Reviews on the mineral provision in ruminants (XI):
IODINE 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 Iodine 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 prof. dr. A.Th. van 't Klooster and dr. M.C. Blok, 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
EDDI		Ethylenediamine dihydroiodide
T_3		Triiodothyronine
T_4		Thyroxine
TRH		Thyroid releasing hormone
TSH		Thyroid stimulating hormone

1 FUNCTIONS OF IODINE IN THE BODY

The only known function of iodine in the body is its essentiality as a component of thyroid hormones (thyroxine or tetraiodothyronine (T_4), triiodothyronine (T_3)). Together with other hormones (e.g. growth hormone) the thyroid hormones control the rate of oxidation and protein synthesis in all tissues. In this way, they are involved in the foetal development, growth, brain development, digestion, thermoregulation, muscle function, immune defence, reproduction and proper function and growth of the skin, hair and wool [5; 70].

2 DISTRIBUTION OF IODINE IN THE BODY AND IODINE KINETICS

Approximately 80% of the total body iodine is stored in the thyroid gland [70]. The remaining part is located mainly in soft tissues. In veal calves fed milk replacer containing 0.57 ppm (DM) I (from EDDI) for 35 days, I concentrations were (ppm fresh tissue) 361 (thyroid gland), 0.17 (liver), 0.16 (kidney), 0.11 (heart) and 0.09 (spleen), whereas the I concentration in plasma was 0.14 µg/mL [32]. The thyroid gland captures up to 90% of the iodine passing through it. The amount of I incorporated into the thyroid gland depends on the I status of the animal and is calculated to be <20 ((more than) adequate), ± 30 (marginal) or up to 65% (severe deficiency) of the dietary I consumption [61]. After uptake by the thyroid gland, iodine is combined with thyrosine to form diiodothyrosine (T_2) and two of these molecules are used to form T_4. The latter molecule is the inactive transport form of the hormone [70]. Triiodothyronine (T_3) can be produced directly in the thyroid gland or from T_4 in the peripheral tissues [6]. Within the thyroid gland, T_4 is stored in a colloidal form as thyroglobulin. A small proportion of the extrathyroidal iodine occurs as free iodine [70]. In histologically normal thyroid glands of heifers, 11-22 mg I (or 600-1500 µg/g wet thyroid weight) is stored [45].

Thyroid hormone secretion by the thyroid gland is hormonally controlled. Thyroid releasing hormone (TRH) formed by the hypothalamus induces thyroid stimulating hormone (TSH) release from the anterior pituitary. The TSH then stimulates T_4 formation from thyroglobulin and T_4 release from the thyroid gland. Finally, circulating T_4 and T_3 exert a negative feedback on the hypothalamus/pituitary system [30].

Iodine is very efficiently absorbed from the gastrointestinal tract. The main sites of absorption are the rumen (70-80% of the daily intake) and the omasum (10%) [52]. In the blood I is mainly transported loosely bound to plasma proteins [70]. Iodine is actively transported through the placenta to maintain a foetal to maternal ratio of up to 8:1 [15]. Iodine easily enters the mammary gland (irrespective of milking) and can subsequently be reabsorbed from the milk in the udder (82-94% reabsorption in both cows and goats) [52]. Milk concentrations during 70 hours following ^{131}I injection can be up to 10 times that in serum in goats [65] and up to 2.6 times that in serum in cows [38].

