The changing epidemiology of iodine deficiency

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Abstract | Globally, about 2 thousand million people are affected by iodine deficiency. Although endemic goitre is the most visible sign of iodine deficiency, its most devastating consequence is brain damage causing mental retardation in children. The relationship between iodine deficiency and brain damage was not clearly established until the 1980s when the term iodine deficiency disorders (IDDs), which encompass a spectrum of conditions caused by iodine deficiency, was introduced. This paradigm shift in the understanding of the clinical consequences of iodine deficiency led to a change in iodine deficiency assessment. The median urinary iodine excretion level has been recommended as the preferred indicator for monitoring population iodine deficiency status since 2001. The 2007 WHO urinary iodine data in schoolchildren from 130 countries revealed that iodine intake is still insufficient in 47 countries. Furthermore, about one-third of countries lack national estimates of the prevalence of iodine deficiency. The picture that has emerged from available data worldwide over the past two decades is that IDDs are not confined to remote, mountainous areas in developing countries, but are a global public health problem that affects most countries, including developed countries and island nations. The recognition of the universality of iodine deficiency highlights the need to develop and apply new strategies to establish and maintain sustainable IDD elimination and strengthen regular monitoring programmes.

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Introduction

Endemic goitre has been synonymous with iodine deficiency for a long time. Countries were often divided geographically into endemic and nonendemic areas on the basis of goitre prevalence. Control efforts were directed towards curing or reducing goitre. With growing clinical and public health research, it became clear that the consequences of iodine deficiency go far beyond those of goitre and thyroid disease.[1] In 1983, the term iodine deficiency disorders (IDDs) was coined to emphasize that iodine deficiency can affect human beings at all stages of the life cycle and have a broad spectrum of adverse effects, including mental and physical impairment, disturbed thyroid function and goitre (Table 1).[2] Children from 2 months to 15 years of age born in areas of moderate to severe iodine deficiency can lose up to 13.5 IQ points.[3,4] At a population level, iodine deficiency has a negative impact on a country's overall health and productivity and hinders its socioeconomic development.[5] The recognition that iodine deficiency is a major public health problem throughout the world has led to a global acceleration of efforts to combat this deficiency.

Despite the progress of IDD elimination programmes, more than 2 thousand million people worldwide are still at risk of insufficient iodine intake.[6,7] In developing countries, 38 million newborn babies per year are not protected from the devastating consequences of iodine deficiency.[8] The problem, however, is not confined to developing countries. Iodine deficiency has been found in highly developed countries where it had been considered to have been eliminated or not exist. This Review examines the paradigm shift in iodine deficiency control strategies over the past few decades and the effect of these changes on the assessment and prevalence trends of iodine deficiency worldwide. The article also highlights the evidence for iodine deficiency in developed countries gathered in the past decade and emphasizes the importance of public health policy to support sustainable IDD elimination and surveillance programmes.

Pathophysiology of iodine deficiency

Thyroid iodine metabolism

Dietary iodine is readily absorbed from the gastrointestinal tract and reaches the circulation in the form of iodide. Iodide is mostly cleared from the circulation by the thyroid gland and kidney.[9] During pregnancy and lactation, the mammary gland also concentrates and excretes iodine in breast milk.[10] The recommended daily iodine intakes by the WHO, UNICEF and International Council for the Control of Iodine Deficiency Disorders (ICCIDD),[11] and by the American Institute of Medicine[12] are shown in Table 2.

In the thyroid gland, iodide is converted back to iodine and concentrated in the follicular cell. The thyroid contains approximately 70–80% of the total iodine of a healthy adult body (~15–20 mg).[9,13] In the iodine-sufficient human, the thyroid takes up ~10% of the iodine from the circulation, whereas in states of chronic iodine deficiency thyroid iodine uptake can be >80%.[14,15] Iodine is an integral constituent of the thyroid hormones T_4 and T_3. Iodine is incorporated into tyrosine residues of the thyroglobulin molecule and stored in the colloid

Competing interests
The authors declare no competing interests.

