The relationship between iodine nutrition and thyroid disease in lactating women with different iodine intakes

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Abstract
Areas with low, adequate and excessive I content in water co-exist in China. Limited data are currently available on I nutrition and thyroid disease in lactating women and their breast-fed infants with different I intakes. This study aimed to evaluate I nutrition in both lactating women and their infants and the prevalence of thyroid disease in areas with different levels of I in water. From January to June 2014, a total of 343 healthy lactating women (excluding those taking anti-thyroid drugs or I supplements within a year of the study, consuming seafood at the time of the study or those diagnosed with congenital thyroid disease) from Beihai in Guangxi province and Jiajiazhuang, Yangcheng, Jicun and Pingyao townships in Shanxi province were selected. Compared with the I-sufficient group, median urinary I concentrations in both lactating women and infants as well as breast milk I levels were significantly lower in the I-deficient group (P<0.001). The prevalence of thyroid disease in lactating women, particularly subclinical hypothyroidism, was higher in the I-excess group than in the I-sufficient group (P<0.05). In areas with excessive water I content, high thyroid peroxidase antibody and high thyroglobulin levels were risk factors for abnormal thyroid-stimulating hormone levels. Our data collectively suggest that excessive I intake potentially causes subclinical hypothyroidism in lactating women. Moreover, enhanced monitoring of I status is important to avoid adverse effects of I deficiency or excess, particularly in susceptible populations such as pregnant or lactating women and infants.

Key words: Iodine nutrition; Iodine excess; Iodine deficiency; Lactating women; Thyroid disease

I is a trace element that is essential for the synthesis of thyroid hormones that are required for normal growth, and therefore important for the development of newborns. Adequate dietary I intake is critical for susceptible populations with high I requirements, such as pregnant and lactating women as well as infants. Several studies have shown that I deficiency during crucial periods of development in infants leads to growth retardation, impaired hearing capacity and reduced cognitive function¹⁻³. A intake is required not only for meeting the mother’s own synthesis of thyroid hormones but also for transferring the hormones to the infant through breast milk (BM) for maintaining normal thyroid function as well as growth and development, particularly of the brain. This additional loss of I is the main reason underlying I deficiency in lactating women⁴⁻⁵. I deficiency can affect thyroid function during lactation, leading to adverse side-effects in the mother, and consequently pose a critical threat to growth and brain development in the breast-fed infant¹⁻². The current daily I intake of 250 μg/d for lactating women has been recommended by WHO/ICCIDD/UNICEF to ensure that I deficiency does not occur in the postpartum period, and the I content of milk is sufficient for infants’ requirements. In both lactating women and infants <2 years of age, median urinary iodine (MUI) concentration below 100 μg/l defines a population with I deficiency. However, limited data are available on the safe urinary iodine (UI) upper limit in lactating women². Recent studies have shown that I excess in women (including lactating women) living in areas with high water I concentrations increases the risk of thyroid disease⁶⁻⁷. Other investigations have linked excessive I intake from BM to subclinical hypothyroidism in preterm Korean infants⁸. Accordingly, adequate I concentration in BM is considered particularly important for breast-fed infants to ensure optimal thyroid hormone storage and prevent neurological development impairment. Few studies to date have focused on the relationship between I intake and thyroid function in lactating women. The main objectives of

Abbreviations: BM, breast milk; FT4, free thyroxin; MUI, median urinary iodine; Tg, thyroglobulin; TgAb, thyroglobulin antibody; TPOAb, thyroid peroxidase antibody; TSH, thyroid-stimulating hormone; UI, urinary iodine.

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the present study were to clarify the status of I nutrition and the prevalence of thyroid disease among lactating women from three regions with different water I contents in China, to ascertain the relationship between I intake and the status of I nutrition as well as thyroid function in lactating women and their infants and to simultaneously provide reference data for appropriate disease control and prevention measures.

