducing caries is likely to be much weaker.

It is known that immunity plays a role in the development of caries, as it does with other diseases. Research is currently in progress to try to develop a vaccine against caries[45-47]. None of the data presented in the present paper provides evidence against immunity as a factor.

Dentists often argue against changes in dietary patterns as a major factor, on the grounds that sugar consumption has remained approximately constant in most developed countries over the past few decades. However, this is a simplistic argument. First, crude industry figures on total sales of sugar in developed countries contain no information on the distribution of sugar consumption with age and time of day. The form of sugar ingested—for example in canned food, soft drinks or processed cereals—may also be important. Second, tooth decay is increasing together with increases in sugar and other fermentable carbohydrates in the diet in several developing countries[48,49]. This was also the case with Australian aborigines, even when their water supplies consisted of bores containing fluoride at close to the "optimal" concentration for the local climate[50,51] Third, there is more to diet than sugar. For instance, there is some evidence, even conceded occasionally by pro-fluoride bodies[52], that certain foods which do not contain fluorides (for example wholegrain cereals, nuts and dairy products) may protect against tooth decay. So the whole question of the relationship between total diet and tooth decay needs much greater input from nutritionists and dietitians.

Perhaps the real mystery of declining tooth decay is why so much effort has gone into poor quality research on fluoridation, instead of on the more fundamental questions of diet and immunity.

The main body of this research was performed while the author was a principal research scientist in the CSIRO Division of Mathematics and Statistics, Canberra. □

