Discovery, The Student Journal of Dale Bumpers College of Agricultural, Food and Life Sciences

Volume 6 Article 6

Fall 2005

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ABSTRACT

Selenium (Se) is deficient in many Arkansas soils; therefore, an experiment was conducted on steers to evaluate the effects of two supplemental Se sources on performance, blood metabolites, and immune function. Thirty Angus-crossbred steers were blocked by weight and assigned within block to one of 15 pens (two steers/pen). Pens were assigned randomly within blocks to one of three dietary treatments consisting of a corn-soybean meal supplement devoid of supplemental Se (negative control, NC) or corn-soybean meal supplements providing 1.7 mg supplemental Se/d as sodium selenite (inorganic Se, ISe) or as Se yeast (organic Se, OSe). Steers were offered fescue hay to allow for approximately 10% refusals, and 1.1 kg/d (as fed basis) of the appropriate grain supplement. Level and source of supplemental Se did not affect average daily gain for the 105-d trial. By d 42, steers fed both sources of supplemental Se had greater blood Se concentrations than those fed the NC. On d 63 and 84, blood Se concentrations differed among all dietary treatments (NC < ISe < OSe), and on d 105 steers fed both sources of supplemental Se had greater blood Se concentrations than NC. Antibody response to vaccination for bovine respiratory viruses, or in vitro lymphocyte blastogenesis did not differ among steers fed the different diets. Both sources of supplemental Se increased blood Se concentrations, the organic source more rapidly than the inorganic source; however, Se level and source had minimal effects on immune function of weaned beef steers.

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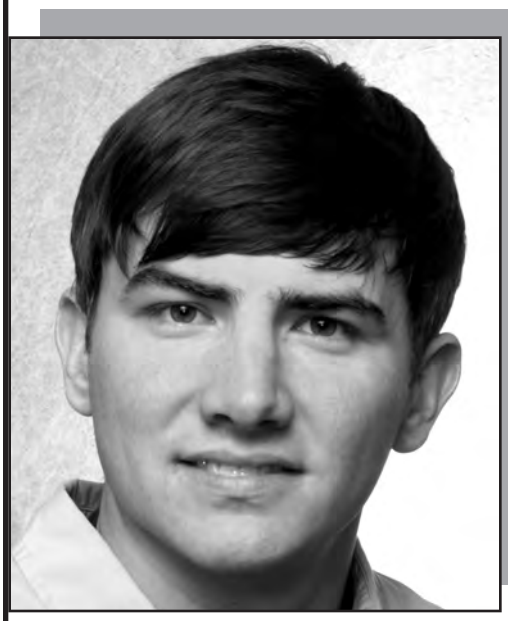
MEET THE STUDENT-AUTHOR



R. Scott Fry

I graduated from Quitman High School in May 2001, and I began my college career at the University of Arkansas in August 2001. I come from a strong agricultural background in rural Arkansas. I have always had a great deal of knowledge about the production aspects of the cattle industry, and I saw an honors research project as a way to explore the world of research in agriculture.

This research project helped me obtain an internship position at USDA-ARS in Beltsville, Md., where I was involved in research with reproductive physiology in turkeys. Experiences like these have really brought out in me a great appreciation for research. I feel that the educational experiences and my involvement on campus at the U of A have really opened many windows of opportunity for me. The U of A and the Bumpers College have proven to be outstanding scholarly environments in which to become more knowledgeable and prepared for the real world.

I am a senior and will graduate in May 2005, and I plan to attend graduate school in the field of animal science. I plan to focus my research on ruminant nutrition and the immune function of beef cattle.

I would like to thank Dr. Beth Kegley and Dr. Ellen Davis for their guidance in my research project. Also I would like to acknowledge Michael Ratcliff, Doug Galloway, and Pete Hornsby for their assistance in the completion of the selenium trial. I would also like to thank Alltech Inc. for the donation of their product. All involved are greatly appreciated.

