Iodine nutrition and thyroid diseases in Chengdu, China: an epidemiological study

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Summary

Objective: To assess the iodine nutritional status and investigate the prevalence of thyroid diseases in a community population in Chengdu, China.

Methods: Eighty school-age children were randomly selected for measurements of urinary iodine concentration. A total of 1500 residents over the age of 18 who had lived in Chengdu for more than 5 years were selected by stratified cluster sampling. Serum thyroid hormone concentrations and thyroid autoantibodies were measured, and thyroid ultrasonography was performed.

Results: The median urine iodine concentration was 184 μg/l in school-age children. The prevalence of clinical hyperthyroidism, subclinical hyperthyroidism, clinical hypothyroidism and subclinical hypothyroidism was 0.97%, 1.95%, 0.90% and 5.55%, respectively. The prevalence of thyroid autoantibodies and thyroid nodules was 15.82% and 16.87%, respectively. The prevalence of clinical hyper- and hypothyroidism was greater in females than in males (P < 0.05). The prevalence of subclinical hyper- and hypothyroidism, thyroid nodules and thyroid autoantibodies increased significantly with age (P < 0.05). The rate of new abnormal TSH was 9.37%, and the average serum Thyroid Stimulating Hormone (TSH) concentrations increased with age. When TSH >0.71 mU/l, the prevalence of positive TPOAb and/or TgAb increased significantly with rising concentrations of TSH (P<0.05); however, the prevalence of thyroid nodules did not increase with escalating concentrations of TSH (P=0.09).

Conclusion: Subclinical thyroid diseases, especially subclinical hypothyroidism and thyroid nodules, are common in an iodine sufficient area. Females and the elderly might benefit from routine screening for thyroid diseases, followed by appropriate detection and treatment.

Introduction

Thyroid diseases are commonly observed in both outpatient and inpatient hospital clients. Numerous studies have reported on the prevalence of various thyroid diseases, but the results of these studies vary. Several factors may affect the prevalence of thyroid disease, especially ethnic and geographic factors.1 Family studies have determined that thyroid autoantibody presentation was contingent upon genetic factors.2 Laurberg et al.3 demonstrated that the environmental factors which may affect thyroid disease include iodine nutrition, geographical conditions, smoking and alcohol consumption. In addition, the prevalence of thyroid diseases is also influenced by gender and age.4

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Iodine is an important micronutrient required for the synthesis of thyroid hormones; both insufficient and excess iodine intake may lead to thyroid diseases. Mainland China began universal salt iodization (USI) in 1996, and has effectively controlled iodine deficiency disorders. However, few studies have been conducted to investigate the effects of USI on the thyroid disease spectrum. The aim of our study is to assess iodine nutritional status and investigate the prevalence of thyroid diseases in a community population in Chengdu, China.

Materials and methods

Population

All subjects participating in this study came from a community in Chengdu (1 of 10 cities undergoing epidemiological investigation regarding thyroid disease in China). In total, 1500 residents over the age of 18 years old who had lived in Chengdu for more than 5 years were selected by stratified cluster sampling. Of these, 1350 subjects completed the investigation and were included in this study, yielding a response rate of 90%. The exclusion criteria were as follows: (i) Pregnant females or postpartum women within 1 year; (ii) individuals taking glucocorticoids, dopamine or dobutamine; (iii) individuals taking antiepileptic drugs (phenytoin, carbamazepine, etc.); (iv) individuals suffering from adrenocortical insufficiency, renal insufficiency or any other serious systemic disease or chronic wasting disease; and (v) individuals receiving amiodarone or an iodine-containing contrast agent within 6 months.

Assessment

All participants in this study provided informed consent and were asked to complete a self-assessment questionnaire that included demographic data, reproductive history, smoking history, previous thyroid disease, family history of thyroid disease, etc. Height, weight and blood pressure were measured in all participants. Fasting blood samples were collected and centrifuged at 704 x g speed for 5 min. Serum was decanted and stored at -20°C until the assay. Fasting urine specimens from 80 school-age children were collected and frozen (-20°C).

