The decline of caries in New Zealand over the past 40 years

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SUMMARY

In New Zealand, as elsewhere, caries prevalence has declined since the 1950s; this has been accompanied by a change in the intra-orai pattern of the disease. This is illustrated by analysis of data for 12-year-old children. However, because treatment services for children in New Zealand are so comprehensive, the DMF index is primarily a count of restorations placed. This treatment overlay can distort the true caries prevalence and has been a confounding factor in assessment of the change in caries over time. Measurement of the fine gradations of ongoing change in the present low-caries-prevalence population requires the use of a more sensitive indicator than the DMF indices. When the timing of various forms of fluoride supplementation is correlated with the decline in caries, the decline continues beyond the time of maximum population coverage with fluoridated water and fluoridated toothpaste. Thus an explanation of the convergence of caries prevalence in fluoridated and non-fluoridated areas since the 1970s may require a re-assessment of the fluoride effect. This convergence, and the overall decline during the last decade without known additional fluoride supplementation, suggest that factors other than fluoride, such as food additives and antibiotics, may have contributed.

In the last 40 years, a dramatic reduction in caries prevalence in all sections of the New Zealand population has occurred. Despite similar changes occurring in many countries with different dental delivery systems, different public health programmes, and different cultures, the reasons for the continuing decline in caries prevalence continue to be debated.

The decline has affected all age groups in both fluoridated (F) and non-fluoridated (NF) areas in varying degrees but, because dental caries experience is cumulative, change can be most easily measured in children. This paper examines and discusses the decline in caries prevalence in the permanent teeth of 12-year-old New Zealand children with exposure to various forms of fluoride supplementation.

CARIES EPIDEMIOLOGY IN NEW ZEALAND

Before assessing the caries prevalence data, it is necessary to understand the way dental care was, until very recently, delivered to almost all school children by the School Dental Service (SDS).

In the early 1920s when dental disease was a major health problem, New Zealand introduced the SDS staffed by operating auxiliaries. The country was divided into a number of areas, each managed by a dentist under national directive. The auxiliaries were responsible for clinical decisions with indirect and, often, distant supervision. Early operative intervention in the carious process, then regarded as irreversible, reduced the need for tooth extraction. As the SDS pre-dated the publication of the DMF index, other ratios were used to monitor progress. The number of fillings placed per child per year in both deciduous and permanent teeth (F/C/Y) and, later, fillings in permanent teeth per child per year (FP/C/Y) were recorded until, in 1980, the DMF index and the percentage of children caries-free were used for statistical purposes.
Because of the emphasis on treatment and the almost total coverage of the child population, DMF scores are counts of restorations already placed; in New Zealand, the D and M components are negligible. The DMF index is, for all practical purposes, an F index determined by the treatment decisions of many uncalibrated operators. The variation which occurs in treatment decisions remains a major confounding factor in caries epidemiology. The effect of treatment prescription was dramatically illustrated in the SDS in 1970 following a directive to restore only lesions into dentine. The year the F/CY ratio was 33 percent lower and, in the years 1976-1981, the reduction was 64 percent. This is not likely to have been a change in disease prevalence, but rather a change in treatment prescription. Treatment differences occur not only between individual operators but also result from the philosophies of dental managers. For example, in 1978 in a fluoridated area, two clinics 2 km apart, but under the same dental manager and serving the same age and socio-economic groups recorded F/CY ratios of 1.3 and 4.7 respectively; in the same year the range between dental districts in F/CY was 1.0-1.4 in F areas, and 1.1-2.0 in NF areas.

The influence of treatment prescription on disease statistics is not a phenomenon peculiar to the SDS or to a particular time - it may occur wherever there are comprehensive treatment services. It is, however, a factor to be considered, especially in small surveys of the effects of public health programmes. National data and surveys, on the other hand, tend to cancel out excessive variability and provide more reliable information.

**EVIDENCE FOR THE DECLINE IN CARIES PREVALENCE IN NEW ZEALAND**

Early SDS data are unsuitable for identifying prevalence trends because of partial population coverage and the ratios used. However, from the 1960s, the two filing ratios and, from 1980, the DMF score, do indicate declining prevalence. Cross-sectional data from studies and surveys confirm the decline.

The F/CY ratio changed little until the change in the diagnostic criteria. Then the ratio declined sharply from 3.3 in 1976 to 1.5 F/CY in 1980, when it stabilised at about this level.

