REVIEW

Human Reproduction and Iodine Deficiency: Is It a Problem in the UK?

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Women suffer from thyroid disorders between four and ten times as frequently as men of the same age. This has been shown to be due to iodine deficiency causing damage to the thyroid during pregnancy and is not true of women who have never been pregnant. Iodine deficiency not only damages women but causes impairment of brain development in their babies. These are the main reasons for the recent increase in the World Health Organization (WHO) recommendation for an iodine intake to 200 mcg/day during pregnancy. Many British women have iodine intakes much below this recommendation and indeed below 100 mcg/day. The WHO was, however, informed by the British government that the population at risk of iodine deficiency in the UK was ‘zero’. The WHO recommendations for action are discussed.

Keywords: iodine deficiency, thyroid disorders, brain development, learning disability, maternal nutrition, pre-pregnancy care, food fortification.

INTRODUCTION

A paper in this journal by Durrant-Peatfield [1] gave an interesting account of ‘thyroid dysfunction and management’ in clinical practice and suggested that hypothyroidism in particular has a higher prevalence than is generally thought. The present paper aims to extend the discussion of the effects of iodine deficiency and hypothyroidism to human reproduction with particular reference to the advice of the World Health Organization (WHO). The paper by Durrant-Peatfield [1, p. 372] noted that the thyroid “is five times more likely to be damaged in women than in men”. Research has shown that this higher percentage of thyroid damage only happens in women who have at some time been pregnant and have embarked on pregnancy with comparatively low iodine intakes. Pregnancy demands higher thyroid hormone secretion and stresses the thyroid, which may be permanently damaged if the iodine supply is inadequate. This discovery was one reason why the WHO sponsored a conference in Brussels in 1992 under the title ‘Iodine Deficiency in Europe: a Continuing Concern’. The proceedings have been published [2]. The greatest emphasis in the proceedings of this conference and the associated literature was not, however, on the damaging effect of a low iodine intake on the pregnant woman but on the effect of the low intake on her baby. The present review retains this emphasis.

Following the 1992 conference the WHO had further meetings at regional and global levels and increased the recommended iodine intake for pregnant women to 200 mcg/day [3]. The present review shows that there are large numbers of women in Britain with iodine intakes below the current WHO recommendations and, indeed, below the recommendations before the present increase. Does this matter? If it does matter, what is to be done?
THE EFFECTS OF IODINE DEFICIENCY ON THE BRAIN OF THE UNBORN CHILD

The WHO report emphasizes that iodine deficiency in pregnant women may cause irreversible brain damage in the developing fetus [4]:

Infants and young children exposed to iodine deficiency may also suffer from brain damage, psychomotor retardation and intellectual impairment. Thus IDD include a broad spectrum of conditions that vary in severity ... iodine deficiency also affects reproductive function, leading to increased rates of abortion, still births, congenital anomalies, low birth weights and infant and young child mortality (p. 2).

It is the effect of iodine deficiency in a mother on the mental and psychiatric development of her children that has come to be regarded during the last 15 years as of the greatest social importance, exceeding any direct consequences of iodine deficiency in adults. European countries are no longer troubled by extreme iodine deficiency resulting in cretinism, but by lesser degrees of maternal iodine deficiency resulting in losses in the mental ability of their children recorded at school age and expressed as a loss in learning ability. The introduction to a paper by Connolly and Pharoah [5, p. 317] (from the University of Sheffield and University of Liverpool, respectively) discusses hearing loss, motor competence and cognitive function. It states:

That iodine deficiency diseases present a spectrum of developmental consequences which vary from the gross to those detected only by careful and precise quantitative measures is of considerable significance biologically and socially. The less severe manifestations may in fact be of greater social significance because many more individuals are affected and put at developmental risk.

The cases of a modest loss of learning ability greatly outnumber the cases of children more seriously affected. The loss of mental ability of subpopulations is only one of the diverse consequences of iodine deficiency but the social and economic consequences of loss of intelligence are far reaching.