Thyroid ultrasonography was performed with a 7.5 MHz ultrasound imager (GE, LOGIQ@50), and thyroid nodule diameter was measured. Thyroid function and thyroid autoantibodies were measured using chemiluminescence immunoassay kits (Roche Kit, Cobas-e601 analyser). The intra-assay and inter-assay coefficients of variation were all <5%. TPOAb, TgAb and TSH were measured in all participants. If TSH was <0.71 mU/l, then free thyroxine (FT4) and free triiodothyronine (FT3) were measured in the same sample. If TSH >6.25 mU/l, only FT4 was measured. Median urinary iodine was measured with an As-Ce Catalytic Chromatographer. The reference values were as follows: FT4 12.0–22.0 pmol/l, FT3 3.1–6.8 pmol/l, TSH 0.71–6.25 mU/l, TPOAb <34 IU/ml and TgAb <115 IU/ml.

Diagnostic criteria for thyroid diseases

Clinical hyperthyroidism (overt thyrotoxicosis) was defined as TSH <0.71 mU/l and FT4 >22.0 pmol/l, and/or TSH <0.71 mU/l and FT3 >6.8 pmol/l. Subclinical hyperthyroidism (subclinical thyrotoxicosis) was based on normal FT4 and FT3 and TSH <0.71 mU/l. Clinical hypothyroidism (overt hypothyroidism) was defined as TSH >6.25 mU/l and FT4 <22.0 pmol/l, whereas subclinical hypothyroidism was based on normal FT4 and TSH >6.25 mU/l. Positive thyroid autoantibodies were defined as TPOAb ≥34 IU/ml and/or TgAb >115 IU/ml.

Statistics

Data processing was performed with SPSS version 16.0. Laboratory values were, respectively, reported as the mean ± SD, percentage, median and interquartile range when appropriate. For continuous variables, non-parametric statistics (Mann–Whitney or Kruskal–Wallis) and parametric statistics (t-test) were used when appropriate. Group differences between the numbers of subjects were analysed using a Chi-squared test. Spearman correlation analysis was used between serum TSH and age. The level of significance was set at 5%.

Results

Clinical characteristics and urine iodine status of subjects

All of the 1500 participants were invited to participate in the study, and 150 subjects of which for not completing the investigation were excluded. In total, 1350 individuals with a mean age of 45.83 ± 15.20 years (range, 18–82 years) participated in this study. Of these, 608 (45%) were males with a mean age of 45 ± 15.81 years, and 742 (55%) were females with a mean age of 46.5 ± 14.65 years. There was no significant difference in age between genders among all subjects (P=0.071). The median urine iodine concentration in 80 school-age children was 184 μg/l.
The prevalence of thyroid dysfunction and influenced factors

Of the 1334 subjects without a history of thyroid disease, the prevalences of clinical hyperthyroidism, subclinical hyperthyroidism, clinical hypothyroidism, and subclinical hypothyroidism were 0.97%, 1.95%, 0.90% and 5.55%, respectively. The prevalences of clinical hyperthyroidism (1.51% vs. 0.33%, \( P = 0.03 \)) and hypothyroidism (1.51% vs. 0.17%, \( P = 0.01 \)) were both significantly greater in females than in males, whereas there were no significant differences in the prevalences of subclinical hyperthyroidism (2.05% vs. 1.82%, \( P = 0.76 \)) and hypothyroidism (5.75% vs. 5.30%, \( P = 0.72 \)) between females and males (Table 1).

The 1334 participants without a history of thyroid disease were divided by age into three groups. The prevalence of subclinical hyperthyroidism \( (P = 0.008) \) and hypothyroidism \( (P < 0.001) \) was significantly elevated with age, but no similar trends were found for the prevalence of clinical hyperthyroidism \( (P = 0.091) \) or hypothyroidism \( (P = 0.62, \) Chi-squared test).

