Although iodine is an essential trace element in humans and plays an important role in thyroid hormones, an excessive intake of iodine causes hypothyroidism (1). In Japan, the average iodine intake is estimated to be higher than the tolerable upper limit value established by the US or European dietary reference intakes (2–4); however, there have been no health problems observed to be caused by this high iodine intake. High iodine intake in Japan is a result of routine consumption of several kinds of edible algae. Among these, some types of brown algae (Laminariaceae sp. and Saccharina sp.) called “kombu” contain high iodine levels of more than 2,000 mg/kg when dried (5), and serve as the largest contributor of iodine intake in the Japanese (6). However, little is known about the absorption of iodine from kombu. In the present study, serum and tissue iodine concentrations were measured in rats fed a diet supplemented with powdered kombu (Saccharina sculpens) or potassium iodide to evaluate the absorption of iodine from kombu. Eighteen male 5-wk-old Wistar rats were divided into three groups and fed a basal AIN93G diet (iodine content, 0.2 mg/kg) or the basal diet supplemented with iodine (183 mg/kg) either in the form of kombu powder or potassium iodine (KI) for 4 wk. There were no differences in weight gain or serum biochemistry tests (alanine aminotransferase and aspartate aminotransferase activity, and total serum cholesterol and triglyceride concentration) after iodine supplementation. In addition, serum levels of the thyroid hormones thyroxine and triiodothyronine, as well as thyroid-stimulating hormone, were not affected. On the other hand, serum and tissue (thyroid, liver and kidney) iodine concentrations were markedly elevated after iodine supplementation. There was no difference in thyroid iodine concentration between KI and kombu supplementation. However, there was a significant difference observed in the iodine concentrations of serum, liver and kidney between the two iodine sources; rats fed KI had iodine concentrations in these tissues 1.8 to 1.9 times higher than those in rats fed kombu powder. These results suggest that the absorption of iodine from kombu is reduced compared to that from potassium iodide.

Materials and Methods

Kombu sample. A dried edible brown algae (Saccharina sculpers; Japanese name, “Gagome kombu”) was purchased in a local retail shop in Osaka, Japan, and was ground into a powder with a mill (Grindomix GM200, Retsch, Haan, Germany). The iodine content of the kombu sample was 3,655 mg/kg. Although this is higher than values (2,100–2,400 mg/kg) listed in the Standard Tables of Food Composition in Japan 2010 (7), it is within the range of the iodine concentration of the several kinds of kombu measured at the authors’ laboratory (2,420–5,600 mg/kg) (8).

Animals and diets. The experimental protocol was reviewed and approved by the Animal Ethics Committee of Kansai Medical University and followed the “Guide for the Care and Use of Experimental Animals” established by the Prime Minister’s Office of Japan. Experimental animals were housed in plastic cages in a room under a controlled 12 h light (8:00 to 20:00) and dark cycle at a temperature of 23 ± 2°C and humidity of 50 ± 10%. The animals were given the experimental diets and deionized water ad libitum during the entire experimental period.

Eighteen male 5-wk-old Wistar rats (Shimizu Lab. Supplies Co. Ltd., Kyoto, Japan; body weight mean ± SE: 121 ± 3 g) were divided into three groups and fed the experimental diets for 4 wk. The first group (control group) was fed a casein-based semi-purified basal diet (AIN93G diet (9)). The second group (kombu group) was fed a diet containing 5% dried kombu powder; this
The digest was diluted with water to 10 mL and filtered with methylammonium hydroxide (TMAH) at 90˚C for 3 h.

Liver and kidney were digested with 1 mL of 5% tetra-

minescence enzyme immunoassay.

Diagnostics Inc., Munich, Germany) utilizing a chemilu-

were measured by commercial kits (Siemens Healthcare

Gifu, Japan) and the following hormone assays. The

by a commercial service (BioGate Co. Ltd., Yamagata-

plastic cage, the amount of feed intake was measured

feeding period, each rat was individually put into the

was 0.2 and 183 mg/kg diet, respectively. During the

exposed of 183 mg/d in rats

were significantly different (p<0.05). These statistical

test for 4 mo at a level of 465 μg/d exhibited

Statistics. Experimental data were assessed by one-

way analysis of variance. When the F value was significant (p<0.05), the Tukey-Kramer multiple range test

were performed on a personal computer (eMac; Apple Computer, Cupertino, CA) with the Mac OS 9.2

operating system and statistical program package Stat-

View-J version 5.0 (Abacus Concept, Berkeley, CA).

Results and Discussion

During the 28 d feeding period, total food intake (mean ±SE, n=6) was 510 ±15, 552 ±17 and 529 ±18 g in the control, kombu and KI groups, respectively; daily iodine intake was 3.6, 3.607 and 3.457 μg/d, respectively. No significant differences were observed in body weight or animal growth among the groups. At the end of the experimental period, the body weight of each group (mean ±SE, n=6) was as follows: control group, 244 ±4; kombu group, 250 ±5; KI group, 244 ±7 g. Similarly, kombu or potassium iodide supplementation induced no changes in liver and kidney weights or serum TG, TCHO, ALT or AST level (data not shown). These results indicated that administration of a large amount of iodine at a level of more than 3 mg/d in rats for 4 wk produced no abnormalities in appetite, growth, lipid metabolism or liver function.