INTRODUCTION

Selenium deficiency is a serious problem in livestock production worldwide. Severe cases can be found in Finland, New Zealand, China, Japan, and the United States. The most Se-deficient areas in the United States are found in the Northwest, Southeast, and the Northeast, including many of the states adjoining the Great Lakes (McDowell, 2003). Large areas of Arkansas' soil are deficient in trace minerals, including Se. With these problems in mind it is important to pay attention to the effects Se deficiency has on the agricultural/beef industry in Arkansas. Arkansas ranks fifteenth nationally in beef cattle production with a cattle population exceeding 1.8 million on 30,000 farms (Arkansas Farm Bureau, 2003). Arkansas' cattle production is mainly a cow/calf system with some grazing of feeder and stocker animals that are ultimately shipped to the high plains and Midwest for finishing and processing. It is very important that producers in Arkansas, as well as throughout the U.S., manage mineral deficiency problems.

Cattle consuming forages from Se-deficient soils are at risk of developing Se deficiency-associated disorders, including white muscle disease; ill thrift syndrome; reproduction dysfunction (neonatal weakness, retained placenta, calf scours, calf pneumonia, abortion, stillbirths, decreased fertility); and diarrhea (McDowell, 2003). Selenium functions to prevent oxidative damage to body tissues (Hoekstra, 1974), and Se deficiency can inhibit antibody production to foreign challenges (Mulhern et al., 1985) and the detoxification of certain toxins (Burk, 1983). Studies have indicated that calves can be severely depleted of Se and Se-dependent glutathione peroxidase (Arthur, 1981) yet exhibit no clinical signs until they are subjected to a stress.

Selenium deficiencies are frequently subclinical, and for this reason, the lack of this mineral may limit calf

performance without the producer being aware of the deficiency. Research shows that Se deficient cattle grow slower and are less efficient than non-deficient cattle (McDowell, 2003). Selenium deficient cattle can also be unresponsive to medication (McDowell, 2003). Borderline deficiencies are often very costly to the producer. Estimates indicate that the use of Se in all animal diets would save about one billion dollars annually in the United States (Phillips et al., 1989).

Different mineral sources differ in bioavailability, which is the proportion of the mineral in the feed that is absorbed and utilized by the animal. Organic Se is potentially a more bioavailable source of Se; however, currently research is limited in this area. The objectives of this study were to determine the effects of supplemental Se from two sources on growth and immune function of weaned beef steers.

MATERIALS AND METHODS

Thirty weaned steers weighing between 215 and 279 kg were obtained from the University of Arkansas Beef Cattle Facility, Savoy. Steers were grazed for 80 d without any supplemental Se, with the intention of them being marginally deficient in Se at the beginning of the trial on 22 Jan., 2004.

Steers were weighed on two consecutive days at the beginning of the trial. Steers were blocked by weight (five blocks), and within the block they were assigned randomly to a pen (three pens/block, two steers/pen). Steers were housed in 3.7 m by 30 m pens with a 3 m concrete feedbunk in the front of each pen. Water was available ad libitum.

Three dietary treatments were used, 1) a control diet (no supplemental Se), 2) the control diet plus 1.7 mg/d supplemental Se from an inorganic source as sodium selenite, or 3) the control diet plus 1.7 mg/d supplemental Se from an organic source as a high Se yeast (Sel-Plex, Alltech, Nicholasville, Ky.). A grain supplement was fed at 1.1 kg/d on an as-fed basis (Table 1). Grain supplements were mixed three times, on d 0, 32, and 66. The grain supplement met the NRC (1996) requirements for protein, vitamins, and other minerals. Grain supplements were sampled at all mixing times. Fescue hay was available ad libitum to allow approximately 10% refusals and feed intake was recorded daily. Each bale of hay was sampled and five composite samples made over the 105 d trial. Hay was fed once daily at 0800 h and the supplement was fed twice daily at 0800 and 1600 h.

Steers were weighed prior to feeding every 21 d and weights were taken on two consecutive days at the end of the trial. Blood was drawn from each steer every 21 d to determine the concentration of Se and the activity of glutathione peroxidase (d 1, 21, 42, 63, 84, and 105). Blood samples were taken via jugular veinpuncture into 10-mL heparinized tubes (BD Vacutainer‘, Franklin Lakes, N.J.). The steers were observed daily for any signs of Se deficiency symptoms.

Blood samples were used to monitor immune function by analysis of in vitro lymphocyte proliferation, macrophage phagocytosis, and in vivo antibody production. On d 98, blood was collected for lymphocyte proliferation assays. Isolation, incubation, labeling with tritiated thymidine, and cell harvesting for lymphocyte proliferation followed the procedures by van Heugten et al. (1994). Cells were incubated for 48 h at 39°C with 5% CO_2, tritiated thymidine was added to each well, and cultures were incubated for an additional 18 h. Cells were harvested on glass fiber mats and the radioactivity was measured as counts/minute on a liquid scintillation analyzer (TRI-CARB 2200CA, Packard Instrument Co., Downers Grove, Ill.).

