

The Influence of Supplements of Selenite, Selenate and Selenium Yeast on the Selenium Status of Dairy Heifers

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Ortman K, Andersson R, Holst H: The influence of supplements of selenite, selenate and selenium yeast on the selenium status of dairy heifers. Acta vet. scand. 1999, 40, 23-34. – The aim of the study was to define possible differences between selenite, selenate and selenium yeast on various aspects of selenium status in growing cattle. Twenty-four Swedish Red and White dairy heifers were fed no supplementary selenium for 6 months. The basic diet contained 0.026 mg selenium/kg feed dry matter (DM). After the depletion period the animals were divided into 4 groups; group I-III received 2 mg additional selenium daily as sodium selenite, sodium selenate, and a selenium yeast product, respectively. Group IV, the control group, received no additional selenium. The total dietary selenium content for groups I-III during the supplementation period was 0.25 mg/kg DM. After the depletion period the mean concentration of selenium in blood (640 nmol/l) and plasma (299 nmol/l) and the activity of GSH-Px in erythrocytes (610 μ kat/l) were marginal, but after 3 months of supplementation they were adequate in all 3 groups. The concentration of selenium in blood and plasma was significantly higher in group III than in groups I and II, but there was no significant difference between groups I and II. The activity of GSH-Px in erythrocytes did not differ between any of the supplemented groups. The animals in the control group had significantly lower concentrations of selenium in blood and plasma and lower activities of GSH-Px in erythrocytes than those in the supplemented groups. The activity of GSH-Px in platelets was also increased by the increased selenium intake. There was no difference in the concentration of triiodothyronine (T3) between any of the groups, but the concentration of thyroxine (T4) was significantly higher in the unsupplemented control group.

cattle; feed supplements; sodium selenite; sodium selenate; GSH-Px; plasma; blood; platelets; thyroxine; triiodothyronine.

Introduction

Sweden is a selenium deficient country and commercial feeds for farm animals have been routinely supplemented with selenium since 1980. By tradition, but also for economic reasons, sodium selenite has been the selenium compound used. However, in recent years the

use of sodium selenite has been questioned (Pehrson 1993b), principally because the selenite ion has pro-oxidative properties (Hafeman *et al.* 1974, Dougherty & Hoekstra 1982, Levan-der & Burk 1986, Spallholz 1994). Its in vivo disadvantages have been highlighted in experi-

ments with mice and rats (*Dougherty & Hoekstra* 1982, *Csallany & Menken* 1986). As an alternative to selenite, organic selenium compounds have been tested and found to be appropriate for the supplementation of dairy cows (*Ortman & Pehrson* 1997). However, organic selenium compounds are more expensive than inorganic selenium compounds.

Sodium selenate is an inorganic selenium compound which is similar in price to sodium selenite, and has the potential advantage that the selenate ion does not have the pro-oxidative properties of selenite. *Kumpulainen et al.* (1985) proposed that selenate should be used to supplement human diets to avoid the disadvantages of selenite.

When evaluating the biological effect of a selenium compound, it is generally accepted that the concentration of selenium in different body tissues may not adequately represent the concentration of the biologically active selenium in those tissues. Instead, the activity of the selenium-containing enzyme glutathione peroxidase (GSH-Px) has been commonly used as an indicator of the active component of the selenium in different tissues. Most often the activity of GSH-Px in blood has been used as long-term indicator of selenium status and the activity of GSH-Px in serum or plasma as a short-term indicator. However, in cattle the activity of GSH-Px in serum is very low and therefore not a suitable parameter (*Carlström* 1979). *Levander* (1983) emphasised the value of using the activity of GSH-Px in platelets as a short-term indicator of the selenium status of human beings, because platelets are turned over rapidly and have a high selenium content. The concentration of selenium in platelets adapts quickly to the dietary intake of selenium, and in that respect they are comparable to the liver, which has been reported to contain the largest pool of labile selenium in the body (*Thompson et al.* 1980, *Levander et al.* 1983b). Most of the

studies on platelets have been done in rats or human beings (*Levander et al.* 1983a, *Levander et al.* 1983b, *Kiem* 1988), and to the authors' knowledge no study has been made in cattle.

In recent years several new specific selenoproteins have been identified (*Zachara* 1992). One of them is the enzyme type I iodothyronine deiodinase, which converts thyroxine (T4) to the more active triiodothyronine (T3). *Arthur et al.* (1988) reported that severely selenium-deficient calves had higher plasma T4 concentrations and lower plasma T3 concentrations than calves which had been supplemented with selenium.

The present study had 3 main objectives:

- to determine whether there were any differences between the concentrations of selenium in blood and the activities of GSH-Px in the erythrocytes of cattle supplemented with sodium selenite, sodium selenate or a selenium yeast product,
- to investigate whether the determination of the GSH-Px activity of the platelets of cattle would be a satisfactory method for the evaluation of their short-term selenium status,
- to investigate whether the metabolism of the thyroid hormones T3 and T4 would be influenced by the form of supplementary selenium given.

