

## THE EFFECT OF FEEDING VARIOUS DIETARY LEAD LEVEL ON THE PERFORMANCES OF BROILERS

Z. SINOVEC, LJILJANA JANKOVIĆ, BRANA RADENKOVIĆ, N. JOVANOVIĆ

*Faculty of Veterinary Medicine, Belgrade*

(Received, 25. August 1999.)

*The 42 day long trial was performed on 94 Hybro broilers divided into four groups. All groups of chickens were fed with commercial feed mixtures and the lead was added as lead acetate of quantities enough to supply 0; 9.2; 92.0 or 920.0 mg Pb/kg food. After 28 days, each group was divided into two subgroups. The first subgroup was continuously fed with diets containing lead and the other subgroup with the control feed mixture without added lead.*

*Increasing dietary lead intake level linearly decreased broiler performances, even at 100 ppm. The lower performance caused by the dietary lead was lessened by excluding the contaminated feed from the broilers diet, but only after a certain period which depended on the dietary level of lead, primarily. Early detection of lead presence in feed and elimination of contaminated mixtures from the diet can reduce negative effects on performance to an acceptable level.*

*Key words: lead, poultry, performance, feeding, broilers*

### INTRODUCTION

Lead is considered to be one of the major environmental pollutants and has been incriminated as a cause of accidental poisoning in domestic animals more than any other substance (NRC, 1972). One of the primary sources of lead contamination in the air, soil and water is combustion of fuel containing lead additives (Underwood, 1977). Substantial amounts are also used in production of batteries, pigments, ceramics, pesticides and plumbing (Paone, 1970). Recently, there has been considerable concern over the level of environmental contamination with toxic metals and its relationship with public health (Doyle, 1979).

The metabolism of dietary lead had been extensively reviewed. Monogastric animals absorb approximately 10 percent of dietary lead (Netahery and Miller, 1975; NRC, 1972). It was concluded (NRC 1980) that chickens are very resistant to lead poisoning (Damron et al., 1969; Vengris and Mare, 1974) and dietary lead at 1000 mg/kg been tolerated by poultry for several months with no visible signs of toxicity.

Damron et al. (1969) conducted replicate four week trials and concluded that broilers can tolerate up to 100 ppm dietary lead, while Latta and Donaldson (1986) found that 1000 ppm of lead depressed growth. Bafundo et al. (1984) performed several trials with young chickens to evaluate lead toxicity. The results of these experiments indicated that 1100-3300 ppm of Pb was toxic to growing chickens, and correlated with the conclusions of Damron et al. (1969) and Simpson et al. (1970) who described some of the adverse effects of lead poisoning on performance.

Berg et al. (1980) found that an increase of dietary lead concentration up to 2000 ppm, in two week old broilers induced a linear decrease of the body weight down to approximately 70% of the control, while Morgan et al. (1975) found that 500 ppm lead depressed growth in quails. Bakalli et al. 1995, conducted four trials to study the toxic effects of supplemental dietary lead in the form of sulfate or acetate salts. Supplementation with both forms of lead caused a linear decrease of body weight, depressed weight gain and had a significantly negative effect on feed: gain ratio. The authors concluded that the statement: "broilers can tolerate 100 ppm of Pb in finished feed" is no longer valid, because significantly depressed body weight gain was observed when only 1 mg Pb/kg feed had been added.

However data in the literature on the influence of exclusion of contaminated feed on broiler performance are scarce and there is no agreement concerning the possible positive effects of such a dietary change. The aim of this study was to evaluate the effects of different dietary lead levels on broiler performance, as well as the effects of subsequent exclusion of the contaminated feed.

#### MATERIAL AND METHODS

**Animals.** Hybro broilers were obtained from a commercial hatchery. The trial was performed on 94 chickens, which were housed in wire floor battery brooders. The usual light/dark cycle, temperature and moisture were maintained throughout the trial.

**Diets.** All groups of chickens were fed with a commercial mixture (table 1) which consisted of standard feed ingredients and contained enough nutrients to provide the requirements of the broiler category. Lead acetate  $[Pb(CH_3COO)_2]$  was added in amounts sufficient to provide 10, 100 or 1000 ppm of dietary lead in the diets offered to the three treatment groups (Table 2).

