The Effects of Iodine Deficiency in Pregnancy and Infancy

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Abstract

Iodine requirements are increased ≥50% during pregnancy. Iodine deficiency during pregnancy can cause maternal and fetal hypothyroidism and impair neurological development of the fetus. The consequences depend upon the timing and severity of the hypothyroidism; the most severe manifestation is cretinism. In moderate-to-severely iodine-deficient areas, controlled studies have demonstrated that iodine supplementation before or during early pregnancy eliminates new cases of cretinism, increases birthweight, reduces rates of perinatal and infant mortality and generally increases developmental scores in young children by 10–20%. Mild maternal iodine deficiency can cause thyroid dysfunction but whether it impairs cognitive and/or neurologic function in the offspring remains uncertain. Two meta-analyses have estimated that iodine-deficient populations experience a mean reduction in IQ of 12–13.5 points. In nearly all regions affected by iodine deficiency, salt iodisation is the most cost-effective way of delivering iodine and improving maternal and infant health.

Keywords: iodine, deficiency, pregnancy, infancy, thyroid, cognition, intelligence, infant mortality.

Iodine (atomic weight 126.9) is an essential component of the hormones produced by the thyroid gland. Thyroid hormones, and therefore iodine, are essential for mammalian life. In the early 1920s, Switzerland was the first country to fortify household salt with iodine to control endemic goiter and cretinism. In the 1970s and 1980s, controlled studies showed that iodine supplementation before and during pregnancy not only eliminated new cases of cretinism but also improved cognitive function in the rest of the population. From 1990 to 2007, global population coverage with iodised salt increased from about 20% to 70%. But the International Council for the Control of Iodine Deficiency Disorders (ICCIDD) estimates that nearly two billion individuals in 2011 continue to have insufficient iodine intake worldwide, including 1/3 of all school-age children, and iodine deficiency remains a public health problem in 32 countries. There are insufficient data from nearly all countries to estimate the prevalence of iodine deficiency in pregnant women.

In healthy adults, the absorption of iodide is >90%. The body of a healthy adult contains 15–20 mg of iodine, of which 70–80% is in the thyroid. In chronic iodine deficiency, the iodine content of the thyroid may fall to <20 mg. Iodine is cleared from the circulation mainly by the thyroid and kidney, and while renal iodine clearance is fairly constant, thyroid clearance varies with iodine intake. In conditions of adequate iodine supply, ≤20% of absorbed iodine is taken up by the thyroid. In chronic iodine deficiency, this fraction can exceed 80%. Despite high fractional clearance of iodine by the thyroid, in chronic severe iodine deficiency, thyroid hormone synthesis falls, leading to hypothyroidism and its sequelae.

The iodine requirement during pregnancy is sharply increased because of: (1) an increase in maternal thyroxine (T₄) production to maintain maternal euthyroidism and transfer thyroid hormone to the fetus early in the first trimester, before the fetal thyroid is functioning; (2) iodine transfer to the fetus, particularly in later gestation; and (3) an increase in renal iodine clearance. Iodine requirements from the US Institute of Medicine (IOM) and WHO for pregnancy, lactation and infancy are shown in Table 1. The recommended method to assess iodine nutrition in pregnant women is the urinary iodine concentration (UIC). Because >90% of dietary iodine eventually appears in the urine, UIC is an excellent indicator of recent iodine intake. UIC is usually measured in spot
urine specimens from a representative sample of women, and expressed as the median, in \( \mu \text{g/L} \). Recommendations for using the median UIC to classify iodine status of pregnant and lactating women, and infants, are shown in Table 2.

In nearly all regions affected by iodine deficiency, salt iodisation is the most cost-effective way of delivering iodine and of improving cognition. Worldwide, the annual costs of salt iodisation are estimated at US$0.02–0.05 per child covered, and the costs per child death averted are US$1000 and per Disability-adjusted life year (DALY) gained are US$34–36. However, in some regions, iodisation of salt may not be practical for control of iodine deficiency, at least in the short term. In these areas, iodised oil supplementation can be used. Iodised oil can be given orally or by intramuscular injection. Usual doses are 200–400 \( \mu \text{g iodine/year} \) and it is often targeted to women of childbearing age and pregnant women. Iodine can also be given as KI or KIO3 as drops or tablets. In order to ensure adequate iodine supply during pregnancy, women should ideally be provided with ample iodine intake (at least 250 \( \mu \text{g/day} \)) for a long period before conception to ensure plentiful intrathyroidal iodide stores.