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Key points

- Despite efforts to monitor changes in the magnitude of iodine deficiency worldwide, prevalence data are crude and are still not available for many countries
- Iodine deficiency is re-emerging in some developed countries; therefore, public awareness and government policies on iodine fortification, supplementation and surveillance of iodine deficiency are warranted
- Currently, the WHO, UNICEF and ICCIDD recommend using median urinary iodine concentration in school-age children as a proxy for the iodine nutrition status of the general population
- The applicability of this recommendation for the most vulnerable population groups, such as pregnant women and young children, needs to be reviewed
- At the population level, close monitoring and surveillance of iodine intake is an important public health measure to ensure optimal iodine nutrition

Table 1 | Health consequences of iodine deficiency

Physiological groups	Health consequences of iodine deficiency
All ages	Goitre Hypothyroidism Increased susceptibility to nuclear radiation
Fetus	Spontaneous abortion Stillbirth Congenital anomalies Perinatal mortality
Neonate	Endemic cretinism including mental deficiency with a mixture of mutism, spastic diplegia, squint, hypothyroidism and short stature Infant mortality
Child and adolescent	Impaired mental function Delayed physical development Iodine-induced hyperthyroidism
Adults	Impaired mental function Iodine-induced hyperthyroidism

Permission obtained from the WHO © WHO, UNICEF & International Council for the Control of Iodine Deficiency Disorders. *Assessment of Iodine Deficiency Disorders and Monitoring Their Elimination: A Guide for Programme Managers* 3rd edn [online]. Accessed: February 2012. http://whqlibdoc.who.int/publications/2007/9789241595827_eng.pdf (2007).[11]

lumen. During hormone secretion, the iodinated tyrosines are removed from the thyroglobulin by proteolysis and T_4 and T_3 are released into the circulation.[16] TSH stimulates all stages of thyroid iodine metabolism, including iodine transport into the thyroid gland and the synthesis and secretion of thyroid hormones.[15,16] Excessively ingested iodide is readily excreted in urine (~90%), with only small amounts being discharged in the faeces and sweat.[16,17]

Thyroid adaptation to iodine deficiency

The thyroid adapts to low dietary iodine intake in several ways, which are mediated through increased secretion of TSH by the pituitary gland. First, TSH stimulates iodide uptake by the thyroid gland, which leads to reduced renal clearance of iodide and hence reduced urinary iodine excretion. Second, TSH stimulates thyroglobulin breakdown and enhances preferential secretion of T_3, and the conversion of T_4 to T_3 in peripheral tissues.[18] In chronic iodine deficiency, despite these changes, the total thyroid iodine content is depleted, which results in hyperplasia of thyroid epithelial cells. The development of goitre is a natural consequence of these processes.[19] The serum thyroglobulin concentration is also markedly elevated in chronic iodine deficiency.[18]

Indicators for assessing iodine deficiency

Total goitre rate

For a long time, iodine deficiency in certain geographical regions was determined by the prevalence of goitre in the population: the total goitre rate (TGR).[19,20] Changes in goitre prevalence, however, lag behind changes in iodine nutrition status after a salt iodization programme has been initiated;[21,22] therefore, goitre prevalence does not accurately reflect the current iodine intake. Baseline assessment of goitre by palpation or by ultrasonography, nevertheless, remains useful,[23,24] and this approach is still applied to evaluate changes in prevalence of iodine deficiency after the introduction of salt iodization programmes in developing countries.[25]

Median urinary iodine concentration

As approximately 90% of absorbed iodine is eventually excreted in the urine,[16] the urinary iodine concentration (UIC) is a good indicator of changes in dietary iodine intake over preceding days or weeks. The WHO, UNICEF and ICCIDD have recommended the median UIC in schoolchildren as the main indicator for assessing and monitoring the iodine nutritional status of a population.[26] The use of UIC to monitor IDD has redefined the epidemiology and distribution of iodine deficiency, as iodine deficiency has now been found to be prevalent in developed countries, major cities, coastal areas and island nations where goitre prevalence is low and iodine deficiency had been considered nonexistent or to have been eliminated. Furthermore, iodine intake above requirements and excessive iodine intake have been detected by measuring UIC. Table 3 shows the epidemiological criteria for assessing iodine nutrition on the basis of UIC and IDD status on the basis of TGR.