The prevalence of thyroid autoantibodies and influenced factors

In 1334 subjects without a history of thyroid disease, 15.82% were measured with positive thyroid autoantibodies. The prevalence of thyroid autoantibodies (TPOAb and/or TgAb) was \( \approx 2 \)-fold greater among women than in men (21.37% and 9.11%, \( P < 0.001 \)). Age subgroup studies observed that the prevalences of thyroid autoantibodies in 18–39-year-old, 40–59-year-old and 60–82-year-old age groups were 12.40%, 17.38% and 19.26%, respectively (Table 2), and increased significantly with age (Chi-squared test for trend, \( P < 0.001 \)). Further analysis found that in the 18–39-year-old group, the prevalence of TPOAb, TgAb, TPOAb and/or TgAb, and TPOAb and TgAb was greater in females than in males (TPOAb: \( P = 0.013 \); TgAb: \( P < 0.001 \); TPOAb and/or TgAb: \( P < 0.001 \); TPOAb and TgAb: \( P = 0.011 \)); similar results were found in the 40–59 age group (TPOAb: \( P = 0.007 \); TgAb: \( P < 0.001 \); TPOAb and/or TgAb: \( P < 0.001 \); TPOAb and TgAb: \( P = 0.003 \)). However, there were no significant differences in the 60–82 age group (TPOAb: \( P = 0.098 \); TgAb: \( P = 0.57 \); TPOAb and/or TgAb: \( P = 0.091 \); TPOAb and TgAb: \( P = 0.69, \) Table 3).

In addition, we grouped the 1334 individuals without a history of thyroid disease by TSH level into five groups (the group criteria were used as reference 4). There was a trend towards increasing prevalence of thyroid autoantibodies with increasing TSH when TSH >0.71 mU/l (TPOAb: \( P < 0.001 \); TgAb: \( P < 0.001 \); TPOAb and/or TgAb: \( P < 0.001 \); TPOAb and TgAb: \( P < 0.001 \); Chi-squared test, Table 4).

The prevalence of thyroid nodules and influenced factors

The prevalence of thyroid nodules was 16.87% in the 1334 subjects without a history of thyroid disease (Table 1), and thyroid nodules were more common in females than in males among these subjects (20.41% vs. 12.58%, \( P < 0.001 \)). The prevalences of thyroid nodules in three age groups were 6.69%, 16.82% and 36.67%, respectively (Table 2), and increased significantly with increasing age (Chi-squared test for trend, \( P < 0.001 \)). In the 18–39 and 40–59 age groups (Table 3), the prevalence of thyroid nodules in females was higher than

<table>
<thead>
<tr>
<th>Thyroid function status</th>
<th>Total</th>
<th>( P )</th>
<th>Thyroid disease-free</th>
<th>( P )</th>
<th>Previous history of thyroid disease</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>( n )</td>
<td>( % )</td>
<td>( n )</td>
<td>( % )</td>
<td>( n )</td>
</tr>
<tr>
<td>Total</td>
<td>608</td>
<td>(45.04)</td>
<td>742</td>
<td>(54.96)</td>
<td>604</td>
</tr>
<tr>
<td>Eu</td>
<td>558</td>
<td>(91.78)</td>
<td>660</td>
<td>(88.95)</td>
<td>558</td>
</tr>
<tr>
<td>OH</td>
<td>2</td>
<td>(0.33)</td>
<td>11</td>
<td>(1.48)*</td>
<td>1</td>
</tr>
<tr>
<td>SH</td>
<td>33</td>
<td>(5.43)</td>
<td>42</td>
<td>(5.66)</td>
<td>32</td>
</tr>
<tr>
<td>OT</td>
<td>3</td>
<td>(0.49)</td>
<td>12</td>
<td>(1.61)*</td>
<td>2</td>
</tr>
<tr>
<td>ST</td>
<td>12</td>
<td>(1.97)</td>
<td>17</td>
<td>(2.99)</td>
<td>11</td>
</tr>
<tr>
<td>Nodule</td>
<td>77</td>
<td>(12.66)</td>
<td>152</td>
<td>(20.49)*</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*\( P < 0.05 \) (compared between females and males). Eu, euthyroid; OH, overt hypothyroidism; SH, subclinical hypothyroidism; OT, overt thyrotoxicosis; ST, subclinical thyrotoxicosis.
those in males (18–39 year, \(P=0.019\); 40–59 year, \(P<0.001\)); however, there was no significant difference between females and males in the 60–82 age group (60–82 year, \(P=0.52\)).

