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Review paper

Sodium selenite as a new rodenticide

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SUMMARY

Rodents are the most destructive group of small mammalian pests considering the overall damage that they cause by feeding and other activities, or as vectors of many disease agents. In practice, chemical rodenticides have been the most widespread and most effective method of control of commensal (*Mus musculus*, *Rattus norvegicus* and *R. rattus*) and most harmful field rodent pests (*Apodemus sylvaticus*, *A. agrarius* and *Microtus arvalis*).

After anticoagulant and vitamin D3 rodenticides, which were introduced worldwide in the 1980s, no other chemical compound has had a comparable role as a rodenticide in practice. In the past decade, commercial baits containing 0.1% sodium selenite have also been registered in Serbia in various formulations both for controlling rodents indoors and in the field.

Data on sodium selenite as a rodenticide have been scarce. The present paper surveys research data reported so far, analyzing and drawing conclusions regarding the validity and feasibility of sodium selenite as a method of rodent control with reference to the available ecotoxicological data.

Keywords: Sodium selenite; Rodents; Rodenticides

INTRODUCTION

Considering the amounts of food eaten or polluted, and damage caused to electric installation and devices, rodents make the most harmful group of pests (Sheikher & Jain, 1997; Aplin et al., 2003). Besides the economic losses, which may be redressed and overcome, rodents also pose a considerable threat to people, domestic and wild animals as pools and vectors of various agents of disease, helmints (nematodes, tapeworms and liver flukes), bacteria and viruses (Begon, 2003; Ćirić et al., 2003; Bonnefoy et al., 2008; Kataranovski et al., 2010).

Over the past several decades, chemical control of rodent pests by anticoagulants has been the most widespread and effective control practice (Buckle & Smith, 1994; Hadler & Buckle, 1992; Guidobono et

al., 2010). Ethical principles (Mason & Littin, 2003; Meerburg et al., 2008), environmental awareness (Singleton et al., 1999; Ramsey & Wilson, 2000) and legislation regulating and limiting the use of existing rodenticides (European Commission, 2007) are some of the reasons for redirecting research towards new and environmentally friendly ways to control rodents. Sodium selenite, a product of new research principles and more acceptable from the environmental aspect, has been registered in Serbia as such a rodenticide. Commercial baits that are currently present on the market contain 0.1% sodium selenite and they are classified into the poison group 2 (Janjić & Elezović, 2010).

Even though sodium selenite has been available on the market for over a decade, information about it as a rodenticide is still scarce. Hitherto research has mostly focused on determining the properties of the most frequent organic and inorganic forms of selenium occurring in nature or its synthetic and formulated products. Our own experiments have been conducted to test baits with 0.1% sodium selenite content in the field (Vukša et al., 2009; Jokić et al., 2010; Jokić et al., 2012), in warehouses (Vukša et al., 2007; Đedović et al., 2011; Jokić et al., 2011a; Đedović et al., 2012) and in the laboratory (Vukša et al., 2006; Jokić et al., 2011b; Jacević et al., 2011). The focus of our present research was on the advantages and deficiencies of sodium selenite as a rodenticide based on its ecotoxicological parameters and other properties of baits containing 0.1% sodium selenite. To make a comprehensive overview and valid assessment of sodium selenite as a rodenticide, its properties have been compared to data in source literature for bromadiolone as the most widely used rodenticide in Serbia.

Our conclusions are based on source literature and our own research data, while information on the effects of 0.1% sodium selenite baits on the environment, primarily on nontarget organisms (e.g. predators and vultures), are still lacking. Also, it is not clear whether all animals had been sacrificed according to ethical principles.

SODIUM SELENITE

Selenium, a naturally occurring dietary trace element can be found in water, air, rocks, plants and food (Adriano, 1986; Cutter, 1989; Johnsson, 1991). As a solid, sodium selenite has a molecular weight of 172.9 and is highly soluble in water (85 g/100 g water at 20° C) (Anonymous, 2009). Solubility of selenium compounds has a crucial role in their toxicity. The highly soluble sodium selenite has demonstrated up to 900-fold higher acute toxicity than the insoluble element selenium (Cummins & Kimura, 1971).