### Methods

A systematic literature search in PubMed, Current Contents Connect® and ISI Web of Science® for articles in English, French, German, Spanish (search terms included: iodine, urinary iodine, iodine deficiency, iodine status, pregnancy, infancy, children, cretinism, cognition, school performance, mental development, intelligence, growth, perinatal mortality, infant mortality, maternal mortality, preterm birth, prematurity, abortion, stillbirth, miscarriage, birthweight, meta-analysis) was conducted. Study reports were also obtained from book chapters and through correspondence with iodine experts around the world. Approximately 3300 abstracts were reviewed. Of these, approximately 450 full-length papers were reviewed. Of these, 27 original studies and two meta-analyses were included in this review. It was decided not to attempt to perform a meta-analysis of the pregnancy studies because: (i) the number of randomised controlled intervention trials of iodine in pregnancy and/or infancy is small; (ii) in many, blinding is uncertain, drop-outs were high and follow-up was inconsistent; and (iii) the studies vary widely in their designs (oral vs. intramuscular delivery of high doses of iodised oil; daily low dose potassium iodide; iodisation of irrigation water; timing of intervention).

### Results

#### Cretinism, cognition and thyroid function

Iodine deficiency has multiple adverse effects on growth and development in animals and humans. These are collectively termed the iodine deficiency

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**Table 1.** Recommendations for iodine intake (\( \mu \text{g/day} \)) for pregnant and lactating women, and infants

<table>
<thead>
<tr>
<th>Age or population group</th>
<th>US Institute of Medicine</th>
<th>Age or population group</th>
<th>World Health Organization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infants 0–12 months</td>
<td>110–130</td>
<td>Children 0–5 years</td>
<td>90</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>220</td>
<td>Pregnancy</td>
<td>250</td>
</tr>
<tr>
<td>Lactation</td>
<td>290</td>
<td>Lactation</td>
<td>250</td>
</tr>
</tbody>
</table>

*Recommended daily allowance.
*Adequate intake.
*Recommended nutrient intake.

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**Table 2.** Epidemiological criteria from the World Health Organization for assessment of iodine nutrition in pregnant and lactating women, and infants based on median or range of urinary iodine concentrations

<table>
<thead>
<tr>
<th>Iodine intake</th>
<th>Pregnant women</th>
<th>Lactating women</th>
<th>Children less than 2 years of age</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;150 ( \mu \text{g/L} )</td>
<td>Insufficient</td>
<td>Insufficient</td>
<td>Insufficient</td>
</tr>
<tr>
<td>150–249 ( \mu \text{g/L} )</td>
<td>Adequate</td>
<td>Adequate</td>
<td>Adequate</td>
</tr>
<tr>
<td>250–499 ( \mu \text{g/L} )</td>
<td>More than adequate</td>
<td>More than adequate</td>
<td>More than adequate</td>
</tr>
<tr>
<td>( \geq 500 \mu \text{g/L} )</td>
<td>Excessive</td>
<td>Excessive</td>
<td>Excessive</td>
</tr>
<tr>
<td>( \geq 100 \mu \text{g/L} )</td>
<td>Adequate</td>
<td>Adequate</td>
<td>Adequate</td>
</tr>
<tr>
<td>( \geq 100 \mu \text{g/L} )</td>
<td>Adequate</td>
<td>Adequate</td>
<td>Adequate</td>
</tr>
</tbody>
</table>