**Methods**

**Survey areas**

Villages for survey were selected based on the national surveillance data on areas with high water I contents, along with historical iodine deficiency disorders (IDD) surveillance data obtained in recent years (23). The coastal area, Tieshangang district (Xinggang, Nankang and Yingpan Towns) of Beihai city, Guangxi province, was selected as the I-deficient region with median water iodine (MWI) ≤ 10 μg/l and with low coverage rates of iodised salt (23). Luocheng village in Jiajiazhuang township and Xiaoguo village in Yangcheng township, both located in Fenyang City from Shanxi province, were selected as areas where residents have sufficient I nutrition (50 μg/l ≤ MWI ≤ 150 μg/l), representing the control group. Chengzi, Donghe, Guixianzhuang and Dongshe villages in Jicun township and Jinhua village in Pingyao township (Fenyang city of Shanxi province) were selected as areas with high I water content (MWI ≥ 300 μg/l) (10).

**Survey subjects**

From January to June 2014, a total of 343 lactating women (including 106, 104 and 133 subjects from the I-deficient, sufficient and excess regions, respectively) were recruited, excluding those taking anti-thyroid drugs or I supplements within a year of the study, consuming seafood at the time of the study and those with a family history of thyroid disease or having congenital thyroid disease. All the participants had lived in the region for ≥ 5 years. Infants whose breast-feeding occurred within a year of the study and with BM as the main source of food were selected.

**Survey indicators**

According to WHO recommendations, the following indicators were adopted to assess I status (23): MUI, goitre rate (GR), thyroid-stimulating hormone (TSH) and thyroglobulin (Tg).

**Survey methods, sample collections and measurements**

A standard questionnaire was designed to acquire demographic information including name, age, personal or family history of thyroid disease (including type of thyroid disease), intake of supplements containing I, smoking habits, economic income, source of drinking water and drinking duration. The questionnaire was administered face-to-face and conducted by well-trained staff.

A single-spot urine sample was collected in the morning in clean plastic tubes and stored at 4°C until batch-analysed for I content using the China Health Standard Method for Determination of Iodine in Urine by As3⁻⁻⁻⁻Ce4⁺⁺⁺⁺ Catalytic Spectrophotometry (24). Urine and standard samples (250 μl) were digested with 1000 μl (NH4)2S2O8 (1.0 mol/l) at 100°C for 60 min. Internal quality control samples for UI were provided by the Chinese National Reference Laboratory for Iodine Deficiency Disorders (NRLIDD).

In the pre-selected I-deficient area (Tieshangang district), each participant provided a water sample of at least 15 ml, whereas in the pre-selected I excess and sufficient villages, twenty-two water samples in total were collected from the central water supply. Water samples were stored at 4°C until

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**Table 1. Diagnostic criteria for thyroid disease**

<table>
<thead>
<tr>
<th>Thyroid disease</th>
<th>Diagnostic criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothyroxinaemia</td>
<td>FT&lt;sub&gt;4&lt;/sub&gt; &lt; 11.5 pmol/l and TSH within the normal range</td>
</tr>
<tr>
<td>Overt hypothyroidism</td>
<td>TSH &gt; 4.20 mIU/l, FT&lt;sub&gt;4&lt;/sub&gt; &lt; 11.5 pmol/l</td>
</tr>
<tr>
<td>Subclinical hypothyroidism</td>
<td>TSH &gt; 4.20 mIU/l, FT&lt;sub&gt;4&lt;/sub&gt; within the normal range</td>
</tr>
<tr>
<td>Overt hyperthyroidism</td>
<td>TSH &lt; 0.27 mIU/l, FT&lt;sub&gt;4&lt;/sub&gt; &gt; 22.7 or FT&lt;sub&gt;3&lt;/sub&gt; &gt; 6.8 pmol/l</td>
</tr>
<tr>
<td>Subclinical hyperthyroidism</td>
<td>TSH &lt; 0.27 mIU/l, FT&lt;sub&gt;3&lt;/sub&gt; and FT&lt;sub&gt;4&lt;/sub&gt; within the normal range</td>
</tr>
<tr>
<td>Autoimmune thyroiditis</td>
<td>TPOAb &gt; 60 U/ml or TgAb &gt; 60 U/ml with overt or subclinical hypothyroidism</td>
</tr>
<tr>
<td>High serum autoantibody values</td>
<td>TPOAb &gt; 60 U/ml or TgAb &gt; 60 U/ml</td>
</tr>
<tr>
<td>Goitre</td>
<td>Thyroid volume 18 ml (female)†</td>
</tr>
</tbody>
</table>

* FT<sub>4</sub>, free tri-iodothyronine; FT<sub>3</sub>, free thyroxin; Tg, thyroglobulin; TgAb, thyroglobulin antibody; TPOAb, thyroid peroxidase antibody; TSH, thyroid-stimulating hormone.

