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ORIGINAL CONTRIBUTION

# Effect of iodine source and dose on growth and iodine content in tissue and plasma thyroid hormones in fattening pigs

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**Abstract**
*Purpose* The aim of the present feeding trial with iodine was to assess pigs' growth performance and carcass characteristics, the iodine accumulation in tissues, and their influences on the thyroid hormones in plasma.
*Methods* Eighty pigs (33–115 kg body weight) were allotted to 5 dietary treatments: a control group (150 μg I/kg), two potassium iodide [KI] groups (4,000 and 10,000 μg I/kg), and two potassium iodate [$KIO_3$] groups (4,000 and 10,000 μg I/kg). Iodine concentration was determined in thyroid gland, liver, kidney, muscle, fat, and skin by ICP-MS. Furthermore, thyroxine ($T_4$) and triiodothyronine ($T_3$) in plasma were evaluated.
*Results* High dietary iodine tended to have a negative effect on younger animals' growth (average daily gain, ADG). However, during the entire growth period, the growth performance and carcass characteristics were not influenced by iodine dosages or sources. Irrespective of iodine source, higher iodine doses of diets affected higher iodine stores in all tested tissues except for abdominal fat. Thus, iodine supplementation with 10,000 μg I/kg feed significantly increased iodine content in thyroid gland (+122%), liver (+260%), kidney (+522%), muscle (+131%), and skin (+321%) compared to the control group. However, there was no significance of thyroid hormones in plasma.
*Conclusions* As a result, pork and fat of pigs showed only low iodine accumulation even in the high-iodine groups. Thus, there should be no risk of an iodine excess in human nutrition and animal health, and the EU-upper level for iodine in pig feed can be maintained.

**Keywords** KI · $KIO_3$ · Iodine dosages · Growth performance · Tissue accumulation · Thyroid hormone · Fattening pigs

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## Introduction

Iodine is an essential trace element which is needed for the synthesis of thyroid hormones in humans and animals. The thyroid hormones $T_3$ and $T_4$ have multiple functions as regulators of important metabolic processes like cell activity, growth, and development of brain function. Iodine deficiency disorders (IDD) like goiter, diminished growth, mental retardation, and skeletal deformations occur in case of deficient iodine supply [1]; however, excess iodine intake leads to hypothyroidism and autoimmune thyroiditis in thyroid hormone production [2].

Besides iodized salts, increasing the iodine content of food of animal origin like meat, milk, and eggs by feeding higher doses of different iodine sources has been applied in recent years [3]. Therefore, the iodine supplementation of animal feed is important not only for animals' health, but also for human nutrition.

On the other hand, in 2005, the European Food Safety Authority (EFSA) indicated that maximum permitted dietary content of iodine in livestock feeds should be established in view of safety of the consumer of animal-derived products [3]. According to the high carryover rates of iodine from feed into milk and eggs [4], the maximum permitted iodine content in feed rations to dairy cows and laying hens was reduced to 5,000 μg per kg of complete

feed (standardized at 88% dry matter). In pig nutrition, the maximum permitted dietary content according to the feed law allowed by the current European Community (EC) was set at 10,000 μg iodine per kg of complete feed [5].

Limiting dietary iodine contents in livestock feeding is relevant not only to the safety of the consumer of animal-derived products but also to the safety of the animal itself. For example, pigs fed with high doses of excessive dietary iodine showed similar symptoms as humans such as increased thyroid weight [6]. This was observed already at 10,000 μg iodine per kg of feed, which matches to the upper margin of permitted dietary iodine contents in pig nutrition. In humans, the NOAEL (no observed adverse effect level) was estimated to range at iodine consumption of 1,800 μg per day [7]. This is equivalent to dietary iodine concentrations of around 4,500 μg per kg of food dry matter (assuming consumption of 400 g food dry matter per day). Consequently, the adverse effect level of excessive dietary iodine seems to be similar in humans and pigs. The pig might therefore serve as a model to study the effect of dietary iodine excess in humans.

