Thyroid function in rats with iodine deficiency is not further impaired by concurrent, marginal zinc deficiency

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The hypothesis tested was that Zn deficiency aggravates impaired thyroid function as induced by I deficiency. In two separate experiments male rats were fed on diets either deficient in Zn or in I, or deficient in both. An identical, restricted amount of food was given to each rat so that body-weight gain of the experimental groups was comparable. Zn deficiency was evidenced by reduced tibiaal Zn concentrations. I deficiency was evidenced by goitre, reduced urinary I excretion, reduced plasma thyroxine concentrations and reduced absolute amounts and concentrations of thyroxine in the thyroid. Zn deficiency had no effect on the raised thyroid weight as induced by I deficiency. Zn restriction from 184 μmol Zn/kg diet to 31 μmol Zn/kg diet, but not to 92 μmol Zn/kg diet, significantly lowered plasma thyroxine concentration. There were no interrelated effects of Zn and I deficiencies on thyroid hormone levels. These results indicate that marginal Zn deficiency does not influence thyroid hormone metabolism in I deficiency.

Rats: Zinc: Iodine: Thyroid

I deficiency is the major cause of disorders such as goitre and cretinism (Hetzel, 1988). Simultaneous deficiency of either retinol (Morley et al. 1978; Obα & Kimura, 1980; Nockels et al. 1984) or Se (Vanderpas et al. 1990) may aggravate the symptoms of I deficiency. Perhaps this also occurs in concurrent Zn deficiency. Sandstead et al. (1967) reported an unexpectedly high incidence of goitre in children with dwarfism caused by Zn deficiency. Furthermore, Wada & King (1986) found in young men a significant lowering of free serum thyroxine (T₄) levels after a period of low Zn intake. In rats Zn deficiency causes reduced triiodothyronine (T₃) levels in serum (Morley et al. 1980) and raised activities of hepatic thyroxine 5’ monodeiodinase (EC 3.8.1.4; Oliver et al. 1987).

There may be an interaction of I and Zn deficiencies with regard to thyroid function. The present study with rats was undertaken to test this hypothesis. We have attempted to use a model suitable for elucidating an interaction between Zn and I intakes in humans in areas where marginal Zn and/or I intake is a problem. Thus, we have induced in rats marginal Zn and I deficiencies without clinical signs.

MATERIALS AND METHODS

Animals and housing

Two separate experiments were performed. The experimental protocols were approved and their execution supervised by the animal welfare officer of the Wageningen Agricultural University. Male, weanling Wistar (Cpb:WU) rats, aged 21 d, were used in both
experiments. They were purchased from Harlan/Cpb, Zeist, The Netherlands. On arrival the rats were housed in groups of five animals in cages with wire-mesh bases (600 x 420 x 190 mm). After 14 d they were individually housed in metabolism cages (3.14 m² x 120 mm) with polycarbonate tops and stainless-steel wire-mesh bases. The cages were placed in a room with controlled ventilation (twenty air changes/h), relative humidity (45–65%), room temperature (20–22°C) and lighting (light on: 06.00–18.00 h).

Diets and feeding

From arrival, all animals were fed ad lib. on the purified control diet for another 14 d. This diet was Zn and I sufficient (184 μmol added Zn/kg; 1.2 μmol added I/kg). The composition of the diet was as follows (g/kg): ovalbumin 151, maize oil 25, coconut fat 25, glucose 709-4, cellulose 30, CaCO₃ 12-4, NaH₂PO₄·2H₂O 15-1, MgCO₃ 1-4, KCl 1-0, KHCO₃ 7-7, KIO₃ 0-25 mg, ZnSO₄·H₂O 33 mg, I and Zn-free mineral premix 10, vitamin premix 12. The composition of the mineral and vitamin premix has been reported elsewhere (Grooten et al. 1991). After the pre-experimental period (day 0 of the experiment), the rats were divided into four groups of twelve rats each and one group of six rats. In both Expts 1 and 2 one group of twelve rats and the group of six rats remained on the control diet. The other groups received a Zn-sufficient I-deficient diet, a Zn-deficient I-sufficient diet or a Zn-deficient I-deficient diet. To prepare the I-deficient diets, KIO₃ was omitted from the control diet. To prepare the Zn-deficient diets, part of the added ZnSO₄·H₂O was removed. In Expt 1 the Zn-deficient diets contained 31 μmol added Zn/kg instead of 184 μmol Zn/kg. In Expt 2 the Zn-deficient diets contained 92 μmol added Zn/kg. Analysis showed that the control diet contained 200 and 182 μmol Zn/kg in Expts 1 and 2. The analysed Zn concentrations of the other diets were as follows: Zn-sufficient I-deficient diets, 194 and 214 μmol/kg in Expts 1 and 2; Zn-deficient I-sufficient diets, 43 and 89 μmol/kg in Expts 1 and 2; Zn-deficient I-deficient diets, 43 and 90 μmol/kg in Expts 1 and 2.

