

Reviews on the mineral provision
in ruminants (XVI):
CONTAMINANTS: CADMIUM, LEAD,
MERCURY, ARSENIC AND RADIO
NUCLIDES

A.M. van den Top

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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 Manganese provision in ruminants the COMV expresses its gratitude to the author, dr. A.M. van den Top.

Utrecht/Lelystad, November 2005.

Professor dr. ir. A.C. Beynen
Chair of the COMV

Dr. M.C. Blok
Secretary of the COMV and Head of the CVB

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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ABBREVIATIONS

The following abbreviations have been used:

ALA-D = delta-aminolevulinic acid dehydratase

BW = body weight

DM = dry matter

DMI = dry matter intake

DSMA = disodium methanearsonate

MSMA = monosodium methanearsonate

1 FUNCTIONS AND TOXIC EFFECTS IN THE BODY

1.1 Lead and mercury

Lead and Hg are not known to be essential to ruminants. These elements have, therefore, to be considered only toxic.

1.2 Cadmium

Within the body, Cd binds to hemoglobin and thionein. By impairing absorption and binding to thionein Cd can be detrimental to Cu, Zn, Fe and Mn absorption and metabolism [37;69;108;118;156]. The remaining Cd accumulates in the kidney (cortex) and causes kidney tissue damage [124]. Besides this, Cd causes hypocalcemia and immunosuppression [59;69].

1.3 Lead

Toxic effects of Pb include irritation of the gastrointestinal tract, degeneration of capillaries, and impairment of heart, liver, kidney, lung and brain functions [15].

1.4 Mercury

The toxicity of organic Hg compounds is considerably greater than that of inorganic compounds. Inorganic Hg salts are caustic and emetic [93]. Mercury has an affinity for sulphhydryl groups (thereby impairing the activity of many enzymes) and metallothionein. Hg poisoning causes fatty liver degeneration and depression of humoral immunity [102;142].

1.5 Arsenic

Both inorganic and aliphatic organic As compounds cause necrotic lesions in several parts of the gastrointestinal tract. These lesions may even progress to perforation. Capillary vessels are damaged, resulting in transudation, bleedings and shock. Trivalent inorganic and aliphatic organic As compounds, in contrast with pentavalent and aromatic organic As compounds, react with sulphhydryl groups of many enzymes. This results e.g. in disturbances of glycolysis and the citric acid cycle enzymes. Arsenate can uncouple the oxydative phosphorylation. A comprehensive survey of these interactions is given in reference [43]. Moreover, in man and in hamsters As has been found to be teratogenic [43;48], but as yet no such effect has been proven in ruminants.

In As-deficient animals activities of several liver enzymes involved in lipoprotein metabolism are reduced when compared with animals receiving sufficient amounts of As [8]⁵. However, no specific biochemical role of As is known.

⁵ No data on As content of the ration or animal species given.

2 SOURCES, DISTRIBUTION AND KINETICS

2.1 Cadmium

Emissions of (tin) mines, smelters, coal and oil products combustion, as well as cigarette smoke, contribute to Cd pollution of air and environment [69]. However, direct inhalation of Cd from polluted air is of minor importance for ruminants [126]. (Super)phosphate fertilizers and sludge also contribute to Cd loading of agricultural soils [61;142]. Among normal, uncontaminated soils, peat and löss soils are reported to have higher Cd concentrations (0.87 and 0.78 mg/kg, respectively) than clay and sand soils (0.41 and 0.32 mg/kg, respectively) [40]. Herbage harvested from areas of high natural soil Cd content can contain up to 3 ppm (DM), and in industrial areas, up to 40 ppm (DM), 2 mg/kg [156] or 4 mg/kg dry soil [75]. In the past, maize silage at 5 km from a Zn processing factory (Budel) was demonstrated to contain 2.7 ppm Cd (DM) [75]. Increasing soil pH decreases Cd uptake by plants [156]. Accordingly, sheep grazing pastures on acidic soils had higher kidney Cd concentrations than their counterparts on alkaline soils [89].

Homeostatic control of Cd metabolism in ruminants [15;83], as well as passage of the placenta [122;126;134], is minor.

Cadmium is mainly recovered from the kidney, the liver showing the next highest concentrations [88]. Within the liver, Cd is bound to thionein [124]. In sheep and cattle, Cd concentrations seemed to attain a plateau after 6-18 months on a certain diet (suggesting some homeostatic control) [135;138;142], whereas kidney cortex increased both with dose time and duration of exposure. However, due to the small number of animals no significant differences could be observed [138]. Female calves accumulated more Cd in their kidneys than did male calves [88]. Similarly, ewes > 3.5 years of age had higher kidney Cd levels than wethers of the same age [106]. Cattle exposed for 8 years to Cd-containing sludge showed 5- to 8-fold increases in kidney and liver Cd concentrations [50;142]. Selected data on tissue Cd distribution are given in Table 18.3.

2.2 Lead

In industrial and urban areas Pb containing dust can be deposited on plants. Although it is not taken up in the plants, it cannot be easily washed off. Roots and tubers are not loaded with Pb in this way. Normal, uncontaminated forage contains up to 10 ppm Pb (DM) [60]. In the past, forage harvested in the vicinity of frequently used highways could contain up to 300 ppm Pb or 210 ppm (DM) [145]. Even up to 100 m away from the roadside increased Pb levels could be found [60]. No recent data (after the abandonment of Pb-containing fuel) on roadside forage Pb levels are available. However, it is not usual that such forage is fed in large amounts to cattle⁶, whereas the Pb content of the total ration will be lower due to "dilution" with (low-Pb) concentrates [153-155]. In some cases small ruminants will be more at risk than cattle because of their use as "lawn mowers" of verges and dikes along highways. In such cases, the direct inhalation of automotive exhaust also contributes to heavy metal loading. In the vicinity of mines and Pb/Zn smelters, forage Pb levels can increase to hazardous altitudes. For instance, downstream a smelter in India grass Pb levels were 163-212 ppm compared with 5 ppm before the start of the smelter [41]. In upright silage silos, Pb may migrate by leaching from the upper layers and accumulate in the silage at the

⁶ Although in some cases roadside forage is grazed by or cut and fed to cattle, in most cases such types of roughage are not suitable for use in the ration of high-producing dairy cattle because of low nutrient content, the presence of rubbish and (toxic) weeds. Application may mainly be considered for dry cow rations.

bottom of the silo [33]. Several cases of intoxications of ruminants from lead-based paint⁷, used motor oil, oil filters and discarded storage batteries have been reported [11;23;139;158]. The latter causes of accidental Pb toxicity mainly occur in animals < 1 year of age, because of their explorative (licking) behavior [23]. Finally, Pb shot (e.g. from clay pigeon shooting) in grass silage [116] or barley [131] occasionally causes Pb toxicity, although cattle grazing such pastures can have normal Pb levels in blood [22] and organs [30].

Accumulation of Pb in calves is not dependent on gender [88]. Homeostatic control of Pb is minor, although lambs retain diminishing proportions of the ingested Pb during a 7-weeks exposure [142]. In pregnant cattle, Pb passes the placenta to the fetus [111]. Lead is mainly accumulated in kidney, liver and bones, whereas muscle levels are much lower [88;124]. Selected data on tissue Pb distribution are given in Table 18.3.

2.3 Mercury

Mercury-containing fungicides (see 18.8), industrial contamination (e.g. from smelters and mining activities), traffic and the application of contaminated sludge are the most important sources of Hg. Besides this, volcanic activity and erosion of rocks contribute to atmospheric Hg concentrations. Theoretically, both contaminated feed and polluted air can contribute to Hg load of animals. Intrapulmonary Hg resorption rate from polluted air is supposed to be much higher than intestinal uptake of dietary Hg. However, in areas with increased air Hg concentrations contamination of the feed is usually such high that the absolute contribution of air Hg to total Hg load of the animal is minor [68;124]. Inorganic Hg can be methylated to methyl Hg, which is much more readily taken up than inorganic Hg compounds (see 18.3). As this mainly occurs in aquatic environments, fish meal is relatively rich in Hg [124]. However, as fish meal has been abandoned as a feed for ruminants [110], in today's ruminant feeding practice this is irrelevant.

