

Impact of serum lead levels on growth, hematology, and medical health of Aberdeen Angus bull calves

O. A. Zavyalov^{1*}, *E. S. Medetov*² and *Y. Kh. Delalov*³

¹Federal Scientific Center for Biological Systems and Agrotechnologies of the Russian Academy of Sciences, Orenburg, Russia

²Kadyrov Chechen State University, Grozny, Russia

Abstract The studies were performed on physiologically healthy Aberdeen Angus bulls. Animals based on data on the content of lead in blood serum, by percentile method, were divided into three groups: group I – up to the 25th percentile; group II – within the limits of 25-75 percentiles; group III – above the 75th percentile. The estimated parameters are: the elemental composition of blood, morphological and biochemical compositions of blood, the antioxidant status of blood serum. It was found that in terms of the average daily gains over the two-month period preceding sampling, bulls of groups II and III were inferior to individuals from group I by 3.8 and 11.3% ($P \leq 0.05$), respectively. The blood serum of group I bulls contained more Ca, Zn, Se, while minimal Cd concentrations were noted. In bulls with a minimum concentration of lead, an increased content of total protein, uric acid, monocytes, erythrocytes, and hemoglobin in the blood was noted. As the concentration of lead increased from minimum to maximum, the activity of the enzymes of primary antioxidant protection – superoxide dismutase and catalase - decreased, against the background of an increase in the level of malonyldialdehyde.

1 Introduction

Lead (Pb) is considered one of the most dangerous and cumulative environmental pollutants that affect all biological systems [1]. In living systems, lead is considered one of the permanent and ubiquitous heavy metals [2]. It is present in all parts of the environment in three main forms: metallic lead, lead salts and organic lead containing carbon [3]. Lead exposure causes clinical pathological changes in the kidneys and endocrine system [4]. High levels of lead in animals lead to reproductive failure [5], decreased productivity [6], accompanied by hearing impairment, neuromuscular weakness and negative changes in cognitive functions in humans and experimental animals [7]. Lead enters the body of animals by breathing and consuming contaminated feed and water. The digestibility of orally administered lead is low. However, due to the low rate of excretion, harmful levels of lead can accumulate in tissues after prolonged intake, even in small amounts [8]. A wide range of

* Corresponding author: Oleg-zavyalov83@mail.ru

negative effects of lead on the mammalian body is mainly associated with an increase in the level of reactive oxygen species in tissues [9]. Exposure to high levels of lead, through the development of oxidative stress, can lead to impaired kidney function [10] and liver [11]. Acute and chronic lead poisoning leads to damage to the blood vessels of the heart, which in some cases can have fatal consequences [12]. Exposure to low levels of lead can cause hypertension in both humans and animals [13]. The mobilization and accumulation of lead in the body depends on a number of factors, such as age, physiological condition, etc. [14]. The analysis of literary sources has shown that despite the sufficiently high level of knowledge of the role of toxic elements and, in particular, lead in the realization of physiological functions and productive qualities of the body of farm animals, the amount of available information on the study of the relationship of lead levels in metabolically active bio-substrates with productivity indicators with a wide list of blood values in beef steers during growing and fattening is quite small, which determines the relevance of such studies.

2 Research methodology

The studies were performed on physiologically healthy bulls of the Aberdeen Angus breed (age 17-18 months; $n=50$). The criterion for selecting animals for the experiment was a high growth rate (at least 1000 g/day) in the last two months preceding the sampling. At the next stage, the young animals selected for research based on data on the content of lead in blood serum, using the percentile method, were divided into three groups: group I – up to the 25th percentile ($n=15$); group II – within the limits of 25-75 percentiles ($n=25$); group III – above the 75th percentile ($n=15$). The basis for the choice of these intervals was previously conducted research [15].

The conditions of feeding and keeping for all examined animals were identical. The feeding rations of the experimental bulls corresponded to the feeding standards established for the corresponding age and sex group of animals [16].

