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SELENIUM AND GLUTATHIONE PEROXIDASE LEVELS IN LAMBS RECEIVING FEED SUPPLEMENTED WITH SODIUM SELENITE OR SELENOMETHIONINE

By

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MOKSNES, KNUT and GUNNAR NORHEIM: *Selenium and glutathione peroxidase levels in lambs receiving feed supplemented with sodium selenite or selenomethionine.* Acta vet. scand. 1983, *24*, 45—58. — Twenty-one 6 months old female lambs were divided into 7 groups and fed a basal diet containing 0.13 mg Se/kg. The basal diet was further supplemented with 0, 0.1, 0.5 or 1.0 mg Se/kg either as sodium selenite or as selenomethionine, and was fed for 10 weeks. Both feed additives produced an increase in the selenium concentration in the tissues analysed. Significant correlations were found between the concentrations of selenomethionine or sodium selenite added to the feed and the subsequent tissue levels. However, the selenium levels seemed to plateau at approximately 0.5 mg Se/kg of supplemented sodium selenite. The total glutathione peroxidase (GSH-Px) activity of the tissues increased when the selenium supplementation increased from 0 to 0.1 mg/kg for both selenium compounds. With further increase in selenium supplementation the GSH-Px activity in the tissues plateaued except in the blood where the activity continued to rise with increasing selenomethionine supplementation. The selenium dependent GSH-Px activity in the liver rose with increasing selenomethionine supplementation, but approached a plateau when 0.1 mg Se/kg as sodium selenite was added to the feed. The selenium concentration in whole blood responded more rapidly to the selenium supplementation than did GSH-Px activity. The experiment indicates that the optimal selenium concentration in the feed is considerably higher than 0.1 mg Se/kg, and that selenium levels of 1.0 mg/kg in the feed do not result in any risk for the animals or the consumers of the products.

dietary selenium; lambs; selenium concentrations; glutathione peroxidase activities; tissues.

About 4,500 cases of nutritional muscular dystrophy (NMD) are reported yearly in sheep in Norway (*Central Bureau of Statistics of Norway* 1979, 1980). This number constitutes about 0.5 % of the total number of lambs. NMD is most common in the

inland area of Norway where the feedstuffs are very poor in selenium (*Frøslie et al.* 1980). In such an area *Lutnæs* (1981) reported the incidence rate of NMD in lambs to be about 1 %. The disease was described by *Slagsvold & Lund Larsen* as early as 1934 and it occurred both in lambs and young cattle. At that time the etiology of the disease was unknown. Its relationship to vitamin E deficiency was discovered later, and in the late 1950's it was shown that the disease could be prevented by adding selenium to the feed (*Muth et al.* 1959).

Selenium was introduced in the prophylaxis and therapy of NMD in sheep in Norway in the early 1960's (*Hansen* 1962), and the benificial effects of selenium were verified (*Mikkelsen & Hansen* 1967, 1968, *Lunde & Ødegård* 1972). It was shown later that feedstuffs of Norwegian origin have a very low content of selenium (*Frøslie et al.* 1980). Selenium is usually administered by injection or by oral administration of high, single doses of sodium selenite or sodium hydroselenite to the ewes or the lambs in the spring. Furthermore, since 1980, sodium selenite has been added to commercial mixtures of concentrates and minerals for most species of domestic animals at a level corresponding to 0.1 mg Se/kg in the feed (*Landbruksdepartementet* 1979).

It is well known that the availability and potency of selenium are, to a large extent, dependent on its chemical form. When selenomethionine, an organic selenium compound, is added to the feed, it gives higher tissue levels of selenium in pigs and chickens than when the same amount of selenium is given as sodium selenite(*Ku et al.* 1972, *Moksnes & Norheim* 1982, *Moksnes* 1983). The purpose of the present investigation was to study the effect of addition of sodium selenite and selenomethionine to the feed, at levels above the minimum requirement, on the selenium concentrations and glutathione peroxidase activities in lambs.

