Reviews on the mineral provision in ruminants (XIV): SELENIUM METABOLISM AND REQUIREMENTS IN RUMINANTS

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PREFACE

In the Netherlands the ´Handleiding Mineralenonderzoek bij rundvee in de praktijk´[1] is a well-known publication that has been used already for decades as a guide to trace and treat mineral disorders in cattle. The fifth edition of this guidebook was published in 1996. The content of this publication was largely identical to that of the fourth edition (1990). Therefore the (independent) committee that is responsible for the contents of the guidebook (the 'Commissie Onderzoek Minerale Voeding'[2], COMV) decided in 2000 that a thorough revision was desired.
The committee was of the opinion that, if possible, the available scientific literature should be summarized and evaluated once again. Furthermore, attention should be paid to the mineral provision of categories of cattle other than dairy cattle, as well as to that of sheep and goats. Finally, the basic principles for the calculation of the mineral requirements should be described in a transparent way.

The intended revision was made possible as the Dutch 'Ministerie van Landbouw, Natuur en Voedselkwaliteit' (LNV), the 'Productschap Diervoeder' and the 'Productschap Zuivel'[3] were willing to subsidize this extensive and ambitious project.
The COMV decided to execute the project as follows.

- External experts, invited by the COMV, should summarize and evaluate the relevant literature in a so-called 'basal document' (with two exceptions to be written in English).
- Subsequently, these documents should be critically evaluated by the COMV.
- These basal documents should then be used to write and arrange the several chapters of the revised 'Handleiding'.

The revised 'Handleiding' is available (in the Dutch language) since October 2005, under the title 'Handleiding mineralenvoorziening rundvee, schapen en geiten.'[4] This book is published by the 'Centraal Veevoederbureau' (CVB; Central Bureau for Livestock Feeding) in Lelystad, as was also the case for the previous edition.

The COMV was of the opinion that the valuable basal documents, that became available during the course of this project, should be published too. By doing so everyone has the possibility to trace the basis for the text of the revised 'Handleiding'. The CVB was gladly willing to issue these documents as CVB Documentation reports. In connection with this the authors and the members of the COMV have disclaimed all rights and have assigned them to the Productschap Diervoeder, of which the CVB is one of the services.
For an overview of the CVB Documentation Reports that will appear in this context, you are referred to an Annex in the back of this report.
For the preparation of the present report on the Selenium provision in ruminants the COMV expresses its gratitude to the author, dr. A.M. van den Top.

Utrecht/Lelystad, September 2005.

Professor dr. ir. A.C. Beynen	Dr. M.C. Blok
Chair of the COMV	Secretary of the COMV and Head of the CVB

The author, Dr. A.M. van den Top, expresses his thanks to the COMV, especially Dr. M.C. Blok, Prof. Dr. A. Th. Van 't Klooster, Dr. ir. A.W. Jongbloed and Dr. J. Veling, for critically reading the manuscript and their advice.

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1 Guidebook on mineral research for cattle in practice.
2 Committee for research on mineral nutrition
3 The Ministry for Agriculture, Nature and Food quality, the Product Board Animal Feed and the Dutch Dairy Board, respectively.
4 Guidebook mineral provision cattle, sheep and goats.

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CONTENT

LIST OF ABBREVIATIONS

Abbreviation	Unit	Description
BW	kg	Body weight
DM		Dry matter
DMI	kg/d	DMI = dry matter intake
GSH-PX		Glutathione peroxidase
SeCys		Selenocystine
SeMet		Selenomethionine

1 FUNCTIONS OF SELENIUM IN THE BODY

In the body, Se is a component of the enzymes glutathione peroxidase (GSH-Px) and iodothyronine-5'-deiodinase [53]. Iodothyronine-5'-deiodinases convert thyroxine (T_4) to triiodothyronine (T_3) and, therefore, Se may influence iodine metabolism. GSH-Px detoxifies peroxide radicals and is an important component of the cellular antioxidant system. Proper function of this system is necessary for cell membrane stability and to prevent tissue damage. GSH-Px shares its function with other enzymes (e.g. superoxide dismutase, catalase) and with non-enzyme radical scavengers, such as vitamin E. The effects of Se deficiency may depend, therefore, on both the rate of free radical production and the activity of the other scavengers [69], although evidence is slender [6].
A more comprehensive list of selenoproteins is given in Table 15.1 of reference [69]. The effects of Se on the reproductive system are reviewed in reference [29].

2 DISTRIBUTION OF SELENIUM IN THE BODY AND SELENIUM KINETICS

In sheep fed a concentrate ration containing 0.38 ppm Se and 30 days after an injection with radioactive Se from Na selenite (100 µCi/animal), tissue Se concentrations in kidney, liver, heart and muscle were (ng Se/g wet weight) 0.642, 0.466, 0.297 and 0.090 [45].
Little is known about the mechanisms of Se absorption or whether or not animals exert homeostatic control over its absorption. The evidence on the influence of the level of Se intake on apparent Se absorption is conflicting [69]. Selenium is absorbed primarily from the small intestine, with little or no absorption from or even net secretion into rumen or abomasum [37; 52]. Selenate has been shown to share an absorptive pathway with molybdate and sulphate and, hence, absorption may be influenced by these compounds [69]. In the plasma, Se is mainly incorporated in selenocysteine-rich proteins, and may be made available for the synthesis of other selenoproteins by the activity of enzymes as selenocysteine ß lyase [69]. In cases of increased dietary Se supply, the main increases in tissue Se concentrations are found in bone and muscle [16], although injected Se is reported to accumulate in the liver [40]. After administration of ^{75}Se to sheep, the highest activity was found in the kidney, whereas muscles had very low activities [56]. In non-ruminant calves fed as much as 10 or 40 ppm Se (DM; from Na selenate), Se was found to accumulate mainly in the liver, yielding high Se levels in the bile (bone Se concentrations not determined) [34]. In pregnant animals, Se efficiently crosses the placenta [53]. The Se status of neonates is, therefore, dependent on the Se status of the dams [27]. In an *in vitro* study with goat mammary gland slices, uptake of Se by the mammary tissue appeared not to be significantly influenced by several plasma cofactors[5], but rather to occur by diffusion. Although mediated transport could not be excluded, no evidence for any form of active transport into the udder tissue could be demonstrated [44]. Upon subcutaneous application of ^{75}SeMet to lactating goats, the butter fat appeared to be free from radioactivity, whereas nearly 70% of the total radioactivity in milk was recovered from the casein fraction, indicating the incorporation of ^{75}SeMet in the methionine fraction of the casein [29].