Blood samples (9 ml) were taken from the tail vein in the morning into vacuum tubes with a blood coagulation activator from Hebei Xinle Sci&Tech Co.Ltd. The blood serum was separated by centrifugation of the samples for 10 min at a speed of 1000 rpm. The tubes were cooled to a temperature of $-18\text{ }^{\circ}\text{C}$ and stored until the moment of elemental analysis. Animal blood was examined using a biochemical automatic analyzer of the Dirui CS-240 brand (DIRUI, China) and a morphological automatic analyzer DF-50 Vet (Shenzhen Dymind Biotechnology Co, China). The activity of the enzyme superoxide dismutase and catalase was determined by the rate of loss of hydrogen peroxide in the incubation medium. The concentration of hydrogen peroxide was determined by reaction with ammonium molybdate. The content of malonyldialdehyde was evaluated using a reaction with thiobarbituric acid by a spectrophotometric method. The elemental composition of blood serum was determined by 24 parameters (B, Na, Mg, Al, P, K, Ca, Mn, Co, Ni, Cu, Ga, Sr, Ag, Cd, In, Ba, Hg, Bi, Cr, Fe, Zn, As, Se) using atomic emission and mass spectrometry with inductively coupled plasma.

The reliability of the differences was assessed using the Student's T-test. The significance level (p) was assumed to be less than or equal to 0.05. The tables show the average values of the indicators and the errors of the arithmetic averages. The Statistica 10.0 application software package was used for data processing.

3 Results

The actual differences between the groups of bulls in terms of serum lead concentration are shown in Figure 1.

2024

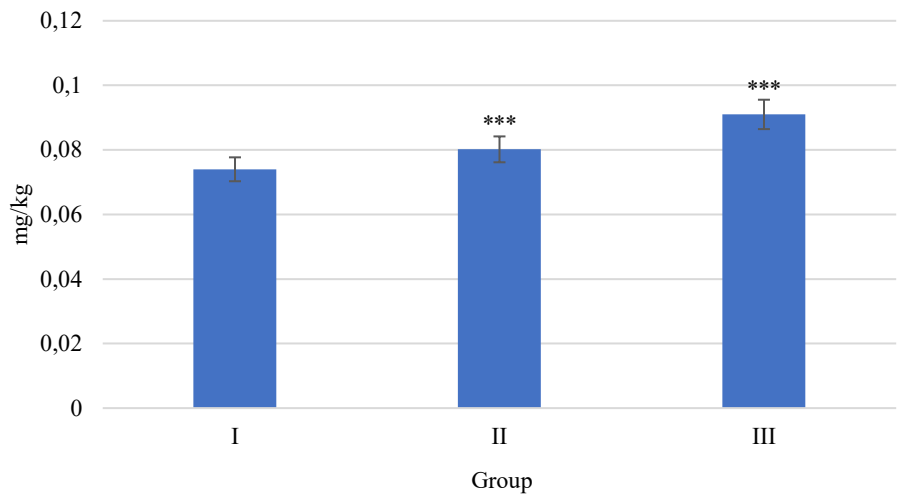


Fig. 1. Average values of lead concentrations in blood serum of bulls of experimental groups, mg/kg

It was found that the blood serum of group III bulls contained 0.030 mg/kg of lead, which is 12.3 ($P\leq0.001$) and 11.1% ($P\leq0.001$) more than in group I and II. At the same time, the range of lead concentrations in the blood serum of group I bulls ranged from 0.0655 to 0.0778 mg/kg, group II from 0.0795 to 0.0861 mg/kg, group III from 0.0852 to 0.101 mg/kg.

A comparative analysis of the intensity of weight growth of experimental animals in the context of the studied groups showed that as lead concentrations in the blood serum of bulls increased, productivity indicators decreased (Fig. 2).