During bovine⁸ and ovine⁹ pregnancy, organically bound Hg passes the placenta more readily than does inorganic Hg. Within the fetus Hg is primarily stored in the liver [93;95;147]. After birth, highest Hg concentrations are found in the kidney [147]. In an experiment using one ram fed radioactive Hg¹⁰, specific activities ($\mu\text{Ci}/\text{kg}$) were highest in the kidneys (25.6)., The liver (4.10) and muscle tissue (0.17) Hg concentrations were much lower. However, related to mass the majority of body Hg was stored in the muscle tissue [74]. Similar data could be obtained from an experiment with Holstein calves receiving an oral dose of ²⁰³Hg¹¹ [9]. However, in an experiment with one goat and one cow (both lactating) loaded with ²⁰³Hg¹² [128], liver ²⁰³Hg concentrations were highest. The reason for the difference with the ram experiment is unclear. Selected data of the latter two experiments are given in Table 1. In Jersey cows fed CH₃HgCl for 14 days, 72% and 7% of total body Hg was found in muscle and liver tissue, respectively [95].

⁷ Lead concentrations of 220 ppm (DM) in pasture grass from contamination with Pb-based paint have been reported [139].

⁸ From CH₃²⁰³HgCl.

⁹ Released from silver amalgam tooth fillings in the dam.

¹⁰ 260 μg Hg from a ²⁰³Hg(NO₃)₂ solution administered through 12 days; after a 3 days recovery period the animal was killed.

¹¹ From either CH₃HgCl or HgCl₂.

¹² From either CH₃HgCl or HgCl₂.

Table 18.1 Selected data on tissue ²⁰³Hg distribution after single oral dosing of ruminants

Ref.	Category	n	t	Hg source	Tissue (% of dose/kg fresh tissue)						
					Kidney	Liver	Lung	Spleen	Heart	Muscle	Skin
[128] ^a	Goat	1	13	CH ₃ HgCl	1.24	6.57	0.60	?	0.32	?	4.89 ^b
	Cow	1	7		1.24	6.35	0.68	?	0.62	?	1.65 ^b
[9]	Calf	3	7	HgCl ₂	0.55	0.15	0.03	0.03	0.0037	0.0029-0.0036	0.0054
	Calf	3		CH ₃ HgCl	4.41	2.25	0.99	1.02	1.23	1.83-2.17	0.35

n = number of animals/group; t = time after dosing (days); ^a intraruminal dosing; ^b skin and hair.

In Dutch cattle slaughtered between 1970 and 1980, kidney and liver Hg concentrations were 0.011 and <0.01 ppm, respectively [143]. Selected data from literature surveys on Hg concentrations in slaughter animals are given in Table 18.3.

Mercury can be absorbed from the gastrointestinal tract, the lungs and the skin [93]. Data on the exact site of Hg absorption from the intestine in ruminants are lacking, but as the rumen wall contains relatively high Hg concentrations compared with the more distal parts of the intestine after oral Hg dosing [9](Table 18.2)¹³, the rumen may be the main site of gastrointestinal Hg absorption. However, as no more data are available, this assumption cannot be substantiated. After absorption ionic Hg (Hg²⁺) is bound to plasma proteins¹⁴, whereas methyl-Hg is mainly transported in the erythrocytes. They can contain up to 20 times more methyl-Hg than plasma [9;93;142]. Metallic Hg (from Hg vapor) not oxidized in the bloodstream can rapidly cross the blood-brain barrier and cause damage to the central nervous system [93].

Table 18.2 Selected data on gastrointestinal Hg concentrations after oral ²⁰³Hg dosing [9].

Tissue	HgCl ₂	CH ₃ HgCl
	(% of dose/kg of fresh tissue)	
Rumen wall	0.57	0.80
Abomasum	0.009-0.024	0.50-0.52
Small intestine	0.010-0.043	0.48-0.54

2.4 Arsenic

Several rodenticides, insecticides, defoliant, fungicides and herbicides contain(ed) As. Moreover, in the proximity of superphosphate plants considerable As contamination (1.6-6 ppm As (DM) in herbage) can be observed [13]. The exact site of As absorption is not clear. After absorption, ± 95% of As is associated with the erythrocytes [43]. Accumulation of As in calves is not dependent on gender [88]. Arsenic is mainly recovered from kidney and liver. Selected data on tissue distribution of As in calves are given in Table 18.3. In case of longer duration of exposure¹⁵, As also deposits in hair, hoofs, bone and teeth. Arsenic crosses the placenta, but the blood brain barrier not very well [43]. For differences in toxicity between several As compounds, see 18.8.

¹³ For more experimental details, see preceding text.

¹⁴ Not specified.

¹⁵ No exact period given.

Table 18.3 Selected data on tissue distribution of Cd, Pb, Hg and As in slaughtered ruminants¹⁶

Ref.	Category	Age (mo)	n	Element	Tissue concentration (µg/kg fresh weight)			µg/L Blood	
					Liver	Kidney	Muscle		
[149]	Calves		20	Cd	6-184	24-24-1440	2-53		
				Pb	20-410	40-930	<10-70		
[94]		4	4	Pb ^c	297	315			
[36]		7	4	Pb ^d	600	800-1200			
[88]		6-12	312	Cd	25.6-25.8	112-137	1.2-1.4	0.4	
				Pb	24.3-26.9	22.2-23.8	8.0-8.4	5.3-5.6	
				As	12.6-15.7	13.8-15.9	3.8-3.6	3.2-3.7	
[68]		Cows		198	Cd	20-65	70-200	1-22	
				198	Pb	192-240	179-190	20-35	
				160	Hg	12	33	5	
			104	As	20-40	15-17	4-12		
			1302	Cd	45-330	188-1872	2-29		
			1449	Pb	5-1480	70-2690	4-92		
			580	Hg	11-31	10-200	2-20		
			544	As	9-37	25-357	1-280		
[143]					Cd	140	550		
					Pb	290	460		
				Hg	< 10	11			
				As	11	47			
[39]		24-36	3	Pb ^e	100	100			
[149]			23	Cd	31-305	121-1200	1-28		
				Pb	30-440	120-810	<10-100		
[34]			42	Pb	100-2000	200-1900			
[120]				Cd	235	1071			
				Pb	1247	2835			
[82]	Bulls	13.5	6?	Pb	290	210			
[152] ^a			8	Cd	80	240	7		
[144]	Lambs	5,5	8	Cd	22	34	3		
				Pb	1222	498	23		
				Hg	14	178	1		
				As	2	5	2		
[49]	Sheep		4	Pb	312	240	48		
[138]		30		Cd ^b	< 120	< 90	<145		
		48			< 120	90	< 145		
		88			161	324	-		
		114			218	594	-		
		120			336	522	377		
		136			209	378	-		
[68]			286	Cd	11-858	16-1654	2-63		
		279	Pb	389-1410	185-314	28-75			
		194	Hg	15-45	42-58	2-5			
		296	As	10-500	15-48	10-12			
[58]	Goats	10-14	6	Cd	230	250			
				Pb	850	3350			
[10]					34	Cd	190-530	1300-3510	
				Pb	540-650	560-680			

n = number of animals; mo = months; ^a the ration contained 0.2 ppm Cd (DM); ^b calculated from values in DM; ^c fed 2 ppm Pb for 28 days; ^d fed 1 ppm Pb for 100 days; ^e fed 0.25 ppm Pb for 118 days; assumed DM contents: liver 24%; kidney 18%; muscle 29% [136]

¹⁶ Duration of exposure is not included in the Table because insufficient data were available.

For Cd and Pb, current EU legislation [45] sets maximum allowable concentrations in bovine and ovine tissues designated for human consumption. Values are given in Table 18.4.

Table 18.4 Maximum allowable concentrations of heavy metals in bovine and ovine tissues designated for human consumption according to EU legislation [45]¹⁷

Element	Liver	Kidney	Meat
	µg/kg fresh weight		
Cd	500	1000	50
Pb	100	100	100

¹⁷ For Hg and As, no EU limits for ruminant tissues are given.

3 ABSORPTION AND METABOLISM

3.1 General

Oral Cd absorption in goats is very low: 14 days after a single dose, only 0.3-0.4% of dose could be recovered from the carcass [83]. In cattle, absorption¹⁸ is stated to be < 0.5% [156]. In contrast, in sheep apparent Cd¹⁹ absorption was 5% at a dietary Cd level of 60 ppm [38], 5% (intake 101 mg/day) or 11% (intake 0.18 mg/day) [126], whereas apparent absorption from a milk diet was assessed to be very high (> 90%) [121]. Possibly these differences are caused by the very small amounts under study, small analytical fluctuations causing relatively large miscalculations [15]. Based on slaughter experiments with sheep, daily Cd accumulations were assessed to be 1 (intake 200 µg) and 3 µg (intake 800 µg), respectively [56]. Oral Pb absorption²⁰ in sheep is reported to be 1.3 % [15]. In Jersey cows fed CH₃HgCl for 14 days, apparent Hg absorption was 59% of dose [95]. Exact data on As absorption in ruminants are not available.