Neonatal TSH concentration

The pituitary production of TSH is regulated by circulating T_4 levels. The serum TSH concentration is inversely related to the serum free T_4 concentration. Iodine deficiency reduces the circulating T_4 level, which results in an elevated serum TSH concentration. Hence, the neonatal blood TSH concentration can be a valuable indicator of iodine deficiency.[27] However, many potential confounding factors exist (such as the day of sample collection after birth, assay methods and contamination by iodine containing antiseptics during delivery) that can discredit the data and their interpretation.[28] Neonatal TSH testing should only be used as one of the indicators of iodine deficiency in a population if a robust universal neonatal screening system exists.

Following the introduction of salt iodization programmes in many countries, the level of household iodized salt coverage is used as an important indicator for assessing the progress of these programmes.[8,11]

Global trend of iodine deficiency

The early 1990s were a turning point in global efforts to combat iodine deficiency. This period of time was marked by several high-level meetings, including the 1990 World Health Assembly,[29] the World Summit for Children in 1990,[30] the Ending Hidden Hunger conference in 1991,[31] and the International Conference on Nutrition in 1992.[32] Subsequently, in 1994, the WHO recommended eliminating IDDs by iodizing all salt for human consumption.[33] This brought about a major shift in the strategy for control of IDD from focused, small-scale salt iodization programmes in 'endemic areas' to a surge of countries adopting more systematic salt iodization programmes. Globally, the proportion of people consuming iodized salt increased from <20% in 1990 to ~70% by the year 2000, which contributed to a considerable reduction in the prevalence of iodine deficiency worldwide.[8,34]

The report on the global prevalence of IDDs published by the WHO in 1993 included data from >120 countries.[20] The report provided the baseline estimates of the magnitude and distribution of iodine deficiency worldwide on the basis of revised TGR criteria, in which countries with TGR >5% are classified as iodine deficient. For the first time, the population at risk of iodine deficiency was quantified. An estimated 1,570 million people or ~30% of the global population were reported to be at risk of iodine deficiency. The global prevalence of goitre was estimated to be 12% and 110 countries were classified as iodine deficient.[20]

Following the implementation of universal salt iodization (USI) programmes, monitoring data became available in many countries. In 2004, the WHO revised information on the global iodine status in the Global Database on Iodine Deficiency.[35] From the revised WHO data, Andersson *et al.* reported that in 54 countries iodine deficiency was a public health problem (median UIC in schoolchildren <100 μg/l).[36] Iodine deficiency was most prevalent in the WHO Region of Europe (59.9%) and least prevalent in the Americas Region (10.0%). The extrapolation of the iodine deficiency prevalence data from the schoolchildren data indicated that about 2 thousand million individuals worldwide had insufficient iodine intake.[35,36] The estimated TGR in 2003 was 15.8%. whereas that reported in 1993 was 12%,[20] which was largely attributed to the increase in the Africa and Europe Regions.[36]

