Original Article

Re-emergence of iodine deficiency in Australia

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Iodine is an essential nutrient for human growth and development. The thyroid gland is dependent upon iodine for production of thyroid hormone. It is a common perception that iodine deficiency is not a major public health concern in mainland Australia, with sporadic studies carried out about a decade ago showing average urinary iodine excretion levels of around 200 µg/day. Recent evidence, however, has shown that the consumption of iodine is declining in Australia. A similar situation has occurred in the USA. The present study was designed to evaluate the urinary iodine excretion (UIE), as the indicator of iodine nutrition, in samples obtained from various demographic groups in the Sydney metropolitian area, namely: schoolchildren, healthy adult volunteers, pregnant women and patients with diabetes. Urinary iodine in spot urine sample was measured in a Technicon II autoanalyser using an in-house, semiautomated method. The results in this communication show that all four study groups had the median UIE below 100 µg/L, the criteria set by the World Health Organization for iodine repletion, and confirm what has been described previously, that iodine deficiency has reemerged in Sydney, Australia. One of the major causes of the reduced iodine intake is the reduction of iodine in milk since the dairy industry replaced iodine-rich cleaning solutions with other sanitisers. Secondly, less than 10% of the population are currently using iodised salt. A national survey into the iodine nutrition status in Australia is urgently required as part of the establishment of a systematic surveillance and legislation is required to iodise all edible salt.

Key words: Australia, children, diabetes, iodine deficiency, pregnant women, Sydney, urinary iodine.

Introduction

The trace element iodine is an essential nutrient for human growth and development. The thyroid gland is dependent upon iodine for production of thyroid hormone and this is the only known physiological function for iodine. The association between iodine deficiency and endemic goitre has been known for centuries, but it is only recently that we have come to appreciate that environmental iodine deficiency can cause a wide spectrum of devastating mental and physical disorders, collectively described as iodine deficiency disorders (IDD).[1] While endemic goitre is the most visible consequence of iodine deficiency, the most significant and profound effects are on the developing brain.[2] The potential impact of iodine deficiency on the intellectual development of large segments of the populations in underdeveloped countries is of particular concern, especially when all of the adverse effects of iodine deficiency can be prevented by long-term, sustainable iodine prophylaxis.

The minimum daily iodine intake, recommended by the World Health Organization (WHO), varies with age, ranging from 50 to 120 µg from infancy to adolescence, 150 µg for adults and 200 µg for pregnant and lactating women.[3] As 90% of ingested iodine is eventually excreted in the urine, measurement of urinary iodine concentration is an accurate reflection of dietary iodine intake. The median urinary iodine concentration in casual urine samples, expressed as micrograms per litre (µg/L), is currently the most practical biochemical marker of community iodine nutritional status and is widely used in population-based surveys.[4] A urinary iodine concentration of 100 µg/L is considered by WHO as the minimal urinary iodine concentration for iodine sufficiency and corresponds roughly to a daily intake of 150 µg iodine.[4] Primary schoolchildren are an appropriate population group for the assessment of iodine deficiency because of their physiological vulnerability and their accessibility for testing. Furthermore, measurement of urinary iodine levels in schoolchildren is important for public health considerations, as this group effectively reflects the current status of IDD in the general population, as well as the extent to which IDD control measures have had an impact on the population.

While there has not been any systematic surveillance of iodine nutrition in mainland Australia, it has generally been claimed that the Australian population has been iodine replete for some decades, with sporadic surveys showing average urinary iodine excretion levels in excess of 200 µg/day.[5] In Tasmania, where endemic goitre was once a very common problem, the Tasmanian Department of Health, through a Thyroid Advisory Committee, has been monitoring goitre rates and urinary iodine excretion in schoolchildren since 1949. Anecdotal reports of declining urinary iodine levels in Tasmanian schoolchildren and the results of small sporadic

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surveys that we have conducted in Sydney prompted this current study. It was designed to evaluate a small group of samples obtained from normal healthy schoolchildren, healthy adults and pregnant women to ascertain if there was any evidence to support the view that iodine deficiency is reemerging in the Australian community.