3 IODINE ABSORPTION AND METABOLISM

3.1 General

The (probably true) absorption of inorganic I is reported to be 80-90% or even almost 100% [26].
The I content of forages can vary considerably, depending on plant species, climate, season, soil type, application of fertilizers [70] and distance from the seaside. (Marine) clay and peat soils have higher I concentrations than sand soils. This is reflected in the milk I content of the cows grazing the pasture grown on this soils (see par. 4.1). However, no simple correlation between soil I and I content of the plants grown on it exist. In a Dutch survey, white clover contained 0.16-0.18 and grass contained 0.04-0.40 ppm I (DM). Nitrogen fertilization decreased herbage I content [25]. Iodine concentrations in plants decreased during summer, the decrease being greater in grasses than in *Leguminosae* [21]. In the same report is was demonstrated that increasing distance from the seaside was associated with lower plant I concentrations. For instance, red clover harvested at 10-50 km from the seaside contained 272 µg I/kg DM, whereas the same plant species at 401-450 km from the seaside contained 112 µg I/kg DM. The I content of the drinking water showed a similar pattern and decreased from 7.6 to 1.1 µg/L over the same distance.

3.2 Differences in iodine metabolism due to different iodine sources

Several iodine compounds have been used as I sources for ruminants. Besides simple anorganic salts (NaI, KIO_3, $NaIO_3$, CaI_2, CuI_2, diiodosalicylic acid) and elemental iodine, some organic compounds have been employed. The main representatives of this group are methyl I and ethylenediamine dihydroiodide (EDDI). The chemical formula of EDDI is $(H_2NCH_2CH_2NH_2).2HI$ [55] and this compound contains 82% I [42].

3.2.1 Cattle

Some studies have been dedicated to differences in absorption between different I sources. In two studies, KIO_3 was found to produce slightly higher milk and blood iodine concentrations than did KI [41; 56], although absorption of KIO_3 may be slower than that of KI [41]. Diiodosalicylic acid (DIS) yielded much lower milk I excretion (0.45 mg I/day) than did NaI (0.79 mg I/day) [47]. Adding I (from KI) to the diet of cows fed $Na^{125}I$ significantly reduced milk ^{125}I secretion (12.2 vs. 7.1% of dose/day) and increased urinary ^{125}I excretion (49.1 vs. 65.9% of dose/day), whereas a similar amount of I from DIS had hardly any influence on milk and urinary ^{125}I output [50]. No significant differences in milk I concentrations could be observed between NaI, elemental I [19; 52], methyl iodide or $NaIO_3$ [52]. When feeding lactating dairy cows a ration containing 1.7-1.9 ppm I (DM) from either KI, CaI_2 or EDDI, no significant differences in milk I concentrations between the sources could be observed [14]. In spring, milk I levels were 63, 61 and 63 µg I/kg milk, respectively. In autumn, corresponding levels were 57, 69 and 58 µg I/kg milk.

3.2.2 Sheep and goats

No information is available on differences in I absorption from different I sources in small ruminants.

3.2.3 Discussion and conclusions

Results of I bio-availability trials in ruminants are summarized in Table 1.

Table 1 Selected results from experiments on bio-availability trials in lactating dairy cattle

Ref.	Sources used	Response criteria	Bio-availability
[41]	KIO_3, KI	Milk I, blood I	KIO_3 = KI
[56]	KIO_3, KI	Milk I, blood I	KIO_3 = KI
[47]	DIS, NaI	Milk I	NaI > DIS
[50]	KI, DIS	Milk I, urine I	KI > DIS
[19; 52]	NaI, I, methyl I, $NaIO_3$	Milk I	no differences between sources
[14]	KI, CaI_2 or EDDI	Milk I	no differences between sources

In summary, nearly all experimental evidence does not show any difference in bio-availability between KI, NaI, KIO_3, $NaIO_3$, CaI_2, elemental iodine, methyl I or EDDI for cattle. Only DIS was shown to be far less available than the other sources and should, therefore, not be used. For sheep and goats, no data are available. Availability of the different I sources is assumed to similar to that for cattle.
Thus, in practice there is no (dis)advantage of one specific I source for use in ruminant nutrition (except DIS), and the cheapest and most convenient source may be used.

