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Comments

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It is well established that there are regional variations in dietary iodine intake with consequent differing prevalences of iodine deficiency disorders (IDD)[1]. Traditionally, North America and Japan were judged to be iodine replete, while other regions, such as parts of Europe, Africa, Asia and South America, showed a variation in patterns of IDD[2]. Since the WHO reports of 1960 and 1994, improvements in iodine intake have been effected mainly as a result of salt iodisation programmes[1]. However, in other countries, notably in the USA and Australia, iodine intake as assessed by urinary iodine (UI) excretion has been reported to be in decline[3–5]. Recent reports[6,7] suggest that the fall in dietary iodine intake in the USA, at least in terms of the UI concentration, may have stabilised as the population UI concentration in 2000 was reported to be 161 μg per l[7], while the value in pregnant women was 149 μg per l[6]. Nonetheless, 9% of pregnant women had a UI concentration $<50\,\mu g\,l^{-1}$, suggesting an iodine deficiency.

The decline in iodine intake in the general population of the United States between the first National Health and Nutrition Examination Survey (NHANES I) and the NHANES III[5] was also seen in pregnant women. However, it was noted that pregnant women tended to have a higher UI excretion when compared with the general population as shown by a lower prevalence of UI concentrations $<50\,\mu g\,l^{-1}$: 1.0 vs. 3.9% in NHANES I and 6.9 vs. 14.9% in NHANES III in pregnant women when compared with the general population in both the surveys. This may reflect the increase in UI concentration during pregnancy which, it was suggested, results from an increased glomerular filtration rate[8], a phenomenon that was confirmed later[9–11]. An increased UI concentration during pregnancy is not a universal finding as both lower and unchanged values have been reported[12]. These regional differences in UI excretion may reflect a UI threshold above which iodine leakage occurs, the so-called 'iodostat'[13]. In iodine deficiency, this threshold may not be reached, while in an area where the population are iodine replete, the threshold may be obscured[9,10]. It has further been suggested that iodine losses in pregnancy may contribute to a negative maternal iodine balance[13–15].

These findings are particularly relevant in posing questions about the correlation between the UI concentration and the thyroid hypofunction or hypothyroxinemia[7]. In NHANES III, otherwise normal individuals may have been excreting at the time of study a concentration of iodine less than $50\,\mu g\,l^{-1}$, a value believed to indicate iodine deficiency, but this may not necessarily reflect long-term patterns[16]. 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[8,9]. This suggests that the thyroid gland may have been undergoing pregnancy-related stress[17] manifested as an increased thyroid volume, increased serum thyroglobulin (Tg) concentration and a decreased serum-free thyroxine concentration. This so-called 'thyroid stress' can be prevented if iodide is administered during pregnancy[18,19].

Although the richest potential dietary sources of iodine come from marine flora and fauna, iodised salt forms a significant source of iodine for people in many countries. However, universal salt iodisation has not been implemented in many countries, while in others implementation is voluntary[1]. In European nations, only 27% of households routinely consume iodised salt. In the absence of iodised salt, the intake of iodine is therefore opportunistic. In northern Europe, milk has been shown to be a major source of dietary iodine intake. However, agricultural practices arising from climatic conditions, for example when cattle are brought in from pasture during the winter months and fed on dietary supplements containing iodine, cause the concentration of iodine in milk to show a seasonal variation so that both the milk iodine and the UI concentration of humans who consume milk are relatively low during the summer months and increase during the winter months[20–22]. Whether this has implications for the iodine status of mothers is unclear. Recent unpublished studies in Ireland and the UK suggest that the concentration of iodine excreted in urine by pregnant women is declining, and this may have implications for maternal and foetal thyroid function.

Despite its inadequacies, the measurement of UI excretion during pregnancy continues to provide the most readily available indicator of iodine deficiency[1]. Other techniques, such as the measurement of thyroid volume or the concentration of Tg in serum, may not be readily available or may be too expensive. Another index of iodine deficiency is the number of borderline high concentrations of thyroid-stimulating hormone (TSH) observed in the course of neonatal screening for

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congenital hypothyroidism[23]. As with measuring the serum Tg concentration, the cost of the assay would prohibit this course as a first-line screening procedure, but it can be a useful resource if already in place. Both the variability of UI concentration during pregnancy and the different ways values are expressed create problems in using this as an index to estimate the prevalence of iodine deficiency in pregnancy. However, as in the general population, the UI concentration can still provide an indicator of iodine status and demonstrate if a requirement exists for iodine supplements, specifically directed at pregnant women. In the absence of such supplementation, the ability to maintain maternal thyroid hormone stores that are adequate to sustain optimal foetal development may depend on thyroid hormone stores before conception which, in turn, rely on the ready availability of dietary iodine provided by iodised salt.

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