Arch. Biol. Sci., Belgrade, 66 (3), 1083-1089, 2014 DOI:10.2298/ABS1403083J

RODENTICIDE EFFICACY OF SODIUM SELENITE BAITS IN LABORATORY CONDITIONS

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Abstract – We examined the acceptance and palatability of baits containing different contents of sodium selenite as a rodenticide, in Swiss mice under laboratory conditions. In a no-choice and choice feeding test, the animals were exposed to baits containing 0.1, 0.05, 0.025 and 0.0125% of sodium selenite. The total bait consumption by Swiss mice in the no-choice feeding test was highly negatively correlated, while total sodium selenite intake was medium-positively correlated to the sodium selenite content in the bait. In the same test, daily intakes significantly depended on the content of sodium selenite in the bait, while the exposure and associated interactions of contents of sodium selenite and exposure had no statistically significant impact. Baits with sodium selenite contents of 0.05 and 0.1% had the most lethal effects. The negative impact of the sodium selenite content on bait acceptance and palatability was confirmed in choice feeding tests. Baits containing 0.05 and 0.1% of sodium selenite displayed the biological potential to be used as a rodenticide. It is necessary to improve its insufficient acceptability and palatability by adding adequate additives to the bait. The results of this study should be verified in experiments with wild rodents.

Key words: Sodium selenite; Swiss mice; acceptability; palatability

INTRODUCTION

In the middle of the last century, the introduction of first-generation anticoagulants significantly improved rodent pest management programs (Hadler and Buckle, 1992). However, their wide use induced resistance development in commensal rodent species, particularly in Western Europe (Rowe and Redfern, 1965) and the USA (Jackson and Kaukeinen, 1972). After the introduction of the use of more potent second generation anticoagulant compounds at the end of the eighties in the last century, it was hoped that the problem of resistance of commensal rodent species will be overcome (Hadler and Shadbolt, 1975; Hadler and Buckle, 1992), however, resistance to most second generation anticoagulant compounds was reported soon after their introduction (Rowe et al., 1981; Greaves et al., 1982; Johnson, 1988).

Rodent species continue to cause considerable damage in agriculture, forestry, animal husbandry and public health (Wood, 1994; Pelz and Klemann, 2004). Here, we discuss another new approach: the use of sodium selenite bait to improve rodent pest management programs.

The biological importance of selenium is reflected in the fact that on the one hand it is essential for humans and animals, and on the other toxic at high concentrations (Rayman, 2000). Selenium is widely distributed in various forms in soils, water, air, vegetation and foods (Adriano, 1986; Johnsson, 1991), in which it very often occurs in the form of sodium selenite (Gerberding, 2003). Sodium selenite is used as a poultry- and livestock-feed supplement, to promote growth and prevent selenium-deficiency diseases (Kamal, 1994). Selenium from orally administered sodium selenite is efficiently absorbed from the gastrointestinal tract (Brown et al., 1972; Gerberding, 2003). The absorption does not appear to be homeostatically controlled, as no difference in absorption was observed between selenium-deficient and selenium-sufficient rats administered with mildly toxic doses of selenium (Brown et al., 1972). The toxic effects of selenite involve the formation of selenotrisulfides in the active sites of enzymes (Frenkel and Falvey, 1988, 1989). Signs of acute selenium toxicity in humans are garlicky or sour breath odor, gastrointestinal disturbances, restlessness, hypersalivation, muscle spasms, hemolysis, liver necrosis, cerebral and pulmonary edema, coma and death (Clark et al., 1996; Vinceti et al., 2001).

Rodenticides based on sodium selenite have been registered in Serbia for rodent control in open-field or storage conditions. Despite being commercially available, however, their acceptance and palatability are not known. The present study was designed to assess the potential of bait with different contents of sodium selenite as a rodenticide, through the determination of lethal effect, acceptability and palatability in laboratory conditions for Swiss mice.

MATERIALS AND METHODS

Animals

Laboratory tests were conducted from October to November, 2010, in the Pesticides and Environmental Protection Research Institute, Applied Zoology Laboratory, Belgrade, Serbia. Experiments on animals were conducted in accordance with ethical principles and approved by the Council of Biotechnical Sciences, University in Belgrade, Serbia, and were in adherence to the guidelines of the Ethical Committee of the Institute for Biological Research "Siniša Stanković", Belgrade, Serbia.

Adult male and female Swiss mice (Institute for Medical Research, Military Medical Academy, Belgrade, Serbia), weighing from 20 to 25 g were used. The animals were housed individually in plastic cages (320x200x135mm) under standard laboratory conditions: 21-24°C, 12 h light/dark cycle, 45-70% relative humidity, water available *ad libitum*. The weights of the Swiss mice did not vary significantly between the treatment groups in the no-choice ($F_{3,92} = 0.62$; $P = 0.60$) and choice feeding tests ($F_{3,76} = 0.29$; $P = 0.83$).

