GOITROGENIC EFFECTS OF ALLYLISOTHIOCYANATE, NITRATE AND NITRITE IN RATS AND ALLEVIATING PROPERTIES OF IODINE AND SELENIUM SUPPLEMENTS

Renata B. Kostogrys1, Pawel M. Pisulewski2, Anna Pecio2, Agnieszka Filipiak-Florkiewicz3

1Department of Human Nutrition, Faculty of Food Technology, The Agricultural University, Krakow, Poland; 2Department of Comparative Anatomy, Faculty of Biology and Earth Sciences, The Jagiellonian University, Krakow, Poland; 3Małopolska Centre of Food Monitoring and Certification, Faculty of Food Technology, The Agricultural University, Krakow, Poland

Key words: goitrogens, iodine supplementation, selenium supplementation, iodine metabolism, thyroid gland

In Poland, a high level of nitrate and nitrite in food and iodine deficiency have been observed in the last years. The effects of potential goitrogens, namely allylisothiocyanate (SCN-), nitrate (NO3-) and nitrite (NO2-) on growth performance, serum hormones (fT4, TSH) and thyroid morphology were investigated in rats. Simultaneously, the potential antigoitrogenic effects of iodine and selenium supplements were studied.

In experiment 1, male Wistar rats of an initial body weight of 95 g were fed four experimental diets, based on AIN93G diet for rodents, with 0 or 2 µg iodine (KIO3) supplement per rat per day. The diets were: AIN93G - control (C), AIN93G + I (C+I), AIN93G + SCN- (6 mg/100 g body weight) (SCN), AIN93G + SCN- (6 mg/100 g body weight) + I (SCN+I), AIN93G + NaNO2 (300 mg/100 g) (NO3), AIN93G + NaNO2 (300 mg/100 g) + I (NO3+I), AIN93G + NaNO2 (25 mg/100 g) (NO2) and AIN93G + NaNO2 (25 mg/100 g) + I (NO2+I). The diets were fed to eight groups of rats (n=6) for 18 days. Feed intake was restricted to 15 g/day/rat. Body mass of rats was monitored weekly. On day 18, the rats were anaesthetised and their blood was drawn by cardiac puncture. The immulite rat TSH application kit was used to determine TSH concentrations in blood serum. Serum fT4 was compared with the control animals. The only exception were the rats fed NO2+I diet, showing still morphological alterations in their thyroid glands. In conclusion, dietary allylisothiocyanate, nitrate and nitrite impair thyroid metabolism in rats and lead to thyroid hypertrophy. At the same time, the goitrogenic effects of allylisothiocyanate and nitrate can be alleviated by dietary iodine whereas the goitrogenic effects of nitrite can be alleviated only by concomitant dietary supplements of selenium and iodine.

INTRODUCTION

Iodine is a specific substrate for thyroid hormone synthesis. The transport of inorganic iodine to follicular cells is maintained by the iodide pump located in the basolateral membrane; this is the rate-limiting step for thyroid hormone synthesis. A number of anions act as competitive inhibitors of iodide transport in the thyroid, including perchlorate (ClO4-), thiocyanate (SCN-), and pertechnetate (TeO42-). Blockage of the iodine-trapping mechanism has a similar disruptive effect on the thyroid-pituitary axis as iodine deficiency. Nitrates also can interfere with normal iodine thyroid metabolism by inhibiting iodine uptake by the thyroid gland, thus leading to the development of goitre in laboratory animals, e.g. rats [Bloomfield, 1961; Horing et al., 1986; Jahrise et al., 1991] and also in humans [van Maanen et al., 1998; Gatseva & Argirova 2008; Vladeva et al., 2000; Tajtakova et al., 2006]. However, Below et al. [2008] demonstrated that a low alimentary intake of nitrate does not influence the thyroid volume in a population with currently sufficient alimentary intake of iodine [Below et al., 2008]. Nitric oxide donors also inhibit iodide transport and organization in cultured bovine thyroid cells [Costamagna et al., 1998]. Nitrates, in contrast to nitrites, are relatively nontoxic, but an elevated nitrate load may produce potential harmful effects via an endogenous conversion of nitrates to nitrates [Jensen, 1995; Panesar & Chan, 2000].

