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Selenium Accumulation in Beef: Effect of Dietary Selenium and Geographical Area of Animal Origin

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Selenium (Se) is an essential nutrient with multiple human health benefits; the single most important dietary source of Se is beef. The Se content of beef varies, and cattle fed a high selenium diet may have Se concentrations in beef that are well above average. Such beef is potentially a unique supplemental source of dietary Se. To examine factors affecting Se accumulation in beef, 16 steers (initial wt 374.4 ± 33.7 kg) were taken from seleniferous or nonseleniferous areas and fed in a 2 × 2 factorial design with diets high or moderate in Se (11.9 or 0.62 mg Se/kg diet). Diets contained 50% alfalfa, 25% wheat, and 25% corn on a dry matter basis. All dietary Se was from agricultural products, and Se in the high Se diet was primarily from high Se wheat and alfalfa hay. A loin muscle biopsy was taken at the start of the trial to determine initial Se content of beef. Steers were slaughtered after 14 weeks of the trial, and edible carcass (round, sirloin, shoulder clod, and ribeye) and organ samples were collected. Diets did not affect growth or feed intake ($P > 0.05$), and Se toxicity signs were not observed. Different cuts of meat had similar Se concentrations, and the Se content of all cuts was increased by both high dietary Se and high Se background. Except for liver and kidney, Se in tissues was increased by seleniferous background ($P < 0.02$) and high dietary Se ($P < 0.001$). Kidney Se concentrations of animals fed the high Se diet were lowest in animals from seleniferous areas ($P = 0.04$), suggesting a possible adaptation to the high Se diet. These results demonstrate that cattle fed diets high in Se from agricultural products will accumulate substantial amounts of Se in the beef without developing signs of Se toxicity and that prior Se status regulates Se accumulation in some organs. They further demonstrate that management practices may be altered so as to make beef a significant source of dietary Se.

KEYWORDS: Selenium; seleniferous; beef; diet; accumulation; wheat; alfalfa

INTRODUCTION

Selenium (Se) has been known to be nutritionally essential for mammals for more than 40 years (*1*). Human Se deficiency is not considered a problem in North America; however, there have been many reports of Se deficiency in domestic animals (*2*). Recently, Clark et al. (*3*) reported a substantial decrease in the risk of certain cancers by persons who consumed Se in excess of the dietary reference intake (DRI = 55 μg Se/d) on a daily basis for 10 years, thus, prompting an interest among many people to increase their Se intakes.

We have previously reported that beef, already the single greatest dietary source of Se for North Americans (*4*), can provide high intakes of selenium depending on its geographic origin (*5*). The concentration of Se in beef varies between geographical areas of the U. S. (*6*) and between areas of low and high soil selenium within the same geographical area (*5*). Selenium from beef is highly bioavailable when assessed by its ability to increase liver Se concentrations and glutathione peroxidase activities (GSH-Px) in Se deficient rats, and Se bioavailability is not affected by retail cut of beef (*4*). In a controlled human feeding trial, high intakes of Se partially from beef increased measures of Se status and improved neuropsychological function (*7*).

Because of the variation in Se content, beef from seleniferous areas (areas with high soil Se) may potentially be a means of supplying extra dietary Se. Hintze et al. (*5*) reported that the Se content of beef from a moderately seleniferous area averaged 0.7 μg Se/g; therefore, a 100 g serving of beef would supply 70 μg Se/serving, more than the amount of Se that is recommended by the DRI. Cattle grown in very seleniferous areas could potentially accumulate much more Se, and it is possible that a 200 μg supplemental amount of Se, the same dosage used in the Clark study (*3*), could be obtained from a single moderate portion of beef. Production of such animals could lead to

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Table 1. Diet Composition and Se Content of Dietary Ingredients Fed to 16 Steers Obtained from a Seleniferous or Nonseleniferous Area

ingredient	% of high Se diet	% of low Se diet	Se concn
seleniferous wheat	25[a]	0	8.10 ± 0.06[b]
nonseleniferous wheat	0	25	0.71 ± 0.01
seleniferous hay	45	0	14.26 ± 0.75
nonseleniferous hay	0	45	0.64 ± 0.06
desugared molasses	5	5	0.33 ± 0.01
corn	25	25	0.39 ± 0.07

[a] Percentages of feed ingredients in ration expressed on dry matter basis. [b] Values expressed in mg Se/kg ± SD.

marketing of a specialty crop, and if Se from beef is shown to have anticarcinogenic properties, publicizing the Se content of beef could be used to counteract the increasingly negative nutritional image of beef.