The 2005 World Health Assembly (WHA) adopted a resolution requiring the WHO Member States to report on the country's iodine deficiency situation every 3 years.[37] The global estimates of the magnitude of iodine deficiency were updated in 2007.[6,7] These data are the most up-to-date global iodine deficiency status estimates (Table 4) and highlight a number of important issues. First, global progress had been made in controlling iodine deficiency; the number of countries where iodine deficiency was a public health problem (median UIC <100 μg/l) had reduced from 54 in 2003 to 47. However, the estimated number of people in the general population having insufficient iodine intake remained at 2 thousand million. This coincided with a minimum increase in the world household iodized salt coverage.[38] Second, the number of countries having more than adequate or even excessive iodine intake had increased from 27 to 34, which demonstrated the needs to strengthen the monitoring of salt iodization. Third, as the median UIC is still used as the proxy indicator of iodine deficiency for the whole population, including special population groups such as pregnant women,[39] only limited data exist to estimate iodine deficiency in pregnant women. Fourth, a substantial number of countries still did not have iodine deficiency status data. Only 130 of the 193 WHO Member States had available data, which makes the 2005 WHA resolution difficult to achieve.[37] In addition, lack of nationally representative data[36] could result in an underestimate or an overestimate of the true iodine deficiency status.

Since 2000, UNICEF has been tracking the progress of USI programmes by using the household iodized salt coverage rate as an indicator.[8] UNICEF global data collected in 2007 showed that the proportion of households in the developing world consuming adequately iodized salt remained at about 70%.[8,38] This lack of progress highlights the challenges that some developing countries face. However, 34 countries had achieved the IDD elimination goal of >90% of households consuming adequately iodized salt by 2007, whereas only 21 had done so in 2002.[8] One of the biggest improvements between 2002 and 2007 was the increased availability of data on household iodized salt consumption; 66 countries did not have any data in 2002 compared with 34 in 2007.

Despite evidence of improvement, a sizable proportion of developing countries still have no iodine deficiency data, and data from developed countries are not available in the UNICEF database. At country level, the alignment of iodine nutrition as assessed by schoolchildren's median UIC in the WHO Global Database on Iodine Deficiency and as indicated from the UNICEF global database of household iodized salt consumption is not always consistent. For instance, India was classified as having optimal iodine nutrition (median UIC 100–199 μg/l) by the WHO,[6] but the household iodized salt consumption in

Table 2 | Recommended iodine intake

Age or population group	Recommended iodine intake (μg per day)
WHO/UNICEF/ICCIDD RNI	
Children 0–5 years	90
Children 6–12 years	120
Children >12 years and adults	150
Pregnant women	250
Lactating women	250
Institute of Medicine RDA	
Infants 0–12 months	110–130
Children 1–8 years	90
Children 9–13 years	120
Children ≥14 year and adults	150
Pregnant women	220
Lactating women	290

Abbreviations: ICCIDD, International Council for the Control of Iodine Deficiency Disorders; RDA, recommended dietary allowance; RNI, recommended nutrient intake.

Table 3 | Epidemiological indicators and criteria for assessing iodine deficiency

Criteria and population group	Iodine intake (iodine nutrition status) or IDD status
Median UIC in children aged ≥6 years and adults (μg/l)	
<20	Insufficient (severe iodine deficiency)
20–49	Insufficient (moderate iodine deficiency)
50–99	Insufficient (mild iodine deficiency)
100–199	Adequate (adequate iodine nutrition)
200–299	Above requirements (more than adequate intake, may pose a slight risk in the general population)
≥300	Excessive (risk of adverse health consequences)
Median UIC in pregnant women (μg/l)	
<150	Insufficient
150–249	Adequate
250–499	Above requirements
≥500	Excessive
TGR in children aged ≥6 years (%)	
≥30	Severe IDD status
20.0–29.9	Moderate IDD status
5.0–19.9	Mild IDD status
0.0–4.9	No IDD or IDD eliminated

Abbreviations: IDD, iodine deficiency disorder; TGR, total goitre rate; UIC, urinary iodine concentration. Permission to adapt obtained from the WHO © WHO, UNICEF & International Council for the Control of Iodine Deficiency Disorders. *Assessment of Iodine Deficiency Disorders and Monitoring Their Elimination: A Guide for Programme Managers* 3rd edn [online]. Accessed: February 2012. http://whqlibdoc.who.int/publications/2007/9789241595827_eng.pdf (2007).[11]

India was reportedly only 51%, which clearly indicates the population iodine intake is not adequate.[38] The estimated number of newborn babies who are not protected from iodine deficiency in India is 13.3 million annually and the total unprotected population in India is 563.2 million.[8] This discrepancy of different measures of iodine deficiency further highlights the importance of establishing systematic national surveillance programmes.