[5] not specified.

3 SELENIUM METABOLISM

3.1 Selenium absorption from different sources

The Se content of plants varies substantially [69] and is dependent on the Se status of the soil (Se content of soil and plants growing on it are highly correlated [53]), plant species, part of the plant (the leaves of the plant containing 1.5-2 times as much Se as the stems [53]) and the season [69]. As the Se content of the soil increases, the Se content of the seeds increases more than that of the vegetative matter [53]. The Se content of legumes tends to be lower than that of grasses, but this difference decreases at lower soil Se concentrations. The Se content of cereals can vary considerably, wheat having higher concentrations than barley, oats and lupine seeds [69]. Selenium absorption and retention from concentrate-based rations are significantly greater than from forage-based rations [37].
The main inorganic forms of Se to be supplemented to ruminants are Na selenate ($Na_2SeO_4.10H_2O$) and Na selenite ($Na_2SeO_3.5H_2O$). Moreover, selenium dioxide (SeO_2) has been suggested to be useful as a component of drenches to prevent Se deficiency. In the case of the use of pluronic drenches to prevent bloat together with Se preparations to prevent Se deficiency, SeO_2 may have advantages over selenate. On standing, both selenates and selenites were precipitated out of solution, whereas SeO_2 was not [19]. No data are available on (differences in) ruminal metabolism of Se compounds. Available organic Se sources are Se-enriched yeast, SeMet and SeCys [6; 35]. Feed ingredients such as brewers grains may be also used as a Se source [49; 53].

3.1.1 Cattle

Either Na selenite or Se-enriched yeast cultures supplemented at 1 mg Se/day to the ration of dairy and beef calves (250 kg initial BW, alfalfa silage/barley/soybean meal ration, 112 days on experiment) had no effect on performance of the animals, but the yeast treatment resulted in significantly higher blood Se concentrations and GSH-Px activities [49].
In an experiment with dairy cows on a grass/clover pasture (30-38 µg Se/kg DM) in their last weeks of lactation (± 12 kg of milk), the addition of 0.64 mg of Se/day as a drench during 56 days revealed no differences in blood Se levels between either Se dioxide or Na selenate treated animals [19]. In another experiment with lactating dairy cows initially yielding 35 kg of milk on a mixed diet (haylage/maize/cottonseed/soybean meal) the addition of either Na selenate or Na selenite (total Se concentrations 0.276 and 0.268 ppm (DM), respectively) during 9 weeks, did not influence serum GSH-Px levels, whereas final serum Se concentrations were significantly higher in the selenate-treated group (1.47 vs. 1.35 µM) [57].
When dairy cows (10-40 kg of milk) were fed alfalfa based diets supplemented with either Na selenite or Se-rich feeds such as brewers grains, plasma and milk Se concentrations were lowest in the selenite-supplemented groups [12]. Maybe the difference was due to a greater bioavailability of Se from brewers grains than from Na selenite. However, comparison of the different groups was difficult as not only the ration constituents, but also the Se concentrations were different. The only groups that were more or less comparable (alfalfa-/maize silage/concentrate + either Na selenite or brewers grains; total Se intake 2.53 and 2.87 mg/day, respectively) resulted in plasma Se concentrations of 0.8 and 1.2 µM and milk Se concentrations of 0.017 and 0.029 ppm, respectively.

3.1.2 Sheep

In sheep fed alfalfa hay with or without barley (0.27 or 0.38 ppm Se in the basal diet, total intake 555 and 843 µg Se/day, respectively), apparent absorption (42.8 vs. 38.5% of dose) and retention (35.4 vs. 29.1% of dose) of Se from Na selenite were significantly higher than

from Se-enriched yeast [37]. On the other hand, in lambs no differences could be observed in GSH-Px activities or performance after supplementing Se as Se yeast or as Na selenite for 3.5 months [35]. The ration consisted of hay and concentrates and the calculated Se intake was 0.12-0.15 mg/day.
No significant differences in serum Se or GSH-Px levels could be demonstrated when either Na selenate or Na selenite were added to the ration of sheep (maize/soybean meal/alfalfa, 0.3 ppm Se (DM) added). Muscle and liver Se concentrations were slightly higher in the selenite-treated group [57]. Selenomethionine caused a lower Se excretion in the urine of wethers than Na selenite (1 ppm of additional Se). After 13 days of treatment, Se concentrations in nearly all organs were significantly higher, whereas blood Se concentrations were only slightly higher in the SeMet group compared to the selenite group [15].

3.1.3 Goats

In a comparison of Na selenite (roughage + selenite added to concentrates) and Se from Se-fertilized roughage (both 0.4 ppm Se (DM)) fed to lactating goats, the latter ration appeared to significantly increase the Se content in hair, whereas the Se content of the milk tended to be higher [10].

3.1.4 Discussion and conclusions

Results of Se bioavailability trials in ruminants are summarized in Table 1.

