Iodine toxicity in ruminants

I. Paulíková, G. Kováč, J. Bíreš, Š. Paulík, H. Seidel, O. Nagy

University of Veterinary Medicine, Košice, Slovak Republic

ABSTRACT: The inadequate intake of iodine is still a topical problem, because it contributes to a reduction in performance through the disturbed health of adult animals and their offspring. The necessity of iodine is frequently described in association with hypothyroidal state. In our work we want to assign the hidden risk of prolonged iodine feeding in ruminants. We discuss the possible sources of iodine intoxication, the clinical signs of the intoxication, its effects on production, reproduction, and thyroid functions; animal susceptibility to intoxication; laboratory and necropsy findings; diagnostic, and therapeutic possibilities.

Keywords: iodine; intoxication; cattle; sheep; goats

Contents

1. Introduction

The role of iodine deficiency in thyroid disorders, especially that of endemic goitre, has been known for two hundred years. Nowadays, over one thousand million people have an inadequate iodine intake (Maberly, 1994).

As with the human population, so too the occurrence of functional thyroid disorders has been increasing in animals, especially in new-born calves and lambs (Körber *et al.*, 1985; Kursa *et al.*, 1996; Bíreš *et al.*, 1996; Sargison *et al.*, 1998).

Iodine is the essential element used by the thyroid for the biosynthesis of thyroid hormones (Thilly *et al.*, 1992). Through these hormones, iodine is involved in controlling metabolism, cell growth and maturation, and the development and growth of tissues (Hetzel, 1989). Iodine deficiency during pregnancy has particularly adverse effects – retardation of foetus development, its death, dead or weak neonates with hyperplastic thyroid – goitre. These findings were reported in cattle (Kursa *et al.*, 1996; Herzig and Suchý, 1996), sheep (Radostits *et al.*, 1994), and goats (Radostits *et al.*, 1994; Bíreš *et al.*, 1996). Importance of iodine for cell mediated immune function has not been confirmed by Šlosarková *et al.* (2002).

As a mineral element, iodine cannot he synthetized in the body. Soil and consequently plants are the primary sources of iodine. Because the iodine concentrations of soils in Central Europe (and so in Slovakia) are relatively low (Anke *et al.*, 1993), the foodstuffs cannot supply an adequate iodine intake for animals. The Slovak region thus belongs to these regions where iodine deficiency disorders occur, both in the human population and farm animals, particularly in sheep and goats (Bíreš *et al.*, 1993, 1996). Substitution and utilization of iodine in pigs and cattle were studied by Herzig *et al.* (1999, 2000, 2001a, 2001b).

The literature reports mostly concern the aforementioned disorders associated with an inadequate iodine intake. However, from the practical point of view, it is important to mention conditions when the iodine acts as a poison to the body. In humans, Pennington (1990) describes six types of excessive iodine intake – thyroiditis, goitre, hypothyroidism, hyperthyroidism, hypersensitiveness, and acute responses. Urinary iodine levels can be used as indicator of iodine intake in cattle (Herzig *et al.*, 1996)

In this work we want to indicate comparable clinical signs in ruminants after prolonged excessive iodine intake.

In some people, prolonged iodine intake at a dose over 10 times of the daily requirement may lead to goitre or thyrotoxicosis (Wolf, 1969; Braverman *et al.*, 1971). In ruminants, iodine intoxication is a less frequent clinical problem because the toxic doses in these species are very high.