Materials and methods

The experimental protocol used in this study was approved by the local ethical committee.

Animals and management

Twenty-four Swedish Red and White dairy heifers were used; in September 1995 they were tied up in a long-stall system, and were divided into 4 groups of 6 animals whose mean ages (and ranges) were as follows: group I, 17 months (11-25); group II, 17 months (11-22); group III, 17 months (11-23); and group IV, 17

months (11-22). The groups were randomly allocated to one of 4 treatments. For the 6 months from September 1995 to the end of February 1996, the 4 groups were fed the same basic ration, to which no selenium was added, except for the 4 youngest heifers, one in each group, which were not brought to the farm until mid-October and were therefore fed the basic diet for only four-and-a-half months instead of 6. From the end of February until the end of May groups I, II and III were fed rations supplemented with selenium in the 3 different forms, while group IV remained on the unsupplemented diet.

Feeding and treatments

The basic diet, which was fed individually during the whole indoor season, was composed of grass silage fed ad libitum, 1 kg oat grain, and 100 g of a selenium-free vitaminised mineral feed daily. Each animal was estimated to consume 8 kg of silage dry matter (DM) per day. The silage contained 0.023 mg Se/kg DM and the oat grain contained 0.06 mg Se/kg DM. The average daily intake of selenium from the basic diet was thus 0.238 mg (0.026 mg/kg DM). The sodium selenite, sodium selenate and the selenium yeast product (ALKOSEL 1000, Primalco Ltd Biotec, Rajamäki, Finland) were each mixed with solid glucose so that a known volume (15 ml) contained 2.0 mg selenium. Group I received sodium selenite, group II received sodium selenate and group III received the selenium yeast product. Group IV remained as an unsupplemented control group. The glucose-selenium mixture containing 2 mg selenium was given to each of the heifers in groups I-III on their grain once a day, and the animals in group IV were given the same volume of glucose, without added selenium, in the same way. The total selenium content of the supplemented ration was thus 2.24 mg (0.25 mg/kg DM) for groups I-III, and remained at 0.24 mg (0.026

mg/kg DM) for group IV. The overall experimental design is summarised in Fig. 1.

Samples

Jugular blood samples were taken from all the heifers one and 2 weeks before the start of the supplementation period (in the Figs. 2-7 the mean value of each of the parameters measured in these 2 samples is presented as the zero-time value) and then at pre-determined intervals during the experiment. All the samples were taken between 7 and 8 a.m. The anticoagulants used were potassium-EDTA for the determination of GSH-Px activity in erythrocytes and selenium in whole blood and plasma, sodium citrate for the isolation of platelets, and sodium heparin for the determination of T4 and T3 in plasma. The blood samples were brought to the laboratory within one hour after collection and, if necessary, centrifuged immediately. The platelets were isolated and their GSH-Px activity was determined on the day of sampling, but all the other samples were stored at -20 °C until analysed.

Laboratory analyses

For the isolation of platelets 20 ml of the citrated blood was transferred into a round-bottomed 25 ml glass tube containing 5 ml of acid-citrate-dextrose solution (ACD). After centrifugation at 220xg for 30 min at 25 °C the platelet-rich supernatant was transferred into coned 10 ml polystyrene tubes. After additional centrifugation at 1400xg for 30 min at 4 °C the pellet containing the platelets was resuspended in a fixed volume of calcium-free phosphate buffer and the platelets were counted manually in a Bürker chamber. After counting, the washed platelets were centrifuged at 1500xg for 20 min at 4 °C, lysed in distilled water, and centrifuged at a maximum speed of 21,500xg for 10 min at 4 °C to pellet the cell debris. The enzyme-containing supernatant was used to as-

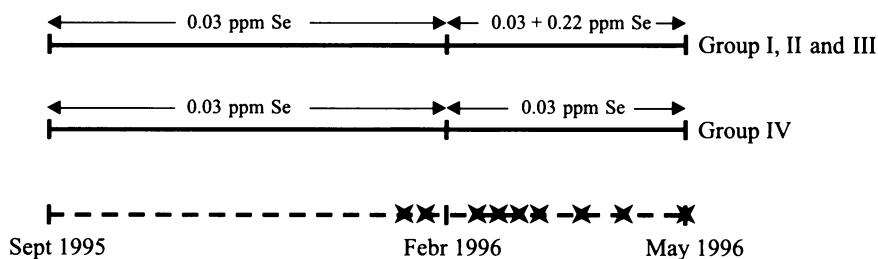


Figure 1. Overall experimental design. Basic feed contained 0.03 mg Se/kg (ppm). During the eleven weeks period from February to May 1996 groups I-III were supplemented with 0.22 ppm Se as sodium selenite, sodium selenate and selenium yeast, respectively. Group IV was an unsupplemented control group.

