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Clinical experimental studies on acute rumen acidosis in sheep with a pre-existing chronic lead and organophosphorus compound intoxication. I. Clinical and haematological changes

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ABSTRACT

Experiments were performed on 15 sheep divided into 2 groups (A - 7 animals; B - 8 animals) in order to monitor the changes in clinical and haematological parameters in chronic lead and pesticide intoxication followed by acute rumen acidosis. A chronic lead and Fosdrin 24 intoxication was provoked in both groups via administration of lead acetate at a dose rate of 15 mg/kg body mass and 3.5 mg/kg body mass of organophosphorus insecticide Fosdrin 24 (2-carbomethoxy-1-methylvinyl-dimethylphosphate) once weekly, 2 hours after morning feeding for 6 months. Before treatment of experimental groups with the toxic combination, blood samplings were performed in 15-day intervals, and after the beginning of the experiment on a monthly basis until the end of the 6th month, for determination of haemoglobin, erythrocyte counts, total and differential leukocyte counts, haematocrit values, erythrocyte sedimentation rate. At the end of the 6th month, acute rumen acidosis was provoked in group B sheep via the administration of 20 ml/kg body mass beet molasses containing 540 g/l saccharose. Haematological and clinical parameters were followed out in dynamics at hours 2, 6, 10, 12, 20 post-treatment and compared with group A sheep. It was concluded that the application of the aforementioned doses of xenobiotics resulted in chronic intoxication with clearly expressed clinical and haematological changes: accelerated heart and respiratory rates, erythropenia, oligochromia and leucocytosis. On the basis of the chronic lead and insecticide intoxication, the acute rumen acidosis aggravated the clinical and haematological changes and resulted in total lethality for animals in group B.

Key words: acute rumen acidosis, sheep, lead chronic intoxication, organophosphorus chronic intoxication

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Introduction

Rumen acidosis is a disease in ruminants on a nutritive basis, manifested by increased release of lactic acid and histamine in rumen and resulting in several metabolic and functional systemic disturbances (DIRKSEN, 1965; AHRENS, 1967; DUNLOP, 1972; CONNOTE, 1981; MICHAELL, 1990). Animals with rumen acidosis also manifest various nervous (AHRENS, 1967; SODHI, 1981; NIKOLOV and GROSEVA, 1995; NIKOLOV, 1998), cardiovascular and respiratory (SODHI, 1981; NIKOLOV and GROSEVA, 1995; NIKOLOV, 1998), organic (NIKOLOV and GROSEVA, 1995; NIKOLOV, 1998), haematological and biochemical changes (DIRKSEN, 1965; AHRENS, 1967; DUNLOP, 1972; CONNOTE, 1981; MICHAELL, 1990; NIKOLOV, 1998).

The accumulation of heavy metals and pesticides in soil, plants, and then in animals is a real problem (VASILEV and NIKOLOV, 1994; YORDANOVA and NIKOLOV, 1994; VASILEV and NIKOLOV, 1995). The modern industrialization and chemicalisation of agriculture create the prerequisites for chronic intoxications in domestic animals, influencing the various clinical and biochemical parameters of blood, rumen content, urine, etc., leading to decreased productivity and very often to slaughtering or euthanasia (VASILEV and NIKOLOV, 1994; YORDANOVA and NIKOLOV, 1994, 1995; NIKOLOV and YORDANOVA, 1995; SIMEONOV and NIKOLOV, 1995; VASILEV and NIKOLOV, 1995). No data exist on the influence of simultaneously occurring lead and organophosphorus compound intoxications in animal organisms on the one hand, or for the development of acute rumen acidosis on the basis of such an existing intoxication, on the other.

This fact provided us with the reason to perform complex experimental studies in small ruminants in order to monitor changes in the clinical and haematological parameters in the course of such pathologies.

Material and methods

Experimental studies were performed on 15 merino sheep with masses 55-62 kg, aged 5-6 years. All sheep were fed and housed in a similar manner and given antihelmintics against both ecto- and endoparasites. The daily ration of each animal consisted of 0.5 kg concentrate, 1 kg alfalfa hay, 2 kg corn silage, with free access to water and salt. After 15 days of adaptation the animals were divided into 2 groups – A (7 sheep) and B (8 sheep). Both groups continued to receive the same ration.

