

Iodine intake and prevention of thyroid disorders: surveillance is needed

There is a need for national monitoring and adjustment of iodine intake as part of a program of prevention of thyroid disorders and their complications

THE WIDESPREAD APPLICATION of public iodine supplementation programs, which cover about 3–4 billion people worldwide,^{1,2} is a response to the paucity of iodine in the natural diet in many regions of the world and the severe public health consequences of iodine deficiency.³

However, in some countries, the tendency to low iodine intake has mostly been corrected by haphazard increases in the iodine content of certain parts of the diet.⁴ In the United Kingdom, dairy products may contain extra iodine as a result of adding iodine to cow feed to increase the animals' reproductive performance or of using iodine-containing cleansing agents in the dairy industry. As reviewed by Phillips,⁵ this unplanned increase in iodine intake has eliminated endemic goitre in Britain during the last 30–40 years. In the United States, London and colleagues encountered cases of unexplained very high iodine intakes (1100–1300 µg per day) in 1964. Subsequently, they discovered that bakers used iodine-containing conditioners in bread and that this caused high levels of iodine intake.⁶ Obviously, such unplanned variation in dietary iodine is a hazardous way of providing a population with an adequate intake of iodine, as mechanisms unrelated to disease prevention can profoundly alter iodine intake. To some extent, Australia may be a country where factors other than disease prevention have modulated the intake of iodine, and iodine intake may now be in an unplanned phase of decrease.⁷

A report by McElduff et al⁸ (page 317) in this issue of the Journal seems to support this proposition. McElduff and colleagues looked at the frequency distribution of whole-blood thyroid-stimulating hormone (TSH) concentrations in newborns in the northern Sydney area. TSH is measured as part of screening for congenital hypothyroidism. In 5%–10% of infants around 72 hours after birth, TSH values were above 5 mIU/L. The World Health Organization (WHO) recommends assessment of TSH concentrations in newborns to detect population iodine deficiency, and specifies that less than 3% of newborns should have a whole-blood TSH concentration over 5 mIU/L. In a subsample of neonates, McElduff et al found that, during pregnancy, their mothers had a median urinary iodine concentration of 109 µg/L, indicating borderline mild iodine deficiency. WHO specifies that the median urinary iodine concentration in adults should be over 100 µg/L, and an extra iodine intake of 50 µg/day in pregnant and lactating women.¹ Thus, the corresponding median urinary iodine concentration of pregnant women would be around 130 µg/L. McElduff et al warn that Sydney may be an area of iodine deficiency, and suggest that iodine intake and risk of disease should be investigated in more detail.

Their concern is well founded. Even if there is no documentation that these borderline iodine values are harmful to a mother and child, the margin of safety is small. Furthermore, in Australia, there is apparently no regular surveillance of population iodine status, or of the variable iodine content of dairy products and other foods. The iodine intake may well be even lower in other sections of the Australian population.

Severe iodine deficiency may cause brain damage and other developmental disorders,³ and goitre and its complications may affect a significant proportion of the population at all levels of iodine deficiency.⁹ Any public healthcare system should evaluate iodine intake and prevent disorders caused by iodine deficiency. Several factors need to be taken into account in such an evaluation and prevention program:

- *The relationship between iodine intake and the risk of thyroid disease is not a simple one.* Even if iodine supplementation may decrease the risk of some thyroid disorders, the risk of other disturbances at a younger age, such as hypothyroidism and Graves' disease, may increase.⁴ Severe iodine deficiency, with a median urinary iodine excretion of less than 25 µg/24 h, is an instance where giving *any* type of iodine supplementation is better than doing nothing. However, at higher levels of intake, careful planning and surveillance are needed.

- *The methods often used for evaluating iodine intake and the risk of disease are not perfect.* Neonatal screening showing more than 3% of TSH values over 5 mIU/L is not, by itself, enough to indicate insufficient maternal iodine intake. Detection of neonatal hypothyroidism requires identification of relatively high TSH levels (20–25 mIU/L), and many TSH assays and screening programs are not designed to identify TSH values around 5 mIU/L with reasonable confidence. Technical aberrations can easily give an increased frequency of elevated blood TSH concentrations. One such aberration occurs with sampling of blood before TSH has fully returned to baseline levels after the early postnatal surge. As discussed by McElduff et al,⁸ early sampling may have contributed to their findings.

- *Iodine deficiency is not the only pathogenetic mechanism leading to an increase in blood TSH levels in neonates.* Iodine has an autoregulatory inhibitory effect on the thyroid gland, with a fall in both thyroid hormone synthesis and secretion. Possibly, this mechanism has been developed to protect against hyperthyroidism induced by a sudden iodine load. In a variety of abnormal states the thyroid gland overreacts, producing hypothyroidism. The thyroid of the fetus and infant is considerably more sensitive to iodine inhibition than the maternal thyroid. Excess iodine intake, rather than iodine deficiency, in mother or infant has been a more common cause of transient neonatal hypothyroidism in

countries with a relatively low iodine intake, such as Germany, Italy and Belgium.⁴ In severe iodine deficiency, iodine supplementation to the mother decreases the abnormally high serum TSH in both the mother and the newborn.¹⁰ On the other hand, in pregnant women with urinary iodine concentrations around 50 µg/L, iodine supplementation decreases TSH levels in mothers, but not in cord blood. TSH levels in the newborn may even be higher after iodine supplementation.¹¹ The finding of McElduff et al of a positive correlation between maternal urinary iodine concentrations during pregnancy and whole-blood TSH levels in neonates needs further elaboration,⁸ but it may be an example of iodine autoregulation of the fetal thyroid.

Should pregnant women living in mild and moderately iodine-deficient areas receive iodine supplements, and does supplementation involve any risk? An increase in iodine intake will improve thyroid function in pregnant women, which is important for early brain development in their infants.¹² There are still things to be learned about the influence of small amounts of iodine on neonatal thyroid function in mild iodine deficiency, and about pituitary/thyroid feedback regulation in the fetus and small infant. A tendency to a slight increase in neonatal TSH level after iodine supplementation may be of little importance, as, in these infants, the serum concentration of T4 (which may be the major thyroid hormone influencing brain development¹²) does not show a concomitant reduction.¹¹ Finally, iodine supplementation imposes no risk of worsening of postpartum thyroid dysfunction in the mother.¹³

In conclusion, pregnant women should not be iodine deficient. To strictly follow WHO guidelines on iodine intake, pregnant women with similar urinary iodine levels to those found by McElduff et al could alter their diet towards more iodine-rich foods, or they could take a small iodine supplement as part of the vitamin and mineral supplements recommended for pregnant women in most countries. However, there is at present no evidence that a supplement will have beneficial effects. Ideally, iodine intake should be evaluated and kept optimal in the entire population, taking into account that unnecessary high iodine intakes may be associated with more hypothyroidism.⁴

The studies by McElduff et al⁸ and other researchers⁷ demonstrate the need for national monitoring and adjust-

ment of iodine intake as part of a program of prevention of thyroid disorders and their complications. Such initiatives normally involve government nutrition or public health agencies in collaboration with experts in thyroid diseases, nutrition and epidemiology and prevention.^{1,2} It would be an added bonus if the program elucidated some of the unresolved issues in the field of population iodine supplementation in developed countries. This would continue the considerable contribution of Australian scientists to the understanding and correction of iodine-deficiency disorders.^{3,14}

Peter Laurberg

Professor and Head
Department of Endocrinology and Medicine

Susanne B Nøhr

Senior Consultant
Department of Gynaecology and Obstetrics
Aalborg Hospital, Aalborg, Denmark
laurberg@aes.nja.dk

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