2. The toxic doses of iodine in ruminants

Severe or fatal intoxication in calves occurrs after the prolonged administration of iodine at a dose of 10 mg/kg per day. Intoxications with marked and mild clinical manifestations are induced by daily iodine doses of 2.2 mg/kg and 0.4 mg/kg, respectively (Mangkoewidjojo *et al.*, 1980). These doses are about 500, 100, 20 times higher than the daily requirement of iodine (approximately 0.02 mg/kg b.w. – NRC, 1978). In adult cows weighing about 600 kg, the daily iodine requirement for synthesis of thyroid hormones is approximately 10 mg (Convey *et al.*, 1978), i.e. 0.016 mg/kg b.w. In practice, intoxication results from the prolonged intake of higher iodine doses ranging from 70 to 600 mg per animal and day (McCauley *et al.*, 1972; Wallace, 1975; Hillman and Curtis, 1980; Olson *et al.*, 1984). In a dairy cow weighing 600 kg this represents 0.12 up to 1.0 mg of iodine per kg of body weight daily. This amount is between 7.5 and 63 times higher than the daily requirement. Sheep are probably less sensitive to iodine intoxication than cattle (Forbes *et al.*, 1932). Clinical disease with a fatal course in lambs weighing on average 30 kg was manifested after an intake of at least 562 mg of iodine in the form of EDDI (ethylenediamine dihydroiodide) or 393 mg of potassium iodide for 3 weeks (McCauley *et al.*, 1973). These doses represent 18.7 and 13.1 mg of iodine per kg b.w. and day, respectively.

3. Possibilities of iodine intoxication in cattle and sheep

Land-locked European countries, including Slovakia, are deficient in iodine to some extent (Delange, 1995; Langer, 1995). However, under certain conditions daily iodine intake may exceed the daily requirement, even several times.

This may be caused by:

– the long-term consumption of a diet containing several components supplemented with iodine
– the incorrect management of mineral feed admixtures over a long time
– the use of iodine compounds over long periods of time as a feed additive for the therapy and prevention of infectious pododermatitis, a complex of respiratory diseases, actinomycosis, mastitis, and infertility.

These possibilities of iodism in cattle and sheep were reported by several authors (Forbes *et al.*, 1932; Blaxter, 1946, 1948; Miller and Swanson, 1973; McCauley *et al.*, 1972, 1973; Wallace, 1975; Buck *et al.*, 1976; Fish and Swanson, 1979; Hillman and Curtis, 1980; Olson *et al.*, 1984) both in experimental and field conditions.

4. Clinical signs of the intoxication

Iodism in cattle is manifested by a persistent cough, hyperthermia, naso-ocular discharge, inappetency, depression, dermatitis, and alopecia (Forbes *et al.*, 1932; Blaxter, 1944, 1946; Schmidt *et al.*, 1971; McCauley *et al.*, 1972; Newton *et al.*, 1974; Wallace, 1975; Hillman and Curtis, 1980; Mangkoewidjojo *et al.*, 1980; Olson *et al.*, 1984; Radostits *et al.*, 1994; White, 1998). In dairy cows, Döcke (1994) described tachycardia, nervousness, loss of weight, and a high level of metabolism. Other reported signs included the occurrence of exophthalmos (Blaxter, 1946; Hillman and Curtis, 1980; Radostits *et al.*, 1994) and infectious diseases, especially diseases of the respiratory system (Forbes *et al.*, 1932; Blaxter, 1946; McCauley and Johnson, 1972; McCauley *et al.*, 1972; Wallace, 1975; Mangkoewidjojo *et al.*, 1980; Olson *et al* , 1984), in some cases death (Mangkoewidjojo *et al.*, 1980) and an increased mortality of the offspring from such dams (Thomas and Moore, 1953; Olson *et al.*, 1984).

In sheep, depression, anorexia, hyperthermia, cough, changes in the respiratory system, and

sometimes death were reported (Forbes *et al.*, 1932; Blaxter, 1948; McCauley *et al.*, 1973). Lacrimation, salivation, and skin lesions were not observed (Blaxter, 1948).

Some clinical and laboratory findings of iodine intoxication of cattle and sheep are summarized in Table 1.