The aim of the present study was to assess whether large doses of iodine affect animal growth and the iodine accumulation rates in different pig tissues not only under various levels of iodine supplementation up to the maximum permitted iodine content in feed rations to pigs, but also from different iodine sources as KI and $KIO_3$.

**Table 1** Composition of basal diets and analyzed nutrient content in the grower and finisher diet of growing pigs

| Ingredient (%) | Grower diet | Finisher diet |
|---|---|---|
| Corn | 55.0 | 53.55 |
| Soybean meal without hulls | 20.0 | 7.0 |
| Barley | 10.0 | 19.0 |
| Peas | 7.5 | 10.0 |
| Wheat bran | 3.0 | 6.0 |
| Rapeseed oil | 1.0 | 1.0 |
| Mineral premix[a] | 3.0 | 3.0 |
| HCl-lysine | 0.20 | 0.20 |
| DL-methionine | 0.10 | 0.10 |
| Threonine | 0.20 | 0.15 |
| Analyzed nutrient composition | | |
| Dry matter (%) | 90.9 | 89.6 |
| Crude ash (%) | 4.9 | 2.4 |
| Crude protein (%) | 17.8 | 14.4 |
| Ether extracts (%) | 4.1 | 4.3 |
| Crude fiber (%) | 3.0 | 3.6 |
| Starch (%) | 45.9 | 50.5 |
| Sugar (%) | 5.6 | 5.0 |
| *Metabolisable energy*[b] *(MJ/kg)* | *14.5* | *14.4* |

Italic values are calculated values, in contrast to the other data, which were measured directly

[a] premix consisting of 22.0% Ca, 7.1% P, 5.0% Na, 450,000 IU/kg Vitamin A, 50,000 IU/kg Vitamin $D_3$, 700 IU/kg Vitamin E, 550 mg/kg Cu, 2,000 mg/kg Zn, 4 mg/kg Se, and 4 mg/kg KI

[b] calculated according to Society of Nutrition Physiology [8]

## Materials and methods

### Animals, diets, and housing

The present study was carried out with a total of 80 castrated male growing pigs (Austrian crossbreed OEHYB: (Large White × Landrace) × Piétrain; initial body weight (BW): 33.3 ± 0.4 kg). Animals were distributed equally among 5 dietary treatment groups with 16 pigs each considering litter and initial body weight. The grower and finisher diets were based on corn, soybean meal, barley, and peas and were produced by Garant, Pöchlarn, Austria (Table 1). The grower diet was fed until the animals reached a live weight of 70 kg; afterward the feed was switched to the finisher diet. Feed and water were provided ad libitum throughout the experiment. For treatment 1–5, diets were supplemented with different types and amounts of iodine (I) as follows: a control group, fed a basic diet that natively contained 20 μg I/kg feed in addition to 130 μg I as potassium iodide [KI] supplemented per kg feed (DM) according to the GfE recommendations [8], two KI groups (4,000 and 10,000 μg I/kg DM), and two potassium iodate [$KIO_3$] groups (4,000 and 10,000 μg I/kg DM). The respective iodine addition did not vary between the grower and finisher diets.

The animals were housed in 10 pens (i.e., two pens per treatment) at the Austrian Pig Testing Facility (Streitdorf, Austria). Pens had fully slatted concrete floors at a room temperature of 20–22 °C, a nipple drinker, and an automatic dry-feeding system. Individual body weight and feed intake were determined continuously using a transponder system.

### Slaughtering and sampling

At the end of the feeding trail, pigs were slaughtered under standardized conditions at an individual body weight of 115 ± 0.3 kg. Slaughtering was conducted using standard procedures in compliance with guidelines from the Austrian Pig Testing Facility. The different tissues samples from thyroid gland, liver (left lobe of the liver), kidney (marrow), abdominal muscle, abdominal fat tissue, and abdominal skin were collected and immediately frozen at −20 °C for later analysis. Blood was collected into heparinized polyethylene tubes (VACUETTE®, greiner bio-one,

Austria). Plasma was obtained by centrifugation of the blood (1,100*g*, 5 min) and stored at −20 °C pending analysis.