Materials and methods

Study participants

The study was undertaken in Sydney in late 1998 and early 1999. Participants in the study included healthy schoolchildren, normal adult volunteers, pregnant women and patients with diabetes attending the outpatient clinics at Westmead Hospital.

Schoolchildren

A random selection of 94 healthy primary schoolchildren, of both sexes, aged between 6 and 13 years from a north-western suburb of metropolitan Sydney cooperated in the study. These children came from a suburb considered to be typical of an upper middle-income community in NSW. Consent forms were obtained from the School Council and parents of the children.

Pregnant women

Spot urine samples were collected from 101 full-term pregnant women attending an antenatal clinic at Westmead Hospital.

Healthy adult volunteers

Urine samples were collected from 86 healthy Institute of Clinical Pathology and Medical Research (ICPMR) staff, of both sexes and aged from 21 to 60 years, who had no previous history of thyroid disease and who had not knowingly taken iodine containing medications or supplements during the previous 6 months.

Diabetic patients

Urinary iodine concentrations were measured on 85 urine samples from diabetic patients submitted to the laboratory for routine microalbumin analysis.

Sample collection

A random urine sample (50–70 mL) was collected with no preservative added. Names, ages and sex were recorded. The urine samples were divided into three × 4 mL aliquots and stored frozen at – 20°C until assay.

Urinary iodine measurement and statistical analysis

Urinary iodine was measured in a Technicon II autoanalyser (Bran & Luebbe, Sydney, Australia) by the Sandell-Kolthoff reaction using an in-house, semiautomated method that has been developed to ensure removal of interfering substances (designated as ICPMR method).[6] While this method has been validated in the past by many different comparative studies, for the purposes of the present study it was decided to compare the results from our in-house method with two other methods to ensure the data obtained was not biased by a methodological artefact. A comparison was performed on samples from schoolchildren (87 out of the total of 94) with a manual acid digestion method developed by Dunn *et al.*[7] This method was known as 'Method A', that is recommended by the International Council for Control of Iodine Deficiency Disorders (ICCIDD) and WHO. Samples analysed by Method A were measured by the National Reference Laboratory for IDD in Beijing, Peoples Republic of China. The other comparative study was undertaken in-house with measurement of urinary iodine by a new technology using inductively coupled plasma mass spectrophotometry (ICPMS) with SPS-5 autosampler (Varian, Palo Alto, California, USA).

The urine samples of the schoolchildren were each tested by the ICPMR method, Method A and ICPMS. Agreement between the three different methods was measured by examining the differences between the methods against their means.[8] The other groups of samples were measured only by the ICPMR method. All results are presented as the median value (μg/L) measured by the in-house ICPMR method.

The statistical analysis was conducted using SPSS for Windows.

Results

Urinary iodine concentration in different groups

The median urinary iodine concentrations of the study groups are presented in Table 1. The median levels were very similar for all groups, being 84 μg/L for schoolchildren, 88 μg/L for pregnant women, 88 μg/L for healthy adults and 69 μg/L for the diabetic patients. Each of these median levels is consistent with mild iodine deficiency.

The frequency distribution of urinary iodine concentrations

Figure 1 presents the frequency distribution of the four groups in the ranges of 0–20, 21–50, 51–100, 101–200 and > 200. The figure shows that 13 (13.8%), 13 (20.6%), 18 (17.8%) and 20 (23.5%) individuals of these groups, respectively, had urinary iodine excretion < 50 μg/L, in the range of moderate iodine deficiency.

Agreement between different methods

The agreements between (a) ICPMR in-house method versus ICPMS (b) ICPMR in-house versus Method A and (c) Method A versus ICPMS, respectively, were measured (Fig. 2).

Table 1. Median urinary iodine excretion

Groups	*n*	Median UIE (μg/L)	Range (μg/L)
Schoolchildren	94	84	28–312
Healthy adults	63	88	12–200
Pregnant women	101	88	20–448
Diabetic patients	85	69	20–234

UIE, urinary iodine excretion.