Selenium occurs in the form of the amino acid selenocysteine in selenoproteins which exert various effects, while maintaining the reduction-oxidation balance in a cell. The discovery that all three deiodinases that convert thyroxine (T4) into triiodothyronine (T3) contain selenocysteine shows how...
the production of the active thyroid hormone is dependent on Se status. The selenoenzyme families of glutathione peroxidases and thioredoxin reductases exhibit powerful antioxidant properties and form a complex defense system that protects thyrocytes against oxidative damage.

The above studies do not provide morphological evidence of goitrogenic effects of allylisothiocyanate (SCN\(^{-}\)) and nitrite (NO\(_2\)) on the thyroid tissue (i.e. thyroid follicles) nor information on the secretion of the thyroid stimulating hormone (TSH) involved in the thyroid metabolism. Additionally, they are lacking information on the potential alleviating effects of iodine or selenium on thyroid metabolism in rats fed the above goitrogens.

Because high levels of nitrate and nitrite in food and iodine deficiency have been observed in Poland, the goitrogenic effect of these substances and alleviating properties of iodine or selenium should be studied.

In view of the above, the objective of the present study (Experiment 1) was to determine the effect of three goitrogen treatments (allylisothiocyanate, nitrate and nitrite) and two levels of iodine supplementation (0 µg iodine (I) supplement or 2 µg iodine (I) supplement (as a potassium iodate)) in rats fed the above goitrogens.

In Experiment 1, male Wistar rats of an initial body weight of 95 g were fed four experimental diets, based on AIN93G diet, with 0 µg iodine (I) supplement or 2 µg iodine (I) supplement (as a potassium iodate) per rat per day. The diets were: AIN-93G- control (C), AIN-93G + I (C+I), AIN-93G + SCN\(-\) (6 mg/100 g body weight) (SCN), AIN-93G + SCN\(-\) (6 mg/100 g body weight) + I (SCN+I), AIN-93G + NaNO\(_3\) (300 mg/100 g) (NO\(_3\)), AIN-93G + NaNO\(_3\) (300 mg/100 g) + I (NO\(_3\)+I), AIN-93G + NaNO\(_2\) (25 mg/100 g) (NO\(_2\)) and AIN-93G + NaNO\(_2\) (25 mg/100 g) + I (NO\(_2\)+I). Table 1A. The levels of goitrogens were chosen on the basis of our previous experiments [Kostogrys et al., 2006a, b]. The diets were fed to eight groups of rats (n=6) for 18 days. Goitrogens and iodine were prepared daily per rat as a water solution and mixed with the diet. Feed intake was restricted to 15 g per rat per day. Body mass of the rats was monitored weekly.

In Experiment 2, male Wistar rats of an initial body weight of 120 g were fed five experimental diets, based on AIN93G rodent diet with 0 µg iodine supplement or 2 µg iodine (I) supplement (as a potassium iodate) per rat per day and with 0 µg selenium supplement or 3.59 µg selenium (Se) supplement (as a Na\(_2\)SeO\(_4\)) per rat per day. The diets were: AIN93G (CON), AIN93G + Se, (Se), AIN-93G + NaNO\(_2\) (25 mg/100 g) (NaNO\(_2\)), AIN-93G + NaNO\(_2\) (25 mg/100 g) + Se (NaNO\(_2\)+Se), AIN-93G + NaNO\(_2\) (25 mg/100 g) + Se + I (NaNO\(_2\)+Se+I), (Table 1B). The diets were fed to five groups of rats (n=6) for 18 days. Goitrogens, selenate and iodine were prepared daily per rat as a water solution and mixed with the diet. Feed intake was restricted to 15 g per rat per day. Body mass of the rats was monitored weekly.

**MATERIAL AND METHODS**

**Animals, housing and feeding**

All experimental procedures complied with the Polish Ethical Standards. Male rats of Wistar strain, approximately five weeks old, were obtained from the Institute of Animal Production in Kraków. They were housed individually in screen-bottomed stainless steel cages, in an isolated room with controlled temperature (25°C) and ambient humidity, with 12-h light-dark cycle. The rats were fed semi-purified AIN-93G diets with complete mineral and vitamin mixture [Reeves, 1993] and had free access to distilled water. All chemicals used in the mineral and vitamin mix were of analytical grade.