Because Zn deficiency is known to cause anorexia (Swenerton & Hurley, 1967; Chesters & Quarterman, 1970), the four experimental groups in each experiment were given an identical, restricted amount of food so that body-weight gain would be comparable. The group of six rats was given the control diet ad lib. The experimental groups received 60% of ad lib. intake. Food was administered daily. The diets were in powdered form and stored at 4°C until required for feeding. Separate batches of diet were made for the two experiments. Twice-distilled water was freely available from polycarbonate bottles with stainless-steel mouthpieces.

Experimental procedures

Each experiment lasted 42 d. During the first 3 weeks the rats were weighed once weekly and during the last 3 weeks twice weekly. Food intake was recorded every 2 d. Urine and faeces were collected quantitatively from each rat during the last 3 d of week 6 of each experiment. In Expt 1 urine was also collected for 3 d in weeks 2 and 4.

In Expt 2 a clinical examination of the rats was carried out by two independent assessors who were unaware of treatment modality. Rats were examined in random order. An apparently healthy rat fed on a commercial, pelleted diet (RMH-B®, Hope Farms, Woerden, The Netherlands) served as reference. The following variables were scored: discharge from nose and eyes, condition of hair-coat, condition of skin on trunk, tail and paws, colour of tail and teeth divergence. The variables were scored on a scale from 0 (similar to reference rat) to 3 (extremely different from reference rat). One assessor examined the rats on day 40 and the other on day 41. This method of clinical examination has been detailed elsewhere (Beynen et al. 1987).
At the end of each experiment blood was taken by orbital puncture, while animals were under diethyl ether anaesthesia, and collected in heparinized tubes. The anaesthetized rats were subsequently killed by CO₂ inhalation. Thyroid gland, tibia and testes were carefully removed. The glands were immediately weighed. Plasma, thyroid gland and tibia samples were frozen at −18° until analysis.

Plasma samples were analysed for T₄ and T₃ by radioimmunoassay (RIA) without previous extraction according to the method of Larsen (1972) with minor modifications as described by Van Hardeveld & Kassenaar (1976). The thyroid was homogenized and T₄ and T₃ were extracted according to the method for thyroglobulin hydrolysis as described by Rolland et al. (1970), and determined as described previously. Thyroid-stimulating hormone (TSH) was measured by the specific RIA for rat TSH as developed by the National Institute of Diabetes and Digestive and Kidney Diseases (National Institutes of Health, Bethesda, MD, USA). Reference preparation RP-2 was used as TSH standard.

Diet samples, tibiae and faeces were dry ashed at 500°C for 17 h. The ash was dissolved in 6 mol HCl/l and Zn was determined by flame atomic absorption spectrophotometry with the use of a Varian AA-475 (Varian Techtron, Springvale, Australia).

I and creatinine in urine were determined as described by Kolthoff & Sandell (1936) and Folin (1914).

Statistics
For each experiment the data from the four groups fed on a restricted basis were subjected to two-way analysis of variance (ANOVA) to disclose I and Zn effects and their interaction. P < 0.05 was pre-set as the criterion of statistical significance. Differences between two groups with one dietary variable (CONTRAST) were evaluated with Student's t test. To take into account the increasing risk of a type I error due to multiple comparisons, Bonferroni's adaptation of the P value was applied. All analyses were done using the SPSS PC+ computer program (SPSS Inc., 1986).