The following paper furthers our investigation of how Se accumulates in beef and conditions that result in high concentrations of Se in beef. Feedstuffs naturally high in Se were fed, in a controlled study, to animals originating from areas of high or moderate forage Se content. We report the pattern of Se accumulation in beef and a possible Se tolerance adaptation of animals raised in seleniferous areas.

EXPERIMENTAL PROCEDURES

Animals and Diet. Sixteen crossbred, yearling steers (initial weight 374.4 ± 33.7 kg) were purchased from two geographic regions. Eight animals were purchased from a seleniferous area of South Dakota that has a history of selenium toxicity and has been found to produce beef high in Se. The remaining eight animals came from the North Dakota State University research herd; beef from animals from this area have been found to have Se concentrations very near the national average (*5*). Animals were assigned to individual pens and fed a diet (**Table 1**) based on alfalfa hay and wheat that contained either normal or high concentrations of Se. High Se alfalfa averaged 14.26 ± 0.75 mg Se/kg dry forage, and high Se wheat averaged 8.1 ± 0.06 mg Se/kg. Animals were allowed access to a Se-free mineral supplement.

Blood and muscle biopsy samples were taken before dietary treatments were imposed to ascertain the initial Se status. Animals were housed in separate stalls and fed individually with ad libitum access to diet and water. Feed consumption was monitored daily, and weekly aliquots of rations were collected for dietary analysis; blood samples were taken every 2 weeks by jugular venipuncture. Animals were fed diets 105 days before slaughter. Blood, kidney, liver, and muscle samples from the round, ribeye, sirloin, clod, shoulder, and diaphragm were collected at the time of slaughter. A veterinarian examined all animals at monthly intervals to determine possible signs of Se toxicity.

Selenium Analyses. Selenium was determined by hydride generation atomic absorption spectrometry according to a previously described procedure (*6*). Briefly, samples were digested in nitric acid with magnesium nitrate present as an aide to prevent Se volatilization. Samples were then ashed in a muffle furnace and resuspended in dilute hydrochloric acid. Selenium determination was by a Perkin-Elmer 5100 AAS with a continuous flow hydride generator. Quality control was maintained by running bovine liver standard (NBS standard 1577b). Glutathione peroxidase was determined by the method of Paglia and Valentine (1967) with hydrogen peroxide as the substrate.

Plasma Se Fractionation. Selenium distribution among plasma proteins was determined by the dual-column method of Deagen et al. (*9*), and fractions generated from column elution were analyzed for Se by a fluorometric procedure. Plasma was pooled across all animals within a treatment.

Statistical Analyses. Data were analyzed as a 2 × 2 factorial design using the general linear models produced in PC SAS. Tukey's studentized range test was used to separate treatment means (*10*).

RESULTS

Performance Measures. Diet and background Se status did not significantly affect feed intake, average daily gain (ADG), or final weight (**Table 2**). Total Se intake averaged 11.6 ± 0.2 or 0.70 ± 0.3 g Se for the high and moderate Se dietary groups, respectively. Monthly veterinary examinations did not detect any signs of selenosis in any animal.

Distribution of Plasma Se. Steers from a seleniferous background fed the high Se diet had plasma Se predominately associated with GSH-Px (**Table 3**) and lesser and approximately equal amounts associated with selenoprotein P (SeP) and albumin. Conversely, nonseleniferous background steers fed the moderate Se diet had plasma Se primarily associated with SeP. SeP represented the majority of the plasma Se in seleniferous background steers fed the moderate Se diet and nonseleniferous background steers fed the moderate-Se diet.