5. The effects on production and reproduction

– a decrease in milk yield and an increase in reproduction disorders (Thomas and Moore, 1953; Schmidt *et al.*, 1971; McCauley *et al.*, 1972; Wallace, 1975; Hillman and Curtis, 1980; Olson *et al.*, 1984; Radostits *et al.*, 1994)

Table 1. Clinical and laboratory findings of intoxication caused by various iodine doses in cattle and sheep

Animals	Toxic dose(s)	Clinical and laboratory findings	Reference
Calves	50–10 mg/kg of dry matter of diet for up to 112 days	decrease in food intake, coughing, nasal discharge	Newton *et al.*, 1974
	250–1 250 mg of iodine (EDDI) per calf and day for 6 months	mucopurulent nasal discharge, seromucous ocular discharge, hypersalivation, thick scaly skin, rough hair, alopecia, pneumonia with the severity apparently dose-related	Mangkoewidjojo *et al.*, 1980
	1 250 mg of iodine (EDDI) daily for 6 months	decrease in persistence of antibody titres to *Brucella* and *Leptospira* organisms after vaccination, decreased lymphocyte mitotic activity, phagocytosis by WBC, and WBC counts	Haggard *et al.*, 1980
	0.625–5.0 mg of iodine per kg of b.w. (EDDI)	nasal discharge, occasional lacrimation, moderate coughing	Fish and Swanson, 1982
Dairy cows	164 mg of EDDI for 3 weeks	lacrimation, coryza, conjunctivitis, coughing, hair loss, scaly dermatitis, exophtalmos	Hillman and Curtis, 1980
	440 mg of EDDI for 1 month	milk production reduced by 15%, coughing	Olson *et al.*, 1984
	300–600 mg of EDDI for 4 years	coughing, naso-ocular discharge, salivation, dry coat	
	600 mg of EDDI for 7 years	pneumonia in calves with 50% mortality, decreased milk production	
	300–600 mg of EDDI for 2 years	pneumonia in calves, decreased milk production, naso-ocular discharge	
	>68 mg of EDDI for 1 year	cows with injured hocks not responding to therapy	
	250–785 mg of EDDI for 3 years	naso-ocular discharge, increased calving interval	
Lambs	94–785 mg of iodine in EDDI or potassium iodide for 3 weeks	lethargy, decreased food intake, retarded growth, hyperthermia, coughing, at larger doses death due to bronchopneumonia	McCauley *et al.*, 1973

EDDI = ethylenediamine dihydroiodide

– the loss of body weight in adult animals (Blaxter *et al.*, 1944, 1946; Schmidt *et al.*, 1971; McCauley *et al.*, 1972; Hillman and Curtis, 1980)
– a decrease in weight gain in calves (Forbes *et al.*, 1932; McCauley and Johnson, 1972; Herrick, 1972; Newton *et al.*, 1974; Leung *et al.*, 1980; Fish and Swanson, 1982).

In sheep, neither the loss of weight in adults (Blaxter, 1948) nor the decreased weight gain in lambs (Forbes *et al.*, 1932; McCauley *et al.*, 1973) were observed.

6. The effects on thyroid functions

A study of the effects of excessive iodine intake on thyroid functions showed more distinct results. Thyroid functions were not altered even after prolonged increased iodine intake in common farm conditions (Convey *et al.*, 1977; Hillman and Curtis, 1980). The same was observed in experimentally-induced iodine toxicosis with the use of 200 up to 300 times higher dose than the daily requirement. In calves, the altered synthesis of thyroid hormones was recorded only after long-term supplementation of an iodine dose 500 times higher than their daily requirement (Leung *et al.*, 1980). An inhibitory effect of excessive iodine intake on the synthesis of thyroid hormones was observed in humans (Nagataki, 1974) and horses (Baker and Lindsey, 1969). Nagataki (1974) suggested that such effects depend not only on the amount and time of iodine exposure, but also on the functional state of the thyroid and species or individual genetic potential. According to Leirer *et al.* (1983), a very high intake of iodine may consequently cause a blockage of thyroid function with a reduction in iodine incorporation into thyrosine residue, leading thus to a hypothyroidal state.

7. Susceptibility to intoxication

Different responsiveness to iodine intoxication was found not only between the animal species (NRC, 1978), but also within the same ruminant species (McCauley *et al.*, 1973; Newton *et al.*, 1974; Mangkoewidjojo *et al.*, 1980; Fish and Swanson, 1982).