† Reference values: FT<sub>3</sub>, 11.5–22.7 pmol/l; TSH, 0.27–4.2 mIU/l; FT<sub>4</sub>, 3.1–6.8 pmol/l; TPOAb, 0–60 U/ml; TgAb, 0–60 U/ml.

† Based on the diagnostic criterion of endemic goitre (23).
Iodine nutritional status

MUI values of lactating women were 51.30, 282.42 and 822.51 μg/l for the I-deficient, sufficient and excess groups, respectively, as shown in Table 3. Compared with the I-sufficient group, MUI values were significantly lower in the I-deficient group ($Z = -11.556; \ P = 0.000$) and significantly higher in the I-excess group ($Z = -10.002; \ P = 0.000$). In parallel, MUI of breast-fed infants and median I content in BM differed significantly among the three groups.

Positive correlations among BM I and UI of lactating women as well as UI of their breast-fed infants are depicted on scatter plots in Fig. 1.

Assessment of thyroid hormones and antibodies

Compared with the I-deficient group, the FT₃ levels of lactating women were significantly higher in the I-sufficient and excess groups (5.25 and 5.25 vs. 4.79 pmol/l; \( P = 0.049, 0.020 \)).

We additionally observed a trend for increased serum FT₄ concentrations with increasing water I content. In the I-excess group, FT₄ levels of lactating women were significantly higher compared with the I-deficient group (14.10 vs. 12.71 pmol/l; \( P = 0.010 \)).

Median TSH concentrations of lactating women in the I-deficient group were significantly lower than those in the I-sufficient group ($Z = -6.828; \ P = 0.000$) and were higher in the I-excess group compared with the I-sufficient group ($Z = -1.864; \ P = 0.062$), although this difference was not statistically significant.

Serum Tg levels varied markedly among the three groups and were higher in the I-deficient and excess groups compared with the I-sufficient group (18.36 and 16.25 vs. 10.53 μg/l; \( Z = -4.286, \ P = 0.000; Z = -2.579, \ P = 0.01 \)).

We observed no significant differences in positive rates for TgAb, TPOAb and the combined antibodies among the three groups (7.55, 13.46 and 15.79% for TgAb; 13.21, 9.62 and 12.79% for TPOAb; and 5.66, 6.73 and 9.02% for TgAb and TPOAb for the I-deficient, sufficient and excess groups, respectively; Table 4).

Non-linear correlations among TSH and Tg and MUI of lactating women were observed, their relationships are depicted on scatter plots in Fig. 2.

Thyroid diseases

Compared with the I-sufficient group, the prevalence of sub-clinical hypothyroidism was significantly higher ($\chi^2 = 4.486; \ P = 0.034$) in the I-excess group. However, we observed no significant differences in the prevalence of other thyroid diseases examined among the three groups (Table 5).
Logistic regression analysis showed that high water I areas (OR 95% CI 1.59–3.60) were risk factors for abnormal TSH.

Factors revealed that water I, positive rates of TPOAb and TgAb, and MUI of breast-fed infants and median I content in breast milk differed significantly among the three groups (\( P < 0.001 \)). Compared with the control group, MUI values were significantly higher in the I-excess group (\( P = 0.001 \)). Compared with the control group, MUI values were significantly lower in the I-deficient group (\( P = 0.002 \)).

Water I content varied significantly among the three groups (\( P < 0.001 \)). The control group was chosen as the reference group.

MUI of lactating women (\( \mu g/l \)) 20 ± 9.4

MUI of infants (\( \mu g/l \)) 60 ± 26

Median I content in breast milk (\( \mu g/l \)) 11.90

Coverage of household use of qualified iodised salt (%) 8.33

Analysis of risk factors for abnormal thyroid-stimulating hormone in lactating women

Rates of abnormal TSH values (including cases of hypothyroidism, subclinical hypothyroidism, hyperthyroidism and subclinical hyperthyroidism) differed significantly among the three groups (\( \chi^2 = 14.511; P = 0.001 \)). Further analysis of associated factors revealed that water I, positive rates of TPOAb and TgAb, serum Tg level and UI are risk factors for abnormal TSH. Logistic regression analysis showed that high water I areas (OR 2.25; 95% CI 1.067–4.932; \( P = 0.033 \)), high TPOAb (OR 11.033; 95% CI 5.035–24.185; \( P = 0.001 \)) and high Tg levels (OR 1.014; 95% CI 1.005–1.024; \( P = 0.002 \)) are risk factors for abnormal TSH levels (Table 6).