The association of fetal growth retardation with mental subnormality and cerebral palsy goes back to classic studies in the 1960s [6]. Maternal iodine deficiency causes general fetal growth retardation of all parts of the body including the brain. In 1988, a conference entitled ‘Iodine and the Brain’ was held in Bethesda, MD, supported by the US Institutes of Health [7]. Experiments were described in the published proceedings of this conference showing that a low iodine intake in sheep and marmosets causes reduced brain weight at birth [7, pp. 177–86]. Losses in brain weight of the human fetus of almost 50% at 8 months caused by iodine deficiency were also reported [7, pp. 249–57]. Low birth weight and small head size have many causes but iodine deficiency is one risk factor.

The 1988 conference was concerned with the difficult question of when the greatest susceptibility to iodine deficiency is found during the human life cycle. Severe deficiency in men, women and children causes lethargy, apathy, inactivity and loss of appetite [8, p. 92]. Lesser degrees of iodine deficiency severe enough to cause thyroid enlargement produce no psychological symptoms in adults. Women with goitre have been found not to suffer from neuropsychological defects [7, pp. 259–87]. A loss of IQ and learning disabilities are the fate of the children of mothers with goitre who were suffering from iodine deficiency during reproduction. Most of the 1988 conference proceedings were therefore concerned with the effects on the fetus and the child of iodine deficiency of the mother. The high susceptibility around conception is discussed later.

Children as well as non-pregnant women are much less susceptible than the unborn child to iodine deficiency. Comparative trials of iodine supplementation of children have not corrected the lower intelligence (IQ) of children from iodine-deficient populations [7, pp. 269–87].
TABLE 1. Goitre or other disorders of the thyroid gland by age and sex, USA, 1992, nationwide interview sample for National Health Interview Survey

<table>
<thead>
<tr>
<th>Cases per 1000 persons</th>
<th>Under 45 years</th>
<th>45–64 years</th>
<th>65–74 years</th>
<th>75 years and over</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>3.3</td>
<td>11.4</td>
<td>18.2</td>
<td>9.1</td>
</tr>
<tr>
<td>Female</td>
<td>13.4</td>
<td>53.7</td>
<td>59.0</td>
<td>51.7</td>
</tr>
</tbody>
</table>

Source: [10, Table 58].

Few clinical studies have been found on the effects of paternal iodine deficiency on children. A few studies on experimental and farm animals have recently been reviewed [9]. In male animals, hypothyroidism is reported to reduce libido and sperm number, motility and density, delay puberty onset and reduce the final testis size, final conception rate and fertility.

PREGNANCY CAN DAMAGE THE THYROID GLAND

Women report more thyroid disorders than men. Data from the US National Health Interview Survey 1992 are shown in Table 1 [10]. Statistics understate the prevalence of thyroid disorders, particularly cases of a less serious character which are often not diagnosed or reported. Nevertheless, Table 1 shows that women in the US at the end of their reproductive years are recorded as having four times as much chronic thyroid disease as men.

In an area of Germany with a population suffering from mild iodine deficiency, using ultrasound, Struve and Ohlen [11] of the University of Kiel showed that women who had had children were at greater risk of goitre and the development of thyroid nodules than women who had never been pregnant. This study covered 106 women aged 36–50 years of whom one-half had had children. Goitre was defined as a thyroid volume exceeding 18 ml.

The influence of pregnancy stress on the thyroid has been studied by teams at the University of Brussels and University of Louvain. Glinier et al. [12] concluded that the higher prevalence of thyroid disorders in women compared with men can be attributed to the glandular stress of pregnancy. This team has also shown that the stimulation of the thyroid by pregnancy is only harmful and damaging to the thyroid in the presence of iodine deficiency [13]. Damage to a mother’s thyroid and goitrogenesis were found to be completely prevented by iodine supplementation. Glinier et al. [13] concluded that:

The results indicate the benefits of supplementing pregnant women with iodine and women with excessive thyroid stimulation with a combination of iodine and L-T4. In conditions of mild iodine deficiency, pregnancy fully justifies the monitoring of thyroid function and volume and therapeutic intervention to avoid hypothyroxinemia and goitrogenesis (p. 268).