* denotes time of sampling.

sess the GSH-Px activity. Typically, the method yielded 100-300 x 10⁶ platelets/ml of citrated blood; however, 5 animals, from 4 different groups, never yielded enough platelets for the measurements of GSH-Px and they were therefore excluded from this part of the experiment, leaving 19 animals.

The GSH-Px activity in erythrocytes and platelets was measured by the method described by *Paglia & Valentine* (1967).

Before the selenium content of the samples was measured they were ashed with nitric acid in closed containers, using a microwave digestion procedure with a final temperature of 190 °C (*Matusiewicz et al.* 1991). After digestion the samples were treated with hydrogen peroxide and hydrochloric acid as described by *Alfthan* (1984), and then diluted 50:1 with a 20% solution of urea and allowed to stand for 30 min. The concentration of selenium was finally determined by atomic absorption spectrophotometry of the hydride, as described by *Sturman* (1985). Plasma total T4 and T3 concentrations were determined by using commercial solid-phase radioimmunoassay kits with ¹²⁵I as tracer (Coat-a-Count, Diagnostic Products Corporation, Los Angeles, CA, USA). The method has been validated for bovine plasma T3 and T4 by *Williams et al.* (1987). To confirm the accuracy of the in-

house methods, serial dilutions of plasma with high concentrations of T3 and T4 were prepared and were shown to produce displacement curves parallel to the respective standard curves. For T3, the intra- and inter-assay coefficients of variation for a quality control sample were 3.3% and 5.3% (mean = 2.6 nmol/l), respectively. For T4 the intra- and inter-assay coefficients of variation for 2 quality control samples were 2.3% and 6.6% (mean = 37.4 nmol/l), and 5.2% and 7.5% (mean = 175 nmol/l).

Statistical analyses

The experiment was designed as a randomised block trial. For the GSH-Px activity of erythrocytes and the selenium concentration of whole blood, a regression line, with time as the independent factor and GSH-Px activity or blood selenium as the dependent factor, was fitted to the results from each animal, and the value of the mean slope for each group was calculated. For plasma selenium, the mean concentration observed during a period when the concentration had reached a plateau was calculated for each group. The differences between the groups were compared by means of a two-way ANOVA, followed by multiple comparisons with a 95% simultaneous degree of confidence, according to the method of Tukey.

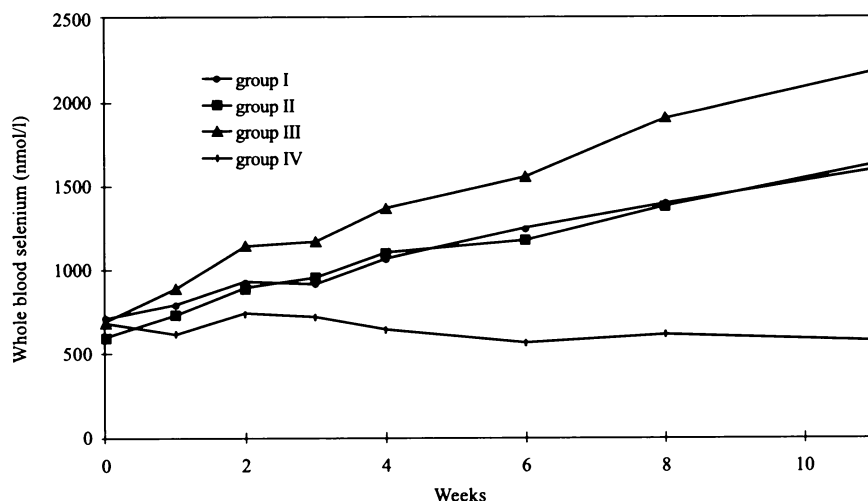


Figure 2. The concentration of selenium in whole blood of heifers during 11 weeks of supplementation with 2 mg selenium daily as sodium selenite (group I), sodium selenate (group II), or selenium yeast (group III) and in unsupplemented control animals (group IV).

The differences between groups for the concentration of T4 in plasma and the activity of GSH-Px in platelets was calculated by using the General Linear Models procedure of SAS (*SAS Institute Inc.* 1988). The calculations were based on the results of all the samples taken after the difference between the supplemented groups and the control group was considered to have been established. The independent variables used were treatment (selenite, selenate, selenium yeast, control), occasion of sampling and block. However, block was not included for the activity of GSH-Px in platelets, because the blocks were incomplete owing to the missing values.

Results

The concentration of selenium in the whole blood of all the supplemented groups increased during the trial and had not reached a plateau after 11 weeks of supplementation (Fig. 2); in contrast, in the control animals, the concentra-

tion decreased slightly during the trial. The differences between the groups were significant, except for that between the selenite and selenate groups.

The concentration of selenium in plasma increased in all the supplemented groups and reached a plateau after 4 weeks of supplementation in groups II and III and after 6 weeks in group I (Fig. 3). The differences between the groups during the plateau period were significant except for that between the selenite and selenate groups.

The activity of GSH-Px in erythrocytes increased in all the supplemented groups but did not reach a plateau during the experiment (Fig. 4). In the control group the activity decreased slightly. There were no significant differences between the selenium-supplemented groups, but all the supplemented groups had significantly higher GSH-Px activities than the control group.