Differences in metabolism due to different sources

3.1.1 3.2.1 General

Exact data on differences in Cd and Pb absorption from different sources in ruminants are virtually lacking.

3.1.2 3.2.2 Lead

Lead phosphate, oxide, carbonate and chloride are reported to be better absorbed than Pb sulfide and metallic Pb [15].

3.1.3 3.2.3 Arsenic

Soluble arsenic compounds, such as arsenites, are readily absorbed from the gastrointestinal tract, whereas pentavalent inorganic As compounds and phenylarsonic compounds are poorly absorbed and largely excreted in the feces [43].

3.1.4 Mercury

3.2.4.1 *Cattle and sheep*

No data are available on differences in Hg absorption from different Hg sources in cattle or sheep.

3.1.4.2 *Goats*

In goats fed Hg from two different compounds during 9 days, apparent Hg absorption was 80% (CH₃HgCl) or 30% of dose HgCl₂ [128].

3.1.5

3.1.6 3.2.5 Conclusion

The scarce data virtually preclude any conclusion as to differences in availability between Cd, Pb, Hg or As sources. However, for goats absorption of Hg from an organic form as CH₃HgCl is much higher than from an inorganic form as HgCl₂. Unless proven otherwise, this is assumed to be also the case for cattle and sheep.

¹⁸ Not specified (true or apparent).

¹⁹ From CdCl₂.

²⁰ Not specified (true or apparent).

Interactions influencing absorption

3.1.7 3.3.1 Interactions of lead, cadmium, mercury and selenium

3.1.7.1 *General*

Selenium has been demonstrated to protect rats, quail and cats against toxic effects of Hg [29]. However, information on similar effects in ruminants is hardly available.

3.1.7.2 *Cattle*

No data are available on interactions of Cd, Pb, Hg and Se in cattle.

3.1.7.3 *Sheep*

3.1.7.3.1 Cadmium

In sheep receiving either 0, 9 or 18 ppm Cd²¹ (DM), the application of 4 heavy intraruminal Se pellets slightly reduced Cd contents of liver, kidney and muscle [72].

3.1.7.3.2 Lead

In sheep, feeding 3 mg Se/animal/day and/or 9.8 mg Pb/kg BW/day²² resulted in increased toxicity of Pb. In the Se + Pb group, all 5 animals died before the end of the experiment (104 days experimental period), whereas 3 of 5 animals died in the Pb group [79].

3.1.7.3.3 Mercury

In an *in vitro* experiment, motility and oxygen uptake of ram spermatozoa were decreased by the addition of Hg²³ to the medium. This effect was counteracted by Se at relatively low Hg concentrations (10^{-5} - 10^{-6} M), but not at high Hg concentrations (10^{-4} - 10^{-2} M) [4].

3.1.7.4 *Goats*

3.1.7.4.1 Cadmium and lead

No data are available on interactions of Cd, Pb and Se in goats.

3.1.7.4.2 Mercury

In goats fed ²⁰³Hg²⁴, no effect of Se on partitioning of ²⁰³Hg excretion via feces or urine could be demonstrated, although bile ²⁰³Hg concentration was significantly lower in Se-treated goats [129].

3.1.7.5 *Conclusion*

Due to lack of data, the possible effect of Se on Hg metabolism in ruminants cannot be judged.

Feeding rations of high Se content to sheep may be contraindicated when Pb intake is high. However, although the Se dose administered (3000 µg/day or 48 µg/kg BW) remains well below toxicity limits (\pm 150 µg/kg BW), it extremely exceeds normal Se requirements (150 µg/day) (Documentation report nr. 46). Therefore, the resented results are not applicable to normal practice. Unless proven otherwise, this is assumed to be also the case for cattle and goats.

²¹ From CdCl₂·2½ H₂O.

²² Selenium from Na₂SeO₃·5H₂O; Pb from PbCO₃. Selenium dose corresponds with 48 µg Se/kg BW.

²³ From HgCl₂; Se from either Na selenite, selenomethionine or selenocystine.

²⁴ 40 µCi ²⁰³Hg/kg BW from CH₃HgCl for 5 days; Se from Na selenite was given at 0.21 mg/kg BW.

3.1.8 3.3.1 Interactions of cadmium and sulfur and of lead and sulfur

3.1.8.1 *Cattle and goats*

No data are available on interactions of Cd and S or of Pb and S in cattle or goats.

3.1.8.2 *Sheep*

3.1.8.2.1 Cadmium and sulfur

Increasing the S content of the diet from 1.9 to 5.9 g/kg DM reduced Cd accumulation in the liver and kidney of sheep by 60% [133]²⁵. Data on the nature of the dietary and supplemented S are not given.

3.1.8.2.2 Lead and sulfur

Lambs given diets containing either 0.7, 2.3 or 3.8 g S/kg of diet and 200 ppm Pb²⁶ survived for 6, 15 and 30 weeks respectively [112]. In a similar experiment using the same diet with 400 ppm added Pb and either 0.7 or 3.7 g S/kg for 6 months, lambs receiving the low S diet had significantly higher kidney (425 vs. 37 ppm) and muscle (1.1 vs. 0.5 ppm) Pb levels [92].

3.1.8.3 *Conclusion*

Although in sheep increasing the S content of the ration diminishes Cd and Pb tissue load, increasing the S content of the ration is usually contradicted because of the negative effects on Cu absorption.

3.1.9 Interactions of lead and cadmium

3.3.3.1 *Cattle and sheep*

No exact data are available on interactions of Pb and Cd in cattle or sheep.

3.1.9.2 *Goats*

In goats experimentally poisoned with Pb²⁷ (28 mg/kg BW) and/or Cd (6 mg/kg BW) for 42 days a mutual antagonistic effect of these elements on the liver and kidney concentrations of their counterparts was observed [58]. Selected data are given in Table 18.9.

3.1.9.3 *Conclusion*

In many cases, Cd and Pb loading of ruminants will occur together. However, as to what extent this ameliorates separate toxicity symptoms of Cd and Pb is insufficiently clear. Moreover, loading of ruminants with one of these highly toxic elements in order to decrease accumulation of the other one is practically irrelevant.

3.1.10 Interactions of cadmium and zinc

3.1.10.1 *Cattle*

In calves fed either 40 or 160 ppm Cd²⁸ from 9 to 20 weeks of age, the addition of 100 ppm Zn to the diet slightly improved feed intake and growth when compared with the non Zn-

²⁵ As cited in reference [142].

²⁶ Oat husks/starch/sucrose/skim milk basal diet containing 0.7 g S/kg; additional S from Na₂SO₄; Pb from Pb acetate.

²⁷ Lead from Pb acetate; Cd from CdCl₂.

²⁸ From CdCl₂; Zn from ZnO.

supplemented groups [109]. Feeding 600 ppm Zn significantly reduced liver and kidney cortex Cd concentrations in calves [71].

3.1.10.2 *Small ruminants*

No data are available on interactions of Cd and Zn in small ruminants.

3.1.10.3 *Conclusion*

As several forms of Cd pollution (sludge, mining activities) also increase the Zn content of the forage, this may ameliorate Cd toxicity symptoms [142]. However, the data presented here are too scarce to recommend addition of Zn in case of Cd toxicity. A dose of 100 ppm resulted only in slight improvements, whereas a dose of 600 ppm Zn has been proven to be detrimental to ruminant health (Documentation Report nr. 44). As no values in between have been investigated, there is no convincing evidence as to the use of Zn additions as a safe measure to combat Cd toxicity.

3.1.11 Interactions of lead and phosphorus

3.1.11.1 *Cattle and goats*

No data are available on interactions of Pb and P in cattle or goats.

3.1.11.2 *Sheep*

In lambs receiving diets containing either 3.3 or 12.6 g P/kg no influence on Pb content of livers and kidneys could be demonstrated [91].

3.1.11.3 *Conclusion*

As yet, there is no evidence as to any effect of P on Pb metabolism in ruminants.

Metabolism

3.1.12 3.4.1 Cadmium and lead

No data are available on metabolism of Cd or Pb compounds in ruminants.

3.1.13 Mercury

Rumen flora of sheep demethylates methyl-Hg compounds, thereby volatilizing metallic Hg [67].

3.1.14 Arsenic

Pentavalent As compounds are partly reduced in the rumen to trivalent As compounds. After ingestion of inorganic compounds²⁹ methylated As compounds (dimethylarsenic acid) are excreted in the urine in large quantities [43;70].