## Analysis

### *Chemical analysis of feed samples*

Diets were analyzed for dry matter (DM), crude ash, crude protein, ether extract, crude fiber, starch, and sugar according to standard procedures [9]. All samples were analyzed in duplicate.

### *Carcass composition*

Carcass measurements preformed included the following parameters: dressing, back fat depth, intramuscular fat, drip loss, and fat meat relation. All slaughter performance parameters were determined by Austrian Pig Testing Facility.

### *Iodine determination*

The iodine content in thyroid, liver, kidney, muscle, abdominal fat tissue, and skin was analyzed by a $HNO_3$–$HClO_4$ digestion method using inductively coupled plasma-mass spectrometry (ICP-MS; ELAN 6100 DRC-e, Perkin Elmer, Ohio) for detection [10]. The samples were freeze-dried and homogenized prior to analysis. A total of 0.1–0.5 g (the sample amounts with high fat content such as abdominal fat tissue and skin were limited to a maximum amount of 0.2 g) was transferred into the microwave PTFE vessels (MLS, MPV-100, Germany), and 4 mL $HNO_3$ (Rotipuran® supra 69%, Carl Roth GmbH, Germany) and 2 mL $HClO_4$ (Rotipuran® supra 70%, Carl Roth GmbH, Germany) were added. Furthermore, 5% $HNO_3$ was prepared as the dilution solution for standards calibration and instrument blank production. The samples were predigested at room temperature for at least 1 h until the 5-step microwave heating program started [10]. In each cycle, 1 blank value and 1 certified standard matrix (CRM) as reference material (BCR®-063R, skim milk powder—Community Bureau of Reference, Belgium) were extracted together with four tissue samples in duplicate. The samples were cooled completely after the microwave program and spiked with ultrapure water ($H_2O_R$, REWA-UPW-2) to a final volume of 25 mL and then stored at 5 °C. Standard solutions were prepared with $KIO_3$ (Fixanal Fluka Analytical potassium iodate 1/60 mol, Germany) for the iodine concentration calibration in samples. The recovery of the method was 105.0 ± 4.2%, which was confirmed using the BCR®-063R. Prior to ICP-MS analysis, the samples were spiked 1:10 with an indium solution (110 ppb in 1% $HNO_3$, Merck, Germany) as an internal standard (IS).

### *Analysis of plasma hormones*

The thyroid hormones thyroxine ($T_4$) and triiodothyronine ($T_3$) in plasma were determined with an enzyme immunoassay competition method (ELISA) with fluorescence detection using a commercial kit (BioMerieux, USA).

### *Statistical analysis*

The results were analyzed using the Statistical Analysis Software System package 9.1.3 (SAS Institute, Cary, NC). Firstly, the data were tested for normal distribution applying the Kolmogorov–Smirnov test. For the normally distributed data, the analysis of variance (ANOVA) was calculated with the GLM procedure of SAS to test the influence of iodine source and supplementation as well as the interaction of both on the growth performance, carcass characteristics and the iodine content of the different tissues, as well as plasma thyroid hormones. In case of significant interaction, the Tukey–Kramer method was applied for a multiple comparison of the least square means of each group. For not normally distributed parameters, the Kruskal–Wallis test (proc NPAR1WAY in SAS) was used. Data are presented as least square means for normally distributed data and means for not normally distributed data, respectively, as well as standard error of means (SEM). The level of significance was defined at $p < 0.05$ and the level of high significance at $p < 0.01$.

## Results

### Growth performance and carcass characteristics

One pig of group 3, one pig of group 4, and two pigs of group 5 were removed from the study due to respiratory disease and are therefore not included in the statistical analyses. The other 76 pigs were healthy throughout the experiment.