In Experiment 1, male Wistar rats of an initial body weight of 95 g were fed four experimental diets, based on AIN93G diet, with 0 µg iodine (I) supplement or 2 µg iodine (I) supplement (as a potassium iodate) per rat per day. The diets were: AIN-93G- control (C), AIN-93G + I (C+I), AIN-93G + SCN\(-\) (6 mg/100 g body weight) (SCN), AIN-93G + SCN\(-\) (6 mg/100 g body weight) + I (SCN+I), AIN-93G + NaNO\(_3\) (300 mg/100 g) (NO\(_3\)), AIN-93G + NaNO\(_3\) (300 mg/100 g) + I (NO\(_3\)+I), AIN-93G + NaNO\(_2\) (25 mg/100 g) (NO\(_2\)) and AIN-93G + NaNO\(_2\) (25 mg/100 g) + I (NO\(_2\)+I). Table 1A. The levels of goitrogens were chosen on the basis of our previous experiments [Kostogrys et al., 2006a, b]. The diets were fed to eight groups of rats (n=6) for 18 days. Goitrogens and iodine were prepared daily per rat as a water solution and mixed with the diet. Feed intake was restricted to 15 g per rat per day. Body mass of the rats was monitored weekly.

In Experiment 2, male Wistar rats of an initial body weight of 120 g were fed five experimental diets, based on AIN93G rodent diet with 0 µg iodine supplement or 2 µg iodine (I) supplement (as a potassium iodate) per rat per day and with 0 µg selenium supplement or 3.59 µg selenium (Se) supplement (as a Na\(_2\)SeO\(_4\)) per rat per day. The diets were: AIN93G (CON), AIN93G + Se, (Se), AIN-93G + NaNO\(_2\) (25 mg/100 g) (NaNO\(_2\)), AIN-93G + NaNO\(_2\) (25 mg/100 g) + Se (NaNO\(_2\)+Se), AIN-93G + NaNO\(_2\) (25 mg/100 g) + Se + I (NaNO\(_2\)+Se+I), (Table 1B). The diets were fed to five groups of rats (n=6) for 18 days. Goitrogens, selenate and iodine were prepared daily per rat as a water solution and mixed with the diet. Feed intake was restricted to 15 g per rat per day. Body mass of the rats was monitored weekly.

**Blood sampling and thyroid gland histological examination**

At the end of Experiments 1 and 2 (18 days) the rats were anaesthetised with thiopental (Biochemie GmbH, Austria; 25 mg/100 g body mass). Blood was rapidly collected by

**TABLE 1A. Composition of experimental diets (%).**

<table>
<thead>
<tr>
<th></th>
<th>C</th>
<th>C+I</th>
<th>SCN</th>
<th>SCN+I</th>
<th>NO(_3)</th>
<th>NO(_3)+I</th>
<th>NO(_2)</th>
<th>NO(_2)+I</th>
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<tr>
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<td>C</td>
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<td>63.3</td>
<td>63.3</td>
<td>63.3</td>
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<td>63.3</td>
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<tr>
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<td>10</td>
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<td>10</td>
</tr>
<tr>
<td>Sucrose</td>
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<td>10</td>
<td>10</td>
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<td>10</td>
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</tr>
<tr>
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<td>7</td>
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</tr>
<tr>
<td>Celulose powder</td>
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<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Mineral mixture</td>
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<td>3.5</td>
<td>3.5</td>
<td>3.5</td>
<td>3.5</td>
<td>3.5</td>
<td>3.5</td>
<td>3.5</td>
</tr>
<tr>
<td>Vitamin mixture</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Choline</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
</tr>
<tr>
<td>Tert-butylhydrochinon</td>
<td>0.0014</td>
<td>0.0014</td>
<td>0.0014</td>
<td>0.0014</td>
<td>0.0014</td>
<td>0.0014</td>
<td>0.0014</td>
<td>0.0014</td>
</tr>
<tr>
<td>Iodine (µg/rat)</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Sodium nitrate (mg/100 g b.w.)</td>
<td>300</td>
<td>300</td>
<td>300</td>
<td>300</td>
<td>300</td>
<td>300</td>
<td>300</td>
<td>300</td>
</tr>
</tbody>
</table>

*AIN-93G mineral mixture, it contains 2 µg iodine/rat; AIN-93G vitamin mixture.
cardiac puncture, transferred to centrifuge tubes with no anticoagulant, and serum was separated by low-speed centrifugation (1500 × g, 15 min). The serum samples were stored at -20°C until analysis. Thyroid glands were carefully excised and fixed in Bouin’s fluid [Kiernan, 1990].

