Fluoride, fluorosis and dental caries

T.W. Cutress
Dental Unit, Medical Research Council, Wellington, New Zealand

Fluoridation of community water supplies as a public health measure has its origin in observations stretching back 100 years(1). Sixty years ago the phenomenon of mottled enamel was linked to a factor in drinking water(2); in 1931 the active factor was identified as the element fluoride(3); a few years later epidemiological studies revealed inverse relationships between fluoride levels in drinking water and the prevalence of dental caries(4). Since the first field trial(5) in 1945 of the effect of artificially increasing the fluoride levels in public water supplies to 1 ppm, numerous studies have been reported world-wide(6). At present 250 million people in 30 countries use artificially fluoridated water with impressive improvements in dental health(7). A 40 to 60% decrease in caries in children is consistently reported. The phenomenon of mottled teeth in communities with 1 ppm water fluoridation is observed at mild levels (in about 20% of children) which appears to be the only disadvantage of regular ingestion of low amounts of fluoride. Lesser concentrations than 1 ppm fluoride in drinking water are correlated with increased prevalence of caries but a relatively small decrease in the prevalence of mild mottled teeth(8).

Fluorine is the most electronegative and reactive of elements and, as one of the more abundant elements, is found in all diets, teeth, and bone. Despite an immense diversity of research the mechanism of action of fluoride in controlling caries remains debatable. The empiricism associated with the use of fluoride is based on a multiplicity of field trials which repeatedly confirm reduction of caries with no detectable adverse systemic effects. This element in diets in trace amounts(9) appears to be the only disadvantage of regular ingestion of low levels of fluoride. Lesser concentrations than 1 ppm fluoride in drinking water are correlated with increased prevalence of caries but a relatively small decrease in the prevalence of mild mottled teeth(8).

The variables of biological response, dosage, socioeconomic and behavioural factors, together with the less than precise methodologies inherent in epidemiological studies have produced vast volumes of data. The overwhelming conclusions of basic, applied and public health research are that the benefit of fluoride on dental caries far outweighs the problems of induced mild fluorosis. Despite continuous and intense research into the toxicological, pharmacological and chemical properties of fluoride over many years, mild fluorosis appears to be the only biologically identified deleterious effect of fluoride, at moderate daily intakes. Claims to the contrary have not been substantiated on independent enquiry(9-11). Nevertheless the possibility of unforeseen adverse effects cannot be (and are not being) ignored.

Toothpaste, salt, rinses, tablets etc. as carriers for fluoride also reduce caries(6). However voluntary, as opposed to involuntary, procedures introduce a whole range of new variables and barriers related to socioeconomic, behavioural and cost which decrease the individual and community benefit.

With a reduction in caries prevalence now apparent and further developments expected in alternative methods of caries control, water fluoridation will no doubt decrease as a priority for disease control. In New Zealand current programmes of monitoring child and adult oral health provide both a realistic and scientific basis on which to make these judgements(12-14).

Three major objections to “mass medication” raised at the time of the original community fluoridation study (1946) still persist. (1) It is ineffective in reducing caries; (2) it is basically harmful to human health; (3) it is against the principle of freedom of choice. However the evidence of the past 40 years has provided no support for the former two objections; the latter is an intangible and outside the scientific scope of the meeting.

Claims(15) of a high prevalence of disfiguring dental fluorosis in Auckland were not substantiated in a recent survey(16) of developmental defects of enamel in children. Although 33% of 1758 9-year-old children living in fluoridated and non-fluoridated areas of the city were observed to have some type of defect of their incisor teeth, the only significant difference between the areas was in the prevalence of diffuse type opacities. (Diffuse opacities - mottled enamel - can be fluoride induced but are not unique to this etiological factor.) Eight percent of children in non-fluoridated areas showed evidence of diffuse opacities, the prevalence was 10% higher in fluoridated areas. This higher prevalence of mottled enamel in optimal fluoridated (1 ppm) areas has been reported in many previous surveys in several countries. In fact this level of mild fluorosis was the major determinant in the recommendations that optimal fluoride levels in drinking water should be set at 1 ppm in temperate climates. Six of the 1758 children were found to have an unsatisfactory appearance due to mottled enamel of the diffuse-patchy type; these children lived in a fluoridated region. A further 13 children had disfiguring developmental defects of enamel which did not conform with fluorosis.

Debate on the effect of fluoride on the appearance of the teeth must include reference to the effects of dental decay on tooth appearance. Before the introduction of fluoride as a protective agent against tooth decay more than 90% of the population could expect to have suffered the disease, the effects showing in terms of unsightly cavities, obvious fillings, missing teeth and even dentures before the age of 20 years. The majority of the adult population currently over the age of 30 years show the disfiguring effects of endemic dental disease, almost 20% being edentulous by the age of 45 years (in 1950 the edentulous rate was 50% by age 50 years.)