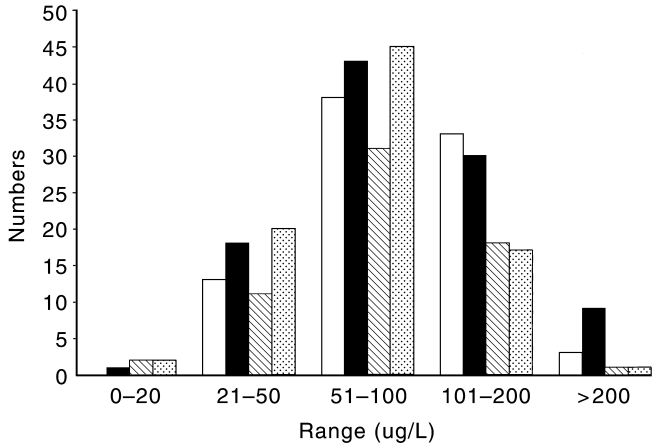

Figure 1. The frequency distribution of urinary iodine excretion in the range of 0–20, 21–50, 51–100, 101–200 and > 200 for the four study groups. Between 13.8% to 23.5% of individuals of the study groups had urinary iodine level below 50 µg/L. (□), schoolchildren; (■), healthy adults; (▨), pregnant women; (▦), diabetic patients.

The differences between two methods were plotted against their average.[8] There was a good agreement among the three methods over the range of 0–100 µg/L, which is the range that we are most concerned about. However, results diverged with increasing urinary iodine concentrations. The 95% confidence intervals for the bias of each pair became – 37.8–54.2, – 41.2–52.2 and –17.6–25.2, respectively.

Discussion

For population studies and classification of iodine nutritional status WHO states that median levels of urinary iodine of 0–20 µg/L, 20–50 µg/L and 50–100 µg/L indicate severe, moderate and mildly iodine deficient populations, respectively.[9] To be iodine replete a given population should have a median urinary iodine level of 100 µg/L. Using these criteria, each of our samples can be classified as suffering from mild iodine deficiency. Our results are very similar to those published recently by Gunton *et al.* who found median urinary iodine levels ranging from 64 µg/L in normal volunteers to 104 µg/L in pregnant women in small samples from a Sydney population.[10] Thus, two independent studies, sampling from different regions in Sydney, have reported very similar results. Comparing these results with data obtained in Sydney nearly a decade ago, it is apparent that iodine intake in our community has decreased by one-half over this time period.[5] A comparison of different methods for measuring iodine in urine indicates that the decline in urinary iodine excretion is real and is not an artefact of the analytical method used to measure iodine.

What is most alarming is the finding that approximately 60% of the pregnant women in our study displayed urinary iodine levels consistent with mild to moderate iodine deficiency. While the severity of the iodine deficiency in these women is not commensurate with the development of severe neurological consequences in their offspring, we know that even mild maternal iodine deficiency can result in a decrease in cognitive capacity and subtle psychomotor defects in the population.[11,12] More recently, a number of studies have shown a significantly increased risk of impaired neurodevelopment in the infants of apparently healthy pregnant women