During the grower period, there was no significant difference in the voluntary feed intake (average daily feed intake, ADFI). However, a significant iodine dose influence ($p < 0.05$) was observed for the growth of the animals (average daily weight gain, ADG). The control group showed the highest ADG, whereas the pigs fed 10,000 µg I/kg $KIO_3$ showed the lowest ADG. Thus, a significant iodine treatment influence for the feed intake per kg of weight gain (feed conversion ratio, FCR) was resulting, where pigs in control group exhibited a significantly lower FCR compared to treatments 2 and 5.

In contrast to the grower period, ADG did not differ between the groups in finisher phase. However, significant interaction between source and dose was shown for ADFI

**Table 2** Influence of iodine source and supplementation (μg I/kg) on growth performance and carcass characteristics

| Treatment | 1 | 2 | 3 | 4 | 5 | SEM | *p*-Values | | |
|---|---|---|---|---|---|---|---|---|---|
| Source | Native/KI | KI | $KIO_3$ | KI | $KIO_3$ | | Source | Dose | Source × dose |
| Supplementation level (μg I/kg) | 20/130 | 4,000 | 4,000 | 10,000 | 10,000 | | | | |
| *Grower period* | | | | | | | | | |
| ADG[1] (g/d) | 933.5 | 886.8 | 859.1 | 846.9 | 796.2 | 10.0 | 0.051 | **0.001** | 0.563 |
| ADFI[1] (kg DM/d) | 2.24 | 2.39 | 2.15 | 2.26 | 2.31 | 0.04 | 0.279 | 0.733 | 0.096 |
| FCR[1,2] (kg/kg) | 2.35[b] | 2.64[a] | 2.51[ab] | 2.66[ab] | 2.86[a] | 0.05 | | | |
| *Finisher period* | | | | | | | | | |
| ADG[1] (g/d) | 813.9 | 800.2 | 784.6 | 778.9 | 802.9 | 9.6 | 0.855 | 0.627 | 0.387 |
| ADFI[1] (kg DM/d) | 2.66[ab] | 2.49[b] | 2.75[ab] | 2.85[a] | 2.63[ab] | 0.04 | 0.780 | 0.437 | **0.011** |
| FCR[1] (kg/kg) | 3.38[ab] | 3.16[b] | 3.56[ab] | 3.61[a] | 3.36[ab] | 0.05 | 0.467 | 0.504 | **0.003** |
| *Growth performance (entire grower-finisher period)* | | | | | | | | | |
| ADG[1] (g/d) | 830.9 | 841.4 | 786.4 | 817.7 | 814.7 | 6.4 | **0.041** | 0.982 | 0.065 |
| ADFI[1,2] (kg DM/d) | 2.42 | 2.35 | 2.46 | 2.43 | 2.45 | 0.03 | | | |
| FCR[1,2] (kg/kg) | 2.85 | 2.92 | 3.10 | 3.09 | 3.11 | 0.04 | | | |
| *Slaughter performance* | | | | | | | | | |
| Dressing (%) | 79.68 | 79.47 | 79.62 | 79.49 | 78.87 | 0.2 | 0.601 | 0.671 | 0.399 |
| Back fat depth[2] (mm) | 24.81 | 25.44 | 24.40 | 23.67 | 23.07 | 0.31 | | | |
| Intramuscular fat (%) | 1.22 | 1.11 | 1.22 | 1.12 | 1.08 | 0.03 | 0.584 | 0.192 | 0.222 |
| Drip loss (%) | 6.33 | 6.23 | 6.37 | 6.41 | 7.81 | 0.32 | 0.289 | 0.533 | 0.388 |
| Fat meat relation (1:…) | 5.13 | 5.08 | 4.98 | 5.17 | 5.38 | 0.07 | 0.734 | 0.341 | 0.353 |

Bold values are statistically significant ($p < 0.05$)

[a, b] Means within rows without a common superscript differ significantly ($p < 0.05$)

[1] *BW* body weight, *ADG* average daily weight gain, *ADFI* average daily feed intake per animal, *FCR* feed conversion ratio

[2] Only the influence of treatment was tested using Kruskal–Wallis test

and FCR. The pigs on treatment 2 differed significantly from pigs on treatment 4 in both of these two parameters (Table 2).