The activity of GSH-Px in platelets increased during the first 3 weeks of supplementation, but

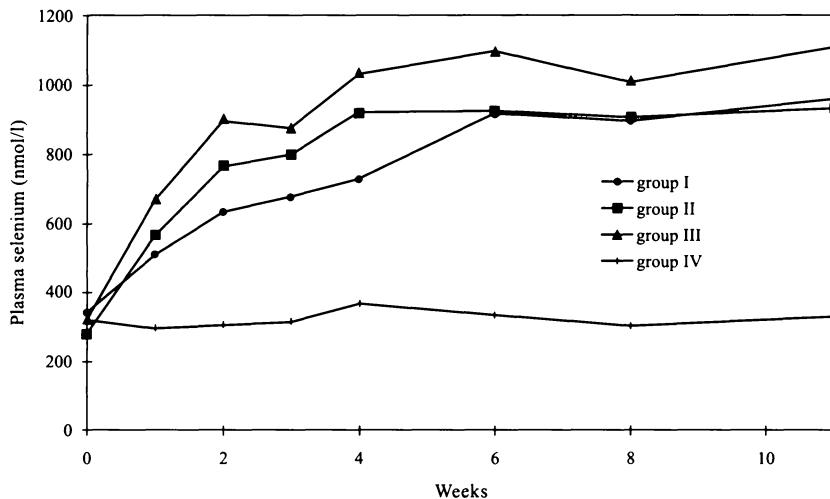


Figure 3. The concentration of selenium in plasma of heifers during 11 weeks of supplementation with 2 mg selenium daily as sodium selenite (group I), sodium selenate (group II), or selenium yeast (group III) and in un-supplemented control animals (group IV).

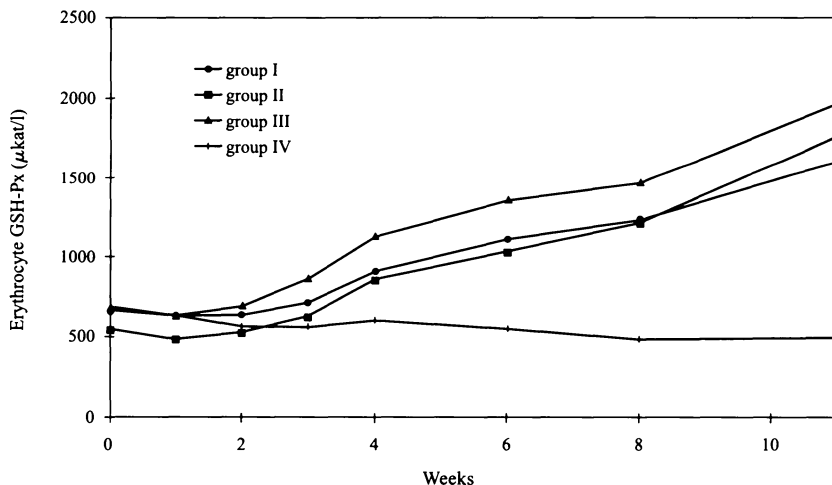


Figure 4. The activity of GSH-Px in erythrocytes of heifers during 11 weeks of supplementation with 2 mg selenium daily as sodium selenite (group I), sodium selenate (group II), or selenium yeast (group III) and in un-supplemented control animals (group IV).

then reached a plateau (Fig. 5). The activity in the group supplemented with selenium yeast (III) was then significantly higher than in the other groups. The group supplemented with selenate

had a significantly higher GSH-Px activity than the control group, but was not significantly different from the group supplemented with selenite, which did not differ from the control group.

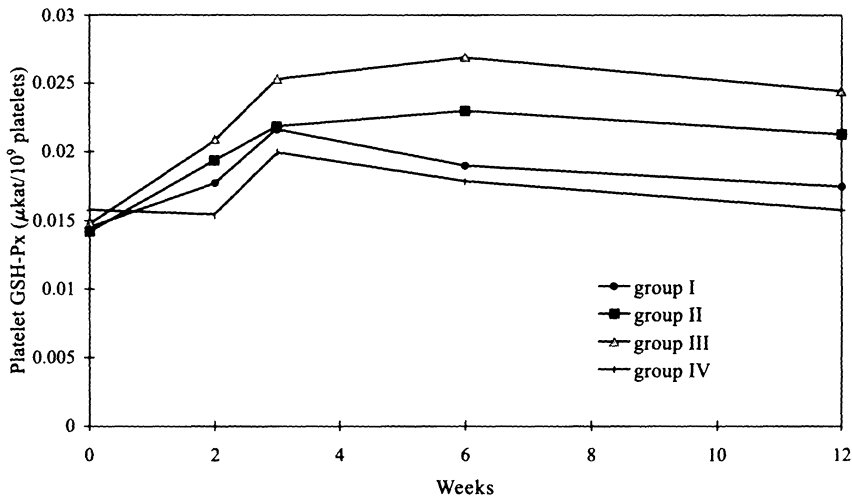


Figure 5. The activity of GSH-Px in platelets of heifers during 11 weeks of supplementation with 2 mg selenium daily as sodium selenite (group I), sodium selenate (group II), or selenium yeast (group III) and in unsupplemented control animals (group IV).