Analyses
Serum free thyroxine (fT4) and serum thyroid stimulating hormone (TSH) concentrations were measured using the lumino-immunoassay LIA-mat F kit (Byk-Sangtec Diagnostica GmbH&Co KG) and The IMMULITE Rat TSH Application kit (DPC Biermann GmbH), respectively.

Thyroid gland histological examination
A part of trachea with the thyroid gland on both sides were removed and fixed in Bouin’s fluid for 3 days. Then the tissues were dehydrated in alcohol, embedded in paraffin and sectioned serially at 7 µm. For histological evaluation the sections were stained with: hematoxylin/eosin and trichrome and colloid were rendered visible by PAS reaction [Kiernan, 1990].

Statistical analysis
The effect of goitrogen treatments was analysed by two-way ANOVA generated by the STATISTICA version 6.1 package (StatSoft, Tulsa, OK.). Where appropriate, treatment means were compared using the Tukey’s multiple range test and p values <0.05 and <0.02 were considered as showing a significant difference between treatment means.

RESULTS

Experiment 1

Body weight
The growth of rats (Figure 1A) was not affected by the potential goitrogens (allyllyoisothiocyanate, nitrate and nitrite). In fact, the growth of rats receiving allyllyoisothiocyanate, nitrate and nitrite, over the period of 18 days, was comparable with that of the control animals, irrespective of supplemental dietary iodine.

TABLE 1B. Composition of experimental diets (%).

<table>
<thead>
<tr>
<th></th>
<th>CON</th>
<th>Se</th>
<th>NaNO₂</th>
<th>NaNO₂+Se</th>
<th>NaNO₂+Se+I</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corn starch</td>
<td>63.3</td>
<td>63.3</td>
<td>63.3</td>
<td>63.3</td>
<td>63.3</td>
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<tr>
<td>Caseine</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Sucrose</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Soybean oil</td>
<td>7</td>
<td>7</td>
<td>7</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Celulose powder</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Mineral mixture</td>
<td>3.5</td>
<td>3.5</td>
<td>3.5</td>
<td>3.5</td>
<td>3.5</td>
</tr>
<tr>
<td>Vitamin mixture</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Choline</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
<td>0.25</td>
</tr>
<tr>
<td>Tert-butylhydroquinon</td>
<td>0.0014</td>
<td>0.0014</td>
<td>0.0014</td>
<td>0.0014</td>
<td>0.0014</td>
</tr>
<tr>
<td>Sodium nitrite (mg/100 g b.w.)</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td>25</td>
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<tr>
<td>Iodine (µg/rat)</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Selenium (µg/100 g b.w.)</td>
<td>3.59</td>
<td>3.59</td>
<td>3.59</td>
<td>3.59</td>
<td>3.59</td>
</tr>
</tbody>
</table>

aAIN-93G mineral mixture, it contains 2 µg iodine/rat; bAIN-93G vitamin mixture; cNa₂SeO₃.

Serum free thyroxine (fT₄) and serum thyroid stimulating hormone (TSH) concentrations
Serum fT₄ concentrations tended to be reduced by allyllyoisothiocyanate, nitrate and nitrite in rats (C – 24.6, SCN – 19.8, NO₃ – 21.8, NO₂ – 21.3 pmol/L, respectively), (Table 2). In contrast, serum TSH levels were significantly increased after administration of SCN (p<0.02) and NO₂ (p<0.05),

FIGURE 1A. The growth of rats from experiment I (g).

FIGURE 1B. The growth of rats from experiment II (g).
At the same time, the rats fed allylthioisocyanate, nitrate and nitrite and receiving iodine supplements (2 µg/day) (SCN+I, NO3+I, NO2+I) showed no changes in serum fT4 and TSH concentrations (Table 2).