Recycling

3.1.15 Cadmium, lead, mercury and arsenic

In sheep fed 60 ppm Cd³⁰ (intake 101 mg Cd/sheep/day) for 163 days bile Cd excretion of sheep was negligible (355 µg/sheep/day) [38]. Other sheep at the same dietary Cd

²⁹ Sodium arsenate and potassium arsenite.

³⁰ From CdCl₂.

concentration had bile Cd concentrations of 0.37 µg/mL [126]. In sheep, daily fecal endogenous Cd loss was assessed to be ± 1 µg/day [56].

No data are available on recycling of Pb, Hg or As in ruminants [142].

Excretion

3.1.16 Cadmium

Bovine milk Cd concentrations have been reported to be (µg/kg) < 0.2 [81], < 10 [73] or 28-38³¹ [135]. When cattle were fed 3 g Cd³² daily for 2 weeks, less than 0.02% of dose was excreted into the milk [85]. Sheep milk Cd concentrations were (µg/kg) ± 0.4 [81] or 23.4-33.5 [32], whereas goat milk Cd concentrations were (µg/kg) <10 [73] or 14.6-18.1 [32]. Near a Pb/Zn smelter³³ mean milk Cd concentration of cattle showing signs of Pb toxicity (see 18.8) was 54 µg/L [41]. When sheep were fed 2 mg Cd/kg BW/day for 70 days mean milk Cd concentration was 3.3 µg/kg [81]. When goats were fed 75 ppm Cd³⁴ for up to 19 months mean milk Cd concentration was 690 µg/kg [7].

Urinary Cd concentrations of cattle from both Cd-contaminated areas (0.08 µg/L) and non-contaminated areas (0.14 µg/L) were very low when compared with fecal Cd concentrations (2.35 and 0.88 ppm (DM)) [66]. In cows fed either 0.25, 1 or 5 ppm Cd (DM)³⁵ for > 1 year urinary Cd concentrations were 0.14-0.16 µg/L [135]. Similarly, in sheep fed 60 ppm Cd³¹ (intake 101 mg Cd/sheep/day) for 163 days urinary Cd excretion of sheep was negligible (36 µg/sheep/day) [38] or less than 0.5% of intake [56]. In goats, 0.15% of an oral dose of ¹¹⁵Cd was excreted via the urine within 96 hours of dosing [126].

During the first 4 days after a single oral Cd dosage of ¹⁰⁹Cd 80-90% of dose is excreted via the feces [15;126]. After this period, excretion declines sharply [83;84]. In fact, Cd is then hardly excreted from the body any more [156].

3.1.17 Lead

Bovine milk Pb concentrations have been reported to be (µg/kg) 0.7-4.9 [100] or 2-79 [15]. Sheep milk Pb concentrations were (µg/kg) 2.6-6.2 [100] or 10.8-19.9 [32], whereas goat milk Pb concentrations were (µg/kg) 4.5 [100] or 5.5-5.6 [32]. In general, values are higher in industrial, mining and urban areas and can occasionally attain very high values. For instance, in the proximity of a Pb/Zn smelter mean milk Pb concentration of 5 cows showing signs of Pb toxicity (see 18.8) was 750 µg/kg [41]. Moreover, 770 µg/kg in colostrum and 320 µg/kg in mature milk of cattle grazing pasture in the vicinity of a smelter were reported [146]. Near a Pb ore concentrating plant, cow's milk contained 50-150 µg/kg [21]. Milk from rural and urban Indian cattle contained 20-580 and 30-770 µg/kg, respectively [140;42]. According to EU legislation, milk suitable for human consumption should contain < 20 µg Pb/kg [45].

Calves fed diets containing 1 to 100 ppm added Pb³⁶ excreted 92-97% via the feces, whereas ± 2% was excreted via the urine [36]. Sheep fed rations containing either 53 or 1003 ppm Pb³⁷ for 7 weeks excreted 99 and 95% of ingested Pb via the feces and 0.4 and 0.06% via the urine for the 53 and 1003 ppm group, respectively [49]. Similarly, in sheep fed diets containing either 3 or 1003 ppm Pb fecal Pb excretion was 89-94% (3 ppm) and 85-87% (1003 ppm). Urinary Pb excretions were usually <1% of intake [24;105].

³¹ Milk Cd concentrations were not significantly influenced by the feeding of 5 ppm added Cd (DM) from CdCl₂ for > 1 year before calving.

³² From CdCl₂.

³³ Also producing Cd.

³⁴ Source not given.

³⁵ From CdCl₂.

³⁶ Wheat straw/lucerne/cracked maize/soybean meal diets; added Pb from newsprint or Pb chromate

³⁷ Added Pb from Pb acetate.3H₂O

3.1.18 Mercury

The vast majority of Hg excretion occurs via the feces, whereas only 0.25% of an oral dose of inorganic Hg was excreted via the urine [74]. In Jersey cows fed CH_3HgCl for 14 days, urinary Hg excretion was 1.1% of dose, whereas 0.2% of dose was secreted into milk [95]. In lactating goats given $^{203}\text{HgCl}_2$ ³⁸, after 9-16 days 94.7% of dose was excreted via the feces, 2.9% via the urine and 0.02% via the milk [64]. In other experiments, 0.3³⁹, 0.2⁴⁰, 1.1⁴¹ % of dose [128] or 0.6%⁴² of dose [65] was recovered from milk of goats. In some experiments, no ^{203}Hg could be detected in cows milk during 7 days after dosing [128]. Bovine milk is reported to contain (mg Hg/kg) 0.0003 (Dutch value) to 0.015 [141]. In goats, half-times of retention of Hg were 78 and 22 days for HgCl_2 and CH_3HgCl , respectively [128].

3.1.19 Arsenic

In an experiment with goats receiving a ration containing either < 35 or 350 $\mu\text{g As/kg}$, milk As concentrations were 15 and 24 $\mu\text{g/kg DM}$ ⁴³ [6].

Trivalent inorganic As compounds are excreted via the bile, whereas within 48 hours 80% of a dose of MSMA is excreted via the urine by goats [43].

³⁸ Single dose of 60-150 μCi from $^{203}\text{HgCl}_2$

³⁹ During 13 days, Hg from $\text{CH}_3^{203}\text{HgCl}$

⁴⁰ During 36 days, Hg from $^{203}\text{HgCl}_2$

⁴¹ During 36 days, Hg from CH_3HgCl

⁴² Period unknown, Hg from methyl ^{203}Hg

⁴³ Assuming a DM content of goat milk of 13% [101], in fluid milk these concentrations would be 2.0 and 3.1 $\mu\text{g/kg}$, respectively.

4 REQUIREMENTS

4.1 Cadmium, lead, mercury and arsenic

Mercury and Pb are not known to be essential to ruminants [124;142]. The dietary As requirement of goats is assessed to be < 50 µg/kg or even < 25 µg/kg. As (in Middle Europe) most feeds contain > 50 µg As/kg, deficiencies are unlikely to occur [8]. Although mild Cd deficiency symptoms have been evoked under extreme circumstances (see 18.7), requirements cannot be defined and under practical circumstances the risk of Cd deficiency seems to be irrelevant.

5 CRITERIA TO JUDGE CONTAMINANT STATUS

5.1 Potential indicators of contaminant status

5.1.1 Cadmium

Data on suitable indicators of Cd status in ruminants are very scarce. Blood Cd concentrations are not useful to assess Cd status as they are only marginally increased in cases of Cd toxicity. Diet, liver and kidney Cd concentrations are reported to be most suitable to judge Cd status. Values are given in Table 18.5 [142].

Table 18.5 Selected values for possible indicators of Cd status in cattle and sheep [142]

	Ration	Liver	Kidney
	ppm (DM)	µg/kg fresh weight	
Normal	0.1-0.2	20-50	30-100
High	0.5-5.0	100-1500	1000-5000
Toxic	>50	50000-160000	100000-250000

For goats, no specific data are available. Unless proven otherwise, the values given in Table 5 are also adopted for goats.

5.1.2 Lead

According to Puls, dietary Pb concentrations for cattle < 1 ppm can be considered normal, 5-20 ppm is high and > 100 ppm is toxic [111]. Other reports give minimum toxic dietary Pb doses for adult cattle of 250 ppm [145] or 2000 ppm (DM) [142]. In alive animals, blood Pb concentrations might be used to diagnose acute Pb poisoning [142]. Blood Pb concentrations < 1.0-1.2 µM are considered normal, whereas values > 1.7-2.0 µM indicate for toxicity [2;41;111;142]. Levels > 4.8 µM are often associated with increased mortality [41]. At constant Pb intake (4.1 mg Pb/kg BW⁴⁴), whole blood Pb concentrations in calves attain a plateau within 3-4 weeks (Figure 18.1) [103]. Similar data were recorded in an experiment with young bulls [82].