On d 38 and 98, monocytes were isolated and phagocytic ability was determined by methods adapted from Monteleone et al. (1999) and Nibbering et al. (1987). In short, peripheral blood mononuclear cells were isolated. Two milliliters of cells suspended in LM Hahn medium (2×10^6 cells/mL) were incubated for 16 h at 39°C in duplicate in six-well plates containing a glass coverslip. Isolation of monocytes occurred because of the propensity of the cells to adhere to the glass surface of the cover slip. Following the 16 h incubation, excess medium was removed, and 2 mL of a 5% porcine-red blood cell (PRBC) suspension were added to the monocytes/macrophages remaining on the glass coverslips. Cells were incubated with PRBC at 39°C and 5% CO_2 for 2 h, after which the coverslips were removed and rinsed with warmed LM Hahn medium to remove non-adherent cells and non-phagocytized PRBC. Cells were then fixed on coverslips and stained (Heme-3, Fisher Scientific, Pittsburgh, Penn.) for visualization under a microscope at 100x. The percentage of phagocytic monocytes and number of PRBC consumed by each phagocytic monocyte were determined by visual assessment of 200 monocytes on each duplicate coverslip.

Glutathione peroxidase (GTH-Px) was measured in whole-blood samples collected through jugular veinpuncture into 10-mL heparinized tubes. The red blood cells (RBC) were washed three times in an isotonic saline solution. The samples were then frozen in 5-mL tubes containing four parts RBC and one part deionized water. Glutathione peroxidase activity was expressed in enzyme units, and results were expressed as units per gram of hemoglobin (Hb). Determination of GTH-Px was performed using a commercial assay kit (OxisResearch™

BIOXYTECH®GPx-340™, Portland, Ore.). Hemoglobin was analyzed via commercial assay (Hemoglobin B, Wako Chemicals USA, Inc., Richmond, Va.).

To measure antibody production, all steers were vaccinated on d 42 with a modified live viral vaccine for infectious bovine rhinotracheitis virus (IBR), parainfluenza 3 (PI3), bovine viral diarrhea (BVD), and bovine respiratory syncytial virus (BRSV) (Titanium, AgriLabs LTD, St. Joseph, Mo.). Blood samples were taken via jugular venipuncture into plain glass tubes prior to vaccination on d 42 and on d 63 (d 21 after vaccination). Steers were revaccinated on d 63 to evaluate the secondary immune response and samples taken on d 70, 77, and 84. Blood samples were stored on ice, taken to the lab, and centrifuged. Serum was stored frozen until analysis for IBR, PI3, BVD, and BRSV. Samples were sent to the Oklahoma Animal Disease Diagnostic Laboratory for analysis.

Steer weights, blood and serum Se, GTH-Px activity, lymphocyte proliferation, macrophage function and antibody production were statistically analyzed using the mixed procedures of SAS (SAS Inst., Cary, N.C.). The experimental unit was pen. The random statement included block, and repeated statement was used for blood data. Least squares means were separated using pair-wise t-test when the F-test was significant ($P < 0.05$).

RESULTS AND DISCUSSION

Negative control supplements mixed on d 0 and d 32 had Se concentrations (dry-matter basis) of 0.22 mg/kg; however, the control supplement mixed on d 66 had Se concentration of 1.14 mg/kg. The hay had Se concentrations ranging from 0.13 to 0.2 with a mean of 0.16 mg/kg. These levels were higher than the analysis received of 0.036 mg/kg on core samples taken prior to the study. When supplement and hay intakes were calculated, the negative control steers were receiving an average of 0.17 mg Se/kg of diet until d 67 when the control diet contained approximately 0.36 mg Se/kg of diet, thus the Se concentrations in all diets were greater than the NRC (1996) requirement for Se of 0.1 mg/kg. There were no symptoms of Se deficiency or morbidity recorded.

There were no differences ($P > 0.10$) between steers fed the different dietary treatments for average daily gain, dry-matter intake, and gain-to-feed ratio (Table 2). Phillips et al. (1989) gave Se boluses to calves grazing Se-deficient forage and reported no positive effect on average daily gain until after d 60; however, blood Se concentrations were increased by d 30 with the Se boluses.