The effect of extensive treatment during childhood on adult dental health was recognised in the 1970s after two surveys of adult oral health and the introduction of the F/CY ratio for evaluation purposes. As with the F/CY, the F/CY/C ratio showed only a small decline between 1970 and 1975, and a sharp drop from 2.6 in 1976 to 1.0 by 1980, after which it remained static until it ceased to be used in 1985.

From 1980 onwards, national DMF data were recorded. The national DMF/CT declined from 5.1 in 1980 to 2.4 in 1988. By 1995 the score was 1.4 DMFT. The scores for F and NF areas converged during the same period. In F areas the DMF dropped from 3.0 in 1985 to 1.3 in 1995, and in NF areas from 3.4 to 1.5 (Figure). The reductions in both F and NF areas have been about 56 percent for the decade. The percentage of children caries-free at the end of SDS treatment increased from 7 percent in 1980 to 26 percent in 1985, and to 30 percent of children in 1995.

Declining prevalence has been accompanied by a change to a markedly skewed distribution curve with the median zero. The number of high-caries-experience children is low, only varying degrees of low experience. The distribution tail of high caries experience children has almost disappeared. Even by the 1988 survey, when the national DMF was still 2.4, only 1.7 percent of children had a DMF greater than 8. This was half the proportion in the 1982 survey only 6 years earlier, and in marked contrast to the 25 percent with a DMFT score greater than 10 in the 1977 survey.

Not only has overall caries prevalence declined greatly, but also the intra-oral pattern of the disease has altered. Even in the late 1970s, interproximal tooth surfaces were frequently carious at an early age. Compound restorations on molars were the norm and, for high-caries-experience children, the proximal surfaces of premolars and upper incisors were frequently affected. Since the 1980s these lesions have become progressively less common, and are rare in the 1990s.

The national monitoring surveys of 1977 and 1982, and the second ICS survey in 1988, verify the trends apparent in the statistical data collected nationally.

**REASONS FOR THE DECLINE IN CARIES PREVALENCE**

Acceptance of fluoride supplementation in various forms being the major reason for the decline in caries is widespread. Nevertheless, epidemiological evidence of change in prevalence does not correlate well with the timetable of the introduction of the various fluoride supplements.

**Water fluoridation**

Two studies monitored the effect of fluoridation in New Zealand - in Hastings from 1954 to 1964 and in Lower Hutt from 1959 to 1969. For 12-year-old children in these towns after 10 years of fluoridation, the DMF scores were respectively 4.3 (45 percent) and 3.7 (29 percent) lower than the historical pre-fluoridation baseline. In the Hastings study, Napier had been proposed as the control population, but the baseline examinations showed that the DMF in Napier was already lower than in Hastings. Diagnostic differences between operators were not considered a possible reason and were not included in the subsequent extensive, but inconclusive investigations of possible causes. The caries prevalence in Napier was regarded as an anomaly, although it was taken as axiomatic that there was in general, a considerable difference between the caries prevalence of children living in F and NF areas - numerous studies overseas had shown that fluoridation was a means of reducing caries prevalence.

A number of large urban areas introduced water fluoridation in the mid-1960s (Figure), but for the SDS did not collect data separately for F and NF areas. By the late 1960s, 54 percent of the population was using fluoridated water, the maximum coverage achieved. No dramatic change was evident in the national F/CY ratio, which was 3.6 in 1968, 3.5 in 1976, and 2.9 in 1975, 10 years after fluoridation had been introduced widely.

The effectiveness of fluoridation appeared evident in the 1977 national survey of 12- and 13-year-olds, children not yet affected greatly by fluoride toothpaste or the change in diagnostic criteria. The DMF score of 6.3 in the F area was of the same order as reported in the two fluoridation studies after 10 years of fluoridation, 5.2 and 6.4. The mean DMF of 7.8 in the NF area, however, was much lower than the DMF scores reported from Hastings.
FIG - DMFT of New Zealand children at about 12 years of age in relation to known fluoride supplementation. References to studies are superimposed. Circle, data from fluoridated areas; triangle, data from non-fluoridated areas.

Palmerston North and Lower Hutt before those towns were fluoridated in the 1950s[13]. The difference between the F and NF areas represented a mean of 1.5 teeth DMF (19 percent) which, although unexpectedly small, was still significant in the relatively high caries prevalence of that time. The mobility of the population, the lack of a true residential history, and the dissemination of fluoride in manufactured products were suggested as the reasons for the small difference. The 1982 survey showed only a 14 percent difference (0.6 DMF teeth), reflecting a greater fall in mean DMF in the NF areas, from 7.8 to 4.2, than in the F area, 6.3 to 3.6. The availability of fluoride toothpaste since the 1970s (Figure) may have reduced the difference in DMF between F and NF areas.

