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Selenium influences growth via thyroid hormone status in broiler chickens

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As there is a possibility that Se influences the growth of animals via thyroid hormone metabolism, the following three experiments were undertaken in order to determine the effects of dietary Se on growth, skeletal muscle protein turnover and thyroid hormone status in broiler chickens. Broiler chickens were raised on a Se-deficient diet until 12 d of age and then used for the experiments. In Experiment 1, twenty-eight birds were randomly assigned to four groups and fed purified diets with the following amounts of Se supplementation: 0·0, 0·1, 0·3 and 0·5 mg Se/kg diet. Dietary Se supplementation significantly increased plasma 3,5,3′-triiodothyronine (T3) concentration and improved growth, while plasma thyroxine (T4) concentration was decreased. In Experiment 2, twenty-eight birds were assigned to four groups and fed either a Se-deficient diet or a Se-supplemented diet (0·3 mg Se/kg diet) with or without the supplementation of iopanoic acid, a specific inhibitor of 5′-deiodinase (5 mg/kg diet). The growth was promoted and feed efficiency was improved by dietary Se supplementation as was also observed in Experiment 1. However, this effect of Se was halted by iopanoic acid supplementation. Hepatic 5′-deiodinase activity was elevated by Se and inhibited by iopanoic acid. In Experiment 3, birds were fed on the following diets to show that Se influences growth of birds via thyroid hormone metabolism: Se-deficient diet, Se-supplemented diets (0·1 and 0·3 mg/kg) and T3 supplemented diets (0·1 and 0·3 mg/kg diet). Lower dietary T3 supplementation (0·1 mg/kg diet) resulted in growth promotion similar to Se supplementation, while higher level of T3 caused growth depression. Furthermore, it was observed that the rate of skeletal muscle protein breakdown tended to be increased by Se similarly to the effect of T3. In conclusion, it was shown in the present study that Se deficiency depresses growth of broilers by inhibiting hepatic 5′-deiodinase activity which causes lower plasma T3 concentration.

Selenium: Thyroid hormone: 5′-Deiodinase: Growth

Since Se was established as being an essential micronutrient in 1957 (Schwartz & Foltz, 1957), most studies concerning Se have concentrated on the role of glutathione peroxidase (GSHPx), which catabolizes H_2O_2 and other organic hydroperoxides (Burk, 1989). In 1986, Jensen *et al.* (1986) observed that dietary Se affects thyroid hormone metabolism, and it was then observed by Beckett *et al.* (1987) that plasma 3,5,3′-triiodothyronine (T3) is produced by 5′-deiodination of thyroxine (T4) in non-thyroidal tissues, particularly the liver and kidney. The reaction is catalysed by type I iodothyronine deiodinase (5′-ID) (Beckett *et al.* 1992), which was verified as being a selenoenzyme (Arthur *et al.* 1990, 1992, 1993; Berry *et al.* 1991; Beckett *et al.* 1992; Kohrle *et al.* 1992). Brain, pituitary gland and brown adipose tissues contain a different selenoenzyme catalysing 5′-deiodinations, type II deiodinase (Davey *et al.* 1995). It has been reported that Se deficiency alters both thyroid hormone synthesis in the thyroid gland and the activity of tissue specific 5′-ID in rats (Kohrle *et al.* 1992). Hepatic 5′-ID activity in the Se-deficient rat is 10-fold lower than that of the Se-supplemented rat (Beckett *et al.* 1992) and plasma T3 concentration is significantly lower than that of the normal rat (Beckett *et al.* 1987). On the other hand, Arthur & Beckett (1994) have reported that the 5′-ID activity in the thyroid of Se-deficient rats is four times greater than in

Abbreviations: GSHPx, glutathione peroxidase; 5′-ID, type I iodothyronine deiodinase; MH, N^{τ}-methylhistidine; T3, 3,5,3′-triiodothyronine; T4, thyroxine.

