Acta Veterinaria (Beograd), Vol. 49. No. 5-6, 313–320, 1999.

UDK 619:616.015.31:636.5.084

EFFECTS OF EXCESSIVE LEVELS OF SODIUM SELENITE ON DAILY WEIGHT GAIN, MORTALITY AND PLASMA SELENIUM CONCENTRATION IN CHICKENS

MIRJANA TODOROVIĆ*, M. MIHAILOVIĆ ** and S. HRISTOV*

*University of Belgrade, Faculty of Agriculture, Nemanjina 6, Zemun
**University of Belgrade, Faculty of Veterinary Medicine, Bulevar JNA, 18, Belgrade

(Received, 3. August 1999.)

One hundred and five, one day old unsexed Hybro chickens divided into 7 groups, were fed basal diets supplemented with 0, 2, 5, 10, 20 and 30 mg Se/kg as sodium selenite for 6 weeks. The Se level of 2 mg/kg feed had no effect on chicken daily weight gain. The lowest level at which dietary Se caused reduction in daily gain was 5 mg/kg. Diets supplemented with 10, 15 and 20 mg Se/kg produced 24.5, 62.7 and 96.6% reductions in daily gain, respectively. Lower gains were evident within the first 11 days for chickens fed diets with 20 or 30 mg Se/kg and after 11 days for those provided with 10 or 15 mg Se/kg. Daily gain for the chickens fed the diet with 5 mg Se/kg was significantly lower only in the fifth week of fattening. Feeding diets with 15, 20 and 30 mg Se/kg caused 26.7, 60 and 80% mortality, respectively. Plasma Se concentrations were increased in all groups given supplmentary Se. Maximal plasma Se levels between 220 and 300 μg/l were reached on the 11th day (except for the group with 2 mg Se/kg) and at that level were maintained to the end of the experiment.

Key words: chickens, sodium selenite, daily weight gain, mortality, plasma selenium concentration

INTRODUCTION

Although naturally occuring and experimentally induced selenium toxicosis has been reported in almost all domestic animals, there have been only limited reports of experimentally induced selenium toxicosis in broiler chickens. The maximal level of dietary selenium that could be safe for chickens is not exactly determined. Many authors believe that decreased weight gain and feed consumption are sensitive parameters of selenium toxicity in animals. The maximum level of dietary selenium that could be tolerated without affecting chicken performance has been stated as 2 mg Se/kg (Cantor et al., 1984), 5

mg Se/kg (Hill, 1974) or 6 mg Se/kg (Moksnes and Northeim, 1982; Moksnes, 1983). Lower weight gain was already found in chickens fed a diet with 4 mg Se/kg (Cantor et al., 1984) or 6 mg Se/kg (Echevarria et al., 1988). Higher selenium levels in the diet led to chichen deaths. The rate of mortality increased with increasing selenium levels in the diet. Andreev et al. (1970) found that chickens fed diet with 60 or 80 mg Se/kg all died between 11 and 32 hours afterwards.

The concentration of selenium in the blood is dependent on the selenium level in the diet. Ducks fed diets with 10, 20 or 40 mg Se/kg exhibited the highest levels of blood selenium at the end of 7, 8 and 12 weeks, respecively (Heinz and Margaret, 1993).

In this stady we investigated the effects of graded levels of sodium selenite up to 30 mg S/kg, on daily weight gain, rate of mortality and plasma selenium concentration during the 6 week period of fattening.

MATERIAL AND METHODS

One hundred and five, one day old unsexed Hybro chickens divided into 7 groups, were fed the basal feed (corn-soyabean meal diet) supplemented with 0, 2, 5, 10, 15, 20 or 30 mg Se/kg as sodium selenite for 6 weeks. Feed and water were available ad libitum. Chickens were weighed weekly and daily gains were calculated. Heparinized blood samples, taken by puncture of the wing vein, were collected weekly from 5 chickens of each group and used for plasma selenium analyses employing the hydride generation technique coupled with atomic absorption spectrometry (Welty et al., 1987).

RESULTS

Effects of graded levels of selenium it the diet on chicken daily weight gains are shown in Figure 1 and 2. Average daily weight gains in the control group and in the group with 2 mg Se/kg in the diet were 29.25 g and 28.13 g, respectively (Figure 1).

Chicken daily gains were lower as birds were provided with the higher Se-containing diets. Thus, in the groups with 5, 10 and 15 mg Se/kg diet daily gains were 88%, 75,2% and 37,3% of those observed in the control group, respectively. Average daily gain in the group with 20 mg Se/kg diet was 1.0 g and in the group with 30 mg Se/kg it was 2.5 g in the first 4 weeks (all birds in this group died by the end of the 4th week). Lower bird gains were evident within the first 11 days for chickens fed diets with 20 or 30 mg Se/kg and after the first 11 days for those provided with 10 or 15 mg Se/kg (Figure 2). Daily gains for the chickens fed the diet with 5 mg Se/kg were significantly lower only in the fifth week of fattening.