A chronic lead and pesticide (organophosphorus insecticide Fosdrin 24 - 2-carbomethoxy-1-methylvinyl-dimethylphosphate) intoxication was provoked in both groups via administration of lead acetate at a dose rate of 15 mg/kg body mass and 3.5 mg/kg body mass, Fosdrin 24 once weekly, 2 hours after morning feeding for 6 months. The doses were individually calculated for each animal. Both compounds were applied dissolved in 0.5 l water via a stomach tube. Before the treatment of experimental groups with the toxic combination a complete physical examination and blood sampling were performed three times at 15-day intervals. The following haematological parameters were determined: haemoglobin, erythrocyte counts, leukocyte counts and haematocrit values (automated analyser Sirano-plus, Germany); differential leukocyte counts (Burker camera counting), erythrocyte sedimentation rate - ESR (Westergren).

After the beginning of the experiment the complete clinical status of sheep was monitored, and blood for determination of the aforementioned parameters was sampled once monthly.

Acute rumen acidosis was provoked at the end of the 6th month in group B sheep via the administration of 20 ml/kg body mass beet molasses containing 540 g/l saccharose. The molasses were given 2 hours after morning feeding, dissolved in 1 l of water, via a stomach tube. Physical examination and blood sampling were performed in dynamics at hours 2, 6, 10, 12, 20 post-treatment in both groups of sheep.

Statistical analysis of data was performed using the t-test at a level of significance at $P<0.05$.

Results

1. Clinical studies

During the control physical examinations (days 30; -15 and 0) there were no deviations from the reference ranges in all animals (Table 1.) Up to the end of the 2nd month (day 60) after the beginning of the experiment, there were no changes in the clinical status of sheep in both groups treated with lead acetate and Fosdrin 24.

Clinical parameters, body temperature, heart rate, respiratory rate and rumen movements at day 60 post-treatment were 39.6±0.1 °C; 89±7/min; 32±5/min and 7±1/5 min, respectively. The animals accepted food and water, ruminated, urinated and defecated normally.

Afterwards, a decrease in appetite was noticed as well as grinding of teeth, weak rumination and unsteady gait. At about the fifth month from the beginning of the experiment we observed alopecia on several skin

Table 1. Changes in clinical parameters of sheep with acute rumen acidosis after chronic lead-organophosphorus compound intoxication

Parameters	Group	Period of investigation													
		Days									Hours				
		-30	-15	0	30	60	90	120	150	180	2	6	10	12	20
Rectal temperature (°C)	A	39.6 ±0.2	39.5 ±0.3	39.7 ±0.2	39.8 ±0.3	39.6 ±0.1	39.1 ±0.2	39.2 ±0.3	38.8 ±0.2	38.5 ±0.1	38.6 ±0.1	38.6 ±0.2	38.5 ±0.1	38.6 ±0.2	38.7 ±0.2
	B	39.7 ±0.1	39.4 ±0.2	39.6 ±0.1	39.7 ±0.2	39.8 ±0.2	39.5 ±0.3	39.1 ±0.2	38.9 ±0.1	38.6 ±0.2	38.6 ±0.1	38.4 ±0.2	38.1 ±0.1	37.6 ±0.2[a]	37.1 ±0.1[a]
Heart rate (1/min)	A	79±2	80±6	83±7	81±6	89±7	119±7[a]	123±9[a]	127±7[b]	132±9[b]	129±7	133±8	128±7	134±6	129±10
	B	80±3	83±4	81±5	83±6	91±5	113±6[a]	120±8[a]	129±6[b]	127±5[b]	130±8	139±8	141±9[a]	158±10[a]	162±8[b]
Respiratory rate (1/min)	A	22±3	24±2	20±3	24±4	32±5	41±4[a]	46±5[a]	48±3[a]	50±4[b]	49±3	51±2	53±6	50±4	52±4
	B	19±2	20±3	21±4	23±3	35±4	42±5[a]	45±6[a]	42±7[a]	56±6[b]	57±3	62±5	69±4[a]	73±5[a]	72±6[a]
Rumen movement (in 5 min)	A	11±1	10±2	9±1	10±2	7±1	6±1	5±2[a]	4±1[b]	4±2[b]	4±1	5±2	4±1	4±2	3±2
	B	10±2	9±2	11±1	10±1	7±2	5±2[a]	4±1[b]	4±2[b]	4±1[b]	4±1	2±1[a]	0	0	0