While other means of control of tooth decay are being used, for the present, control of dental caries appears to be linked to fluoride availability in trace amounts.

Recent reviews by WHO-FDI(7) acknowledge the potential of fluoridated salt and dentifrices as public health measures for reducing the prevalence of caries. Nevertheless such organisations endorse their policies that, where possible, water fluoridation should be introduced as the principle means of primary prevention of caries upheld by the Department of Health and the Dental Association in New Zealand.

REFERENCES

Selenium supplementation alternatives: their effects on animal production, glutathione peroxidase and selenium levels in ewes and lambs

A.K. Metherell, J.L. Owens, C.G. Mackintosh and K. Turner*
Invermay Agricultural Research Centre and
*Invermay Animal Health Laboratory
Private Bag, Mosgiel, New Zealand

INTRODUCTION
Selenium supplementation of ewes and lambs is an important management practice, necessary to achieve satisfactory production, in many areas of New Zealand (Andrews et al. 1968). Until recently direct administration of soluble selenium compounds either orally or by injection was the only method of selenium supplementation allowed under the Animal Remedies Act 1967. In 1982 regulations permitting selenium topdressing were gazetted (New Zealand Gazette 1982.) Recommendations for selenium topdressing have been based on complete coverage of the grazed area on a farm to give protection to all stock for 12 months, although partial application has been suggested (Watkinson 1983).

On properties under extensive grazing and/or low stocking rates complete coverage is an expensive method of supplementation. Alternatives are:
1. The topdressing of limited areas of pasture for grazing at strategic times.
2. The use of slow release selenium formulations such as intra-ruminal bullets or injectable barium selenate paste (Cawley & McPhee 1984).

This experiment compares oral dosing with sodium selenate, the strategic grazing of selenium topdressed pasture in autumn and the injection of barium selenate. Effects on ewe fertility, the growth and survival of lambs, levels of glutathione peroxidase (a selenium-containing enzyme) and selenium in blood were measured.

EXPERIMENTAL
Five groups of about 40 Merino ewes (4 to 5 years of age) and their lambs grazed extremely selenium deficient pastures (3 - 17 µg Se/kg DM) on irrigated flats on a Mackenzie high country yellow-brown earth at Tara Hills High Country Research Station, Omarama. Treatments first applied in April 1983 were:
1. Oral
Oral dosing of ewes with 5 mg Se as sodium selenate, 3 weeks before joining with rams and 4 weeks pre-lambing; and of lambs with 2 mg Se at tailing (4 weeks of age) and weaning (10-12 weeks of age).
2. Barium selenate
Subcutaneous injection of ewes in the neck with 50 mg Se as barium selenate paste (Deposel® ; Robert Young & Co (N.Z.) Ltd, Petone), 3 weeks before joining.
3. Topdressing 4
Grazing of recently selenium topdressed pasture (650 µg Se/kg DM), during the flushing and mating period, beginning 3 weeks before joining and continuing for 4 weeks.
4. Topdressing 8
Grazing of recently selenium topdressed pasture (650–550 µg Se/kg DM), during the flushing and mating period, beginning 3 weeks before joining and continuing for 6 weeks.
5. Control
No selenium supplementation.

RESULTS
Animal performance
There were more barren ewes in the control group than in the selenium supplemented groups (Table 1). Mating records showed that almost all barren ewes in the control group had been mated in the first cycle but did not return to service in the second cycle. This is consistent with embryonic loss as the cause of barrenness. There were no differences between treatments in the number of lambs born per ewe lambing.

The very low number of lambs tailed per ewe mated in the control group (Table 1) reflects both barrenness and perinatal lamb mortality (Table 2). Post-mortem examination of lambs from the control group in every case showed lesions typical of white muscle disease, a myopathy of cardiac or skeletal muscle. A lower lamb tailing percentage in the oral treatment group than in the barium selenate and topdressing treatments (Table 1) was due to higher lamb mortality in the first 28 days (Table 2). Cardiac lesions were observed in some lambs dying in the oral treatment group, but there was no indication of white muscle disease in lambs from the topdressing or barium selenate groups which died before 28 days. However, there were lesions of white muscle disease in lambs from the topdressing-4 treatment which died at 10 weeks or older.

Lamb birth weight did not differ between treatments, but the surviving lambs in the control treatment had much lower weight gains than lambs from the other treatments. There were small differences in liveweight between lambs in treatments 1, 2, 3 and 4. At 4 and 8 weeks of age lambs in the oral treatment were lighter, but subsequently liveweight gains were greater in the oral treatment. Post-weaning gains in the topdressing treatments were very low (Table 3).

Blood selenium and glutathione peroxidase status
At the start of the experiment the mean whole blood selenium and glutathione peroxidase (GSHPx) levels in the ewes were 17 µg/L Se and 2.8 kU/L GSHPx respectively. The GSHPx status of control ewes fell to extremely low levels, less than 1 kU/L GSHPx, through the course of the experiment. The oral