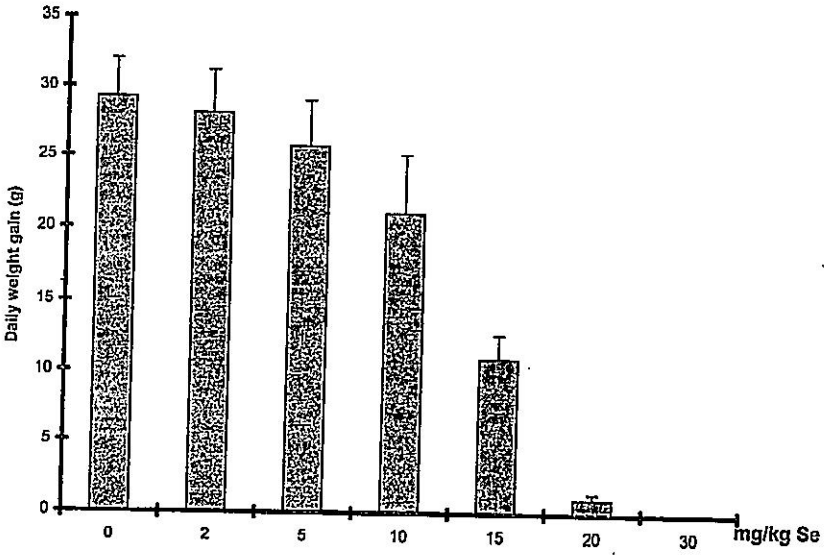

Figure 1. Daily weight gains (calculated for the whole period) in relation to the dietary levels of selenium

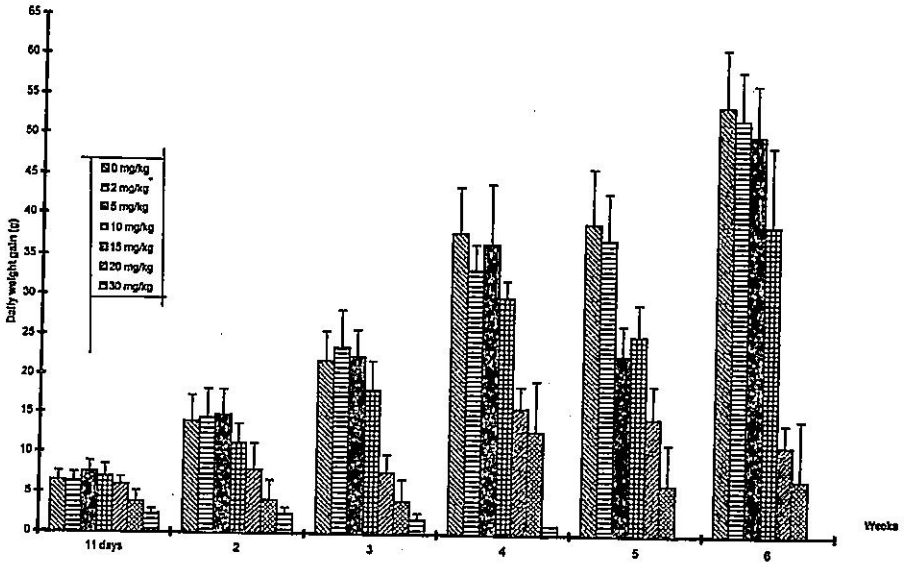

Figure 2. Daily weight gain (g) of chickens fed with graded levels of selenium

One chicken in the control group and one in the group with 2 mg Se/kg died during the 6 week fattening period (Table 1). Necropsy analyses showed that the cause of these deaths was not related to excess of selenium. No chicken died in the groups with 5 and 10 mg Se/kg. At 15, 20 and 30 mg Se/kg, selenium caused 26.7, 60.0 and 80.0% mortality, respectively. Most chickens died in the first three weeks of the experiment.

