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# Excretion routes and distribution of selenium in sheep tissues after selenite loading*

**V. Petrović[1], K. Boldižárová[1], Š. Faix[1], J. Kowalczyk[2], M. Czauderna[2], M. Mellen[3] and L'. Leng[1,4]**

*[1]Institute of Animal Physiology, Slovak Academy of Sciences*
*Soltesovej 4, 040 01Košice, Slovak Republic*
*[2]The Kielanowski Institute of Animal Physiology and Nutrition, Polish Academy of Sciences*
*05-110 Jabłonna, Poland*
*[3]Slovak Agriculture University*
*Tr. A. Hlinku 2, 949 76 Nitra, Slovak Republic*

ABSTRACT

Excretion routes of selenium (Se) and its contents in liver and muscle were investigated in sheep after intravenous infusion of sodium selenite. Blood Se level was increased 12 times and began to fall immediately after the end of selenite infusion. Se excretion by urine and faeces reached the highest values 3 h and from 12 to 24 h later, respectively. Selenite loading resulted in a 5-fold higher Se level in the liver but no change of this parameter was found in muscle 3 days after selenite infusion. The results suggest that some Se-metabolite(s) is(are) secreted into the digestive tract of ruminants.

KEY WORDS: ruminants, selenium metabolism, glutathione peroxidase

## INTRODUCTION

Selenium (Se) is well known to be an essential trace element for animal health. In most European countries the natural Se content in feeds is only 0.03-0.12 mg/kg of DM with values more common at the lower end of this range. Intake of such feeds can result in serious Se deficiency and health problems, especially in highly productive animals. Therefore feeds are widely supplemented with various

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[4] Corresponding author: e-mail: lleng@saske.sk

Se sources to reach a final Se level of 0.3-0.5 mg/kg of DM. Ruminants with apparent nutritional Se deficiency are frequently treated with injection preparations containing inorganic Se-compounds like selenite or selenate.

The aim of this work was to determine the excretion routes and tissue distribution of Se after intravenous loading of sheep with sodium selenite.

## MATERIAL AND METHODS

Five Merino sheep, 37-39 kg BW, were adapted to a diet with a Se content of 0.11 mg/kg of DM for 3 weeks and individually housed in metabolic cages. The control quantitative collection of faeces and urine lasted 3 days and afterwards control samples of liver and muscle were obtained under Rometar anaesthesia. Five weeks later the sheep were placed again into metabolic cages and given intravenously 4.55 mg of Se as a priming dose followed by a two-hour long infusion of 11.7 μg/ml/min of Se as sodium selenite. The total amount of Se administered was 5.95 mg per animal. After the infusion, the quantitative collection of faeces and urine was again carried out for 3 days. Sampling of liver and muscle tissue was done after euthanasia of the animals. Se levels in blood, urine, faeces, feeds and tissues were analysed by the fluorimetric method of Rodriguez et al. (1994). Blood glutathione peroxidase (GPx) activity was determined as in Paglia and Valentine (1967). Statistical analyses of results was done by one-way ANOVA with the Dunnet post-test for Se excretion and GPx dynamics and by the paired Student t-test for tissue Se contents. Values are means ± SEM.

## RESULTS AND DISCUSSION

Intravenous selenite loading induced a huge increase in the blood Se level (from 0.78±0.11 to 9.47±0.88 μmol/l; $P<0.01$). The post-infusion Se level in blood was already reduced by half after 7 h and this pattern was followed in all further samples collected up to the 24 h. After this time the Se blood level was almost stable and its value (2.48±0.17 μmol/l) at 72 h post-infusion tended to be higher but not significantly different from the control. The measured control value of Se blood level corroborates the low selenium status of sheep fed a diet without supplementing any Se-source (Boldižárová et al., 2003).

Data on Se levels in urine and its urinary excretion during post-infusion periods are summarized in Table 1. The Se level in urine showed the highest values between 3 and 5 h after the end of infusion and then it began to fall. Amounts of Se excreted in urine followed almost the same pattern with values measured in

collection periods between 10 to 72 h that were not significantly different from the control value. The explanation for the increase in both urinary Se levels and excretion during the initial 5 h after infusion while the Se level in the blood was even sharply falling is based on the metabolism of selenite in the blood stream. Intravenously dosed selenite is taken up rapidly by RBC, where is reduced by glutathione to $H_2Se$ within a few minutes (Shiobara et al., 1998). After efflux into plasma, $H_2Se$ is bound to albumin *via* 17 disulfide bonds and transported to the liver for synthesis of selenoprotein P, and the surplus of $H_2Se$ is methylated for subsequent excretion (Symonds et al., 1981). This means that the post-infusion increase in both urinary levels and excretion of Se reflects methylation of the infused Se.