A paper by Glinier was discussed at the 1993 symposium on iodine deficiency in Europe and in reply to the discussion Glinier stated that: “pregnancy acts as a revealing factor to uncover underlying marginal iodine deficiency because of the increased needs for thyroid hormones …” (p. 189).

When the iodine supply is adequate the maternal thyroid adjusts to pregnancy and lactation with only minor alterations in thyroid function and volume. The thyroid enlargement and dysfunction associated with iodine deficiency in pregnancy and lactation is generally reversible. However, in a minority of cases of thyroid dysfunction following
pregnancy, the thyroid has been shown not to return to normal even several years post-partum. A study from the University of Wales found that 23% of patients with post-partum thyroiditis were hypothyroid 3.5 years post-partum [14]. A study from Sweden reported 31% long-term hypothyroidism following post-partum thyroiditis [15].

Thyroid dysfunction has been reported in women who have had a miscarriage [16]. Iodine deficiency can, in fact, cause miscarriage. In women who are even mildly iodine deficient the thyroid dysfunction may be aggravated by each successive pregnancy.

Permanent thyroid dysfunction can generally be treated successfully, but the milder disorders in particular are often not treated. Depressed levels of thyroid hormones reduce the body’s capacity for protein synthesis and tissue renewal and accelerate ageing [17]. Thyroid dysfunction should be diagnosed and treated.

Enlargement of the thyroid gland or goitre is perhaps the easiest sign of iodine deficiency to record, in particular with modern ultrasonic equipment. Ultrasonic techniques have been developed which enable the volume of the thyroid gland to be measured with reasonable accuracy [18, 19]. A sonographer requires approximately 2 min for one examination and can examine approximately 200 individuals per day [2, p. 113]. Portable apparatus weighing less than 2 kg is available. Figure 1 shows the size distribution of thyroid glands measured by ultrasound in 1397 German women and 303 Swedish women [20].

DANGERS OF IODINE FORTIFICATION FOR SUSCEPTIBLE INDIVIDUALS

The UK chapter in the symposium [2] says nothing at all under the heading ‘Conclusions’ about the effect of iodine intake on pregnancy outcome but says that: “There are no plans for any national regulation of iodine supplementation. Attention has been drawn to the dangers of iodine supplementation in susceptible individuals” (pp. 326–7).

Iodine supplementation can aggravate thyrotoxicosis in individuals with defective thyroid glands. The tenth edition of the American report Recommended Dietary Allowances suggests that iodine intake only causes thyrotoxicosis in individuals who have been exposed
TABLE 2. Mean daily iodine intake (mcg), Great Britain, 1990

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>16–64</td>
<td>16–24</td>
<td>25–34</td>
<td>35–49</td>
<td>50–64</td>
<td>16–64</td>
<td></td>
</tr>
<tr>
<td>years</td>
<td>years</td>
<td>years</td>
<td>years</td>
<td>years</td>
<td>years</td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>226</td>
<td>146</td>
<td>158</td>
<td>172</td>
<td>171</td>
<td>163</td>
</tr>
<tr>
<td>Lower 2.5 percentile</td>
<td>99</td>
<td>61</td>
<td>53</td>
<td>75</td>
<td>67</td>
<td>63</td>
</tr>
</tbody>
</table>

Source: [23].

to years of iodine deficiency and who have, in some measure, adapted to a low iodine intake [21]. Evidence in support of this view is given in the symposium. The iodine intake in Japan is much higher than in Europe because of the customary consumption of more seafoods, particularly seaweeds. A report in the symposium on iodine nutrition in Japan [2, p. 141] showed the mean intake to be around 1000 mcg/day or approximately six times the mean British intake, with some individuals consuming 10 times more. A survey of 60,000 individuals in Shinsyu, with a mean iodine intake of approximately 1000 mcg/day, found 0.2% hypothyroidism and 0.08% hyperthyroidism using serum hormone measurements. These Japanese figures for thyroid dysfunction in an area with a high iodine intake are much lower than those recorded in Europe or indeed in the US, as seen in Table 1.