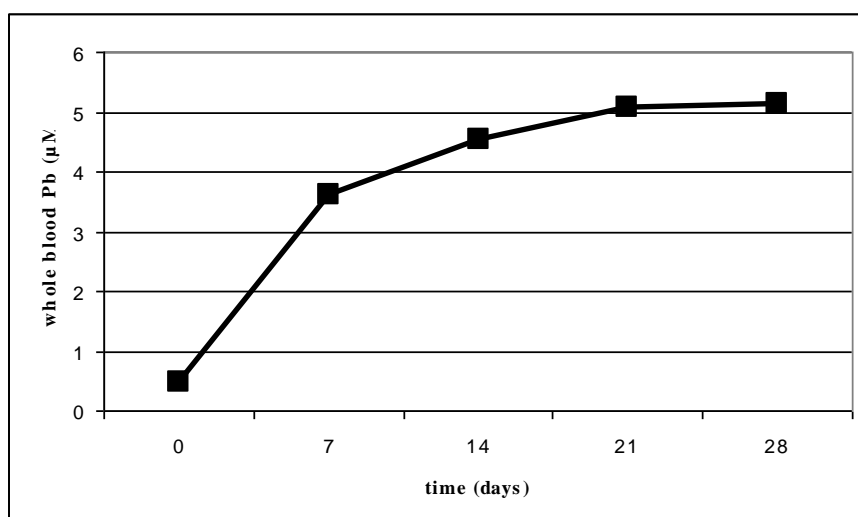


Figure 18.1 Time course of whole blood Pb concentrations in calves at constant dietary Pb intake [103]

However, blood Pb levels can be > 4.8 µM without clinical signs of toxicity [41;60]. Acute Pb toxicity symptoms are usually evoked by some kind of stress (birth, turning out to pasture). Then, Pb accumulated during the previous period is suddenly released, causing clinical symptoms [60]. In this case, blood Pb levels may be insufficiently informative. Analysis of delta-aminolevulinic acid dehydratase (ALA-D) activity in erythrocytes is recommended [60],

⁴⁴ From Pb acetate.

but storage is critical⁴⁵, analysis is difficult and in calves activity shows age-related changes (2-3 fold increase from 1 to 9 weeks of age and then a return to 1-week values by 9 months) [26;107]. This is important because many of the intoxications occur in young calves. Activities are expressed in nmol of porphobilinogen/mL of erythrocytes/hr. Calves < 1 year with values < 200 nmol/mL should be suspected of Pb poisoning, whereas values < 50-100 are indicative of Pb poisoning in all age classes [26;111]. ALA-D values decrease strongly within 1 week and even within 24 hours after the start of Pb dosing (at dietary concentrations >0.3 mg Pb/kg BW⁴⁶) and remain low (at ± 20% of pre-Pb-dosing values) until almost all Pb is removed from the body [47;111;119;160]. The extent of the decline in ALA-D activities is not dose-dependent [77]. For a survey of the biochemical interrelationship of ALA-D and Pb, see reference [46].

Moreover, in dead animals both liver⁴⁷ and kidney should be sampled as one of them may not contain diagnostic Pb levels. Concentrations > 10 ppm Pb are considered diagnostic of Pb toxicity. In chronic Pb toxicity, hair Pb levels are useful, values > 10 ppm (DM) being indicative [111].

5.1.3 Mercury

Calves and heifers that had died from Hg toxicity had liver and kidney Hg concentrations of 2.4-11.7 and 4.6-91 ppm, respectively [25;57;132].

Proposals for maximum allowable concentrations for Hg in kidney, liver and muscle tissue are given in Table 18.6. Blood, urine, fecal and milk Hg concentrations are not sufficiently reliable indicators of Hg toxicity [111].

Table 6 Proposed maximum allowable concentrations for Hg in kidney, liver and muscle tissue

Reference	Qualification	Ration	Kidney	Liver	Muscle
		ppm DM	ppm		
[111]	Normal	<0.10	<0.09	<0.06	
	High	1.0-5.0	14-146	2.0-40	
	Toxic	>4.0	>43		
[68]	Normal		<0.05		
[25]	Toxic		>0.5	>0.5	>0.5
[151]	Toxic		>0.05	>0.05	>0.05

No information as to underlying calculations is given. Mainly dietary Hg concentrations should be used with caution, as organic Hg compounds are far more toxic than inorganic ones [111].

Evidence as to choose a reliable indicator of Hg toxicosis is scarce. As taking liver biopsies from living ruminants is relatively easy, liver Hg concentrations seem to be the best indicator of Hg status in alive ruminants. Blood, urine, fecal and milk Hg levels should not be used. Post mortem, the kidney is the tissue of choice. One should not rely solely on laboratory data, but also take into account the clinical picture, as tissue Hg levels depend on the causative Hg compound [111]. Due to the nature of Hg poisoning, in several cases (e.g. soil licking) dietary levels are often difficult to calculate. Moreover, toxicity of the different Hg compounds differs considerably, whereas the occurrence of Hg poisoning also depends on the duration of exposure. Therefore, dietary Hg levels are of limited value to judge Hg status of animals.

5.1.4 Arsenic

Arsenic poisoning can be confirmed by liver and kidney As concentrations > 3 ppm [1].

⁴⁵ In a refrigerator or in wet ice samples can be stored 3-6 days; samples cannot be stored in a normal freezer, but only at -196 °C after rapid freezing in liquid N₂ [26].

⁴⁶ In one experiment (calves on a milk substitute diet) ALA-D did not react to 1.5 mg Pb carbonate/kg BW three times weekly [76].

⁴⁷ Even in formalin fixed tissues [27].

6 DEFICIENCY

6.1 General

6.1.1 Cadmium

When goats were Cd depleted during 10 years on semi-synthetic rations⁴⁸ (< 15 µg/kg of ration), mild deficiency symptoms such as lower milk yield, fat and protein concentrations as well as poor fertility were observed when compared with a similar ration containing 300 µg Cd/kg [6]. Selected data are given in Table 18.7.

Table 18.7 Selected on Cd depletion in goats [6].

		Cd concentration (µg Cd/kg) ^a	
		< 15	300
Milk	Yield (mL/day)	730	1010
	Fat (g/day)	23	35
	Protein (g/day)	22	28
Reproduction	Success of first insemination (%)	46	73
	Abortion rate (%)	12	0
	Dead kids at birth (%)	43	8

^a Extra Cd from CdCl₂ · H₂O

6.1.2 Lead and mercury

As neither Pb nor Hg has been proven to be essential to ruminants, discussing the possibility of the occurrence of a deficiency is irrelevant.

6.1.3 Arsenic

Although As compounds are mainly known as toxic agents, As is also reported to be essential in very small amounts. As deficiency could occur at dietary concentrations < 50 µg/kg, whereas 350-500 µg/kg should suffice for normal growth, reproduction and milk (fat) yield. In experiments with 2 groups of pregnant and lactating goats and their kids receiving rations⁴⁹ containing either < 35 or 350 µg As/kg, the animals receiving 350 µg As/kg performed better than those in the group with < 35 µg As/kg of diet [6;8]. Selected data are given in Table 18.8.

Table 8 Selected data on the influence of As on growth, reproductive performance and milk yield of goats.

Ref.			As-content of ration (µg As/kg) ^a	
			< 10	350
[8]	Birth weight	Kg	2.8	3.1
			Growth	BW on 91st day of life (kg)
	Reproduction	Success of 1st insemination (%)	47	69
			Abortion rate (%)	33
	Milk	Milk yield (mL/day)	800-1400	1200-1600
		Milk fat (g/day)	31-53	45-71
	Life expectancy	Deaths, kids (%)	32	3
		Deaths, dams (%)	40	25
			< 35	350
[6]	Growth	BW on 91st day of life (kg)	16.2	18.6
	Reproduction	Success of 1st insemination (%)	57	75
		Abortion rate (%)	15	0.8
	Milk	milk yield (mL/day)	806	1010
		milk fat (g/day)	32	35
		milk protein (g/day)	25	28

^a Extra As from As₂O₃

⁴⁸ Potato starch, sugar, casein, urea and sunflower oil.

⁴⁹ Potato starch/sugar/casein/urea/sunflower oil ration [6].