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1. Murray, J. J. & Rugg-Gunn, A. J. *Fluorides in Caries Prevention* 2nd Edn (Wright, Bristol, 1982).
2. Kruger, B. J. *Aust. J. Dent.* **59**, 162-166 (1955).
3. McEniery, T. M. & Davies, G. N. *Community Dent. oral Epidem.* **7**, 42-50 (1979).
4. Lawson, J. S., Brown, J. H. & Oliver, T. I. *Med. J. Aust.* **1**, 124-125 (1978).
5. Burton, V. J., Rob, M. I., Craig, G. C. & Lawson, J. S. *Med. J. Aust.* **140**, 405-407 (1984).
6. Sutton, P. R. N. *Med. J. Aust.* **141**, 394-395 (1984).
7. Barnard, P. D. *NHMRC Spec. Rep. Ser. 8*, 30-43 (National Health & Medical Research Council, Canberra, 1956).
8. Barnard, P. D., Clements, F. W. *Int. dent. J.* **26**, 320-326 (1976).
9. Carr, L. M. *Aust. dent. J.* **28**, 269-276 (1983).
10. Anderson, R. J., Bradnock, G. & James, P. M. C. *Br. dent. J.* **150**, 278-281 (1981).
11. Kalsbeek, H. *J. dent. Res.* **61** (Special Issue), 1321-1326 (1982).
12. Colquhoun, J. *Community Dent. Oral Epidemiol.* **13**, 37-41 (1985).
13. Dental Health Education & Research Foundation, News Release, Sydney, 15 December 1980.
14. Barnard, P. D. *Dent. Outlook* **6**(4), 46-47 (1980).
15. Martin, N. D. & Barnard, P. D. *Dent. Outlook* no. 23, 6-7 (May, 1970); no. 33, 2-3 (May, 1972); no. 41, 6-7 (December, 1973).
16. Carr, L. M. *Aust. dent. J.* **21**, 440-444, (1976).
17. Australia, Director-General of Health, *Annual Report 1983-84*, Table 69 (1984); *Annual Report 1981-82*, Table 66 (1982). Australian Government Publishing Service, Canberra.
18. Carr, L. M. *Aust. dent. J.* **27**, 169-175 (1982).
19. Anderson, R. J. *Br. dent. J.* **150**, 354-355 (1981).
20. Rose, D. & Marier, J. R. *Environmental Fluoride, 1977* (National Research Council of Canada, Ottawa, 1977).
21. Waldbott, G. L., Burgstahler, A. W. & McKinney, H. L. *Fluoridation: the Great Dilemma* (Coronado, Lawrence, Kansas, 1978).
22. Tsutsui, T., Ide, K. & Maizumi, H. *Mutat. Res.* **140**, 43-48 (1984).
23. Tsutsui, T., Suzuki, N., Ohmori, M. & Maizumi, H., *Mutat. Res.* **139**, 193-198 (1984).
24. Tsutsui, T., Suzuki, N. & Ohmori, M. *Cancer Res.* **44**, 938-941 (1984).
25. Diesendorf, M. *Commun. Hlth Stud.* **4**, 224-230 (1980).
26. McClure, F. J. (ed.) *Fluoride Drinking Waters* (US Department of Health, Education & Welfare, Public Health Service, Bethesda, Maryland, 1964).
27. Amrit, T. & Joshi, J. L. *Conference Int. Soc. Fluoride Res.*, New Delhi, November, Abstr. 15 (1983).
28. Forsman, B., *Commun. dent. Oral Epidem.* **2**, 132-148 (1974).
29. Imai, Y., *Jap. J. Dent. Health* **22**, 144-196 (1972).
30. Zimmermann, E. R., Leone, N. C. & Arnold, F. A. *J. Am. med. Ass.* **50**, 272-277 (1955).
31. Hewat, R. E. T. *New Zealand dent. J.* **45**, 157-167 (1949).
32. Ziegelbecker, R. *Fluoride* **14**, 123-128 (1981).
33. UK Dept of Health, Report No. 122, HMSO, London (1969).
34. Sutton, P. R. N. *Fluoridation: Errors and Omissions in Experimental Trials* 2nd edn (Melbourne University Press, Melbourne, 1960).
35. *Report of the Royal Commission into Fluoridation of Public Water Supplies* (Government Printer, Hobart, Tasmania, 1968).
36. Royal College of Physicians, *Fluoride, Teeth and Health* (Pitman Medical, London, 1976).
37. Anderson, R. J., Bradnock, G., Beal, J. F. & James, P. M. C. *J. dent. Res.* **61** (Special Issue), 1311-1316 (1982).
38. Truin, G. J., Plasschaert, A. J. M., Konig, K. G. & Vogels, A. L. M. *Commun. Dent. oral Epidem.* **9**, 55-60 (1981).
39. Koch, G. *J. dent. Res.* **61** (Special Issue), 1340-1345 (1982).
40. Glass, R. L. *Caries Res.* **15**, 445-450 (1981).
41. DePaola, P. F., Soparkar, P. M., Tavares, M., Allukian Jr, M. & Peterson, H., *J. dent. Res.* **61** (Special Issue), 1356-1360 (1982).
42. Leverett, D. H. *Science* **217**, 26-30 (1982).
43. James, P. M. C., Anderson, R. J., Beal, J. F. & Bradnock, G. *Commun. Dent. oral Epidem.* **5**, 67-72 (1977).
44. Ekstrand, J. & Ehrnebo, M. *Caries Res.* **14**, 96-102 (1980); Ekstrand, J. & Koch, G., *J. Dent. Res.* **59**, 1067 (1980).
45. Lehner, T., Russell, M. W. & Caldwell, J. *Lancet* **i**, 995-997 (1980).
46. McGhee, J. R. & Michalek, S. M., *Am. Rev. Microbiol.* **35**, 595-638 (1981).
47. Smith, G. E., *Trends pharmac. Sci.* (in the press).
48. Newbrun, E. *Science* **217**, 418-423 (1982).
49. Sheiham, A. *Int. J. Epidemiol.* **13**, 142-147 (1984).
50. Barrett, M. J. & Williamson, J. J. in *Aust. dent. J.* **17**, 37-50 (1972).
51. Brown, T. in *Better Health for Aborigines* (eds Hetzel, B. *et al.*) 97-101 (University of Queensland Press, Brisbane, 1974).
52. Australian Nutrition Foundation, *Dent. Outlook* **11**(2), 47-51 (1985).
53. Fejerskov, O., Antoft, P. & Gadegaard, E., *J. dent. Res.* **61** (Special Issue), 1305-1310 (1982).
54. von der Fehr, F. R. *J. dent. Res.* **61** (Special Issue), 1331-1335 (1982).55. Mansson, B., Holm, A. K., Ollinen, I.
55. Mansson, B., Holm, A. K., Ollinen, I. & Grahnen, H. *Swed. dent. J.* **3**, 193-203 (1979).
56. Andlaw, R. J., Burchell, C. K. & Tucker, G. J. *Caries Res.* **16**, 257-264 (1982).
57. Mainwaring, P. J. & Naylor, N. M. *J. dent. Res.* **60**, 1140 (1981).
58 Mitropoulos, C. M. & Worthington, H. V. *J. dent. Res.* **60**, 1154 (1981).
59. Downer, M. C. *J. dent. Res.* **61**, 1336-1339 (1982).
60. Palmer, J. D. *Br. dent. J.* **149**, 48-50 (1980).
61. Anderson, R. J. *Br dent. J.* **150**, 218-221 (1981).
62. Zacherl, W. A. & Long, D. M. *J. dent. Res.* **58**, 227 (1979).
63. Hewat, R. E. T. & Eastcott, D. F., *Dental Caries in New Zealand*, pp. 75-76, 79 (Medical Research Council of New Zealand, 1955).