3.3 Interactions influencing iodine absorption and metabolism

3.3.1 Interactions of iodine and selenium

3.3.1.1 Cattle

In two herds of dairy calves at pasture, the application of an intraruminal Se pellet (Se group) increased basal T_3 levels, whereas basal T_4 levels were decreased when compared with the control group not receiving a Se pellet [71]. In one of the two herds (16 calves/herd) thyrotropin releasing hormone (TRH) caused significantly higher peak T_3 levels in the Se group than in the control group.

3.3.1.2 Sheep and goats

No experimental evidence is available on effects of dietary Se additions on I metabolism in goats.

3.3.1.3 Conclusion

Although Se might influence I metabolism, insufficient data are available to judge this effect.

3.3.2 Interactions of iodine and goitrogens

3.3.2.1 General

Several plant species contain variable amounts of goitrogens, which can increase the I requirements of ruminants 2- to 4-fold). When damaged, white clover (*Trifolium repens*) and cassava can release HCN, which can be converted into thiocyanate in the animal. Several *Brassica* species (kale, rape, turnips) contain glucosinolates. These goitrogens affect I uptake by the thyroid gland, and their effect can be overcome by increasing dietary I concentrations. Low-glucosinolate cultivars of rape (for the production of low-glucosinolate rape-seed meal (“Canola meal”)) have been developed. Some types of *Brassica* seeds contain thiouracil goitrogens, which hamper the iodination of tyrosine in the thyroid gland. This effect is more difficult to overcome by increasing the dietary I content [60; 70].

3.3.2.2 *Cattle*

As dietary goitrogens are excreted in the milk of lactating ruminants, rumen breakdown is certainly not complete [62; 70]. When rape-seed meal was added to a casein/starch diet fed to cows and calves during 8 weeks (20% rape-seed meal added at the expense of both casein and starch), milk I concentrations decreased from 108 to 30 µg/kg (Tower[5]) or from 153 to 33 µg/kg (Turret) [62]. Tower rape-seed meal fed in concentrations of up to 18.9% of diet to dairy cows did not influence TSH, T_4 and T_3 levels, whereas diets containing 13.2 and 18.9% Midas rape-seed meal plasma TSH levels were increased and T_4 levels were decreased as measured by the TRH test[6]. Soybean meal served as a control. Dietary thiocyanate (5 g NaSCN on the first day and 2 g on the next 5 days) has been demonstrated to increase urinary I excretion in calves [48]. In thiocyanate-treated vs. non-treated calves, 54 vs. 34% of an oral dose of ^{125}I were excreted via the urine during the 6-day collection period. Feeding soybean meal instead of roasted soybeans significantly decreased TSH, T_4 and T_3 levels of steers [67]. When calves (135 kg BW) were fed up to 1400 g cottonseed meal, faecal excretion of daily orally administered ^{131}I (from NaI) increased up to 94%, whereas urinary ^{131}I excretion decreased up to 35% [51]. However, no suitable data on the effect of dietary goitrogens on I metabolism in ruminants are available to quantify this interaction [70].

3.3.2.3 *Sheep*

Thyroid weight of lambs increased linearly (from 67.4 to 120.8 mg/kg BW) as the concentration of glucosinolate (from rape-seed meal) in the diet was raised up to 17.5 mmol/kg [70].

3.3.2.4 *Goats*

Goats fed grass containing 34 g NO_3^-/kg DM developed small, hyperplastic thyroid glands, whereas control goats (NO_3^- content of the ration not given) had normal thyroids [63]. Thyroid weights were significantly lower in the nitrate-treated (0.55 g) than in the control animals (1.90 g). No thyroid hormone determinations were reported.

3.3.2.5 *Conclusion*

The occurrence of goitrogens in the ration may increase dietary I requirement of ruminants. Due to lack of data effects cannot be quantified. A tentative proposal for I requirements of ruminants fed high-goitrogen rations has been made by the ARC [5] (see par. 5).