Over the entire experimental period, ADFI and FCR were not influenced by the iodine supplementation. However, a significant iodine source influence in ADG was observed with lower ADG values in the $KIO_3$ groups compared to the KI groups.

The carcass composition of pigs did not differ between pigs fed the control diets and pigs fed the iodine diets (Table 2).

### Iodine content in organs and tissues and concentration of thyroid hormones

The highest iodine concentration was measured in the thyroid gland and then followed by kidney, liver, muscle, skin, and abdominal fat tissue (Table 3). Iodine concentrations in thyroid gland, kidney, and skin showed high significant ($p < 0.01$) iodine dose influences. The increasing iodine content of feed heightened iodine concentration in muscle samples too; however, significant interaction of the factors highlighted that these increases were not similar for KI and $KIO_3$. In liver, the iodine concentration increased in a linear manner with the higher iodine supplementation and showed a significant treatment influence. Additionally, no significant treatment effect was observed in abdominal fat tissue. Likewise, neither $T_4$ and $T_3$ thyroid hormones nor their ratio was influenced by the iodine supplementation.

## Discussion

Despite the advances and a significant decrease in the number of IDD, an estimated two billion individuals still have an insufficient iodine intake, especially in Africa, Asia, and Europe [11]. Due to the native low iodine content in most foods, iodized salt is most popular used in human nutrition to compensate low iodine intake. $KIO_3$ is commonly used for supplementation, due to its higher stability compared to KI [12]. Moreover, there are approximately more than 4.5 billion livestock in the world, which are fed insufficient iodine amounts cause of the poor iodine content in plant matter [11, 13]. In recent years, alternative iodine prophylaxis methods were designed to improve animal health as well as human nutrition. For example, the iodine content from animal feed was transferred into food of animal origin like milk, eggs, and meat [4, 14].

**Table 3** Influence of iodine supplementation on iodine content in tissues (DM) and thyroid hormone concentrations

| Treatment | 1 | 2 | 3 | 4 | 5 | SEM | *p*-Values | | |
|---|---|---|---|---|---|---|---|---|---|
| Source | Native/KI | KI | $KIO_3$ | KI | $KIO_3$ | | Source | Dose | Source × dose |
| Supplementation level (μg I/kg) | 20/130 | 4,000 | 4,000 | 10,000 | 10,000 | | | | |
| *Tissues iodine contents* | | | | | | | | | |
| Thyroid gland (g/kg) | 9.68 | 16.42 | 17.55 | 20.60 | 22.28 | 0.7 | 0.195 | **<0.0001** | 0.797 |
| Liver[1] (μg/kg) | 146.4[c] | 265.2[b] | 270.9[b] | 457.0[a] | 596.7[a] | 24.0 | | | |
| Kidney (μg/kg) | 171.8 | 471.4 | 664.2 | 1,185.2 | 950.4 | 71.4 | 0.866 | **<0.0001** | 0.091 |
| Muscle (μg/kg) | 120.7[b] | 101.4[b] | 142.6[b] | 362.6[a] | 194.9[ab] | 23.7 | 0.192 | **0.001** | **0.033** |
| Abdominal fat tissue (μg/kg) | 8.6 | 14.5 | 14.6 | 21.0 | 12.2 | 1.4 | 0.160 | 0.508 | 0.150 |
| Skin (μg/kg) | 15.8 | 31.7 | 50.4 | 70.5 | 62.5 | 3.9 | 0.456 | **<0.0001** | 0.065 |
| *Thyroid hormones* | | | | | | | | | |
| $T_3$ (nmol/l) | 0.71 | 0.77 | 0.83 | 0.84 | 0.74 | 0.02 | 0.730 | 0.330 | 0.121 |
| $T_4$ (nmol/l) | 55.93 | 54.95 | 57.37 | 59.07 | 53.72 | 0.99 | 0.510 | 0.921 | 0.084 |
| $T_3/T_4$ ratio | 0.010 | 0.010 | 0.010 | 0.010 | 0.010 | 0.0004 | 0.937 | 0.275 | 0.580 |