Table 1. Composition of complete feed mixtures, %

| Composition ingredient, % | Mixtures |       | Chemical composition, % | Mixtures |       |
|---------------------------|----------|-------|-------------------------|----------|-------|
|                           | I        | II    |                         | I        | II    |
| Maize, raw                | 58.90    | 67.20 | Moisture                | 11.14    | 11.46 |
| Soyabean meal, solvent    | 12.00    | 10.00 | Ash                     | 5.66     | 5.24  |
| Sunflower meal, solvent   | 14.00    | 10.00 | Crude protein           | 21.45    | 18.31 |
| Fish meal                 | 6.00     | 4.00  | Ether extract           | 6.92     | 6.52  |
| Meat and bone meal        | 3.00     | 3.00  | Crude fiber             | 4.06     | 3.55  |
| Fat                       | 3.50     | 3.00  | N-free extract          | 50.77    | 54.92 |
| Dicalcium-phosphate       | 0.40     | 0.40  | Ca                      | 1.02     | 0.87  |
| Limestone, ground         | 0.70     | 0.70  | P                       | 0.79     | 0.69  |
| Iodised salt              | 0.33     | 0.50  | ME, MJ/kg               | 14.17    | 14.09 |
| Methionin                 | 0.10     | 0.10  | Lysine                  | 1.17     | 0.96  |
| Lysine                    | 0.10     | 0.10  | Meth.+Cyst.             | 0.80     | 0.68  |
| Vitamin-mineral premix    | 1.00     | 1.00  | Tryptophan              | 0.27     | 0.23  |

Table 2. Dietary lead level in feed for broilers, mmg/kg

| Mash | G r o u p |       |        |         |
|------|-----------|-------|--------|---------|
|      | K         | 0 - I | 0 - II | 0 - III |
| I    | 0.00      | 11.19 | 99.38  | 995.85  |
| II   | 0.00      | 10.26 | 102.41 | 983.11  |
| Mean | 0.00      | 10.72 | 100.89 | 989.48  |
| SEM  | -         | 0.21  | 0.55   | 2.33    |

Design of the experiment. Broilers were divided into four groups and the trial lasted from hatching till the end of fattening. Three groups of chickens were fed with contaminated feed for 28 days, and after that each group was subdivided in to two subgroups. One subgroup was fed continuously with the contaminated feed till the end of the trial (42nd day), while the other subgroup was given feed without added lead.

Data and sample collection. Performance and health status were monitored during the trial. Body weight and feed consumption were measured on the 28th and 42nd day of the trial. From the measured data, average daily gain and gain/feed ratio were calculated. Dietary lead was determined (AOAC, 1980) by flame atomic absorption spectrophotometry (Perkin Elmer 3300).

Statistical analysis. All data obtained were statistically processed (Snedecor and Cochran, 1971) and an appraisal was made of the significance of differences between mean values among the groups of chickens.

## RESULTS AND DISCUSSION

Broilers of the control group had a harmonious conformation and proper developed bone and muscle tissue, a vivid temperament and were in good condition. All clinical findings were normal. Their appetite was good, and droppings were normally formed. No disturbances of health were observed in the experimental groups, except for weak signs of lead poisoning seen in the group fed with 1000 ppm of dietary lead. These broilers were less active and less vigorous than the others were. Also the color of the droppings was darker in the second, and especially in the third experimental group. However, no mortality was recorded during the trial.

The results of measurement of body weight and weight gain are summarized in table 3. Both parameters were depressed by increasing dietary lead content. Average body weights in all experimental groups were most similar at the beginning of the trial, without significant differences. During the trial, average body weights showed significant differences associated with the regime of nutrition. Both 100 and 1000 ppm dietary lead gave the first significant depression of body weight after two weeks treatment only, and differences were significant after a four week period. This was similar to the findings of Morgan et al. (1975) and Berkley et al. (1995). Further use of contaminated feed caused greater depression of body weight and a nonsignificantly lower body weight was recorded in the first experimental group, too. Exclusion of the contaminated feed from the diet moderated the growth depression, but the effects were measurable only after a recovery period lasting two weeks for the lower dietary lead level.

