Iodine, milk, and the elimination of endemic goitre in Britain: the story of an accidental public health triumph

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Abstract
Objective—To determine how iodine deficiency and endemic goitre disappeared in Britain.
Design—Review of surveys of endemic goitre and iodine nutrition.
Main results—Endemic goitre was widespread in Britain but has declined, most notably since the 1960s. Its disappearance was probably due to changes in farming practice, especially iodine supplementation in dairy herds which has resulted in iodine contamination of milk and dairy produce.
Conclusions—Although iodization of dairy herds offers an indirect method of counteracting iodine deficiency, it is haphazard and there should be careful and continuous monitoring of iodine intakes in the population.

Although endemic goitre and other diseases caused by iodine deficiency remain a significant global health problem, few doctors practising in the 1990s will recall that endemic goitre was at one time widespread in Britain. Its elimination is the story of an unplanned and accidental public health triumph. While many other countries introduced national iodization programmes to eliminate iodine deficiency by adding iodine to salt or bread, this was never carried out in Britain. Yet endemic goitre has disappeared and iodine intakes in the UK have risen progressively during the last half century. The most reasonable explanation seems to be that changes in farming practice which led to iodine contamination of milk and dairy produce together with the policy of successive governments in encouraging milk consumption led to the eradication of this age old disease.

Endemic goitre in Britain
In his 1836, Treatise on English Bronchocele, Inglis recorded that endemic goitre was as common in the Yorkshire dales as in Geneva or any of the Alpine valleys.1 Children could be seen at play with pieces of black velvet tied around their necks to charm away the goitre evil. Other early records reported that goitre and even cretinism, usually associated with severe iodine deficiency, could be found in several English Counties including Norfolk, Monmouthshire, and Cornwall.2 Cretinism was said to be notorious in Chiselborough, Somerset.3 By the turn of the century an English goitre belt was recognised which extended from the West Country through Somerset into the Cotswold and Chiltern Hills and northwards into Derbyshire and the Peak District. There were well defined offshoots affecting both north and south Wales (fig 1). In 1924, a survey of 12 year old schoolchildren in England and Wales was carried out by school medical officers at the request of the Board of Education.4 This study is remarkable, not least for its sheer size comprising observations on 375 000 children. It confirmed the geographical pattern of the disease shown by previous studies and revealed a high prevalence of visible goitre (up to 30% in some communities) which led to calls for a national iodization programme. The war of 1939–45 brought the subject into new prominence because goitre appeared to be unusually common in young women drafted into factories for war work. The Medical Research Council (MRC) carried out several special surveys which showed visible goitre to be present in 50% of the adult women in Hook Norton, Oxfordshire, 43% of girls in Sherborne, Dorset; and in 26% of boys and girls in St Albans, Hertfordshire.5 In contrast, only 2% of children showed thyroid enlargement in Maldon, Essex where the drinking water is rich in iodine. In 1944 and again in 1948 the MRC urged the general adoption of iodized salt throughout the UK as a means of preventing goitre but no action followed.6

Figure 1 Areas of England and Wales where endemic goitre has been prevalent in the past.4

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A number of ad hoc surveys carried out in subsequent years showed that thyroid enlargement continued in many areas well into the 1960s. Schools in north Oxfordshire, which had been included in the 1948 MRC survey, were resurveyed in 1958 using the same methods and classification. Goitre rates were similar in boys but had actually increased in girls from 27% in 1948 to 40% in 1958. Later surveys showed goitre to be still present during the early 1960s in Sheffield and South Wales and detailed metabolic studies of patients presenting with non-toxic thyroid enlargement in Glasgow showed that they had low dietary iodine intake and low plasma inorganic iodine concentrations. Subsequently, the disease appears to have declined and a 1990 survey showed that thyroid enlargement was no longer detectable in schoolchildren in a traditionally iodine deficient area of South Wales.