Table 1 Summarized results of bioavailability trials with different Se sources for ruminants

Reference	Category	Sources used	Bioavailability
[49]	Dairy and beef calves	Na selenite, Se-enriched yeast	Se-yeast = selenite (performance) Se-yeast > selenite (blood Se and GSH-Px)
[19]	Lactating dairy cows	Se dioxide, Na selenate	dioxide = selenate (blood Se)
[57]	Lactating dairy cows	Na selenite, Na selenate	selenate = selenite (serum GSH-Px) selenate > selenite (serum Se)
[12]	Lactating dairy cows	Na selenite, brewers grains	brewers grains > Na selenite
[37]	Sheep	Na selenite, Se-enriched yeast	selenite > Se-yeast (apparent Se absorption + retention)
[35]	Lambs	Na selenite, Se-yeast	Se-yeast = selenite (GSH-Px, performance)
[57]	Sheep	Na selenite, Na selenate	selenite = selenate (serum Se, GSH-Px)
[15]	Wether lambs	Na selenite, SeMet	SeMet > selenite (tissue Se, urinary Se excretion)
[10]	Lactating goats	Na selenite, Se- fertilized roughage	roughage > selenite (hair)

For all ruminants, the differences between the several inorganic sources (Na selenite, Na selenate, Se dioxide) are minor. There is conflicting evidence as to the difference between Se-enriched yeast and inorganic Se sources, so no clear advantage of one of these components can be derived from the literature. Selenomethionine seems to have advantages over selenite, but evidence is scarce. Although SeMet is the predominant form of Se occurring in feeds and forages, considerable differences occur as to apparent Se availability from either feeds or inorganic Se sources. Possibly incorporation of SeMet (from supplements) into bacterial proteins and differences in adsorption of Se-containing bacteria to the insoluble particulate fraction of rumen contents (thereby lowering Se absorption in the

small intestine) contribute to the observed differences. Thus, apparent Se availability appears to be greater (52.8 vs. 41.8%) from a concentrate than from a lucerne hay diet [69].
As a conclusion, due to lack of data no superior Se source for use in ruminants can be designated.

3.2 Interactions influencing selenium absorption

3.2.1 Interactions of selenium and calcium

3.2.1.1 Cattle

In dry, pregnant cows (n = 11), apparent Se absorption was influenced by the Ca content of the ration (maize silage/hay/concentrates; Se varying from 896 to 1682 µg/day; 0.07-0.14 ppm Se (DM)) [24]. Calcium levels applied ranged from ± 4 to 14 g/kg DM. Depending on the Ca content of the ration, the apparent Se absorption varied between ± 25 and 45% and the relation could be described by the equation
$A = -0.355 + 1.974\ C - 1.195\ C^2$ $(R^2 = 0.775)$

in which A = apparent Se absorption
C = Ca concentration of the ration (% (DM)).

This interaction implies that the apparent Se absorption is maximal when the Ca content of the ration is about 8 g/kg DM.
However, in dairy calves (n = 4/group) fed diets (maize/soybean meal/grass pellets/ cottonseed hulls) containing either 1.7, 6.7, 13.1 or 23.5 g Ca/kg (extra Ca from $CaCO_3$) and 0.062 ppm Se, no significant Ca effect on apparent ^{75}Se absorption or tissue ^{75}Se concentrations could be demonstrated [3].

3.2.1.2 Sheep and goats

No experimental evidence is available on interactions of Se and Ca in small ruminants.

3.2.1.3 Conclusion

Evidence on influences of Ca on Se metabolism does not agree. Due to scarcity of data, no unequivocal conclusion can be drawn.

3.2.2 Interactions of selenium and Iron

For non-ruminants, conflicting results have been found as to the influence of dietary Fe on Se metabolism [1; 47]. However, although Fe has been mentioned as a dietary component interacting with Se metabolism in ruminants [11], convincing evidence is lacking as yet.

3.2.3 Interactions of selenium and sulphur

Due to the chemical similarity of Se and S, interactions between these elements have been suggested. In several cases, S has been reported to influence Se absorption [6; 51]. A survey of results is given in Table 2.

Table 2 Selected results from experiments on the influence of S on Se metabolism

Reference	Category	total Se (ppm)	total S (g/kg)	S-source	Observations
[72]	Ewes and lambs	0.01 or 0.22	3.9 or 5.7	K_2SO_4/selenite/ DL-Met	*higher incidence of degenerative heart lesions in sulate-group vs. control group *higher levels of ASAT, LDH and MDH in both low-Se and S-supplemented groups
	Ewes	0.13 or 0.16	2.8 or 7.8	Na_2SO_4	* higher ^{75}Se-activity in small intestine, uterus and omasum, but no differences in Se-excretion due to additional S
[30]	Ewes and lambs	0.04 or 0.21	1.7 or 5.0	Na selenite/ Na_2SO_4/Cys/Met	* higher incidence of WMD in sulphate-group vs. control group
[62]	Sheep	0.35, 0.88 or 1.34 (DM)	2.15 or 3.97 (DM)	Na_2SO_4	* lower liver Se concentrations at the high S level
[58]	Sheep	0.25	0.5, 1.1, 1.7, or 2.4	Na_2SO_4	* 22 (0.5 S) and 12 % (2.4 S) of a ^{75}Se dose excreted via the urine
[36]	Beef calves	(DM) 0.14 (hay) and 0.23-0.25 (concentrates)	2.0 or 7.5 g/kg (concentrates)	$CaSO_4$	* no significant differences in performance, blood GSH-Px, SOD, ASAT, AChE, G6PD activities or Se concentrations
[33]	Dairy cows	0.14 or 0.27 ppm (DM)	2, 4 or 7 g/kg DM	$MgSO_4$/$CaSO_4$	* lower DMI, apparent and estimated true Se availability at increasing S levels * positive Se balance at S level <4 g/kg DM

WMD = white muscle disease; ASAT = aspartate aminotransferase; LDH = lactate dehydrogenase; MDH = malic dehydrogenase; Cys = cystine; Met = methionine, SOD = superoxide dismutase; AChE = achtylcholinesterase; G6PD = glucose-6-phosphate dehydrogenase

As yet, due to conflicting results, quantification of the effect of S on Se metabolism is impossible. The ARC [6] suggests an additional factor may influence this interaction.