In addition, the change in diagnostic criteria in 1976 would also have reduced the DMF scores.

Since 1986, the national DMF data for F and NF areas has continued to converge in real terms; the 1995 difference was 0.3 DMF teeth (Figure), a 20 percent difference at the overall prevalence level of 1.4 DMF teeth in 1995. However, at such a low prevalence level, a 20 percent difference is clinically meaningless.

Fluoride tablets
At the time water fluoridation was introduced in 1955, the prevailing opinion was that the cariostatic effect of fluoride was achieved by systemic incorporation in the developing enamel. Fluoride tablet use by children up to 12 years of age was advocated in NF areas at a dosage of 1 mg/day to equate with the intake from water fluoridated at 1 ppm. However, the recommended dosage was reduced[15] after a survey of enamel defects in 1982[16]. From 1995, tablet use has been limited to professional prescriptions for high-risk individuals. Although fluoride tablets, regularly used, may be effective in reducing caries prevalence[14], most of the benefit can be explained by other dentally beneficial practices[17].

Topical fluorides
Topical fluoride was sometimes used in the SDS from 1950 but, from 1976, all children, except low-risk children using fluoridated water, were treated with a topical fluoride application of 2 percent sodium fluoride at each 6-month recall. Initially, the aim was to expose undiagnosed incipient lesions of enamel to fluoride, but later, fluoride was applied to diagnosed lesions of enamel as an empirical treatment. A controlled field trial of this empirical treatment showed no benefit[18].

Fluoride mouth-rinsing
Some school-based mouth-rinsing programmes were implemented prior to 1980. A 5-year double-blind study on high-caries-risk children, for whom operative intervention was determined by one examiner, failed to demonstrate any caries inhibition[19], although positive results had been reported overseas[20]. The conclusions of many mouth-rinsing studies are now questioned because historical baseline data, used in a period of declining caries prevalence, can give an illusion of benefit[21]. The New Zealand results have proved to be in line with the lack of effectiveness of mouth-rinsing as a public health measure in a moderate-caries-experience population[22].
Fluoride toothpaste

Fluoride toothpaste became readily available in the 1970s and dominated the market by the 1980s (Figure). Fluoride toothpaste is commonly linked to the decline in caries prevalence in the last 20 years. However, some children, especially among the more disadvantaged sections of the population, do not use toothpaste. In the most recent survey of 1,027 children, only 137 (13 percent) had a DMF score greater than 4, and just 17 of these had 8 or more teeth affected. Even for the 137 children in the tail of the distribution curve, the intra-oral pattern of caries was mainly of simple pit and fissure fillings. Fluoride toothpaste should have had its maximum expected effect by 1988, but a continued fall in the mean DMF score has been recorded yearly since that time (from 2.4 to 1.4), the number of children caries-free has increased, and there have been further changes in the intra-oral pattern.

Fissure sealants

Fissure sealants were introduced in the SDS in the late 1980s. Sealants protect the tooth surfaces least affected by fluoride, and provide a complementary public health adjunct to water fluoridation. However, the potential benefit is less with the decline in caries prevalence. The efficacy of the public health use of sealants on premolars is limited because of the low caries susceptibility of these teeth, a situation which may now apply to molars. At the present time, sealant use would be most effective when limited to high-caries-risk groups.

Although the effect has not been quantified, sealants may have contributed to some extent to the decline in prevalence in 12-year-old children since 1988. However, sealants would not have affected the intra-oral pattern.

DISCUSSION

In most scientific fields, accurate measurement is a necessary first step in investigations. However, the most sensitive index in use at present, the DMF surface index, does not identify the full magnitude of the change in caries prevalence. For example, small developmental pit lesions on the occlusal and buccal surfaces of molars are equated with large restorations involving occlusal and proximal surfaces. The differences in the maintenance requirements and the life expectancy of a tooth with such different restorations are vast, yet the score for both is 2. If continuing change is to be adequately monitored and the reasons for it established, a more sensitive index weighted to give value to the site and size of the lesion is necessary. Nevertheless, even from the relatively coarse DMFT index, alteration in the prevalence and nature of dental caries is marked.