Bold values are statistically significant ($p < 0.05$)

[a, b] Means within rows without a common superscript differ significantly ($p < 0.05$)

[1] Only the influence of treatment was tested using Kruskal–Wallis test

In this context, a feeding trial was conducted, analyzing the influence of rising iodine supplementations and different sources on animal performance as well as the iodine content in tissues and thyroid hormone concentration.

The effect of iodine supplementation on growing fattening pigs was different in the grower and finisher phase. Briefly, high iodine intake depressed the weight gain of pigs (ADG) significantly. However, these findings disappeared in the finisher phase. These results suggest that high-iodine diets may be more harmful to young animals than to more mature animals, as young animals showed similar feed intake compared to more mature animals despite their lower body weight. Furthermore, results of the performance trial showed significantly impaired FCRs in treatments 2 (4,000 μg I/kg as KI) and 5 (10,000 μg I/kg as $KIO_3$) compared to the treatment 1 (150 μg I/kg as KI) during grower period, and during the finisher period in treatment 4 (10,000 μg I/kg as KI) compared to the treatment 2 (4,000 μg I/kg as KI). However, considering the entire grower-finisher period, these changes in FCR in the higher iodine groups were no longer significant. Similar results for the whole fattening period were shown in other studies [15–18], but the present study showed that different iodine source may also affect the ADG. Especially compared to the study of Schone et al. [19], who fed 10,000 μg I/kg as KI to grower-finisher pigs, higher ADG (10%) and ADFI (3%) and lower FCR (3%) were observed in the present study. Furthermore, in agreement with other porcine dose–response studies [15–18], the present study reported no iodine supplementation and source influence on any carcass parameters.

In the present study, a dose-depended increase was shown between the control group and the iodine supplementation groups among most of tissues except in abdominal fat tissues (Table 3). In general, comparable published dose–response experiments in growing pigs resulted in lower values than the present study [15–18]. These studies applying iodine concentrations from 100 to 8,000 μg I/kg diet, showed different muscle iodine contents in the range of about 4–17 μg I/kg of fresh matter (FM) in studies of Franke et al. [17] and Schone et.al. [18], 32–50.6 μg I/kg FM in the study of He et al. [16], as well as 23–138 μg I/kg of dry matter (DM) as reported by Rambeck et al. [15]. However, these observations in iodine content are only one-fortieth to half of the concentrations observed in the present study.

Iodine as an essential trace element is present in a very low concentration (<0.5 mg/kg) in the body. Several methods have been reported for total iodine determination, including Sandell–Kolthoff method [20], isotope-dilution method [21], and methods by ICP-MS applying various digestion methods, such as the tetramethylammonium hydroxide (TMAH) digestion [22, 23] or the acid digestion method with a mixture of nitric acid and perchloric acid ($HNO_3$–$HClO_4$) [10, 24, 25]. For acid digestion, high oxidation power from $HNO_3$–$HClO_4$ mixture could disintegrate the biologic sample to its full extent without any iodine losses [10]. Therefore in the present study, the acid digestion method ($HNO_3$–$HClO_4$) using ICP-MS technique was chosen for the total iodine determination.