3.4 Recycling

Iodine is recycled via secretion into the abomasum [70]. Moreover, presence of labelled I in the rumen after parenteral administration of ^{131}I indicates for I secretion via saliva and/or rumen epithelium [52]. This allows the secreted iodine to be extensively recycled. Moreover, T_4 is excreted via the bile. Only ± 10% of this T_4 is reabsorbed from the intestine. Therefore, T_4 excretion substantially contributes to faecal I excretion [52].

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[5] Tower, Turret and Midas are different varieties of rape-seed; Midas rape-seed contains relatively high goitrogen concentrations

[6] The TRH test was performed by intravenous injection of 300 µg TRH and determining TSH and thyroid hormones afterwards in all experimental groups

3.5 Excretion

Excretion of I mainly occurs via the urine. Besides this, lactating animals secrete significant amounts into faeces and milk. By day 18 after a single oral dose of ^{131}I, ± 40% of dose was excreted in the urine, 30% in the faeces and 8% in milk [52]. The I is lost from the body as iodide [70]. However, when I concentrations in the milk replacer of veal calves were raised from 0.57 to 200 ppm (DM), bile I concentrations increased from 0.26 to 8.2 µg/mL [32]. This indicates for the bile being another route of excretion when dietary I intake is high. Unfortunately, no data on urinary I concentrations were recorded in this experiment. When two lactating goats were given a single oral dose of ^{125}I, the partition of radioactive I excretion into milk, urine and faeces appeared to be temperature-dependent [39]. Results are given in Table 2.

Table 2 Selected results of ^{125}I excretion data in lactating goats after a single oral dose of ^{125}I [39]

Temperature °C	Milk	Urine	Faeces	Total
	%			
33	16.8	52.5	9.8	79.1
5	2.6	71.2	18.1	91.9

At both temperatures, urine is the main I excretion pathway. At higher ambient temperatures, T_4 output from the thyroid gland is lower and less I might be taken up by the thyroid gland and retained in the body. Moreover, as the I-concentrating mechanism is sensitive to T_4, lower T_4 levels are associated with increased milk I concentrations [39]. However, as only two goats were used, these data have to be considered with caution.

4 IODINE REQUIREMENTS

Theoretically, the I requirements of adult animals are determined by the endogenous (inevitable) losses and the secretion into milk. In growing and pregnant animals I is also deposited in growing (foetal) tissues. However, no data are available on endogenous I losses and in all species the I content of the milk is very variable. Moreover, I requirement depends on the ambient temperature, whereas the criteria of adequacy are not consistent [70]. Thus, a proper calculation of minimal I requirements is impossible.

4.1 Cattle

4.1.1 Dairy cattle

In pregnant cows, in calves hardly any I is present up to 70 days of gestation. From 75 to 118 days the thyroid gland develops, whereas the greatest increase in I content occurs near term. Pregnancy, however, is considered not to increase I requirements to any significant degree [61].