Rodenticides

Lethal effects, acceptability and palatability of baits containing 0.1; 0.05; 0.025 and 0.0125% of sodium selenite (anhydrous, pure min. 99%), supplied by Alfa Aesar, France, were determined for Swiss mice under laboratory conditions. Plain baits were prepared by mixing coarse ground grains (wheat:barley:corn = 30:40:30), wholegrain wheat flour and corn oil (cholesterol free) in the ratio 90:5:5. Baits were made by applying the appropriate amount of sodium selenite to previously prepared plain bait. The test of active ingredient content in the prepared samples was performed in the laboratories of the City Institute for Public Health, Belgrade, Serbia.

Rodenticide baiting tests

In the pre-test period, the animals were given untreated food for laboratory mice, produced by the Veterinary Institute Subotica, Serbia. At the beginning of each assay, the individual weight of the mice was measured. During the trial, ordinary food was removed from the cages. The daily amount of bait eaten by each animal was recorded. After completion of the exposure period, treated bait was removed and control bait was provided for the recovery period. At all times during the treatment, animals were caged individually.

The lethal effects of baits with different sodium selenite contents on Swiss mice were determined in a no-choice feeding test, according to EPPO standards (2004). Six animals (three females and three males) were used in each assay. Animals were exposed for 24 h to bait containing 0.1, 0.05, 0.025 and 0.0125% of sodium selenite. Since complete mortality could not be achieved at any concentration after 24 h exposure, the exposure periods were adjusted to 24, 48, 72 and 96 h.

Bait acceptance and palatability were determined according to Johnson and Prescot (1994), in a choice feeding test. Twenty animals (ten females and ten males) were used in each assay. Over a 96 h period, in bowls placed on the opposite sides of the cage, individuals were offered toxic and non-toxic (plain) bait. After each measurement of the bait, the location of the bowls was switched.

Histopathological examination

From dead mice, the heart, liver, spleen and kidneys were excised and tissue samples were fixed in 10% neutral formalin for 5 days. Transmural tissue samples were dehydrated in graded alcohol, xylol and embedded in paraffin blocks. Finally, 2 μm thick paraffin sections were stained with hematoxylin and eosin (H&E) and analyzed using an Olympus-2 microscope (Tokyo, Japan).

Computations and statistical analyses

One-way analysis of variance (ANOVA) was used to compare the average weights of Swiss mice among each group in no-choice and choice feeding tests.

To analyze the results of the no-choice feeding experiment, a linear regression test was used to compare relationships between consumption as a dependent, and treatment level as an independent variable. Two-way ANOVA was used to compare the effects of active ingredients and exposure as independent variables to the daily-consumed amounts of bait. The resulting data (daily consumption) were transformed using sqrt(x+1) before analysis. Means for daily consumptions were separated using Tukey's test. Individuals that died before the end of the exposure period were not included in the analysis. Survival analysis of the tested mice was done by Kaplan-Meier estimator. A log-rank test was used to compare differences between the groups. The data were processed by the Stat for Windows, R.4.5. software package.

Bait acceptance and palatability ratio were calculated as described by Johnson and Prescott (1994).

RESULTS

Bait consumption was highly correlated with the content of sodium selenite in the bait. The correlation factor was the lowest for the exposure period of 72 h (r = - 0.61), and the highest for the exposure of 24 h (r = - 0.77). Total sodium selenite intake was weakly correlated with the contents of sodium selenite in the bait at the exposure of 24 and 48 h (r = 0.26) and 72 h (r = 0.30). For the exposure of 96 h, a medium correlation factor was calculated, r = 0.39. The sodium selenite content in bait vs daily consumption in the no-choice feeding test was significant ($F_{3.80} = 87.96$; $P < 0.05$), while exposure and associated interactions of sodium selenite contents vs exposure were not significant ($P > 0.05$).

The lowest average daily bait intake was recorded in the treatments with 0.1% of sodium selenite for 24 and 48 h exposures, contrary to the treatment with 0.0125% of sodium selenite, for all periods, where the highest average daily bait intake was recorded (Table 1). Compared to other treatments, where no statistically significant differences were recorded, a higher bait consumption was recorded in the treatment with 0.025% of sodium selenite for the 24 h exposure.

A minimum lethal dose of 30.0 mg/kg was recorded for the treatment with 0.025% of sodium selenite for the 48 h exposure, while the average maximum lethal dose was noted in the treatment with 0.1% sodium selenite for the 72 h exposure with 179.5 mg/kg.

