Effect of Selenium and Iodine Supplementation on Growth Rate and on Thyroid and Somatotropic Function in Dairy Calves at Pasture

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ABSTRACT

The effects of Se and I supplementation on growth rate and on thyroid and somatotropic function were examined for heifer calves from two herds fed pasture. Supplementation of calves with intraruminal Se pellets increased the basal plasma concentration of 3,5,3′-triiodothyronine and reduced the basal plasma concentration of thyroxine for both herds. For one herd, supplementation with Se increased the triiodothyronine response to challenge with thyrotropin-releasing hormone, increased BW gain, and tended to increase the plasma concentration of IGF-I. The plasma concentration of growth hormone was unaffected by Se supplementation. Supplementation with I increased the response of thyroid hormones to thyrotropin-releasing hormone but did not increase BW gain. Interaction between Se and I treatment within the herds was not apparent for any outcome variable. These data suggest that the effects of Se deficiency in grazing calves may be mediated by alterations in thyroid hormone metabolism but apparently are not mediated through modulation of the peripheral concentration of growth hormone.
(**Key words**: selenium, iodine, thyroid hormones, growth)

Abbreviation key: **GH** = growth hormone, $\mathbf{T_3}$ = 3,5,3′-triiodo-L-thyronine, $\mathbf{T_4}$ = L-thyroxine, **TRH** = thyrotropin-releasing hormone.

INTRODUCTION

Nutritional degenerative myopathy (white muscle disease) and poor growth rate of calves are common manifestations of Se deficiency (17). Although the antioxidant role of Se and vitamin E in the prevention of myopathy is well understood (14), the mechanism by which Se deprivation causes growth retardation has not been established. Growth rate responses to Se supplementation of calves on pasture commonly occur in the absence of signs of muscular degeneration (19), which suggest the existence of a mechanism that does not involve abnormal peroxide metabolism and that is independent of the enzyme glutathione peroxidase.

Type I iodothyronine-5′-deiodinase is an Se-dependent enzyme (5). The deiodinase enzymes are responsible for the deiodination of L-thyroxine ($\mathbf{T_4}$), converting it to its more active form, 3,5,3′-triiodo-L-thyronine ($\mathbf{T_3}$). Type I is the major deiodinase in liver, kidney, and skeletal muscle, and type II is the major deiodinase in brain, pituitary, and brown adipose tissue (11).

The peripheral concentration of T_3 was reduced, and concentration of T_4 was increased, in calves fed a synthetic diet that was deficient in Se compared with concentrations of T_3 and T_4 in calves fed the same diet supplemented with Se (2). In rats fed a diet deficient in Se, similar changes in thyroid hormone concentration were noted (3). Although the peripheral concentration of growth hormone (**GH**) was unchanged, pituitary concentration of GH was lower in rats fed the diet that was deficient in Se than it was in rats fed a diet supplemented with Se. Plasma thyrotropin concentration was increased in rats fed the diet deficient in Se, despite elevated concentrations of total and free T_4 in plasma. The researchers of that study concluded that those changes were consistent with impaired production of T_3 by type II 5′-deiodinase in the pituitary (3) and suggested a role for Se in growth that may be mediated by changes in thyroid hormone metabolism. Precisely how Se affects somatotropic function is unclear; if peripheral concentration of GH is unaffected, perhaps Se deficiency affects mediators of GH action such as IGF-I.

Evidence exists that combined Se and I deficiencies have important metabolic consequences (1). Deficiency of either of these nutrients decreases thyroid I, T_4, and T_3 concentrations in rats, suggesting that Se deficiency could exacerbate the effects of low I intake.

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Although I deficiency of livestock is recognized (4), few studies have investigated the effect of I supplementation on the growth of grazing cattle.

Our objective, therefore, was to test the hypothesis that supplementation with Se, I, or both alters the growth rate and the plasma concentrations of T_4, T_3, GH, and IGF-I in grazing dairy calves.