Table 2. Demographic characteristics of lactating women from three different water iodine groups
(Normally distributed mean values and standard deviations; non-normal distributed 25th and 75th percentiles)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Beihai (I-deficient areas)</th>
<th>Yangcheng and Jiaijiazhuan (I-sufficient areas)</th>
<th>Pingyao and Jicun (I-excess areas)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>Median 26-59, Sample size 106</td>
<td>Median 25-53, Sample size 104</td>
<td>Median 25-49, Sample size 133</td>
</tr>
<tr>
<td>Height (m)†</td>
<td>Median 1.57, Sample size 0.06</td>
<td>Median 1.59, Sample size 0.06</td>
<td>Median 1.59, Sample size 0.05</td>
</tr>
<tr>
<td>Weight (kg)†</td>
<td>Median 58.53, Sample size 6.97</td>
<td>Median 60.26, Sample size 11.89</td>
<td>Median 62.07, Sample size 13.02</td>
</tr>
<tr>
<td>Smoking</td>
<td>None, Sample size 0</td>
<td>None, Sample size 0</td>
<td>None, Sample size 0</td>
</tr>
<tr>
<td>MUI (( \mu g/l ))</td>
<td>Median 2.25, Sample size 1.18–4.05</td>
<td>Median 57.5, Sample size 19.00–83.33</td>
<td>Median 464.8, Sample size 339.60–508.73</td>
</tr>
<tr>
<td>I content of iodised salt (( \mu g/kg ))</td>
<td>Median 20.94, Sample size 2.92</td>
<td>Median 11.90, Sample size 4.71</td>
<td>Median 11.73, Sample size 3.77</td>
</tr>
<tr>
<td>Coverage of household use of qualified iodised salt (%)</td>
<td>Median 14.29, Sample size 8.33</td>
<td>Median 8.33, Sample size 4.71</td>
<td>Median 11.73, Sample size 3.77</td>
</tr>
</tbody>
</table>

MUI, median urinary iodine.

Table 3. Status of iodine nutrition in lactating women and infants from the three water iodine groups
(Medians, sample sizes and 25th to 75th percentiles)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Beihai (I-deficient areas)</th>
<th>Yangcheng and Jiaijiazhuan (I-sufficient areas)</th>
<th>Pingyao and Jicun (I-excess areas)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MUI of lactating women (( \mu g/l ))</td>
<td>Median 51.30, Sample size 113</td>
<td>Median 282.42, Sample size 98</td>
<td>Median 822.51, Sample size 125</td>
</tr>
<tr>
<td>MUI of infants (( \mu g/l ))</td>
<td>Median 64.85, Sample size 28</td>
<td>Median 427.01, Sample size 90</td>
<td>Median 1222.41, Sample size 124</td>
</tr>
<tr>
<td>Median I content in breast milk (( \mu g/l ))</td>
<td>Median 41.47, Sample size 103</td>
<td>Median 346.11, Sample size 91</td>
<td>Median 942.33, Sample size 991</td>
</tr>
</tbody>
</table>

MUI, median urinary iodine.

Analysis of risk factors for abnormal thyroid-stimulating hormone in lactating women

Rates of abnormal TSH values (including cases of hypothyroidism, subclinical hypothyroidism, hyperthyroidism and subclinical hyperthyroidism) differed significantly among the three groups (\( \chi^2 = 14.511; P = 0.001 \)). Further analysis of associated factors revealed that water I, positive rates of TPOAb and TgAb, serum Tg level and UI are risk factors for abnormal TSH.

Logistic regression analysis showed that high water I areas (OR 2.25; 95% CI 1.067–4.932; \( P = 0.033 \)), high TPOAb (OR 11.033; 95% CI 5.035–24.185; \( P = 0.001 \)) and high Tg levels (OR 1.014; 95% CI 1.005–1.024; \( P = 0.002 \)) are risk factors for abnormal TSH levels (Table 6).