In addition, TSH subgroup study failed to show a significant increasing trend for the prevalence of thyroid nodules with increasing TSH level when TSH >0.71 mU/l (Chi-squared test, \(P=0.09\), Table 4).

### The median serum TSH level and influenced factors

The median serum TSH level in the thyroid disease-free population (1334) was 2.64 (1.81–3.86)mU/l, and the median TSH in females was higher than that in males (2.98 vs. 2.44, \(P<0.001\)). A total of 125 (9.37%) subjects had an abnormal serum TSH level. There were 86 subjects (6.45%) with an elevated TSH level, 25 of which had TSH >10 mU/l (5 males, 20 females). The other 61 individuals had a value between 6.25 and 10 mU/l (28 males, 33 females). Thirty-nine subjects (2.9%) had a value lower than 0.71 mU/l (13 males, 26 females). The median TSH value was not significantly greater in females than in males, both in the 18–39 year (2.47 vs. 2.38, \(P=0.299\)) group and the 60–82 year (3.28 vs. 2.70, \(P=0.069\)) group. However, in the 40–59 age group, the TSH level in females was significantly higher than that in males (2.98 \(P=0.019\); 40–59 year, \(P<0.001\)).

### Table 1

<table>
<thead>
<tr>
<th>Group (year)</th>
<th>Total (n)</th>
<th>OT n (%)</th>
<th>ST n (%)</th>
<th>OH n (%)</th>
<th>SH n (%)</th>
<th>Nodules n (%)</th>
<th>Ab+ n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>18–39</td>
<td>523</td>
<td>8 (1.53)</td>
<td>18 (3.44)</td>
<td>4 (0.76)</td>
<td>15 (2.87)</td>
<td>35 (6.69)</td>
<td>65 (12.40)</td>
</tr>
<tr>
<td>40–59</td>
<td>541</td>
<td>4 (0.74)</td>
<td>5 (0.92)</td>
<td>5 (0.92)</td>
<td>34 (6.28)</td>
<td>91 (16.82)</td>
<td>94 (17.38)</td>
</tr>
<tr>
<td>60–82</td>
<td>270</td>
<td>1 (0.37)</td>
<td>3 (1.11)</td>
<td>3 (1.11)</td>
<td>25 (9.26)</td>
<td>99 (36.67)</td>
<td>52 (19.26)</td>
</tr>
<tr>
<td>Total</td>
<td>1334</td>
<td>13 (0.97)</td>
<td>26 (1.95)</td>
<td>12 (0.90)</td>
<td>74 (5.55)</td>
<td>225 (16.87)</td>
<td>211 (15.82)</td>
</tr>
</tbody>
</table>

Ab (+), positive TPOAb and/or TgAb; TPOAb & TgAb, positive TPOAb and TgAb simultaneously.