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Se-sufficient rats. Chanoine *et al.* (1993) have reported that the thyroid gland is a major source of circulating T3 in rats, accounting for approximately 55 % of total T3 production. In any case, T4 is synthesized exclusively in the thyroid gland, and thought to be a prohormone requiring activation by 5′-monodeiodination to form biologically active T3. The contribution of intrathyroidal conversion of T4 to T3 to T3 homeostasis appears to be important, but the exact contribution remains to be determined.

It is well documented that the thyroid hormones play important roles in growth and protein turnover (Hayashi *et al*. 1991; Hayashi, 1993), therefore, Se deficiency might affect protein turnover followed by growth retardation, as T3 production is impaired.

In the present study, three experiments were carried out to investigate the effect of dietary Se on thyroid hormone status, growth and skeletal muscle protein breakdown in broiler chickens. The first experiment was designed to clarify the relation between Se level, plasma thyroid hormone and growth. To further characterize the effects of dietary Se on thyroid hormone metabolism in the second experiment, we used iopanoic acid, a deiodinase inhibitor, to prove that the effect is correlated with the change in 5′-ID activity. The third experiment was designed to compare the effect of dietary Se and dietary T3 on growth and skeletal muscle protein breakdown.

Table 1. Composition of the purified diet

Ingredients	g/kg
Isolated soyabean protein	250·0
Dextrose	607·4
Glycine	4·0
Methionine	6·0
Corn oil	40·0
Cellulose	30·0
Vitamin mix*	2·6
Mineral mix†	60·0
Calculated nutrient composition:	
ME (μJ/kg)	13·81 (3300 kcal/kg)
CP	212·5
Lysine	12·5
Met + Cys	10·0
Ca	12·0
NPP	6·5
Determined Se content (mg/kg)	0·05

ME, metabolizable energy; CP, crude protein; Met, methionine; Cys, cysteine; NPP, non-phytate phosphorus.

* Provided (per kg diet): thiamin 2·6 mg, vitamin A (retinol) 1365 μg cholecalciferol 13 mg, vitamin E (DL-α-tocopheryl acetate) 6·5 mg, menadione (2-methyl-1,4-naphtohydroquinone diacetate) 650 μg, riboflavin 6·5 mg, pyridoxine 1·3 mg, cyanocobalamin 26 μg, pantothenic acid 10·4 mg, nicotinic acid 26 mg, choline chloride 780 mg, biotin 65 μg, folic acid 520 μg.

† Provided (per kg diet): NaCl 6 g, $CaCO_3$ 14·8 g, $MgSO_4.7H_2O$ 6 g, $CaHPO_4.2H_2O$ 20·7 g, K_2HPO_4 10·0 g, KCl 1 g, Mn 60 mg ($MnSO_4$ 360 mg), Zn 60 mg ($ZnCO_3$ 150 mg), Fe 80 mg ($FeSO_4.7H_2O$ 500 mg), Cu 10 mg ($CuSO_4.5H_2O$ 30 mg), I 0·76 mg (KI 1 mg), Co 1 mg ($CoCl_2$ 1·7 mg), Mo 3·3 mg ($Na_2Mo_4.2H_2O$ 8·3 mg).

Materials and methods

Animals and diets

Experiment 1. A total of forty 1-d-old Arbor Acres male broiler chickens, obtained from a commercial hatchery (Kumiai Hina Center, Kajiki Kagoshima, Japan), were housed in an electrically-heated battery brooder and were given a Se-deficient diet for 12 d (Se-depletion period) with free access to deionized water. The Se-deficient diet was formulated according to the National Research Council (1994) and the composition is shown in Table 1. Twenty-eight birds of similar body weight (199 (SEM 8) 8 g) were then selected and randomly assigned to one of four dietary treatments (0·0, 0·1, 0·3 or 0·5 mg Se/kg diet). Sodium selenite was used to supplement Se. The diets were given for 9 d starting at 12 d of age. All the birds were individually housed in a metabolism cage in a temperature-controlled room (25°C) with free access to feed and deionized water. Body weight was measured on the 3rd, 6th and 9th day of the treatment period. Feed intake was recorded daily. At 21 d of age, all the birds were killed by decapitation and blood samples were collected into heparinized tubes and centrifuged at 1250 ***g*** for 15 min at 4°C. The plasma samples were then stored at −20°C until analysed.