**Thyroid follicle histology**

The rats fed allylthioisocyanate (SCN), nitrate (NO3) and nitrite (NO2) showed a series of morphological alterations (Figure 2A and 3A) in their thyroid glands (high follicular epithelial cells and reduced amount of colloid). The height of the epithelial follicle cells was apparently increased in the thyroid gland of rats fed SCN, NO3 and NO2, compared with the control animals (C), thus indicating increased follicle activity (Figure 3A). In addition, mild to moderate irregularity of follicles and decreased amount of colloid were observed in the thyroid glands of rats fed SCN, NO3 and NO2. Moreover, the vascularity of the thyroid tissue from rats fed SCN, NO3 and NO2 was much more developed, compared with the control animals (C), (Figure 2A). Interestingly, the goitrogenic effects of SCN and NO2 could be fully compensated by dietary iodine supplements. Namely, the rats fed SCN+I and NO3+I those receiving iodine supplement (2 µg/d) showed no histological changes in their thyroid glands. In contrast, goitrogenic effects of dietary NO2 in rats could not be alleviated by iodine supplement (2 µg/day). In more detail, in spite of iodine supplementation, the NO2+I rats showed consistent histological changes in their thyroid glands, notably cell hyperplasia and hypertrophy.

**Experiment 2**

**Body weight**

The growth of rats was not affected by nitrite intake (NaNO2) nor by Se (NaNO2+Se) or Se+I supplements (NaNO2+Se+I). In fact, the growth of rats receiving NaNO2 over the period of 18 days was comparable with that of the control animals (CON), irrespective of dietary iodine or selenium (Figure 1B).

**Thyroid follicle morphology**

The histological examination of thyroid glands showed a series of morphological alterations after nitrite administration (NaNO2) (high follicular epithelial cells and reduced amount of colloid). Equally, a series of morphological alterations was observed in thyroid glands of rats fed nitrite, receiving Se supplementation only (NaNO2+Se). In contrast, the rats fed nitrite and receiving simultaneous selenium and iodine supplementation (NaNO2+Se+I) showed similar thyroid morphology to those fed the control diet (CON) (Figure 2B).

**DISCUSSION**

In the present study, no negative effects of goitrogens (allylthioisocyanate, nitrate and nitrite) on body weight of rats were evidenced in Experiment 1. Also, iodine supplementation had no effect on body weight of these animals. The same was true for the effects of nitrite intoxication and the effects of iodine and selenium supplements in Experiment 2. These findings could result from too short experimental periods (18 days), during which the potential toxic effects of goitrogens were not manifested. In contrast to our findings, nitrate intoxication may severely suppress the growth of rats [Chow et al., 1980; Fritsch et al., 1980; Ogur et al., 2000; Zaki et al., 2004]. However, the above experiments were conducted for much longer periods of time (2–14 months). The same effect was reported in studies by Bilczuk [1976], Fritsch et al. [1980] and Chow et al. [1980], but again, these experiments were conducted for 6-14 months. The potential causes of above effects were either a reduction in food and water intake or an increase in protein catabolism or decreased plasma T3 and T4 levels [Zaki et al., 2004].

In the present study, the administration of goitrogens altered thyroid hormonogenesis by decreasing (insignificantly) serum fT4, and increasing (p<0.02 for allylthioisocyanate and p<0.05 for nitrite) serum TSH levels (Table 2). Similarly, Schone et al. [1991] showed that under iodine deficiency conditions, allylthioisocyanate acts negatively on thyroid metabolism by decreasing T3 concentration below detection level. This was also the case for T4 concentration in pigs receiving potassium thiocyanate (a decrease from 1.18 to 0.25 mmol/L) [Schone et al., 1997]. The finding that dietary allylthioisocyanate decreased T3 concentrations can be explained by decreased iodine uptake by the thyroid gland. Langer & Štolc [1965] showed that adult rats (200 g) intoxicated with allylthioisocyanate had a lower thyroid 131I uptake, compared with the control animals. Similar findings were reported by Kahl & Bobek [1971]. In line with the above effects of allylthioisocyanate, nitrate administration in drinking water significantly decreased plasma T3 and T4 levels in rats [Zaki et al., 2004]. In turn, serum fT4 was decreased in rats intoxicated with nitrite [Kostogrysz et al., 2006b]. The above effects of the studied goitrogens may be due to the inhibition of iodine transmembrane transport by a competitive iodine inhibitor (e.g. nitrate) to thyroid epithelial cells. The iodine binding may be blocked by nitrate either indirectly, i.e. by inhibition of Na+/K+ ATPase complex or directly, i.e. by inhibition of sodium-iodide symporter Na+/I– [Chung, 2002; Dohan & Carrasco, 2003].
FIGURE 2A. Thyroid gland from experiment 1 stained with hematoxylin/eosin (A – C group; B – C+I group; C – SCN group; D – SCN+I group; E – NO3 group; F – NO3+I group; G – NO2 group; H – NO2+I group; nk – blood vessels).
both involved in iodine trapping by these cells. The increased serum TSH concentrations, observed in our studies, could be expected. Namely, in a number of experiments, feeding animals with iodine-deficient diets decreased the concentrations of circulating $fT_4$ thyroid hormone and increased the release of TSH from the pituitary gland. Thus, the effects observed in our studies suggest the same negative feedback mechanism, involving the thyroid-pituitary hormonal axis, similar to that produced upon iodine deficiency [Kanno et al., 1992].