RESULTS

Weight gain
Initial body weights (day 0 of the experiment) in Expts 1 and 2 were 125.1 (SE 1.3) and 131.4 (SE 1.4) g (n 54). Feed intake of the control groups fed ad lib. was 18.9 (SE 0.5) and 19.1 (SE 0.6) g/d (n 6) in Expts 1 and 2. Final body weights of the experimental groups in Expts 1 and 2 are shown in Table 1. Restricting feed intake to 60% of ad lib. intake had reduced body weight by about 40% at the end of the two experiments. In Expt 1, restricted feeding for 42 d of the Zn-deficient diets with 31 µmol added Zn/kg diet lowered group mean body weight by 6% when compared with restricted feeding of either the control or I-deficient diet. In the course of Expt 2, when the Zn-deficient diets contained 92 µmol added Zn/kg, body-weight gain of the four groups fed restrictedly were superimposable (Table 1); no clinical abnormalities were detected.

Zinc status
The Zn-deficient diets produced significantly reduced tibial Zn concentrations, the effect being greater in Expt 1 than in Expt 2 (Table 1). Relative testes weight was lower in Zn-deficient than in Zn-sufficient rats in Expt 1. This was not seen in Expt 2 when the Zn-deficient diets contained more Zn than in Expt 1. The amount of I in the diet had no effect on the indicators of Zn status. Ad lib. v. restricted feeding of the control diet did not affect the tibial Zn concentration and lowered relative testes weight (Table 1).
Table 1. **Body weight and indicators of zinc status in rats fed on the experimental diets**

(Results are means with pooled standard errors for twelve animals per group for rats fed restrictedly and for six animals per group for rats fed *ad lib.*).

| Feeding regimen... | Restricted |  |  | Ad lib. control | Effects of  
| Diet*... | Control | I-deficient | Zn-deficient | I+Zn-deficient | Pooled se | I v. Zn v.  
<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<th>Mean</th>
<th>se</th>
<th>I+Zn deficiency (ANOVA)†</th>
<th>CONTRAST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body wt (g)</td>
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<td></td>
<td></td>
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</tr>
<tr>
<td>Expt: 1</td>
<td>206.1</td>
<td>205.7</td>
<td>193.9</td>
<td>194.7</td>
<td>2.7</td>
<td>334.7</td>
<td>9.8</td>
<td>Zn c, d</td>
</tr>
<tr>
<td>2</td>
<td>217.5</td>
<td>218.3</td>
<td>220.2</td>
<td>218.8</td>
<td>2.8</td>
<td>356.0</td>
<td>10.2</td>
<td>—</td>
</tr>
<tr>
<td>Tibial Zn (μmol/g dry wt)</td>
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<td></td>
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<td></td>
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</tr>
<tr>
<td>Expt: 1</td>
<td>3.20</td>
<td>3.21</td>
<td>1.43</td>
<td>1.51</td>
<td>0.04</td>
<td>3.15</td>
<td>0.08</td>
<td>Zn c, d</td>
</tr>
<tr>
<td>2</td>
<td>3.21</td>
<td>3.18</td>
<td>2.18</td>
<td>2.19</td>
<td>0.05</td>
<td>3.26</td>
<td>0.06</td>
<td>Zn c, d</td>
</tr>
<tr>
<td>Testes wt (g/kg body wt)</td>
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<td></td>
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<tr>
<td>Expt: 1</td>
<td>14.4</td>
<td>14.8</td>
<td>9.4</td>
<td>8.3</td>
<td>0.5</td>
<td>9.3</td>
<td>0.2</td>
<td>Zn c, d, e</td>
</tr>
<tr>
<td>2</td>
<td>14.8</td>
<td>14.6</td>
<td>14.4</td>
<td>15.0</td>
<td>0.4</td>
<td>9.6</td>
<td>0.4</td>
<td>—</td>
</tr>
</tbody>
</table>

The following comparisons were found to be significantly different by CONTRAST: c, Zn deficient v. Zn sufficient when I was sufficient (*P < 0.017*); d, Zn deficient v. Zn sufficient when I was deficient (*P < 0.025*); e, *ad lib.* v. restricted feeding when I and Zn were sufficient (*P < 0.017*).