### Table 2

<table>
<thead>
<tr>
<th>Group (year)</th>
<th>Gender</th>
<th>n</th>
<th>TPOAb (+) n (%)</th>
<th>TgAb (+) n (%)</th>
<th>Ab (+) n (%)</th>
<th>TPOAb and TgAb (+) n (%)</th>
<th>Nodules n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>18–39</td>
<td>Male</td>
<td>264</td>
<td>16 (6.06)</td>
<td>10 (3.79)</td>
<td>18 (6.82)</td>
<td>8 (3.03)</td>
<td>11 (4.17)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>259</td>
<td>32 (12.36)</td>
<td>36 (13.90)</td>
<td>47 (18.15)</td>
<td>21 (8.11)</td>
<td>24 (9.27)</td>
</tr>
<tr>
<td>40–59</td>
<td>Male</td>
<td>213</td>
<td>16 (7.51)</td>
<td>7 (3.29)</td>
<td>18 (8.45)</td>
<td>5 (2.34)</td>
<td>21 (9.86)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>328</td>
<td>50 (15.24)</td>
<td>54 (16.46)</td>
<td>76 (23.17)</td>
<td>28 (8.53)</td>
<td>70 (21.34)</td>
</tr>
<tr>
<td>(\geq)60</td>
<td>Male</td>
<td>127</td>
<td>14 (11.02)</td>
<td>14 (11.02)</td>
<td>19 (14.96)</td>
<td>9 (7.09)</td>
<td>44 (34.65)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>143</td>
<td>26 (18.18)</td>
<td>19 (13.29)</td>
<td>33 (23.07)</td>
<td>12 (8.39)</td>
<td>55 (38.46)</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>1334</td>
<td>154 (11.54)</td>
<td>140 (10.49)</td>
<td>211 (15.82)</td>
<td>83 (6.22)</td>
<td>225 (16.87)</td>
</tr>
</tbody>
</table>

### Table 3

<table>
<thead>
<tr>
<th>Group (year)</th>
<th>Gender</th>
<th>TPOAb (+) n (%)</th>
<th>TgAb (+) n (%)</th>
<th>Ab (+) n (%)</th>
<th>TPOAb and TgAb (+) n (%)</th>
<th>Nodules n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>18–39</td>
<td>Male</td>
<td>16 (30.77)</td>
<td>11 (28.21)</td>
<td>15 (38.46)</td>
<td>8 (20.51)</td>
<td>9 (23.07)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>45 (7.85)</td>
<td>47 (8.20)</td>
<td>70 (12.22)</td>
<td>22 (3.84)</td>
<td>88 (15.36)</td>
</tr>
<tr>
<td>40–59</td>
<td>Male</td>
<td>72 (11.32)</td>
<td>59 (9.28)</td>
<td>97 (15.25)</td>
<td>34 (5.35)</td>
<td>109 (17.13)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>13 (21.31)</td>
<td>13 (21.31)</td>
<td>16 (26.23)</td>
<td>10 (16.39)</td>
<td>12 (19.67)</td>
</tr>
<tr>
<td>(\geq)60</td>
<td>Male</td>
<td>12 (48.00)</td>
<td>10 (40.00)</td>
<td>13 (52.00)</td>
<td>9 (36.00)</td>
<td>7 (28.00)</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>154 (11.54)</td>
<td>140 (10.49)</td>
<td>211 (15.82)</td>
<td>83 (6.22)</td>
<td>225 (16.87)</td>
</tr>
</tbody>
</table>

Ab (+), positive TPOAb and/or TgAb; TPOAb & TgAb, positive TPOAb and TgAb simultaneously.

### Table 4

<table>
<thead>
<tr>
<th>TSH levels mU/l</th>
<th>n</th>
<th>TPOAb (+) n (%)</th>
<th>TgAb (+) n (%)</th>
<th>Ab (+) n (%)</th>
<th>TPOAb and TgAb (+) n (%)</th>
<th>Nodules n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TSH &lt;0.71 (A)</td>
<td>39</td>
<td>12 (30.77)</td>
<td>11 (28.21)</td>
<td>15 (38.46)</td>
<td>8 (20.51)</td>
<td>9 (23.07)</td>
</tr>
<tr>
<td>0.71≤TSH &lt;2.5 (B)</td>
<td>573</td>
<td>45 (7.85)</td>
<td>47 (8.20)</td>
<td>70 (12.22)</td>
<td>22 (3.84)</td>
<td>88 (15.36)</td>
</tr>
<tr>
<td>2.5≤TSH &lt;6.25 (C)</td>
<td>636</td>
<td>72 (11.32)</td>
<td>59 (9.28)</td>
<td>97 (15.25)</td>
<td>34 (5.35)</td>
<td>109 (17.13)</td>
</tr>
<tr>
<td>6.25≤TSH &lt;10 (D)</td>
<td>61</td>
<td>13 (21.31)</td>
<td>13 (21.31)</td>
<td>16 (26.23)</td>
<td>10 (16.39)</td>
<td>12 (19.67)</td>
</tr>
<tr>
<td>TSH≥10 (E)</td>
<td>25</td>
<td>12 (48.00)</td>
<td>10 (40.00)</td>
<td>13 (52.00)</td>
<td>9 (36.00)</td>
<td>7 (28.00)</td>
</tr>
<tr>
<td>Total</td>
<td>1334</td>
<td>154 (11.54)</td>
<td>140 (10.49)</td>
<td>211 (15.82)</td>
<td>83 (6.22)</td>
<td>225 (16.87)</td>
</tr>
</tbody>
</table>