MATERIAL AND METHODS

Animals and feeding

Twenty-one 6 months old female lambs (Norwegian Dala sheep) were divided into 7 groups. The basal diet consisted of 0.6 kg hay and 0.2 kg commercial mixtures of concentrates (Table 1) per lamb per day. The hay contained 0.05 mg Se/kg dry matter (DM) and the concentrates 0.35 mg/kg DM resulting in a basal diet containing 0.13 mg Se/kg DM. The basal diet was

supplemented with 0, 0.1, 0.5 or 1.0 mg Se/kg either as sodium selenite or as seleno-DL-methionine, and was fed to the lambs for 10 weeks. The selenium supplement was first mixed in lactose in the ratio 1 to 1000, and then mixed into the concentrates. Blood samples were collected before, and 2, 5, and 10 weeks after the start of the experiment. Whole blood samples were analysed for selenium and glutathione peroxidase (GSH-Px). Alanine aminotransferase (ALAT), aspartate aminotransferase (ASAT) and gamma glytamyl transpeptidase (γ-GT) in the serum samples were determined using standard methods, and the results were found to be within normal range. The lambs were immunized twice, after 4 and 7 weeks, with 5 different vaccines to study the effect of the selenium supplementation on humoral and cellular immunity. These results will be reported later. Immediately after the experimental feeding period the lambs were slaughtered and bled, and examined for lesions. Tissue samples were taken from certain organs for selenium and GSH-Px analyses.

Table 1. Composition of the commercial concentrates mixture.

Diet *	
Herring meal, extra quality	8.0 %
Ground sorghum	14.0 %
Ground rye	10.0 %
Ground barley	33.4 %
Ground oats	21.0 %
Wheat brand	6.1 %
Molasses	5.0 %
Mineral mixture	2.0 %
Vitamins	0.5 %
Guaranteed content:	
Crude protein	14.3—16.3 %
Digestible crude protein	11.5—13.5 %
Fat	3—4 %
Feed units (F.f.u.) per 100 kg	94—98
Calculated content:	
Calcium (Ca)	0.5 %
Phosphorus (P)	0.6 %
Salt (NaCl)	1.0 %
Added per kg:	
Vitamin A	4.000 I.U.
Vitamin D_3	2.000 I.U.
Selenium (analysed)	0.35 mg/kg

* Commercial concentrates from Arex-Møllesentralen, Oslo.

Analytical methods

Samples of liver, kidney, leg muscle and cardiac muscle destined for selenium analyses were frozen at —20°C immediately after collection. Material for analysis was taken in a semifrozen state to prevent loss of fluid from the samples. The tissue samples were analysed according to a modification of a fluorimetric method (*Ihnat* 1974, *Norheim & Nymoen* 1981). Results are expressed as µg Se/g on a wet weight basis. Samples of liver, leg muscle and cardiac muscle were taken as soon as possible after slaughter and immediately frozen in liquid nitrogen and kept below —80°C until they were analysed for GSH-Px activity. Whole blood samples for selenium and GSH-Px analyses were refrigerated for analysis the following day. Serum samples were frozen at —20°C until they were analysed for ALAT, ASAT and γ-GT.

For GSH-Px analysis, between 1 g and 5 g of tissue sample were cooled on ice and homogenized in 20 ml 0.15 mol/l potassium chloride. The homogenates were centrifuged at 48,200×g in a refrigerated centrifuge maintained at 0°C for 2 h. All the samples were analysed for total GSH-Px activity using cumene hydroperoxide as enzyme substrate, as described by *Paglia & Valentine* (1967). The liver samples were also analysed for selenium dependent GSH-Px activity using hydrogen peroxide as substrate. A molar concentration of hydrogen peroxide corresponding to half the amount of cumene hydroperoxide was used.

RESULTS

The addition of selenomethionine to the diet increased the selenium concentration in all tissues analysed (Table 2). The liver responded more than other tissues and reached a maximum of approximately 2.5 µg Se/g at the highest dietary level. On average, when compared to the control group, selenium levels increased by a factor of about 9 in liver, 4 in whole blood, 2.5 in heart muscle and 4.5 in skeletal muscle when 1.0 mg Se/kg as selenomethionine was added to the feed. Significant correlations were found between the amount of selenium added to the ration and the selenium concentration in all tissues analysed (Table 3).