Indeed, minor variations in results may be due to the different digestion and detection methods applied, as Franke et al. [17] and Schone et.al. [18] used the TMAH digestion method prior to ICP-MS detection, whereas Rambeck et al. [15] and He et al. [16] used the

Sandell–Kolthoff reaction. However, the major influencing factor on iodine contents in tissue might be the different iodine sources used in these studies. Thus, KI was supplied in feed in studies of He et al. [16] and Schone et.al. [4], whereas $KIO_3$ was used in studies of Rambeck et al. [15] and Franke et al. [17]. Thence, using different iodine sources, chemically and technically different methods may affect results, which could partly explain the high deviation of the present study′s results compared to the other studies. Furthermore, with 10,000 μg I/kg diet, KI showed more accumulation than $KIO_3$ in extrathyroidal tissues except liver and raised the muscle content to a significant extent. This finding is in accordance with some isotope studies that human iodine bioavailability from $KIO_3$ was about 10% lower than from KI [26, 27]. Anyway, all these results from different studies are important, indicating the need for further investigation on this topic.

Thyroid represents not only the store but also the control center of the iodine and thyroid hormone household [19]. Compared to the thyroid gland, the extrathyroidal tissues contain only traces of iodine (Table 3): The ratios of iodine concentration in these extrathyroidal tissues to the thyroid amount to 1: 100,000 and those of the abdominal fat tissue to the gland even to 1: 1,000,000. Nevertheless, both thyroid hormones, $T_3$ and $T_4$, were neither affected by the dose nor affected by the source of iodine as their concentrations remained relatively constant over this wide range of iodine supplementation (150–10,000 μg I/kg diet). In contrast to this study, similar $T_3$ but 20% lower $T_4$ contents were shown in the study of Schone et al. [19], who observed a significantly decreasing effect on $T_3$ with 10,000 KI/kg diet. However, comparison of results is exacerbated by the different methods applied for measuring thyroid hormones in plasma of the pigs, as radioimmunoassay kit was used in the study of Schone et al. [19] and ELISA was used in the present study.

Excessive iodine intake affects not only the animal‘s health, but may also affect consumers as iodine is concentrated in foods via carryover from feed [6]. The present study resulted in maximum concentration of 362.6 μg I/kg in pork, which, according to NRC [28], should be safe for animal health and human nutrition. In contrast, the carryover of iodine into milk and eggs is much higher than into pork [4, 14], a fact also confirmed by Franke et al. [17].

In conclusion, this study shows that iodine supplementation and different iodine sources in pig diets have negative effects on growth of younger animals. However, the overall growth performance, carcass characteristics, and both thyroid hormones remained similar to the control. Hence, further research on piglets would be required to determine appropriate upper limits for iodine intake in younger animals. Increasing the iodine supplementation had a significant effect concerning the iodine enrichment in most tissues, except for abdominal fat tissue. Irrespective of iodine supplementation, the thyroid gland contained more than 80% of the total iodine amount in animal bodies. The contribution of pork and fat of pigs to the iodine intake of humans is marginal even at a high iodine supplementation of feed (10,000 μg I/kg). Therefore, a reduction of the EU-UL of iodine content in feed of grower-finisher pigs is unnecessary, regarding human nutrition and the health of the animals.

