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REVIEW ARTICLE

SELENIUM IN THE DIET OF RUMINANTS

Barna Kruzhel[1], Małgorzata Bąkowska[2], Stakh Vovk[3], Ewa Nowakowska[2], Pavkovych Sergei[1]

[1]Lviv State Agrarian University, Ukraine
[2]West Pomeranian University of Technology, Szczecin, Poland
[3]Institute of Agriculture in the Carpathian Region, National Academy of Agricultural Sciences of Ukraine

Abstract. The results of the research show that there is a deficiency in Se in the environment in most regions. It is crucial to supplement selenium deficiency in ruminants from deficient regions as long-lasting lack of this element may lead to many diseases. Selenium serum or blood level can be an indicator of the supply of this element in ruminants (intravitally) as well as its content in the liver and kidneys. The most commonly used criterion for assessing the selenium status in livestock is its serum or blood content. At a low concentration of this element in the serum/blood of ruminants, its supplementation is necessary.

Key words: diseases, ruminants, selenium

INTRODUCTION

Studies on the role of selenium in animals are carried out for many years. This element belongs to the so-called trace elements, which are necessary for keeping the proper functions of an organism.

Selenium is not evenly distributed in the environment, there are areas with soils either poor or rich in this element. Most soils in the world have low content of this element [Kabata-Pendias and Pendias 1999, Borowska 2002, Cartes et al.

Corresponding author – Adres do korespondencji: Barna Kruzhel, PhD, Lviv State Agrarian University, Department of Biotechnology and Animal Science, Lviv–Dubljany, 80381, Ukraine, e-mail: barna53@mail.ru

2005, Broadley et al. 2006]. According to Kabat-Pendias and Pendias [1999], the average content of selenium in soils on a global scale is equal to 0.33 mg · kg^{-1}. Sandy soils generally have typically low quantities of this element (from 0.06 to 0.4 mg · kg^{-1}), while the soils that originate from the formations rich in selenium (tertiary loams) may contain significantly higher quantities of this element (2.3 ÷ 4.2 mg · kg^{-1}). Deficit of this element occurs mainly in certain areas of China, Canada, Denmark, New Zealand and the Nordic countries [Yläranta 1990, Mäkelä-Kurtto and Sippola 2002, Hartikainen 2005]. Borowska [2010] claims that the so-called selenium provinces can be unique in the world. The content of selenium in these regions can even reach 1200 mg · kg^{-1} (e.g., Japan, Canada, USA).

This element as a component of selenoproteins plays an important structural and enzymatic function. According to Flohe et al. [2000] and Rayman [2000], the main selenoproteins include glutathione peroxidase, iodothyronine deiodinase, selenoproteins P and W and thioredoxin reductase. Selenium is an inactivator of toxic heavy metals [Rayman 2000], neutralizes the effect of aflatoxins [Navarro-Alarcon et al. 1998, Maehira et al. 2002], causes apoptosis in tumor cells [Griffin 1982, Behne et al. 1996], prevents or delays the cell aging by protecting the mitochondrial membranes [Wesołowski and Ulewicz 2000], protects the cell lipids from damaging effects of reactive oxygen species [Musik et al. 2003].

The consequences of selenium deficiency in ruminants

Many authors draw attention to the fact that the concentration of selenium in the body of animals depends primarily on the feed consumed, which enrichment largely depends on the content of this element in the soil [Pilarczyk et al. 2004, 2008 a, b, 2009, 2010, 2011 a, b, c, Sablik et al. 2011, Seremak et al. 2011, Flueck et al. 2012, Kawecka et al. 2013, Nowakowska 2013] (Table 1). According to Mayland [1994], and Kabat-Pendias and Pendias [1999], the content of selenium in the feed below 0.010 mg · kg^{-1} d.w. can cause deficiency symptoms in animals.