Table 1. Dynamics of selenium concentrations in urine and amounts of Se excreted in sheep after intravenous infusion of selenite

| Parameter | Collection periods of urine (hours after the end of selenate infusion) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | C | 0-1 | 1-2 | 2-3 | 3-5 | 5-10 | 10-18 | 18-30 | 30-72 |
| Concentration µmol/l | 0.03± 0.01 | 53.5± 21.0 | 71.4± 19.8 | 106.8± 18.6** | 125.5± 31.4** | 86.1± 21.0* | 46.8± 10.3 | 21.6± 7.8 | 2.9± 0.4 |
| Amount excreted nmol/min | 0.013± 0.01 | 15.6± 1.5 | 26.9± 4.5** | 26.7± 4.4** | 47.7± 7.0** | 30.6± 4.7** | 15.8± 2.5 | 5.5± 0.8 | 0.5± 0.2 |

C-control value before selenite infusion; *P<0.05;**P<0.01, significantly different from control

Table 2. Dynamics of selenium concentrations in faeces and amounts of Se excreted in sheep after intravenous infusion of selenite

| Parameter | Collection periods of faeces (hours after the end of selenite infusion) | | | | | | |
|---|---|---|---|---|---|---|---|
| | C | 0-12 | 12-24 | 24-36 | 36-48 | 48-60 | 60-72 |
| Concentration µmol/kg of DM | 2.69± 0.36 | 5.20± 0.21 | 12.34± 0.76** | 14.65± 1.28** | 11.71± 1.68** | 8.69± 0.95** | 7.79± 1.05* |
| Amount excreted nmol/12 h | 298.3± 43.0 | 290.4± 49.5 | 905.3± 176.0** | 536.4± 44.6 | 761.4± 45.7** | 332.0± 111.4 | 594.0± 274.9 |

C-control value before selenite infusion; DM- dry matter; *P<0.05; **P<0.01, significantly different from control

The Se level and amount excreted in faeces also increased after the end of infusion and followed a similar pattern as in urine (Table 2). The amount of Se excreted in faeces reached a peak between 24 and 36 h post-infusion and this finding suggests secretion of methylated Se-metabolite(s) by saliva, bile and pancreatic juice into the digestive tract. Within of 72 h post-infusion, the portion of Se excreted by urine and faeces from the total amount infused was found to be 48.1 and 1.97%, respectively.

The Se level in liver tissue was found to be increased from 10.39±0.63 to 53.32±2.66 μmol/kg of DM ($P<0.001$) by the end of the experiment. The unchanged Se level in muscle (control: 1.07±0.06 vs 72 h after infusion: 1.08±0.04 μmol/kg of DM, NS) confirms the inability of inorganic Se sources to create significant body deposits of this trace element. The activity of GPx in whole blood was not influenced by selenite infusion and varied between 352.1±52.1 U/g of Hb (control) and 271.9±38.9 U/g of Hb (72 h after the end of infusion).

## CONCLUSIONS

Our results demonstrate the low potential of sodium selenite to build up body deposits of Se in ruminants. The increased Se content in faeces induced by selenite infusion indicates that Se-metabolites are secreted into the digestive tract of sheep.

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## STRESZCZENIE

**Drogi wydalania oraz dystrybucja selenu w tkankach owiec po podaniu seleninu**

Badano drogi wydalania selenu (Se) i jego zawartość w wątrobie i mięśniach owiec po dożylnej infuzji seleninu. Największy wzrost wydzielania Se w moczu stwierdzono po 3 godz. od momentu zakończenia infuzji Se, a w kale od 12 do 24 godz. Podczas infuzji poziom Se w wątrobie wzrósł pięciokrotnie, natomiast w mięśniach nie stwierdzono zmiany zawartości Se po 3 dniach od momentu zakończenia infuzji. Wyniki te świadczą o sekrecji metabolitów Se do układu trawiennego przeżuwaczy.