Summary. In confirmation of Klieneberger's observation, the L-organism was cultivated from rats. It was also cultivated from a human suppurating lesion. The strains were morphologically and culturally similar to those of Klieneberger, and maintained their character during 2 to 5 months' observation. The L-organism exerts a definite pathogenic action on mice.

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Effects of Minute Amounts of Lead in the Diet of the Dog.

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The observation that 100 mg. of lead per kilo of diet did not cause any apparent change in the growth, blood and reproductive performance of the rat,[1] led to the extension of the study to another species, namely, the dog. Two litters of 3 puppies each were divided into 3 groups, so that one dog from each litter was in each group, and fed a diet containing 41.2% casein, 29.4% sucrose, 18.3% lard, 7.2% butter, 2.6% bone ash and 1.3% of Cowgill-Karr salt mixture.[2] Seventy grams of dried yeast and 10 gm. of cod liver oil were added to each kilo of diet. Measured amounts of lead acetate were mixed with the salts so that the increment of lead for each group was 0, 25 and 100 mg. of lead per kilo of diet, respectively. The puppies grew well on this ration. Litter I, born of a 12-kilo mongrel, gained an approximate average of 1500 gm. per month and litter II, born of a fox terrier, gained about 700 gm. per month. The added lead in the diets of groups 2 and 3 did not seem to affect the comparative growth of the dogs. One dog in group 3 was a bit smaller than its litter mates, but this animal was quite plump and its decreased size probably was due to the fact that it had more of a smaller breed in its genetic constitution.

Weekly examinations were made of the blood of each dog for hemoglobin content, erythrocyte count and basophilic granulations (stippling) when stained with Wright's blood stain.

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1 Horwitt, M. K., and Cowgill, G. R. In process of publication, Reported at Meetings of the American Society of Biological Chemists at Memphis, April, 1937.

2 Cowgill, G. R., *J. Biol. Chem.*, 1923, **56**, 725.

After 225 days on the experimental diet, the hemoglobin contents of the blood of the dogs in gm. % were 13.7 and 13.3 for group 1, 13.7 and 13.3 for group 2, and 12.3 and 12.2 for group 3. The erythrocyte count expressed in millions of cells per cubic mm. of blood was 6.30 and 6.10 for group 1, 6.60 and 6.80 for group 2, and 8.91 and 7.59 for group 3. No stippled cells were found until after 2 months on the experimental diet when a considerable amount of basophilic stippling became apparent in both dogs from group 3 (102 mg. per kilo). This apparent disagreement with the suggestion by Aub, *et al.*,[3] that dogs' blood does not stipple may be explained by the fact that we were dealing with puppies which because of their youth, were probably more susceptible to lead. No stippled cells were found in the blood of the dogs in groups 1 and 2.

The observation that the comparative increase in erythrocytes in group 3 was not associated with a corresponding increase in the hemoglobin content of the blood indicates that the individual erythrocytes were deficient in hemoglobin.

After 7 months on the experimental diet one of the dogs in group 3 became ill quite suddenly with bloody diarrhea and severe colic contractions. The dog was sacrificed, a rib removed for lead analysis, and representative sections of the brain, lung, kidney, and intestines saved for pathological analysis. A few days later the 2 dogs on the control diet developed an upper respiratory infection. At this time all of the remaining 5 dogs were sacrificed and sections of their organs saved for analysis. Table I presents the results of analysis of samples of blood taken from the dogs prior to death together with the results of the lead analysis of the ribs.[4]

The animal from group 3 which became ill had 230 mg. of lead per kilo of bone and had ingested the highest amount of lead. This was the only animal whose tissues on examination showed any pathological changes that might be interpreted as lead poisoning and even in this animal the changes were doubtful.

TABLE I.
Comparison of Lead Content of Blood and Bones with Amounts of Lead Ingested.

	Group 1 2 mg./kilo		Group 2 27 mg./kilo		Group 3 102 mg./kilo	
Mg. lead per kilo of blood	.40	.36	.62	.45	.67	.72
Mg. lead per kilo of bone	6.1	13.5	36.7	69.8	230.	198.
Gm. lead ingested	.094	.066	1.269	.756	4.59	2.86
Age of dogs in days	232.	292.	232.	293.	226.	295.
Final wt., kg.	12.5	5.0	12.8	5.5	9.3	5.0

[3] Aub, C. A., Fairhall, T., Minot, A. S., and Reznikoff, P., *Medicine Monographs*, 1926, **7**, 139.

[4] Description of authors' method to appear in *J. Biol. Chem.*, July, 1937.

Except for the appearance of occasional stippled erythrocytes, the other dog in group 3 was apparently normal. A neurological examination† failed to show anything abnormal in any of the dogs. X-ray examination of the bones of all the animals was made during various stages of growth in an effort to detect lead deposition, but it was not possible to notice any significant differences between the X-ray photographs of the dogs.

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Effect of Vitamin E Deficient and Muscular Dystrophy-Producing Diet on Metabolism of Guinea Pigs.

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It has been found[1,2] that excised skeletal muscle from animals exhibiting nutritional muscular dystrophy shows an increased consumption of oxygen. The thyroid glands from rats deprived of vitamin E are markedly hypoplastic.[3] Endocrine gland disturbances have frequently been observed in clinical muscular dystrophy.

This report is concerned with the results of metabolic rate determinations made upon guinea pigs kept on diet 13 of Goettsch and Pappenheimer[4] supplemented by 8 cc. of fresh tomato juice daily. This diet is deficient in vitamin E and also allows the development of muscular dystrophy. Ten young guinea pigs (5 male and 5 female) served as experimental animals. Control guinea pigs of the same age were selected from the same stock and kept upon a diet of grains, alfalfa and lettuce. Measurements of oxygen consumption were made at a room temperature of 28°C. in a multiple closed circuit apparatus (Benedict).[5] The determinations were made at approximately one week intervals. A total of 116 determinations were made on the experimental animals and 55 on the control group. Food was withheld for 24 hours before each experiment. The presence of the typical lesions of experimental muscular dystrophy was established by examination of the skeletal muscle removed at necropsy.

† We are indebted to Dr. James C. Fox, Jr., for making this examination.

1 Victor, J., *Am. J. Physiol.*, 1934, **108**, 229.

2 Madsen, L. L., *J. Nutrition*, 1936, **11**, 471.

3 Singer, E., *J. Physiol.*, 1936, **87**, 287.

4 Goettsch, M., and Pappenheimer, A. M., *J. Exp. Med.*, 1931, **54**, 145.

5 Benedict, F. G., *J. Nutrition*, 1930, **3**, 161.