Iodine deficiency in developed countries

Other than geographical location, iodine deficiency has been perceived to be associated with under-development.[20] Increasing evidence from the measurement of urinary iodine excretion shows that iodine deficiency is not confined to developing countries. For example, until the early 1990s, Australia was iodine replete.[40] Since then the re-emergence of iodine deficiency has been well documented.[41–49] The National Iodine Nutrition Study in schoolchildren, which was performed across five mainland states in 2003–2004, confirmed that Australia is mildly iodine deficient, with a weighted national median UIC of 96 μg/l. Median UIC was lowest in the two most populated states on the eastern seaboard.[50]

One of the explanations for iodine deficiency in Australia is that only a minority of people purchase iodized salt and the food industry does not use iodized salt in food production and preparation.[51] For over four decades, milk contaminated with iodine residues from iodophors used in the dairy industry has been the main source of iodine in the Australian diet, contributing at least 50% of the daily intake.[52] As a consequence of the replacement of iodine-containing sanitizers by other chemicals in the dairy industry in the 1990s, the iodine content in milk has been drastically reduced. Therefore, milk is no longer a rich source of iodine in Australia. A similar situation pertains to New Zealand, where iodine deficiency has also been reported in schoolchildren, infants and toddlers, and pregnant women.[53–56] Vanderpump and colleagues showed a similar result in the UK in 2011 in a cross-sectional survey of schoolgirls in nine cities.[57] The median UIC was 80 μg/l and the proportion of schoolgirls with UIC <100 μg/l was 69%.

Historically, a large part of Western Europe has been affected by iodine deficiency,[58,59] and as recently as 2003 up to 60% of the European population was iodine deficient.[36,60] With various voluntary measures, the prevalence of iodine deficiency has reportedly decreased by 30% since 2003.[61] Most governments of developed countries, however, still give low priority to addressing iodine deficiency in their populations.[61,62] Australia is one of few developed countries that has launched mandatory food fortification, requiring that bread must contain iodine (from iodized salt) in response to the demonstration of the re-emergence of iodine deficiency in its population.[63] This initiative, while expected to improve iodine nutrition in the general population, is unlikely to provide sufficient iodine nutrition to pregnant women.[64–66]

Iodine deficiency in Pacific islands

Iodine deficiency has not been perceived as a major public health problem in the Pacific island nations because of the long-held belief that the people who live in these islands regularly eat seafood, a rich natural source of iodine, as part of their normal diet. This assumption has held because available data are very limited. IDD was documented in the highlands of Papua New Guinea as early as 1960.[67] More recent data published in 2005 showed that schoolchildren from the Southern Highlands Province were moderately iodine deficient (median UIC 48 μg/l).[68] The Fijian 1994 survey reported a TGR of 48.6% among schoolchildren and of 14.1% among pregnant women. The median UIC was 26 μg/l in schoolchildren, while in pregnant women it was between 74 μg/l and 137 μg/l, depending on the location where they came from.[69] The 1998 survey in Timor-Leste showed that all 13 districts had a TGR >5%, indicating iodine deficiency was a public health problem,[11] and seven out of the 13 districts had a TGR ≥20% (M. Li, unpublished work).