Table 1. Tissue iodine concentration in rats fed experimental diets.

<table>
<thead>
<tr>
<th>Experimental group</th>
<th>Serum (μg/mL)</th>
<th>Liver (μg/g)</th>
<th>Kidney (μg/g)</th>
<th>Thyroid (μg/g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0.10 ± 0.01</td>
<td>0.13 ± 0.01</td>
<td>0.09 ± 0.01</td>
<td>108 ± 15</td>
</tr>
<tr>
<td>Kombu</td>
<td>4.80 ± 0.27</td>
<td>1.62 ± 0.09</td>
<td>2.09 ± 0.11</td>
<td>440 ± 47</td>
</tr>
<tr>
<td>KI</td>
<td>8.76 ± 0.25</td>
<td>2.99 ± 0.15</td>
<td>3.82 ± 0.16</td>
<td>412 ± 30</td>
</tr>
</tbody>
</table>

Values are means ± SE (n=6). Means in the same column not sharing a common superscript differ significantly (p<0.05).

Statistics. Experimental data were assessed by one-way analysis of variance. When the F value was significant (p<0.05), the Tukey-Kramer multiple range test was performed to determine which pairs of the means were significantly different (p<0.05). These statistical tests were performed on a personal computer (eMac; Apple Computer, Cupertino, CA) with the Mac OS 9.2 operating system and statistical program package StatView-J version 5.0 (Abacus Concept, Berkeley, CA).

Results and Discussion

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Table 1 shows the thyroid weight and serum thyroid hormone concentration of rats in each group. No differences were observed in thyroid weight or serum thy-
roid hormone concentration among the experimental groups. In addition, the serum TSH concentration was below the detection limit (<0.1 μIU/mL) in all rats. These results indicate that the thyroid function in rats was not affected by dietary iodine exposure of 183 mg/kg diet.

In a previous study by Gao et al., dietary iodine exposure for 4 mo at a level of 465 μg/d caused hyperthyroid-
Comparison of Absorption of Kombu Iodine and Potassium Iodide

ism and the exposure for 6 mo caused hypothyroidism in rats (10). In the present study, the iodine dose (about 3,500 μg/d) was higher than the dose used in the previous study; it is possible that the dosing period, rather than the dosing level, may be more critical in changing the thyroid function due to excess iodine intake. On the other hand, in some human studies, approximately 30 mg/d of iodine intake caused hypothyroidism within 3 wk (11, 12). Since the dry weight of the adult human diet is about 500 g/d, dosing of 30 mg/d corresponds to a dietary level of about 60 mg/kg; if a human were to intake iodine at the dietary level used in the present study (183 mg/kg diet), hypothyroidism should be caused within 1 mo. Thus, rats may have a lower susceptibility to high iodine intake than humans.

Table 2 shows the iodine concentration in the serum, liver, kidney and thyroid of rats fed the experimental diets. Rats fed the iodine-supplemented diets showed remarkably higher tissue iodine concentrations than rats fed the control diet. There were significant differences in the iodine concentration of serum, liver and kidney between the kombu and KI groups; iodine concentration in these tissues in the KI group was 1.8 to 1.9 times higher than those in the kombu group. Although a recent study showed that more than 95% of the iodine was in the form of the iodide ion in kombu, and a large part of the iodide was easily released by porcine pancreatic enzymes in vitro (13), the present results suggest that absorption of iodine from kombu is less than that from KI.

There was no difference in thyroid iodine concentration between the KI and kombu groups, suggesting that iodine concentration is strictly regulated in the thyroid; therefore, the excess iodine intake did not affect the thyroid function.

The causal factor underlying the inconsistency in the absorption of kombu iodine between the present in vivo study and previous in vitro studies (13) is unclear. Kombu contains sodium and potassium at a high level. Based on the Standard Tables of Food Composition in Japan 2010 (7) and the composition of the AIN93G diet (9), the sodium and potassium contents of the diet of each group is as follows: control and KI groups, 1.019 mg/kg and 3.600 mg/kg; kombu group, 2.519 mg/kg and 6.450 mg/kg diet, respectively. That is, it means that the kombu group had taken in almost twice the amounts of sodium and potassium as compared with other groups. Although not measured, it can expect that the high intake of sodium and potassium caused an increase in the amount of drinking water and urine. The increase in urine amounts may have accelerated urinary excretion of iodine; we can consider the possibility of its having resulted in a low retention of iodine in the kombu group.

While it is still unknown why the large intake of iodine from kombu does not cause serious health problems in Japan, the present results suggest that the lower absorption of iodine from kombu, which may be less toxic than the inorganic iodide, may be a reason the possible adverse effects are infrequent. Thus, further study is necessary to clarify the digestibility and absorption of iodine from edible algae including kombu as well as the effect of high intakes of sodium and/or potassium on iodine retention.

REFERENCES