The analysis of blood Se (Fig. 1; day x treatment interaction, $P < 0.001$) revealed that on d 0 the steers to be fed organic Se had the lowest initial blood Se ($P < 0.05$). On d 21 there were no differences in blood Se concentrations between steers fed the different diets. By d 42, steers fed both sources of supplemental Se had greater ($P < 0.05$) blood Se concentrations than those fed the negative control diet. On d 63 and 84, blood Se concentrations differed ($P < 0.05$) among all dietary treatments (with negative control < inorganic Se < organic Se). Also, the increase in Se in the grain supplement on d 66 related to an increase in all blood Se concentrations on d 84 and 105. On d 105, steers fed both sources of supplemental Se had greater ($P < 0.01$) blood Se concentrations than the negative control. Beck et al. (2005) reported that supplementation of calves with Se yeast increased Se concentration in whole blood by 3.1 times compared with the negative controls and 1.6 times compared with calves fed inorganic Se.

The serum Se (Fig. 2; day x treatment interaction, $P < 0.001$) concentrations on d 0 were similar to that of the blood Se concentration with the steers to be fed organic Se being lower than that of the steers to be fed the control diet ($P < 0.05$). On d 21, 42, and 63, steers fed both sources of supplemental Se had greater ($P < 0.05$) serum Se concentrations than those fed the negative control diet. On d 84, serum Se concentrations differed among all the dietary treatments ($P < 0.05$), with the steers fed the organic source having the highest concentration and the steers fed the control diet having the lowest concentrations. On d 84 and 105, an increase occurred that was closely related to that of the increase in Se concentration seen in the grain supplement after mixing on d 66. On d 105, the steers fed the inorganic source had higher concentrations of serum Se than those of steers fed the negative control diet ($P < 0.05$).

Glutathione peroxidase (GTH-Px) activity (Fig. 3; day x treatment, $P < 0.05$) differed on d 0 compared to that of the blood- and serum-Se concentrations, when the steers to be fed the organic Se had higher GTH-Px activity than steers to be fed the negative control diet ($P < 0.05$). On d 21, 63, and 84 there were no differences in GTH-Px activity between the dietary treatments. However, on d 42 there was an increase in the GTH-Px activity in steers fed the negative control diet ($P < 0.05$), and on d 105 steers fed the organic source had higher GTH-Px activity than those steers fed the negative control ($P < 0.05$). Beck et al. (2005) found that supplemental Se did not enhance glutathione peroxidase activity. This variation may reflect the slower turnover rate of GTH-Px in the red blood cell compared to serum Se concentrations, which respond to Se supplementation more rapidly (Underwood and Suttle, 1999).

Lymphocyte proliferation on d 98 (Table 3) was not affected by the dietary treatments ($P < 0.48$). Beck et al.

(2005) found that there were no differences (P > 0.47) in lymphocyte proliferation with either unstimulated cultures or cultures stimulated with ConA, PWM, or PHA. Supplemental Se has resulted in varying effects on lymphocyte proliferation of ruminants as reported in the literature (Beck et al., 2005). Cao et al. (1992) reported that Se deficiency in dairy cows resulted in a decreased response of isolated peripheral blood lymphocytes to mitogen stimulation using ConA. Low dietary Se does not consistently affect cell-mediated immune response in ruminants (Stabel and Spears, 1993).

The functions of macrophages are very critical to the immune function of the animal. The function of macrophages (called monocytes when found in blood) is to engulf foreign materials that are invading the system. There was a tendency (P = 0.15) for diet to affect the number of PRBC ingested in vitro (Table 3). Macrophages from steers supplemented with inorganic Se phagocytized more (P = 0.06) porcine red blood cells than did macrophages from negative control steers, with macrophages from organic Se-supplemented steers being intermediate. However, the percentage of phagocytic macrophages was not affected by diet. In contrast, Beck et al. (2005) found supplemental Se did not affect the number of PRBC ingested by macrophages; but the percentage of phagocytic macrophages was increased by supplemental Se yeast as compared with the control or with supplemental sodium selenite.