Sodium selenite also resulted in increased selenium concentration in whole blood and tissues (Table 2). The selenium concentration in liver increased with each increase in dietary sodium

Table 2. Tissue and whole blood selenium content (mean and range) on day of slaughter of lambs kept on different levels of selenomethionine or sodium selenite. The results are given in μg/g wet tissue or μg/ml blood.

	Basal diet	Added selenomethionine (mg Se/kg)			Added sodium selenite (mg Se/kg)		
		0.1	0.5	1.0	0.1	0.5	1.0
Liver	0.28	0.52	1.46	2.46	0.55	1.54	2.06
n=3	(0.26—0.30)	(0.50—0.54)	(1.22—1.61)	(2.21—2.77)	(0.45—0.63)	(1.29—1.85)	(1.68—2.42)
Muscle	0.06	0.09	0.11	0.26	0.08	0.12	0.11
n=3	(0.04—0.08)	—	(0.10—0.12)	(0.24—0.28)	—	(0.10—0.13)	(0.10—0.12)
Heart	0.18	0.26	0.26	0.46	0.23	0.34	0.27
n=3	(0.15—022)	(0.24—0.27)	(0.26—0.27)	(0.43—0.51)	(0.20—0.26)	(0.33—0.35)	(0.24—0.30)
Kidney	1.40	1.45	1.49	1.85	1.33	1.81	1.81
n=3	(1.20—1.61)	(1.35—1.52)	(1.45—1.56)	(1.68—2.04)	(1.18—1.47)	(1.58—2.14)	(1.37—2.01)
Blood	0.13	0.26	0.34	0.50	0.26	0.33	0.30
n=3	(0.10—0.19)	(0.25—0.27)	(0.32—0.38)	(0.48—0.50)	(0.24—0.28)	(0.31—0.36)	(0.27—0.33)

Table 3. The selenium concentrations (μg Se/g wet weight) in liver (y_1), skeletal muscle (y_2), cardiac muscle (y_3) and whole blood (y_4) from lambs as a function of selenium concentrations (μg Se/g dry weight) added to the ration (x) in the form of selenomethionine, and in liver (y_5), skeletal muscle (y_6), cardiac muscle (y_7) and whole blood (y_8) as a function of the selenium concentrations added to the ration in the form of sodium selenite.

Regression function	n	r	P
$y_1 = 2.18\ x + 0.30$	21	0.98	< 0.001
$y_2 = 0.19\ x + 0.05$	21	0.94	< 0.001
$y_3 = 0.24\ x + 0.19$	21	0.91	< 0.001
$y_4 = 0.33\ x + 0.17$	21	0.94	< 0.001
$y_5 = 1.79\ x + 0.38$	21	0.94	< 0.001
$y_6 = 0.05\ x + 0.07$	21	0.73	< 0.001
$y_7 = 0.09\ x + 0.21$	21	0.58	< 0.01
$y_8 = 0.12\ x + 0.20$	21	0.63	< 0.01

n = number of samples analysed, r = correlation coefficient and P = probability of r = 0 (t-test).

selenite. Increasing selenium concentrations were found in whole blood, cardiac and skeletal muscle following dietary supplement up to 0.5 mg/kg and then they tended to plateau. Despite this plateau effect there seems to be a significant correlation between the concentration of sodium selenite added to the feed and the subsequent tissue levels (Table 3).

Dietary selenomethionine increased the total GSH-Px activity in all tissues analysed when the supplementation was increased from 0 to 0.1 mg/kg (Table 4). There were great variations in the liver GSH-Px activity within the groups when using cumene hydroperoxide as enzyme substrate, so it was difficult to assess the effect of increasing dietary selenium on the total GSH-Px activity. However, the selenium dependent GSH-Px activity, determined with hydrogen peroxide as substrate, rose with increasing levels of selenomethionine. In whole blood the GSH-Px activity rose with increasing levels of selenomethionine whereas a pleteau effect was observed with both cardiac and skeletal muscle at higher selenium levels.

Dietary sodium selenite also increased the total GSH-Px activity in all tissues analysed when the selenium supplementation increased from 0 to 0.1 mg/kg (Table 4). When the sodium selenite supplementation was increased from 0.1 to 1.0 mg/kg, the GSH-Px activity plateaued when using cumene hydroperoxide.