Perhaps, this may explain cases of excessive iodine intake in cattle with no clinical signs, or manifested only by a mild cough and slight naso-ocular discharge (Long *et al.*, 1953, 1956; Kuebler, 1957; Buck *et al.*, 1976; Fish and Swanson, 1982), which may be attributed to the expectorant effect of iodine (Beckman, 1961). According to some authors, the development of clinical iodine toxicosis in cattle (Olson *et al.*, 1984) and sheep (McCauley *et al.*, 1973) depends, apart from the amount and exposure time, on such factors as stress, concomitant diseases, and nutritional disorders. The different severity of clinical manifestation may also depend on the type of iodine compound. This was recorded in laboratory animals (Webster *et al.*, 1957), calves (Newton *et al.*, 1974; Fish and Swanson, 1982), and lambs (McCauley *et al.*, 1973). A higher tolerance was observed with an organic iodine compound (EDDI) compared with inorganic iodine substances (calcium iodate, potassium iodate). However, the biological efficacy of organic and inorganic iodine compounds at normal doses is almost the same (Miller and Swanson, 1973). Risk of iodine intoxication appears when the EDDI is used for the therapy and prevention of infectious pododermatitis, because recommended doses per animal and day (400–500 mg for 2–3 weeks, or 50–100 mg continually for a longer time) (Harris, 1978) are several times higher than the nutritional requirements. A decrease in body weight or weight gain is preceded by a decrease in food intake, which was observed in dairy cows (McCauley *et al.*, 1972), sheep (Blaxter, 1948), calves (Forbes *et al.*, 1932; McCauley and Johnson, 1972; Newton *et al.*, 1974), and lambs (Forbes *et al.*, 1932; McCauley *et al.*, 1973). The cause of decreased food intake is not well known. Studies with iodine administration by means of gelatinous capsules (McCauley *et al.*, 1973) or oral tube (Leung et al., 1980) indicate that, in addition to the reduced palatability of the food (Newton *et al.*, 1974; Fish and Swanson, 1982), some unknown factors are involved.

8. Laboratory findings in iodine intoxication

During iodine toxicosis some changes in biochemical indices are found. Hillman and Curtis (1980) reported significant increase in serum glucose, AST, and blood urea nitrogen. A significant decrease in serum cholesterol and urinary creatinine was also observed (Long *et al.*, 1953; Hillman and Curtis, 1980). However, other authors (Olson *et al.*, 1984) did not confirm these findings. On the other hand, there was evidence that increased iodine intake correlates with increase in iodine concentrations in the blood serum of dairy cows

(Long *et al.*, 1953, 1956; Kuebler, 1957; Harris, 1978; Hemken, 1979; Olson *et al.*, 1984; Maas *et al.*, 1989), calves (Newton *et al.*, 1974; Fish and Swanson, 1982), and lambs (McCauley *et al.*, 1973), and also in the milk and urine of dairy cows (Convey *et al.*, 1977; Harris, 1978; Binnerts, 1979; Hillman and Curtis, 1980; Hemken, 1979).

Alterations in the immune system occur during the continuous administration of the EDDI form of iodine at the daily dose of 50 mg, which is the dose commonly used for the prevention of infectious pododermatitis in cattle (Harris, 1978). In animals, changes in the quantity and functional state of leukocytes and other components of the immune system were found. These changes resulted in lymphopenia, neutrophilia (Hillman and Curtis, 1980), leukopenia, a decrease in lymphocyte blastogenic response to T and B cellular mitogens, depressed phagocytic capacity, and a shortening of the persistence of the antibody titre in response to bacterial mitogens (Haggard *et al.*, 1980). These findings clearly indicate suppression of cellular and humoral immunity, possibly with inadequate response to infectious agents. This may explain the field observations of the increased occurrence of refractory infectious diseases in animals fed foods supplemented with excessive amounts of iodine (McCauley and Johnson, 1972; McCauley *et al.*, 1972; Wallace, 1975; McCauley *et al.*, 1973; Olson *et al.*, 1984).