In the past decade there have been anecdotal reports of goitre on the island of Tanna in Vanuatu. An iodine nutrition survey was carried out in 2007 to determine the iodine nutritional status of the schoolchildren, and the prevalence and possible cause of goitre. The median UIC among schoolchildren was 49 μg/l; 72% of children had UIC <100 μg/l and 51% had UIC <50 μg/l, respectively.[23] Furthermore, around 30% of boys and girls had thyroid gland volumes greater than the international standard 97th percentile for their age.[70] Known goitrogenic substances, such as thiocyanate and its precursors, are found in a number of foods (including cassava and sweet potatoes)[71] that are part of the Ni-Vanuatu diet, but

consumption of foods containing goitrogenic substances did not seem to be associated with thyroid size. Iodine deficiency is the most probable cause of enlarged thyroid glands. Against common perception, <40% of children living on the island reported eating seafood on a weekly basis, and among them only 30% ate fresh fish. These findings clearly indicate that even residents of island nations do not necessarily eat enough fresh seafood to ensure an adequate daily iodine intake.[23]

Iodine deficiency during pregnancy

Dietary iodine requirements increase during pregnancy.[72] Although the full effect of mild maternal iodine deficiency on the offspring is still not fully understood,[73] severe maternal iodine deficiency and hypothyroidism have been associated with impaired neurological development in the offspring.[74,75] Globally, national representative survey data on iodine nutrition during pregnancy are limited. Maternal iodine deficiency, defined as median UIC <150 μg/l,[11] however, has been well documented in both developing and developed countries.

The Thai cyclical iodine monitoring programme in pregnant women began in the year 2000.[76] Each year, 15 provinces, covering four regions in the north, north-east, central, and south of Thailand, were randomly selected and maternal urine samples were collected at delivery for the measurement of iodine content. The overall maternal median UICs from 2000 to 2003 were 153 μg/l, 112 μg/l, 107 μg/l and 115 μg/l. The lowest UIC levels were found in the north-east region, below 100 μg/l in all four years, and the highest in the south, in the range of sufficient iodine intake.[76] Pregnant women living in some communities in southern Thailand, however, were also found to be iodine deficient.[77] In these communities, the median UIC was 75 μg/l in the first trimester and the estimated daily iodine intake was <250 μg per day.[77] Insufficient iodine intake has also been found among pregnant women in Nigeria[78] and India.[79]

In the USA, although the median UIC more than halved between 1971–1974 and 1988–1994 (from 320 μg/l to 145 μg/l) in the general population,[80] it had stabilized at 164 μg/l in two recent surveys in 2005–2006 and 2007–2008.[81] However, a trend of insufficient iodine intake among pregnant women exists. The median UIC of women of reproductive age in 1988–1994 was significantly lower than in 1971–1974, at 126.6 μg/l (95% CI 120–135 μg/l) for nonpregnant women and 140.5 μg/l (95% CI 124–180 μg/l) for pregnant women.[80,82] The median UIC was borderline among 326 pregnant women in 2000–2006 (153 μg/l).[83] Data from the National Health and Nutrition Examination Survey (NHANES) during 2005–2008 indicate that the median UIC of a small sample of pregnant women ($n = 184$) had further declined to 125 μg/l (95% CI 86–198 μg/l) and 57% of this group had a UIC of <150 μg/l.[81] The country-wide assessment of iodine status among over 3,600 pregnant women in Portugal found that the median UIC value was 85 μg/l in continental Portugal, 70 μg/l in Madeira and 50 μg/l in Açores. Only 17% of the women met the WHO criteria of adequate iodine nutrition (UIC >150 μg/l).[84] Iodine deficiency in pregnant women appears to be widespread in developed countries and has been reported in Australia,[41,42,44,46] New Zealand,[56] Switzerland,[27,85] the UK[86] and Ireland,[87] and in 2011 in France[88] and Spain.[89] It has also been reported in Hong Kong.[90]

