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.

E. L. WOOD AND H. M. HINES.

From the Department of Physiology, State University of Iowa.

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.

TABLE I.

Exper. Condition	Days on Diet	Body wt. in gm. Initial	Maximum	Terminal	Ce. O_2 used per M^2 of Surface Area per Hr. Initial	Final	Aver. all tests
Controls	118	258	511	495			6278
Males, Diet 13	69	342	422	386	6897	6574	6314
Females, Diet 13	102	278	412	390	6193	6645	6433

The data (Table I) show that no significant difference exists between the metabolic rates, expressed as cc. of O_2 used per hour per square meter of surface area, of control and dystrophy animals. Furthermore, the rates of oxygen utilization by the animals in the early days of the experiments were essentially the same as that found in the terminal experiments on the same animals.

If an endocrine disturbance existed in such animals, reared under the above conditions, it was not of such a nature or degree as to be reflected in their rates of oxygen utilization. These observations on the intact animal offer no explanation as to the cause of the increased oxygen consumption by excised skeletal muscle from animals under similar dietary conditions.

Summary. Guinea pigs reared on a vitamin E deficient diet and exhibiting typical experimental muscular dystrophy show normal metabolic rates as determined by oxygen consumption measurements.

9383 P

Recovery of Influenza Virus from Chicago Epidemic.

J. MARION CLAMPIT AND F. B. GORDON. (Introduced by G. M. Dack.)

From the Department of Bacteriology and Parasitology, University of Chicago.

In the original isolation of the virus of human influenza, ferrets were used as the experimental animal.[1] Later it was found that after a few passages in ferrets the virus became pathogenic for young white mice.[2] Since some of the strains required only one or 2 ferret passages before becoming pathogenic for mice, numerous attempts have been made to infect mice by means of material taken directly from patients. Francis and Magill[3] have recently succeeded

1 Smith, W., Andrews, C. H., and Laidlaw, P. P., *Lancet*, 1933, **2**, 66.

2 Andrews, C. H., Laidlaw, P. P., and Smith, W., *Lancet*, 1934, **2**, 859.

3 Francis, T., and Magill, T. P., PROC. SOC. EXP. BIOL. AND MED., 1937, **36**, 132.