The iodization and marketing of milk in Britain

Research during the 1920s showed that iodine supplementation of livestock could improve their reproductive performance. This provided an economic incentive for the iodine supplementation of dairy herds, which was achieved by providing iodized salt licks for cattle (introduced by Boots in 1928) and subsequently the use of iodine enriched cattle feed (marketed by Spillers in 1937). The result was a spectacular rise in the iodine content of milk, particularly during the winter months when pasture is in short supply and cattle are dependent on iodine rich artificial feed. The use of iodinated casein, given to cows as a lactation promoter, and iodophor disinfectants, used for cleaning teats and bulk milk tankers, also contributed to the iodine contamination of milk.

In parallel with this increase in iodine content, successive governments put into effect policies to increase the consumption of milk and dairy produce. Before the 1930s milk had been a luxury only affordable by wealthier families. A survey in 1932 found a mean intake of only 3.1 pints per week. The subsequent establishment of the Milk Marketing Board and the Milk Act (1934) and The National Milk Scheme (1940), which provided free milk or at a subsidised price for all expectant mothers and young children, greatly boosted consumption.

By the early 1950s average liquid milk consumption had nearly doubled. In figure 2 data on milk consumption have been combined with published analyses of its iodine content to show how the amount of iodine provided by average milk consumption in the UK has increased since 1931. By the 1980s the iodine content of milk during the winter was such that milk alone could almost satisfy the recommended daily requirement of 150 μg/day. Indeed, the iodine content in some milk samples was so high that this led to concerns about possible toxic effects. The increase in the iodine content of milk and related dairy products is likely to have been the major explanation of the threefold rise in dietary iodine intakes, from 80 μg to 255 μg/day, which occurred between 1952 and 1982. Iodine intakes were also boosted by the iodine enrichment of meat and eggs, probably due to the same processes of spillover from the use of iodine in animal feeds. It is noteworthy, however, that over the same period the consumption of traditional iodine rich foods such as fish and sea food declined. Other northern European countries, particularly Scandinavia, have also benefited from iodine in milk and dairy produce because of the use of iodine rich foods throughout the long winters. This may explain why iodine deficiency has declined in northern Europe but not in southern Europe where pasture is available for a greater part of the year and artificial feedstuffs are less often used.

Iodine induced thyrotoxicosis

Iodization is not without its side effects and it has been known for many years that an increase in the incidence of hyperthyroidism often occurred after iodization. It usually affected older subjects who had a longstanding goitre and is due to increased sensitivity of the thyroid gland to iodine in amounts which would be well tolerated by normal subjects. There is evidence that this has occurred in Britain. Mortality from thyrotoxicosis peaked in the decade 1931–40,
and both the mortality and incidence of thyrotoxicosis due to toxic multinodular goitre have remained highest in those areas of Britain shown in figure 1 which once had endemic goitre. While the increase in dietary iodine in these iodine deficient areas of Britain has benefited the young by eradicating goitre, the price may have been of precipitating thyrotoxicosis in older men and women in the same areas.

Conclusion
Countries with endemic iodine deficiency have tended to solve the problem by the compulsory iodization of staple foodstuffs, for example bread or salt. The British experience, however, suggests that the same result can be achieved by ensuring that dairy herds are iodine replete and allowing the resulting iodine contamination of milk and dairy produce to increase human iodine intakes. While compulsory iodization of salt or bread has the advantage that it can be precisely controlled, consumer opposition allied with stringent regulations to control the use of food additives has tended to prevent iodization in recent years, most notably in Germany. Allowing the spillover of iodine from dairy herds to effect iodization offers an alternative but has the disadvantage of being considerably more haphazard as the amounts of iodine entering the human food chain will depend on both farming practice and the levels of consumption of key foodstuffs, in particular liquid milk. As this solution has been adopted by default in Britain, it is nevertheless essential that iodine intakes are carefully monitored to ensure that they remain adequate to prevent the re-emergence of iodine deficiency and its adverse health consequences.

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1 Inglis J. Treatise on English bronchocoele, with a few remarks on the use of iodine and its compounds. London, 1836.
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