Table 4. Tissue and whole blood GSH-Px activities (mean and range) on day of slaughter of lambs kept on different dietary levels of selenomethionine or sodium selenite. The results are given in μkat/kg wet tissue or μkat/l blood.

	Basal diet	Added selenomethionine (mg Se/kg)			Added sodium selenite (mg Se/kg)		
		0.1	0.5	1.0	0.1	0.5	1.0
Muscle	18	31	32	35	30	32	30
n=3	(12—25)	(28—36)	(25—37)	(28—40)	(24—35)	(29—36)	(29—31)
Heart	254	326	317	381	315	346	338
n=3	(203—353)	(320—337)	(283—349)	(357—397)	(297—347)	(320—360)	(300—377)
Blood	243	586	774	864	702	593	604
n=3	(115—493)	(392—701)	(665—869)	(741—933)	(573—797)	(517—657)	(581—633)
Liver	629	729	615	753	800	589	556
n=3	(434—880)	(703—774)	(433—865)	(647—880)	(753—887)	(553—660)	(507—633)
Liver*	141	281	303	344	301	322	310
n=3	(97—207)	(267—297)	(293—320)	(317—363)	(280—323)	(303—337)	(287—327)

* Measured with hydrogen peroxide as enzyme substrate.

Table 5. Selenium concentrations (mean and range) and GSH-Px activities (mean and range) in whole blood of lambs as affected by duration of selenium feeding and different levels of dietary selenium as sodium selenite or selenomethionine.

Added selenium (mg/kg)	Selenium concentrations (μg/ml) Duration of selenium feeding (weeks)				GSH-Px activity (μkat/l) Duration of selenium feeding (weeks)			
	0	2	5	10	0	2	5	10
0	0.05 (0.02—0.12)	0.10 (0.08—0.13)	0.10 (0.06—0.16)	0.13 (0.10—0.19)	141 (56—299)	136 (24—303)	181 (50—373)	243 (115—493)
0.1 Se-meth	0.05 (0.01—0.13)	0.13 (0.10—0.18)	0.15 (0.12—0.19)	0.26 (0.25—0.27)	164 (82—304)	159 (62—333)	247 (128—401)	586 (392—701)
0.5 Se-meth	0.05 (0.02—0.09)	0.21 (0.20—0.24)	0.27 (0.22—0.33)	0.34 (0.32—0.38)	210 (69—285)	385 (288—460)	567 (376—685)	774 (665—869)
1.0 Se-meth	0.12 (0.09—0.18)	0.28 (0.27—0.32)	0.38 (0.32—0.46)	0.50 (0.48—0.54)	271 (160—432)	405 (267—620)	593 (465—821)	864 (741—933)
0.1 Na_2SeO_3	0.06 (0.02—0.09)	0.13 (0.11—0.15)	0.16 (0.15—0.17)	0.26 (0.24—0.28)	153 (98—195)	165 (85—208)	260 (213—303)	702 (573—797)
0.5 Na_2SeO_3	0.05 (0.01—0.09)	0.20 (0.18—0.23)	0.24 (0.20—0.26)	0.33 (0.31—0.36)	195 (54—355)	228 (83—376)	334 (203—428)	593 (517—657)
1.0 Na_2SeO_3	0.10 (0.07—0.12)	0.20 (0.19—0.22)	0.23 (0.19—0.27)	0.30 (0.27—0.33)	217 (128—272)	268 (182—347)	359 (298—399)	604 (581—633)

The activity in the liver also plateaued when using hydrogen peroxide as a substrate.

The selenium concentrations and total GSH-Px activities in whole blood of the lambs were dependent upon the duration of the selenium feeding (Table 5). The increase in whole blood selenium responded quickly and showed the highest increase in the first 2 weeks of the feeding period. The total GSH-Px activity showed no, or only a slight increase after 2 weeks in all groups except in the groups supplemented with 0.5 or 1.0 mg Se/kg as selenomethionine. Here, a marked increase in their GSH-Px activity was observed. For the rest of the feeding period both selenium concentrations and GSH-Px activities in all groups increased according to the duration of the experiment.

