uptake of magnesium-28 is not a generalized reaction in tissue damage.

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[1] Brandt, J. L., Glaser, W., and Jones, A., *Metabolism*, **7**, 355 (1958).
[2] Rogers, T. A., and Mahan, P. E., *Proc. Soc. Exp. Biol. Med.*, **100**, 235 (1959).
[3] Carmichael, E. B., *J. Pharm. Exp. Therap.*, **35**, 193 (1929).

PATHOLOGY

Urinary Selenium and Dental Caries

Cadell and Cousins reported recently on the concentration of urinary selenium and prevalence of dental caries among two groups of children 5–14 years of age in New Zealand[1]. From Table 1 of their article, all but two of the children in both groups had urinary selenium-levels in the range of 0·10–0·49 p.p.m. However, the mean values of urinary selenium given for the first and second group were 0·021 p.p.m. and 0·030 p.p.m. respectively. Obviously there is an error in the presentation of the data.

It is stated by Cadell and Cousins that: "The suggested direct relationship between urinary selenium concentrations and the prevalence of dental caries in American children is not substantiated by an investigation in New Zealand". In view of the data presented by them, this statement seems unwarranted and could lead to erroneous conclusions.

None of their subjects in both groups, having the same magnitude of dental caries, had values of urinary selenium in excess of 0·050 p.p.m. In our work, cited by Cadell and Cousins[2,3], more than half the children in the areas of high prevalence of caries had values of urinary selenium greater than 0·050 p.p.m. On the other hand, in the area with low prevalence of caries, the great majority of the children had values of less than 0·050 p.p.m. Admittedly, the possible dangers to health from the cumulative effects of daily ingestion of small amounts of selenium have been largely unexplored. In one case, however, the presence in the urine of a patient of selenium at a level of about 0·040 p.p.m. was considered to be suggestive of toxic amounts of ingestion[4].

Apparently, Cadell and Cousins are under the impression that a direct relationship could exist between prevalence of caries and all levels of urinary selenium concentration. Such an assumption does not seem correct. It is like claiming that an inverse relationship exists between prevalence of caries and any amount of fluorides ingested, which, of course, is not the case. It is well known that small amounts of selenium are protective against dietary necrotic liver degeneration in rats and also prevent exudative diathesis in chicks[5,6]. On the other hand, the ingestion of excessive amounts of selenium is harmful to animals[7]. If by further work it is shown that selenium is connected with caries, there certainly must be a threshold value of selenium intake, below which there is no influence on susceptibility to caries. Thus it may well be that the ingestion of minute amounts of selenium, such as indicated by the mean urinayr values shown by Cadell and Cousins, does not exert any influence, inhibitory or conducive, on the prevalence of caries.

The description presented by Cadell and Cousins regarding the methods used for assessing caries prevalence in their subjects is quite inadequate. Why did they combine prevalence of caries in primary and in permanent teeth? We have shown that the selenium content of primary teeth appears to be higher than that of the permanent teeth[8]. This becomes an important point in assessing prevalence of caries if the amount of selenium incorporated into the teeth during the period of formation of the crowns influences their future susceptibility to caries.

There is no doubt that further work is necessary for a better understanding of the possible relationship between selenium and caries. However, New Zealand does not appear to be suitable for such investigations since, according to Cadell and Cousins, that country has widespread areas deficient in selenium and also no known seleniferous areas exist there.

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[1] Cadell, P. B., and Cousins, F. B., *Nature*, **185**, 863 (1960).
[2] Hadjimarkos, D. M., *et al.*, *J. Pediatrics*, **40**, 451 (1952).
[3] Hadjimarkos, D. M., and Bonhorst, C. W., *J. Pediatrics*, **52**, 274 (1958).
[4] Lemley, R. E., and Merryman, M. P., *J. Lancet*, **61**, 435 (1941).
[5] Schwarz, K., and Foltz, C. M., *J. Amer. Chem. Soc.*, **79**, 3292 (1957).
[6] Patterson, E. L., *et al.*, *Proc. Soc. Exp. Biol. and Med.*, **95**, 617 (1957).
[7] Trelease, S., and Beath, O. A., "Selenium", 165 (New York, 1949, published by authors).
[8] Hadjimarkos, D. M., and Bonhorst, C. W., *Oral Surg. Oral Med. and Oral Path.*, **12**, 113 (1959).

Histochemical Demonstration of Some Hydrolytic Enzymes in Atheromatous Aortas

In connexion with other investigations made in this Department on the pathogenesis of atherosclerosis, our attention was directed to the histochemical changes in early atheromatous plaques. Barrows and Chow[1] suggested that a disturbance in the relative concentrations of enzymes within arterial tissue may result from arteriosclerosis. Relatively little is known, however, about the localization of enzymes in atheromatous aortas. Paterson *et al.*[2] demonstrated alkaline phosphatase in early atheromatous plaques, using Gomori's calcium–cobalt technique. They assumed that the activity was due to vascularization of the intima in early plaques.

We have made a histochemical study of the phosphatases[3], esterase (with the naphthol *AS* method[4]), and leucyl aminopeptidase[5], in atheromatous aortas. The samples were obtained fresh at autopsy and fixed overnight with cold, neutral 10 per cent formalin: sections were cut in a cryostat at 10μ. The test for phosphatase was made at different *p*H's, and the highest activity was obtained at *p*H 5·8–6·2. This is in good accord with the results of Kirk and Praetorius[6]. This phosphatase was found by histochemical methods in atheromatous plaques only (Fig. 1) and could not be demonstrated in perfectly normal aortas. The activity was most intense at the margins of the plaques.