Hetzel [2], the executive director of the International Council for the Control of Iodine Deficiency Disorders (ICCIDD), said in a discussion at the European meeting in Brussels in 1992 that:

The age group susceptible to iodine induced hyperthyroidism results from long-standing iodine deficiency. Once you have corrected the iodine deficiency, this cohort progressively disappears. We should include the problem of iodine-induced hyperthyroidism in the group of disorders connected to iodine deficiency. In this respect the Japanese data, which were presented today, are important from the point of view of public health (p. 148)

A statement in 1994 by the WHO [22, pp. 6–7] entitled *Iodine and Health* concluded that:

Issues relating to the safety of universal salt iodization have been carefully examined by WHO ... The benefits to be derived from universal salt iodization ... and the absence of significant adverse effects among others in the same areas who are not iodine deficient, far outweigh any risk of excess intake for a small minority.

IODINE DEFICIENCY IN THE UK

The WHO [4] estimated the number of people probably ‘at risk’ of iodine deficiency in Europe at many millions. The WHO [4] report lists the world’s nations and their own estimates of the size of populations ‘affected’ and ‘at risk’ from iodine deficiency diseases. Thus, for example, the report shows the populations affected as being 5% in Belgium, 5% in France, 10% in Germany and 2.5% in The Netherlands. The UK reported to WHO that: “Population affected 0, population at risk 0 … No national data … it is generally considered that the iodine status of the general population is adequate” (p. 39). The daily intake of women as recorded by the Dietary and Nutritional Survey of British Adults in 1990 [23] is shown in Table 2 and Fig. 2 [24].

How do these recorded intakes compare with the recommendations? As can be seen from Table 2 and Fig. 2, a large percentage of women in the UK have iodine intakes well below the allowances recommended by the Department of Health [25]. Further, as evidenced in
Table 4 and Fig. 2, the iodine intake of many women in the UK is even further below the recommendations of the WHO [3]. The main difference between Tables 3 and 4 is in the recommendations for pregnancy and lactation. The UK Dietary Reference Values report says: ‘Pregnancy—no increment’ and ‘Lactation—no increment’. The WHO recommends an extra 50 mcg/day of iodine during both pregnancy and lactation. The WHO had previously recommended only an extra 25 mcg/day for pregnancy and lactation in line with American recommendations, but the symposium on iodine deficiency in Europe in 1993 recommended an increase in the supplements for pregnancy and lactation to 50 mcg/day [2].

Table 3. Dietary reference values (RNIs) for iodine, UK, 1991

<table>
<thead>
<tr>
<th>Age</th>
<th>RNI (mcg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–3 months (formula fed)</td>
<td>50</td>
</tr>
<tr>
<td>4–12 months</td>
<td>60</td>
</tr>
<tr>
<td>1–3 years</td>
<td>70</td>
</tr>
<tr>
<td>4–6 years</td>
<td>100</td>
</tr>
<tr>
<td>7–10 years</td>
<td>110</td>
</tr>
<tr>
<td>11–14 years</td>
<td>130</td>
</tr>
<tr>
<td>15–18 years</td>
<td>140</td>
</tr>
<tr>
<td>Over 18 years</td>
<td>140</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>No increment</td>
</tr>
<tr>
<td>Lactation</td>
<td>No increment</td>
</tr>
</tbody>
</table>

Source: [25].
WHO RECOMMENDATIONS TO ELIMINATE IODINE DEFICIENCY DISEASES

The elimination of iodine deficiency was included in the declaration signed by 190 heads of state following the World Summit for Children in September 1990. The symposium of 1993 [2] concluded with recommendations which fell under two headings: those aimed at governments and intended to improve the iodine intake of whole populations, and those aimed at individuals and health services. The greatest emphasis was placed on government action to improve the whole population’s iodine intake:

The governments and the European Community are called upon to initiate adequate legislation, and other necessary measures to ensure the availability and use of iodized salt, which will generally be the most appropriate measure for iodine supplementation [2, p. 477].