Experiment 2. Forty birds were raised exactly as in Experiment 1 until 12 d of age. Then twenty-eight birds of similar body weight (about 238 (SEM 8) g) were selected and randomly assigned to one of four dietary treatments for 9 d (0 or 0·3 mg Se/kg diet, with or without 5 mg iopanoic acid/kg diet). Iopanoic acid was purchased from Sigma Chemical Co., (St Louis, MO, USA). The experiment was conducted in a similar way to Experiment 1.

Experiment 3. The third experiment was conducted similarly to the previous experiments except excreta was collected to measure N^{τ}-methylhistidine (MH) excretion which is an index of skeletal muscle protein breakdown. The birds were fed the Se-deficient diet for the first 12 d and thirty-five birds of similar body weight (about 232 (SEM 8) g) were selected and randomly assigned to one of five treatment groups: 0 mg Se/kg diet; 0·1 mg Se/kg diet; 0·3 mg Se/kg diet; 0·1 mg T3/kg diet; 0·3 mg T3/kg diet. The excreta samples were collected for 2 d starting at 19 d of age. The collected samples were stored at −20°C until analysed. At 21 d of age, all the birds were killed by decapitation and blood samples were collected as described in experiment 1 (p. 728). The chemicals were purchased from Nacalai Tesque Inc. (Kyoto, Japan) unless otherwise stated.

Plasma glutathione peroxidase activity measurement

Plasma GSHPx activity was measured by the modified method of Hawkes & Kimberly (1990). The unit of activity was defined as the amount of enzyme required to prevent the reduction of 1 μmol dichloroindophenol per min at 25°C, pH 7·8. Each plasma sample (1 ml) was preincubated with 0·8 ml 2·3 mM-glutathione for 2·5 min, then 0·8 ml 1·5 mM-peroxide was added and the enzyme reaction went on for 14 min. Then, 0·8 ml 0·68 mM-dichloroindophenol was added, reacted for 7 min and absorbance was determined with a double-beam spectrophotometer (UV-150-02, Shimadzu Corporation, Kyoto, Japan) at 630 nm. The unit shown in the present experiment can be converted to the unit reported by Paglia & Valentine (1967) by multiplying by 167.

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Measurement of thyroid hormone

Plasma T4 and T3 concentrations were measured by commercial kits. T4 concentration was measured by Enzymun-Test T4 (Boehringer-Mannheim, Mannheim, Germany) and T3 concentration was measured by Enzymun-Test T3, Elisa-Auto T3 (International Reagents Corporation, Kobe, Japan).

Selenium content determination

The plasma and liver Se content were determined by the fluorometric method (Bellanger, 1995). Samples were digested at 160°C for 150 min with a mixed digestion solution (13 M-HNO_3–12 M-$HClO_4$ (2:1, v/v)). The pH of digested solution was adjusted to 1·0–1·5 with 6 M-NaOH, then a fluorescence derivative was made with 1 ml 0·1 % 2,3-diaminonaphthalene. The fluorescence compound was extracted with 2·0 ml cyclohexane (Wako Pure Chemical Industry Ltd, Osaka, Japan), and was measured by F-2000 fluorescence spectrophotometer (Shimadzu Corporation) at excitation wavelength of 379 nm and emission wavelength of 521 nm.