Since the work of Dean, the inverse relationship between fluoride availability and the prevalence of dental caries has been generally accepted. Fluoride has been used in New Zealand in various ways, but only in water and toothpaste. It has had significant public-health impact. Until the mid-1980s, fluoride in water and toothpaste seemed to explain all the changes in caries prevalence. However, this inverse relationship has become less significant. When the timing of the introduction of the various forms of fluoride supplementation is examined against the trend lines for caries prevalence (Figure), the correlation found in the 1930s has lessened with time and, in the last decade, an inverse relationship has ceased to exist.

In the light of current knowledge about the cariostatic effect of fluoride, the preventive effect of water fluoridation should have become obvious nationally when 50 percent of the population was exposed to fluoridated water; the effect should probably have peaked for 12-year-old children by the early 1970s. However, the data available (PFIC/Y ratios), showed only a small reduction. The DMF scores for F and NF areas in 1977 also showed only a moderate benefit for children using fluoridated water, 1.5 less teeth DMF. The smaller than expected difference between F and NF areas was due to the decline being greater in NF than in F areas. The score for the NF area, when evaluated against the historical pre-flouridation data, also suggests that a decline was occurring prior to 1977. This interpretation of the 1977 data is not new; similar re-evaluations of the New Zealand pre- and post-flouridation data have already been published. Some evidence from other countries suggests an unrecognised early beginning to the decline, unrelated to fluoride. However, in New Zealand, all change in F areas was attributed to fluoridation, and in NF areas to its flow-on effects.

After 1982, fluoride toothpaste and the reduction in overprescription of restorative treatment had an increasing impact on caries prevalence. Certainly from this time the DMF scores for 12-year-old children in both areas lowered progressively and, in 1995, approximated each other with only an 0.3 DMF difference. Changes in the intra-oral pattern of caries and in the distribution curve began to be apparent from the late 1970s. Before fluoride toothpaste was generally available, many children had compound cavities on molars and premolars and lesions on anterior teeth despite use of fluoridated water; however, these gradually become less common in both F and NF areas. Toothpaste has possibly been a more successful vehicle for fluoride delivery than expected, but this does not explain the reduced prevalence in those sections of the population who do not use toothpaste. Other factors may be involved as, theoretically, fluoridated toothpaste should have had its maximum effect on the statistics for 12-year-old children by the late 1980s when it first saturated the market (Figure).

By the time fissure sealants started to be used in the SDS in 1988, the decline in caries prevalence was already well established, and they are unlikely to account for the recent prevalence changes.

Diet, particularly sugar, is a key factor in caries aetiology, but total sugar consumption levels appear to have remained static for most of the twentieth century. However, other dietary changes in the last 30 years have gathered momentum in the past decade. The types of foods eaten have changed and this has been linked to documented socio-economic changes in society. The shelves of supermarkets are crowded with pre-prepared and packaged convenience foods, many of which have added preservatives and colourings, some known to be bacteriostatic.

Food production techniques have also changed, and intensive farming often involves the use of antibiotics which are then in the food-chain. The use of antibiotics for childhood ailments is widespread and could add further to a dietary intake. Some evidence suggests that medicinal antibiotic use does have an effect on the oral flora. The current rarity of green stain also suggests some change in the oral flora over time. During examination of some thousands of primary school children in the 1980s, my subjective clinical impression was that mouths were becoming cleaner and pre-examination prophylaxis less
often necessary. If this impression has validity, it would be indicative, like green stain, of a more comprehensive change in the oral cavity than dental caries alone.

Whatever the reason, it is certain that, in 12-year-old children, caries prevalence almost halved between 1988 and 1995, from an already low level and without any known additional fluoride supplementation. This is one of the two outstanding anomalies in the caries prevalence data of the last 40 years. The other is the reduced prevalence in NF areas and the declining difference in DMF scores between F and NF areas. The rapidity and the extent of these changes, particularly during the past decade, require explanation and suggest either recent or intensifying contributory causes.

Knowing why there has been a decline in caries prevalence will become very relevant if the present trends are reversed. A return to the prevalence seen in children even a decade ago would present real problems now that money for health care is limited and the workforce fragmented. Attempts to attribute all the change in caries prevalence to fluoride divert attention from other investigations. Food additives and gratuitous antibiotic intake are examples of the sort of factors which merit further study. It may be time to acknowledge that fluoride supplementation is only a partial cause of the decline in prevalence and that epidemiological studies aimed at identifying other contributing causes are urgently needed.

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