[a]P<0.05; [b]P<0.01

Group A (n=7) treated p.o. with lead acetate at a dose of 15 mg/kg body mass and 3.5 mg/kg body mass, Fosdrin 24 once weekly for 6 months.
Group B (n=8) treated p.o. with lead acetate at a dose of 15 mg/kg body mass and 3.5 mg/kg body mass, Fosdrin 24 once weekly for 6 months, and the end of the 6th month treated with 20 ml/kg body mass beet molasses containing 540 g/l saccharose.

Table 2. Changes of haemoglobin, RBC, WBC, HTC and ESR in sheep with acute rumen acidosis after chronic lead-organophosphorus compound intoxication

Parameters		Group	Period of investigation													
			Days									Hours				
			-30	-15	0	30	60	90	120	150	180	2	6	10	12	20
Hb (g/l)		A	95±8	96±7	95±6	96±7	94±3	90±4	85±5	79±2[a]	74±3[b]	74±3	73±2	74±3	75±3	74±4
		B	97±6	98±7	95±8	94±4	90±4	87±3	86±4	80±3[a]	73±4[b]	77±2	85±2	99±1[a]	112±4[b]	115±5[b]
RBC ($\times 10^{12}$/l)		A	10.5 ±1.2	10.2 ±1.3	10.3 ±1.2	10.2 ±1.4	10.0 ±1.2	9.1±1.4	8.2 ±1.4[a]	7.6 ±0.7[b]	7.4 ±0.5[b]	7.4±0.2	7.3±0.1	7.5±0.1	7.3±0.2	7.4±0.3
		B	10.3 ±1.4	10.2 ±1.2	10.4 ±1.3	10.1 ±1.5	9.9±1.6	8.4±1.2	8.1 ±1.1[a]	7.8 ±0.9[a]	7.6 ±0.8[b]	7.7±0.9	7.9±0.8	9.1 ±0.3[a]	10.8 ±0.2[b]	12.6 ±0.4[b]
WBC ($\times 10^{9}$/l		A	8.6±1.3	8.7±1.4	8.7±1.3	8.9±1.2	9.2±1.4	10.5 ±1.4[a]	14±1.6[a]	14.1 ±1.3[b]	16.7 ±1.4[b]	16.6 ±1.3	16.5 ±1.2	16.1 ±1.3	16.5 ±1.2	16.3 ±1.5
		B	8.7±1.2	8.6±1.1	8.8±1.2	8.8±1.3	9.4±1.5	10.8 ±1.6[a]	13.4 ±1.5b	13.9 ±1.7[b]	16.2 ±1.8[b]	16.3 ±1.4	17.1 ±1.3	17.8 ±1.4	19.3 ±1.6[a]	21.8 ±2.1[b]
HTC (%)		A	35±3	36±4	34±2	36±3	37±5	35±4	32±3	28±4	27±3[a]	28±3	27±4	26±3	28±4	28±3
		B	36±5	35±4	37±3	35±4	36±4	34±5	33±4	27±3	26±2[a]	27±3	27±4	35±3[a]	37±4[a]	42±5[b]
ESR	mm for 1h	A	16.2 ±1.2	16.4 ±1.1	16.7 ±1.2	16.8 ±1.3	16.5 ±1.1	17.9 ±1.2	18.8 ±0.9	19.2 ±1.3	20.2 ±0.3[a]	20.3 ±0.4	20.5 ±0.3	20.1 ±0.5	20.4 ±0.1	20.3 ±0.5
		B	16.4 ±1.1	16.7 ±0.9	17.0 ±0.9	16.9 ±1.2	17.1 ±0.9	18.3 ±1.1	18.9 ±1.0	19.4 ±0.9	21.0 ±0.5[a]	20.9 ±0.6	20.1 ±0.4	17.5 ±0.8	16.7 ±0.9	15.1 ±0.2[a]
	mm for 2h	A	33.2 ±3.1	34.1 ±4.2	33.9 ±3.8	34.1 ±4.2	33.9 ±3.9	34.5 ±4.3	35.2 ±4.4	36.4 ±4.2	38.3 ±4.1[a]	38.4 ±3.9	38.2 ±3.7	38.6 ±3.2	39±4.1	39.1 ±4.2
		B	33.5 ±4.1	33.9 ±3.8	34.1 ±2.9	34.2 ±3.2	34.5 ±3.3	34.8 ±3.9	35.6 ±4.1	36.9 ±4.1	38.9 ±4.4[a]	39.0 ±4.2	38.9 ±4.1	36±3.2	35±3.1	32±2.8[a]

