It is concluded that chloride ions are actively transported across the frog corneas and this ionic transport represents the only source of current across this membrane, and probably it is directly related to corneal hydration and transparency. The ATPase found in the frog corneas would seem to be different from that found in sodium transporting systems[6] and might not be related to the chloride transport found. Though this does not mean that chloride transport is not ATP dependent, it is possible that another mechanism might be involved in the transport of chloride across biological membranes.

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[1] Davson, H., *Brit. J. Ophth.*, **33**, 175 (1949).
[2] Maurice, D. M., *J. Physiol.*, **112**, 367 (1951).
[3] Donn, A., Maurice, D. M., and Mills, N. L., *Amer. Med. Assoc. Arch. Ophth.*, **62**, 748 (1959).
[4] Ussing, H. H., and Zerahn, K., *Acta Physiol. Scand.*, **23**, 110 (1951).
[5] Boyle, P. J., and Conway, E. J., *J. Physiol.*, **100**, 1 (1941).
[6] Skou, J. C., *Prog. in Biophys.*, **14**, 131 (1964).

Vanadium and Dental Caries

THE effects of trace elements other than fluoride on dental caries have recently received increased attention. The results of numerous investigations, reviewed elsewhere[1], have indicated that selenium is capable of increasing the susceptibility to caries, particularly when it is consumed during the formation of the teeth and incorporated into their structure. On the other hand, molybdenum appears to have a beneficial effect on caries[2].

In recent years, vanadium has been investigated as a caries-inhibiting trace element. Vanadium was found to decrease caries when given to hamsters and rats either during the time of tooth formation or post-developmentally[3,4]. However, further animal experiments failed to substantiate these findings[5,6].

The only investigation of the effect of vanadium on caries in man was conducted in Wyoming among children[7]. This State was selected because it contains widespread deposits of vanadium. For ascertaining the dietary intake of vanadium by the children, its concentration in water samples was determined. This was based on the premise that water is the main vehicle for vanadium ingestion in man, as is the case with fluoride. The majority of the water samples contained minute amounts of vanadium ranging from 0·03 to 0·09 p.p.m. with a few above the latter level. It was concluded that the consumption of water containing vanadium as low as 0·07–0·09 p.p.m. was responsible for decreasing dental caries in children as compared with those drinking water with vanadium at levels of 0·03–0·06 p.p.m.

In the light of recent findings, it appears that, in general, water does not constitute an important source of vanadium and that its concentration cannot serve as a reliable criterion of the total amount of this element ingested daily. An investigation of 194 public water supplies in cities located in forty-nine States was conducted by the U.S. Public Health Service. It revealed the presence of minute amounts of vanadium in the samples ranging from 0·0004 to 0·07 p.p.m. with a mean content of 0·006 p.p.m.[8]. In a second investigation by the U.S. Public Health Service, it was shown that from a total of 247 water samples taken from five major rivers (Ohio, Mississippi, Colorado, Missouri, Columbia) and the Great Lakes, vanadium was detected in only five samples[9]. No vanadium was found in the samples from the Ohio and Mississippi Rivers and the Great Lakes.

It is concluded that in epidemiological studies on the association between vanadium intake and caries, the concentration of this element in water alone cannot be relied on as an index reflecting the total amount ingested daily. It is known that the same is true with regard to selenium, even in seleniferous regions[10]. Other methods should be used for assessing the dietary vanadium intake in population groups.

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[1] Hadjimarkos, D. M., *Amer. Med. Assoc. Arch. Environ. Health*, **10**, 893 (1965).
[2] Ludwig, T. G., *et al.*, *Nature*, **186**, 695 (1960).
[3] Geyer, C. F., *J. Dent. Res.*, **32**, 590 (1953).
[4] Kruger, B. J., *J. Austral. Dent. Assoc.*, **3**, 298 (1958).
[5] Muhler, J. C., *J. Dent. Res.*, **36**, 787 (1957).
[6] Buttner, W., *J. Dent. Res.*, **42**, 453 (1963).
[7] Tank, G., and Storvick, C. A., *J. Dent. Res.*, **39**, 473 (1960).
[8] Taylor, F. B., *J. Amer. Water Works Assoc.*, **55**, 619 (1963).
[9] Kroner, R. C., and Kopp, J. F., *J. Amer. Water Works Assoc.*, **57**, 150 (1965).
[10] Hadjimarkos, D. M., and Bonhort, C. W., *J. Pediat.*, **59**, 256 (1961).

Antagonism by Prostaglandins of the Responses of Various Smooth Muscle Preparations to Sympathomimetics

PROSTAGLANDINS are known to produce two types of effects on smooth muscle. First, they may produce direct and relatively short-lived effects, such as stimulation of the isolated uterus or relaxation of the isolated tracheal chain preparation. Secondly, in doses too low to produce a direct effect, they may produce a long-term potentiation of the effects of other stimulants. These two effects have been investigated in the isolated guinea-pig uterus[1]. The long-term potentiation of the effects of other stimulants which is induced by brief exposure of the guinea-pig uterus to low concentrations of PGE_1 or PGE_2 appears to be non-specific with respect to the potentiated stimulant, and can be demonstrated with a variety of stimulants including vasopressin, oxytocin and histamine, and also with electrical field stimulation[2]. The potentiating effect, however, is fairly specific to prostaglandins of the PGE series. This type of effect is not restricted to the guinea-pig uterus and can be demonstrated on the isolated rat colon or vas deferens (Fig. 1).

Another indirect long-term effect of prostaglandins of both the PGF and the PGE series is described here, and consists of a depression of the responses of various isolated smooth muscle preparations to sympathomimetic substances—to adrenaline, noradrenaline, phenylephrine, and isopropyl noradrenaline.

Some tissues in which this effect has been examined are shown in Table 1. In some of these, prostaglandins have

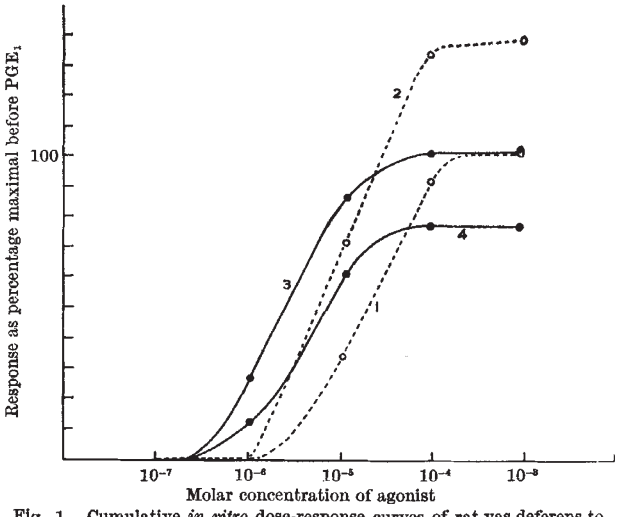

Fig. 1. Cumulative *in vitro* dose-response curves of rat vas deferens to carbamyl-choline (interrupted line) and adrenaline (solid line). Curves 1 and 3 were obtained before exposure to prostaglandin. Curves 2 and 4 were obtained 90 min after exposure for 5 min to PGE_1, at 4×10^{-6} g/ml.