7 TOXICITY

7.1 General

A survey on the clinical and pathological symptoms of Cd, Pb, Hg and As intoxications is given in reference [15]. Currently, heavy metal contamination does not pose a great risk to human health in the Netherlands [124].

7.2 Cadmium

It is not clear as to what extent toxic doses for ruminants depend on the chemical form of Cd. For Cd from CdCl₂, Cd succinate and Cd propionate 50-300 ppm (DM) is reported to cause poor growth, BW loss, poor milk yield, anemia, sterility, abortions and dead calves at birth [68;69]. When 40 or 160 ppm Cd from CdCl₂ was fed to calves from 9-20 weeks of age, only few clinical symptoms occurred [109]. However, when either 640 or 2560 ppm Cd were fed, the health status of the animals deteriorated rapidly. In the 2560 ppm Cd group, all animals died within 2 to 8 weeks. Clinical symptoms were unthriftiness, rough coat, mouth lesions, dry and scaly skin, sore and enlarged joints and impaired vision. These symptoms resemble those of Zn deficiency and may be related to the mutual antagonism between these two elements [142]. Selected data on tissue Cd concentrations on the 640 and 2560 ppm Cd groups are given in Table 18.7. For Cd propionate, 640 mg Cd/kg DM caused serious intoxication in adult cattle [68]. Disturbance of kidney function occurs when kidney Cd concentrations exceed \pm 300 ppm (DM)⁵⁰ [156].

As Cd accumulates in tissues, public health considerations should be taken into account. In an experiment with sheep, 0.6 ppm (DM) of the total ration was stated not to be exceeded in order to prevent Cd accumulation to levels hazardous to public health. At Cd levels between 0.6 and 30 ppm (DM) in rations for ruminants their livers and kidneys should not be used as human food [117]. According to the NRC, dietary concentrations for cattle and sheep should not exceed 0.5 ppm [97]. Maximum allowable Cd and Pb concentrations in liver and kidney according to EU legislation are given in Table 18.4. As the relation between dietary and tissue Cd concentrations is complex, the lower limit mentioned (0.6 ppm (DM)) can be adopted.

7.3 Lead

Lead poisoning may be either acute or chronic. In acute cases, clinical signs usually start within 2-3 days after consumption of a fatal dose and include head pressing, circling, blindness, convulsions, staggering, muscle twitching, teeth grinding, salivation, constipation or diarrhoea and oligouria. In chronic cases, some kind of stress (birth, turning out to pasture) may suddenly evoke a seizure and death. Before, anorexia, BW loss, tympany, obstipation, lower milk yield, abortion and anemia can be observed [2;15;28;35;41;107;113;116]. In lambs, chronic Pb intoxication has been demonstrated to be associated with slowed learning [28]. Post mortem, osteoporosis and hyperemia and bleedings of the kidney cortex, as well as hydronephrosis can be observed [15;31].

In adult cattle, 5-15 mg Pb/kg BW⁵¹ or 1000 ppm Pb (DM) caused poor growth and performance; 6-7 mg Pb/kg BW caused toxicity signs⁵². In calves, ingestion of a single bolus of 200-400 mg Pb/kg BW and, for adult cattle, 600-800 mg Pb/kg BW is lethal [15;159]. Moreover, 3-25 mg Pb/kg BW⁵³ caused Pb toxicity, whereas 2.7 mg Pb/kg BW resulted in death of calves on milk diets within 20 days [68]. In calves consuming diets⁵⁴ containing either 1 or 102 ppm Pb for 100 days no differences in performance between the groups could

⁵⁰ Assuming a DM content of bovine kidney of 21% [16], this corresponds with 63000 μ g Cd/kg fresh weight.

⁵¹ From Pb acetate.

⁵² Not specified.

⁵³ From either Pb acetate, Pb oxide or Pb carbonate.

⁵⁴ Wheat straw/lucerne/cracked maize/soybean meal.

be observed [36]. Calves receiving milk substitute diets are more sensitive to Pb⁵⁵ than similar calves receiving a grain/hay diet [160]. Dietary Pb levels > 15 ppm (DM) already cause detectable alterations in ALA-D activity [15]. Added dietary levels up to 1000 ppm Pb⁵⁶ fed during 84 days only slightly reduced performance in sheep [49]. In Pb-poisoned cattle and goats high liver and kidney Pb have been reported [5;23;34;58]. Selected data are given in Table 18.9.

Table 18.9 Selected data from an experiment with goats receiving extra Pb and/or Cd

Ref.	Category	Pb	Cd	liver	kidney	liver	kidney
				Pb		Cd	
added (mg/kg BW)				µg/kg fresh weight			
[23]	Cattle			17.300	88.600		
[34]				1.000-165.000	16.000-526.000		
[5]	Calves	220-1.100 ^a		9.300-12.6000	50.500-297.000		
[109]			640 ppm			507.000-3.789.000	2.281.000-4.929.000
			2560 ppm			430.000-3.178.000	695.000-3.419.000
[58]	Goats ⁵⁷	28	0	12.870	84.240	410	370
		0	6	1.040	4.190	5.470	13.480
		28	6	7.080	20.950	4.720	5.840

^a From various sources

Cattle grazing grass containing 163-212 ppm Pb had blood Pb concentrations of 1690-1932 µM [41].

A maximum dietary value of 30 ppm Pb has been proposed for cattle and sheep rations [97]. It is not clear if this is sufficient to prevent Pb accumulation in liver and kidney beyond maximum allowable EU levels (Table 18.4). However, this limit may suffice to prevent Pb toxicity in cattle. Recalculation to DM values is questionable.

7.4 Mercury

Acute Hg toxicity has been evoked by the accidental consumption of Hg-containing fungicides, as HgCl₂ (calomel), HgCl, HgO, HgI, phenyl mercuric acetate, methyl mercury dicyandiamide⁵⁸ and CH₃HgCl [25;132;142;157]. Within plants, the leaves contain higher Hg concentrations than do stems [130].

Clinical symptoms of Hg toxicity are salivation, lacrimation, excessive thirst, depression, diarrhoea, alopecia, eczema, incoordination, recumbency and refusal to drink. Eventually the animals can die from Hg toxicosis [57;68;132;137]. However, sudden death may be the predominant symptom [25]. Post mortem, tubular degeneration and glomerulonephritis of the kidneys, edema and bleedings can be observed [51;57;132].

Toxic doses depend upon the compound consumed. Some examples are given in Table 18.10.

⁵⁵ Equal doses of 5 mg Pb/kg BW from Pb acetate . 3H₂O for 7 days for both groups.

⁵⁶ From Pb acetate . 3H₂O.

⁵⁷ Lead (28 mg/kg BW) from Pb acetate; Cd (6 mg/kg BW) from CdCl₂. Experimental poisoning during 42 days.

⁵⁸ For this particular compound, it is not clear as to what extent toxicity originates either from the Hg or from the other parts of the molecule.

Table 18.10 Examples of toxic doses of different Hg compounds for cattle and goats. For ease of comparison, values are calculated for an adult, lactating cow of 650 kg BW and consuming 22 kg DM. Body weight and DMI of goats was assumed to be 70 kg and 3.5 kg, respectively. **Bold = given values; italic = calculated values.**

Ref.	D ^a	Category	Compound	Daily dose (mg)	mg/kg BW	mg/kg BW ^{0.75}	mg/kg feed DM
[25]	± 3 w	cattle	phenyl mercuric acetate	<i>179</i>	<i>0.275</i>	<i>1.4</i>	<i>8.1</i>
[68]	?		alkyl Hg ^b	<i>0.07</i>	<i>0.0001</i>	<i>0.0005</i>	<i>0.003</i>
	?		alkyl Hg ^b	<i>312</i>	<i>0.48</i>	<i>2.4</i>	<i>14</i>
			CH ₃ -Hg-dicyandiamide	<i>146</i>	<i>0.225</i>	<i>1.1</i>	<i>6.6</i>
[102]	90 d	goat	HgCl ₂ ⁵⁹	100-150	<i>1.4-2.1</i>	<i>4.1-6.2</i>	<i>29-43</i>

^a duration of exposure; ^b not specified

The NRC suggests a maximum tolerable dietary level of 2 mg Hg/kg feed for cattle [97]. No separate values are given for goats. However, regarding the data presented in Table 18.10 this level does not seem to be safe under all circumstances.