Selenium deficiency in cattle and sheep can cause nutritional muscular dystrophy (white muscle disease), which manifests in the stiffness of the limbs, difficulty in grasping the food and swallowing, aspiration pneumonia and myoglobinuria [Rederstorff et al. 2006, McCann and Ames 2011]. Selenium deficiency in cattle may also cause diarrhea in young animals and the increase in cirrhosis cases in beef cattle. In cows, placenta retention may occur after delivery, inflammation of the endometrium, fetal death due to their impaired development, extension of the calving and parturitional intervals, and lower breeding performance of bulls [Hulland 1985, Bostedt and Schramel 1990, Zust et al. 1996, Radostits et al. 2000, Suttle 2010].

Table 1. Selected Se content in ruminants from different regions

Tabela 1. Wybrane stężenia Se u przeżuwaczy z różnych regionów

Location Rejon	Tissue Tkanka	Se content Stężenie Se	Reference Piśmiennictwo
		Cows – Krowy	
Ukraine – Ukraina	serum – surowica	0.085 μg · ml^{-1}	Pilarczyk et al. [2005]
Poland – Polska	serum – surowica	0.034 μg · ml^{-1}	Pilarczyk et al. [2005]
		0.074 μg · ml^{-1}	Sablik et al. [2011]
		0.020 μg · ml^{-1}	Pilarczyk et al. [2013 c]
		233.7–237.6 nmol · dm^{-3}	Bombik et al. [2010]
Norway – Norwegia	blood – krew	0.090 μg · ml^{-1}	Kommisrud et al. [2005]
Czech Republic – Czechy	plasma – osocze	0.031 μg · ml^{-1}	Slavík et al. [2007]
Czech Republic – Czechy	blood – krew	0.038–0.095 μg · ml^{-1}	Pavlata et al. [2002]
USA	serum – surowica	0.038–0.077 μg · ml^{-1}	Segerson et al. [1981]
Slovenia – Słowenia	serum – surowica	0.014–0.017 μg · ml^{-1}	Zust et al. [1996]
Romania – Romunia	blood – krew	0.026 μg · ml^{-1}	Pop et al. [2011]
Romania – Romunia	serum – surowica	0.011 μg · ml^{-1}	Pârvu et al. [2003]
	hair – włosy	0.140 μg · g^{-1}	
Columbia – Kolumbia	serum – surowica	0.052–0.097 μg · ml^{-1}	Fenimore et al. [1983]
		Water buffalos – Bawoły rzeczne	
India – Indie	serum – surowica	0.190 μg · ml^{-1}	Prasad and Arora [1991]
Poland – Polska		0.021 μg · ml^{-1}	Sablik et al. [2011]
Pakistan – Pakistan		0.100 μg · ml^{-1}	Akhtar et al. [2009]
		Sheeps – Owce	
Ukraine – Ukraina	serum – surowica	0.485 μg · ml^{-1}	Pilarczyk et al. [2005]
Poland – Polska	serum – surowica	3.28^{-1}0.23 mmol · l^{-1}	Pięta and Patkowski [2007]
	wool – wełna	6.51^{-1}4.24 mmol · l^{-1}	
Poland – Polska	serum – surowica	0.031 μg · ml^{-1}	Pilarczyk et al. [2005]
Poland – Polska	liver – wątroba	3.62 μg · g^{-1} w.w.	Słupczyńska et al. [2009]
	kidneys – nerki	1.59 μg · g^{-1} w.w.	
	muscle – mięśień	0.04 μg · g^{-1} w.w.	
Iran – Iran	blood – krew	0.028 μg · l^{-1}	Karimi-Poor et al. [2011]
USA	blood – krew	0.090 μg · ml^{-1}	Coggins [2006]
USA	liver – wątroba	0.173 μg · g^{-1} w.w.	Cox [2006]
Turkey – Turcja	serum – surowica	0.304 μg · ml^{-1}	Pamukcu et al. [2001]
German – Niemcy	serum – surowica	0.045 μg · ml^{-1}	Humann-Ziehank et al. [2013]
		Goats – Kozy	
Poland – Polska	liver – wątroba	0.047 μg · g^{-1} w.w.	Pilarczyk et al. [2013 b]
	muscle – mięsień	0.027 μg · g^{-1} w.w.	
	kidneys – nerki	0.537 μg · g^{-1} w.w.	
	lungs – płuca	0.056 μg · g^{-1} w.w.	
	heart – serce	0.035 μg · g^{-1} w.w.	
Republic of Burundi Republika Burundi	kidney – nerka	1.194 μg · g^{-1} w.w.	Benemariya et al. [1993]
	liver – wątroba	0.427 μg · g^{-1} w.w.	
Czech Republic – Czechy	blood – krew	0.068 μg · ml^{-1}	Pechowa et al. [2012]
Croatia – Chorwacja	blood – krew	0.019–0.036 μg · ml^{-1}	Antunović et al. [2013]
Mexico – Meksyk	serum – surowica	0.020–0.021 μg · ml^{-1}	Ramírez-Bribiesca et al. [2001]
USA	serum – surowica	0.010 μg · ml^{-1}	Harris [1987]
USA	blood – krew	0.076–0.499 μg · ml^{-1}	McComb et al. [2010]
France – Francja	serum – surowica	0.021 μg · ml^{-1}	Morand-Fehr [1981]