Table 3. Body weight\* and weight gain\* of broilers

| Trial days         | G r o u p                     |                             |                              |                             |
|--------------------|-------------------------------|-----------------------------|------------------------------|-----------------------------|
|                    | K                             | 0 - I                       | 0 - II                       | 0 - III                     |
| Body weight, g     |                               |                             |                              |                             |
| 1.                 | 43.09±3.94                    | 43.06±3.35                  | 43.06±3.73                   | 43.04±4.61                  |
| 28.                | 1041.58±137.20 <sup>a,x</sup> | 1007.88±105.72              | 958.00±166.41 <sup>b,x</sup> | 925.42±131.11 <sup>y</sup>  |
| 42.                | 1847.50±149.86 <sup>a</sup>   | 1778.50±143.02 <sup>a</sup> | 1655.50±191.40 <sup>b</sup>  | 1678.50±206.89 <sup>b</sup> |
| 42**               | 1847.50±227.69 <sup>a,x</sup> | 1837.00±152.83 <sup>b</sup> | 1820.50±172.21 <sup>y</sup>  | 1532.00±162.04              |
| Weight gain, g/day |                               |                             |                              |                             |
| 1-28.              | 35.66±4.77 <sup>a,x</sup>     | 34.53±3.62                  | 32.79±5.85 <sup>b,x</sup>    | 31.51±4.53 <sup>y</sup>     |
| 28-42.             | 57.36±4.19 <sup>a</sup>       | 56.07±7.09 <sup>a</sup>     | 51.71±7.40 <sup>a</sup>      | 53.08±9.43 <sup>b</sup>     |
| 28-42**            | 57.26±8.16 <sup>a,x</sup>     | 57.76±4.68 <sup>b</sup>     | 57.48±5.90 <sup>y</sup>      | 44.19±4.24 <sup>y</sup>     |

\*Values expressed as X±Sd \*\*Uncontaminated feed

<sup>a, b, c</sup>Mean values within column with unlike superscript letters were significantly different (p<0.05, LSD test)<sup>x, y, z</sup>Mean values within column with unlike superscript letters were significantly different (p<0.01, LSD test)

Although body weight can be valuable for estimation of quality and nutritive value of the feed, daily weight gain is a more reliable parameter (table 3). The first decline of body weight gain was observed after two and three weeks of treatment with 1000 or 100 ppm lead, respectively. After the first phase of fattening with contaminated feed, the daily weight gain differences established between control and experimental groups fed with higher dietary lead levels were highly significant ( $p < 0.01$ ). As previously mentioned further consumption of contaminated feed caused a greater decline of body weight gain, which was also observed in the first group. The weight gain depression was moderated following the exclusion of the contaminated feed from the diet, but only after a two-week period of recovery from the lower dietary lead level. The phenomenon of compensatory growth could be seen in this group after two weeks of using uncontaminated feed.

Use of lead contaminated feed resulted in a greater feed intake in the experimental groups, compared to the control group (table 4). This was probably a result of higher loss of wasted feed in proportion to the dietary lead levels. In the second phase of fattening the observed differences between the first two experimental groups were not dose-dependent, but the waste of feed in the third experimental group was more peculiar and exceeded that of the control group by 20 per cent.

Table 4. Feed intake and feed: gain ratio

| Trial days         | G r o u p |        |        |         |
|--------------------|-----------|--------|--------|---------|
|                    | K         | 0 - I  | 0 - II | 0 - III |
| Feed intake, g/day |           |        |        |         |
| 1-28.              | 60.86     | 62.62  | 63.34  | 66.33   |
| 28-42.             | 122.35    | 134.14 | 133.14 | 147.86  |
| 28-42.**           | 122.90    | 124.21 | 122.28 | 146.43  |
| Feed: gain ratio   |           |        |        |         |
| 1-28.              | 1.69      | 1.81   | 1.94   | 2.12    |
| 28-42.             | 2.15      | 2.35   | 2.31   | 3.35    |
| 28-42.**           | 2.15      | 2.24   | 2.36   | 2.27    |

\*\* Uncontaminated feed

Feed:gain ratio (Table 4) is a derivation of daily gain and feed consumption, representing at the bottom line, one of the best markers of feed quality. The feed: gain ratio was increased by 7-25% in the first phase of the trial, following the dietary lead levels in the contaminated feed. In the second phase, further consumption of contaminated feed caused higher feed: gain ratio, but the difference between the first two experimental groups did not depend on dietary lead level, while in the third experimental group a significant increase of feed gain ratio was recorded. Excluding the contaminated feed from the diet moderated the increase of feed gain ratio and the differences between experimental groups were not dose dependent.

Increasing dietary lead level was followed by a linear decrease of broiler performance (Berg et al., 1979), even at 100 ppm. The poorer performance caused by the dietary lead was meliorated after excluding the contaminated feed from the diet of broilers, but only after a certain period which depended on the dietary lead level and on satisfaction of broiler requirements, especially in calcium (Berg et al., 1979) and methionine (Latta and Donaldson, 1986). Thus, early detection of lead in feed and elimination of contaminated mixtures from the diet can diminish the negative effects on performance and reduce the losses to an acceptable level.

Lead acts as a cumulative toxin (Bakalli et al., 1995) and tends to accumulate in the bones. Consequently, the majority of body lead (about 90%) can be found in the skeleton (Shroeder and Tipton, 1986, Rabinowitz et al. 1976) and appears to be relatively immobile. The results from this study could be explained by relative mobilization of accumulated lead. Other factors accounting for the difference between data obtained in this and in earlier studies could be the duration of the trial which was much longer than most.