Table 1. Daily consumption, mortality and mean lethal and nonlethal doses of sodium selenite baits under different times of exposure in no-choice feeding tests (means[1] followed by the same letter are not significantly different; Tukey test at 0.05, df = 9.80)

Concentration of a.i. in bait (%)	Exposure (h)	Mean daily consumption (g a.i./100g b.w.)		Total mortality (n=6)	Lethal dose (mg a.i. kg^{-1} b.w.)		Non-lethal dose (mg a.i. kg^{-1} b.w.)		Days to death	
		Means	SE		Means	SE	Means	SE	Means	Range
0.1	24	2.44 ab	0.29	2/6	32.94	2.77	20.23	1.62	2.5	1-4
	48	2.48 a	0.82	2/6	90.21	32.59	29.52	9.24	2.0	-
	72	4.56 abc	1.88	4/6	179.54	46.46	33.36	4.91	3.0	2-4
	96	6.51 abc	2.19	6/6	139.59	37.00	-	-	3.2	1-5
0.05	24	5.86 abc	0.95	2/6	41.57	2.48	23.15	4.18	2.5	2-3
	48	4.35 abc	0.79	3/6	48.68	2.55	38.37	16.84	4.0	3-5
	72	4.13 abc	0.52	4/6	72.39	6.27	41.28	6.01	4.0	3-5
	96	4.47 abc	0.77	4/6	103.06	19.52	62.49	13.11	4.0	-
0.025	24	7.28 c	0.83	0/6	-	-	18.19	2.07	-	-
	48	5.44 abc	0.22	1/6	30.00	-	26.62	1.13	5.0	-
	72	6.67 bc	0.96	3/6	64.10	7.58	35.98	2.34	4.66	3-6
	96	5.81 abc	0.89	3/6	66.94	16.24	49.33	7.71	4.33	4-5
0.0125	24	16.44 d	1.36	0/6	-	-	20.55	1.70	-	-
	48	16.37 d	0.85	0/6	-	-	40.93	2.12	-	-
	72	14.95 d	0.79	1/6	51.53	-	56.98	3.48	4.0	-
	96	13.81 d	0.78	3/6	69.40	6.28	68.72	6.13	5.66	5-7

[1] Untransformed means and standard errors are shown in the tables

The death of animals after consumption of bait with different sodium selenite contents occurred in the period of one to seven days from the beginning of the feeding. The shortest period from sodium selenite intake to death was noted in the feeding with 0.1% of sodium selenite, for 24 h and 96 h exposures, while the longest period was observed in the treatment with 0.0125% of sodium selenite for the 96 h exposure. Most of dead animals (31/38) did not show visible symptoms of illness before death. In other dead animals (7/38), the first visible poisoning symptoms in the form of tremor and feeding disruption were observed 3-12 h before death. Two individuals (male and female) that showed poisoning symptoms in the form of tremor and short-term feeding disruption recovered after consuming 38.3 and 38.6 mg a.i. per kg b.w. of the toxicant, respectively.

Pathomorphological examination of all treated animals showed an increase in thoracal and abdominal cavity fluids. The surfaces of the examined organs (heart, liver, spleen and kidneys) had a normal color. On the other hand, studious histopathological examination of these tissues showed that the ingestion of a lethal dose of sodium selenite caused severe, diffuse and massive degenerative or necrotic and vascular

Table 2. Bait acceptance and palatability with different concentrations of sodium selenite in baits during 96 h of exposure in choice feeding tests with Swiss mice

Content of a.i.[1] (%)	Bait acceptance[2] (%)	Palatability[3]	Total mortality (n=20)	Days to death	
				Mean	Range
0.1	13.11	0.15	5/20	4.0	2-6
0.05	14.08	0.16	2/20	5.0	-
0.025	21.39	0.27	0/20	-	-
0.0125	40.45	0.68	0/20	-	-

[1] Content of sodium selenite in baits;
[2,3] Bait acceptance and palatability of sodium selenite-treated baits calculated according Johnson and Prescott (1994) formula.

alterations in all treated animals. These irreversible tissue-damage symptoms appeared uniformly in each of the examined sections, and were located in the middle areas. The illness caused by the sodium selenite appeared to be independent of the treatment concentration.

The analysis of Swiss mice survival in the no-choice feeding test indicates that the recorded lethal effects depend on treatment level (content of a. i. in the bait).

Survival analysis using a log-rank test revealed that the lethal effects caused by feeding with bait containing 0.05 and 0.1% of sodium selenite do not significantly differ ($P = 0.4659$). In addition, no statistically significant difference was observed between the lethal effects of consuming bait containing 0.0125 and 0.025% of sodium selenite ($P = 0.2811$).

The highest mortality rate of Swiss mice (100.0%) was recorded in the treatment with 0.1% of sodium selenite for the 96 h exposure, while in the treatment with 0.025% of sodium selenite for the 24 h exposure, as well as for treatments with 0.0125% of sodium selenite for the 24 h and 48 h exposures, no mortality was recorded.