3.2.4 Interactions of selenium and arsenic

Although As has been mentioned to influence Se metabolism [11], no suitable reports on the effect of As on Se metabolism in ruminants are available. In rats, As has been demonstrated to increase the toxicity of several methylated Se compounds, as trimethylselenonium [39; 54], methylseleninic acid, dimethylselenoxide, selenobetaine (methylester), Se-methylselenocysteine and SeMet [39]. On the other hand, Se reduced methylation of As, which may increase As toxicity [66].

3.2.5 Interactions of selenium and lead

3.2.5.1 Cattle

In dairy calves (70 d of age, 89 kg BW) on a maize/soybean meal/grass pellets/cottonseed hulls ration, the addition of 1000 ppm Pb (from $PbSO_4$; Pb-group) to the basal ration (2 ppm Pb, control group) significantly influenced ^{75}Se metabolism [48]. The experiment lasted for 28 days; on day 24, a single oral dose of 500 µCi of ^{75}Se was administered. By day 28, ± 65% of dose was excreted via the faeces in the Pb-group, whereas faecal excretion in the control group was ± 52% of dose. At the same time, urinary ^{75}Se excretion was lower in the Pb group (± 2.5% of dose) compared with the control group (± 4% of dose). ^{75}Se accumulation in kidney, liver, pancreas, small intestine, heart, spinal cord and muscle was significantly lower in the Pb-group compared with the control group.

3.2.5.2 Sheep and goats

As yet, no data are available on influences of Pb on Se metabolism in small ruminants.

3.2.5.3 Conclusion

As argued in Documentation Report nr. 48, Pb concentrations in forage can range from 500-1300 ppm Pb (DM) in heavily contaminated areas. Assuming a DM content of the ration of the dairy calves mentioned (see par. 3.2.5.1) of 90%, the Pb level applied in the control group will be ± 1100 ppm (DM). Therefore, the Pb level applied in the calf experiment will be applicable in practice. Under conditions of heavy Pb contamination, decreased Se absorption due to the influence of Pb will have to be accounted with. Due to scarcity of data, this effect cannot be quantified.

3.2.6 Interactions of selenium and copper

Contrary to most interactions, that reduce the availability of trace elements, Cu has been shown to increase liver Se concentrations in sheep [62; 75]. When rations (no data given) containing 6.7 vs. 17.0 ppm Cu (DM) were fed to sheep, liver Se concentrations increased from 2.30 to 3.43 ppm (DM) [62]. However, in another experiment ^{75}Se content of muscles was significantly decreased[6] after feeding extra Cu[7] [75]. Moreover, there appeared to exist a three-way interaction with dietary S: when dietary S concentrations were highest (3.97 vs. 2.15 g/kg DM), S reduced the hepatic Se concentration, which was most pronounced at the high Cu intake [62]. In another experiment [68], no consistent influence of injected Cu on the growth response of lambs to injected Se could be demonstrated. In lactating goats, as much as 100 ppm of dietary Cu has been demonstrated not to influence Se metabolism [4].

3.3 Recycling

In sheep, Se excretion via the bile can be up to 28% of intake [40]. Most of this is subsequently reabsorbed [69], although a part contributes to the faecal endogenous losses. Faecal endogenous losses seem to increase with increasing DMI [69].

3.4 Excretion

Normally, the faeces is the main route of excretion of Se. Besides this, considerable amounts of Se can be excreted via the urine and thus contribute to Se homeostasis. Finally, Se can be excreted by exhalation [69].
Adding 10 instead of 0.2 ppm Se (DM) to a milk substitute ration of calves during 6 weeks increased biliary Se concentrations from 0.006 to 0.39 ppm (DM) [34]. When 40 ppm of additional Se was fed during another 12 days, biliary Se concentration was 2.75 ppm (DM). In sheep given 38.6 µg of ^{75}Se (as SeO_4) by rumen puncture, approximately 69% of the dose had been excreted in the faeces in 7 days [56], whereas <5% of the total dose had been excreted via the urine. However, urinary Se excretion can vary considerably. In sheep, approximately 7-10% of a dietary Se dose from yeast or selenite supplements was excreted via the urine, whereas urinary excretion of feed Se was 18-24% of intake [37]. On concentrate-based diets compared with forage-based diets, urinary Se output tends to be higher. Finally, when sheep and goats were Se-loaded from fly ash grown sweet clover (ration containing 23% sweet clover containing 66 ppm Se (DM)), urinary Se concentrations

[6] From 16,9% to 11.6% of dose at 13 days after dosing.
[7] 10 ram lambs per group, lucerne hay/shelled maize/oats diet containing 1.2 ppm Mo, 1,6 g S/kg and either 6.4 or 16.4 ppm Cu in the DM

increased on average 200-fold, whereas faecal Se concentrations increased on average 45-fold [16]. In sheep of low selenium status and in negative Se-balance, urinary Se excretion can be 40-50% of intake [40; 69].

4 SELENIUM REQUIREMENTS

The Se requirements of adult animals are determined by the endogenous (inevitable) losses and the secretion into milk. In growing and pregnant animals Se is also deposited in growing (foetal) tissues. However, using the factorial approach to estimate Se requirements is difficult because the Se deposition in tissues, conceptus and milk depends on the Se-intake [16; 53].

4.1 Cattle

For dry cows, as well as for sheep and goats, apparent Se availability from feed is between 30 and 60 %. Although data are scarce, true Se availability is reported to be between 40 and 65 % [53]. For calculation of Se requirements, a value of 40% for true Se availability is chosen. In pre-ruminant calves, apparent Se absorption has been calculated to be 60% [34].

