- 12. R. E. Beyer, Fed. Proc., 19, Suppl. 5, 145-151 (1960); Fed. Proc., 22, 874 (1963).
- 13. B. Chance, G. Hollunger, and B. Hagihara, Biochem. Biophys. Res. Commun., 8, 180 (1962).
- 14. N. Zuntz and S. Schumberg, Studien zur Physiologie des Marsches, Berlin (1901).

## EFFECT OF VITAMIN D INTAKE ON LEAD ACCUMULATION IN CHICKENS

R. E. Andrushaite, V. K. Bauman, and Academician A. R. Val'dman\*

UDC 546.815:577.161.2

KEY WORDS: lead; vitamin D; lead content in tissues; chickens.

The action of lead on animals is determined not only by the degree of pollution of the external environment and the quantity of lead directly entering the body, but also by the physiological state of the animal and its intake of certain other substances [3]. It has frequently been shown that the toxic action of lead is potentiated by a diet deficient in calcium and phosphorus [7, 8].

The question of the role of vitamin D in the toxic effect of lead has not yet been settled. Besides data showing that rats receiving a diet with vitamin D absorb more lead than animals reared on a diet deficient in this vitamin [9] there have been observations showing that the blood lead concentration of children is increased in the presence of vitamin D deficiency [10].

The object of this investigation was to make a comparative study of the accumulation of lead in animals with a deficient vitamin D intake. Chickens, which are a convenient model with which to study the action of vitamin D, were used in the experiments [1].

## EXPERIMENTAL METHOD

The chickens were reared in battery cages with free access to food and water. The basic diet (BD) contained the following components (in %): cornflour 43.0, barley flour 24.5, casein 10.0, sunflower waste 17.0, yeast 3.0, chalk 1.0, bone meal 1.0, common salt 0.5.

Chickens kept on this diet develop characteristic signs of rickets by the 21st-25th day of life: retardation in growth, untidy plumage, softness of the beak, a low serum calcium and phosphorus level, and absence of calcium-binding protein in the duodenum [1]. The chickens received vitamin D from the first days of life with the diet in a dose of 500 i.u./kg (BD + D) or as single intramuscular injections (500 i.u. in 0.1 ml propylene glycol) 72 h before sacrifice. The addition of lead in doses of 40, 100, and 200 mg/kg diet was made in the form of a solution of lead acetate.

The chickens were killed at the age of 25 days and lead was determined in the tibia, kidneys, and liver after incineration at 600°C and dissolution in 6 M hydrochloric acid; the blood lead level also was determined [4] with a Perkin-Elmer 403 atomic absorption spectrometer. All the data given below are mean values of 8-12 individual determinations.

## EXPERIMENTAL RESULTS

The results confirm the known fact [2] that when the diet is deficient in vitamin D the live weight of chickens is on average 40% lower than in the control group (BD + D). The addition of lead caused some tendency for the weight of the chickens to decrease, but no statistically significant changes were observed (Table 1).

With an increase in the lead content in the diet, the blood lead level of the chickens rose (Table 2). However, this relationship was not linear, for with higher doses of lead in

\*Academy of Sciences of the Latvian SSR.

Laboratory of Biochemistry and Physiology of Animals, Institute of Biology, Academy of Sciences of the Latvian SSR, Salaspils. Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 93, No. 2, pp. 30-32, February, 1982. Original article submitted July 28, 1981.