Hepatic type I iodothyronine deiodinase activity measurement

The hepatic 5′-ID activity was measured as the rate of radiolabelled I released from labelled reverse T3 (3,3′,5′-triiodo-L-thyronine) according to the method of Freeman & McNabb (1991). The liver samples (0·5 g) were homogenized in three volumes ice-cold 50 mM-morpholinopropane sulfonic acid (Sigma Chemical Co.) buffer, containing 1 mM-EDTA at pH 7·4. The homogenate was centrifuged at 1000 ***g*** at 4°C for 10 min and the supernatant was stored at −20°C until analysis. The reaction was done with an incubation volume of 80 μl, which consisted of 25 μl supernatant, 20 μl dithiothreitol (80 mM), 10 μl morpholinopropanesulfonic acid buffer, 20 μl unlabelled reverse T3 (3,3′,5′-triiodo-L-thyronine, 12·8 μM; Sigma Chemical Co.) and 5 μl ^{125}I-labelled reverse T3 (12·8 μM, L-3,3′,5′ triiodothyronine, 31 MBq/μg; New England Nuclear Corp., Boston, MA, USA). Boiled liver homogenate was used as a reaction blank. After vortexing, the tubes were placed in a shaking waterbath (37°C, 90 strokes/min) for 15 min. The reaction was terminated by adding 20 μl of bovine serum albumin (40 g/l) (Sigma Chemical Co.), followed immediately by the addition of 150 μl ice-cold TCA (200 g/l). The tubes were held in ice-cold water for 15 min, then total radioactivity was determined. After the determination, all the reaction solution was centrifuged at 4000 ***g*** at −10°C for 30 min, then 170 μl supernatant was applied to an ion-exchange column (Dowex AG 50W-X8 (H^+); Bio-Rad Laboratories, Hercules, CA, USA), which was prewashed with 10 ml 1·74 M-acetic acid. The ^{125}I was eluted with four 2 ml aliquots of 1·74 M-acetic acid, and the radioactivity in the acetic-acid fraction was counted. The protein content of the liver homogenate was determined by the Lowry method (Lowry *et al.* 1951).

Measurements of N^{τ}-methylhistidine excretion rate

The MH content of excreta samples was measured by the method of Hayashi *et al.* (1987) using HPLC. Dietary MH content was analysed similarly. The MH content of the experimental diet was 0·06 μmol/g, and this was subtracted from the total excreted MH. The rate of skeletal muscle protein breakdown was calculated by dividing the amount of excreted MH derived from skeletal muscle by the calculated amount of MH in the skeletal muscle using the method of Hayashi *et al.* (1985).

Statistical analysis

The results were analysed using Statview 4·11 (Abacus Concepts, Inc., Berkeley, CA, USA). One-way factorial ANOVA was applied and Fisher's PLSD (protected least significant difference) was selected for multiple comparison. Data are presented as means with their standard errors. Results were considered statistically significant at the $P < 0·05$ level.

Results

Experiment 1

Results obtained in Experiment 1 are summarized in Fig. 1. Growth rates of the birds consuming Se-supplemented diets (0·1, 0·3 or 0·5 mg Se/kg diet) were all greater compared with that of the Se-deficient birds, but significance ($P < 0·05$) was observed only when the 0·5 mg Se/kg diet was given (Fig. 1(a)). Dietary Se had no significant effect on feed intake and feed efficiency (Fig. 1(a)). The liver and plasma Se levels were significantly increased when birds were fed 0·3 or 0·5 mg Se/kg diet. However, there were no significant differences between the birds fed 0·1 mg Se/kg diet and the Se-deficient diet (Fig. 1(b)). Se deficiency caused a significantly lower plasma T3 concentration and a higher plasma T4 concentration ($P < 0·05$; Fig. 1(c)).

Experiment 2

Results obtained in Experiment 2 are summarized in Fig. 2. Se supplementation significantly ($P < 0·05$) improved the growth rates and feed efficiency as was shown in Experiment 1. However, the growth promoting effect of Se was not observed when iopanoic acid was given. The improvement in feed efficiency by Se supplementation was also halted by iopanoic acid (Fig. 2(a)). Plasma T4 concentration was significantly decreased ($P < 0·05$) by Se and this effect was negated by iopanoic acid (Fig. 2(b)). Plasma T3 concentrations were significantly higher in the Se group, and iopanoic acid neutralized the effects of Se. Se supplementation increased the hepatic 5′-ID activity and this increase was also negated by iopanoic acid (Fig. 2(c)).