4.1.1 Dairy cattle

The endogenous faecal Se loss varies between 0.011 and 0.019 mg/kg DMI [37], whereas in lactating cows (milk yield on average 26 kg) consuming ± 2.5 mg Se/day urinary Se excretion was on average 0.5 mg/day [25; 33; 53]. Related to BW, total endogenous Se loss is assessed to be 0.25 µg/kg BW. However, as this would result in extremely low requirements, arbitrarily a value of 0.50 µg/kg BW is chosen for all ruminants.
Selenium content of growing tissues is reported to be 50 µg/kg growth [69]. No qualification of this estimate is given. This value is used for all ruminants.
In pregnant cows consuming a ration containing 0.3 ppm Se (DM), the conceptus accumulates approximately 0.055 mg of Se/day during the last trimester of gestation [31]. No more data are available on Se requirements during other stages of pregnancy. As only one, relatively high Se level (0.27 ppm Se (DM) (total mixed ration, 10-12 kg DMI)[8] has been employed, this estimate is probably not “minimal”. For calculation of requirements, a value of 0.06 mg/day is adopted.
Colostrum is reported to contain 0.063 mg Se/kg [8]. The Se content of mature milk is assessed to be 0.016 [61], 0.026 [8] or 0.01 to 0.025 [53]. For calculation of requirements, a value of 0.02 mg Se/kg is chosen for all ruminants. Dairy cows fed 0.78 to 11.47 mg Se/day during 90 days had final milk Se concentrations of 0.010 to 0.037 mg/kg, respectively [12]. In a Dutch survey, milk Se concentrations appeared to range from 0.003 to 0.011 mg Se/kg milk [9]. In this survey, milk from farms on sand and peat soils had the lowest values (mean 0.004 mg Se/kg), whereas milk from farms on marine clay contained on average 0.008 mg Se/kg. On average, a value of 0.01 mg Se/kg milk seems to be a reasonable estimation for Dutch dairy cattle. Regarding the lower levels mentioned, this value couldn’t be classified to be “minimal”.

4.1.1 Beef cattle

No separate calculations are recommended for the Se requirements of beef cattle as compared to dairy cattle [52].

[8] Besides this, the animals were also given lucerne hay/grass hay containing <0.1 ppm Se (DM) during the early dry period.

4.2 Sheep

Calculation of the Se requirements of sheep is essentially similar to that of cattle. Endogenous loss is similar to that of cattle. Selenium content of growing tissues is reported to be 0.044 mg/kg growth [20]. Unless proven otherwise, the values for cattle are used.
Sheep colostrum contains 0.021 to 0.030 mg Se/kg [29], whereas sheep milk contains 0.028 mg Se/kg [14] or 0.010 to 0.018 mg Se/kg [29]. On average, a value of 0.02 mg Se/kg is considered to be a reliable estimation. However, Underwood and Suttle estimate the Se content of milk to be 0.006 mg/kg [69]. In deficient ewes, colostrum and milk Se content can be <0.010 mg Se/kg [29].
No data are available on Se requirements for gestating sheep.
Wool contains 0.2 ppm Se (DM)[5; 16]. Therefore, as wool yield varies with breed from 1-3.6 kg of DM/year [6], the Se excretion via wool is 0.2-0.7 mg/year, or 0.5-2.0 µg/day.

4.3 Goats

For goats, data on endogenous Se loss, as well as on Se content of growing tissues, are lacking. Unless proven otherwise, the values for cattle are used.
Goat colostrum contains (mg/kg) 0.135 (day 1) to 0.065 (day 2) [8], whereas mature goat milk contains (mg/kg) 0.01 [16], 0.02 [46; 61] or 0.023 [8]. On average, these values for mature milk are 0.02 mg Se/kg. However, when goats are Se-loaded from fly ash-grown sweet clover, (23% sweet clover containing 66 ppm Se (DM)), milk Se concentrations increased on average 60-fold [16].
No data are available on Se requirements for gestating goats.
Goat hair is reported to contain 0.3 ppm Se (DM) [16].

4.4 Conclusion

The following equation can be used to calculate the required Se-concentration of ruminant rations:

$$C = \frac{(BW \times 0.5) + (kg\ milk \times 20) + (kg\ growth \times a) + b}{10 \times A_{Se} \times DMI}$$

in which
- C = required dietary Se concentration (ppm (DM))
- BW = body weight (kg)
- A_{Se} = true Se absorption (40 %)
- DMI = dry matter intake (kg/day).
- a = Se content of growing tissues (50 µ/kg growth for cattle and 40 µg/kg growth for sheep)
- b = amount of Se needed for gestation (0.06 mg/day for cattle during the last trimester); for sheep and goats, the amount of Se needed for gestation is unknown; related to metabolic BW, a value of 0.01 mg/day is calculated.

5 ALLOWANCES

Using the equation mentioned (see par. 4.4), some examples of dietary Se requirements and allowances have been tabulated (Table 3).

Table 3 Examples of calculated Se requirements (ppm Se (DM)) and allowances (including a safety margin of 50%). Assumed body weight: cow 650 kg, sheep 75 kg and goat 70 kg.

Category	DMI	Requirement	Allowance	
	kg	mg/day	mg/day	Ppm (DM)
Growing female cattle				
4 months, 850 g growth/day, 130 kg BW	3.9	0.27	0.40	0.10
9 months, 700 g growth/day, 250 kg BW	5.6	0.41	0.62	0.11
16 months, 625 g growth/day, 400 kg BW	7.3	0.58	0.87	0.12
Dairy cattle (650 kg BW)				
Cow, dry, pregnant, 8-3 wk a.p.	11.5	0.95	1.43	0.12
Cow, dry, pregnant, 3-0 wk a.p.	11.0	0.95	1.43	0.13
Cow, lactating, 20 kg of milk	18.5	1.64	2.47	0.13
Cow, lactating, 40 kg of milk	23.5	2.48	3.71	0.16
Beef cattle, intermediate type				
1000 g growth/day, 100 kg BW	3	0.25	0.38	0.13
1200 g growth/day, 250 kg BW	6	0.46	0.69	0.12
1100 g growth/day, 500 kg BW	9	0.76	1.14	0.13
Veal calves				
1150 g growth/day, 150 kg BW	4.5	0.33	0.50	0.11
1450 g growth/day, 275 kg BW	7	0.53	0.79	0.11
Sheep (75 kg BW)				
growing lamb, 0.3 kg growth/day, 40 kg BW	1.6	0.08	0.12	0.08
Sheep, pregnant, last trimester	1.9	0.12	0.18	0.10
Sheep, lactating, 3 kg of milk, nursing 2 lambs	2.6	0.25	0.38	0.14
Goats (70 kg BW)				
goat, pregnant, last trimester	1.7	0.11	0.17	0.10
goat, lactating, 4 kg of milk	3.2	0.26	0.40	0.12