The I content of bovine milk is very variable and vulnerable to environmental contamination. Especially the use of iodine udder washes [66] and teat disinfectants (iodophors) [11] can cause abberatingly high values, although these treatments are not always associated with high milk I concentrations [31; 66]. This depends i.a. on I concentration of the solution used, way of application to the udder or teat (dipping, spraying, wiping) and post-treatment cleaning of the udder. In an American study, milk I content (total of 1572 milk samples, µg I/kg) was reported to be 148 (no external I treatment of the udder), 167 (I-containing teat dip), 202 (I in backflush systems) or 251 (both I-containing teat dips and I in backflush systems) [13]. Similarly, in a French study (848 samples form 537 dairy factories) 95% of the samples had I contents between 10 and 250 µ/kg [7]. Samples contained on average 23-44 (summer) or 93-97 µg I/kg (winter). When properly determined, the milk I content still appears to depend on the season, soil type (especially the top soil [31]) and nutrition: in Dutch surveys, summer milk I concentrations were 12-33 µg/kg, whereas winter results were 25-49 µg/kg (samples from 20-50 dairy plants) [11; 37]. The higher winter values have been suggested to originate from the higher amounts of concentrates used [31]. Dutch milk I concentrations from dairy plants were demonstrated to depend on the soil type [12]. The concentrations were (µg/kg) 27 (marine clay; north and southwest), 9.7 (sand and peat; east and middle) and 13.4 (composite soils; northwest, middle and south). It is not clear as to what extent this effect was interrelated with the proximity to the seaside (see paragraph 3.1). In an American survey of bulk tank milk samples (175 herds), much higher values were observed, as milk I concentrations were on average 466 µg/kg [66]. These high values were not related to the use of iodine teat dips. In the same report, the influence of dietary I concentrations was demonstrated by feeding EDDI to 6 dairy cows (1 g/head/day via rumen fistula) for 2 weeks. Mean milk I concentration increased from 210 to 6225 µg/kg. Milk yield was not influenced by I treatment. Other reports on the I content of bovine milk also supply a wide range of data (µg/kg): 10-80 [33], 50 [1], 20-140 [43], 5-300 [36] or 30-300 [61]. Finally, breed differences in milk I content could not be observed [11]. Colostrum can contain ± 2-3 times higher values than milk [36; 70].

The I content of bovine milk is more or less linearly related to the dietary I content. Herd bulk milk I concentration has been demonstrated to be correlated to the dietary I intake (0 to 36 mg I/head/day from EDDI) [10] or from 0 to 1000 mg EDDI/head/day (2 animals per group [49]). Similarly, a significant correlation between dietary I intakes (restricted to those of ± 10 mg head/day) from pasture and concentrates and milk I concentration was calculated [3]. The equation was bulk milk I content (µg/kg) = 0.37 x (average I intake (mg/cow/day)) + 0.05

(number of farms not clear; no statistical data given).

Apparently, the animal does not have a clear limit for absorption or transfer of I into milk [27]. Recovery of a certain dose of supplementary I in the milk was calculated to be 5-10 [40], 10 [11] to 30% [70] of dietary intake. The I content of the milk is inversely related to the level of milk yield [11] and thus increases as lactation progresses [52].
The I content of bovine hair is sensitive to feed I content. In cows fed rations containing either 70, 150, 230, 390 or 710 µg I/kg DM, pigmented hair I contents were 151, 316, 458, 1080 or 1645 µg I/kg DM [22].

4.1.2 Beef cattle

No separate calculations need to be made for the I requirements of beef cattle. Dietary I concentrations of 0.5 ppm should be sufficient, unless the ration contains goitrogenic substances [60].

4.2 Sheep

In ovine foetuses, the iodine uptake commences at the beginning of the second trimester of pregnancy [26]. No data are available on (increased) I requirements for pregnancy.
Sheep milk I content is extremely variable (79-1831 µg/L), depending on dietary I intake, weather (lower milk I concentrations during rainfall periods in grazing animals) and season (milk I concentrations were highest at the end of summer, and lowest in spring) [9]. Other reported values are 80-389 [15] and 145-937 (summer) or 116-1183 µg I/kg (winter) (6 and 9 samples, respectively) [7]. No data are available on the I content of wool.

4.3 Goats

Data on goat milk I content are scarce. Values of 70 [46], 199 [15] and 247 µg I/kg [22] were reported (breeds not given). The latter value was the mean value after feeding 18 lactating goats for 56 days with a ration containing 500 µg I/kg DM. In the same report, 19 goats fed a similar ration containing 40 µg/kg DM had a mean milk I concentration of 6 µg/kg.
The I content of goat hair is reported to be 192 µg/kg DM (500 µg I/kg dietary DM for 52 weeks) [22]. In the same experiment, goats fed a ration containing 40 µg I/kg DM during 52 weeks had a mean hair I content of 68 µg/kg DM.