Discussion

Thyroid hormones play an important role in growth and development of the human body, particularly in the brain. The I intake of breast-fed infants relies solely on concentrations in BM, the only source of I after birth, to meet the infant’s requirements for the synthesis of thyroid hormones(15). Thus, brain development in infants is positively associated with the status of I nutrition and thyroid function of the mother. Previously, IDD were widespread in China. In recent years, IDD has been effectively controlled using the Universal Salt Iodization (USI) programme.

In China, thirty-one million people are currently living in high water I areas (7). Considering the complicated geographical environment of China, we evaluated the status of I nutrition and iodised salt in Beihai was rather low, as cheaper sea salt was rarely used to avoid interference from I in salt, thus ensuring minimal contribution to UI. Coverage of qualified iodised salt in Beihai was rather low, as cheaper sea salt was used by the residents in this coastal city. The use of iodised salt in Beihai was rather low, as cheaper sea salt was rarely used to avoid interference from I in salt, thus ensuring minimal contribution to UI.
Iodine intake and disease in lactating women

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Fig. 1. Positive correlations among breast milk (BM) I, urinary iodine (UI) concentrations of lactating women and UI concentrations of their infants on a scatter plot. Positive correlations are evident between BM I and UI of lactating women, UI of infants and UI of lactating women and BM I and UI of infants (r \(0.879, P = 0.000; r 0.623, P = 0.000; r 0.733, P = 0.000\)).

coverage of qualified iodised salt were relatively low owing to the usage of non-iodised salt.

A number of international researchers and the WHO have recommended that I nutrition from BM is optimal at concentrations of 100–200 μg/l to ensure normal development in infants\(^1\)\(^\text{(20-22)}\). When the MUI of infants <2 years of age is above 100 μg/l, the current I nutrition status of the population is considered adequate. In our study, the median I concentration in water from Beihai in Guangxi province was only 2.2 μg/l, clearly reflecting the lack of I in the external environment. In addition, median BM I was 41.47 μg/l and MUI was 64.85 μg/l in infants, which were lower than the recommended values. These results clearly reveal deficient I nutritional status among lactating women and infants from Beihai. In contrast, MUI was as high as 822.51 and 1222.41 μg/l, respectively, in lactating women and infants from Pingyao and Jicun villages in Shanxi province, indicating excessive I nutritional status in these cases. The above findings support the recommendation that UI concentration should be monitored in lactating women and infants, especially for populations living in high or low water I areas.

Owing to loss of I in BM and urine during lactation, dietary I requirements are increased in lactating women, and I metabolism is enhanced with increasing I intake. The BM I content increased with UI concentration in lactating women in our study. The I intake of breast-fed infants relies solely on concentrations in BM. BM I and UI of breast-fed infants were positively correlated, confirming that the I nutrition status of infants is affected by the I nutrition status of their mothers\(^1\)\(^\text{(23,24)}\).

Previous studies have reported increasing TSH levels with I intake in adults\(^1\)\(^\text{(18,25)}\). Consistent with earlier data, we observed an increase in the median concentration of TSH in lactating women from three regions with different water I contents. The FT\(_3\), FT\(_4\), TSH and UI concentration were low in Beihai, an area with low I levels in drinking water. However, GR was >5%, which was consistent with a report documenting high GR in lactating women in I-deficient areas\(^1\)\(^\text{(26)}\). Accordingly, we speculated that Beihai city is a mild I-deficient area with a low prevalence of thyroid disease.

We compared the utility of TSH and Tg as biomarkers of I status in lactating women using two methods. (1) Groups were classified based on the three different I nutrition areas (Table 4). In this case, TSH increased with the water I content. However, median Tg levels of lactating women in both I-deficient and
excess groups (18.36 and 16.25 μg/l, respectively) were significantly higher than those in the I-sufficient group (10.53 μg/l), showing lower values in the middle and higher values on both sides (approximate V-shape). (2) Groups were classified based on the different UI concentration levels (Fig. 2). In this study, the TSH level was increased and subsequently decreased with increased levels of UI, similar to data from a previous study [27] (approximate inverted ‘U curve’). In children, a ‘U curve’ relationship between TSH and UI has been reported [27]. However, no obvious correlation was evident between median Tg and UI concentration in children, suggesting that Tg is a useful biomarker for I deficiency and excess I intake [26,29].