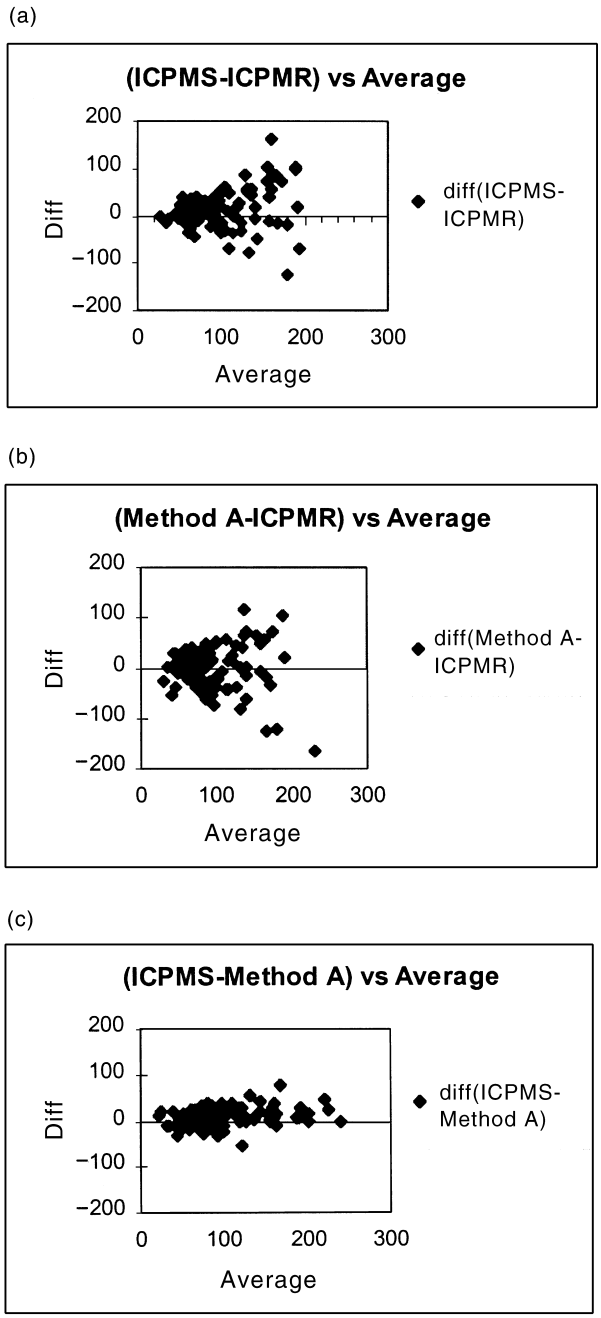

Figure 2. The agreement of different urinary iodine measurement methods was examined by plotting the different between the results by two methods against the average of the two results. (a) Inductively coupled plasma mass spectrophotometry (ICPMS) versus Institute of Clinical Pathology and Medical Research (ICPMR); (b) Method A versus ICPMR and (c) ICPMS versus Method A. There was a good agreement in the range 0–100 µg/L. The results diverged in the higher values.

with decreased serum thyroid hormone levels.[13,14] While we have no data on circulating thyroid hormone levels in our study group it is possible that moderate iodine deficiency during pregnancy may result in decreased maternal thyroid hormone production and cause subtle, but significant, psychomotor defects in the children born of these women.

There is a growing body of evidence supporting the view that iodine consumption is declining in the Australian population. Milk contaminated by iodophores has been the major source of iodine in the Australian diet for the past 40 years. Iodophores are compounds of iodine linked to surfactants

that act as carriers or solubilising agents for iodine. A small amount of free iodine is released in solution, thereby minimising toxicity while preserving moderate germicidal activity of the element.[15] Since the early 1960s iodophores have been used in the dairy industry, in most parts of Australia, for cleaning and sterilising milking apparatus and containers.[16] While the dairy industry monitors milk iodine concentrations in milk tankers, we are not aware of any monitoring of milk at the retail level. Obviously, there is considerable and unpredictable variation in milk iodine levels throughout the country. However, anecdotal evidence indicates that iodine concentrations in milk of up to 300 μg/L were not unusual until recent times. Over the past few years there appears to be a trend in the dairy industry towards use of non-iodine containing germicidal agents and this the most likely cause of declining iodine intake in our community.

Iodised salt is widely available commercially, but only 10% of all households' purchase iodised salt for domestic use. Furthermore, as table salt is not the major source of salt in our diet, the contribution iodised table salt makes to iodine nutrition is probably insignificant. Most of the salt in our diet comes from salt added in the preparation and processing of food. As far as we can ascertain, iodised salt is not used in the food industry. Thus, we have an extraordinary situation in Australia where the community has been dependent upon contamination of a staple food for an essential micronutrient.

Iodine deficiency disorder is not just an important public health issue in the underdeveloped world. It could also be a major threat to all industrialised nations if vigilance is not maintained and if it is not properly managed. Alarms have been raised recently from other parts of the world, particularly New Zealand, Europe and the USA, where a similar trend of declining iodine nutrition has been observed.[17,18] Accordingly, the results of the present study and that of Gunton *et al.*[10] raise serious concerns about the adequacy of iodine intake in Australia and the need for more information through a systematic national survey of both process and outcome indicators of iodine deficiency.[20]

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