[a]P<0.05; [b]P<0.01

Group A (n=7) treated p.o. with lead acetate at a dose of 15 mg/kg body mass and 3.5 mg/kg body mass, Fosdrin 24 once weekly for 6 months. Group B (n=8) treated p.o. with lead acetate at a dose of 15 mg/kg body mass and 3.5 mg/kg body mass, Fosdrin 24 once weekly for 6 months, and the end of the 6th month treated with 20 ml/kg body mass beet molasses containing 540 g/l saccharose.

areas, with reddening of skin and pruritus. Diarrhoea, depression, emaciation, periodical colics and lameness occurred in 90% of sheep in group A. Both wool and gingivae became grey with vesicular ulcerative inflammation of the oral cavity in 70% of experimental animals. After auscultation of the lungs there were moist rales and accelerated vesicular breathing. Some sheep were in a state of excitement, followed by depression. Heart and respiratory rates were significantly elevated from the third month onwards, reaching: 132±9/min; 50±6/min; rumen movements were 4±2/5 min (Table 1.)

After the induction of acute rumen acidosis in sheep in group B the clinical status changed abruptly as early as the 6^{th} hour post-treatment. Two sheep died at hour 6, another two at hour 10, one at hour 12 and the other three at hour 20. During the development of the acute rumen acidosis, followed in dynamics up to post-treatment hour 20, body temperature decreased, and heart and respiratory rates increased, with a weak, soft, barely detectable pulse. The rate and intensity of rumen movements decreased (2±1/5 min at hour 6 and 0/5 min at hour 10). Mucosae became cyanotic. The sheep refused food, did not ruminate, ground their teeth and showed increased thirst. All sheep suffered profuse diarrhoea up to hour 6. Prior to death they were in a comatose state with no reactions to light or sound.

Animals in group A, untreated with molasses, showed no deviations in clinical parameters compared to those at day 180 (Table 1.)

2. Haematological studies

Changes in haematological parameters of sheep in both groups, before and after the start of the experiment, are presented in Table 2. It was observed that during the three control studies (days 30; -15 and 0), the values of haemoglobin, red and white blood cell counts, the haematocrit and the ESR were within physiological ranges. After treatment of both groups with lead acetate and the insecticide Fosdrin 24, the haematological parameters changed in a different direction and at a different rate. Haemoglobin concentrations decreased, although not statistically significantly, to the end of the fourth month (day 120; 85±5 g/l). From the fifth month onwards the oligochromemia was clearly expressed ($P< 0.01$) with average values of 73±4 g/l. The changes in red blood cells counts showed the same tendency toward decrease during development of chronic lead and Fosdrin 24 intoxication.

The oligocythemia was well expressed after the 120^{th} day ($P<0.05$) in both groups, with average counts of 7.4±0.3 10^{12}/l ($P<0.01$) at day 180 and haematocrit values of 26±2% ($P<0.05$) (Table 2.)

Total leukocyte counts increased significantly ($P<0.05$) after the third month ($10.8\pm1.6 \quad 10^9$/l), reaching $16.2\pm1.8 \quad 10^9$/l ($P<0.01$) at the end of the sixth month. ESR (1 h) and ESR (2 h) values increased up to the 6th month, corresponding positively with simultaneously decreased erythrocyte counts.