However, we assumed that children and lactating women are different populations, the latter being affected by the external environment, metabolism of the body itself, diet and small sample size, and further research on a larger scale is warranted. Our preliminary data indicate that TSH and Tg are not suitable as biomarkers for evaluating I nutrition in lactating women.

In our comparative analysis of I-deficient and sufficient areas, increased prevalence of thyroid disease (hypothyroidism, subclinical hypothyroidism, hyperthyroidism and subclinical hyperthyroidism, referred to as abnormal TSH) of lactating women was observed in I-excess areas. In high water I areas, high TPOAb and high Tg levels were determined as risk factors for abnormal TSH among lactating women. However, the influence of I deficiency on abnormal TSH was not established in this study, possibly because only mild I deficiency was present in our survey area of Beihai, Guangxi. Notably, our results are analogous with previous findings of increased incidence of subclinical hypothyroidism in pregnant women under high I conditions [30,31]. The prevalence of hypothyroxinaemia was evaluated as 14.42% in I-sufficient areas, compared with an earlier study showing 10.54% in six I-sufficient areas in China [27]. Taking into consideration the complicated geographical environment of China, household coverage of adequately iodised salt and demographic characteristics in the scope of the current survey, we propose that excessive I intake may lead to thyroid disease, especially subclinical hypothyroidism.

I deficiency results in a wide spectrum of adverse effects throughout the life cycle. Among these, the effect of I deficiency on infant intellectual development is of the greatest concern. No clear results on the association between loss of intelligence and I excess have been obtained to date. In this study, we focused on the status of I nutrition in relation to the prevalence of thyroid disease in lactating women. Urine samples were additionally collected from breast-fed infants to determine UI levels (as urine samples were difficult to collect for infants, we were unable to obtain all the counterpart samples from both lactating women and their infants). Moreover, the infants examined in this study were too young to obtain blood samples, and therefore we planned to use experimental animals for further research. Another limitation of this study was the lack of dietary assessment in individuals (e.g. water consumption was not measured).

Monitoring of the I status of schoolchildren alone is not adequate. The Ministry of Health in China recommended careful monitoring of the I nutrition status in lactating women as part of the state monitoring for preventing and controlling tasks.

### Table 4. Thyroid parameters of lactating women in the three water iodine groups*  
(Numbers and percentages; mean values and standard deviations; medians and 25th to 75th percentiles)

<table>
<thead>
<tr>
<th>Thyroid Parameter</th>
<th>Beihai (I-deficient areas)</th>
<th>Yangcheng and Jiajiazhuang (I-sufficient areas)</th>
<th>Pingyao and Jicun (I-excess areas)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>FT3 (pmol/l)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>79</td>
<td>4.76</td>
<td>25</td>
</tr>
<tr>
<td>SD</td>
<td>36</td>
<td>1.04</td>
<td>31</td>
</tr>
<tr>
<td>FT4 (pmol/l)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>12</td>
<td>13.47</td>
<td>14</td>
</tr>
<tr>
<td>SD</td>
<td>78</td>
<td>2.52</td>
<td>76</td>
</tr>
<tr>
<td>TSH (mIU/l)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>1.08</td>
<td>2.17</td>
<td>1.44</td>
</tr>
<tr>
<td>P25–P75</td>
<td>0.76–1.57</td>
<td>1.44–3.11</td>
<td>1.60–3.51</td>
</tr>
<tr>
<td>Tg (μg/l)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median</td>
<td>18.36</td>
<td>10.53</td>
<td>16.25</td>
</tr>
<tr>
<td>TgAb-positive rate</td>
<td>6</td>
<td>5.66</td>
<td>7</td>
</tr>
<tr>
<td>TPOAb-positive rate</td>
<td>14</td>
<td>13.21</td>
<td>10</td>
</tr>
</tbody>
</table>