There are then recommendations for the quality control and monitoring of iodine in foods. The UK chapter [2, p. 326] explains that iodized salt accounts for only 2.5% of salt consumption in the UK and that there is no legal requirement for fortification.

Several nations have already gone far in eliminating the problem. Between 1923 and 1952 the 26 cantons of Switzerland introduced iodized salt one by one and then introduced national control by a commission reporting to cantonal health ministers [2, p. 368]. Canada has a federal law requiring the addition of iodine to table salt [2, p. 131].

The Netherlands has legislation specifying the minimum concentration of iodine in household salt and legislates separately for iodine in salt used in bread making. Epidemiological surveys in the Netherlands have shown that the iodization of household salt is not completely effective, partly because of decreased salt consumption, which is recommended. A Swiss paper by Bürgi et al. [26, p. 587] stated that: “if salt intake continues to diminish the iodine content of salt will have to be raised”. The Netherlands [2] chapter also points to the need for iodine in salt because of goitrogens in the environment. Nitrate from agricultural fertilizer is goitrogenic and has been found to contaminate drinking water and vegetables [2, pp. 329–33]. Investigation has shown, however, that nitrate was only goitrogenic for women consuming fewer than 100 mcg/day of iodine. In parts of the UK, water supplies are also contaminated with agricultural fertilizer. While the Netherlands [2, p. 329] chapter in the symposium suggests that bread iodization is very effective, it is concluded that still further protection is needed by young women on slimming diets and elderly people on low-salt diets.

A range of processed foods, in addition to bread, contain added salt and most people obtain most of their salt intake from processed food. In countries such as the Netherlands

<table>
<thead>
<tr>
<th>Age range or state</th>
<th>Intake (mcg/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–12 months</td>
<td>50</td>
</tr>
<tr>
<td>1–6 years</td>
<td>90</td>
</tr>
<tr>
<td>7–12 years</td>
<td>120</td>
</tr>
<tr>
<td>12 years to (and through) adulthood</td>
<td>150</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>200</td>
</tr>
<tr>
<td>Lactation</td>
<td>200</td>
</tr>
</tbody>
</table>

'The allowances can be regarded as serving the same purpose as estimates of population minimum mean intakes sufficient to meet normative requirements. Source: [3].
and Switzerland that require salt to be iodized, processed food is consequentially iodized. If the salt in a neighbouring country is not iodized the processed food may not be iodized and its importation into countries requiring iodization may be subject to complaints. There is a strong argument for harmonization and international agreement on iodization requirements to avoid creating new obstacles to international trade. The Swiss paper already referred to says: “The main threat to the iodized salt programme comes from international trade regulations” [26, p. 587].

The general recommendations at the end of the symposium of 1993 [2, p. 477], which aimed at increasing the iodine intake of whole populations, were followed by specific recommendations which related only to mothers and infants.

The symposium explained the limitation of the specific recommendations [2]:

The thyroids of pregnant women and neonates are particularly sensitive to the effects of iodine deficiency. This is of special concern because adequate thyroid hormone is essential for normal brain development during early life (p. 478).

The monitoring of the iodine intake of mothers and infants in Europe by periodic analysis of urinary iodine levels, and the measurement of thyroid stimulating hormone (TSH) and other thyroid hormones (T₄ and T₃) ‘to the extent feasible’, are recommended in the symposium. It is also recommended that the “mother’s diet should be systematically supplemented with iodine whenever necessary by vitamin/mineral tablets as prescribed by physicians” [2, p. 478].

THE NEED FOR IODINE BEFORE CONCEPTION

There are great difficulties in these recommendations because the evidence shows that, to be wholly effective, iodine deficiency has to be corrected before ovulation and conception. The importance of the prevention of very early iodine supplementation was first demonstrated by Pharoah et al. [27, p. 308] who reported in the Lancet in 1971: “It is concluded that iodised oil is effective in the prevention of endemic cretinism and that, for it to be effective, it should be given prior to conception”. The trial which took place in Papua New Guinea included 16 500 people of whom half were injected with iodized oil. The children of the families who participated were followed for 15 years. A summary in 1988 repeated that iodine supplementation after conception appeared to be ineffective. However, iodine supplementation before conception significantly reduced the mortality of the children over the 15 years and improved both their motor and cognitive functions as shown by the tests reported in Pharoah and Connolly’s papers [28].