7.5 Arsenic

Several organic and inorganic As compounds can evoke As toxicity. Among them, the pesticides arsenic trioxide, arsenic pentoxide⁶⁰, sodium and sodium and potassium arsenite, potassium arsenate, lead arsenate, calcium arsenate, monosodium and disodium methanearsonate (MSMA and DSMA, respectively) are the most hazardous for livestock. In general, arsenites are 5-10 times more toxic than arsenates [1;43]. Organic arsenicals are less toxic than inorganic ones and include aliphatic (cacodylic acid, acetarsonic acid) and aromatic (MSMA and DSMA) compounds. Most of them have been used (or are still used) as rodenticides, insecticides, defoliants, fungicides, herbicides etc.. The toxicity depends on chemical form, solubility, oxidation state, animal species involved and duration of exposure [43]. Acute As poisoning of grazing cattle has been reported after the accidental consumption of lead arsenate⁶¹ from a discarded and subsequently rusted container [80]. Kidney and liver As concentrations were 18.5-31.1 and 15.7 ppm, respectively. In the latter case, both Pb and As may have contributed to the clinical symptoms.

As As is rapidly excreted, chronic poisoning is rarely observed. Clinical symptoms of acute (inorganic and aliphatic organic) As toxicity include severe colic, profuse salivation and foetid, bloody diarrhoea, ruminal atony, dullness, high body temperature, dehydration, polyuria followed by anuria, shock and sudden death. Animals can die within 3-12 hours after ingestion. Post mortem, congestion and inflammation of rumen, abomasum and intestine can be observed. Chronic cases of As poisoning are characterized by poor condition, wasting and desquamation of the skin. Animals poisoned by arsanilic acid show ataxia, hyperesthesia and irreversible blindness [43;80;90].

Dietary levels of 50 or 100 ppm As (from inorganic and organic As compounds, respectively) are reported to be the maximum tolerable level for both cattle and sheep⁶² [97]. Without further evidence, also dietary concentrations > 5 ppm are stated to be toxic, presumably for goats [8]. For cattle, 40 mg As/kg BW should be lethal [68]. More specifically, cattle and sheep were poisoned by 200 (cattle) and 250 (sheep) mg cacodylic acid/kg BW. For cattle, 50 mg MSMA/kg BW or 150 mg DSMA/kg BW were lethal. For sheep, 300 mg MSMA/kg BW or 150 mg DSMA/kg BW were lethal. Moreover, organic arsenicals accumulate in plants [43]. As evidence is scarce, the levels of 50 and 100 ppm for inorganic and organic As compounds in cattle and sheep rations can be adopted. It is not clear why the goat should be

⁵⁹ From HgCl₂; 1-1.5 L of drinking water containing 100 mg Hg/L was consumed by each of 6 goats.

⁶⁰ At this moment (September 2003) still in use as a wood preservative (CTB, Wageningen).

⁶¹ Containing 39% Pb and 10% As [80].

⁶² For a 650-kg cow consuming 22 kg DM (45% DM in the ration), this corresponds with 3.4 or 6.8 mg/kg BW. For a 70-kg sheep consuming 3 kg DM (80% DM in the ration), this corresponds with 2.7 or 5.4 mg/kg BW.

at least 10 times as sensitive for As toxicity as cattle and sheep. However, by lack of data the limit of 5 ppm can be adopted for goats.

Direct measures in toxicity cases

7.1.1 General

Several poisonings mentioned in this chapter (Pb, As, organic Hg) may in part be very similar. As far as possible, the contaminated feed or the toxic substance should be removed and other feed given. Supportive symptomatic therapy (rehydration) is always indicated [43]. More specific therapies include the use of CaNa_2EDTA (cattle: 110 mg/kg BW intravenous) 2 doses 6 hours apart every other day for 3 treatments (Cd poisoning, acute Pb poisoning) [2;15].

In case of Hg poisoning or inorganic As poisoning, dimercaprol (BAL) can be used. The dose is 3 mg/kg BW⁶³, given as an intramuscular injection every 4 hours during 2 days. This treatment may give rise to more severe clinical signs as BAL mobilizes As from the tissues before chelating it. BAL is contraindicated in cases of severe renal or hepatic insufficiency and is itself toxic either, resulting in nephrotoxicity, hypertension, tremors, convulsions and coma. Another more advantageous possibility is the use of Na thiosulphate (30-40 mg/kg BW intravenously or 60-80 mg/kg BW orally 3 times daily during 3-4 days). BAL and Na thiosulphate can be used together. [1;3;15;43].

7.1.2 Cadmium

In case of Cd poisoning, administration of CaNa_2EDTA (see above) is indicated. Although evidence is not convincing, increasing the Zn content of the ration (> 100 ppm extra Zn) can be considered [15].

7.1.3 Lead

In acute Pb poisoning, animals should be given MgSO_4 as a laxative (cattle: 500-1000 g orally; sheep 20-50 g) to remove remaining Pb in the gastrointestinal tract. Administration of CaNa_2EDTA (see above) is indicated [2]. A combined therapy of thiamine s.c. (3-5 mg/kg BW twice daily [2] or 75 mg/kg BW) and EDTA (110 mg/kg BW i.v.) has been proven to increase biliary Pb excretion of sheep by 842% [99] and to alleviate clinical symptoms [2]. In goats, thiamine alone (20 mg/kg BW) was insufficiently useful [78].

In chronic Pb poisoning, especially when neurologic symptoms are obvious, treatment is senseless.

7.1.4 Mercury

In both inorganic and organic Hg poisoning, Na thiosulphate and BAL can be used (see above). However, in case of neurological signs caused by alkyl Hg poisoning any treatment is senseless [3]. Administration of KI and/or Na thiosulfate is ineffective [137]. Although adding Se to the diet of quail can protect these animals from Hg toxicosis, it is unclear as to what extent this also applies for ruminants (see 18.3.3.1).

7.1.5 Arsenic

In case of organic As compound poisoning, no specific antidote is available. Activated charcoal should be administered in a dose of 1-4 g/kg BW (1g charcoal/3-5 mL water). One hour later, Na_2SO_4 should be given as a laxative.

In inorganic As compound poisoning, dimercaprol or BAL can be used (see above). Other drugs as dimercaptosuccinate have not been tested in cattle, whereas D-penicillamine is too expensive for use in cattle [1;15;43].

⁶³ BAL = British Anti Lewisite; given as a 5% solution in 10% benzyl benzoate in peanut oil [43].

8 RADIONUCLIDE CONTAMINATION

8.1 Radionuclides involved

Under natural circumstances, food, feed and water are slightly radioactive. This is caused by the isotopes ^{40}K , ^{14}C and ^{87}Rb . The radioactivity exposure caused by human activities is < 1% of natural radioactivity of food, feed and water [124].

In case of atmospheric nuclear bomb explosions or nuclear power plant accidents, such as the Chernobyl accident, the situation is totally different. Radionuclides from rain can contaminate forage, and in this way enter the food chain. Among the human foods involved, contaminated ruminant milk is the most important hazard to human health. Many of the radionuclides released at such events are relatively short-lived and, therefore, do not impose an important threat to human health. The main radionuclides of interest are ^{131}I , ^{132}I , ^{134}Cs , ^{137}Cs , ^{89}Sr and ^{90}Sr . Besides these, isotopes of e.g. Pu and Am are sources of minor importance. Although ^{132}I has a very short half-life (2.2 hours), it may be present as a fission product of ^{132}Te [53;86]. Ingested radioiodine has been identified as an important cause of thyroid cancer in children after the Chernobyl accident [63].

8.2 Radionuclide distribution and excretion from the body

The effective half-life of an element is determined by both its physical (decay) and biological half-life (elimination from the body). This difference is mainly important for radionuclides with relatively long physical half-lives (^{134}Cs : 2.4 years; ^{137}Cs 30 years; ^{90}Sr : 28 years) [53].

Considering radioiodine, after uptake⁶⁴ (true absorption approaching 100% (Documentation Report nr. 43)) [63], radioactive I isotopes are rapidly transported to and accumulated in the thyroid gland (\pm 20-30% of dose) [53;124]. Approximately 35% of dose is excreted via the urine, 25% is excreted via the feces and 10-20% of dose is excreted via the milk [53]. Excretion into milk starts within 30 minutes after ingestion [53] and peaks within a few days [123]. Incorporated in thyroid hormones, they can be distributed throughout the body via the blood. However, as the half-life is relatively short (8 days), radioactivity level decreases rapidly and important contamination of edible parts of ruminant carcasses is unlikely.

Cesium absorption⁶⁵ is reported to be \pm 80% [63]. Within the body the ^{134}Cs and ^{137}Cs isotopes behave similar to K. This implies that edible parts of ruminant carcasses (meat) can be seriously contaminated. Cesium is excreted into milk much more slowly than I, excretion peaking some 3 weeks after first ingestion. In meat Cs-isotopes are \pm 3 times more concentrated than in milk. The effective half-life of radioactive Cs in cattle is \pm 100 days [52;123].