MATERIALS AND METHODS

Calves

The experiment used 5-mo-old Friesian heifer calves from two herds, herds 1 (n = 90) and 2 (n = 65). The calves grazed predominantly ryegrass (*Lolium perenne*) and white clover (*Trifolium repens*) pasture on their farms of origin. The Se content of the pasture measured between 0.02 and 0.03 mg/kg of DM for both herds. The Se requirement for calves grazing pasture has been estimated to be 0.03 mg/kg of DM (6). The I concentration of the pasture, measured at the start of the experiment, was 0.27 and 0.44 mg/kg of DM for herds 1 and 2, respectively. The I requirement for calves has been estimated to be 0.25 mg/kg of DM (15).

Analysis of Cu concentrations in liver and serum samples previously collected from calves in herd 2 indicated a Cu deficiency (6). All calves in this herd received an s.c. injection of 676 mg of Ca-Cu EDTA (Coprin®; Mallinckrodt Veterinary, Upper Hutt, New Zealand) prior to the start of the trial. The Cu status of herd 1 was adequate based on current recommendations (6). Anthelmintic treatment of the calves consisted of 8 mg/kg of BW of levamisole (Nilverm®; Mallinckrodt Veterinary) or 4.5 mg/kg of BW of oxfendazole (Systamex®; Mallinckrodt Veterinary) every 4 wk for herds 1 and 2, respectively.

Experimental Design

Calves were randomly assigned within herd to one of four groups according to a 2 × 2 factorial design, balanced for age and BW. Treatments were 1) unsupplemented, 2) supplemented with Se, 3) supplemented with I, and 4) supplemented with Se and I.

Calves that were supplemented with Se received two intraruminal Se pellets (Permasel®; Mallinckrodt Veterinary) administered orally. These 30-g intraruminal pellets contained 10% elemental Se in a finely divided matrix of Fe, which released Se at a rate of about 3 mg/d for at least 1 yr (12). Calves that were supplemented with I received an i.m. injection of 4 ml of iodinized poppyseed oil (Lipiodol®; Rhône-Merieux, Wellington, New Zealand). This depot injection provided a total of 1.6 g of I or 4.4 mg/d per calf if I is assumed to be released from the injection site at a constant rate for 1 yr (8).

Blood sampling and BW gain. Prior to supplementation, blood samples were obtained from 8 calves per group in each of the two herds. The same calves were subsequently sampled at 6 and 9 wk following commencement of treatment. On each occasion, blood was drawn from the jugular vein into plain and heparinized tubes and held at 4°C. Heparinized blood was centrifuged within 4 h of collection, and plasma was frozen at –20°C until assays for basal T_3, T_4, GH, and IGF-I were performed. Clotted blood was submitted directly to the Ministry of Agriculture and Fisheries Animal Health Laboratory (Hamilton, New Zealand) for analysis of serum Se. Heifer BW was monitored at the start of the trial and at subsequent sampling dates using electronic scales (AG500; Tru Test, Hamilton, New Zealand).

Thyrotropin-releasing hormone challenge. Nine weeks after commencement of treatment, 4 calves from each treatment group in each herd were chosen using a formal random sampling procedure and were assigned to challenge from thyrotropin-releasing hormone (**TRH**).

The calves were housed inside a building and tethered in individual stalls on grates with rubber matting. Calves were allowed 8 d to adapt to a pelleted lucerne hay diet (Se concentration, 0.02 μg/kg of DM; I concentration, 0.08 mg/kg of DM) that was fed once daily at 1600 h; calves had continuous access to water. The amount of feed offered was 1.2 times maintenance requirements, calculated using the metabolizable energy requirements for calves (10) and BW measured on entry to the building. Adaptation to the diet was assessed by weighing orts. There were no orts after 3 d of the pelleted diet.

Intravenous cannulas, consisting of polyethylene tubing (1 mm i.d.; Dural Plastics and Engineering, Dural, Australia), were placed in the jugular vein of each calf on d 9, and patency was maintained using 100 IU/ml of heparin in sterile, pyrogen-free saline. Feed was withheld the afternoon of d 9. On d 10, 10-ml blood samples were obtained according to the following schedule: –20, –10, –5, 5, 10, 20, 40, 60, 120, 240, 480, 720, and 960 min relative to TRH administration at time zero. Synthetic TRH (TRH®; Roche, Auckland, New Zealand) was diluted in saline to a concentration of 10 μg/ml and administered i.v. at a dose of 0.33 μg/kg of BW (13).