* The control diets in the two experiments contained 1.2 μmol added I and 184 μmol added Zn/kg. The I-deficient diets did not contain added I. The Zn-deficient diets contained either 31 μmol (Expt 1) or 92 μmol (Expt 2) added Zn/kg.

† The ANOVA effect of Zn deficiency was significant (*P < 0.05*) as indicated.
Table 2. *Indicators of iodine status in rats fed on the experimental diets*  
(Results are means with pooled standard errors for twelve animals per group for rats fed restrictedly and for six animals per group for rats fed *ad lib.*).

| Feeding regimen… | | | | | | \hline
<table>
<thead>
<tr>
<th>Diet*</th>
<th></th>
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<th></th>
<th></th>
<th>Restricted</th>
<th>Ad lib. control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinary I (µmol I/mol creatinine)</td>
<td>Control</td>
<td>I-deficient</td>
<td>Zn-deficient</td>
<td>I+Zn-deficient</td>
<td>Pooled se</td>
<td>Mean</td>
<td>se</td>
</tr>
<tr>
<td>Expt: 1</td>
<td>317.6</td>
<td>87.3</td>
<td>323.0</td>
<td>98.1</td>
<td>16.4</td>
<td>288.2</td>
<td>22.0</td>
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<tr>
<td>2</td>
<td>310.2</td>
<td>67.1</td>
<td>345.4</td>
<td>60.6</td>
<td>16.3</td>
<td>231.7</td>
<td>23.9</td>
</tr>
<tr>
<td>Thyroid wt (mg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expt: 1</td>
<td>16.1</td>
<td>19.5</td>
<td>14.2</td>
<td>19.5</td>
<td>0.8</td>
<td>22.9</td>
<td>0.6</td>
</tr>
<tr>
<td>2</td>
<td>18.4</td>
<td>22.9</td>
<td>16.2</td>
<td>22.8</td>
<td>1.0</td>
<td>24.3</td>
<td>0.5</td>
</tr>
<tr>
<td>Plasma T₄ (nmol/l)</td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Expt: 1</td>
<td>86.5</td>
<td>62.4</td>
<td>68.3</td>
<td>56.4</td>
<td>4.3</td>
<td>86.8</td>
<td>5.7</td>
</tr>
<tr>
<td>2</td>
<td>68.6</td>
<td>60.0</td>
<td>71.1</td>
<td>52.8</td>
<td>3.7</td>
<td>81.5</td>
<td>4.4</td>
</tr>
<tr>
<td>Plasma T₃ (nmol/l)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expt: 1</td>
<td>1.28</td>
<td>1.30</td>
<td>1.16</td>
<td>1.27</td>
<td>0.08</td>
<td>1.50</td>
<td>0.12</td>
</tr>
<tr>
<td>2</td>
<td>1.06</td>
<td>1.31</td>
<td>1.10</td>
<td>1.22</td>
<td>0.07</td>
<td>1.38</td>
<td>0.12</td>
</tr>
<tr>
<td>Thyroid T₄ (nmol T₄/mg thyroid)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expt: 1</td>
<td>15.9</td>
<td>9.2</td>
<td>17.8</td>
<td>8.0</td>
<td>1.2</td>
<td>15.3</td>
<td>1.6</td>
</tr>
<tr>
<td>2</td>
<td>19.0</td>
<td>9.6</td>
<td>20.1</td>
<td>8.1</td>
<td>1.0</td>
<td>17.9</td>
<td>1.4</td>
</tr>
<tr>
<td>TSH (nmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expt: 1</td>
<td>0.33</td>
<td>0.45</td>
<td>0.39</td>
<td>0.66</td>
<td>0.07</td>
<td>0.43</td>
<td>0.05</td>
</tr>
<tr>
<td>2</td>
<td>0.24</td>
<td>0.68</td>
<td>0.34</td>
<td>0.67</td>
<td>0.09</td>
<td>0.48</td>
<td>0.02</td>
</tr>
</tbody>
</table>