Selenium Accumulation in Tissues and Organs. Initial plasma Se concentrations averaged 555 ± 25 and 122 ± 27 ng Se/mL for animals from seleniferous and nonseleniferous backgrounds, respectively (**Figure 1**). Plasma Se concentrations in animals from the nonseleniferous region fed the moderate Se diet did not change throughout the study; likewise, there was very little change in animals from the seleniferous area fed the high Se diet. However, plasma Se in animals from a nonseleniferous background fed the high Se diet immediately began to increase, and after 2 months of feeding, plasma Se concentrations were similar to animals from the seleniferous area fed the high Se diet. Plasma Se from animals from a seleniferous background fed a moderate Se diet began to decline immediately and by the end of the study were not different from animals from a nonseleniferous background fed moderate Se diets. The majority of the change in plasma Se occurred during the first month of the study.

Initial whole blood GSH-Px activities (**Figure 2**) were less affected by area of origin and averaged 514 ± 36 and 314 ± 39 EU/mg HB for the seleniferous and nonseleniferous areas, respectively. Glutathione peroxidase activities in animals fed the high Se diet, regardless of background, increased throughout the feeding trial. Glutathione peroxidase activities in animals fed the moderate Se diet moved toward a mean, and by the end of the study, the average GSH-Px activity of both groups fed the moderate Se diet was identical.

Initial muscle Se concentrations (**Table 4**) averaged 2.1 ± 0.1 and 0.4 ± 0.1 mg Se/kg for animals from seleniferous and nonseleniferous backgrounds, respectively. The final Se concentration in all retail cuts of beef was significantly affected by diet and Se background, and Se concentrations were similar for all cuts. The Se concentrations of all organs except liver were significantly affected by Se background and dietary Se intake.

The rank order of Se concentrations in edible beef was seleniferous background/high Se diet (2.10 ± 0.39 μg Se/g) > seleniferous background/moderate Se diet = nonseleniferous background/high Se diet > nonseleniferous background/moderate Se diet (0.43 ± 0.09 μg Se/g). Selenium concentrations in the liver and kidney did not follow this pattern. Kidney and liver Se concentrations of nonseleniferous background steers fed a high Se diet were higher than kidney and liver Se concentrations of seleniferous background steers fed a high Se diet.

DISCUSSION

Numerous studies have investigated the effects of feeding supplemental Se, primarily the inorganic salts sodium selenite and selenate, to livestock with the goal of preventing Se

Table 2. Average Daily Gain (ADG), Feed Intake, and Se Intake of 16 Steers Obtained from a Seleniferous or Nonseleniferous Area and Fed Diets Either High or Adequate in Se

	animal origin						
dietary Se	seleniferous region		nonseleniferous region		ANOVA		
(mg Se/kg)	11.9	0.62	11.9	0.62			
N	4	4	4	4	diet	bkgrnd	interaction (DXB)
initial wt (kg)	390 ± 47[a]	387 ± 16	360 ± 41	355 ± 5	NS[b]	NS	NS
final wt (kg)	493 ± 59	498 ± 22	467 ± 38	474 ± 31	NS	NS	NS
ADG[c]	1.1 ± 0.23	1.14 ± 0.14	1.14 ± 0.18	0.95 ± 0.45	NS	NS	NS
feed intake (kg)[d]	950 ± 60	1014 ± 50	915 ± 109	959 ± 157	NS	NS	NS
Se intake (g)[e]	11.8 ± 0.7	0.7 ± 0.3	11.4 ± 1.1	0.6 ± 0.1	0.0001	NS	NS

[a] Values are means ± SD. [b] Not significant (NS). [c] ADG (kg/d). [d] Feed intake is total amount of feed consumed throughout the trial. [e] Se intake is total Se consumed throughout the trial.