Table 4 | Insufficient iodine intake in populations by WHO Regions

WHO Regions	Insufficient iodine intake (UIC <100 μg/l)	
	School-aged children *n* in millions (%)	General population *n* in millions (%)
Africa	58.5 (41.4)	316.7 (42.0)
Americas	11.6 (10.6)	98.6 (11.0)
East Mediterranean	43.3 (48.8)	259.3 (47.2)
Europe	38.7 (52.4)	459.7 (52.0)
South-East Asia	73.1 (30.3)	503 6 (30.0)
West Pacific	41.6 (22.7)	374.7 (21.2)
Worldwide	266.8 (31.5)	2,012.6 (30.7)

Abbreviation: UIC, urinary iodine concentration. Adapted from Andersson, M. *et al. Best Pract. Res. Clin. Endocrinol. Metab.* **24**, 1–11 (2010), with permission from Elsevier.[7]

The slow progress in scaling up salt iodization programmes in some developing countries has justified the WHO and UNICEF jointly recommending that in areas of moderate and severe iodine deficiency (median UIC <50 μg/l or TGR >20%) and in countries in which salt iodization programmes are not successfully scaling up within 2 years, all pregnant and breastfeeding women should be provided with an iodine supplement.[91] Concerned by the possibility of inadequate iodine intake among pregnant women in North America, the American Thyroid Association recommended iodine supplementation during pregnancy and lactation in the USA and Canada in 2006.[92] Similarly, the National Health and Medical Research Council of Australia has recommended that all women in Australia should take a 150 μg iodine supplement per day during pregnancy and lactation, informed by the fact that mandatory iodine fortification of bread is not likely to provide adequate dietary iodine to this special subgroup of the population.[93,94]

The median UIC in schoolchildren is currently recommended as the indicator for assessment of iodine nutrition in a population.[11] The validity and the representativeness of this indicator for specific population groups, such as pregnant and lactating women, have been questioned in the past few years. Instances have arisen in which median UIC data in schoolchildren indicated sufficient iodine intake, but the median UIC was <150 μg/l in pregnant women.[6] In a study carried out in 11 provinces in China, the median UICs for pregnant and lactating women from the same community in most of these provinces were well below those of schoolchildren by about 50 μg/l and 40 μg/l, respectively.[95] A study from Thailand has reported that median UICs indicated iodine deficiency in pregnant mothers (108 μg/l), but iodine sufficiency in their school-aged children (200 μg/l).[96] In the US NHANES, children aged 6–11 years had a median UIC of ≥200 μg/l, whilst it was only 125 μg/l in pregnant women.[81] These pieces of evidence demonstrate that the assumption that population iodine nutrition is sufficient if the median UIC is adequate in schoolchildren is frequently incorrect.

Clearly, use of the median UIC in schoolchildren as a surrogate marker for that in pregnant women is unacceptable and often misleading. Monitoring programmes should move to direct collections of nationally representative data from pregnant women themselves.

Conclusions

The USI programmes implemented in more than 120 countries have changed the global epidemiology of iodine deficiency. USI should remain the primary strategy for sustainable elimination of iodine deficiency throughout the world. Different sets of barriers and challenges to USI exist in both developing countries and developed countries. Action at country and local levels is, therefore, important. Whereas the quality and availability of data have improved markedly, programme monitoring still needs to be strengthened to enable collection of nationally representative data for both iodine deficiency and possible iodine excess from the programmes. Increasing iodine deficiency in developed countries, particularly in pregnant women, requires the governments of these countries to address this problem. To ensure adequate iodine nutrition during pregnancy and lactation, women should be provided with iodine supplements if iodine nutrition is not optimal.

Review criteria

This manuscript is based on the authors' knowledge of the topic and extensive review of the literature. PubMed was searched for full-text articles in the English language published up until the end of August 2011 using the search terms "iodine deficiency", "iodine deficiency disorders", "iodine status", and "goitre/goiter". We also searched the WHO and UNICEF publications on iodine deficiency, and the International Council for the Control of Iodine Deficiency Disorders publications.

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Author contributions

M. Li researched the data for the article. Both authors wrote the article, provided substantial contributions to discussions of the content and reviewed and/or edited the manuscript before submission.