Much research has been done on the contribution of thyroid hormones, which require iodine, to the growth and development of the ovarian follicle and the ovum. A gene passes through stages during conversion to a structural or body protein, and thyroid hormones are needed at the first transcription stage; they are called transcription enhancers [29–31]. High rates of gene transcription are, of course, essential during maturation of the ovum and sperm before conception and this requires an adequate concentration of thyroid hormones around the germ cells.

A slow-down in the rates of cell replication and growth is a first consequence of a reduction in the concentration and local availability of thyroid hormones, notably T₄. This reduction in growth rate is mediated to different degrees by hormones particularly concerned with growth. The family of growth hormones called insulin-like growth factors (IGFs) are found in all parts of the body partly regulated by thyroid hormones [32–35]. The rate of growth of the ovarian follicle and subsequent size of the corpus luteum are partly controlled by IGFs and thyroid hormones. A review by Giudice [36] on IGFs and ovarian follicular development includes 320 references.

Animal experiments have shown that if iodine deficiency begins during the period
Table 5. Women’s food sources of iodine, Great Britain, 1994

<table>
<thead>
<tr>
<th>Food</th>
<th>Amount (mcg/day)</th>
<th>Daily intake (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk and milk products</td>
<td>72</td>
<td>39</td>
</tr>
<tr>
<td>Cereal products</td>
<td>26</td>
<td>14</td>
</tr>
<tr>
<td>Total beverages</td>
<td>18</td>
<td>10</td>
</tr>
<tr>
<td>Fish</td>
<td>15</td>
<td>8</td>
</tr>
<tr>
<td>Confectionery, sugar and preserves</td>
<td>12</td>
<td>6</td>
</tr>
<tr>
<td>Meat and meat products</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td>Egg and egg products</td>
<td>9</td>
<td>5</td>
</tr>
<tr>
<td>Potatoes</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Fruit</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Vegetables</td>
<td>3</td>
<td>2</td>
</tr>
</tbody>
</table>

Source: [24].

preceding mating, it causes a much more serious range of congenital malformations than if it begins only a few days after mating [37]. Low T₄ levels, which may be a consequence of iodine deficiency, can cause mutations in males and females that may be inherited in their F₁ offspring and in the following F₂ generation [38, 39].

HOW CAN IODINE INTAKE BE INCREASED?

The WHO recommends that the iodization of salt should be introduced by legislation in all countries which have not already done so, as already discussed. There have now been three generations of experience of the iodization of salt, which has been found not to be completely effective. The soundness of the WHO recommendation is not questioned, but implementation leaves some people still iodine deficient.

The fortification of salt is likely to prove increasingly inadequate as the populations of Britain and other countries respond to the advice that they are eating too much salt for the good of their health. The men’s median sodium intake in the UK is 3.320 g/day and the women’s intake is 2.313 g/day, both compared with a recommendation of 1.6 g/day [23]. It is hoped that less salt will be added to processed food as the years go by and it is undesirable to encourage the addition of more iodized salt to food to promote iodine consumption. This is one reason for studying other ways of increasing iodine intake.

The contribution of the main classes of food to women’s iodine intake is shown in Table 5 [24]. The importance of milk and milk products is apparent. The daily consumption of milk varies widely in practice from one woman to the next and the adult survey shows standard deviations for milk and milk products that are 70 and 80% of the mean [24]. It is indeed possible to have a diet with an ample intake of meat, cereals, vegetables and fruit that is iodine deficient. An adequate intake of iodine requires an adequate intake of milk and milk products or of seafood.

It is seen in Table 6 that fish has a much higher iodine content even than dairy produce [40]. Fish can, indeed, readily provide all individual iodine requirements. Fish has, however, become relatively more expensive in recent years and, as can be seen from Table 5, today fish makes only a limited contribution to women’s iodine intake. Total fish production is limited and stocks are declining and may continue to do so.