Information about the effects of sodium selenite on the environment is scarce. Sodium selenite is a salt, and one of the most common water-soluble selenium compounds. As its LC_{50}/96 h for fish is less than 10 mg/L, sodium selenite may be assumed to be potentially toxic or highly toxic to organisms living in water. Even though it is thermostable, sodium selenite is recommended not to be exposed to flame (Anonymous, 2009). It is one of the most widespread forms of naturally-occurring selenium (Kamal, 1994; Anonymous, 2003). It is used as a food additive for animals to promote their growth and prevent diseases (Kamal, 1994). According to Anonymous (2003), human organism discharges most of consumed selenium within the next 24 h. At low concentrations, selenium is a trace element beneficial to humans and animals, while its higher concentrations are highly toxic (Rayman, 2000), and symptoms of human poisoning include a garlic-like bad breath, stomach ache, hypersalivation, muscle contraction, paralysis, coma and death (Clark et al., 1996; Vinceti et al., 2001; See et al., 2006).

Selenium toxicity is believed to be based on selenium trisulfide exchange at the active sites in enzymes (Frenkel & Falvey, 1988, 1989), and it becomes especially evident when selenium is consumed with food. In plants and animals, selenium is primarily localized in the protein fraction (Ferretti & Levandar, 1976).

Oral intakes of high sodium selenite doses (at least 8 times greater than those normally supplied in adequate diet) cause negative effects on reproductive organs. Such effects include an increased number of abnormal sperms, testicular hypertrophy, degeneration and atrophy in male rats (Turan et al., 1999) and impact on estrous cycles in female rats and mice (Anonymous, 2003; Anonymous, 2009). Analyzing hepatocytes in rats, selenite presence in organisms has been found to affect amino acid cycles (Anundi et al., 1982; Hogberg & Kristoferson, 1979). Selenium consumed orally becomes almost fully digested, i.e. by 95-100%. The absorption process is not homeostatic-controlled and no difference has been observed in the absorption processes in rats suffering from selenium deficiency or overdose after mean toxic doses were applied (Brown et al., 1972). Most of the selenium entering a body is quickly discharged, normally within 24 h (Anonymous, 2003).

The acute oral LD_{50} of sodium selenite is 9.79 mg/kg for the Swiss Webster strain of laboratory mouse and 11.20 mg/kg for Wistar laboratory rat (Jacević et al., 2011).

Pletnikova (1970) and Henschler and Kirschner (1969) reported the acute oral LD_{50} of sodium selenite for laboratory mice to be 7 and 48 mg/kg, respectively.

The sodium selenite LD_{50} for rats has been determined as 3, 5.5 and 7 mg/kg, depending on whether its administration is respectively intravenous, dermal or oral (Morss & Olcott, 1967; Cummins & Kimura, 1971). Oral application has been reported to cause high susceptibility to sodium selenite in rabbits with the LD_{50} of 2 mg/kg (Anonymus, 2009). Minimum lethal doses of sodium selenite, expressed as mg selenium/kg body weight, reported for larger animals are 13-18 mg/kg for pigs and 9.9-11.0 for cows (Miller & Williams 1940).

The water-soluble sodium selenite is a commercial product intended for soil enrichment with selenium (Anonymous, 2003). Rayman (2000) detected a direct link between selenium levels in plants and soil. In regions with high selenium levels in soil, cases of human intoxication have been reported after consumption of plant food with high selenium contents. On the other hand, human diseases (e.g. Keshan disease) caused by selenium deficiency in food have been reported in areas with selenium deficiency in soil (Yang et al., 1983).

The selenium from selenite sources is metabolized by reduction and methylation. Liver erythrocytes have a primary role in selenium metabolic processes, i.e. transformation of inorganic to organic forms of selenium, and synthesis of discharged metabolites (Beilstein & Whanger, 1986a,b; Ganther et al., 1966; Diplock et al., 1973). In their experiments on rats, Kato et al. (1992) confirmed the dominant role of the liver in ^{75}Se-selenite metabolism. In that context, the liver is also one of the first organs that are affected by selenium toxicity (Levander, 1986). According to Jaćević et al., (2006), oral intakes of sodium selenite may cause cardiotoxic effects in laboratory mice. Cardiac damage has also been observed in mice exposed for 12 weeks to 0.2 mg selenium/kg/day in food containing sodium selenite (Skowerski et al. 1997). Ultrastructural examination revealed cardiomyocytes that had numerous damaged mitochondria, a large number of lipid droplets, and numerous lysosomes.

Selenium is discharged in urine, feces and expired air, but urinary excretion is considered the primary route (Kamal, 1994).