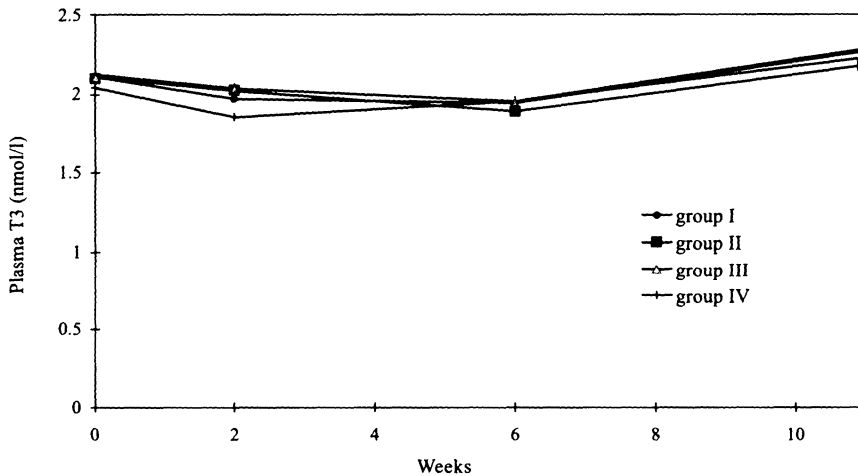


Figure 6. The concentration of triiodothyronine (T3) in plasma of heifers during 11 weeks of supplementation with 2 mg selenium daily as sodium selenite (group I), sodium selenate (group II), or selenium yeast (group III) and in unsupplemented control animals (group IV).

There were no significant changes in the concentration of T3 in the plasma of the 4 groups during the trial (Fig. 6). However, the mean concentration of plasma T4 decreased in the

first 2 weeks after the period of selenium supplementation began, but increased again until the end of the trial (Fig. 7). On the basis of the samples taken 6 and 11 weeks after supplement-

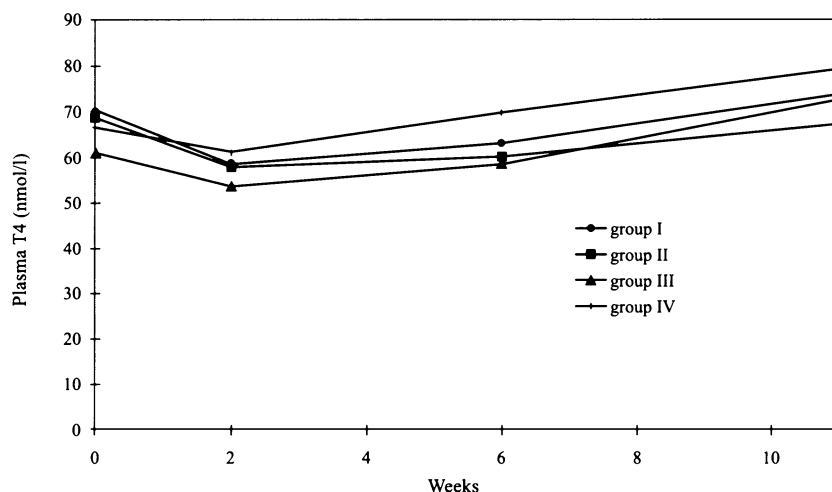


Figure 7. The concentration of thyroxine (T4) in plasma of heifers during 11 weeks of supplementation with 2 mg selenium daily as sodium selenite (group I), sodium selenate (group II), or selenium yeast (group III) and in unsupplemented control animals (group IV).

tation with selenium began, all the supplemented groups had significantly lower concentrations of T4 in plasma than the control group, but they were not significantly different from each other.

Discussion

After being fed the selenium-deficient diet for 6 months the mean whole blood selenium concentration of all the 24 animals was 640 nmol/l (range: 344-1088), their mean plasma selenium concentration was 299 nmol/l (range: 191-381) and the mean activity of GSH-Px in their erythrocytes was 611 μ kat/l (range: 206-1059). These values indicate that most of the heifers were either deficient or had a marginal selenium status, and that some of them might have been at risk of nutritional muscle degeneration (Pehrson 1993a). Thus, the animals were in a condition comparable with that of animals in Sweden before 1980, when animal feedstuffs were not supplemented with selenium.