Supplies of iodine are not, however, limited and are inexpensive. In 1994 the WHO [22, p. 2] said that: “Sea fish, other sea food, and seaweed are rich sources of iodine suitable for human consumption”. Because of their high iodine content and edibility, seaweeds are much the cheapest source of iodine. Seaweed is cultivated in Japan as a food. Such high intakes of iodine would aggravate the illness of a minority of men and women with existing
thryoid disease. It does not seem wise to advocate a more general use of dried seaweeds in UK domestic households.

There are many ways to overcome the difficulties suggested in papers around the world. The suggestions and recommendations fall under the two headings of fortification and supplementation. The iodization of salt is an example of fortification and iodine pills for pregnant women offer an example of supplementation.

Fortification through the control of farm animal feeding is currently receiving major attention. The WHO [22, p. 2] refers to the importance of iodine: “In milk products, eggs, poultry and meat from animals whose diet contained sufficient iodine”. A paper from the German Institute for Animal Nutrition says [41, p. 11]:

> We propose a new strategy to fight iodine deficiency disease. This includes the enriching with iodine along the food chain either via food of animal origin or vegetable origin. Iodine may be supplemented either by addition of iodate or, even more promising, by addition of iodine rich algae (seaweed).

Preliminary feeding experiments done by us have shown that the iodine content in pork, chicken meat and milk rises proportionally to the amount of iodine in the feed of pigs, chicken and cows.

Kaufmann and Rambeck [42] emphasized that seaweeds contain a range of other valuable nutrients. There are over 800 recorded species of seaweeds around the British Isles, mostly edible and only a few poisonous. Seafoods are carriers of a range of B vitamins, essential minerals and polyunsaturated fatty acids. Farm animals also need iodine in their diet [43]. The addition of more iodine to animal feedstock may be one right way of fortifying human foods, but there are advocates of the direct fortification of human foods, for example of bread, as practised in the Netherlands. Most processed food in the UK has a low iodine content. Controlled increases in iodine intake in food manufacture would cost very little.

There is a history making possible a comparison of the merits of supplementation of individual diets and the general fortification of foodstuffs, not only with iodine but with other nutrients such as iron, vitamin A and folic acid. Achieving and sustaining a high coverage with supplementation programmes has proved difficult, requiring a substantial effort by health services. Minorities have benefited greatly but there has been widespread failure to reach the minorities most in need. This is the current experience with the folic acid supplementation of women in anticipation of pregnancy in the UK. Fortification, in contrast, has been shown to be both sustainable and cost-effective.

The World Bank, in its 1993 World Development Report, stated that the fortification of foods was one of the most cost-effective public health interventions. A decision to introduce iodine fortification in the UK needs to be preceded by a study of the distribution of iodine.

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**TABLE 6. Iodine content of some common foods**

<table>
<thead>
<tr>
<th>Food</th>
<th>mcg 100 g⁻¹</th>
<th>mcg MJ⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fish (cod)</td>
<td>110</td>
<td>342</td>
</tr>
<tr>
<td>Eggs</td>
<td>53</td>
<td>87</td>
</tr>
<tr>
<td>Whole milk</td>
<td>15</td>
<td>55</td>
</tr>
<tr>
<td>Bananas</td>
<td>8</td>
<td>20</td>
</tr>
<tr>
<td>Meat (beef)</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Bread (white)</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Potatoes (old)</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Cabbage</td>
<td>2</td>
<td>18</td>
</tr>
<tr>
<td>Pulses (peas)</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Fruit (pears)</td>
<td>1</td>
<td>6</td>
</tr>
</tbody>
</table>

Source: [40].
deficiency. If it is decided to enrich manufactured foods, a limited number of manufacturers who can provide good population coverage have to be chosen and procedures have to be agreed. Every programme must then be monitored. There are historical examples of the recrudescence of iodine deficiency following the relaxation of monitoring [44]. A programme to increase the intake of iodine in the UK should be under statutory control.

REFERENCES

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