Once collected into citrate anticoagulant tubes, blood samples were immediately placed on ice. The samples were centrifuged within 1 h; plasma was separated and then frozen at –20°C for the thyroid hormone, GH, and IGF-I assays.

Assay of Hormones and Se

Total concentrations of T_3 and T_4 were determined using solid-phase radioimmunoassay kits (Coat-a-Count®; Diagnostic Products, Los Angeles, CA). Sensitivity of the T_3 assay was 0.05 nmol/L, and the half-displacement concentration was 1.94 nmol/L. The intraassay and interassay coefficients of variation were 4.8 and 7.0%, respectively. Sensitivity of the T_4 assay was 1.29 nmol/L, and the half-displacement concentration was 78.9 nmol/L. The intraassay and interassay coefficients of variation were 5.1 and 7.7%, respectively.

The concentration of GH was determined by a double-antibody radioimmunoassay (7) using pituitary-derived bovine GH for iodination with ^{125}I (USDA-bGH-I1; 3.2 IU/mg) and for reference standards (USDA-BGH-B1; 1.9 IU/mg). Sensitivity of this assay was 0.79 ng/ml, and the half-displacement concentration was 15.5 ng/ml. Intraassay and interassay coefficients of variation were 9.1 and 15.8%, respectively.

The concentration of IGF-I was determined for the three baseline plasma samples collected immediately before TRH challenge. The method used a double-antibody radioimmunoassay after acid-ethanol extraction as described elsewhere (9). The sensitivity of this assay was 1.06 ng/ml, and the half-displacement concentration was 3.73 ng/ml. Intraassay and interassay coefficients of variation were 9.3 and 8.5%, respectively.

Assay of serum Se concentration was performed by the Ministry of Agriculture and Fisheries Animal Health Laboratory (Hamilton, New Zealand) using a fluorometric method developed by Watkinson (18).

Statistical Analysis

Analysis was performed using analysis of variance and covariance and the general linear models procedure of SAS (16). Herd, Se treatment, and I treatment were the main effects; Se and I treatment were nested within herd. The initial models tested included terms for interaction between Se and I treatments within herd, but interactions were not significant ($P > 0.1$) for any of the independent variables measured. The final reduced models followed the general equation:

$$Y_{ijk} = \mu + H_i + S(H)_{j(i)} + I(H)_{k(i)} + \beta_l X_{lijk} + e_{l(ijk)}$$

where

Y_{ijk} = dependant variable,
μ = overall mean,
H_i = effect of the herd (i = 1 or 2),
$S(H)_{j(i)}$ = effect of the Se treatment group j within herd i (j = 1 or 2),
$I(H)_{k(i)}$ = effect of the I treatment group k within herd i (k = 1 or 2),
β_l = coefficient of regression of Y on the continuous covariable (X_l), and
$e_{l(ijk)}$ = residual error.

Means for daily BW gain were adjusted by using initial BW as a covariable. Means for the basal plasma concentrations of Se and hormones were adjusted by using the pretreatment values as covariables. The tabulated hormonal responses to TRH for the main effects were calculated as the peak hormone concentration and the area under the entire response curve and above the baseline concentration. Means reported are least squares means and standard errors of the means. Plots of the hormonal response to TRH for each factor combination (Figure 1) were calculated without subtracting the baseline concentration. Instead, the baseline value was used as a covariable.

RESULTS

No interaction between Se and I supplementation was apparent for any of the outcomes measured ($P < 0.1$); therefore, the tabulated results are presented in terms of the main effects only.

Serum Se Concentration and BW Gain

Serum Se concentration of the calves that were not supplemented with Se indicated that these calves remained deficient in Se throughout the study (Table 1). A minimum serum Se concentration of 150 nmol/L (12 ng/ml) is recommended for cattle grazing pasture (6). Serum Se concentration of the calves supplemented with Se increased to a mean of over 800 nmol/L (63 ng/ml). The mean daily BW gain of calves in herd 2 that were supplemented with Se was 20% higher than that of calves receiving no Se ($P = 0.01$; Table 1). Supplementation with Se had no effect on the growth rate of calves in herd 1. Supplementation with I had no effect on serum concentration of Se or the growth rate of calves.