Ab (+), positive TPOAb and/or TgAb; TPOAb & TgAb, positive TPOAb and TgAb simultaneously.

OH, overt hypothyroidism; SH, subclinical hypothyroidism; OT, overt thyrotoxicosis; ST, subclinical thyrotoxicosis; Ab (+), positive TPOAb and/or TgAb.
greater than in males (3.09 vs. 2.39, \( P < 0.001 \), Figure 1). In the Spearman correlation analysis, the TSH level was significantly positively correlated with age in both females and males (females \( r = 0.167, \ P < 0.001 \); males \( r = 0.087, \ P = 0.032 \)).

**Discussion**

In our study, the median urinary iodine concentration (UIC) in school-age children was 184 \( \mu \)g/l. Based on the median UIC, according to the criteria from WHO/UNICEF/ICCIDD 2007 for assessing iodine nutrition, iodine nutrition was sufficient in the Chengdu area, which had once been an iodine-deficient but non-iodine-deficiency-disease area before USI conducted in 1996 (data from Center for Disease Control and Prevention of Sichuan Province, China). The prevalence of subclinical hypothyroidism was 5.55% in the subjects of our study. Teng et al. found that in an iodine insufficient area, and 0.91% people had subclinical hypothyroidism. A survey conducted in iodine replete areas in China observed that the prevalence of subclinical hypothyroidism was 4.72% in adults aged 18–45 years. The TSH level was correlated with increased iodine intake. However, the mechanisms between iodine nutrition and TSH are complicated. The relatively high iodine nutrition status may affect thyroid follicular cells apoptosis, trigger and exacerbate autoimmune thyroiditis, which increases the likelihood of subclinical or clinical hypothyroidism. Except for the autoimmune factors, high iodine intake could induce elevation of serum TSH levels through inhibiting pituitary D2 activity in Wistar rats, which suggested that non-autoimmune factors might also play an role in the underlying mechanisms of iodine-induced subclinical and clinical hypothyroidism. We also found that the TSH level increased with age in both females and males. Serum TSH level was demonstrated to be paralleled associated with age. Increased TSH concentration in aging rats have been reported in some studies. However, the mechanisms regulating age effects on serum TSH levels have not been elucidated and should be studied in the future. With the exception of iodine nutrition and age, other factors might affect the prevalence of thyroid diseases. Therefore, even in an area with sufficient iodine, thyroid diseases, especially subclinical hypothyroidism and its influenced factors, such as increased age, should remain the focus of clinical attention.