Experiment 3

Results are shown in Fig. 3. The growth rate was higher than for the control diet when 0·3 mg Se/kg diet was added. The growth rate was also improved by 0·1 mg T3/kg diet while the growth was retarded when 0·3 mg T3/kg was

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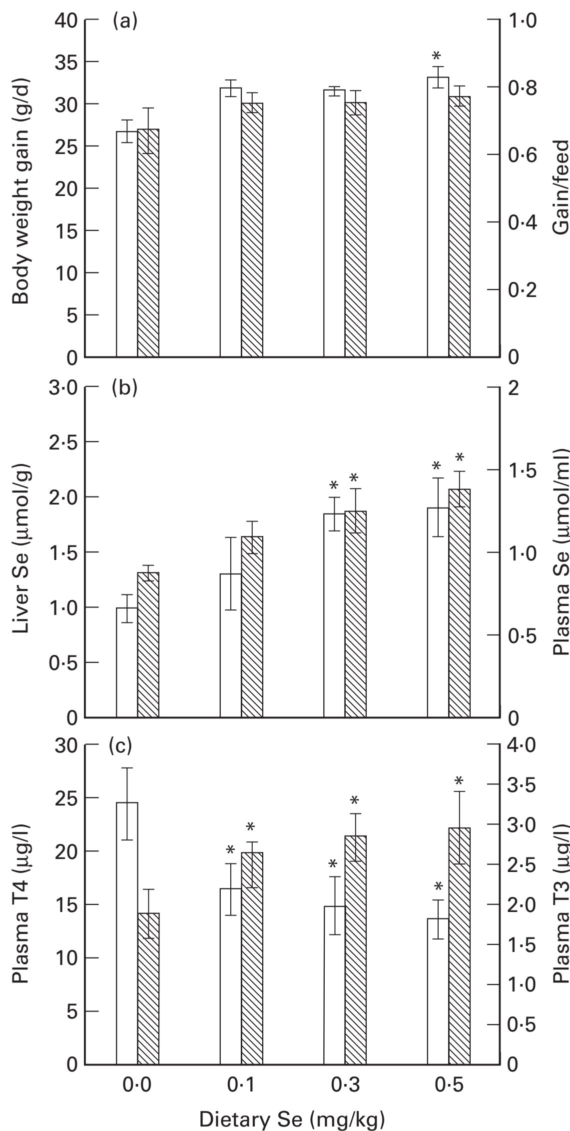

Fig. 1. Effects of dietary Se on (a), body-weight gain (□) and gain/feed (▧), and (b), Se contents in liver (□) and plasma (▧), and (c), plasma concentrations of thyroxine (T4) (□) and 3,5,3′-triiodothyronine (T3) (▧) in broiler chickens. For details of dietary composition see p. 728 and Table 1. Values are means for seven birds with their standard errors shown by vertical bars. Mean values were significantly different from the control (0 mg Se/kg diet): $^{*}P < 0{\cdot}05$.

