neither loss of the nervous layer nor mere thinning of the presumptive epidermis are responsible for its loss of competence.

Summary. 1. The anterior and posterior presumptive epidermis of the early gastrula of *Hyla regilla* possess essentially the same degree of competence for neural plate formation. 2. In the middle and late gastrula stages the posterior presumptive epidermis is less competent than the anterior. 3. Thinning of the ectoderm by removal of at least a large part of its inner layers of cells (the so-called nervous layer) in the advanced blastula stage does not impair the competence of the ectoderm.

9883 P

Influence of Vitamin D in Experimental Lead Poisoning.

ALBERT E. SOBEL, OSCAR GAWRON AND BENJAMIN KRAMER.

From the Pediatric Research Laboratory and the Division of Biochemistry, The Jewish Hospital of Brooklyn.

In the present investigation a study was made of the effect of the antirachitic vitamin upon the concentration of lead in the blood and upon the amount of lead deposition in the bones. This was undertaken because it has been noted previously that the severity of both experimental and clinical lead poisoning is increased under the influence of vitamin D,[1, 2, 3] or during the summer months when the sunlight is rich in ultraviolet rays.

A series of young rats, 23-26 days of age, was placed on a lead-containing diet. Half of these animals received approximately 33 Steenbock units of viosterol in halibut liver oil daily (Mead Johnson) after the first 5 days on the lead-containing diet. The composition of the diet is given in Table I. It is essentially a rickets-producing diet where 3% of $Pb(OH)_2 . 2PbCO_3$ was substituted for 3% of $CaCO_3$. At the end of the experimental period the concentration of lead in the whole blood and the amount deposited in the bones was determineed. The results of the experiments are presented in Tables II and III.

1 Shelling, D. H., PROC. SOC. EXP. BIOL. AND MED., 1932, **30**, 248; Shelling, D. H., and Hopper, K. B., *Bull. Johns Hopkins Hospital*, 1936, **58**, 193.

2 Krehbiehl, O. F., personal communication to Dr. Shelling from Dr. C. E. Willoughby of the Chemical Experiment Station, E. I. DuPont de Nemours Company.

3 Blackman, S. S., *Bull. Johns Hopkins Hosp.*, 1937, **61**, 1.

TABLE I.

Diet.
70 parts Corn Meal (Quaker Oats)
16 " Wheat Gluten
10 " Brewer's Yeast (Mead's)
3 " $2(PbCO_3)Pb(OH)_2$
1 " NaCl
Ca = 0.03 g. per 100 g. of diet
Pb = 2.4 " " " " " "
= 11.6 mM. " " " " "
P = 0.246 g. " " " " "
= 7.9 mM. " " " " "

TABLE II.
Experiment I (Duration—22 days).

	Lead Diet	Lead Diet + Vitamin D
Number of animals	8	8
Avg. change in wt., g.	+3.3	+1.3
Avg. wt. fat free dry femurs, mg.	63.3	66.9
Avg. wt. ashed femurs, mg.	17.9	23.2
Avg. % ash of femur	28.3	34.7
Avg. lead in femurs, mg.	0.101	0.285
Avg. % lead in ashed femur	0.56	1.23

TABLE III.

	Lead Diet	Lead Diet + Vitamin D
Experiment II (Duration—34 Days).		
No. of animals	5	8
Avg. change in wt., g.	+1.4	—0.8
Avg. wt. of lead in tibia, mg.	0.18	0.30
Avg. lead in tibia, %	0.34	0.57
Avg. lead in whole blood, mg. per 100 cc.	1.25	2.13
Experiment III (Duration—23 Days).		
No. of animals	11	10
*Avg. change in wt., g.	—1.6	—3.0
Avg. lead in whole blood,	0.65	1.04
Mg. per 100 cc.	0.71	1.30
Ca, mg. %	8.9	8.7
P, mg. %	3.1	6.2
Ca × P	27.6	54.0

*Divided into two groups.

From these results, a definite influence of the antirachitic vitamin may be readily observed. Both the lead concentration in the blood and the amount of lead present in the bones was about twice as high in the vitamin-fed animals as in those not receiving this addition. These findings help to explain the previously observed increased severity of lead poisoning under the influence of vitamin D or ultraviolet rays, for with increased lead levels in the blood, one usually finds more acute symptoms of plumbism.

The relation of higher concentrations of lead in the blood to in-

creased deposition in the bones is not wholly unexpected. Such phenomena are observed in the deposition of inorganic bone normally. As a rule, the higher the concentration of the bone forming elements in the blood plasma the greater the ease of deposition. Lead in this case may be considered as one of the bone-forming elements, *i. e.*, a part of the inorganic matter of bone, although not a normal one. The mechanism of deposition of lead salts seems to follow the phenomena observed with the normal bone-forming constituents, namely, calcium and phosphate, *i. e.*, the higher the value of the product formed by multiplying the concentrations of these two constituents in the blood serum, the greater the ease of deposition. Another parallel worth mentioning is that vitamin D, as a rule, is instrumental in raising the above product and thereby promotes the normal formation of inorganic bone, just as it promotes deposition of lead in the bone.

Conclusions. Vitamin D causes a rise in the concentration of lead in the blood stream and in the bones of rats suffering from lead poisoning.

9884 P

Influence of Dietary Calcium and Phosphorus upon Action of Vitamin D in Experimental Lead Poisoning.

ALBERT E. SOBEL, IRVING B. WEXLER, DAVID D. PETROVSKY AND BENJAMIN KRAMER.

From the Pediatric Research Laboratory and the Division of Biochemistry, The Jewish Hospital of Brooklyn.

In the preceding paper[1] a marked effect of vitamin D on the blood and bone lead concentrations of lead-fed animals was demonstrated. In view of the great importance attached to the dietary calcium and phosphorus content in lead poisoning[2, 3, 4] investigations were un-

1 Sobel, A. E., Gawron, O., and Kramer, B. K., PROC. SOC. EXP. BIOL. AND MED., 1938, **38**, 433.

2 Shelling, D. H., PROC. SOC. EXP. BIOL. AND MED., 1932, **30**, 248; Shelling, D. H., *The Parathyroids in Health and in Disease*, St. Louis, 1935, C. V. Mosby Company; Shelling, D. H., and Hopper, K. B., *Bull. Johns Hopkins, Hosp.*, 1936, **58**, 137.

3 Gray, I., *J. Am. Med. Assn.*, 1935, **104**, 200.

4 Aub, J. C., *J. Am. Med. Assn.*, 1935, **104**, 87; Aub, J. C., Fairhall, L. T., Minot, A. S., and Reznikoff, P., *Lead Poisoning-Medicine Monographs*, Baltimore, Williams and Wilkins Co., 1926, 7.