Basal Hormone Concentrations

Basal hormone concentrations in plasma obtained 6 wk after treatment are presented in Table 2. Concentrations of thyroid hormones were higher, and concentration of GH lower, for calves in herd 1 than for calves in herd 2 ($P < 0.05$).

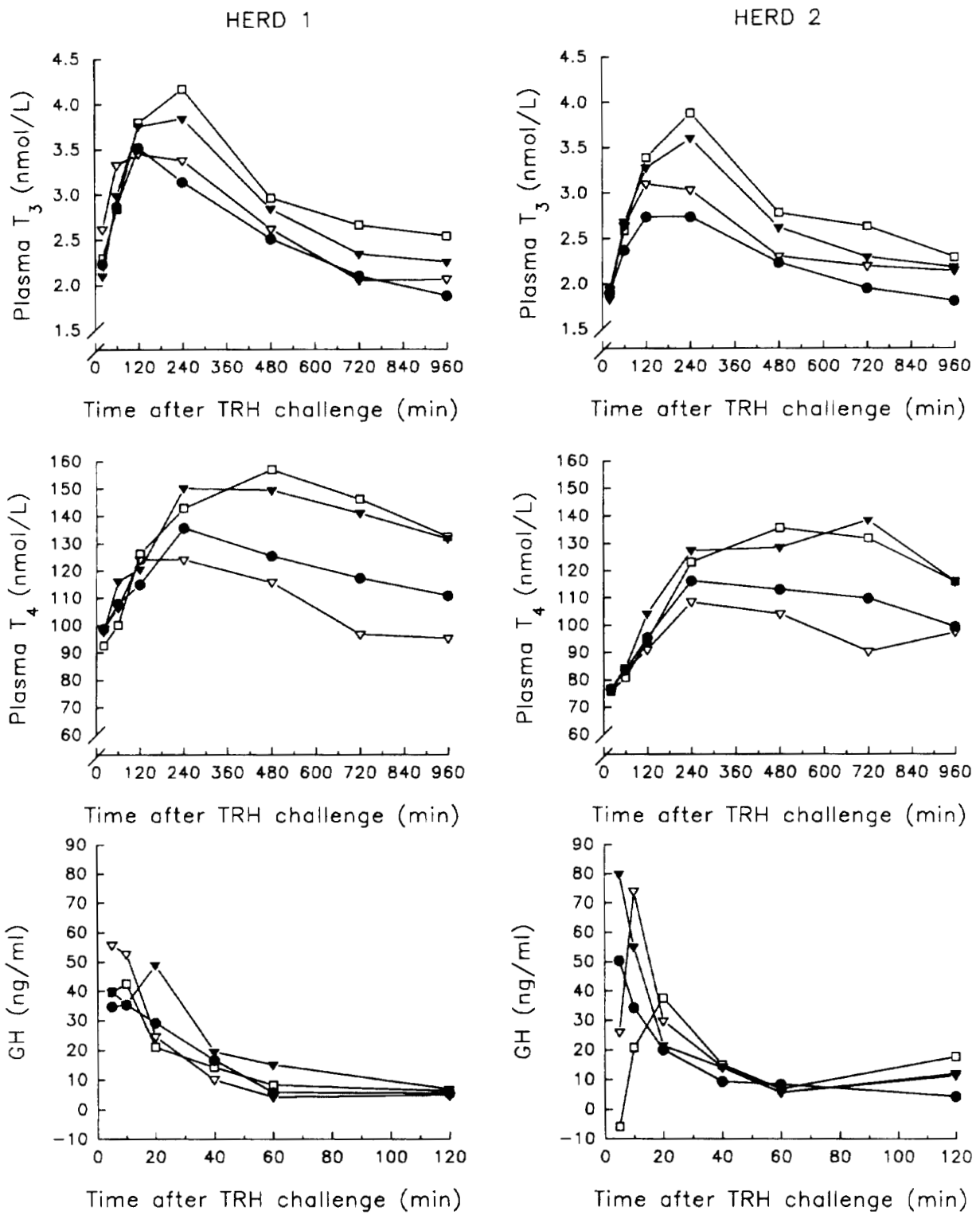

Figure 1. Comparison of the effects of treatment of calves (n = 4 per group for each herd) with two intraruminal Se pellets (▽), 4 ml of 40% I in poppyseed oil injected i.m. (▼), both Se and I (□), or no treatment (●) on plasma concentrations of 3,5,3′-triiodo-L-thyronine (T_3), L-thyroxine (T_4), and growth hormone (GH), following challenge with thyrotropin-releasing hormone (TRH) administered i.v. Curves have been adjusted for differences in baseline values. There was no interaction between Se and I treatments. Pooled SEM of the T_3 response (as measured by the area under the hormone curve) at 240 min was 0.18 and 0.13 nmol/L, pooled SEM of the T_4 response at 720 min was 6.5 and 9.7 nmol/L, and pooled SEM of the GH response at 20 min was 17.8 and 18.3 ng/ml, for herds 1 and 2, respectively.

TABLE 1. Least squares means (LSM) of serum Se concentration and daily BW gain in heifer calves supplemented with Se or I.[1]

	Herd 1					Herd 2				
	Calves	Serum Se		Gain		Calves	Serum Se		Gain	
	(no.)	(nmol/L)[2]		(kg/d)		(no.)	(nmol/L)		(kg/d)	
Treatment		LSM	SE	LSM	SE		LSM	SE	LSM	SE
Se–	40	121.4	25.7	0.79	0.02	27	126.8	25.3	0.56	0.02
Se+	39	825.6	26.6	0.81	0.02	27	857.9	25.1	0.67	0.02
I–	41	485.1	26.5	0.80	0.02	26	483.0	25.0	0.62	0.02
I+	38	461.9	25.7	0.80	0.02	26	501.7	24.9	0.63	0.02
Effect[3]					P					
Se		<0.01		NS			<0.01		<0.01	
I		NS		NS			NS		NS	
Herd							NS		<0.01	

[1]Treatments were Se, two intraruminal Se pellets, and I, 4 ml of 40% (wt/wt) I in poppyseed oil injected intramuscularly; + = supplemented, and – = not supplemented.