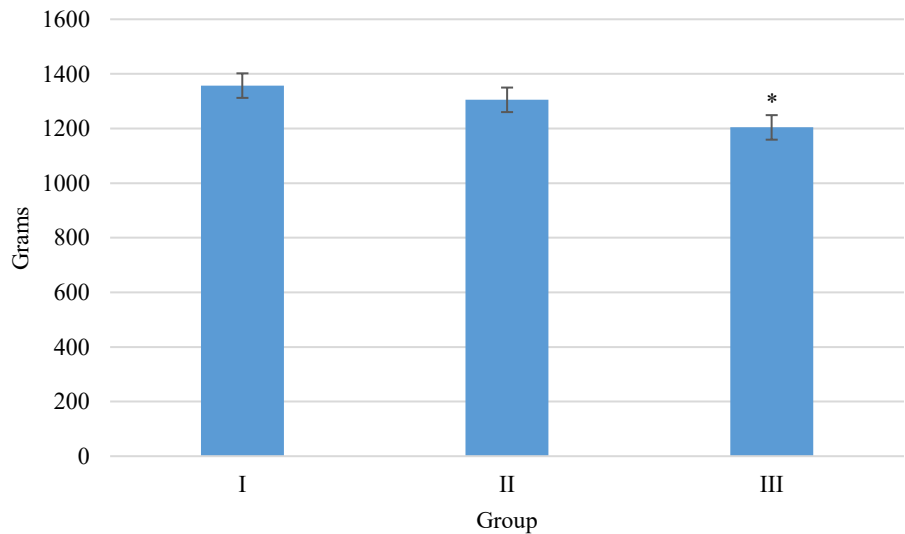


Fig. 2. Average daily increments of Aberdeen Angus bull calves depending on the concentration of lead in blood serum, g/day.

Thus, in terms of average daily gains over the two-month period preceding sampling, the bulls of groups II and III were inferior to individuals from group I by 3.8 and 11.3% ($P\leq$), respectively.

The elemental composition of blood serum differed in the content of individual chemical elements in the context of the compared groups (Table 1).

Table 1. Distribution of chemical elements depending on the concentration of lead in the blood serum of Aberdeen Angus bulls, mg/kg.

Element	Group		
	I	II	III
B	0.571±0.0469	0.851±0.131	0.625±0.0998
Na	3307.6±47.01	3261.3±22.03	3313.2±16.54
Mg	22.75±0.531	22.01±0.458	21.75±0.601
Al	1.32±0.331	1.03±0.156	2.01±0.509
P	95.52±2.65	97.60±1.63	96.58±3.69
K	208.2±3.12	210.5±4.62	211.4±4.54
Ca	128.5±3.68	120.8±5.22	118.8±2.22*
Mn	0.0372±0.0029	0.0412±0.0008	0.0611±0.0109
Co	0.0082±0.0004	0.0094±0.0004	0.0082±0.0006
Ni	0.131±0.0066	0.149±0.0059	0.135±0.0109
Cu	0.554±0.0158	0.621±0.0518	0.623±0.0411
Ga	0.0072±0.0001	0.0071±0.0002*	0.0073±0.0002
Sr	0.239±0.0114	0.247±0.0069	0.257±0.0149
Ag	0.0011±0.0003	0.0011±0.0001	0.0037±0.0006
Cd	0.0032±0.0002	0.0037±0.0001*	0.0037±0.0001*
In	0.0212±0.0005	0.0223±0.0006	0.0212±0.0007
Ba	0.0811±0.0074	0.0792±0.0043	0.0913±0.0054
Hg	0.114±0.0268	0.334±0.1204	0.134±0.0551
Bi	0.0024±0.0005	0.0032±0.0007	0.0033±0.0005
Cr	0.0521±0.0035	0.0512±0.0019	0.0561±0.0025
Fe	3.91±0.426	4.64±0.707	5.21±0.670
Zn	0.857±0.0133	0.805±0.0175*	0.762±0.0255**
As	0.0532±0.0042	0.0621±0.0052	0.0513±0.0029
Se	0.0544±0.0018	0.0492±0.0013*	0.0432±0.0023**

* P≤0.05; ** P≤0.01 compared to group I

In particular, it was found that the blood serum of group I bulls contained more Ca – by 1.7 and 8.2 (P≤0.05)%, Zn – by 6.6 (P≤0.05) and 12.5% (P≤0.01), Se – by 10.6 (P≤0.05) and 25.9% (P≤0.01), at the same time, minimum Cd concentrations were noted by 13.5% (P≤0.01), respectively, in relation to animal units of groups II and III.

As the lead level increased, individual indicators of the morphological and biochemical composition of the blood in the examined animals changed (Table 2).

Table 2. Morphological and biochemical composition of the blood of Aberdeen Angus bull calves depending on the level of lead.