Antibody response to vaccination for bovine respiratory viruses did not (P > 0.35) differ among steers fed the different diets nor were there any day x treatment interactions (P≥0.58) (data not shown). The steers at the beginning were all naïve to IBR and BVD, but not BRSV and PI_3. Selenium level and source had minimal effects on the antibody response of these weaned beef steers. There were day effects revealed in all titers over the 42 d period, with titers peaking at d 35. Humoral response has been improved by increasing dietary Se and/or vitamin E in several studies (Finch and Turner, 1996; Stabel and Spears, 1993; Swecker et al., 1989). Antibody responses have been most consistent when Se is supplemented with vitamin E in the diet (Spears, 2000).

In conclusion, the level and source of supplemental Se did not affect average daily gain of steers over the 105 d trial. Both sources of supplemental Se increased blood Se, the organic source increasing more rapidly than the inorganic source of Se. Selenium level and source had minimal effects on the immune function of the beef steers, perhaps due to the basal level of Se in the control diet.

ACKNOWLEDGMENTS

Funding was provided by the Dale Bumpers College of Agricultural, Food and Life Sciences Undergraduate Research Grant Program.

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Table 1. Ingredient and nutrient composition of grain supplement (dry-matter basis).

Item	%
Ingredient	
Corn	70.86
Soybean meal	12.60
Dicalcium phosphate	0.94
Limestone	1.74
Salt, white	1.53
Rumensin premix[a]	+
Vitamin premix[b]	+
Trace mineral premix[c]	+
Molasses	1.94
Fat	10.4
Calculated nutrient composition[d]	
Crude protein	14.86
NE_m, Mcal/kg	2.16
NE_g, Mcal/kg	1.42

[a]Provided 150 mg of monensin/d.

[b]Provided 11,000 IU vitamin A, 2,200 IU vitamin D, and 26 IU vitamin E/d.

[c]Provided 200 mg zinc as zinc sulfate, 60 mg copper as copper sulfate, 0.6 mg cobalt as cobalt carbonate, and 2.9 mg iodine as calcium iodate/d.

[d]Values calculated with the Oklahoma State Univ. Ration Calculator 1999 (as-fed version) software (www.ansi.okstate.edu/software/OSUNRCAF.xls).

Table 2. Growth performance, feed intake, and gain:feed ratio for steers fed different levels and sources of supplemental selenium for 105 d.

Item	Control	Inorganic Se	Organic Se	Standard error
Average daily gain, kg	0.44	0.45	0.40	0.034
Dry-matter intake, kg	5.19	5.13	5.08	0.073
Gain:feed	0.085	0.089	0.078	0.0073

Table 3. In vitro lymphocyte proliferation and macrophage activity for cells isolated from steers fed different levels and sources of supplemental selenium.

Item	Control	Inorganic Se	Organic Se	Standard error
Lymphocyte proliferation, counts/min				
PHA[a]	62,771	65,549	56,610	10,376
PWM[b]	25,650	25,402	21,772	4,678
ConA[c]	25,219	19,070	16,924	4,858
Macrophage phagocytosis				
Phagocytic cells, %	9.33	11.73	10.79	2.449
Number PRBC ingested/cell	0.96	1.31	1.11	0.117

[a]Lymphocytes cultured in the presence of phytohemagglutinin (PHA; 20μg/mL) to stimulate proliferation of peripheral blood T lymphocytes associated with cell-mediated immunity.
[b]Lymphocytes cultured in the presence of pokeweed mitogen (PWM; 15 μg/mL) to stimulate proliferation of B lymphocytes associated with humoral immunity.
[c]Lymphocytes cultured in the presence of concanavalian A (ConA; 40 μg/mL) to stimulate proliferation of peripheral blood T lymphocytes associated with cell-mediated immunity.