Regarding the considerable variations in several parameters of the factorial approach (e.g. A_{Se}, Se concentration in milk) and the assumptions made, the above estimations of Se requirements are relatively rough. Judgement as to what extent these estimates are "minimal" is therefore, precluded. As can be derived from Table 3, in most cases 0.1 ppm Se (DM) should be sufficient to cover the animals' needs. This is in accordance with most recommendations. For beef cattle and goats, 0.1 ppm is recommended [46; 52]. Similarly, 0.1 ppm (DM) is recommended to be sufficient for all ruminants [32], supposing the vitamin E supply is adequate (10-15 mg vitamin E/kg DM [6; 32]. Some more examples are given in reference [69] (Table 15.8). Slight differences may arise from slightly other assumptions.
On the other hand, for dairy cattle, the NRC recommends 0.3 ppm Se (DM). Such a high requirement is not supported by the factorial estimation as given above. The recommended level of 0.3 ppm Se (DM) is reported to be necessary to maintain whole blood (2.3 µM) or plasma (1.0 µM) Se concentrations at levels considered to be adequate to prevent Se-responsive mastitis [53].
For reproducing sheep, based on the attainment of a plateau in tissue GSH-Px levels a requirement of 0.12 ppm (DM) was estimated [55].

6 CRITERIA TO JUDGE SELENIUM STATUS

6.1 Criteria to judge selenium status

Diagnosis of Se-responsive disorders is hampered as the symptoms are unspecific and biochemical indicators have several limitations [69].

6.1.1 Selenium concentrations in soil and ration

Selenium concentrations in soil (<0.5 mg Se/kg) can yield low Se concentrations of the crop (<0.05 ppm (DM)). However, even at forage levels of 0.02-0.03 ppm (DM) not all Se-deficiency-like disorders in animals consuming these forages may be Se-responsive [69], although forage concentrations <0.10 ppm (DM) are considered deficient [59]. In general, Se uptake by plants is lowest at a soil pH of ± 6, the Se uptake being higher at both higher and lower pH values [18]. However, this effect depends on soil type and cut number. Selenium uptake by ryegrass from clay / loamy soils is significantly lower than from sandy soils. In sandy soils, the pH-effect can be less clear or even opposite to that of loamy soils. All effects become less clear in successive cuts.

6.1.2 Blood parameters of selenium status

Both plasma or serum Se concentrations and GSH-Px activities in whole blood are suitable, highly correlated indicators of Se status[9] [13; 69]. In goats, both of them increased within 24 hours after Se supplementation (0.1 mg/kg BW from Na selenate, lucerne ration containing 20 ppm Se (DM)) [76]. However, both of them have several limitations. Both lack of clinical symptoms of Se deficiency accompanying low serum Se values (0.25 µM) and positive responses with regard to fertility to Se supplementation up to 1.1 µM Se in serum have been reported [69]. In cattle, serum Se values <0.10-0.12 µM [69] or even <0.3 µM [59] are considered to be deficient. For sheep, corresponding values are 0.25-0.50 µM, and for lambs 0.5-0.7 µM [69].
For whole blood GSH-Px activities in lambs, activities <100-150 U/g Hb are considered to indicate for deficiency. This is in accordance with Dutch reference values for cattle ([65] and Table 4). For mature sheep, values are similar to those presented for cattle in Table 4 [65]. For goats, reference values are supposed to be similar. More detailed surveys are given in Tables 15.6 and 15.7 of reference [69].
Experimental evidence on the value of tissue Se levels (e.g. liver, muscle) to judge Se status is slender and, hence, only tentative proposals can be given [69].

Table 4 Dietary and plasma/serum Se levels and GSH-Px activities for cattle [13; 59].

	Se [59]		GSH-Px [13]
	Ration	Plasma / serum	Whole blood
	ppm (DM)	µM	U/g Hb
Deficient	<0.10	0.03-0.3	< 120
Marginal	0.10-0.25	0.4-0.8	
Adequate	0.30-1.00	1.0-3.8	
High	3.00-4.00	31.6-44.3	> 600
Toxic (chronic)	>5.0	>44.3	
Toxic (acute)	>80		

[9] GSH-Px (U/g Hb) = 3.261 x Se (µg/kg) – 40.553; r = 0.93, P < 0.001 (GSH-Px activity to be measured in erythrocytes and Se content in whole blood) [13].

6.2 Conclusions

Both plasma/serum Se concentrations and GSH-Px activities of erythrocytes can be used to judge Se status. However, as Se concentration determination (as well as the determination of milk Se concentrations) is more expensive and difficult [11], the determination of GSH-Px activity of erythrocytes is more advantageous. On the other hand, one should be aware of the fact that Se concentrations decline earlier than GSH-Px activities [69]. As animals fed solely roughage (e.g. yearlings) may have a very low Se status, sampling of these animals is recommended in order to reveal a low farm Se status [13]. Determinations of Se in soil (insufficiently reliable) and in tissues (insufficiently validated) are not recommended.