Indicator	Group		
	I	II	III
Glucose, mmol/L	2.66±0.676	2.53±0.109	1.64±0.712
Total protein, g/L	89.92±1.45	85.28±1.50*	84.18±2.03*
a-Amylase, Units/l	30.83±0.542	30.25±0.412	30.17±0.909
ALT, Units/l	41.35±1.18	44.18±2.19	47.17±2.43*
AST, Units/l	132.2±4.53	144.4±4.41	145.4±3.81*
Total bilirubin, µmol/l	1.86±0.139	1.88±0.143	1.57±0.246
Cholesterol, mmol/l	2.73±0.0909	2.64±0.0937	2.83±0.171
Triglycerides, mmol/l	0.0982±0.0483	0.0793±0.0268	0.187±0.0385
Urea, mmol/L	3.32±0.153	2.98±0.121	3.23±0.326
Creatinine, µmol/L	138.1±4.18	131.5±4.79	135.3±5.72

Uric acid, $\mu\text{mol/L}$	30.16 ± 0.7984	$27.75\pm0.714^*$	$26.23\pm0.559^{**}$
Leukocytes $10^9/\text{l}$	9.07 ± 0.5567	9.677 ± 0.409	8.47 ± 0.551
Neutrophils, %	32.95 ± 3.09	28.14 ± 2.86	30.57 ± 0.821
Relative content of lymphocytes %	57.05 ± 3.83	59.05 ± 2.77	56.91 ± 0.710
Monocytes, %	9.54 ± 0.529	9.45 ± 0.561	$7.16\pm0.409^*$
Eosinophils, %	2.22 ± 0.412	2.62 ± 0.331	2.52 ± 0.279
Basophils, %	0.633 ± 0.123	0.652 ± 0.143	0.567 ± 0.114
Relative neutrophil content, $10^9/\text{l}$	2.99 ± 0.328	2.75 ± 0.319	2.62 ± 0.205
Relative content of lymphocytes, $10^9/\text{l}$	5.18 ± 0.451	63.84 ± 58.02	4.82 ± 0.314
Monocytes, %	7.17 ± 0.809	9.54 ± 0.929	9.54 ± 0.561
Eosinophils, $10^9/\text{l}$	0.193 ± 0.0379	0.248 ± 0.0354	0.212 ± 0.0322
Basophils, $10^9/\text{l}$	0.0531 ± 0.0117	0.0612 ± 0.0169	0.0453 ± 0.0134
Erythrocytes, $10^{12}/\text{l}$	5.59 ± 0.111	$5.20\pm0.132^*$	$5.13\pm0.124^*$
Hemoglobin concentration, g/l	103.5 ± 2.52	$94.75\pm3.36^*$	$93.12\pm2.06^*$
Hematocrit, %	22.75 ± 1.37	19.88 ± 0.755	21.33 ± 0.895
Mean volume of one red blood cell, $10^{-15}/\text{l}$	40.85 ± 0.902	40.71 ± 0.452	39.45 ± 1.10
Average hemoglobin content in erythrocyte, pg	19.77 ± 0.876	18.12 ± 1.18	$17.31\pm0.760^*$
Erythrocyte saturation index with hemoglobin, g/l	458.3 ± 14.09	468.3 ± 17.23	$421.3\pm9.87^*$
RDW-CV Index, %	15.18 ± 1.09	13.63 ± 1.038	14.42 ± 1.01
RDW-SD index, fl	24.87 ± 1.57	22.36 ± 1.59	22.83 ± 1.35
Platelet count, $10^9/\text{l}$	309.6 ± 40.97	329.2 ± 39.14	318.6 ± 51.96

* $P\leq0.05$; ** $P\leq0.01$. *** $P\leq0.001$ compared to group I

As can be seen from the data obtained, in bulls with a minimum concentration of lead in the blood serum, an increased content of total protein was noted – by 5.4% ($P\leq0.05$) and 6.8% ($P\leq0.05$), uric acid – by 8.7% ($P\leq0.05$) and 15.0. monocytes – by 0.09 and 2.4% ($P\leq0.05$), erythrocytes – by 7.5 ($P\leq0.05$) and 9.0% ($P\leq0.05$), hemoglobin – by 9.3 ($P\leq0.05$) and 11.1% ($P\leq0.05$) in relation to the group of animals with minimal and average lead levels. At the same time, the indicators of the average hemoglobin content in the erythrocyte and the level of erythrocyte saturation with hemoglobin in group I bulls were higher relative to group III individuals by 14.2% ($P\leq0.05$) and 8.9% ($P\leq0.05$). It should also be noted that there was a significant increase in the concentrations of liver enzymes – alanine aminotransferase and aspartate aminotransferase in the blood serum of group III bulls relative to group I by 14.1 ($P\leq0.05$) and 10% ($P\leq0.05$), which indirectly indicates an increased detoxification process against the background of an increase in the exchange pool of lead in the body.

