Dietary Iodine Intake in Pregnancy: An Update

Abstract:
Iodine deficiency, by depriving the thyroid gland of sufficient raw material to produce thyroid hormones, can be either acute or chronic. While the consequences of severe iodine deficiency (median urinary iodine excretion UI < 20 ug/L) can be extreme, including irreversible mental retardation, those arising from more moderate deficiency may take a more subtle form. There is no evidence of a threshold for the development of hypothyroidism and to date there are no recent studies in Ireland or the UK linking iodine intake to neuropsychological development as has been frequently reported. NHMRC report on iodine deficiency in South Tipperary showed that a higher percentage of schoolchildren with slight or established goitre had what was termed poor intelligence. The most recent studies showing borderline iodine deficiency in Ireland have been reported by findings on the iodine status of UK schoolgirls reported in the Lancet by Vanderpump and colleagues. This report was timely, as despite the absence of recent evidence, it had been assumed that the UK population was iodine sufficient. The study carried out on 737 schoolgirls aged 14-15 years found that urinary iodine (UI) concentration, a measure of dietary iodine intake, showed a median of 80.1µg/L with 51% having values <50µg/L and 17% < 25µg/L. These values are indicative of a mild to moderate iodine deficiency state (WHO). The lowest UI values came from the Belfast centre where a median value of 64.7µg/L with 31% of values < 50 µg/L were found. The relatively low values from N. Ireland support earlier findings reported in 2006 in first trimester (T1) pregnant women studied in the National Maternity Hospital, Dublin (NMH) which a median UI of 52 µg/L was observed with 48 % of values indicative of moderate iodine deficiency. Although a significant source of iodine for people in many countries. However mandatory universal salt iodisation (USI) has not been implemented globally, while in many areas implementation is voluntary. Without salt iodization, the intake of iodine is opportunistic, which in the absence of iodine supplementation results in significant negative maternal iodine balance. The common cause underlying the continuing existence of iodine deficiency in the Irish and UK populations, in contrast to those of other developed countries, is the low availability of iodised salt; < 5% of salt sold is iodised. In iodised countries, the finding of diminishing population UI values in the absence of adequate iodised salt in Australia and New Zealand has resulted in the introduction of mandatory iodisation of salt in commercially baked bread. In the absence of iodine supplementation, the ability to maintain adequate thyroid hormone production may depend on a woman's thyroid hormone stores before conception. These in turn reflect long-term dietary iodine intake or previous parity, as it has been shown that multiparous women have larger thyroids than women who have only had one pregnancy. The presence of adequate pre-existing iodine stores may explain why pregnant women in Ireland and the UK display relatively normal thyroid hormone levels despite having daily iodine intakes lower than those recommended by the WHO as indicated by their UI values. Another factor possibly contributing to normal thyroid hormone production in the absence of apparently inadequate iodine intake may be the ability of the placenta to store iodine. Since it has also been demonstrated that placental iodine storage increases with increased iodine intake, this may be iodine consumed over the gestation period that will be sufficient to meet thyroid hormone requirements for fetal development, even if UI does not appear to suggest iodine intake below WHO recommendations. There are a number of possible solutions to the problem of inadequate dietary iodine intake particularly in developed countries. The solution of universal salt iodisation recommended by the WHO has the advantage of being a simple intervention which can be introduced without any change in dietary habits, in particular table salt iodine intake, but simply involves the addition of an iodinated substance, usually potassium iodide (KI) or potassium iodate (KIO3) to table salt at a concentration of 20 to 40 mg/kg. The population most at risk from iodine deficiency disorders are fetuses, particularly those conceived in the spring or early summer months. However to achieve protection of this cohort, females of child bearing age would require preconception iodine supplementation. The recent report of Mohri et al. supports this view as their finding that previous supplementation was the most effective in reducing risks of inappropriately low FT4 levels during pregnancy. Such supplementation forms the basis of the USP institute recommendations. However USI does not involve increased salt consumption. It could reduce the presentational problems in light of ongoing campaigns to combat hypertension by reducing salt consumption. Another method to reduce the iodine intake in the manufacture of commercially prepared salt and in accountability of USI is discussed in Australia and New Zealand. These methods have the advantage of supplying iodine to the entire population. However if the major at risk group, females of child bearing age subject to non-planned pregnancies, were to be targeted, perhaps the now accepted principle of preconception folate supplementation could be applied to iodine with the potential of providing fetal brain development protection at minimal risk and cost.

References
10. Mohri M, Trimarchi F, Vermiglio F. Maternal thyroid function in different conditions of iodine nutrition in pregnant women exposed to moderate low dietary iodine intake: an epidemiological study Clinical Endocrinology 74: 762-769