**Notes:**
- T₄, thyroxine; T₃, triiodothyronine; TSH, thyroid-stimulating hormone.
- The following comparisons were found to be significantly different by CONTRAST: a, I-deficient v. I-sufficient when Zn was sufficient (*P* < 0.017); b, I-deficient v. I-sufficient when Zn was deficient (*P* < 0.025); c, Zn-deficient v. Zn-sufficient when I was sufficient (*P* < 0.017); e, *ad lib.* v. restricted feeding when I and Zn were sufficient (*P* < 0.017).
- The control diets in the two experiments contained 1.2 µmol added I and 184 µmol added Zn/kg. The I-deficient diets did not contain added I. The Zn-deficient diets contained either 31 µmol (Expt 1) or 92 µmol (Expt 2) added Zn/kg.
- The ANOVA effects of I and Zn deficiency were significant as indicated.
Iodine status

The diets without added KIO₃ clearly lowered the excretion of I in urine (Table 2); this effect tended to be somewhat more marked in Expt 2 than in Expt 1. Zn deficiency did not affect I:creatinine in urine. In Expt 1 the ratios were also determined after 2 and 4 weeks, the values being 128.8 (± 6.6) and 96.7 (± 5.0) μmol I/mol creatinine for the I-deficient rats (n = 24). I deficiency significantly raised group mean thyroid weight in both experiments by on average 30%. Relative thyroid weight, also, was elevated significantly by I restriction (results not shown). There was no interaction of I and Zn deficiency with regard to thyroid weight.

Ad lib. v. restricted feeding of the control diet did not alter I:creatinine in urine. In rats fed ad lib., absolute group mean thyroid weight was somewhat higher (Table 2) and relative weight lower (not shown). The latter was due to the higher amount of body fat in rats fed ad lib. instead of restrictedly; the difference in body fat was observed clearly at autopsy.

The I-deficient diets consistently lowered T₄ concentrations in plasma and thyroid (Table 2). I deficiency also significantly reduced the amount of thyroid T₄ when expressed per whole thyroid (results not shown). In Expt 1 Zn deficiency also reduced plasma T₄; this was not seen in Expt 2 when Zn deficiency was less pronounced. Zn deficiency did not influence thyroid T₄ concentration. T₃ concentration in plasma was significantly raised by I deficiency in Expt 2. In Expt 1 this I effect was not found. Concerning T₃ and T₄ concentrations in plasma, there were no interactions between I and Zn deficiency. TSH levels were significantly elevated in I-deficient rats. The amount of Zn in the diet did not affect plasma TSH concentrations.

The rats given the restricted amount of the control diet had thyroid T₄ concentrations similar to that of their counterparts fed ad lib. In Expt 2, but not in Expt 1, T₄ concentrations in plasma tended to be lower in rats fed on a restricted basis. Rats fed on restricted amounts of the control diet had lower group mean T₃ concentrations than those fed ad lib. Restricted feeding lowered group mean TSH levels significantly in Expt 2. This was not seen in Expt 1 (Table 2).

DISCUSSION

The lowered tibial Zn levels in rats fed on the low-Zn diets indicate that the animals were indeed Zn deficient. As would be expected, in Expt 1 using diets containing 31 μmol added Zn/kg, tibial Zn concentrations were lower than those in Expt 2 using diets with 92 μmol added Zn/kg. The control group fed on restricted amounts of feed had tibial Zn concentrations similar to those of the control group fed ad lib. This suggests that restricted feeding per se had no impact on Zn status of the rats. The observed lowering effect of Zn deficiency on testes weight in Expt 1 has been reported earlier (Hurley, 1969; Giugliano & Millward, 1984). In Expt 2 this was not seen, indicating that 92 μmol added Zn/kg diet is sufficient for testes development despite the fact that it lowered tibial Zn concentration. In Expt 2 the rats were examined clinically, but no abnormalities could be detected. This also indicates that the rats fed on the diets with 92 μmol added Zn/kg in Expt 2 had a marginal Zn status without clinical signs.