At the end of the 6th month, on the basis of the chronic lead and Fosdrin 24 intoxication, the treatment of group B animals with molasses resulted in a various pattern of changes in both red and white blood cells (Table 2.) The amount of haemoglobin and the erythrocyte counts increased at hours 2-6, continuing up to hour 20, when average values were 115 ± 5 g/l and $12.6\pm0.4 \quad 10^{12}$/l, respectively ($P<0.01$). Haematocrit increased to $42\pm5\%$ at hour 20 ($P<0.01$). Total leukocyte counts continued to increase up to $21.8\pm2.1 \quad 10^9$/l at hour 20 ($P<0.01$). The erythrocyte sedimentation rate decreased and at the end of the experiment (hour 20) was 32 ± 2.8 mm (ESR hour 2) and 15.1 ± 0.2 mm (ESR hour 1) ($P<0.05$).

The changes in the differential blood counts during the development of the chronic lead and Fosdrin 24 intoxication showed neutrophilia, eosinopenia, lymphopenia and basophilic stippling of erythrocytes. These anomalies were further expressed after induction of the acute rumen acidosis.

Discussion

Our studies showed that the simultaneous and prolonged action of lead and Fosdrin 24 upon the animal organism had a different impact on its health. Body temperature was not influenced by the combined chronic lead and Fosdrin 24 intoxication up to day 180. Increased heart and respiratory rates reflected the functional and morphological disorders of the cardiovascular and respiratory system after the chronic combined intoxication. Our results are also corroborated by other authors (VASILEV and NIKOLOV, 1994, 1995; NIKOLOV and YORDANOVA, 1995).

The rate and intensity of rumen movements decreased as a result of the chronic toxic action of lead and Fosdrin 24 upon the motor, secretory, chemical and microbiological functions of rumen (YORDANOVA and NIKOLOV, 1994; NIKOLOV and YORDANOVA, 1995; VASILEV and NIKOLOV, 1995). After the induction of the acute rumen acidosis on the basis of the chronic intoxication, the toxic metabolites formed in rumen (lactate, histamine etc.) exerted an additional harmful effect on animal organs and systems, manifested by respective, more vigorous changes in clinical parameters: decrease in body temperature, acceleration of both

heart and respiratory rates, lack of rumen movements. Similar interpretations have also been reported by others (DIRKSEN, 1965; AHRENS, 1967; ANGELOV and NIKOLOV, 1994; NIKOLOV and GROSEVA, 1995; NIKOLOV, 1998).

The influence of heavy metals (lead and cadmium) and pesticides (organophosphorus compound) upon the haemopoietic system is such that they suppress the formation of erythrocytes during the development of chronic intoxication (NIKOLOV and YORDANOVA, 1995; SIMEONOV and NIKOLOV, 1995). This erythropenia has an influence on the amount of haemoglobin (oligochromemia), haematocrit values and the ESR (VASILEV and NIKOLOV, 1994, 1995; NIKOLOV and YORDANOVA, 1995; SIMEONOV and NIKOLOV, 1995). The clinical manifestations of the gastrointestinal troubles observed in our experiment showed that the prolonged action of the xenobiotics lead and Fosdrin 24 in sheep provoked leukocytosis and neutropenia. Loss of wool, the grey colour of gingivae and skin were all a result of chronic lead intoxication (YORDANOVA and NIKOLOV, 1994; VASILEV and NIKOLOV, 1994, 1995; NIKOLOV and YORDANOVA, 1995). During the development of acute rumen acidosis on the basis of chronic lead and pesticide intoxication, rumen osmotic pressure increased as a result of its filling with fluids coming from the blood (pH 3-4) (DIRKSEN, 1965; AHRENS, 1967; DUNLOP, 1972; CONNOTE, 1981; MICHAELL, 1990; NIKOLOV and GROSEVA, 1995; NIKOLOV, 1998). On the other hand, the onset of profuse diarrhoea as early as hour 3 led to a significant degree of systemic dehydration (SODHI, 1981; MICHAELL, 1990; NIKOLOV, 1998). This exsiccation influenced the systemic homeostasis and resulted in haemoconcentration and consequently in changes in the red blood picture and the ESR. The increased white blood cell counts were most probably due to profound intestinal disorders. This suppresses the haemopoietic function, and then functions of the cardiovascular, respiratory, urinary and nervous systems (DIRKSEN, 1965; AHRENS, 1967; DUNLOP, 1972; CONNOTE, 1981; MICHAELL, 1990; NIKOLOV and GROSEVA, 1995; NIKOLOV, 1998).