After 11 weeks of supplementation with either sodium selenite, sodium selenate or the selenium yeast product the animals in groups I, II and III had whole blood and plasma selenium concentrations and erythrocyte GSH-Px activities within adequate ranges, while the selenium status of the animals in the control group had not changed. In the supplemented animals the plasma selenium concentration reached a plateau, but the whole blood selenium and the activity of GSH-Px in erythrocytes did not. This difference might have been due to the relatively short experimental period; the life-span of erythrocytes is about 160 days and as a result these parameters should not have reached a plateau until all the red blood cells from the depletion period had been replaced. Whole blood selenium and the activity of GSH-Px in erythrocytes are therefore suitable for monitoring long-term selenium status, whereas plasma selenium concentration, which reached a plateau within 6 weeks, should be more suitable for monitoring short-term selenium status.

There was a clear difference between the efficacy of the organic and inorganic selenium compounds as selenium supplements with respect to blood and plasma selenium concentrations; the animals that received the selenium yeast had higher concentrations of selenium in whole blood and plasma than the animals supplemented with selenite and selenate. These results are in accordance with earlier experiments (Nicholson *et al.* 1991, Ortman & Pehrson 1997). There was also a tendency for the selenium yeast group to have a higher activity of GSH-Px in their erythrocytes than the other supplemented groups, although the difference was not significant. However, the experiment ended before the GSH-Px activity had reached a plateau and it is possible that the difference might have become significant if a plateau had been reached. Earlier findings in ruminants have been variable; Pehrson *et al.* (1989), Nicholson *et al.* (1991) and Malbe *et al.* (1995) observed higher activities of GSH-Px in cattle supplemented with organic selenium than in cattle supplemented with inorganic forms of selenium, whereas Ortman & Pehrson (1997) observed no difference.

One reason for suspecting that selenite has a pro-oxidative effect has been the reports of increased activities of GSH-Px when selenite has been fed in higher than optimal doses, even up to toxic levels (Hafeman *et al.* 1974, Oh *et al.* 1976, Pehrson & Johnsson 1985). It has been suggested that this effect may indicate that high doses of selenite increase the activity of GSH-Px in order to counteract the pro-oxidative properties of the compound. If this was the case it would be expected that animals fed selenite would have higher activities of GSH-Px than animals fed organic selenium or selenate, which should not have any pro-oxidative properties. However, in this experiment there was no tendency for the animals fed selenite to have higher GSH-Px activities than the animals in

any of the other supplemented groups. The experiment therefore provided no further evidence that selenite has a pro-oxidative effect when fed to cattle at a dose corresponding to 2 mg of selenium daily.

The results also provided evidence that there was no difference between the efficacy of selenite and selenate as selenium supplements for cattle, whether the parameter considered was selenium in whole blood, selenium in plasma, the activity of GSH-Px in erythrocytes or platelets, or the concentrations of T3 or T4 in plasma. In contrast, Podoll *et al.* (1992) reported significantly higher serum selenium concentrations when dairy cattle were supplemented with selenate rather than selenite, although the difference was small. Henry *et al.* (1988) also found that selenate had a higher bioavailability than selenite when it was fed to young wethers, but they used a dose of 6 mg/kg dietary DM, which is considered to be toxic (Sandholm 1993). On the other hand, Serra *et al.* (1994) found that selenite and selenate were absorbed and retained equally well when they were used to supplement young wethers with physiological doses of selenium. Even if the potentially adverse effects of the oxidative properties of selenite on farm animals remain uncertain, it seems evident that sodium selenate is a satisfactory alternative to sodium selenite, since it is at least as effective in improving the selenium status of young cattle.

Levander (1983) found that the activity of GSH-Px in platelets could be used to assess the short-term selenium status of human beings and rats. The results of this experiment indicate that the activity of GSH-Px in platelets should also be useful for monitoring the selenium status of cattle, and that, in terms of this parameter, the selenium yeast had the highest bioavailability. However, the levels of enzyme activity were probably close to the lower detection limit of the assay and the measurements were very

variable. The results must therefore be interpreted with caution. Improvements to the method, for example by improving the procedure for lysing the platelets, and by adapting the method of *Paglia & Valentine* (1967) to the lysate, would probably give a method with greater validity and precision and consequently more reliable results.

Rey et al. (1994) reported that in selenium-deficient human beings platelets were abnormally aggregatable, because of the functional role of GSH-Px in the production of thromboxan A₂. As a result, in selenium-deficient subjects, the platelets might aggregate and sediment during the initial centrifugation and few platelets would be obtained. However, the 5 heifers in this study which consistently gave poor yields of platelets were distributed among the 4 groups, and the poor results therefore seem not to have been associated with their selenium status.

The activity of GSH-Px in the platelets tended to reach a plateau after 3 weeks of selenium supplementation, whereas the plasma selenium concentration reached a plateau within 6 weeks. However, the small difference between the time at which these 2 parameters reached a plateau, and the difficulties experienced in measuring platelet GSH-Px activity suggests that at present plasma selenium remains preferable for monitoring the short-term selenium status of cattle. However, GSH-Px activity is a more satisfactory indicator of the biologically active pool of selenium, and as the activity of GSH-Px in bovine plasma is very low, the measurement of GSH-Px activity in platelets might have some advantages for assessing the short-term selenium status of cattle, provided that the method can be improved adequately.