Table 1. Mortality of chickens fed diets supplemented with excessive levels of selenite

Added Se (mg/kg)	Weekly number of dead chickens						Total (%)
	1 wk	2 wk	3 wk	4 wk	5 wk	6 wk	
0				1			6.7
2		1					6.7
5							
10							
15		1	1	2			26.7
20	2	1	4	2			60.0
30	4	4	2	1			80.0

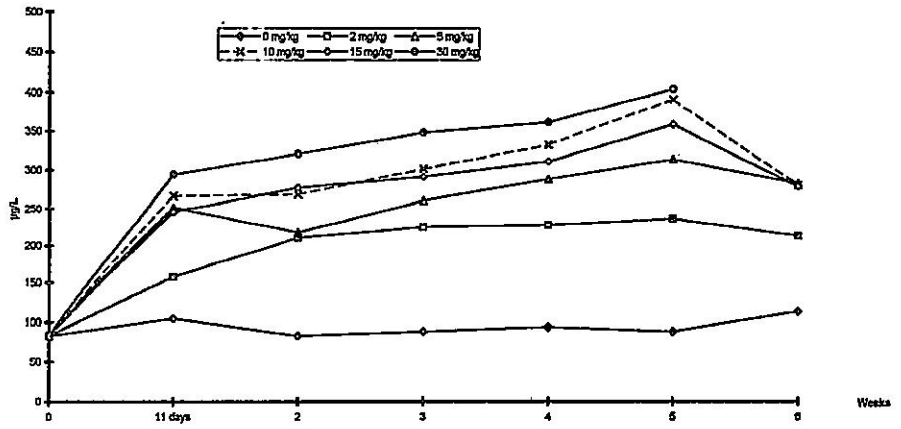

Figure 3. Plasma Se concentrations (μg/L) in chickens fed diets with graded levels of Se

The plasma Se concentrations increased in all groups of chickens given supplementary selenium (Figure 3). Depending on the dietary Se level, maximal plasma Se between 250 and 300 μg/l was reched in the 11th day in all supplemented groups, except in the group with 2 mg Se/kg. With small variations these levels were maintained to the end of the experiment. In the group

fed 2 mg Se/kg die, plasma Se concentration continued to rise to the end of the second week reaching a value of 220 μg/l and then plateaued.

DISCUSSION

We found that a Se level of 2 g/kg feed had no effect on chicken performance as measured by daily weight gain. Cantor et al. (1984) observed that an intake of 2 mg Se/l drinking water had no adverse effects in broilers but an intake of 4 mg Se/L led to lower weight gain and feed intake. However, Hill (1974) reported that chickens tolerated 5 mg Se/kg, while Moksnes and Norheim (1982) and Moksnes (1983) did not find any significant effects on chicken growth with 6 mg Se/kg feed. In our study, the lowest level at which dietary Se became toxic to chickens (as measured by reduction in daily weight gain) was 5 mg/kg feed. This Se level resulted in a 12% reduction of average daily gain. Echevarria et al. (1988) found lower growth in chickens fed a diet with 6 mg Se/kg. Diets supplemented with 10, 15 and 20 mg Se/kg caused 24.5%, 62.7% and 96.6% reduction in average daily gain. Similar to our results are those obtained by Lowry and Baker (1989) who reported a 61% reduction in weight gain of young chickens when the diet was supplemented with 15 mg Se/kg. In our experiment lower gains were evident within the first 11 days for chickens fed diets with 20 or 30 mg Se/kg and after the first 11 days for those provided with 10 or 15 mg Se/kg. Daily gains for the chickens fed the diet with 5 mg Se/kg were significantly lower only in the fifth week of the experiment.

Graded levels of Se up to 10 mg Se/kg feed did not cause chicken death. At 15, 20 and 30 mg Se/kg feed selenium caused 26.7, 60 and 80% mortality, respectively. Most chickens died in the first three weeks of fattening. Echervarria et al. (1988) found that diets up to 9 mg Se/kg feed, Arnold et al. (1972) up to 8 mg Se/kg and Heinz et al. (1990) up to 10 mg Se/kg did not lead to chicken deaths. Andreev et al. (1970) reported that 60 or 80 mg Se/kg feed caused the death of all chickens after 11 - 32 hours. Significantly lower mortality than in our experiment was observed by Jensen (1986) and El Begearmi and Combs (1982). Because the highest levels of Se resulted in severe reduction in feed consumption, Se induced starvation may have been related to chicken mortality.

Organ and tissue Se concentrations were directly proportional to dietary Se concentrations when animals were fed adequate but not excessive Se (Meyer et al., 1981). However, Goehring et al. (1984) found that blood Se was equally sensitive in response to excessive dietary Se intake. This was evident also in our study. Namely, at the 11th day in all supplemental groups except the lowest (2 mg Se/kg), plasma Se concentrations reached maximal levels of between 250 and 300 μg/l depending on the dietary Se level. At those

levels Se concentrations were maintained to the end of experiment. At 2 mg Se/kg feed Se concentrations continued to rise to the end of the second week reaching a value of 220 μg/l and then plateauted.