DISCUSSION

A close relationship between the amount of selenium added to lambs' feed and the subsequent levels of selenium found in blood and tissues has been demonstrated in the present investigation. Little difference was found in the ability of sodium selenite and selenomethionine to raise the levels of selenium in blood and tissues up to a feed supplement of 0.5 mg Se/kg DM. However, when the supplementation was increased to 1.0 mg Se/kg, selenomethionine seemed to be more potent than sodium selenite. Selenomethionine and sodium selenite seem to give a different distribution pattern of the selenium deposits in the body of sheep compared to pigs and chickens. In pigs and chickens, organic selenium is more potent at all levels than inorganic selenium (*Ku et al.* 1972, *Moksnes & Norheim* 1982, *Moksnes* 1983).

In the present investigation, we found higher tissue levels of selenium than did *Oh et al.* (1976a) who used sodium selenite at dietary levels of 0.5 mg Se/kg. This discrepancy may be due to factors such as the basal selenium content in the feed, age of the animals and duration of the supplementation. *Ehlig et al.* (1976) added selenomethionine and sodium selenite at levels of 1.0 mg Se/kg to the feed of lambs for 10 days. The basal feed contained 0.30 mg Se/kg. They found lower concentrations in the tissues than found in the present investigation. This discrepancy is most probably due to the short duration of their experiment. These authors also found that lambs treated with selenomethionine had higher tissue levels of selenium than those given sodium selenite.

Further, they estimated the absorption, retention and "biological value" of selenomethionine and sodium selenite in lambs and concluded that the selenium retention was greater for selenomethionine than selenite. This difference was caused by a higher rate of urinary excretion of selenium in the lambs given sodium selenite. There was no significant difference in the apparent rates of absorption of the 2 selenium compounds. *Mykkänen et al.* (1982) studied the absorption of (^{75}Se)selenite and L(^{75}Se)methionine following intraluminal administration to chicks using an in vivo ligated loop procedure. They found that the percentage of selenite absorbed decreased as the concentration of stable selenium in the dose increased, indicating a saturable transfer process. A similar increase in the luminal selenomethionine did not affect the absorption of selenomithionine, which offers a possible explanation for the differences in the bio-availabilities of these compounds.

In the present experiment, dietary supplementation of 0.1 mg Se/kg either as sodium selenite or selenomethionine, to a diet containing 0.13 mg Se/kg, increased the total GSH-Px activity in blood and the tissues analysed. Above this level, 0.23 mg total Se/kg in the feed, the GSH-Px activity in the tissues approached a plateau. However, in blood, the GSH-Px activity increased further in the selenomethionine group, but not in the sodium selenite group. *Oh et al.* (1976 a, b) added sodium selenite to lambs feed originally very low in selenium and found that the GSH-Px activity in the tissues increased only up to a dietary level of 0.12 mg Se/kg. This discrepancy may be due to the differences in the selenium content of the basal feed and the duration of the experiment. Genetic variations must also be considered. According to *Sandholm et al.* (1981) genetic factors may influence the variation of erythrocyte GSH-Px activity in sheep.

When comparing the effect of supplementation of selenomethionine and sodium selenite on the GSH-Px activity in blood and tissues there are generally only small differences. It seems, however, that sodium selenite is superior to selenomethionine at a dietary level of 0.1 mg Se/kg, while selenomethionine gives the highest selenium values at a dose level of 1.0 mg Se/kg.

The 2 selenium sources differ only slightly in their ability to raise the activity of selenium dependent GSH-Px in liver. When using hydrogen peroxide as enzyme substrate the GSH-Px activity increased with increasing selenomethionine supplementation but

approached a plateau when the feed supplement of sodium selenite was 0.1 mg/kg. The activity of selenium dependent GSH-Px in relation to the total GSH-Px activity increased from 22 % in the control group to 38 % in both groups supplemented with 0.1 mg Se/kg. A further increase was observed in the animals supplemented with 0.5 mg Se/kg. No differences were, however, observed between the groups given 0.5 and 1.0 mg Se/kg. *Sunde* (1980) found that only 10—15 % of the total selenium in sheep liver was accounted for by the selenium incorporated into GSH-Px as compared to 74 to 84 % of the whole blood selenium. *Beilstein et al.* (1981) found in lambs given (^{75}Se)selenite that selenocysteine appears to be the predominant form of selenium in ovine heart and liver. *Jørgensen et al.* (1981) found that less than 10 % of the total GSH-Px in pigs was due to non-selenium dependent GSH-Px.