A decrease in the efficiency of the antioxidant system is observed in cows at high milk yields, particularly when combined with selenium deficiency [Flohe et al. 2000, Gladyshev 2001, Jankowiak et al. 2015]. Therefore, it is important to adequately supply females in selenium, which determines the efficacy of the antioxidant system [Castillo et al. 2006, Pilarczyk et al. 2012]. Selenium deficiency during high lactation increases the risk of mastitis [Villar et al. 2002].

Myocardial damage may occur in calves as a result of selenium and vitamin E deficiency. Three-month-old animals and younger are affected most frequently. Symptoms of deficiency of this element include: arrhythmia, murmurs, shortness of breath, congestive heart failure, which in turn may lead to sudden death [Rederstorff et al. 2006, McCann and Ames 2011].

Selenium deficiencies in sheep in New Zealand and Australia were the cause of 20–50% of infertility in females and also an increased lamb loss was observed [Suttle 2010, Ren et al. 2011]. These authors showed that Se deficiency also negatively affected the rams (the number of sperm in the ejaculate, sperm concentration and motility). Fetal death may also occur in pregnant ewes as well as placenta retention after birth [Ren et al. 2011].

The consequences of excess selenium in ruminants

Excess of selenium causes selenosis, a disease which is characterized by muscle degeneration, heavy breathing, roughness and matt hair, cardiovascular disorders [Ramadan et al. 2001, Deore et al. 2002, Terzano et al. 2005]. Poisoning associated with the intake of excess selenium in ruminant farming is occasional. There were no such cases reported in Poland and Ukraine. Selenium lethal dose (LD_{50}) in adult cattle is 0.501 mg $\cdot$ kg^{-1} body weight, while in lambs it is 0.455 mg $\cdot$ kg^{-1} [Grace 1994, Tinggi 2005]. Blood selenium levels of >2 mg $\cdot$ l^{-1} in cattle and >0.6–0.7 mg $\cdot$ l^{-1} in sheep are associated with selenosis with borderline toxicity at 1–2 mg $\cdot$ l^{-1} in cattle [Levander 1986, WHO 1987].

Standards of selenium supply in ruminants

Selenium serum or blood level can be an indicator of the supply of this element in ruminants (intravitally) as well as its content in the liver and kidneys. Puls [1994] points that the concentration of selenium in the liver is a better indicator of selenium level than the kidney test. Oh et al. [1976] demonstrated that lambs fed with fodder poor in selenium had always a higher concentration of this element in the kidney compared to liver. The opposite situation was observed when a feed rich in this element was supplied to lambs (higher concentration of selenium in

the liver than in the kidneys). Mahan and Kim [1996] believe that the assessment of selenium status of the organism should involve blood or serum tests.