Finally, Sr absorption from the intestine heavily depends on the Ca intake and requirement. True Sr absorption is reported to vary from 12-72% [19]. The distribution of radioactive Sr isotopes resembles that of Ca. Therefore, they are mainly accumulated in the skeleton. However, besides this Sr isotopes can be excreted into milk in considerable amounts. Milk has therefore to be carefully monitored and, if necessary, withdrawn from human consumption [124].

8.3 Monitoring of radioactive contamination

In the Netherlands, a national network for radiological monitoring (LMRV) is installed at i.a. dairy industries and slaughterhouses. Under normal circumstances, it is kept on stand-by. This network continuously monitors radioactive contamination of animal-derived food (particularly milk and meat). In case of nuclear accidents, grass (and also vegetables etc.) is also investigated [86;87;123].

⁶⁴ Besides intake via feed and water, I isotopes can also be inhaled. However, this way of entry is unimportant [53].

⁶⁵ Not specified (true or apparent).

8.4 Countermeasures to combat radioactive contamination of animal derived foods

8.1.1 Grazing ban and milk radioactivity levels

After a nuclear accident, in contaminated areas a grazing ban can be issued to prevent intake of contaminated grass and/or soil. This can be achieved by stalling and feeding uncontaminated feed (“clean feeding”), or by transporting the animals to uncontaminated areas [12;63]. A grazing ban is in force when radioactivity levels from ^{131}I in/on grass exceed 5000 Bq/m^2 [86]. If radioactive contamination of animal products has occurred, banning meat and milk from the human food chain can considerably reduce radioisotope intake. Specific measures with regard to radioactive contamination of milk are taken when milk radioactivity levels exceed 500 (^{131}I), 1000 (radiocesium), 125 (radiostrontium) or 20 Bq/L (alfa-radiators as ^{239}Pu and ^{241}Am) (Table 10 in reference [86]). Besides this, uptake of radionuclides from the intestine can be diminished by specific countermeasures.

8.4.2 Element-specific countermeasures

Reviews of specific countermeasures have been published in references [55;63;96;125;148].

8.4.2.1 *Radioiodine*

As I transport into milk is an active process, the transfer of radioiodine to milk can be diminished by feeding extra stable I to lactating ruminants. Experimental data suggest at least 1 g stable I per day to be necessary to reduce the ^{131}I radioactivity in milk by a factor of 2-3. Administration of stable I before rather than after the start of ingestion of radioiodine is more effective in reducing radioiodine in milk. On the other hand, feeding severely I-deficient rations for a short time would increase radioiodine accumulation into the thyroid of the lactating animal and thus decrease its concentration in milk [63].

However, both measures cannot be applied. Addition of amounts up to 1 g stable I/day may already cause I toxicity in ruminants (Documentation report nr. 43). After stable I administration, reduction of radioiodine contamination of the milk is rather small and the total I concentrations of the milk extremely exceed maximum permissible WHO levels of 0.5 mg/kg ⁶⁶. In other words, milk will still be unsuitable for human consumption. Feeding I-deficient rations is not practical. First, current, site-specific radioiodine deposition data are needed and second, within a few days large amounts of I-deficient feeds have to be available⁶⁷ [63].

When milk is contaminated, measures to allow for decay of radioisotopes can be taken. Possible destinations of contaminated milk (in order of merit) include feeding to animals, spreading on agricultural land, anaerobic digestion, discharge to sea and dilution and/or processing (e.g. to milk powder). All of these measures are theoretically possible but the final decision depends on the radionuclides involved and commercial, social, logistical and political considerations [63;86].

8.4.2.2 *Radiocesium*

Radiocesium can be bound in the intestine and, hence, made less available for uptake by the animal. These binders include hexacyanoferrates (commonly referred to as “Prussian Blue”, “Berliner Blau” or “Berlijns Blauw”) and clay minerals (e.g. bentonite). Ammonium-ferric(III)-cyano-ferrate(II) (AFCF), although rather expensive, is very effective in binding Cs [54;55;63;114;150]. Moreover, it is not toxic to animals and man [104] and, therefore, admitted in the EU as an antidote against Cs transfer into edible tissues [44]. For use in free-

⁶⁶ A single administration of 1 g of KI (= 765 mg I) to lactating goats (DMI not given) resulted in milk total I concentrations of 20 mg/kg [18].

⁶⁷ Depending on the distance to the source of radioiodine emission and the size of the contaminated area involved.

ranging animals, salt licks and ruminal boli containing AFCF have been developed [17]. Wax-coated variants can be used when delayed onset of AFCF release is wanted in order to increase its activity at the time of slaughter. In this way, the “active” period of the boli can be extended from 4-8 weeks to 9-11 weeks. Despite the increased costs (boli, animal handling), this measure has been calculated to be 2.5 times less expensive than clean feeding. In lambs (38 kg BW), 21 mg of AFCF reduced ¹³⁴Cs transfer by 45%; 1 mg AFCF/kg BW reduced ¹³⁴Cs from feed to muscle by 60%. When manure of cows receiving AFCF was used as a fertilizer, a reduction of Cs transfer to the grass was observed [63].

The feasibility and effect of application of K-containing fertilizers⁶⁸ and clay minerals, as well as disking, ploughing and reseeded to reduce Cs uptake by plants considerably depends on soil type and mineral status. The effects are usually most obvious the second year after application and decline thereafter and these measures may, therefore, be repeated every 4-5 years [63].

Feeding 25 g/day of bentonite to reindeer has been proven to be effective and cheap, but for reasons of increased water requirements and relatively small effect (20% reduction of biological half-life) this feeding practice has been discontinued [14].

8.4.2.3 Radiostrontium

The metabolism of radiostrontium is intimately associated with Ca metabolism. Recently, a relationship for the transfer of Sr to milk has been calculated [20;62]:

$$F_{mSr} = \frac{0.11 \times [Ca]_{milk}}{I_{Ca}}$$

in which:

F_{mSr} = transfer coefficient of Sr from ration to milk (day/L)
 $[Ca]_{milk}$ = Ca content of the milk (g/L)
 I_{Ca} = daily Ca intake (g/day)

Application of this equation implies that doubling the dietary Ca intake halves the Sr transfer from ration to milk. In order to prevent negative effects of Ca supplements on the absorption of other essential nutrients, Ca addition (to dairy cattle rations) should be limited to 100-200 g Ca/day (or 1-2% Ca of DMI), which would correspond with reductions of F_m of 40-60% [63].

The addition of stable Sr to prevent Sr uptake has been proven to be useless because of the much higher dietary Ca concentrations overruling its effect. On the other hand, other potential Sr binders such as zeolite A, other clay minerals and alginates are either unpalatable, expensive and/or too unspecific and may, therefore, cause trace element deficiencies [63].

As no data are available on the effective half-life of Sr, assessment of duration of Ca application is difficult. However, regarding the physical half-life of Sr, Ca addition should be continued for years. In such cases, care should be taken to avoid trace element deficiencies. On the long term, liming of soils can reduce Sr uptake by plants. The effect is usually most outstanding in the first year after contamination [63].

8.5 Conclusions and advises

Silage harvested during periods of heavy contamination (e.g. rainfall) should be stored as remote as possible from buildings (> 20 m), in order to reduce radioactivity burden for man and animals. If this roughage is fed during the next winter season, radioisotope excretion will increase. In case of hay, inhalation of dust should be avoided as much as possible [52].

⁶⁸ In the Netherlands, for instance, K content of most soils is already very high [127]. Then, application of K-containing fertilizers is not practical.

For radioiodine, the only useful measure is stalling and clean feeding. If radioiodine is the only or the predominant radioisotope, contaminated feed can be stored to allow for decay. For radiocaesium, AFCF can be administered to exposed animals as a cost-effective measure. Besides this, agricultural measures as ploughing and reseedling can reduce radioisotope burdens.

For radiostrontium, increasing the Ca content of the diet of lactating animals is the only cost-effective measure. The level of 2% Ca of daily DMI should not be exceeded to prevent disturbances of trace element metabolism (mainly Zn [98]). Phosphorus should be increased either to maintain Ca/P ratios. Trace element status of the animals should be monitored.

Surprisingly, the specific countermeasures are not clearly mentioned in the official Dutch "Nationaal Plan voor de Kernongevallenbestrijding" and related publications [86;87]. Introduction of these countermeasures in these reports is recommended.

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