Table 3. Distribution of Selenium among Plasma Proteins of Cattle from Regions of Different Se Background and Fed Diets Either High or Adequate in Se as Determined by the Tandem Column Chromatography System of Heparin–Sepharose and Reactive Blue Method of Deagan et al. (1993)[a]

	animal origin			
dietary Se	seleniferous region		nonseleniferous region	
(mg Se/kg)	11.9	0.62	11.9	0.62
N	4	4	4	4
plasma Se (ng)	491	142	311	108
GSH-Px %	45	32	34	27
albumin %	25	11	26	26
SeP %	24	48	32	40

[a] Samples were from the final blood collection and were pooled by treatment.

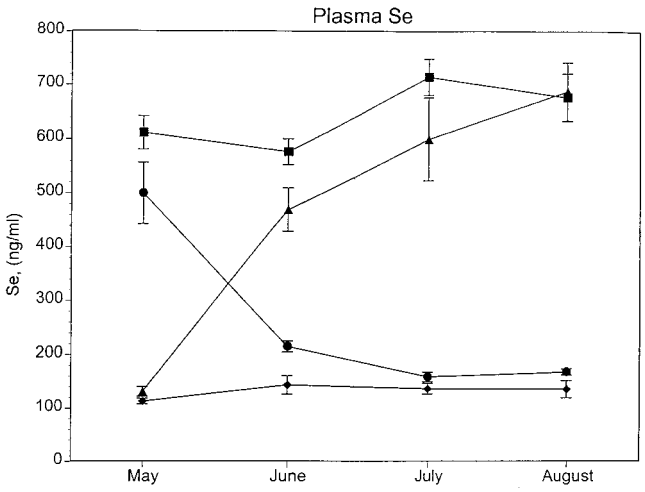

Figure 1. Plasma Se concentration of steers from a seleniferous area fed a high Se diet (■); seleniferous area fed a moderate Se diet (●); nonseleniferous area fed a high Se diet (▲); or a nonseleniferous area fed a moderate Se diet (◆). Values are means ($N = 4$) ± standard error.

deficiency (*11*−*13*). Relatively few studies have examined Se accretion in beef of animals fed feedstuff sources of Se such as high Se hay or wheat. We have previously demonstrated that cattle originating from seleniferous areas produce beef with higher concentrations of Se than cattle originating from nonseleniferous areas (*5*). Animals used in the previous study were cull cows from differing geographic regions raised under different management conditions and forage availability. In the present study, cattle were kept under defined conditions and fed feedstuffs that varied only in Se concentration. Results of this study clearly demonstrate that short-term feeding of beef

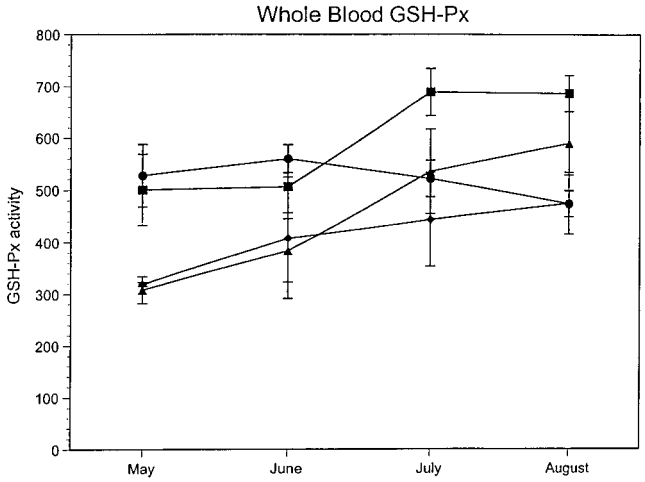

Figure 2. Whole blood glutathione peroxidase activities of steers from a seleniferous area fed a high Se diet (■); seleniferous area fed a moderate Se diet (●); a nonseleniferous area fed a high Se diet (▲); or a nonseleniferous area fed a moderate Se diet (◆). Values are means ($N = 4$) ± standard error.

steers with a ration containing more than 10 mg Se/kg results in a substantial accumulation of Se in the muscle, and such a feeding regimen does not result in any signs of selenosis.