Several studies have demonstrated via cross-sectional survey that the prevalence of TPOAb was \( \sim 7\%–10\% \) and 5–10% for TgAb. We found the rates of TPOAb were 11.54%, and 10.49% for TgAb in populations with sufficient iodine intake. A survey that selected females without thyroid disease in an iodine excess area in China found that the rates of TPOAb were 17.6%, and 14.3% for TgAb. Sang et al. reported that the percentage of TPOAb- and TgAb- positivity was higher in children from the high iodine area compared with the adequate iodine area in Hebei province, China. High iodine has been reported to initiate and exacerbate infiltration of thyroid lymphocytes in genetically susceptible BB/W rats and NOD.H-2h4 mice. Further, high iodine combined with TgAb might enhance antigenicity and promote lymphocyte proliferation. Excessive iodine intake might lead to autoimmune thyroiditis. Li et al. from China observed that both in the baseline and 5-year-follow-up study, the higher incidence of hypothyroidism in thyroid autoantibody-positive subjects was associated with higher iodine intake. The increased iodine intake might be a risk factor for autoimmune prone subjects to develop subclinical and clinical hypothyroidism. In addition, we observed that the prevalence of thyroid autoantibodies increased significantly with increasing age and TSH levels (when TSH >0.71 mU/l). These findings seem to indicate that a relatively high level of TSH might be required to maintain euthyroid function when thyroid autoantibodies are present. Therefore, elevated TSH levels and the presence of thyroid autoantibodies may be risk predictors for the development of subclinical and clinical hypothyroidism in the general population with sufficient iodine nutrition, even when TSH levels are within the normal range.

Thyroid ultrasonography (US) may increase detection of thyroid nodules. A previous study demonstrated that thyroid nodules were found in
19–67% of patients by US. The prevalence of thyroid nodules was 16.87% by US in our study. Significant differences in the prevalence of thyroid nodules were found between females and males. Similarly, as shown in other studies, thyroid nodules were more common in females than in males. In a Chinese population, women were more susceptible to thyroid nodules compared with men (OR: 2.809; 95% CI: 2.444, 3.228). However, we did not find a different prevalence of thyroid nodules between females and males who were older than 60 year, highlighting the need for an expanded sample size to further validate these findings. Our observations further found that thyroid nodules were more prevalent in older participants and increased with age in iodine sufficient area, which had once been iodine deficient area before USI conducted in 1996. This suggests that, although iodine deficiency may lead to a increased risk of thyroid nodule, iodine supplementation did not decrease the incidence of thyroid nodules. The result of anatomical changes by the long-standing iodine deficient adaptation could not be corrected by iodine supplementation.

Approximately 43.75% (7/16) of subjects with previous thyroid disease presented with an abnormal TSH level in our study. The Colorado thyroid study, which reported that nearly 40% of participants who had received thyroid medications maintained an abnormal serum TSH level. However, of the thyroid disease-free subjects, 9.37% (125/1334) had an abnormal serum TSH level, most of whom exhibited subclinical hypothyroidism (6.45% elevated and 2.92% declined). The TSH level was greater in females than in males. A thyroid investigation conducted in Ningbo, an iodine sufficient city in China, found that 6.3% of participants without a history of thyroid disease had an increased TSH level, whereas 0.87% had a low TSH level. Therefore, these findings suggest that in the iodine sufficient area there are considerable subjects with a functionally abnormal thyroid gland, whether with previous thyroid history or not.

In summary, the prevalence of thyroid dysfunction, especially subclinical hypothyroidism, was relatively high despite sufficient iodine nutrition. Some studies have demonstrated that elderly women who suffer from subclinical hypothyroidism are at an increased risk for atherosclerosis and myocardial infarction; subclinical hyperthyroidism is a risk factor for atrial fibrillation in elderly populations. Therefore, we suggest that elderly populations with subclinical thyroid disease should be examined for thyroid function. In our study, the TSH level and the prevalence of thyroid autoantibodies were higher in females; both increased significantly with age. It would be beneficial to undertake a longitudinal follow-up study for elderly females with positive thyroid autoantibodies and subclinical thyroid disease.

Our investigation has several limitations. First, the study was not performed on subjects under the age of 18 year, and the proportion of the study population over the age of 60 year was relatively small. Second, the study was cross-sectional and hence did not include individual change over time. Third, laboratory tests of FT4 and/or FT3 were performed only in populations with an abnormal TSH level, not in populations with a normal TSH level. Finally, the median UIC was measured only in school-aged children and not in the entire study population. Therefore, we would have a follow-up study for their changes of thyroid of the population and survey about the relationship between the prevalence of thyroid diseases and the individual iodine nutritional status.

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References

Iodine nutrition and thyroid diseases