One of the important blood indicators related to the productive qualities of cattle is the level of antioxidant protection and lipid peroxidation. The study showed that as the concentration of lead increased from minimum to maximum, the activity of primary antioxidant defense enzymes decreased: superoxide dismutase – by 7.5 and 9.9% ($P\leq0.05$); catalase – by 7.1 and 9.4% ($P\leq0.05$), against the background of an increase in the level of malonyldialdehyde by 7.1 and 9.4% ($P\leq0.05$), respectively (Table 3).

Table 3. Indicators of antioxidant status and lipid peroxidation of blood serum of Aberdeen Angus bull calves, depending on the level of lead.

Indicator	Group		
	I	II	III
Malonyldialdehyde, μm	29.05 ± 0.927	31.59 ± 1.11	$33.59\pm1.21^*$
Superoxide dismutase, units/mg of protein	45.56 ± 1.10	42.13 ± 1.24	$41.03\pm1.66^*$
Catalase, units/mg of protein	43.52 ± 1.05	40.42 ± 1.11	$39.42\pm1.34^*$

* $P\leq0.05$ compared to group I

4 Discussion

The study showed that as the concentration of lead increased from minimum to maximum, the intensity of weight growth of bulls decreased by up to 11%. An explanation of the possible reason for the relatively low productivity of bulls with an increased content of lead in blood serum is possible taking into account data on the negative effect of this element on animal health, including through the development of oxidative stress [17]. In our study, confirmation of the development of oxidative stress in animals is a significant increase in the level of malonyldialdehyde, as one of the reliable and frequently used markers of oxidative stress in animals. The increased accumulation of reactive oxygen species caused by the development of oxidative stress leads to the inhibition of antioxidant defense mechanisms [18], while the antioxidant reactions of cattle to oxidative stress require a large amount of energy, which theoretically could be used to realize the genetic potential of weight growth. It has also been established that oxidative stress can be accompanied by a decrease in the commodity characteristics of the products obtained, as was shown by the example of dairy cows [19]. The results obtained in our experiment revealed a close relationship between the concentration of lead in blood serum and the metabolism of minerals. An explanation of this fact is possible taking into account previous studies demonstrating the existence of a significant relationship between lead levels and concentrations of other essential elements. Thus, it was found that many toxic properties of lead are due to its ability to replace essential elements: calcium [20], magnesium, iron [21], zinc [22], selenium [23], as well as cobalt and copper [24]. The negative dynamics of changes in hematological parameters of blood detected in our experiment under the influence of various levels of lead may be related to the ability of the latter to directly affect the hematopoietic system, limiting hemoglobin synthesis by suppressing the activity of a number of key enzymes associated with heme synthesis [25]. Lead also helps to reduce the life expectancy of circulating red blood cells by increasing the fragility of cell membranes [26].

5 Conclusions

Thus, it can be concluded that bulls with a minimum concentration of lead in the blood serum had higher rates of intensity of weight growth and were characterized by increased concentrations of Ca, Zn, Se, total protein, uric acid, monocytes, erythrocytes, hemoglobin. In the blood serum of animals with a high lead content, there was a reduced concentration of antioxidant defense enzymes – superoxide dismutase and catalase, against the background of a relatively high content of malonyldialdehyde.

The research was carried out with the financial support of the Russian Science Foundation under project No. 23-26-00045

References

1. R. C. Patra, D. Swarup, R. Naresh, P. Kumar, D. Nandi, P. Shekhar, S. Roy, S. L. Ali, *Eco-toxicol Environ Saf* **66**, 127-131 (2007)
2. M. A. Assi, M. N. Hezmee, A. W. Haron, M. Y. Sabri, M. A. Rajion, *Vet World* **9**(6), 660-671 (2016)
3. M. Ahamed, M. K. J. Siddiqui, *Clin. Chim. Acta* **383**(1), 57-64 (2007)
4. S. H. Jadhav, S. N. Sarkar, R. D. Patil, H. C. Tripathi, *Arch. Environ. Con. Toxicol* **53**(4), 667-677 (2007)
5. M. Ahamed, M. K. J. Siddiqui, *Clinical Nutrition* **26**(4), 400-408 (2007)