## References


1. Kotwal A, Priya R, Qadeer I (2007) Goiter and other iodine deficiency disorders: a systematic review of epidemiological studies to deconstruct the complex web. Arch Med Res 38:1–14
2. Pennington JA (1990) A review of iodine toxicity reports. J Am Diet Assoc 90:1571–1581
3. European Food Safety Authority (2005) Opinion of the scientific panel on additives and products or substances used in animal feed on the request from the commission on the use of iodine in feedingstuffs. EFSA J 168:1–42
4. Flachowsky G, Schone F, Jahreis G (2006) Iodine enrichment in food of animal origin. Ernährungs-Umschau 53:17–21
5. European Union (2005) Commission regulation (EC) of 8 September 2005 amending the conditions for authorisation of a number of feed additives belonging to the group of trace elements. The Commission of the European Communities
6. Schone F, Rajendram R (2009) Iodine in farm animals. In: Preedy V, Burrow G, Watson R (eds) Comprhensive handbook of iodine. Elsevier Academic Press, Oxford
7. Scientific committee on food (2002) Opinion of the scientific committee on food on the tolerable upper intake level of iodine. European Commission, Bruxelles/Brussels
8. GfE (2008) Communications of the committee for requirement standards of the society of nutrition physiology—prediction of metabolisable energy of compound feeds for pigs. DLG Verlag, Frankfurt/Main
9. Naumann C, Bassler R (1997) Die chemische Untersuchung von Futtermitteln. VDLUFA-Verlag, Darmstadt
10. Benkhedda K, Robichaud A, Turcotte S, Beraldin FJ, Cockell KA (2009) Determination of total iodine in food samples using inductively coupled plasma-mass spectrometry. J AOAC Int 92:1720–1727
11. Zimmermann MB (2009) Iodine deficiency. Endocr Rev 30:376–408
12. Kelly FC (1953) Studies on the stability of iodine compounds in iodized salt. Bull World Health Organ 9:217–230
13. Food and Agriculture of the Organization of the United Nations (2006) Livestock's long shadow: environmental issues and options. FAO, Rome
14. Yalcin S, Kahraman Z, Yalcin SS, Dedeoglu HE (2004) Effects of supplementary iodine on the performance and egg traits of laying hens. Br Poult Sci 45:499–503
15. Rambeck WA, Kaufmann S, Feng J, Hollwich W, Arnold R (1997) Improving the human iodine supply by iodination of swine feed. Tierarztl Prax 25:312–315
16. He ML, Hollwich W, Rambeck WA (2002) Supplementation of algae to the diet of pigs: a new possibility to improve the iodine content in the meat. J Anim Physiol Anim Nutr (Berl) 86:97–104
17. Franke K, Schone F, Berk A, Leiterer M, Flachowsky G (2008) Influence of dietary iodine on the iodine content of pork and the distribution of the trace element in the body. Eur J Nutr 47:40–46

18. Schone F, Zimmermann C, Quanz G, Richter G, Leiterer M (2006) A high dietary iodine increases thyroid iodine stores and iodine concentration in blood serum but has little effect on muscle iodine content in pigs. Meat Sci 72:365–372
19. Schone F (1999) Iodine deficiency, iodine requirement and iodine excess of farm animals–experiments on growing pigs. Berl Munch Tierarztl Wochenschr 112:64–70
20. Sandell ED, Kolthoff IM (1937) Microdetermination of iodine by a catalytic method. Mikrochemica Acta 1:9–25
21. Windisch W, He J, Kirchgessner M (1999) Isotope-dilution technique for determination of endogenous faecal excretion and true absorption of iodine in 125I labelled rats. J Anim Physiol Anim Nutr 82:205–215
22. Sturup S, Buchert A (1996) Direct determination of copper and iodine in milk and milk powder in alkaline solution by flow injection inductively coupled plasma mass spectrometry. Anal Bioanal Chem 354:323–326
23. Gelinas Y, Krushevska A, Barnes RM (1998) Determination of total iodine in nutritional and biological samples by ICP-MS following their combustion within an oxygen stream. Anal Chem 70:1021–1025
24. Knapp G, Maichin B, Fecher P, Hasse S, Schramel P (1998) Iodine determination in biological materials—options for sample preparation and final determination. Fresenius J Anal Chem 362:508–513
25. Larsen EH, Ludwigsen MB (1997) Determination of iodine in food-related certified reference materials using wet ashing and detection by inductively coupled plasma mass spectrometry. J Anal Atom Spectr 12:435–439
26. Murray MM, Pochin EE (1951) Thyroid uptake of iodine from ingested iodate in man. J Physiol 114(Suppl):1–14
27. Murray MM (1953) The effects of administration of sodium iodate to man and animals. Bull World Health Organ 9:211
28. NRC (2005) Mineral tolerance of animals. National Research Council of the National Academies, Washington, D.C