Administration of iodine supplement in Experiment 1, alleviated negative effects of allylisothiocyanate, nitrate and nitrite on thyroid hormonogenesis in rats. However, in spite of this, thyroid morphology was negatively affected by nitrite intoxication (see below).

Goitrogen treatments led to changes in thyroid gland morphology in rats, in Experiments 1 and 2 (Figures 2A, 3A, 2B). In fact, allylisothiocyanate, nitrate and nitrite intoxication resulted in both hyperplasia and hypertrophy of the thyroid gland. The height of the epithelial follicle cells was increased, mild to moderate irregularity of follicle was found, and a decrease in the amount of follicular colloid was observed, in the intoxicated animals. These changes were essentially the same as in iodine-deficient animal models. For example, long-term administration of a low iodine diet has been reported to cause follicular hyperplasia and hyper trophy in rats [Kanno et al., 1992], similar to that observed in our studies. Thus, the observed effects suggested the same negative changes in thyroid morphology as produced by io-

FIGURE 2B. Thyroid follicles stained with Pasini’s trichrome (I – CON, II – Se, III – NaNO$_2$, IV – NaNO$_2$ + Se, V – NaNO$_2$ + Se + I).
FIGURE 3A. Thyroid gland from experiment 2 stained with PAS (A – C group; B – C+I group; C – SCN group; D – SCN+I group; E – NO3 group; F – NO3+I group; G – NO2 group; H – NO2+I group; k – colloid).
dine deficiency [Kanno et al., 1992]. Moreover, in fish intoxicated with glucosinolates [Burel et al., 2000], the height of the epithelial follicle cells was decreased (by 200%) and the amount of follicular colloid was decreased, compared with the control animals. Also, Langer & Stolc [1965] found that in iodine-deficient rats, allylisothiocyanate intoxication (2.5 and 5 mg per animal) significantly increased thyroid weight-defined as a goitrogenic effect. This effect of alli-

... well as the level of nitrate intoxication. In view of the above, allylisothiocyanate, nitrate and nitrite may be considered as competitive inhibitors of iodine binding by thyroid gland, thus affecting the thyroid-pituitary hormonal axis and changing thyroid morphology, in a way similar to that of iodine deficiency.

Administration of iodine supplement in Experiment 1, alleviated goitrogenic effects of allylisothiocyanate and nitrate in rats and was ineffective in animals intoxicated with nitrite. Interestingly, the goitrogenic effects of nitrite could be alleviated only by administration of iodine and selenium, as observed in Experiment 2. To offer an explanation, nitrites and nitrites are both oxidation products and ready sources of nitric oxide (NO). NO reacts rapidly with super-

... nitrite. Interestingly, the goitrogenic effects of nitrite intoxication in rats can be alleviated only by concomitant supplementation of iodine and selenium.

CONCLUSIONS

Allylisothiocyanate, nitrate and nitrite have no effect on the growth of rats in 18-day experiments. On the other hand, intoxication of rats with allylisothiocyanate and nitrate im-

... 2009]. Because nitrite is more toxic than nitrate, iodine is not sufficient to allevi-


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ACKNOWLEDGEMENTS

There is no conflict of interest. RBK – research and writing. PMP – comments. AP – research. The study was support-

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22. 517–528 (in Polish)

Received April 2009. Revision received November 2009 and accepted March 2010.