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COMMENTARY

Iodine deficiency in industrialized countries*

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The recent report of poor iodine status in the UK[1] is a wake-up call to health authorities in industrialized countries who have been slow to grasp that iodine deficiency (ID) is not confined to developing countries. Australia, New Zealand, Belgium, Norway and now the UK are all mildly iodine deficient. In the US, iodine intakes have fallen, and it is uncertain whether status is adequate in pregnancy, leading to calls for iodine supplementation.[2] Among the WHO regions, Europe has the highest prevalence of ID (>40% of children have low iodine intakes) and the lowest household coverage with iodized salt.[3] Iodine-deficient soils are common in the industrialized countries; historic 'goitre belts' include the Alps and Apennines in Europe, Midwestern North America, southern Australia and inland areas of England and Wales. ID in these areas will occur unless iodine enters the food chain through addition of iodine to foods (e.g. iodization of salt) or dietary diversification introduces foods produced outside the iodine-deficient area.

Iodine deficiency has multiple adverse effects on growth and development, collectively termed the ID disorders.[4] The most important are mental and motor deficits, because adequate thyroid hormone (of which iodine is an essential component) is critical for normal brain development and function. Moderate to severe ID in populations causes sporadic cretinism and reduces mean IQ by 12–13.5 points.[4] The effect of milder ID found in most industrialized countries is less certain, but recent controlled trials in school children in Europe[5] and New Zealand[6] have shown clear benefits of iodine repletion on cognitive function and fine motor skills. In adults in industrialized countries affected by milder ID, sustained iodine prophylaxis reduces rates of nodular goitre and hyperthyroidism, and their associated health costs.[7]

Why have iodine intakes in industrialized countries like the US, the UK and Australia fallen in recent years? The major dietary sources of iodine in typical 'Western' diets are bread (containing iodized salt) and dairy products.[4] But some of the iodine in bread (from iodized dough conditioners) and nearly all of the iodine in dairy products is adventitious: dietary iodine supplements given to cows and residues of disinfectant iodophors used for cleaning teats and milk storage containers end up in milk and can sharply increase its low native iodine content. Over the past few decades, remarkably, adequate dietary intakes in many industrialized countries have depended on these 'accidental' and unregulated iodine sources! But intake from these sources may be falling, for commercial and legislative reasons. Take Australia as an example. In Australia in 1975, the mean iodine concentration in cows' milk was nearly 600 μg/l. Concerned about potential toxicity, the government specified a milk iodine limit of 500 μg/l in 1982. This led to the replacement of many iodophors by chlorine-containing sanitizers. By 2001, median iodine content of milk samples in Sydney had fallen to 140 μg/l. In 2004, a national survey[8] found that children in south-eastern Australia were iodine deficient and 9% of girls had goitre by thyroid ultrasound, leading to the introduction of a national programme in 2009 to iodize all salt used in bread making.

Could a similar fall in adventitious iodine intake explain the new UK findings? In the nineteenth and twentieth centuries, the UK goitre belt extended from the West Country through Somerset into Derbyshire and the Peak District and also included north and south Wales; occasional cases of cretinism were reported. In these areas in the 1940s, visible goitre was present in half of adult women and the Medical Research Council recommended an iodized salt programme, but no action was taken. ID in the UK remained a health problem into the late 1950s and early 1960s; in 1958, 40% of girls in Oxfordshire had goitre. Goitre then disappeared. Phillips suggested that this 'accidental public health triumph' resulted from increasing use of iodophors in dairying and higher milk intake;[9] iodine-containing bread conditioners may also have contributed. Mainly due to these adventitious sources, dietary iodine intakes in the UK increased between 1952 and 1982 from 80 μg to 255 μg/day. However, since then, milk intakes in the UK have fallen and, although data are scarce, iodine compounds used in the dairy and baking industries have been at least partially replaced by noniodine alternatives. As dietary iodine from these adventitious sources falls, the UK is vulnerable to ID because <5% of household salt is iodized.[10]

Although recent small regional iodine surveys in the UK suggested that ID had re-emerged,[10] the study by Vanderpump *et al.*[1] on behalf of the British Thyroid Association is the first national survey of iodine nutrition in the UK in over 60 years. It reports a median urinary iodine concentration (UIC) in 14- to 15-year-old schoolgirls (n = 737) of 80 μg/l (IQR 57-109), with 16% of girls having a

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UIC < 50 μg/l but only 1% with a UIC < 20 μg/l. Although the sampling frame was not proportionate to size (it surveyed urban centres) and the overall median UIC was not population weighted, according to WHO criteria for classifying iodine nutrition in populations based on the median UIC,[3] the data suggest the UK is mildly iodine deficient. In a multivariate regression, sampling in summer (when milk iodine concentrations are at their nadir) and low intake of milk were predictors of low UIC.

What can be done to ensure adequate iodine intakes in industrialized countries? In nearly all countries, the best strategy to control ID is the addition of iodine to salt; it is simple, effective, safe and inexpensive.[3] Worldwide, nearly 70% of households in low-income countries have access to iodized salt, and the annual costs of salt iodization are estimated at only US$ 0.02–0.05 per child covered.[4] However, because ca. 90% of salt consumption in industrialized countries is from purchased processed foods, if only household salt is iodized, it will not supply adequate iodine. Thus, to successfully control ID in industrialized countries, it is critical to convince the food industry to use iodized salt in their products. Because iodine at ppm levels in foods does not cause any sensory changes, and, in most countries, the price difference between iodized and noniodized salt is negligible, there are no major barriers to its use in foods. In Denmark and the Netherlands, nearly all salt used by the baking industry is iodized, and this controls ID. Switzerland's long-running iodized salt programme has been successful because ca. 60% of salt used by the food industry is iodized on a voluntary basis. The current global push to reduce salt consumption to prevent chronic diseases and the policy of salt iodization to control ID do not conflict: iodization methods can fortify salt to provide adequate iodine even if per capita salt intakes are reduced to <5 g/day, as long as all salt consumed is iodized.

Since 1990, control of ID using iodized salt has been an integral part of national nutrition programmes in many developing countries in Africa, Latin America and Asia. It is remarkable that high-income countries in Europe and North America have donated substantial funds and expertise to these programmes, but have not yet corrected ID in their own populations.

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