Suppression of Thyroid Function during Ingestion of Seaweed “Kombu” (Laminaria japonica) in Normal Japanese Adults

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Abstract. The effect of ingesting seaweed “Kombu” (Laminaria japonica) on thyroid function was studied in normal Japanese adults. Ingesting 15 and 30 g of Kombu (iodine contents: 35 and 70 mg) daily for a short term (7–10 days) significantly increased serum thyrotropin (TSH) concentrations, exceeding the normal limits in some subjects. The serum free thyroxine (FT$_4$) and/or free 3,5,3′-triiodothyronine (FT$_3$) concentrations were slightly decreased within the normal limits. During long term daily ingestion of 15 g of Kombu (55–87 days), the TSH levels were elevated and sustained while the FT$_4$ and FT$_3$ levels were almost unchanged. Urinary excretion of iodine significantly increased during ingestion of Kombu. These abnormal values returned to the initial levels 7 to 40 days after discontinuing the ingestion of Kombu. Based on these findings that thyroid function was suppressed during ingestion of Kombu, though the effect was reversible, we recommend Japanese people avoid ingesting excessive amounts of seaweed.

Key words: Excess iodine, Seaweed ingestion, Hypothyroidism, Kombu (Laminaria japonica), Urinary iodine excretion

Since iodine is an essential element of thyroid hormones, changes in iodine intake may cause various thyroid disorders. It is well known that iodine deficiency induces hypothyroidism, whereas excess iodine may induce not only hyperthyroidism but also hypothyroidism. A number of studies have shown that excess intake of iodide or iodine-containing drugs resulted in hypothyroidism, particularly in subjects living in iodine-sufficient area, neonatal or elderly persons, patients with underlying thyroid disorders such as autoimmune thyroid diseases, subacute thyroiditis, thyroidectomy and so on [1–3]. Since most Japanese people eat iodine-rich foods such as seaweed, Japan is considered as an iodine-sufficient area. Thus, cases of hypothyroidism or goiter due to excess iodine intake have been reported. Namba et al. [4] demonstrated that administration of iodine-containing tablets to normal Japanese people resulted in a rise in serum thyrotropin (TSH) with a slight decline in serum free thyroxine (FT$_4$) levels and enlarged thyroid volume. However, Takamura et al. [5] reported that iodine-containing tablet or solution was effective for rapid blockage of thyroid function but iodine-rich food was not effective.

Therefore, we designed this study to determine whether ingestion of seaweed affects thyroid function in normal Japanese adults.

Subjects and Methods

A total of 23 experiments were performed in 13 normal adults (8 male and 5 female healthy volunteers, 27 ± 12 years old) whose serum TSH, FT$_4$ and free 3,5,3′-triiodothyronine (FT$_3$) concentrations were within normal limits and anti-thyroid antibodies were negative.
The subjects ingested ordinary Japanese foods without seaweed throughout the experimental period. Then Kombu (Laminaria japonica) was added to the ordinary diet as follows: the daily amounts of 15 g (experiments A and C) or 30 g (experiment B) of Kombu which contain 35 mg or 70 mg of iodine, for 7–10 days for the short-term experiment (A, B) and 55–87 days for the long-term experiment (C). Kombu as “Tororo Kombu” (iodine content was 2.3 mg (1.8 × 10⁻⁵ mol)/g (dry weight)) was purchased from Oguraya Co. Ltd., Japan.

Serum TSH, FT₄, and FT₃ concentrations and urinary iodide excretion before, during and after discontinuation of Kombu ingestion

| Table 1. Serum TSH, FT₄, FT₃ concentrations and urinary iodide excretion before, during and after discontinuation of Kombu ingestion |
|-----------------|-----------------|-----------------|
| (A) 15 g Kombu, short term (n = 6) |
| | before | during (7–10 days) | after (7–14 days) |
| TSH (µU/ml) [0.40–4.40] | 2.15 ± 0.32 | 4.31 ± 0.57 [[4/6]] | 2.33 ± 0.47 |
| | p<0.05<sup>t</sup>, w<sup>p<0.05</sup> | p<0.05<sup>ts</sup>, w<sup>p<0.05</sup> | p<0.05<sup>t</sup>, w<sup>p<0.05</sup> |
| FT₄ (ng/dl) [0.8–1.7] | 1.21 ± 0.07 | 1.14 ± 0.11 | 1.23 ± 0.13 |
| | ns<sup>ts</sup> | ns<sup>ts</sup> | ns<sup>ts</sup> |
| FT₃ (pg/ml) [2.0–4.0] | 3.17 ± 0.09 | 2.93 ± 0.10 | 3.13 ± 0.12 |
| | p<0.05<sup>t</sup> | p<0.05<sup>t</sup> | ns<sup>ts</sup> |
| Urinary iodide (iodine mg/day) | 0.56 ± 0.07 | 17.2 ± 2.24 | 1.40 ± 0.33 |
| | p<0.001<sup>t</sup> | p<0.005<sup>t</sup> | ns<sup>ts</sup> |

(B) 30 g Kombu, short term (n = 14)