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disorders and are one of the most important and common human diseases. They result from inadequate thyroid hormone production due to lack of sufficient iodine. Iodine deficiency during pregnancy impairs the neurological development of the fetus. In areas of severe chronic iodine deficiency, maternal and fetal hypothyroxinemia can occur from early gestation onwards. Thyroid hormone is required for normal neuronal migration and myelination of the brain during fetal and early postnatal life, and hypothyroxinemia during these critical periods causes irreversible brain damage, with mental retardation and neurological abnormalities. The consequences depend upon the timing and severity of the hypothyroxinemia (Table 3). The most severe manifestation of in utero iodine deficiency is cretinism. Two classic forms of cretinism – neurological and myxedematous – have been described. The three characteristic features of neurological cretinism in its fully developed form are severe mental retardation with squint, deaf mutism and motor spasticity. The typical myxedematous cretin has a less severe degree of mental retardation than the neurological cretin. But myxedematous cretins have all the features of severe hypothyroidism present since early life, including severe growth retardation, incomplete maturation of the facial skeleton, puffy features, thickened and dry skin, dry and rare hair, and delayed sexual maturation. Whether mild-to-moderate maternal iodine deficiency produces more subtle changes in cognitive and/or neurologic function in the offspring remains uncertain. But two prospective case–control studies in iodine-sufficient women have reported even mild thyroid dysfunction during pregnancy may impair cognitive development of the offspring.

In a landmark trial in an area of severe iodine deficiency in Papua New Guinea, alternate families received saline (control) or iodised oil injection. The primary outcome was the prevalence of cretinism at 4 and 10 years follow-up, with more sensitive diagnostic tests applied at the 10-year follow-up. Iodine supplementation was associated with a significant reduction in the prevalence of endemic cretinism: at 4 years of age, the relative risk [95% CI] was 0.27 [0.12, 0.60] and at 10 years of age, the relative risk [95% CI] was 0.17 [0.05, 0.58]. A long-term follow-up was done on a small sample of non-cretinous children at 11 and 15 years of age, but found no significant differences in motor and cognitive function between the children born to supplemented families and controls.

In a study in Zaire, participants were pregnant women attending antenatal clinics in an area of severe iodine deficiency with a 4% cretinism rate. Pregnant women were randomly allocated to two groups: one received iodised oil injection, the other an injection of vitamins. Women were on average 28 weeks pregnant when they were treated. Psychomotor development scores were measured in the offspring at ~72 months of age, but there was a loss to follow-up of ~50% in both groups. The psychomotor development scores were significantly higher in the iodine group (mean psychomotor development score, 91 ± 13 vs. 82 ± 14 in the controls); the prevalences of psychomotor scores ≤60 were 0.5% in the iodine group vs. 9.7% in the controls.

In a study in western China, an area of severe iodine deficiency and endemic cretinism, participants were groups of children from birth to 3 years and women at each trimester of pregnancy. Untreated children 1–3 years of age, studied when first seen, served as controls. The intervention was oral iodised oil and treated children and the babies born to the treated women were followed for two years. The main outcomes were neurologic examination, head circumference, and indexes of cognitive and motor development. A small subsample was followed out to ~7 years of age. The prevalence of moderate or severe neurologic abnormalities among the infants whose mothers received iodine in the first or second trimester was 2%, as compared with 9% among the infants who received iodine in the first or second trimester was 2%, as compared with 9% among the infants who received iodine in the third trimester (through the treatment of their mothers) or after birth. Treatment in the third trimester of pregnancy or after delivery did not improve neurologic status, but head growth and developmental quotients improved slightly. Treatment at the end of the first trimester did improve neurologic outcome. The prevalence of microcephaly was 27% in the untreated children compared with 11% in the treated children. The mean (±standard deviation) developmental quotient at 2 years of age was higher in the treated than in the untreated children (90 ± 14 vs. 75 ± 18). In the long-term follow-up study, development of children (range 4–7.3 years) whose mothers received iodine during pregnancy, and children who received iodine first in their second year, was examined. A second group of children (range 5.8–6.9 years) whose mothers received iodine while pregnant were examined 2 years later. Head circumference was improved for those who received iodine during pregnancy (compared with