[2]Mean serum Se of 16 calves per group 6 wk after commencement of supplementation; nanograms per milliliter = nanomoles per liter/12.67.

[3]NS = $P > 0.10$; interaction of Se and I within herds was not significant.

Supplementation with Se increased plasma T_3 concentration and decreased plasma T_4 concentration for calves in both herds. Plasma GH concentration was unaffected by Se treatment. Supplementation with I had no effect on basal hormone concentrations except for a tendency to increase T_4 concentration for calves in herd 2 ($P = 0.07$).

Response to TRH Challenge

Supplementation with Se increased the baseline concentration of T_3, peak T_3 concentration, and the area under the T_3 response curve in herd 2 ($P < 0.05$; Table 3; Figure 1) but not in herd 1. Peak concentration of T_4 and the area under the T_4 response curve were not affected by Se supplementation of either herd (Table 4; Figure 1), but supplementation decreased baseline T_4 concentration for calves in herd 1 ($P < 0.05$).

Supplementation with I increased peak concentrations of T_3 and T_4 and areas under the curves for T_3 and T_4 for both herds. This effect was in addition to the effect of Se supplementation (Figure 1), and there were no statistical interactions. No clear trend was apparent for baseline concentrations of T_3 and T_4 (Tables 3 and 4).

Baseline plasma GH concentration and the GH response to TRH challenge were unaffected by supplementation (Table 5; Figure 1); however, concen-

TABLE 2. The effect of Se or I supplementation on least squares means (LSM) for basal plasma concentrations of 3,5,3'-triiodo-L-thyronine (T_3), L-thyroxine (T_4), and growth hormone (GH) in heifer calves (n = 16 per group).[1]