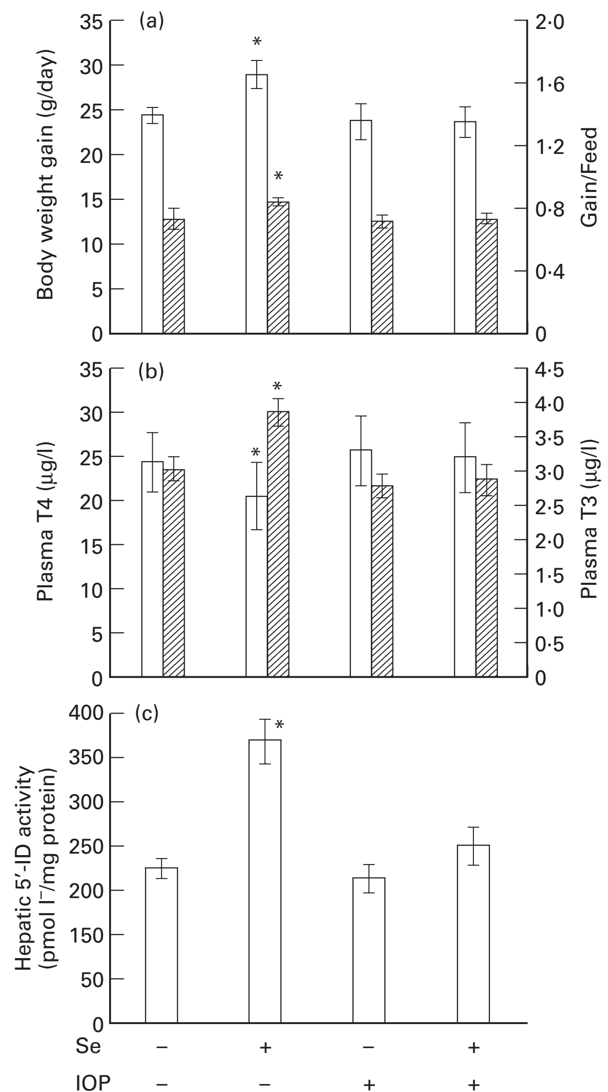

Fig. 2. Effects of dietary Se and iopanoic acid (IOP) on (a), body-weight gain (□) and gain/feed (▨), and (b) plasma concentrations of thyroxine (T4) (□) and 3,5,3′-triiodothyronine (T3) (▨), and (c), hepatic type I iodothyronine deiodinase (5′-ID) activity in broiler chickens. For details of dietary composition see p. 728 and Table 1. Values are means for seven birds with their standard errors shown by vertical bars. Mean values were significantly different from the control (−Se, −IOP): $^{*}P < 0{\cdot}05$.

added. Both dietary Se and T3 had similar effects on feed efficiency (Fig. 3(a)). The supplementation of the diet with 0·1 mg T3/kg caused a similar plasma T3 level as Se supplementation, while 0·3 mg T3/kg diet caused a much higher plasma T3 level (Fig. 3(b)). Dietary T3 supplementation had a tendency to decrease the plasma T4 level (Fig. 3(b)). Se addition increased both hepatic 5′-ID activity and plasma GSHPx activity but dietary T3 supplementation had no significant effect on the activities of both enzymes (Fig. 3(c)). Dietary Se supplementation tended to increase the rate of skeletal muscle protein breakdown (Fig. 3(d)) indicating an increased muscle protein degradation due to increased plasma T3.

Discussion

Although the present experiment was not designed to determine the recommended amount of dietary Se intake, the suitable dietary Se level is suggested to be about 0·3 mg/kg diet, based on the analyses of plasma GSHPx activity and liver Se contents. Plasma Se concentration seemed to reach plateau at a highest supplementation level (about 0·5 mg/kg). The suitable Se level suggested in the present experiment is higher than the National Research Council (1994) recommendation (0·15 mg/kg diet), but corresponds with the US Food and Drug Administration allowance (Ullrey, 1992). However, hepatic 5′-ID activity could not be used for this purpose in the present experiment

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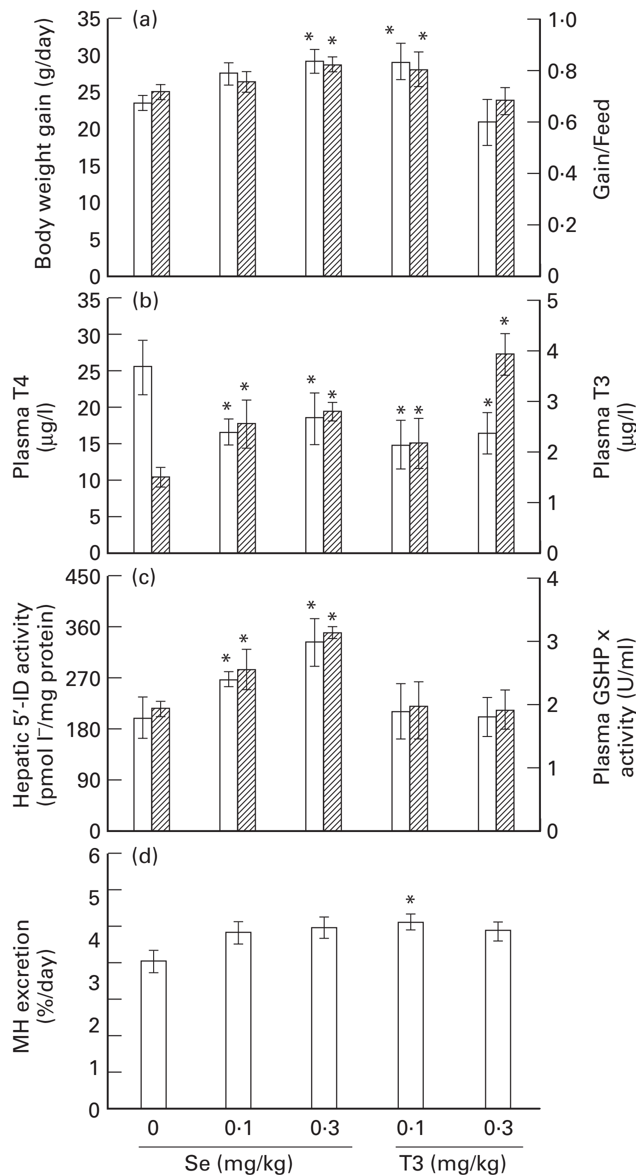

