

Iodine and pregnancy: a call to action



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See [Articles](#) page 331

Iodine, a crucial micronutrient, is essential for thyroid hormone production and for normal in-utero neuro-development. During pregnancy, iodine intake must be increased by 50% because of physiological increases in maternal thyroid hormone production, an increase in maternal renal iodine losses, and fetal iodine needs for thyroid hormone production. Iodine deficiency affects 2 billion people worldwide and is the main cause of preventable mental impairment. Every year, 38 million newborn babies in developing countries are affected by iodine deficiency despite ongoing and concerted efforts by WHO, the UN, and the International Council for the Control of Iodine Deficiency Disorders.¹

Although severe iodine deficiency is not an issue in developed countries, such as the UK, USA, and Australia, moderate deficiency has re-emerged as an important public health concern. This re-emergence is a direct consequence of insufficient cohesive public health policies to eradicate iodine deficiency. Starting in the 1920s in the USA, iodine deficiency was eliminated through voluntary salt iodisation. Later, in the USA, UK, and Australia, adequate iodine intake within populations was serendipitously assured by the use of iodophors in the dairy industry, and of iodate conditioners in bread manufacturing in the USA. Because of decreases in dairy intake in the UK, changes in the use of iodophors in dairy production in the USA and Australia, decreases in the use of iodate dough conditioners by US bread manufacturers, and probable decreases in iodised salt consumption in the USA, moderate iodine deficiency has re-emerged. A 2011 cross-sectional study² of 810 UK schoolgirls aged 14–15 years showed moderate iodine deficiency with a median urinary iodine excretion of 80.1 µg/L (IQR 56.9–109.0).

Although the association between severe iodine deficiency and neurodevelopment is well understood, research is scarce on the effects of more subtle degrees of maternal iodine deficiency on child development. In *The Lancet*, Sarah Bath and colleagues³ document their examination of associations between maternal urinary iodine concentration and child cognitive development (IQ and reading ability at age 8–9 years) in 1040 first-trimester pregnant women in the UK. The pregnant women were mildly-to-moderately iodine deficient, with a median urinary iodine concentration

of 91 µg/L (IQR 53.8–143); lower than the optimum range of 150–249 µg/L recommended by WHO. The urinary iodine concentration in these study participants is similar to that in participants of other, more recent, UK studies. Such deficiency is probably caused by poor availability of iodised salt, few UK recommendations for increased iodine intake in pregnancy, and insufficient use of iodine-containing prenatal supplements.

In adjusted and unadjusted analyses, Bath and colleagues found that the children of women with urinary iodine to creatinine ratios of less than 150 µg/g creatinine had significantly lower scores for verbal IQ (odds ratio 1.58, 95% CI 1.09–2.30), reading accuracy (1.69, 1.15–2.49), and reading comprehension (1.54, 1.06–2.23) than did children of women with ratios of 150 µg/g or more. The investigators noted a possible dose-effect relation, with child neurocognitive scores worsening going from maternal ratios of 150 µg/g or more, to 50–150 µg/g, to less than 50 µg/g.

Strengths of the study include its prospective design, large sample size, the undertaking of several sensitivity analyses, and adjustment for many potential confounders. An important limitation is the use of urinary iodine as a marker for individual iodine status. Because of a large amount of diurnal and day-to-day variation in urinary iodine concentrations, an estimated ten spot-urine samples are needed to achieve 20% precision in estimates of individual iodine intake.⁴ In this context, that the investigators could show a clear relation based on one maternal urinary iodine concentration is surprising, even with adjustments for urine creatinine and dichotomisation by greater and less than 150 µg/g and child cognition. Other study limitations include the potential for residual confounding and for socio-economic bias resulting from the fact that only 56% of the Avon Longitudinal Study of Parents and Children (ALSPAC) cohort had a measure of IQ.

Most,^{5–7} but not all,^{8,9} previous observational studies have shown that mildly low maternal thyroid function is associated with poorer neurocognitive outcomes in children. Women in most of these studies were iodine sufficient. Three uncontrolled, non-randomised trials^{10–12} in regions of mild-to-moderate iodine deficiency have studied the effects of iodine supplementation in pregnancy on child developmental outcomes. Two

reported improved developmental test scores at age 3–18 months in children of women supplemented with 200–300 µg iodine daily, starting early in gestation. However, the third noted decreased psychomotor development index scores in children of mothers supplemented with 150 µg or more daily iodine. Placebo-controlled, randomised clinical trials being done in Thailand and India are designed to establish the effects of iodine supplementation on obstetric outcomes and child development in pregnant women with mild-to-moderate iodine deficiency.¹³

Bath and colleagues' study,³ along with data from Vanderpump and colleagues,² should be regarded as a call to action to public health policy makers in the UK. Absence of a public health policy in the face of clear documentation of moderate iodine deficiency and strong evidence of its deleterious effect on the neurodevelopment of children is ill advised. Nor should unmonitored and adventitious dietary iodine sources continue to be relied on. Until measures are taken to ensure that iodine needs can be met by usual dietary sources, pregnant and breastfeeding women should insist that the prenatal vitamins they are prescribed contain iodine.

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