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<table>
<thead>
<tr>
<th>Quality assessment</th>
<th>Summary of findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of studies and study design</td>
<td>Heterogeneity of results?</td>
</tr>
<tr>
<td>Cretinism: overall quality of evidence grade = high</td>
<td></td>
</tr>
<tr>
<td>5 RCTs</td>
<td>No, all studies showed a positive effect of intervention on reduction in incidence of cretinism.</td>
</tr>
<tr>
<td>Cognitive development: overall quality of evidence grade = high</td>
<td></td>
</tr>
<tr>
<td>4 RCTs</td>
<td>Yes, all studies showed a positive effect of an increase in development or intelligence quotient.</td>
</tr>
<tr>
<td>Birthweight (g): overall quality of evidence grade = low</td>
<td></td>
</tr>
<tr>
<td>2 RCT</td>
<td>Difficult to assess; poor description of findings.</td>
</tr>
<tr>
<td>Infant mortality rate: overall quality of evidence grade = moderate</td>
<td></td>
</tr>
<tr>
<td>2 RCTs</td>
<td>Yes, both studies showed a reduction.</td>
</tr>
</tbody>
</table>
those receiving iodine at age 2) and for those supplemented before the end of the second trimester (relative to those supplemented during the third trimester). Iodine before the third trimester predicted higher psychomotor test scores for children relative to those provided iodine later in pregnancy (~5.4%) or at 2 years (~16%).

In a randomised Peruvian trial, women of child-bearing age from Andean villages in an area of severe iodine deficiency with a 1–3% cretinism rate were studied. A subsequent reanalysis reassigned children to two groups, iodine-deficient or iodine-sufficient at time of cognitive testing, based on their UIC and T4. This analysis found a significant higher IQ score in the iodine-sufficient group compared with the iodine-deficient group (85.6 ± 13.9 vs. 74.4 ± 4.8).

In two villages in Ecuador with severe intellectual disability and a cretinism rate of up to 8%, one village received iodine treatment, and one did not and served as an iodine-deficient control. Participants were all women of child-bearing age, pregnant women and children, and coverage with iodine was estimated to be about 90%. The treatment village received an injection of iodised oil and was then followed at 4-year intervals for ~20 years, and the effects on the offspring were recorded. No more cretins were born in the treatment village than in the control village. Two years after treatment had begun, the mean developmental quotient in infancy was not significantly different between villages. However, mean IQ measured in first and second grade children was higher by ~10 points in the treatment village than in the control village. Five years after treatment began, the treated group was divided into three groups: (i) children born after treatment had begun; (ii) children whose mothers had received iodine during pregnancy; and (iii) children whose mothers had received iodine prior to conception. The later group had significantly higher IQ than the first two groups (76.8 vs. 72.3 vs. 65.2, respectively). Studies done several years later in these children also suggested that iodine treatment late in pregnancy or afterward had no benefits of children’s IQ at 3–5 years of age, but treatment early in pregnancy or prior to conception improved IQ (83.7 ± 13.4 vs. 72.7 ± 14.0 in treated vs. control villages).

Endemic cretinism is the extreme expression of the abnormalities in physical and intellectual development caused by iodine deficiency, but the cognitive deficits associated with iodine deficiency may not be limited to remote, severely iodine-deficient areas. Many authors have argued that even mild-to-moderate iodine deficiency in pregnancy, still present in many industrialized countries, may affect cognitive function of the offspring. The controlled trials of iodine treatment in mild-to-moderately iodine-deficient pregnant women discussed below did not report data on infant or child development. However, several reported measures that might be surrogate markers of future infant development, including maternal and newborn thyroid function.

Romano et al. gave 120–180 μg iodine as iodised salt or control daily beginning in the first trimester to healthy pregnant Italian women (n = 35; median UIC 31–37 μg/L). In the treated group, median UIC increased threefold and thyroid volume did not change. In the controls, there was no change in UIC, but a 16% increase in thyroid volume. Treatment had no effect on maternal thyroid-stimulating hormone (TSH). Pedersen et al. randomised pregnant Danish women (n = 54) to receive either 200 μg iodine/day as KI solution or no supplement from 17 weeks to term. In the treated group, median UIC increased from 55 μg/L to 90–110 μg/L in treated group. Maternal thyroid volume increased 16% in the treated group vs. 30% in controls. Maternal thyroglobulin (Tg) and TSH, and cord Tg were significantly lower in the treated group. No significant differences were found between groups comparing maternal or cord T₄, triiodothyronine (T₃) and free (F)T₄. In a double-blind, placebo-controlled trial, Glinnoor et al. supplemented pregnant Belgian women (n = 120; median UIC 36 μg/L; biochemical criteria of excess thyroid stimulation) with 100 μg iodine/day or control from ~14 weeks to term. Treatment had no significant effect on maternal or cord T₃, FT₄ and T₃/T₄ ratio. The treated women had significantly higher UIC, smaller thyroid volumes, and lower TSH and Tg concentrations, compared with controls. Newborns of the treated group also had significantly higher UIC, smaller thyroid volumes and lower Tg concentrations compared with controls.