#### REFERENCES

1. AOAC 1980. Official Methods of Analysis. 14<sup>th</sup> ed, Washington D. C.
2. Bafundo, K. W., Baker, D. H., Fitzgerald, P. R. (1984): Lead toxicity in the chick as affected by ...p173Xess copper and zinc and by *Eimeria acervulina* infection. *Poult. Sci.*, 63, 1594-1603.
3. Bakalli, R. I., Pesti, G. M., Regland, W. I. (1995): The agnitude of lead toxicity in broiler chickens. *Vet. Hum. Toxicol.* 37, 15-20.
4. Berg, L. R., Nordstrom, O. J., Ousterhout, L. E. (1980): The prevention of chick growth depression due to dietary lead by increased dietary calcium and phosphorus levels. *Poult. Sci.*, 1860-1863.
5. Damron, B. L., Simpson, F. C., Harms, H. R. (1969): The effect of feeding various levels of lead on the performance of broilers. *Poult. Sci.*, 48: 1507-1508.
6. Doyle, J. J. (1979): Toxic and essential elements in bone - a review. *J. Anim. Sci.*, 49, 482-497.
7. Latta, M. Dorothy, Donaldson, W. E. (1986): Lead toxicity in chicks: Interaction with dietary methionine and choline. *J. Nutr.*, 116, 1561-1568.
8. Morgan, G. W., Edens, F. W., Thaxton, P., Parkhurst, C. R. (1975): Toxicity of dietary lead in Japanese quail. *Poult. Sci.*, 54, 1636-1642.
9. Neathery, M. W., Miller, J. W. (1975): Metabolism and toxicity of cadmium, mercury and lead in animals: A review. *J. Dairy Sci.*, 58, 1767.
10. National Research Council (1972): Lead: Airborne Lead in Perspective. National Academy of Sciences, Washington, D. D.
11. National Research Council (1989): Mineral Tolerance of Domestic Animals. National Academy of Sciences, Washington, D. C.
12. Paone, J. (1970): Mineral Facts and Problems. Bureau of Mines Bull. 650. U.S. Department of the Interior, Washington, D. C.
13. Rabinowitz, M., Wetherill, G. W., Kopple, J. D. (1976): Study of human lead metabolism by use of stable isotope traces. *Environ. Health Prospect.*, 7, 145-153.
14. Schroeder, H. A., Tipton, J. H. (1968): The human body burden of lead. *Arch. Environ. Health*, 17, 965-972.
15. Simpson, C. F., Damron, B. L., Harms, H. R. (1970): Abnormalities of erythrocytes and renal tubules of chicks poisoned with lead. *Am. J. Vet. Res.*, 31: 515-523.

16. *Snedecor, W. G., Cochran G. W. (1971): Statistical Methods. The Iowa State. Univ. Press.*
17. *Underwood, E. J. (1977): Trace Elements in Human and Animal Nutrition, 4th ed., Academic Press, New York.*
18. *Vengris, V. E., Mare, C. (1974): Lead poisoning in chickens and the effect of lead an interferon and antibody production. Can. J. Comp. Med., 38, 328-335.*

#### UTICAJ ISHRANE BROJLERA SMEŠAMA SA RAZLIČITIM KOLIČINAMA OLOVA NA PROIZVODNE REZULTATE

Z. SINOVEC, LJILJANA JANKOVIĆ, BRANA RADENKOVIĆ, N. JOVANOVIĆ

#### SADRŽAJ

Ogled je izveden na ukupno 94 Hybro brojlera podeljenih u četiri grupe, a trajao je 42 dana. Sve grupe brojlera hranjene su komercijalnim smešama, a olovo je dodato u formi olovo-acetata u količini dovoljnoj da obezbedi 0; 9,2; 92,0 ili 920,0 mg Pb/kg hrane. Nakon 28 dana, svaka grupa je podeljena u dve podgrupe. Jedna podgrupa je hranjena kontaminiranim smešama do kraja ogleda, a druga hranom bez dodatog olova.

Pad proizvodnih rezultata upravo proporcionalno je vezan sa sadržajem olova u hrani, čak i pri količinama od 100 ppm. Slabiji proizvodni rezultati izazvani prisustvom olova u hrani mogu se ublažiti isključivanjem kontaminirane hrane, ali tek nakon određenog perioda koji, pre svega, zavisi od sadržaja olova u hrani. Rana detekcija prisustva olova u hrani i isključivanje iz upotrebe može proizvodne gubitke svesti na prihvatljiv nivo.