EFFICACY OF SODIUM SELENITE PRODUCTS IN THE LABORATORY

The efficacy of commercial products containing 0.1 % sodium selenite has been tested in the laboratory on animals from wild populations of the house mouse *Mus musculus*, brown rat *Rattus norvegicus* and black rat *R. rattus* (Vukša et al., 2006; Jokić et al., 2011b), and the laboratory mouse and Wistar rat (Jaćević et al., 2006; Jacevic et al., 2011). The properties and efficacy of baits containing 0.1% technical grade concentration of sodium selenite have been examined in experiments with laboratory mice (Swiss Webster strain) and common voles (Jokić, 2012). Bioassays were conducted with and without food, according to EPPO's standard methodology (2004b) and OECD/OCDE standard (2001), over the period 2005-2011.

Wild house mice deaths occurred over 9-14 days, brown rats died for 5-11 days, and black rats for 6-14 days. There was no difference in susceptibility between the sexes.

Laboratory mice and common voles started to die 1-5 and 3-9 days, respectively, after ingesting baits with 0.1 % technical concentrate of sodium selenite in a no-choice bioassay. Common voles exposed to the same laboratory conditions died 4-12 days after ingesting 0.005% bromadiolone baits. The lowest lethal doses of sodium selenite for laboratory mice and common voles were 32.9, and 13.3 mg/kg body weight, respectively. The lowest lethal dose of bromadiolone against common voles was up to 3.5-fold lower than the sodium selenite lethal dose, i.e. 3.8 mg/kg body weight. Experimental data also revealed a high toxicity of sodium selenite. Mortality of common voles was caused by four-fold lower doses of 0.1 % sodium selenite baits than 0.005% bromadiolone. Different LD_{50} values of sodium selenite against laboratory mice were detected depending on whether the active ingredient was applied by bait ingestion or by gavage. The LD_{50} recorded after application by gavage was more than three-fold lower than the lethal dose recorded after bait ingestion. Compared to the oral LD_{50} of bromadiolone, which was 1.75mg/kg, the oral LD_{50} of sodium selenite was up to six-fold higher against laboratory mice.

The gender of examined rodents was not found to have effect on their susceptibility to baits based on sodium selenite and bromadiolone. Data from that study also showed a positive correlation between the survival time and dose. Considering the speed of human metabolism and discharge of selenium, we may assume that the metabolic processes of organic forms of selenium in an

organism, i.e. the speed of its metabolism, may affect product efficacy and animal survival.

In a choice feeding bioassay, the acceptability and palatability of attractant-free baits containing 0.1% sodium selenite (technical grade concentrate) were significantly lower than those of baits containing 0.005% bromadiolone against laboratory mice and common voles. So far, no criterion has been formulated for classifying the acceptability and palatability of rodenticides with mechanisms of activity other than the mechanism of anticoagulants. According to Schmolz (2011), acceptability of any anticoagulant product exceeding 25% should be considered appropriate and sufficient to expect such bait to be successful in rodent control. As the mechanisms and speed of activity of sodium selenite and bromadiolone are different, it is not possible to assess the acceptability of sodium selenite baits using that criterion. Anyhow, as successful rodent control largely depends on bait acceptability and palatability (Buckle & Smith, 1994), the present data show that the efficacy of baits containing 0.1% sodium selenite depends on the choice of additives, i.e. attractants.

SODIUM SELENITE EFFICACY IN THE FIELD

Applying standard EPPO (2004a) methodology, trials were conducted to test the efficacy of several formulations of commercial products with 0.1% sodium selenite and 0.005% bromadiolone in the field against field rodents (Vukša et al., 2009; Jokić et al., 2010; Jokić et al., 2012). Common voles and striped field mice were found predominating, depending on locality, plant crop and its growth stage, and evaluation that was conducted either by trapping or by visual observation of characteristic signs. The average sodium selenite efficacy of 56-86% in controlling the present species of small rodents was similar or statistically significantly lower than the average 78-89% efficacy recorded by baits with 0.005% bromadiolone.

Baits containing sodium selenite showed an efficacy that was below 50% or twice as low in several cases as the efficacy of bromadiolone baits (unpublished data). Based on data from field trials, it is not possible to determine whether the time of year, type of crop or its growth stage have any effect on bait efficacy. Weather conditions, precipitation in particular, are known to affect bait efficacy (Buckle & Smith, 1994). As sodium selenite is readily soluble in water, rainfall can be expected to cause losses of that active ingredient in baits and its leaching into soil. Paraffinized baits are the most frequent and economical form of products that retain compactness, increase resistance to moisture and prevent degradation and leaching. However, added paraffin reduces bait acceptability and palatability (Buckle & Smith, 1994). As sodium selenite was found to have low acceptability and palatability in the laboratory, a new formulation based on encapsulation may be expected to increase and uniform its efficacy in the field.