Liesenkötter et al. reported results from a quasi-random, controlled trial of 230 μg iodine/day from 11 weeks to term in pregnant German women (n = 108;
median UIC 53 μg/g creatinine; goiter rate 42.5%). Median UIC increased to 104 μg/g creatinine in treated group, and median thyroid volume was significantly lower in the newborns of the treated women compared with controls (0.7 vs. 1.5 mL, respectively). Treatment had no significant effect on maternal TSH, T<sub>3</sub>, T<sub>4</sub>, thyroid volume or Tg, and had no effect on newborn TSH. In a placebo-controlled, double-blind trial, Nohr et al. gave a multinutrient supplement containing 150 μg iodine/day or control to pregnant Danish women positive for anti-thyroid peroxidase antibodies (TPO-Ab) (n = 66) from 11 weeks to term. Median UIC was significantly higher in the treated women at term, but there were no differences in maternal TSH, FT<sub>4</sub> or Tg between groups. Finally, in a prospective, randomised, open-label trial, Antonangeli et al. supplemented pregnant Italian women (n = 67; median UIC 74 μg/g creatinine) with 50 μg or 200 μg iodine/day from 18–26 weeks to 29–33 weeks of gestation. Median UIC was significantly higher in the 200 μg group than in the 50 μg group (230 vs. 128 μg/g creatinine). However, there were no differences in maternal FT<sub>3</sub>, FT<sub>4</sub>, TSH, Tg or thyroid volume between groups.

**Maternal goiter**

In areas of mild-to-moderate iodine deficiency, pregnancy has often been suggested as an environmental factor contributing to a higher prevalence of goiter and thyroid disorders in women, compared with men. But the data to support this are scarce. In European studies, an uncontrolled prospective study in 10 women, a retrospective study and a cross-sectional study in smoking women suggest goiters formed during pregnancy may only partially regress after parturition.

**Birthweight and infant growth**

In a severely iodine-deficient area of western China, iodine repletion of pregnant women (n = 295) improved head circumference and reduced the prevalence of microcephaly from 27% to 11% (P = 0.006). In Zaire, treatment with iodised oil during pregnancy resulted in 3.7% higher birthweights compared with offspring of untreated mothers. In a region of endemic goiter area in Algeria, treatment of pregnant women (n = 1536) with oral iodised oil just before conception or during the first trimester significantly increased birthweights (6.25%) compared with non-treated women. Household use of iodised salt correlated with increased weight-for-age and mid-upper-arm circumference during infancy in a large Asian study.

**Infant mortality**

Infant survival is improved in infants born to women whose iodine deficiency is corrected before or during pregnancy. Delong et al. added potassium iodate to irrigation water over a 2- to 4-week period in three area of severe iodine deficiency in China and found a large reduction in both neonatal and infant mortality in the following 2-3 years compared with areas that did not receive iodine. The median UI increased in women of child-bearing age from <10 to 55 μg/L, while the infant mortality rate (IMR) decreased in the three treated areas from a mean of 58.2 to 28.7/1000 births, from 47.4 to 19.1/1000, and from 106.2 to 57.3/1000. Similar results were also observed for neonatal mortality; the odds of neonatal death were reduced by about 65% in the population who had iodine treatment. Iodised oil given intramuscularly to iodine-deficient pregnant women in Zaire at ≥28 weeks of gestation decreased infant mortality. In severely iodine-deficient women, the IMR in infants of treated and untreated mothers was 113/1000 and 243/1000 births, respectively, and in women with mild or moderate iodine deficiency, the IMR with and without treatment was 146/1000 and 204/1000 births.