The most commonly used criterion for assessing the selenium status in livestock is the serum or blood content. According to Grace [1997], biochemical standards used in the diagnosis of serum selenium deficit in ruminants are as follows: less than 0.041 g · ml^{-1} – deficiency; 0.041–0.079 g · ml^{-1} – marginal level; above 0.079 g · ml^{-1} – physiological level. According to Smith et al. [1998], the following biochemical standards should be used in the diagnosis of whole blood selenium deficit in ruminants: less than 0.14 g · ml^{-1} – deficiency; 0.14–0.20 g · ml^{-1} – marginal level; above 0.20 g · ml^{-1} – physiological level. According to Puls [1994], biochemical standards used in the diagnosis of selenium deficit in the liver of ruminants should be as follows: less than 0.1 g · ml^{-1} of the above – deficiency; 0.1–0.24 g · ml^{-1} – marginal level; 0.25–1.2 g · ml^{-1} – optimal level.

Selenium supplementation in ruminants

The results shown in Table 1 confirm that in some geographical areas there is a deficiency of selenium in ruminants. At a low concentration of this element in the serum/blood of ruminants, its supplementation is necessary. Selenium may be supplemented in the inorganic or organic form. European Union permits supplementation of ruminant feed additives with sodium selenite, sodium selenate and selenium yeast *Saccharomyces cerevisiae*. Sodium selenite is most frequently applied in the feed industry. Selenium yeast are a source of organic selenium (mainly selenomethionine).

The bioavailability of selenium depends on the species of animals. For pigs, Se is available in both: organic and inorganic compounds. It was found that in pigs 77% of Se in the form of selenite was resorbed in the gastrointestinal tract, while in sheep it was only about 29% [Grela and Sembratowicz 1997]. Mineral selenium compounds in ruminants are strongly reduced by rumen bacteria to non-assimilable forms. According to Gerloff [1992], the factor that reduces the absorption is low rumen pH, typical for the high-yielding cows that receive high doses of energy feed.

Pilarczyk et al. [2004] found a significant improvement in reproductive indices (fertility – 96%, and fecundity – 137.5%) after application of sodium selenite in ewes. According to the authors, lamb losses in the experimental group were lower (9.2%) than in the control group (12.1%). Similar effects were obtained using selenium yeast [Pilarczyk et al. 2013 a]. Deaths in the group of lambs derived from the ewes fed selenium yeast were significantly lower, body weight of lambs on the day of birth was higher, the number of live-born lambs was also higher compared to the control group. Lambs derived from the mothers that were admi-

nistered selenium yeast had significantly higher body weight at 33 and 90 days of age compared to lambs of the mothers not receiving Se. Reproductive indices (fertility and fecundity) were also improved as a result of the application of selenium yeast in ewes. Segerson et al. [1986] established that the number of lambs born alive from control and Se-treated ewes was 1.61 and 1.81, respectively, while the number of lambs weaned per ewe was 1.1 and 1.5, respectively.

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SELEN W ŻYWIENIU PRZEŻUWACZY

Streszczenie. Liczne wyniki badań wskazują, że na większości obszarów istnieją niedobory selenu w środowisku. Wskaźnikiem zaopatrzenia organizmu w selen może być jego stężenie w surowicy lub krwi (przyżyciowo), jak i również jego zawartość w wątrobie oraz nerkach (pośmiertnie). Najczęściej stosowanym kryterium oceny statusu selenowego u zwierząt gospodarskich jest jego zawartość w surowicy lub krwi. Ważne jest, aby uzupełniać niedobór selenu u zwierząt bytujących w regionach niedoborowych, zwłaszcza u przeżuwaczy, gdyż długotrwały deficyt tego pierwiastka może doprowadzić do licznych schorzeń.

Słowa kluczowe: choroby, przeżuwacze, selen

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