SODIUM SELENITE EFFICACY IN PROTECTED ENVIRONMENTS

Over a period of eight years, different formulations of commercial products containing 0.1 % sodium selenite and 0.005% bromadiolone have been experimentally tested in mills, mixing and storage facilities (Vukša et al., 2007; Đedović et al., 2011; Jokić et al., 2011a; Đedović et al., 2012) in order to determine their efficacy in controlling commensal rodents.

Wild populations of house mouse showed uniform susceptibility to sodium selenite. Nominally, its efficacy of 82-98% was not significantly different from 71-97% bromadiolone efficacy in our earlier trials or 60-100% efficacy reported by Rowe et al. (1981). Compared to bromadiolone, whose average 86-95% efficacy was uniform, the efficacy of sodium selenite in controlling wild populations of brown rat was lower and mostly nonuniform, ranging from 61% to 91%. On many trial sites, sodium selenite baits showed low efficacy against commensal rodents. Other accessable good-quality food sources caused a reduced intake of sodium selenite baits, which eventually led to low bait efficacy.

PATHOMORPHOLOGICAL CHANGES IN TISSUES AND ORGANS

As part of our research, a pathomorphological study was conducted on tissues and organs of mice (Swiss strain) and rats (Wistar strain) fed for three days on baits with 0.009% sodium selenite, and diffuse bleeding was detected in the pericardium, along the alimentary tract and in subcutaneous tissues and muscles. Microscopic visualization revealed characteristic signs on kidneys, livers and spleens. Kidneys and spleens of all animals were specked with

numerous bleeding spots, while liver surfaces had mosaic pattern, decaying texture and wrinkled edges. Bladder blood vessels were injected and the urine had dark brown colour. Other organs had normal appearance (unpublished data).

Two-days of oral ingestion of baits with 0.009% sodium selenite induced prominent pathohistological changes in rodent hearts, especially in male rats and mice (Jaćević et al., 2006).

The administration of different single doses of sodium selenite (4, 10, 14 and 18 mg/kg) caused severe, diffuse and massive degenerative and vascular changes in rats and mice of both genders (Jacevic et al., 2011). The affected cardiomyocytes had extensive sarcoplasmatic vacuolisation or pale eosinophilic sarcoplasm and lacked cross-striation. In these irregular, round to ovoid cells, nuclear polymorphism was present with large, round to rectangular shapes and prominent nucleoli. Affected areas were observed in the subepicardium, myocardium and endocardium. Blood vessel thickening, as well as vacuolisation of endothelial cells, were observed, too. Interstitial haemorrhages appeared uniformly in each of the sections examined. It was located either in the middle myocardial or in the subendocardial area.

Increasing doses of sodium selenite (4, 10, 14 and 18 mg/kg) were applied to separate groups of mice and rats by the *po* route. We noticed prominent histopathological changes in the heart, liver, spleen and kidneys. Histopathological findings revealed signs of inflammation, haemorrhage, degeneration and rapid loss of normal cell architecture in all examined tissues of sodium selenite-treated animals.

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Natrijum selenit kao novi rodenticid

REZIME

U odnosu na ukupnu štetnost koju nanose ishranom i drugim aktivnostima, ali i kao prenosioci većeg broja prouzrokovača različitih obolenja, glodari spadaju u najštetnije vrste sitnih sisara. U praktičnim uslovima primene, rodenticidi hemijskog porekla predstavljali su najprimenljiviji i najefektniji način suzbijanja štetnih komensalnih (*Mus musculus*, *Rattus norvegicus* and *R. rattus*) kao i najštetnijih vrsta poljskih glodara (*Apodemus sylvaticus*, *A. agrarius*, *Microtus arvalis*).

U odnosu na antikoagulantne rodenticide i vitamin D3, koji su uvedeni u primenu do 80-ih godina prošlog veka, u svetu, u praktičnim uslovima primene, ni jedno novo jedinjenje nije zauzelo značajniju ulogu kao rodenticid. Proteklih desetak godina u našoj zemlji registrovani su i komercijalno dostupni mamci sa sadržajem 0.1% natrijum selenita, različitih formulacija, kao rodenticidi za suzbijanje glodara u zaštićenim i uslovima otvorenog polja.

Za sada je malo dostupnih podataka o natrijum selenitu kao rodenticidu. Cilj ovog rada je predstavljanje rezultata dosadašnjih istraživanja, njihova analiza i donošenje zaključaka o mogućnosti i opravdanosti primene natrijum selenita za suzbijanje glodara, sa osvrtom na dostupne ekotoksikološke podatke.

Ključne reči: Natrijum selenit; glodari; rodenticidi