EDITORIAL

GOITRE AND FLUOROSIS IN KENYA

The thyroid gland synthesizes and secretes thyroxine and tri-iodothyronine both of which are hormones essential for normal metabolism in many animal species including man. The principal extrinsic ingredient of these hormones, iodine, is avidly taken up by the thyroid gland by a process which results in the thyroid maintaining concentration gradients for iodine against plasma of levels reaching 1000-fold. This, however, assumes the presence of iodide in plasma chiefly from dietary sources. A daily dietary intake of 100-150 mcg of iodide (1) is required to maintain a physiological plasma iodide concentration of approximately 5 mcg per litre. When dietary iodide levels are inadequate to maintain this blood level because of either absolute or relative deficiency physiological feedback mechanisms result in enlargement of the thyroid gland (goitre) in an attempt to enhance its iodide-trapping activity. Goitre is a common finding in many parts of the world including the central highland regions of Kenya. When the prevalence of goitre exceeds 10 per cent in a given population, the people in that region are, by definition, said to suffer from endemic goitre (2).

The main goitrogenic factor worldwide is dietary iodide deficiency. In most cases food is the major source of dietary iodide, providing about 90% of the total intake, the rest coming from water (1). Enzyme-dependent hereditary biosynthetic defects are a rare cause of goitre. Other goitrogens such as inorganic anions, notably perchlorate and thiocyanate, which inhibit iodide transport mechanisms within the thyroid gland, are rarer causes of goitre. These latter two groups of goitrogenic factors do not contribute significantly to incidence of endemic goitre in large communities.

Areas which have endemic goitre in Kenya are highlands in the central parts of the country where there are no lakes from which iodide-rich foodstuffs, such as fish, could be found. Iodized salt has been mandatorily available in Kenya for many years. In deed, most of the cases of goitre from these areas do not show iodide deficiency on biochemical evaluation. Many of these patients manifest clinical and laboratory features of simple goitre (normal plasma levels of thyroxine, triiodothyronin, thyroid stimulating hormone, and normal thyroid iodideuptake values). It therefore would appear unlikely that absolute iodide deficiency per se would account for endemic goitre in Kenya. Furthermore, a study of iodide levels in salt conclusively showed that salt is a significant source of iodide throughout the Republic of Kenya (3). Indeed the goitrous areas appeared to have higher levels of iodide in salt than the non-goitrous areas. Thus, Meru, Muranga and Nyeri which have a high prevalence of goitre had 35.82, 33.79 and 33.08 of lodide as mg potassium iodate (KIO₃) per kg salt, respectively whereas non-goitrous areas such as Busia and Bungoma had 22.07 and 10.42 mg KIO3 per kg sall, respectively (3). It would therefore appear that iodization of salt alone may not be sufficient to reduce the prevalence of goitre in an endemic region.

It is interesting that the same areas which suffer from endemic goitre in Kenya also have the highest prevalence of fluorosis in the country. Indeed, many cases

of fluorosis in Kenya have concurrent goitre.

The following are representative figures of fluoride concentrations in water in parts per million in various parts of central Kenya (3): Nairobi, 19; Thika, 20; Kiambu, 16; Athi River, 15; Embu, 8.4; Nakuru, 3.5 and Naiyasha, 40.

Fluorine, chlorine, iodine, bromine are all halogens. Fluorine is the most reactive and iodine the least reactive of the halides given. Concurrent occurrence of fluorosis and endemic goitre may have a causal relationship, in that fluorides could competitively be taken up by the thyroid gland and thereby inhibit the iodide transport mechanism and reduce available substrate for thyroid hormone formation. Theoretically, the goitre that follows could be prevented by plentiful intake of dietary iodide which would be sufficiently large to enable adequate quantities of iodide to enter the thyroid gland by simple diffusion. This could explain the absence of goitre in areas which suffer from fluorosis but which, fortunately, have abundant dietary iodide. Thus the endemicity of goitre in Kenya could be a consequence of relative iodide deficiency in the central highland regions where there is a simultaneously relative abundance of fluoride. Excessive fluoridation of toothpastes which are widely available and aggressively advertised throughout Kenya may aggravate the problem of fluorosis and goitre. The significance of such a relationship have far-reaching implications from a public health point of view and merits careful study.

A recent weekly publication questioned the wisdom of fluoridating toothpastes in Kenya in view of the high levels of fluoride in water in Kenya (4). Dr. Firoze Manji (4) of the University of Nairobi is quoted as having reported that it was time both the medical and dental professions seriously thought about fluorosis, an unnecessary disease. He reports that "the geology of Kenya renders it an area endemic in fluorosis. Large quantities of volcanic ash, rich in fluorides are present in the surface soils, especially those of the Rift Valley, the Central and the Eastern provinces. Analysis of Kenyan water shows unacceptably high fluoride content in several areas. Countries such as Kenya, where fluorosis is endemic, do not need fluoridation of their water supplies. Defluoridation should be sought to prevent the long-term consequences of excessive fluoride intake". This statement is fully endorsed and also the need for fluoridating toothpaste in Kenya is questioned.

Although the above observations do not constitute universally accepted facts, they nevertheless form a hypothesis which may warrant serious attention since both endemic goitre and fluorosis are significant medical problems in Kenya.

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CURRENT MEDICAL PROGRESS WEEKLY CLINICAL CONFERENCES

TO BE HELD AT

THE AGA KHAN HOSPITAL, NAIROBI

It has been decided to alter the format of these meetings and amalgamate with the monthly clinical conference. From the end of June there will be a Weekly Clinical Conference held on Thursdays at 12.30 for 12.45 p.m.

Sandwiches and coffee will continue to be served. These meetings will replace the monthly clinical conference previously held on the 1st Tuesday of the month.

DR, A.F. BAGSHAWE

ANNOUNCEMENT

The Sixth Bienniel General Meeting and Conference of the Pan African Association of Neurological Sciences will be held in Tunis, Tunisia from 27 to 30 April 1983. For further information contact:

> Professor Mongi Ben Hamida Centre de Neurologie La Rabta Tunis Tunisia