4.4 Conclusion

As insufficient data are available, factorial estimation of I requirements of ruminants is precluded. Only tentative assessments are possible.

5 ALLOWANCES

5.1 Survey of recommendations

In Table 3 recommendations for I allowances as given in the literature are presented.

Table 3 Recommended I allowances for ruminants (ppm (DM))

Ref.	Season	Cattle	Sheep	Goats	Category
[5]	Summer	0.11	0.11		
	Winter	0.52	0.54		
				0.65	Mature, 2 year
				0.20	Mature, 4 year
[59]			0.10-0.80		The higher level indicated for pregnancy and lactation
[61]		0.33			Maintenance
		0.45			Lactation
[60]		0.5 (as fed)			Beef cattle
[46]				0.4-0.6	

For goats, the AFRC [2] adopts similar values as proposed by the ARC [5] for sheep and cattle. For rations rich in goitrogen containing plants (see 3.3.2), an allowance of 1.8 [35] to 2 ppm I (DM) [5] is suggested.

5.2 Conclusion

Unless otherwise proven, 0.15 ppm (DM) (summer) and 0.50 ppm (DM) (winter) seem to be defendable estimations of I requirements of lactating dairy cattle and sheep. For beef cattle, 0.5 ppm should be sufficient. For goats, AFRC recommendations 0.20-0.65 ppm I (DM) (depending on age) may be adopted. For goitrogen-rich rations, 1.8 ppm I (DM) may be recommended.

6 CRITERIA TO JUDGE IODINE STATUS

6.1 Suggested indicators of iodine status

In overt I deficiency, the enlargement of the thyroid gland in the throat region is both visible and palpable. However, this is a late symptom of I deficiency. Determinations of thyroid weight, histology and iodine content have been shown to be insufficiently sensitive (e.g. the same I concentrations and histological observations could be obtained from both normal and I-deficient animals) and less invasive methods are available [70]. Similar remarks can be made with respect to the use of several I determinations in serum. These include serum-precipitable I (SPI), protein-bound I (PBI) and butanol-extractable I (BEI) and serum inorganic I. All of these are insufficiently sensitive and do not supply exact information concerning T_4 function.

In adult animals, plasma thyroid-stimulating hormone (TSH) concentration has been reported to be the most sensitive indicator of iodine status. When dietary I supply is insufficient, TSH levels will increase to maintain circulating T_4 and T_3 concentrations. If I deficiency proceeds, T_4 concentrations will decrease and, finally, in very severe I deficiency T_3 concentrations decrease [6]. In seven cows treated with 6-n-propyl-2-thiouracil (4.0 mg/kg BW/day for ± 60 days), TSH levels were 9.5-11.5 ng/mL, whereas in seven control cows not treated with this compound values were 2-2.5 ng/mL [54]. However, as no threshold values for TSH in ruminants have been reported, this determination cannot be applied. In general, serum T_4 levels are not very reliable to judge I status due to the many factors influencing it. Thyroxine levels are usually higher in winter than in autumn or summer, whereas levels are depressed by intestinal parasitism and in early lactation. In newborn animals, T_4 levels can be three times as high as those of their dams, but decrease to “adult” values within some 2 months. Thus, for newborn animals ratios of newborn:dam T_4 concentrations have been suggested to indicate for I deficiency in the offspring. Values are given in Table 4 [70]. In newborn lambs and kids, plasma T_4 levels were well related to the ability to withstand and recover from hypothermia. Lambs with T_4 levels > 40 nmol/L after birth did not show any benefit from extra dietary I supply [15]. If hypothyroidism is caused by Se deficiency or thiouracils, conversion of T_4 into T_3 is affected. Then, T_3 rather than T_4 determinations should be performed. However, apart from the relatively late response of T_3 levels to I deficiency, T_3 levels are influenced by feed and water restriction and high ambient temperatures. Marginal bands for both T_3 and T_4 levels in adult animals, as well as dam/newborn ratios are given in Table 4 [70].