The metabolism of thyroid hormones was affected by the low selenium diet in this study, although the selenium status of the control animals was better than in the trial of *Arthur et al.* (1988). The concentration of T4 was signifi-

cantly higher in the unsupplemented control group than in the supplemented groups, but there were no differences between the supplemented groups. In contrast, the concentrations of T3 were very similar in all 4 groups. It is possible that there are compensatory systems which actively maintain the concentration of the more metabolically active hormone T3 nearly constant. For example, the activity of the enzyme iodothyronine deiodinase is actually induced in selenium-deficient animals, despite it being a selenium-containing enzyme, and moreover, the degradation of T3 to T2 becomes inhibited when the supply of selenium is limited (*Arthur* 1993). This suggests that the animals in the control group were only marginally deficient, rather than severely deficient in selenium.

In conclusion, organic selenium as a yeast product increased the selenium concentration in blood and plasma more efficiently than selenite or selenate, but no difference was found between organic and inorganic compounds in their capacity to increase GSH-Px activity in blood or to influence the T4 levels. No difference in bioavailability was found between selenite and selenate.

Acknowledgements

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References

- Alfthan G*: A micromethod for the determination of selenium in tissues and biological fluids by single-test-tube fluorimetry. *Anal. Chim. Acta* 1984, 165, 187-194.
- Arthur JR*: The biochemical functions of selenium:

- relationships to thyroid metabolism and antioxidant systems. Rowett Research Institute, 1993, Annual Report.
- Arthur JR, Morrice PC, Beckett GJ: Thyroid hormone concentration in selenium deficient and selenium sufficient cattle. *Res. Vet. Sci.* 1988, 45, 122-123.
- Carlström G: On the determination of glutathione peroxidase. The distribution of the enzyme between blood plasma and erythrocytes in different animals. *Summaries of XXI World Vet. Congr. Moscow*, 1979, 55.
- Csallany AS, Menken BZ: Effect of dietary selenium on hepatic organic solvent-soluble lipofuscin pigments. *J. Am. Coll. Toxicology* 1986, 5, 79-85.
- Dougherty JJ, Hoekstra WG: Stimulation of lipid peroxidation *in vivo* by injected selenite and lack of stimulation by selenate. *Proc. Soc. Exp. Biol. Med.* 1982, 169, 209-215.
- Hafeman DG, Sunde RA, Hoekstra WG: Effect of dietary selenium on erythrocyte and liver glutathione peroxidase in the rat. *J. Nutr.* 1974, 104, 580-587.
- Henry PR, Echevarria MG, Ammerman CB, Rao PV: Estimation of the relative biological availability of inorganic selenium sources for ruminants using tissue uptake of selenium. *J. Anim. Sci.* 1988, 66, 2306-2312.
- Kiem J: Selenium in platelets. *Biol. Trace Elem. Res.* 1988, 15, 83-88.
- Kumpulainen J, Salmenperä L, Siimes MA, Koivistoinen P, Perheentupa J: Selenium status of exclusively breast-fed infants as influenced by maternal organic or inorganic selenium supplementation. *Am. J. Clin. Nutr.* 1985, 42, 829-835.
- Levander OA: Considerations in the design of selenium bioavailability studies. *Federation Proc.* 1983, 1721-1725.
- Levander OA, Alfthan G, Arvilommi H, Huttunen JK, Kataja M, Koivistoinen P, Pikkariainen J: Bioavailability of selenium to Finnish men as assessed by platelet glutathione peroxidase activity and other blood parameters. *Am. J. Clin. Nutr.* 1983a, 37, 887-897.
- Levander OA, Burk RF: Report on the 1986 A.S.P.E.N. research workshop on selenium in clinical nutrition. *J. Parent. Ent. Nutr.* 1986, 10, 545-549.
- Levander OA, DeLoach DP, Morris VC, Moser PB: Platelet glutathione peroxidase activity as an index of selenium status in rats. *J. Nutr.* 1983b, 113, 55-63.
- Malbe M, Klaassen M, Fang W, Myllys V, Vikerpuur M, Nyholm K, Sankari S, Suoranta K, Sandholm M: Comparisons of selenite and selenium yeast feed supplements on Se-incorporation, mastitis and leukocyte function in Se-deficient dairy cows. *J. Vet. Med. A* 1995, 42, 111-121.
- Matusiewicz H, Sturgeon RE, Berman SS: Vapour-phase acid digestion of inorganic and organic matrices for trace element analysis using a microwave heated bomb. *J. Anal. Atom. Spectrom.* 1991, 6, 283-287.
- Nicholson JWG, McQueen RE, Bush RS: Response of growing cattle to supplementation with organically bound or inorganic sources of selenium or yeast cultures. *Can. J. Anim. Sci.* 1991, 71, 803-811.
- Oh S-H, Sunde RA, Pope AL, Hoekstra WG: Glutathione peroxidase response to selenium intake in lambs fed a torula yeast-based, artificial milk. *J. Anim. Sci.* 1976, 42, 977-983.
- Ortman K, Pehrson B: Selenite and selenium yeast as feed supplements for dairy cows. *J. Vet. Med. A* 1997, 44, 373-380.
- Paglia DE, Valentine WN: Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. *J. Lab. Clin. Med.* 1967, 70, 158-169.
- Pehrson B: Diseases and diffuse disorders related to selenium deficiencies in ruminants. *Norw. J. agric. Sci.* 1993a, Suppl. 11, 79-93.
- Pehrson B: Selenium in nutrition with special reference to the biopotency of organic and inorganic selenium compounds. *Biotechnology In The Feed Industry, Proc. Alltech's 9th Annual Symposium*, Nicholasville, KY, USA, 1993b, pp. 71-89.
- Pehrson B, Johnsson S: Selenium and glutathione peroxidase in blood and tissues and growth and feed efficiency in young bulls at different dietary selenium levels. *Zbl. Vet. Med. A* 1985, 32, 492-501.
- Pehrson B, Knutsson M, Gyllenswärd M: Glutathione peroxidase activity in heifers fed diets supplemented with organic and inorganic selenium compounds. *Swedish J. agric. Res.* 1989, 19, 53-56.
- Podoll KL, Bernard JB, Ullrey DE, DeBar SR, Ku PK, Magee WT: Dietary selenate versus selenite for cattle, sheep and horses. *J. Anim. Sci.* 1992, 70, 1965-1970.
- Rey C, Vericel E, Nemoz G, Chen W, Chapuy P, Lagarde M: Purification and characterization of