Although animals from seleniferous areas fed a high Se diet finished the study with the greatest concentration of Se in the edible beef, Se concentrations in muscle actually declined over the course of the feeding period. It is possible that the test diet was lower in Se than the natural diet consumed the previous year. However, this is doubtful because we have extensively sampled the high Se region that the steers were obtained from and have never found Se concentrations in forage as great as that used in the high Se ration. We infer that there is a maximum amount of Se that can be put into muscle, and the muscle was probably near saturation before feeding began. Assuming that 58% of carcass weight is muscle and Se is evenly distributed throughout carcass muscle, 580 mg of total Se was present in muscle of animals from a seleniferous background fed the high Se diet before feeding, and 621 mg was present at slaughter, an increase of 41 mg. Conversely, animals from a nonseleniferous background fed the high Se diet contained 110 mg of muscle Se before the study began and 406 mg at slaughter, a 3.7-fold increase (**Table 5**).

The transfer of dietary Se to muscle in steers from a seleniferous area fed the high Se diet was inefficient. These steers consumed approximately 11 841 mg of Se throughout the feeding period, but only 0.34% (41 mg) was retained in muscle. Steers from the seleniferous area fed the moderate Se

Table 4. Se Concentrations in Organs and Tissues of Cattle from Regions of Different Se Background and Fed Diets Either High or Adequate in Se

	animal origin						
dietary Se	seleniferous region		nonseleniferous region		ANOVA		
(mg Se/kg)	11.9	0.62	11.9	0.62			
N	4	4	4	3	diet	bkgrnd	interaction
biopsy[a,b]	2.48 ± 0.66[a]	1.71 ± 0.44[a]	0.51 ± 0.07[b]	0.35 ± 0.07[b]	NS	0.0001	NS
round	2.10 ± 0.39[a]	1.22 ± 0.14[b]	1.45 ± 0.23[b]	0.43 ± 0.09[c]	0.0002	0.0001	NS
clod	2.16 ± 0.37[a]	1.21 ± 0.11[b]	1.56 ± 0.24[b]	0.40 ± 0.07[c]	0.0002	0.0001	NS
sirloin	2.05 ± 0.39[a]	1.19 ± 0.05[b]	1.61 ± 0.19[a,b]	0.40 ± 0.05[c]	0.0003	0.0001	NS
ribeye	2.06 ± 0.22[a]	1.20 ± 0.08[b]	1.50 ± 0.27[b]	0.35 ± 0.07[c]	0.0001	0.0001	NS
diaphragm	1.91 ± 0.01[a]	0.64 ± 0.08[c]	1.42 ± 0.34[b]	0.31 ± 0.05[c]	0.0013	0.0001	NS
liver	4.69 ± 1.04[a]	0.97 ± 0.14[b]	5.94 ± 2.35[a]	0.89 ± 0.15[b]	NS	0.0001	NS
kidney	3.58 ± 0.42[b]	2.12 ± 0.12[c]	4.73 ± 0.79[a]	2.45 ± 0.54[b,c]	0.02	0.0001	NS

[a] Means are mg Se/kg wet tissue ± standard deviation. [b] Means with different superscripts within same row are significantly different from each other ($P < 0.05$).

Table 5. Conversion of Dietary Se to Edible Muscle Se of Cattle from Regions of Different Se Background and Fed Diets Either High or Adequate in Se

	animal origin			
dietary Se	seleniferous region		nonseleniferous region	
(mg Se/kg)	11.9	0.62	11.9	0.62
N	4	4	4	3
initial muscle Se (mg)	580.3	397.1	110.2	74.6
final muscle Se (mg)	621.2	364.5	406.3	91.6
total Se intake (g)	11.8 ± 0.7[a]	0.7 ± 0.03	11.4 ± 1.1	0.7 ± 0.1
% dietary Se to muscle	0.34	0	2.6	2.6

[a] Values are means ± standard deviation.

diet had a net loss of total carcass muscle Se. They started the trial with 397 mg and ended at 365 mg. In contrast, steers from the nonseleniferous area, regardless of dietary treatment, converted 2.6% of dietary Se to muscle Se (see **Table 5**).