7 DEFICIENCY

Selenium deficiency causes white muscle disease (WMD) or nutritional muscular dystrophy. Young cattle may develop the disease when turned out to spring pasture. It is characterized by leg weakness and stiffness, flexion of the hock joints, muscle tremors, arrhytmia, tachycardia, and abnormal breathing without neural involvement. Muscles are striated and calcified. The animals often die from cardiac failure due to impairment of the heart muscle. Impairment of disease resistance in Se deficiency is demonstrated by a shorter duration of mastitis when Se-deficient cattle are treated with Se. The incidence of mastitis can be reduced by vitamin E supplementation (2000-4000 IU/day). Selenium alone reduces the duration, but not the incidence of clinical mastitis [64]. An excellent vitamin E supply possibly prevents the occurrence of clinical symptoms in animals with low Se and GSH-Px levels in the blood [70]. Resistance against viral infections in calves or nematode infections in lambs are not impaired by chronic Se deficiency [26; 69]. In sheep lambs, WMD can occur from 0 to 12 months, but most commonly occurs at 3-6 weeks. Goat kids are believed to be more susceptible than are either lamb or calf [26; 53; 60; 69]. Moreover, Se can be involved in the pathogenesis of retained placenta [29]. Both Se- [71] and combined Se/vitamin E-treatment have been shown to be effective in some cases of retained placenta in cattle [6], and in several studies the condition could be prevented when Se was fed or injected during late gestation [53]. Finally, sometimes cases of infertility, abortion, poor wool growth and periodontal disease could be successfully treated by Se therapy [6; 29]. Erythrocytes of Se-deficient goats were more susceptible to haemolysis than those of goats receiving a sufficient amount of Se [22]. The effects of a Se-deficiency can be aggravated by concurrent deficiencies of Cu and Mn [47]. In general, based on milk Se levels peat and sand soils are considered to be Se deficient [9], and thus awareness of Se deficiency symptoms is indicated.

As soon as the Se content of ruminant diets falls below 0.08 ppm (DM), the frequency of clinical signs of Se-responsive disorders increases progressively [6]. For cattle and sheep, levels of 0.02-0.05 ppm Se (DM) are marginal [2; 69], whereas levels <0.03 ppm (DM) are regarded to be inadequate [6; 42]. Therefore, minimal dietary allowance for all ruminants is considered to be 0.1 ppm (DM). Many forages contain less than this concentration (Table 5). However, forage Se concentrations are not the best indicator of Se-deficiency risk [32; 52; 69].

Table 5 Selenium concentrations in Dutch forages (BLGG, Oosterbeek, 2003)

Type of forage	Harvesting period	Mean	Range (upper and lower limits)
		ppm (DM)	
Grass silage	1997-2002	0.048	0.037-0.058
Fresh grass	2002	0.029	0.023-0.045
Maize silage	1997-2002	0.019	0.009-0.043

In goats, Se deficiencies have rarely been reported [2;23], although these animals require both Se and vitamin E. Minimal requirements for vitamin E (d-alfa-tocopherol) are considered to be 10-15 mg/kg DM. When Se supply is marginal, even higher amounts of vitamin E may be necessary [2]. When highly unsaturated fat is fed to non-ruminant calves and lambs, extra vitamin E should be supplied at a level of 3 mg d-alfa-tocopherol or 4.5 mg dl-alfa-tocopheryl acetate per g polyunsaturated fatty acids (PUFA) [32].

Measures in deficiency and toxicity cases have been reviewed [69] and are summarized below.

7.1 Direct measures in deficiency cases

7.1.1 Direct continuous supplementation

Offering Se-fortified salts (26-30 mg Se/kg; either in loose or block form) *ad libitum* can be a convenient and cheap method of supplying extra Se to sheep. However, due to individual variation in salt consumption not all animals are protected. In one study e.g. 7-33% of ewes were left unprotected against Se deficiency [69].

7.1.2 Direct discontinuous supplementation

Direct supplementation of subcutaneous injections or oral drenches (usually either as Na selenite or Na selenate) can be applied in doses providing 0.1 mg/kg BW at intervals of 1-3 months. The optimal oral dose may rather be 0.2 mg/kg BW. It is important that the animals are treated at responsive moments (mating / insemination, late pregnancy, weaning) to optimise the treatment effect [69].

7.1.3 Slow release oral supplementation

Slow release boluses have been used in the prevention of white muscle disease in all ruminants. However, heavy boluses (containing Fe and Se) cannot be used in lambs or kids, whereas in cattle considerable losses (7-56%) of administered boluses have been observed. Soluble-glass boluses cause fewer problems in all kinds of ruminants. Parenteral application of Ba selenate (1 mg Se/kg BW) has been shown to be effective, although the recommended site of injection and the withdrawal period have to be observed with caution [69].

7.1.4 Indirect selenium supply via fertilization

Fertilization of pastures or grain crops with selenite solutions has several disadvantages. Selenium is poorly absorbed by plants, especially when soil pH is low. Residual effects are short-lived (up to 15 months [74]), whereas hazardously high Se levels can occur in the plants immediately after application of the fertilizer. Annual application of 10 g Se/ha (as selenate) to 25-33% of the grazing area combined with rotational grazing is more prudent [69]. A spring application of 10 g Se/ha (from selenate) produced herbage containing on average 1.86 ppm (DM) in the first cut. Selenite is far less potent in increasing herbage Se content than is selenate [63]. The application of a slow-release $BaSeO_4$ granulate (10-20 g Se/ha) is another safe way to supply extra Se for up to 3 years [73; 74]. However, the time lag between application and effects on the animals may be up to 6 weeks and, hence, direct treatment of the animals may be necessary to overcome this period [69].