Table 4 Marginal bands for T_3, T_4 and desired dam/newborn ratios in sheep and cattle (derived from reference [70])

Indicator	Cattle	Sheep
Dam/newborn ratio	2-2.5	1-1.5
	Adult	
Serum T_4 (nM)	25-50	20-30
Serum T_3 (nM)	2.0-2.5	1.0-1.7

Urinary I excretion can be used to assess I status. A urinary excretion < 15 µg I/day [6] or < 100-250 µg I/L (cattle) [64] is considered to indicate for I deficiency, whereas concentrations > 500 µg/L are indicative for excessive I intake [64]. However, when goitrogens are the cause of inhibition of the uptake of I into the thyroid gland, urinary I excretion may be normal or relatively high. Under these circumstances, determination of urinary I excretion cannot be used [6].

The same remark may be valid for the use of milk I concentrations. The occurrence of goitre in lambs was associated with milk I concentrations of their dams of 45-98 µg/kg [9]. This is in accordance with milk I concentrations below 80 µg/kg [44] or below 70-100 µg/kg [70] reported to indicate for I deficiency in sheep. For lactating cattle, milk I concentrations below 30-50 µg/kg [70] or < 25 µg/kg [3] have been suggested. According to Puls [64], values from 8-25 µg/kg indicate for a deficiency, whereas values between 30-300 are considered adequate. However, considering the extreme (seasonal) variability of milk I concentrations [9], these values have to be used with caution. In general, values < 25 µg I/kg milk indicate for a deficiency. Moreover, as iodine can be accumulated in milk by active transport into the udder, milk I concentrations are only suitable to determine I deficiency. Higher values are insufficiently informative to indicate for (more than) adequate supply.

6.2 Conclusions

As most of the suggested indicators of I status have one or more disadvantages, the use of at least two different indicators is recommended. In lactating animals, milk I levels can be used to detect simple I deficiency (low total I content of the ration), whereas urinary I concentrations can be used in non-lactating animals or in herds where I-containing teat dips or udder washes are employed. As a second indicator determination of serum T_4 and, if goitrogens are suspected to cause the I deficiency, also serum T_3 levels can be used. Additionally, newborn animals from suspected herds should be checked for goitre.

7 DEFICIENCY

Clinical I deficiency is characterized by a visible enlargement of the thyroid gland (in the newborn animal [26]), which is specific for this disease. This condition is called “goitre” (or “goiter”) [70]. Iodine-deficient ewes have been demonstrated to bear less lambs [68]. This was in part due to a lower number of twins in iodine-deficient as compared with iodine-sufficient ewes. Moreover, iodine deficiency increased perinatal lamb and calf mortality. Calves and lambs may be hair-/woolless, weak or dead at birth. In adult animals, signs may be reduced wool yield, irregular cycling, low conception rate, retained placenta in the female and decreased libido and poor semen quality in the male [26; 59; 60]. In goats, kids from I-deficient does have lower birth weights and a higher incidence of goitre [34]. Moreover, in I-deficient pregnant goats incidence of abortion is higher, whereas perinatal mortality of kids is high and vitality and growth are poor [22; 23; 28; 69].
In cattle, dietary I deficiency (0.06 ppm I) was associated with elevated activities of iodothyronine deiodinase (thyroidal type I) and cytosolic glutathione peroxidase in the thyroid gland, but not in the liver and pituitary [73].

7.1 Direct measures in deficiency cases

7.1.1 Direct continuous supplementation

Iodine can be added to the ration of housed ruminants as mineral mixes incorporated in total mixed rations or concentrates. Supplementation of 2-5 mg I/head/day from any commonly used I salt (except diiodosalicylic acid) is usually sufficient [16]. Moreover, seaweed (containing 1.9 [42] to 4-6 g I/kg [70]) and salt licks (0.1 g I/kg [70]) can be applied. When salt licks are used outside, KI and $Ca(IO_3)_2$ are less suitable because they readily volatilise and leach. More favourable sources are CuI_2 [70] or CuI [29]. Fertilization of grassland with I-compounds has proven to be rather insufficient, as the remaining effect is only minor in successive cuts [25].