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<td></td>
<td>before</td>
<td>during (7–10 days)</td>
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<td>TSH (µU/ml)</td>
<td>1.54 ± 0.20</td>
<td>3.09 ± 0.41 [[4/14]]</td>
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<td>p&lt;0.005&lt;sup&gt;t&lt;/sup&gt;, 0.01&lt;sup&gt;†&lt;/sup&gt;</td>
<td>p&lt;0.01&lt;sup&gt;t&lt;/sup&gt;, 0.05&lt;sup&gt;s&lt;/sup&gt;</td>
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<td>FT₄ (ng/dl)</td>
<td>1.36 ± 0.04</td>
<td>1.21 ± 0.00</td>
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<td>p&lt;0.01&lt;sup&gt;ts&lt;/sup&gt;</td>
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<tr>
<td>FT₃ (pg/ml)</td>
<td>3.20 ± 0.08</td>
<td>2.98 ± 0.07</td>
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<td>p&lt;0.01&lt;sup&gt;ts&lt;/sup&gt;, 0.05&lt;sup&gt;s&lt;/sup&gt;</td>
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<td>Urinary iodide (iodine mg/day)</td>
<td>1.11 ± 0.26</td>
<td>48.8 ± 9.6</td>
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<td>p&lt;0.001&lt;sup&gt;t&lt;/sup&gt;, 0.01&lt;sup&gt;†&lt;/sup&gt;</td>
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The differences in data between groups were ana-
lyzed parametrically by the t test and non-parametrically by Wilcoxon and Friedman tests.

**Results**

Table 1 summarized changes in serum TSH, FT$_4$ and FT$_3$ concentrations and urinary excretion of iodide, before, during and discontinuation of Kombu ingestion (15 g (A, C) or 30 g (B) of Kombu for the short-term (A, B) and long-term (C) experiments). During ingestion of Kombu, serum TSH concentrations increased significantly with peak values over the normal limits in some subjects (4/6 (A), 4/14 (B) and 1/3 (C) subjects) in all experiments. Serum FT$_4$ (B) and FT$_3$ (A, B) levels were lowered slightly within normal limits in the short-term experiments and these values were almost unchanged in long-term experiments. Urinary excretion of iodide were elevated to about 30-fold (A, C) and 44-fold (B) the initial levels. These abnormal values returned to the initial levels 7 to 40 days after discontinuation of Kombu ingestion. An example is shown in Fig. 1.

**Discussion**

Previous reports demonstrated that thyroid functions were suppressed in normal subjects following administration of drugs containing various amounts of iodine as follows: 0.25–1.5 mg as Na I [11], 0.42–3.8 mg as NaI [12], 12.5 mg as Lugol’s solution [13], 27 mg as licorice lecithin-bound iodine [4], 114.8 mg as KI [14] per day. In the present study, 35 or 70 mg of iodine as 15 or 30 g of seaweed Kombu were administered daily, and the daily excretion of urinary iodine as iodide which is known to be the most valid index of absorbed iodine was estimated to be about 20 or 50 mg (57–71% of the iodine administered as Kombu).

The present study demonstrated that daily ingestion
of Kombu over the short term resulted in a significant increase in serum TSH with a slight decrease in serum FT$_4$ and/or FT$_3$ concentrations. During ingestion of Kombu for long term, serum TSH levels were elevated and sustained but FT$_4$ and FT$_3$ concentrations were almost within normal limits. FT$_4$ and FT$_3$ concentrations were influenced by various factors such as the degrees of inhibitory effect of iodine on thyroid function, increase of TSH by the negative feedback mechanism, responsiveness and compensatory enlargement of thyroid gland to TSH. The inhibitory action of excess iodine (acute Wolff-Chaikoff effect) [15] is known to be temporary (escape from the Wolff-Chaikoff effect [16]). However, the inhibitory effect of Kombu ingestion in the present study persisted for at least 3 months. These findings indicate that thyroid function in normal Japanese adults was suppressed during ingestion of Kombu, although the suppression was slight and reversible.

It is well known that Japan is an iodine-rich areas probably because of seaweed ingestion. Urinary iodine excretion by normal Japanese has been reported to be as high as 0.06 to 9.3 mg per day [9, 10, 17–19] compared to 0.03 to 0.7 mg by people from other countries [9]. Furthermore, various cases of hypothyroidism caused by excess iodine ingestion have been reported, such as endemic goiter in Hokkaido where Kombu is routinely eaten with meals [20], and newborn babies with transient hypothyroidism or persistent hyperthyrotropinemia born to mothers who consume excessive iodine [21], a woman with hypothyroidism and anorexia nervosa who took iodine-rich diet [22], a woman with hypothyroidism caused by drinking “Ne-Kombu” water in which roots of Kombu have been dipped, as a folk remedy for hypertension [23], a boy with growth retardation due to hypothyroidism caused by eating Kombu everyday as a snack [24] etc. Recently, Kasagi et al. [25] reported that one third of cases of consecutive hypothyroidism in Japan became euthyroid after iodine restriction.

Thus the Ministry of Health, Labour, and Welfare, Japan recommended an upper intake level of iodine as
3 mg per day [26]. The amount of 15–30 g of Kombu used in the present study seems to be a lot but it is the amount normally eaten during meal for many Japanese people who favor seaweed. The absorbed iodine (20–50 mg) estimated by urinary excretion significantly exceeded the recommended upper intake level (3 mg). Therefore, we want to warn Japanese people against consuming excessive amounts of seaweed, which are promoted as healthy foods.

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References