8 TOXICITY

8.1 General aspects

Toxicity of Se can be either acute or chronic. Animals suffering from acute Se toxicity can show salivation, respiratory distress, pulmonary congestion, circulatory failure and degenerative changes in the heart, liver and kidney. Chronic Se poisoning is characterized by dullness, lack of vitality, emaciation, roughness of coat, hair loss, soreness and sloughing of the hooves, stiffness and lameness due to erosion of the joints of the long bones. Sudden collapse and death may occur. The clinical picture of chronic Se-toxicosis was often referred to as “blind staggers” (mainly associated with consumption of Se accumulating plants) or “alkali disease” (associated with intake of grass from Se-rich soils). However, experimental chronic Se toxicosis mainly caused epidermal alterations. Therefore, as the animals are unwilling to walk, starvation may aggravate the disease and contribute to the clinical picture [69].
Some soils are Se-rich, whereas some plants specifically accumulate Se. The Se accumulating plants can be divided into obligate and facultative Se accumulator plants. The obligate Se accumulator plants (*Astragalus* and *Haplopappus spp.*, and *Xylorrhiza glabriuscula* and *Stanleya pinnata*) require high amounts of Se for their growth. They can contain up to 10,000 ppm of Se [41] (or 125-4800 mg/kg DM [69]). Facultative Se accumulator plants do not need high amounts of Se for their growth, but can accumulate several hundred ppm of Se when grown on soils high in available Se. The more alkaline the soil, the more readily Se is taken up [41; 43]. On the other hand, the higher the S content of the soil the lower is the Se uptake by plants. Possibly the relatively low Se content of herbage on heavily fertilized pastures can be attributed to this antagonism [42]. Selenium in (accumulator) plants is present in the more toxic organic forms (methylselenocysteine, selenocystathionine and selenomethionine) [69]. Among the obligate accumulators, in the Netherlands only *Astragalus glycyphyllos* rarely occurs [28]. The other species mentioned do not occur. On the other hand, facultative accumulators such as *Aster spp.* and *Atriplex spp.* can occur abundantly [28]. In the USA, white sweet clover (*Melilotus alba*) growing on fly ash has been demonstrated to contain on average 66 ppm Se (DM), the upper limit in subsamples being 205 ppm Se (DM)[16]. In the Netherlands, sweet clover can also be an abundantly occurring plant, although it is not a typical pasture plant. No data are available on Se contents of Dutch *Atriplex* species, neither have plant-born Se toxicity cases been reported from the Netherlands. In fact, based on milk Se levels many peat and sand soil areas may be Se-deficient, whereas the remaining clay and löss areas are considered adequate in Se [9]. Higher forage Se levels are associated with an arid climate, as is evident in parts of Ireland and India [69]. Therefore, although some attention has to be paid to Se-accumulating plants, under Dutch circumstances their relevance seems to be minor.
Principally, the combustion of coal and wastes as well as industrial activities such as ore-processing discharge large quantities of Se into the atmosphere. However, it is not clear as to what extent these sources contribute to chronic Se toxicosis [69].
For all ruminant species, LD_{50} values of 0.15-1.9 mg/kg BW for injected and 1.9-8.3 mg/kg BW for oral inorganic Se are reported. Selenite, selenate and SeMet are more toxic than elemental Se or selenides [42; 69]. The NRC reports acute toxicity for dairy cattle when 10-20 mg Se/kg BW is fed, whereas injection of ± 0.5 mg Se/kg BW resulted in a 67% mortality rate [53]. Chronic Se toxicity can occur in cattle fed rations containing 5-40 ppm Se for several weeks or months [53], although a daily intake of 0.5 ppm (8.4 mg) Se has been reported to significantly increase lactate dehydrogenase and aspartate aminotransferase activities in grazing dairy cows when compared with control cows consuming 0.2 ppm (3.2 mg) Se [38]. This may be indicative for liver damage. A level of 3 ppm (DM) is considered to be the maximum tolerable dietary level for all ruminants [2; 32]. However, when non-ruminating calves on a milk-substitute ration containing 0.088 ppm Se (DM) were fed as

much as 40 ppm additional Se (DM, from Na selenate) during 12 days (after a period of 6 weeks on a diet containing 10 ppm (DM)), no clinical abnormalities due to Se toxicosis were observed, whereas feed intake and growth were only slightly reduced [34]. The authors suggested the extreme tolerance of the non-ruminant calf to Se toxicosis could be related to the casein content of the milk substitute, as casein has been demonstrated to protect rats against the toxic effects of a ration containing 10 ppm Se. The reason for this effect is not clear.

8.2 Direct measures in toxicity cases

As Se toxicity is incurable, no direct measures can be taken to treat this condition. Therefore only preventive measures can be taken [69]. Changing the ration by the removal of Se-rich feeds (or supplements) and introduction of feeds poor in Se is necessary.

9 PREVENTION

9.1 Short-term prevention strategies

Supplying either bromobenzene (cattle), As (25 mg from Na arsenite; rats, cattle), sulfate mixture (30 g/day; buffaloes) or Hg (chicks, quail) have been reported to give some protection against Se toxicity [69].

9.2 Long-term prevention strategies

Treating the soil with S or gypsum ($CaSO_4$) have been mostly ineffective in reducing Se uptake by plants, although raising the S: Se ratio of the soil has sometimes been successful. Heavy dressings with superphosphate (containing $CaSO_4$) have been indicated as the cause of Se deficiency in grazing animals, possibly because of increased soil S content [69]. However, in a German study no influence of the application of 50 vs. 0 kg S/ha/year on the Se content of pasture grass could be observed [67].

9.3 Conclusion

Each of these preventive methods has clear disadvantages with respect to residues and human and animal health (bromobenzene, As, Hg) or interaction with other trace elements (e.g. S with Cu), whereas the reported results are doubtful.

Table 6 Inventory of Se allowances for cattle, sheep and goats as used in some foreign countries (ppm (DM)).

Country	**Ref.**	***Allowance***				
		Cattle	**Ref.**	**Sheep**	**Ref.**	**Goat**
Great Britain	[2; 6]	0.1				
USA[a,b]	[52; 53]	0.3 (DM; dairy cattle) 0.1 (beef cattle)	[51]	0.1	[50]	?
Germany	[17]	0.15 (growth) 0.20 (mature)		?	[7]	0.1-0.2
France	[21]	0.1 (is deficiency limit)				

[a] Allowances for cattle are expressed in mg/kg feed as fed; as DM contents of the feeds are not given, allowances cannot be calculated in ppm (DM)
[b] minimum requirements

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ANNEX: OVERVIEW OF THE SERIES OF CVB DOCUMENTATION REPORTS 'REVIEWS ON THE MINERAL PROVISION IN RUMINANTS'

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