There was a much greater effect of dietary Se on plasma Se concentrations than on whole-blood GSH-Px activities. This was because plasma Se is related to short-term Se intake, whereas whole blood GSH-Px is more related to long-term status. The effect of dietary Se on plasma Se accumulation was further examined by separation of Se contained in individual proteins. Under most conditions, SeP, albumin, and plasma GSH-Px contain the majority of Se in serum and SeP accounts for approximately 60% of plasma Se (*20*, *21*). In the present study, SeP concentrations fluctuated greatly among treatments and was the largest pool of Se only in animals fed the moderate Se diet. The largest pool of Se in animals fed high Se diets, especially those from a seleniferous background, was in plasma GSH-Px. In contrast, Holstein calves used by Awadeh et al. (*22*) had increased SeP and decreased GSH-Px when fed high Se diets as selenite, while steers fed adequate Se diets had decreased SeP and increased GSH-Px. This difference probably reflects differences in metabolism of Se salts vs organic forms of Se.

A surprising finding was that the greatest concentrations of Se in the kidney and liver were not found in animals from the seleniferous area fed the high Se diet but rather from animals from the nonseleniferous area fed the high Se diet. This implies that animals from seleniferous areas have developed physiologic adaptations that allow them to retain a much lower percentage of dietary Se. Selenium is excreted after methylation by S-adenosylmethionine (SAM). Hasegawa et al. (*23*) reported that mice treated with 20 mg/kg selenocysteine had significantly lower liver methionine adenosyltransferase activity and suggested that SAM biosynthesis may be a limiting factor for Se excretion. Perhaps cattle from seleniferous areas adapt to high Se intakes by upregulation of Se methylation through increased SAM biosynthesis.

If the goal is to produce beef with high concentrations of Se, would it not be easier to add inorganic Se to feedlot rations rather than feed high Se feedstuffs? Several factors argue against this. First, Se is not on the Generally Recognized As Safe list and therefore cannot, without a veterinarian's recommendation, be added to rations beyond 0.25 mg Se/kg diet. Second, many people perceive Se as a toxin, but the consumer is interested in consuming foods that have high amounts of a "healthy nutrient". If rations were formulated with high concentrations of exogenous Se, eventually there would be publicity that feedlots were adding a toxin to their diet. Such publicity would further the "negative image" of meat and not enhance it. Finally, more Se from organic sources is incorporated into tissue than inorganic sources of Se (*12*, *14*, *15*).

The chemical form of Se consumed by the animals used in this study was not determined, but many assume that the speciation of Se in forages is similar to that in wheat grain (i.e., selenomethionine; SeMet) (*16*). In actuality, it is unclear what form of Se predominates in grass. Peterson and Butler (*17*) demonstrated that 60−80% of Se in pasture grass was associated with the protein fraction (probably SeMet) and 20−30% was in the form of seleno-amino acids. Wu et al. (*18*) demonstrated that grasses contain several different Se species including Se-methylselenocysteine, SeMet, and selenocysteine. Overall, the predominate form of Se found in the high Se forages used in this study was most likely SeMet, which has been demonstrated to be more bioavailable than inorganic Se (*19*). This accounts for the large effect of geographical area of origin on Se content of beef; areas where Se in soil is taken up by plants that are subsequently consumed by cattle produce beef high in Se (*5*). Thus, the most practical means of producing high Se beef is by feeding feedstuffs naturally high in Se. Because most nutritional guidelines suggest reducing the intake of red meat, there is a substantial nutritional implication to production of high Se beef as in this study. A modest serving of beef is approximately 100 g, and thus, a serving of high Se meat from this study would provide 200 μg of Se, which is the same amount of Se considered to be protective against colon, lung, and prostate cancer by Clark et al. (*3*). The form of Se certainly has a major effect on its efficacy against cancer (*24*); thus, the actual health benefits of high Se beef depend on results of specific studies and characterization of the chemical form of Se found in high Se beef. Red meat has been implicated as a risk factor for colon cancer (*25*); it would be of interest to determine if high Se beef could mitigate that risk.

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