- glutathione peroxidase from human blood platelets. Age-related changes in the enzyme. *Biochim. Biophys. Acta* 1994, 1226, 219-224.
- Sandholm M*: Acute and chronic selenium toxicity. *Norw. J. agric. Sci.* 1993, Suppl. 11, 37-50.
- SAS Institute Inc.*: SAS/STAT users guide release 6.03 Ed. SAS Institute Inc., Cary NC, USA. 1988.
- Serra AB, Nakamura K, Matsui T, Harumoto T, Fuji-hara T*: Inorganic selenium for sheep I. Selenium balance and selenium levels in the different ruminal fluid fractions. *AJAS* 1994, 7, 83-89.
- Spallholz JE*: On the nature of selenium toxicity and carcinostatic activity. *Free Radic. Biol. Med.* 1994, 17, 45-64.
- Sturman BT*: Development of a continuous-flow hydride and mercury vapor generation accessory for atomic absorption spectrophotometry. *Appl. Spectrosc.* 1985, 39, 48-56.
- Thompson KG, Fraser AJ, Harrop BM, Kirk JA*: Glutathione peroxidase activity in bovine serum and erythrocytes in relation to selenium concentrations of blood, serum and liver. *Res. Vet. Sci.* 1980, 28, 321-324.
- Williams JE, Miller SJ, Mollet TA, Grebing SE, Bowman DK, Eilersieck MR*: Influence of frame size and zeranol on growth, compositional growth and plasma hormone characteristics. *J. Anim. Sci.* 1987, 65, 1113-1123.
- Zachara BA*: Mammalian selenoproteins. *J. Trace Elem. Electrolytes Health Dis.* 1992, 6, 137-151.
- Sammandrag**
Effekten av tillförsel av selenit, selenat och selenjäst på kvigors selenstatus.
- Under en sexmånaders period gavs 24 rekryteringskvigor en icke- selenberikad foderstat innehållande 0,026 mg selen/kg torrs substans (ts). Djuren delades därefter in i 4 grupper. Grupp I erhöll under 3 månader via fodret ett dagligt tillskott på 2 mg selen i form av natriumselenit. Grupp II gavs samma mängd selen som natriumselenat och grupp III samma mängd organiskt selen i form av ett selenjästpreparat. Grupp IV (kontrollgruppen) erhöll inget selentillskott. Det totala seleninnehållet i de berikade foderstaterna var 0,25 mg/kg ts. När selensupplementeringen påbörjades var seleninnehållet i helblod och plasma 640 resp. 299 nmol/l och GSH-Px aktiviteten i erytrocyterna 610 µkat/l, vilket innebär att djuren hade ett selenstatus som låg nära bristnivån. Vid försökets slut hade samtliga supplementerade grupper ett fullgott selenstatus, medan inga förändringar hade skett i kontrollgruppen. Djuren i grupp III hade vid försökets slut signifikant högre halt av selen i blod och plasma än de i grupp I och II, under det att inga skillnader förelåg beträffande GSH-Px aktiviteten i erytrocyter. GSH-Px aktiviteten i trombocyter ökade också efter selentillförseln. Det förelåg ingen skillnad mellan några grupper vad avser trijodtyronin (T3) i plasma, däremot hade kontrollgruppens djur en signifikant högre halt av thyroxin (T4) i plasma.

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