Fig. 3. Effects of dietary Se and 3,5,3′-triiodothyronine (T3) on (a), body weight gain (□) and gain/feed (▨), (b), plasma concentrations of thyroxine (T4) (□) and T3 (▨) and (c), type I iodothyronine deiodinase (5′-ID) activity of liver (□) and gluathione peroxidase (GSHPx) (▨) activity of plasma, and (d), rate of skeletal muscle protein breakdown as measured by N^{τ}-methylhistidine (MH) excretion in broiler chickens. Values are means for seven birds, with their standard errors shown by vertical bars. Mean values were significantly different from the control (0 mg Se/kg diet): *$P < 0.05$.

because only four levels of Se, including the control, were examined, while hepatic 5′-ID activity significantly responded to the dietary Se levels.

Iopanoic acid, a specific monodeiodinase inhibitor (Harrison *et al.* 1996), depressed the hepatic 5′-ID activity when added to the Se-supplemented diet, and decreased the plasma T3 concentrations to the control levels. As expected, the growth promotion due to Se supplementation was completely halted by the addition of iopanoic acid (Fig. 2(a)), indicating that the growth promotion due to Se could relate to the hepatic 5′-ID activity.

More than thirty selenoproteins have been identified (Mitchell *et al.* 1996). Among them, GSHPx and iodothyronine deiodinase were shown to be functional selenoproteins. Se has long been believed to affect growth through the GSHPx activity. GSHPx plays a role in the detoxification of H_2O_2, which protects the cell from injury caused by peroxides. GSHPx activity continues to increase until the Se level has met the needs of the animal. However, it has also been shown that the relationship between GSHPx and growth is not simple (Jensen *et al.* 1986). It is possible that Se affects growth through the influence on 5′-ID activity. Thyroid function is known to be altered by many environmental factors, such as energy intake and dietary composition in addition to ambient temperature. Hypothyroidism may be induced by severe nutrient deficiency, high ambient temperature and anti-thyroidal substances. However, it is not yet clear whether thyroid hormone status induced by ambient temperature, nutrient deficiency etc., influences growth in animals. In the present study, we induced a reduction in plasma T3 concentration by both Se deficiency and administration of iopanoic acid. The impairment of hepatic 5′-ID activity followed by a decreased T3 production were clearly shown in the group of birds who were Se deficient and fed iopanoic acid. Both treatments caused clear growth depression.

Se deficiency caused a decrease of plasma T3 concentrations (Fig. 1(c)), and this might be a reason why growth was impaired in the Se-deficient birds. In fact, lower dietary T3 (0·1 mg/kg) stimulated the growth of Se-deficient birds. Growth impairment was also observed when iopanoic acid was given to the Se-fed birds. However, higher level of dietary T3 (0·3 mg/kg; catabolic dose) depressed the growth of birds as was expected. It is also important that dietary Se had a trend to increase the rate of skeletal muscle protein breakdown. It was shown that T3 increases the skeletal muscle protein breakdown rate in rats (Brown *et al.* 1981).

In conclusion, the present study strongly indicates that dietary Se improves the growth of broilers because Se is needed to synthesize 5′-ID which catalyses the conversion of T4 to T3.

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