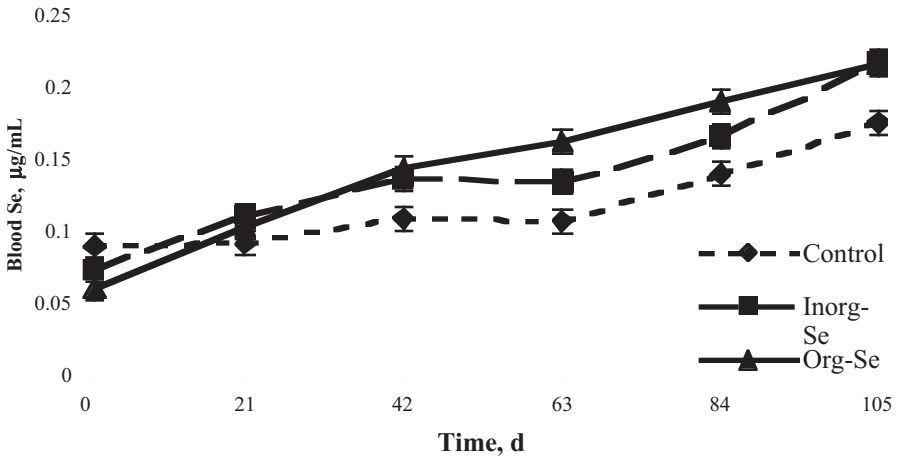

Fig. 1. Effect of dietary selenium concentration and source on steer blood selenium concentrations throughout the 105 d trial.

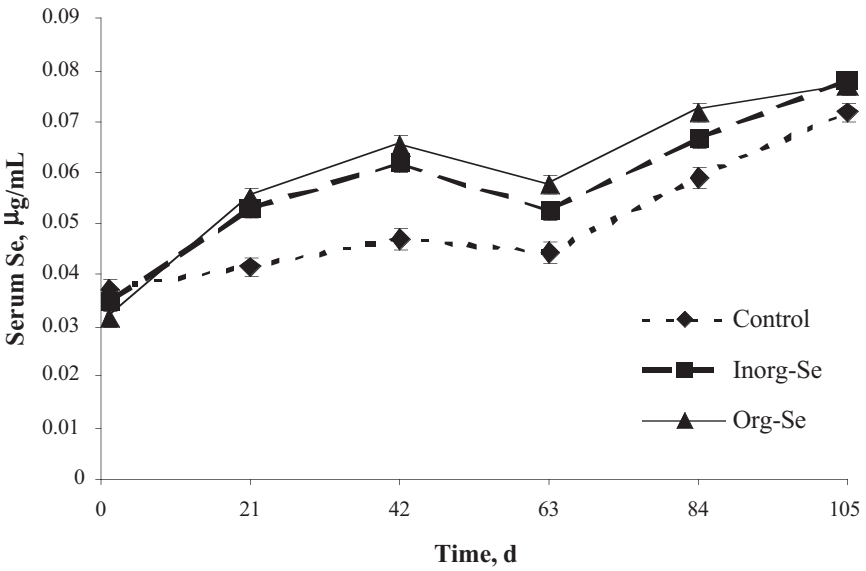

Fig. 2. Effect of dietary selenium concentration and source on steer serum selenium concentrations throughout the 105 d trial.

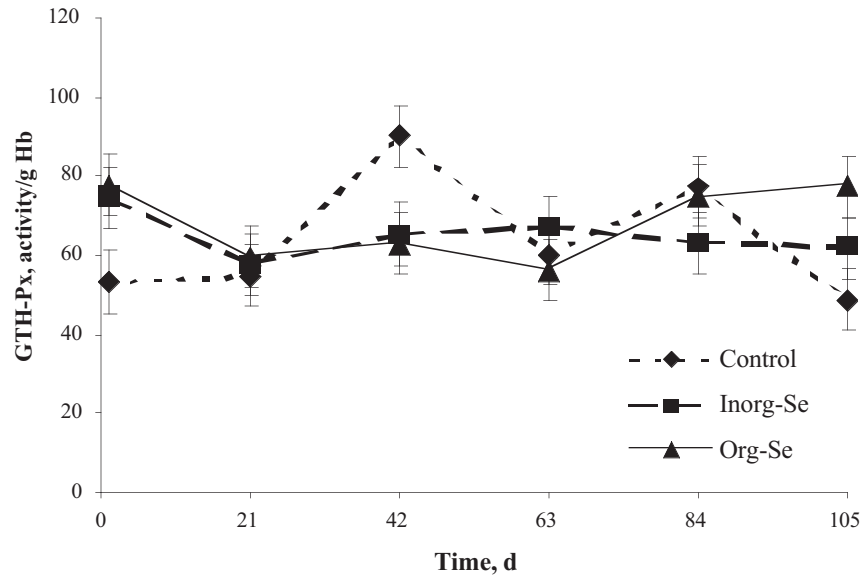

Fig. 3. Effect of dietary selenium concentration and source on steer glutathione peroxidase activity per gram of hemoglobin throughout the 105 d trial.