Infant survival may also be improved by iodine supplementation in the newborn period. A randomised, placebo-controlled trial of oral iodised oil (100 mg iodine) was conducted in an area of presumed iodine deficiency in Indonesia to evaluate the effect on mortality. The iodine or placebo was given in conjunction with oral poliovirus vaccine; infants (n = 617) were treated at ≥6 weeks of age and were followed to 6 months of age. There was a significant 72% decrease in risk of infant death during the first 2 months of follow-up. In a large cross-sectional study in Indonesia, use of adequately iodised salt was associated with a significantly lower prevalence of child malnutrition and mortality in neonates, infants and children aged <5 years.

**Meta-analyses**

A meta-analysis was done of 21 observational and experimental studies including a control group of the
effect of iodine deficiency on mental development (Table 4). Of these, 16 studies were in children, 4 included adults, and 2 included infants; the age range was 2–45 years. The final meta-analysis included 2214 participants (mainly children) and IQ was used as the main outcome measure. The studies were all done in areas of moderate-to-severe iodine deficiency. The IQs of non-ID groups were on average 13.5 IQ points higher than those of the iodine-deficient groups. In the second meta-analysis by Qian et al., inclusion criteria were all studies conducted in China, comparing children (<16 years old) living in naturally iodine-sufficient areas with those: (i) in severely iodine-deficient areas; (ii) children in iodine-deficient areas born before the introduction of iodine prophylaxis; and (iii) children in iodine-deficient areas born after the introduction of iodine prophylaxis. IQ was measured using the Binet or Raven’s Scales. The effect size was an increase of 12.45, 12.3, 4.8 IQ points, respectively, for the iodine-sufficient group and the later two groups, compared with those in iodine-deficient areas. Compared with severely iodine-deficient children, there was an increase of ≈12 IQ points for children born more than 3.5 years after iodine prophylaxis was introduced.

Comments

The five early intervention trials, in severely iodine-deficient populations, were ground breaking studies done under difficult conditions in remote areas. The Papua New Guinea study had the strongest design and clearly demonstrates that iodine treatment in a population with high levels of endemic cretinism sharply reduces or eliminates incidence of the condition. The Zaire and China trials report development of non-ID groups were on average 13.5 IQ points higher than those of the iodine-deficient groups. In the second meta-analysis by Qian et al., inclusion criteria were all studies conducted in China, comparing children (<16 years old) living in naturally iodine-sufficient areas with those: (i) in severely iodine-deficient areas; (ii) children in iodine-deficient areas born before the introduction of iodine prophylaxis; and (iii) children in iodine-deficient areas born after the introduction of iodine prophylaxis. IQ was measured using the Binet or Raven’s Scales. The effect size was an increase of 12.45, 12.3, 4.8 IQ points, respectively, for the iodine-sufficient group and the later two groups, compared with those in iodine-deficient areas. Compared with severely iodine-deficient children, there was an increase of ≈12 IQ points for children born more than 3.5 years after iodine prophylaxis was introduced.

Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%. Although there are too few studies to draw firm conclusions, the available data suggest iodine prophylaxis in severely iodine-deficient populations, or iodine supplementation of severely deficient pregnant women or infants, may reduce the IMR by ≥50%.

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### Table 4. Summary of findings and overall assessment of quality of evidence from meta-analyses of observational studies comparing cognition in iodine-deficient to iodine-sufficient populations

<table>
<thead>
<tr>
<th>Quality assessment</th>
<th>Summary of findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intelligence quotient: overall quality of evidence: moderate</strong></td>
<td><strong>Generalisability to population of interest</strong></td>
</tr>
<tr>
<td>No. of studies</td>
<td>Design</td>
</tr>
<tr>
<td>21, but 3 were omitted because of heterogeneity, so final number was 18</td>
<td>Mainly cross-sectional studies, some intervention trials. IQ measured by varying methods.</td>
</tr>
<tr>
<td>37</td>
<td>Cross-sectional studies of children from mothers supplemented during pregnancy or before, compared with after. IQ measured in all studies using the Binet or Raven’s Scales.</td>
</tr>
</tbody>
</table>

IQ, intelligence quotient; SD, standard deviation.
tially irreversible adverse impact of iodine deficiency during early life on neurodevelopment, regional and national public health programs should focus on effective and sustained iodine prophylaxis of pregnant women and infants.

Conflicts of interest
The author has no conflicts of interest to declare.

References