7.1.2 Direct discontinuous supplementation

Intramuscular injection of 1 mL iodised oil (400 mg I (exact source not given); Lipiodol) has been reported to significantly increase sheep milk I concentrations for up to 14 months when compared with untreated animals [9]. The application of I-containing drenches is not recommended because it is too cumbersome. If applied, two oral doses of 280 mg KI or 360 mg KIO_3 after the third and fourth months on pregnancy can be given to ewes given a ration containing kale [70].

7.1.3 Slow release oral supplementation

In sheep, several intraruminal devices supplying 500-1100 µg I/day (I source not given) through 3 years have been tested [17; 70]. Iodine deficiency was not observed during the supplementation period.

8 TOXICITY

Iodine can cause cumulative and chronic toxicity. The main cause of I toxicity is the faulty use or preparation of mineral supplements, e.g. for the control of foot rot or lumpy jaw (actinomycosis) [55; 66]. Besides this, the feeding of large amounts of seaweed (containing 4-6 mg I/kg) can pose animals at risk [70]. Clinical I toxicity symptoms are anorexia, nasal discharge, excessive tear and saliva production, rapid breathing and coughing from tracheal congestion, decreased appetite and milk production, poor growth, hair loss, dermatitis and abortion. Both increased and decreased body temperature and increased heart rates can be observed [4; 32; 55; 59; 70].

In veal calves fed a milk replacer containing either 0.57, 10, 50, 100 or 200 ppm (DM) I (from EDDI) from 3 to 38 days of age, only the 200 ppm level reduced growth, DMI, feed efficiency and DM digestibility [32]. Both at the 100 and 200 ppm levels clinical I toxicity symptoms were observed. However, as the I levels in muscle, spleen, heart, kidney and liver started to increase at the 50 ppm level (when compared with the lower levels), a maximum tolerable I level below 50 ppm (DM) was suggested. In ruminating heifer calves fed up to 174 ppm I (DM), performance was slightly depressed at I intakes > 42 ppm I (DM) [18]. Maximum tolerable levels (ppm (DM)) of 50 (sheep and cattle) [57; 59; 70], 5 (dairy cattle) [61], 8-20 (cattle, sheep and goats) [2; 5] or 50 ppm (beef cattle) [60] have been suggested.

8.1 Direct measures in toxicity cases

Except lowering the I content of the ration no direct measures to be taken in I toxicity cases are reported for ruminants. Cows suffering from endometritis after abortion normally respond to antimicrobial therapy [55].

9 PREVENTION OF DEFICIENCY

9.1 Short-term prevention strategies

No separate short-term prevention measures except the application of intraruminal boluses [70] have been recommended.

9.2 Long-term prevention strategies

No suitable long-term prevention strategies are available. Iodine fertilization of pastures is rather inefficient because of low uptake of the I by the plants and small residual effects in later cuts [25; 70].

Table 5 Inventory of I allowances for cattle, sheep and goats as used in some foreign countries (ppm (DM))

Country	**Ref.**	**Allowance**				
		Cattle	**Ref.**	**Sheep**	**Ref.**	**Goat**
United Kingdom	[2; 5; 70]	0.5 (no goitrogens); 2 (goitrogens present); 0.15 (summer)				
USA[a,b]	[72]	0.33 (dry, gestation) 0.45 (lactation) 0.5 (beef cattle)	[59]	0.1-0.8 (no goitrogens)	[58]	?
Germany	[20]	0.25 (growth) 0.50 (mature)		?	[8]	0.3-0.8
France	[24]	0.2-0.8 (0.15 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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