The application of molasses at a dose of 20 mg/kg body mass in sheep provoked acute rumen acidosis, and the studied clinical and haematological parameters changed later (at hour 24) compared to those in sheep with acute rumen acidosis on the basis of chronic lead and insecticide intoxication (ANGELOV et al., 1994; NIKOLOV and GROSEVA, 1995). This evidenced that the chronic action of lead and insecticide Fosdrin 24 on all studied parameters was hazardous at a higher degree and on this basis, the provoked rumen acidosis aggravated pathological

changes and resulted in a lethal issue for animals in group B earlier - up to hour 20 - than in acute rumen acidosis without previous treatment of sheep.

In conclusion, our complex experimental studies, performed for the first time, showed that the simultaneous application of the chosen doses of lead acetate and Fosdrin 24 provoked clinical intoxication in an early period (month 3) with changes in the studied clinical parameters. On this basis, the induction of acute rumen acidosis was clinically manifested at hour 2, and experimental animals died consecutively at hours 6, 10, 12 and 20. Erythrocyte counts, the amount of haemoglobin and haematocrit, decreased during the development of the chronic lead and pesticide intoxication, and ESR was accelerated as a sequel to the systemic toxic action of xenobiotics. Acute rumen acidosis led to systemic dehydration, thus increasing the aforementioned parameters. The gastro-intestinal disorders during chronic lead and pesticide intoxication, and acute rumen acidosis, were aggravated, influencing the leukocyte counts towards an increase from the beginning to the end of the experiments.

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SAŽETAK

Pokusi su izvedeni na 15 ovaca podijeljenih u dvije skupine (A - 7 životinja; B - 8 životinja) s ciljem praćenja kliničkih i hematoloških pokazatelja u kroničnom trovanju olovom i pesticidima uz naknadnu akutnu acidozu buraga. Kronično trovanje olovom i Fosdrinom 24 izazvano je u obje skupine davanjem tjednih doza olovnog acetata od 15 mg/kg tjelesne mase i organofosfornog insekticida Fosdrina 24 (2-karbometoksi-1-metilvinil-dimetilfosfat) od 3,5 mg/kg tjelesne mase, dva sata nakon jutarnjeg hranjenja tijekom 6 mjeseci. Prije tretiranja pokusnih skupina s tim otrovima uzimani su uzorci krvi u 15-dnevnim intervalima, te nakon početka pokusa jednom mjesečno tijekom 6 mjeseci, a radi utvrđivanja hemoglobina, broja eritrocita, ukupnog i diferencijalnog broja leukocita, vrijednosti hematokrita i brzine sedimantacije eritrocita. Na kraju šestog mjeseca izazvana je akutna acidoza buraga davanjem ovcama iz skupine B po 20 ml/kg tjelesne mase melase od šećerne repe s po 540 g/l saharoze. Hematološki i klinički pokazatelji su praćeni nakon 2, 6, 10, 12, 20 sati po treatmanu i uspoređivani s ovcama iz skupine A. Zaključeno je da je davanje opisanih doza ksenobiotika dovelo do kroničnog otrovanja s jasno izraženim kliničkim i hematološkim promjenama: ubrzanim disanjem i radom srca, eritropeniom, oligokromijom i leukocitozom. Nakon kroničnog trovanja olovom i insekticidom, akutna acidoza buraga pojačala je kliničke i hematološke promjene te izazvala ugibanje svih životinja iz skupine B.

Ključne riječi: akutna acidoza buraga, ovca, kronično trovanje, olovo, organofosforni pesticidi