In Expt 1, using diets with only 31 μmol added Zn/kg, the Zn-deficient animals had significantly lower body weights than their counterparts that were also fed restrictedly (Table 1). Thus, in Expt 1, non-specific Zn effects related to growth retardation cannot be excluded. Therefore, we repeated the experiment with low-Zn diets containing 92 μmol added Zn/kg instead of 31 μmol added Zn/kg.

To monitor I status of the rats, urinary I:creatinine ratios were determined. After 6 weeks the ratio was lowered by about 75% in the rats fed on diets without added I. In Expt
the reduction was somewhat greater than that in Expt 1. Based on the time-course of the I:creatinine ratio in urine, it can be concluded that the I-deficient status was reached gradually. Restricted feeding per se did not influence urinary I:creatinine. The control groups fed either ad lib. or restrictedly had similar ratios. Thus, the rats fed on restricted amounts of the diets supplemented with 1.2 μmol I/kg can be considered I sufficient. I intake of rats fed ad lib. was on average 0.02 μmol I/d, and that of rats fed restrictedly was 0.01 μmol/d. Apparently, this difference in I intake did not affect I status.

I deficiency raised thyroid weight and reduced thyroid T₄ in both experiments which was accompanied by lower plasma T₄ levels. This can be explained by a lowered T₄ synthesis and/or increased T₃ synthesis. Monodeiodination of T₄ to T₃ might also be enhanced (Laurberg, 1984). As would be anticipated (Schröder-Van der Elst, 1991), I deficiency raised plasma TSH concentrations.

Restricted feeding v. ad lib. feeding raised relative thyroid weight, which relates to the lower degree of adiposity in restrictedly-fed animals. Restricted feeding had no significant effect on plasma concentrations of T₄ and T₃. However, plasma T₄ may be lowered by more extreme restriction of feed intake (Schröder-Van der Elst & Van der Heide, 1992). Restricted feeding did not alter thyroid T₄ concentrations. In Expt 2 plasma TSH concentrations were significantly lower in animals fed on restricted amounts of the control diet than in their counterparts given the control diet ad lib. This was not seen in Expt 1. Thus, in essence, the animals fed on a restricted basis did not differ much from those fed ad lib. concerning thyroid function.

In Expt 1 Zn deficiency did not affect thyroid T₄ content, but significantly lowered plasma T₃. Possibly, T₄ release from the thyroid is impaired by Zn deficiency or T₄ monodeiodination into T₃ is enhanced. The latter possibility is supported by results of Oliver et al. (1987) who found an elevated activity of hepatic 5'-monodeiodination in Zn-deficient rats. The suggestion by these authors that this leads to a higher level of T₃ in plasma is not confirmed by our findings. Moreover, Morley et al. (1980) reported that Zn deficiency in rats lowers plasma T₃. Such a tendency was seen in Expt 1, but only when the diet was I sufficient. In Expt 2 there were no effects of Zn deficiency on plasma T₄, which most probably relates to the lesser degree of Zn deficiency. In both experiments there was no interactive effect of I and Zn deficiency on thyroid T₄ concentration and plasma T₄ and T₃ concentrations.

Inconsistent effects of Zn deficiency on plasma TSH levels have been found by various workers (Root et al. 1979; Morley et al. 1980; Oliver et al. 1987). We observed raised group mean TSH levels in both experiments. Since plasma T₃ in the Zn-deficient group in Expt 1 was lowered, the rise in TSH might be a feedback response to stimulate thyroid hormone production. In Expt 1 there tended to be an interaction between I and Zn deficiencies with respect to plasma TSH: combined I and Zn deficiencies raised group mean plasma TSH in a synergistic manner. However, this was not seen with the lower degree of Zn deficiency in Expt 2.

In summary, under the present experimental conditions I and Zn deficiencies had predictable effects on thyroid function and Zn status respectively. However, there appeared to be no interaction between Zn and I deficiencies with regard to thyroid function as assessed using the measures described.

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REFERENCES


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