In choice feeding tests, the increase in the active ingredient (sodium selenite) concentration decreased the acceptability and palatability of bait by the Swiss mice.

DISCUSSION

In accordance with recommendations on the protection of experimental animals (Meerburg et al., 2008; Kostomitsopoulos and Đurašević, 2010), our studies were performed with a minimum number of animals and concentration of sodium selenite. Because of this, it was not possible to calculate the LD_{50} of sodium selenite for the laboratory mouse, but it was possible to determine the lethal effect, bait acceptance and palatability with the aim of assessing its potential as a rodenticide. According to the results obtained in the no-choice feeding test, the increase in sodium selenite content in the bait adversely affects the total amount of bait eaten by Swiss mice. However, although the amount of bait eaten decreased, the total sodium selenite intake increased, particularly in treatments with contents of 0.05 and 0.1% of sodium selenite. In addition, the average daily intake by Swiss mice in the no-choice feeding test, in all exposure periods, increased with the decrease in sodium selenite content in the tested bait. The average daily bait intake of 0.1% of sodium selenite was 6.3 times lower than the average daily intake of bait with 0.0125% of sodium selenite. Compared to the average daily intake of bait with 0.0125% of sodium selenite, the daily intake of bait with 0.05% and 0.025%, for all exposure times was from 2.1 to 3.7 times lower.

In the treatments with 0.1% and 0.05% of sodium selenite in the no-choice feeding tests, two individu-

als (two males) during the first 24 h did not consume the bait and were excluded from the experiment.

According to previous studies (Jaćević et al., 2011), the oral LD_{50} of sodium selenite (anhydrous, pure min. 99%) was 8.9 and 11.2 mg/kg for male and female Swiss mice, respectively, and was in accordance with the earlier published data of Pletnikova (1970), where the LD_{50} value for mice was 7 mg/kg. Henschler and Kirschner (1969) reported a LD_{50} of sodium selenite of 48 mg/kg. Our results show that an average lethal dose of sodium selenite in the no-choice feeding tests is 3.7-fold higher at the shortest exposure, while at the longest exposure period it was 9.6-fold higher than the obtained oral LD_{50} value with the same form of sodium selenite in *per os* application. In comparison with the same LD_{50} values of sodium selenite, the average non-lethal dose, for the shortest and the longest bait exposure periods, was two and five times higher, respectively.

With an increase in sodium selenite content and exposure period, the mortality percentage of Swiss mice in the no-choice feeding test also increased. In addition, no statistically significant difference between the lethal effects caused by consumption of bait with 0.05 and 0.1% sodium selenite was noted. The time of death of half of the Swiss mice individuals consuming these baits, after all of the exposure periods, was about 95 h. Bait with 0.025 and 0.0125% sodium selenite content did not cause the death of half of the individuals.

Based on the results of pathohistological examination of dead individuals, death occured as the result of substantial degenerative changes to the inner tissues of the heart, liver, spleen and kidneys. Previous investigations (Jaćević et al., 2006) show that by the ingestion of a higher amount of sodium selenite, degenerative changes in the heart of Swiss mice occur.

To acquire successful rodent control with bait, the acceptability and palatability of the bait must be good enough to stimulate intake of the required lethal dose (Salmon and Dochtermann, 2006). In practical use, besides the content of the active ingredient, environmental conditions (Salmon and Dochtermann, 2006), carriers of the active ingredient (Prakash et. al., 2003) and bait additives (Marsh, 1988) can significantly affect bait acceptability. There is little data available on the acceptability of rodenticides by Swiss mice. According to Marshall (1984), bait acceptability for Swiss mice in choice feeding tests, with 0.0750 mg/kg content of cholecalciferol, was 52.5%. In our experiments, the highest acceptability in the choice feeding test was displayed by bait with the lowest content of sodium selenite (40.45%). Compared to this, the acceptability of bait with 0.1% sodium selenite was three times, while the palatability was 4.5 times lower. Bait containing 0.05% sodium selenite showed 2.8- and 4.3-fold lower acceptability and palatability, respectively. Bait with 0.0125 and 0.025% content of sodium selenite did not cause the death of the Swiss mice.

Based on the results obtained, it can be concluded that bait with 0.1 and 0.05% of sodium selenite possess some potential as rodenticides in urban and agricultural environments. Further research aimed at finding the appropriate active ingredient carrier and bait additives that could improve bait acceptability and palatability, are necessary. Field studies with wild rodents are necessary to confirm these findings and to refine or optimize the use of sodium selenite in rodent bait to improve rodent pest management programs.

Acknowledgments - This study was supported by Ministry of Education, Science and Technological Development of the Republic of Serbia (Grants III 46008 and OI 173039).

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