REFERENCES

1. *Andreev, M.N., Minina, L. A. and Bronnikova, K. A.*, 1970. Optimal, toxic and lethal doses of sodium selenite for chickens and hens. Vet. Med. Nauki 4, 19-25.
2. *Arnold, R. L., Olson, O. E. and Carlson, C. W.* 1972. Dietary selenium and arsenic and their effects on tissue and egg selenium. Poult. Sci. 52,52, 847-852.
3. *Echevarria, M. G., Henry, P. R., Ammmerman, C. B., Rao, P. V. and Miles, R. D.* 1988. Estimation of the relative bioavailability of inorganic selenium sources for poultry. I. Effect of time and high dietary selenium on tissue selenium uptake. Poult. Sci. 67, 1259-1301.
4. *Cantor, A.H., Nash, D. M. and Johnson, T. H.* 1984. Toxicity of selenium in drinking water of poultry. Nutr. Rep. Int. 29, 683-688.
5. *El Begearmi, M. N. and Combs, Jr. G. F.* 1982. Dietary effects on selenite toxicity in the chicken Poult. Sci. 61, 770-776.
6. *Goehring. T. B., Palmer, I.S., Olson, O.E., Labal, G.W. and Wahlstrom, R.C.* 1984. Effects of seleniferous grains and inorganic selenium on tissue and blood composition and growth performance of rats and swine. J. Anim. Sci. 59, 725-732.
7. *Heinz, G. H. and Margaret, A. F.*, 1993. Over winter survival of mallards fed selenium. Arch. Environ. Contam. Toxicol. 25, 90-94.
8. *Heinz, G. H., Pendleton, G. W., Krinitsky, A. J. and Gold, L. G.* 1990. Selenium accumulation and elimination in mallards. Arch. Environ. Contam. Toxicol. 19, 374-379.
9. *Hill, C.H.* 1974. Reversal of selenium toxicity in chicks by mercury, copper and cadmium. J. Nutr. 104, 593-598.
10. *Jensen, L. S.* 1986. Effects of monensin on response of chicks to deficient and toxic level of dietary selenium. Poult. Sci. 65, 1163-1166.
11. *Lowry, K. R. and Baker, D. H.* 1989. Amelioration of selenium toxicity by arsenicals and cysteine. J. Anim. Sci. 67, 959-965.
12. *Meyer, W. R., Mahan, D. C. and Moxon, A. L.* 1981. Value of dietary selenium and vitamin E for weanling swine as mesured by performance and tissue selenium and glutathione peroxidase activities. J. Anim. Sci. 52, 302-311.
13. *Moksnes, K.* 1983. Selenium deposition in tissues and eggs of laying hens given surplus of selenium as selenomethionine. Acta Vet. Scand. 24, 34-44.
14. *Moksnes, K. and Norheim, G.* 1982. Selenium concentrations in tissues and eggs of growing and laying chickens fed sodium selenite at different levels. Acta Vet. Scand. 23, 368-379.
15. *Welty, B., Wolynetz, M. S. and Verlingen, M.* 1987. Interlaboratory trial on the determination of selenium in lyophilized human serum, blood and urine using hydride generation atomic absorption spectometry. Appl. chem. 59, 927-936.

EFEKTI VISOKIH KONCENTRACIJA NATRIJUM SELENITA NA DNEVNI PRIRAST, MORTALITET I NIVO SELENA U PLAZMI PILIĆA

MIRJANA TODOROVIĆ, M. MIHAILOVIĆ i S. HRISTOV

SADRŽAJ

Sto pet Hibro pilića starih jedan dan, podeljenih u 7 grupa, hranjeno je osnovnom hranom dopunjenom sa 0, 2, 5, 10, 15, 20 i 30 mg Se/kg u obliku natrijum selenita, u toku 6 nedelja. Nivo selena od 2 mg/kg hrane nije izazvao smanjenje dnevnog prirasta. Viši nivoi selena u hrani (10, 15 i 20 mg/kg) imali su za posledicu smanjenje dnevnog prirasta za 24,5, 62,7 i 96,6%. Niži dnevni prirast bio je evidentan već u toku prvih 11 dana ogleda kod grupa koje su dobijale u hrani 20 i 30 mg Se/kg i posle prvih 11 dana kod grupa koje su dobijale 10 ili 15 mg Se/kg. Dnevni prirast pilića koji su u hrani dobijali 5 mg Se/kg bio je signifikantno niži samo u petoj nedelji ogleda. Ishrana sa 15, 20 i 30 mg Se/kg imala je za posledicu mortalitet u iznosu 26,7, 60 i 80%. Koncentracija Se u plazmi bila je povećana kod svih grupa kojima je dodavan Se u hrani. Maksimalna koncentracija Se u plazmi, između 220 i 300 μg/l, bila je postignuta 11-og dana (osim u grupi sa 2 mg Se/kg) i na tom nivou se održavala do kraja ogleda.