FT3, free tri-iodothyronine; FT4, free thyroxin; Tg, thyroglobulin; TgAb, thyroglobulin antibody; TPOAb, thyroid peroxidase antibody; TSH, thyroid-stimulating hormone.
* Differences among the three groups were analysed by ANOVA or the Mann–Whitney test.
† The control group.
‡ The sample sizes.
§ Compared with the I-deficient group, the FT3 levels of lactating women were significantly higher in the I-sufficient groups (P < 0.049).
¶ Compared with the I-deficient group, the FT4 levels of lactating women were significantly higher in the I-excess groups (P = 0.020).
†† In the I-excess group, FT3 levels of lactating women were significantly higher compared with the I-deficient group (14.10 vs. 12.71 pmol/l, P = 0.010).
‡‡ Median TSH concentrations of lactating women in the I-deficient group were significantly lower compared with the I-sufficient group (Z = –2.868, P = 0.000).
††† Serum Tg levels were higher in the I-deficient group than the I-sufficient group (Z = –2.579, P = 0.01).
programme in 2011(8). UI concentrations among lactating women were monitored in thirty-one provinces and corps. According to the monitoring outcomes, MUI values among most lactating women were at the national level. However, both water I content and household coverage of adequately iodised salt were low in Beihai, Guangxi, where the prevalence of thyroid disease was not high but UI levels were low in lactating women and infants. These results clearly indicate that the status of I nutrition in lactating women and infants remains deficient in a number of regions in China. To ensure that brain development in infants is not affected as a result of I deficiency, the relevant governmental departments and the salt industry should

![Image of graphs showing the relationship between MUI of lactating women (µg/l) and TSH (mIU/l), Tg (µg/l) levels.](https://www.cambridge.org/core/terms. For details, visit https://www.cambridge.org/core/terms.

Table 5. Prevalence of thyroid disease among lactating women from the different water I groups*(Number of cases and percentages)

<table>
<thead>
<tr>
<th></th>
<th>Beihai (I-deficient areas)</th>
<th>Yangcheng and Jiajiazhuang (I-sufficient areas)†</th>
<th>Pingyao and Jicun (I-excess areas)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>106</td>
<td>104</td>
<td>133</td>
</tr>
<tr>
<td>Hypothyroxinaemia (%)</td>
<td>16 98</td>
<td>15 42</td>
<td>14 53</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>4 377</td>
<td>4 358</td>
<td>9 677</td>
</tr>
<tr>
<td>Subclinical hypothyroidism</td>
<td>1 94</td>
<td>6 777</td>
<td>19 1429‡</td>
</tr>
<tr>
<td>Hyperthyroidism</td>
<td>2 89</td>
<td>4 358</td>
<td>6 451</td>
</tr>
<tr>
<td>Subclinical hyperthyroidism</td>
<td>2 89</td>
<td>2 92</td>
<td>2 150</td>
</tr>
<tr>
<td>Autoimmune thyroiditis</td>
<td>4 777</td>
<td>5 81</td>
<td>9 677</td>
</tr>
<tr>
<td>High serum autoantibody values</td>
<td>16 90</td>
<td>17 83</td>
<td>26 195</td>
</tr>
<tr>
<td>Goitre prevalence (%)</td>
<td>6 66</td>
<td>2 96</td>
<td>1 75</td>
</tr>
<tr>
<td>Thyroid nodules prevalence (%)</td>
<td>17 70</td>
<td>23 12</td>
<td>24 80</td>
</tr>
</tbody>
</table>

* Differences among the three groups were analysed by χ² test.
† The control group.
‡ Compared with the I-sufficient group, the prevalence of subclinical hypothyroidism was significantly higher (χ² = 4.486; P = 0.034) in the I-excess group.
increase co-operation and supervision to put effective targeted measures of I supplementation in place. Analogously, it is important to prevent excessive I intake through stopping the provision of iodised salt and implementing effective measurements for water improvement (such as finding other drinking water sources with no excessive I) in high water I areas. Public awareness of the adverse effects of inadequate I intake on infant development needs to be enhanced. Further implementation of scientific I supplementation strategies composed of measures that are contextually appropriate, diverse for the general population and specific for the key ones is necessary.

**Conclusion**

Excessive I intake may induce subclinical hypothyroidism in lactating women. Moreover, adequate I nutrition is essential for lactating women and infants, especially those living in I-deficient areas. Enhanced monitoring of I status is important to avoid adverse effects of I deficiency or excess, particularly in susceptible populations such as pregnant or lactating women and infants.

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**References**