9. Necropsy findings

The majority of pathological changes are related to the respiratory system, in some cases to the thyroid and adrenal glands. The severity of the lesions depends strongly on the amount of iodine intake. In cattle, gross findings in the respiratory system are characteristically tracheitis, bronchopneumonia, and pleurisy (Mangkoewidjojo *et al.*, 1980). Enlargement and hyperaemia of the mediastinal lymphnodes are observed in some cases. In the lung tissue, there is exudative inflammation at the centres of the affected areas, hypertrophy of the bronchial mucosal membrane, necrosis in the bronchioles, and fibrinous exudate in the alveoli. Microscopical findings may consist of changes in the tracheal mucosal membrane (squamous metaplasia), loss of cilia, and slight lymphocytic and neutrophilic infiltration of the lamina propria. At the lumen and the wall of the bronchi, bronchioles, and alveoli, inflammatory infiltration was found with the presence of neutrophils, lymphocytes, and macrophages. Peribronchial lymphatic follicles are hyperplastic, lymph vessels are dilated or spastic. Squamous metaplasia of the salivary glands was observed after excessive EDDI intake (Mangkoewidjojo *et al.*, 1980).

McCauley *et al.* (1973) observed suppurative bronchopneumonia and fibrinous pneumonia in sheep intoxicated with iodine.

The evidence for thyroid alteration by increased iodine intake in cattle and sheep was not conclusive (McCauley *et al.*, 1973). While some authors (Convey *et al.*, 1978) did not report thyroid enlargement in dairy cows even with an iodine intake about 200 to 400 times higher than their daily requirement, Wallace (1975) observed thyroid hypertrophy in dairy cows with an iodine intake only 10 times higher (107 mg per day) than their daily requirement. Similar findings were also observed in calves. While Newton *et al.* (1974) described some cases of increased thyroid weight with a 25–200 times higher intake, others (Leung *et al.*, 1980) did not find thyroid hypertrophy even with an iodine intake 500 times higher than their requirement. Increase in thyroid weight after excessive iodine intake was also described in horses (Baker and Lindsey, 1969) and pigs (Newton and Clawson, 1974), as well as in humans (Suzuki *et al.*, 1965; Wolf, 1969).

Similarly, different findings were recorded in the relation between iodism and the adrenal glands. Wallace (1975) reported the hyperplasia of adrenal glands in lactating cows with estimated iodine intake of 107 mg per day. Newton *et al.* (1974) found an increased weight of adrenal glands in calves fed iodine at a dose of 25 to 100 mg/kg. These authors suggested that hyperplasia of this endocrine gland may be the result of stress caused by consuming a diet with high content of iodine. However, the significance of these observations is questionable because Newton *et al.* (1974) in another experiment within their work did not confirm the previous findings. The adverse effect on the weight of adrenal glands and on serum glucocorticoid levels in calves fed up to 1 250 mg of iodine daily was also reported by other authors (Leung *et al.*, 1980).

10. Diagnosis

It is impossible to make a diagnosis of chronic intoxication without laboratory examinations. Except

for the analysis of iodine concentrations in the blood serum, milk, and urine, indices of metabolic profile have no diagnostic value. Confirmation of the diagnosis in suspect herds requires the determination of iodine in the aforementioned samples completed by examination of the diet (Olson *et al.*, 1984). Only in this way, may we clarify the basic problem.

11. Therapeutic possibilities

No specific antidotes against iodism are available. Remission of the problem requires the elimination of iodine sources. This will lead to a gradual decrease in iodine body levels and a reduction in the clinical manifestation of iodine toxicosis (Olson *et al.*, 1984).

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Corresponding Author

MVDr. Iveta Paulíková, University of Veterinary Medicine, Komenského 73, 041 81 Košice, Slovak Republic
Tel.: +421 55 633 80 71, fax: +421 55 63 23 66, e-mail: pauliko@uvm.sk