6. V. Kalashnikov, A. Zajcev, M. Atroshchenko, L. Kalinkova, T. Kalashnikova, S. Miroshnikov, A. Frolov, O. Zav'yalov, *Environmental Science and Pollution Research* **25(22)**, 21961-21967 (2018)
7. G. Flora, D. Gupta, A. Tiwari, *Interdiscip. Toxicol* **5(2)**, 47-58 (2012)
8. N. Ercal, H. Gurer-Orhan, N. Aykin-Burns, *Curr. Top. Med. Chem* **1(6)**, 529-539 (2001)
9. S. Miroshnikov, O. Zav'yalov, A. Frolov, M. Poberukhin, I. I. Sleptsov, F. Sirazetdinov, *Environmental Science and Pollution Research* **26(18)**, 18554-18564 (2019)
10. A. E. A. Moneim, M. A. Dkhil, S. Al-Quraishy, *J. Hazard. Mater* **194**, 250-255 (2011)
11. T. O. Omobowale, A. A. Oyagbemi, A. S. Akinrinde, A. B. Saba, O. T. Dara-mola, B. S. Ogunpolu, J. O. Olopade, *Environ. Toxicol. Pharm.* **37(3)**, 1202-1211 (2014)
12. A. Navas-Acien, E. Guallar, E. K. Silbergeld, S. J. Rothenberg, *Health Perspect. J.* **115(3)**, 472-482 (2007)
13. D. Bagchi, H. G. Preuss, *J. Inorg. Bi-ochem* **99(5)**, 1155-1164 (2005)
14. R. A. Al Naimi, D. Abdulhadi, O. S. Zahroon, E. H. Al-Taae, *Al-Anbar J. Vet. Sci.* **4**, 26-39 (2011)
15. S. Miroshnikov, O. Zav'yalov, A. Frolov, I. Bolodurina, A. Skalny, V. Kalashnikov, A. Grabeklis, A. Tinkov, *Biological trace element Research* **180(1)**, 56-62 (2017)
16. A. P. Kalashnikov, V. I. Fisinin, V. V. Shcheglov, N. G. Pervov, N. I. Klejmenov, N. I. Strekozov, B. D. Kal'nickij, I. A. Egorov, E. A. Mahaev, V. G. Dvalishvili, V. V. Kalashnikov, V. L. Vladimirov, N. V. Gruzdev, A. T. Mysik, N. A. Balakirev, A. I. Ficev, M. P. Kirilov, V. A. Krohina, P. A. Naumenko, S. V. Vorob'eva et al., *Normy i raciony kormleniya sel'skohozyajstvennyh zhivotnyh* (Izdatel'stvo «Znanie», Moskva, 2003)
17. S. J. Flora, *Free Radic Biol Med.* **51(2)**, 257-81 (2011)
18. A. Kapusta, B. Kuczyńska, K. Puppel, *PLoS ONE* **13(3)**, 0193512 (2018)
19. O. A. Zav'yalov, M. Y. Kurilkina, G. M. Topuria, *IOP Conference Series: Earth and Environmental Science. The proceedings of the conference AgroCON-2019*, 012076 (2019)
20. K. Y. Hwang, B. S. Schwartz, B. K. Lee, P. T. Strickland, A. C. Todd, J. P. Bressler, *J. Toxicol. Sci.* **62(2)**, 280-288 (2001)
21. T. I. Lidsky, J. S. Schneider, *Brain* **126(1)**, 5-19 (2003)
22. R. C. Patra, D. Swarup, P. Kumar, D. Nandi, R. Naresh, S. L. Ali, *Science of the Total Environment* **404(1)**, 36-43 (2008)
23. Y. Wang, Y. L. Ou, Y. Q. Liu, Q. Xie, Q. F. Liu, Q. Wu, T. Q. Fan, L. L. Yan, J. Y. Wang, *Biol Trace Elem Res.* **145(2)**, 127-35 (2012)
24. M. López Alonso, F. Prieto Montaña, M. Miranda, C. Castillo, J. Hernández, J. Luis Benedito, *Biometals* **17(4)**, 389-97 (2004)
25. J. Castro, D. Chirinos, E. Ríos, *Toxicol* **33(2)**, 88-92 (2016)
26. I. Baranowska-Bosiacka, I. Gutowska, M. Rybicka, P. Nowacki, D. Chlubek, *Neurol Neurochir. Pol. J.* **46(6)**, 569-578 (2012)