	Herd 1						Herd 2					
	T_3		T_4		GH		T_3		T_4		GH	
	(nmol/L)				(ng/ml)		(nmol/L)				(ng/mL)	
Treatment	LSM	SE	LSM	SE	LSM	SE	LSM	SE	LSM	SE	LSM	SE
Se–	2.06	0.11	89.2	3.0	7.72	1.29	1.81	0.11	65.7	3.1	14.13	1.28
Se+	2.48	0.11	77.9	3.3	8.00	1.30	2.13	0.11	56.1	3.0	15.36	1.28
I–	2.27	0.12	80.6	3.3	7.56	1.29	1.88	0.10	57.2	3.1	13.63	1.28
I+	2.27	0.10	86.5	3.1	8.29	1.29	2.06	0.11	64.6	3.0	15.91	1.28
Effect[2]						P						
Se	<0.01		<0.01		NS		0.02		0.02		NS	
I	NS		NS		NS		NS		0.07		NS	
Herd							0.04		<0.01		0.02	

[1]Treatments were Se, two intraruminal Se pellets, and I, 4 ml of 40% (wt/wt) I in poppyseed oil injected intramuscularly; + = supplemented, and – = not supplemented.

[2]NS = $P > 0.10$; interaction of Se and I within herds was not significant.

TABLE 3. The effect of Se or I supplementation on the plasma 3,5,3'-triiodo-L-thyronine (T_3) response to intravenous administration of thyrotropin-releasing hormone in heifer calves (n = 8 per group).[1]

	Herd 1			Herd 2		
Treatment	T_3 Baseline	T_3 Peak[2]	T_3 AUC[3] (nmol/L per min)	T_3 Baseline	T_3 Peak	T_3 AUC (nmol/L per min)
	(nmol/L)			(nmol/L)		
Se–	1.81	1.89	826	1.49	1.52	755
Se+	1.94	2.01	950	1.89	1.85	946
I–	2.02	1.65	674	1.63	1.31	638
I+	1.72	2.24	1102	1.75	2.05	1067
SE	0.11	0.10	57	0.11	0.10	57
Effect[4]	*P*			*P*		
Se	NS	NS	NS	0.02	0.03	0.03
I	0.06	<0.01	<0.01	NS	<0.01	<0.01
Herd				NS	0.02	NS

[1]Treatments were Se, two intraruminal Se pellets, and I, 4 ml of 40% (wt/wt) of I in poppyseed oil injected intramuscularly; + = supplemented, and – = not supplemented.

[2]Peak T_3 concentration above the baseline concentration.

[3]Area under the T_3 curve and above the baseline concentration.

[4]NS = $P > 0.10$; interaction of Se and I within herds was not significant.

tration of IGF-I tended to increase for calves in herd 2 that were supplemented with Se ($P = 0.06$; Table 5).

DISCUSSION

Supplementation of calves with intraruminal Se pellets increased the basal plasma concentration of T_3 and reduced the basal plasma concentration of T_4. This result is in agreement with that of Arthur et al. (2), who reported similar changes in thyroid hormone concentration for supplemented calves fed a synthetic diet that was deficient in Se. In the present study, supplementation with Se increased the T_3 response to TRH challenge, increased BW gain, and tended to increase the plasma concentration of IGF-I for one herd; however, the concentration of GH was unaffected by Se supplementation. Arthur et al. (2) reported no change in BW gain in response to Se

TABLE 4. The effect of Se or I supplementation on the plasma L-thyroxine (T_4) response to intravenous administration of thyrotropin-releasing hormone in heifer calves (n = 8 per group).[1]

	Herd 1			Herd 2		
Treatment	T_4 Baseline	T_4 Peak[2]	T_4 AUC[3] (nmol/L per min)	T_4 Baseline	T_4 Peak	T_4 AUC (nmol/L per min)
	(nmol/L)			(nmol/L)		
Se–	108.4	46.8	30,463	80.6	54.9	35,743
Se+	85.3	50.4	28,125	74.0	47.8	30,487
I–	98.0	37.8	16,990	68.4	39.7	23,181
I+	95.7	59.4	41,597	86.2	62.9	43,048
SE	6.8	5.5	4,358	6.8	5.5	4,358
Effect[4]	*P*			*P*		
Se	0.02	NS	NS	NS	NS	NS
I	NS	0.01	<0.01	0.07	<0.01	<0.01
Herd				<0.01	NS	NS

[1]Treatments were Se, two intraruminal Se pellets, and I, 4 ml of 40% (wt/wt) I in poppyseed oil injected intramuscularly; + = supplemented, and – = not supplemented.

[2]Peak T_4 concentration above the baseline concentration.