The difference in response of GSH-Px activity to hydrogen peroxide and organic hydroperoxide substrates can be explained by the discovery of a non selenium-containing GSH-Px that catalyze the reaction of organic hydroperoxides like cumene hydroperoxide, but not hydrogen peroxide. *Prohaska et al.* (1977) identified this non-selenium containing GSH-Px as one or several of the glutathione-S-transferases.

The increase in the blood selenium concentration and total GSH-Px activity during the feeding period shows that the selenium concentration responds more rapidly to selenium supplementation than does the GSH-Px activity. Similarly, *Peter et al.* (1980) found that the total erythrocyte GSH-Px activity did not respond as fast as the whole blood selenium concentration in ewes drenched with 25 mg selenium as sodium selenite. They explained the different response between selenium concentration and GSH-Px activity by the fact that the whole blood GSH-Px activity results mostly from the erythrocyte GSH-Px activity and thus is more dependent than whole blood selenium concentration on rates of erythropoesis, erythrocyte turnover and erythrocyte pool size.

According to *Sunde* (1980) sodium selenite is metabolically closer to GSH-Px than selenomethionine. If this is correct, one would expect sodium selenite to elicit a more rapid response than selenomethionine with respect to GSH-Px activity. In the present study, however, the total GSH-Px activity showed the most rapid

response when 0.5 and 1.0 mg Se/kg as selenomethionine was added to the feed.

It is difficult to draw any firm conclusion from the present experiment as to the optimal selenium concentration in lambs' feed and which form of selenium should be chosen. However, it is clear that addition of both sodium selenite and selenomethionine to a basal diet containing 0.13 mg Se/kg does produce beneficial effects on the GSH-Px activity which indicates that the optimal selenium concentration in the feed is considerably higher than 0.1 mg/kg. Since sheep in Norway are fed concentrates for only short periods of the year, it would be necessary to add high levels of selenium to the feed in order to build up a sufficient deposit of selenium in these animals. It has been shown in this experiment that adding selenium to concentrates at levels corresponding to 1.0 mg Se/kg in the feed do not result in any risk for the animals or the consumers of the products.

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SAMMENDRAG

Selen og glutationsperoksydase hos lam gitt forskjellige mengder natriumselenitt og selenomethionin i fôret.

Enogtyve 6 måneder gamle søyelam ble delt i 7 grupper og fôret med et grunnfôr inneholdende 0,13 mg Se/kg (tørrvekt). Fôret ble tilsatt henholdsvis 0, 0,1, 0,5 og 1,0 mg Se/kg som natriumselenitt eller selenomethionin og fôret i 10 uker. Både natriumselenitt og selenomethionin økte selenkonsentrasjonen i de undersøkte organene, men natriumselenitt nådde et maksimum når tilskuddet kom opp i 0,5 mg Se/kg. Signifikante positive korrelasjoner ble funnet mellom nivåene av selen i de undersøkte organene og den mengden selen som ble tilsatt fôret som natriumselenitt eller selenomethionin. Den totale GSH-Px aktiviteten i organene økte når selentilskuddet økte fra 0 til 0,1 mg/kg for begge selenforbindelsene. Når selentilskuddet økte videre fra 0,1 mg/kg fikk vi ingen videre økning i GSH-Px aktiviteten bortsett fra i blod hvor aktiviteten økte med økende tilskudd av selenomethionin. Den selenavhengige GSH-Px aktiviteten i lever økte med økende tilskudd av selenomethionin, mens den nådde et platå ved 0,1 mg Se/kg som natriumselenitt. Selenkonsentrasjonen i blodet svarte raskere på selentilskuddet enn tilfellet var for GSH-Px aktiviteten. Det er vist i dette forsøket at det optimale selennivået i fôret ligger vesentlig høyere enn 0,1 mg/kg, og at et selennivå i fôret på 1,0 mg/kg ikke medfører noen risiko for dyrene eller konsumentene av produktene.

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