[3]Area under the T_4 curve above the baseline concentration.

[4]NS = $P > 0.10$; interaction of Se and I within herds was not significant.

supplementation, despite Se intakes of less than 0.015 mg/kg of DM for the calves in the control group.

The response of T_3 and T_4 to TRH was increased in calves in both herds that were supplemented with I; however, basal concentrations were largely unchanged, and there was no growth rate response to I. This result is in agreement with that of Barry et al. (4), who reported that supplementation with iodinized poppyseed oil resulted in no increase in the BW gain or serum concentrations of T_3 and T_4 for cattle grazing pasture with an I concentration of 0.18 mg/kg of DM. The minimum I requirement for growth of calves fed pasture may be less than the 0.25 mg/kg of DM currently recommended by the NRC (15).

In the present study, plasma GH concentrations (basal or responsive to TRH) were not significantly affected by Se supplementation. This result agrees with the findings of Arthur et al. (3) for rats and suggests that the effects of Se deficiency in grazing calves may not be mediated by alterations in the peripheral concentration of growth hormone. Deficiency of Se could alter somatotropic function through effects on the endocrine or paracrine production of IGF-I, secretion of IGF-II, the number of somatotropic receptors, or the peripheral concentration of IGF-binding proteins. The concentration of IGF-I tended to be increased in calves in herd 2 that had been supplemented with Se.

A BW gain response was noted only for calves in herd 2, despite the similar serum Se concentrations of the two herds (Table 1). Overall, BW gain (Table 1) and thyroid hormone concentrations (Table 2) were lower for calves in herd 2 than for calves in herd 1, but supplementation with I did not affect growth or the hormonal response to Se supplementation for either herd. It is difficult to draw conclusions from comparisons between herds because of the possibility of confounding herd factors.

A lack of I may exacerbate the adverse effects of Se deficiency on thyroid hormone metabolism (3), which may partially explain the inconsistent growth rate response to Se supplementation. An interaction between Se and I may have important implications for livestock grazing pasture grown on soils with low concentrations of both Se and I. No such interaction was noted under the conditions of the present study; however, the intake of I for calves might have been in excess of minimum requirements during this study. Further controlled experimentation is required to determine whether an interaction between Se and I occurs at lower I intakes.

CONCLUSIONS

These results suggest that the adverse effects of Se deficiency on grazing cattle may be mediated by altered thyroid hormone metabolism but not through modulation of peripheral concentrations of GH. Interactions between Se intake and the other determinants of thyroid metabolism, most notably I intake, require further study and may lead to formulation of more precise recommendations for Se supplementation of grazing calves.

TABLE 5. The effect of supplementing heifer calves (n = 8 per group) with Se or I on the plasma growth hormone (GH) response to intravenous administration of thyrotropin-releasing hormone and plasma IGF-I.[1]

	Herd 1				Herd 2			
	GH Baseline	GH Peak[2]	GH AUC[3]	IGF-I[4]	GH Baseline	GH Peak	GH AUC	IGF-I
Treatment	(nmol/L)		(nmol/L per min)	(ng/ml)	(nmol/L)		(nmol/L per min)	(ng/ml)
Se–	7.44	45.1	986	244.9	7.30	38.3	886	179.4
Se+	6.03	60.6	1058	243.4	8.23	42.8	848	210.0
I–	7.80	46.5	688	257.2	7.60	38.0	544	183.7
I+	5.66	59.1	1356	231.1	7.93	43.2	1191	205.7
SE	1.84	8.6	283	10.9	1.84	8.6	283	10.9
Effect[5]	*P*							
Se	NS	NS	NS	NS	NS	NS	NS	0.06
I	NS	NS	NS	NS	NS	NS	NS	NS
Herd					NS	NS	NS	<0.01

[1]Treatments were Se, two intraruminal Se pellets, and I, 4 ml of 40% (wt/wt) of I in poppyseed oil injected intramuscularly; + = supplemented, and – = not supplemented.

[2]Peak GH concentration above the baseline concentration.

[3]Area under the GH curve and above the baseline concentration.

[4]Mean baseline concentration.

[5]NS = $P > 0.10$; interaction of Se and I within herds was not significant.

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