24 and 48 hours after the time of injection.

Results and discussion. Within one to 2 minutes following injection of dimethyl selenide near the median lethal dose, the animals entered a state of hyperpnea with the elimination of an overwhelming garlic-like odor on the breath. Convulsions in the mice, but not in the rats, were common. This was usually followed by exitus within a few hours, with the fatal doses, although some animals died as late as 36 hours after injection. In all cases, the signs of hyperpnea were gone after the first 2 or 3 hours.

Median lethal doses (L.D. 50) for the rat and mouse at 24 hours were determined (13,14) by plotting per cent mortality on probit against log dosage graph paper. It was found that the L.D. 50 for 24 hours for the mice was 1.3 g Se as dimethyl selenide (1.8 g dimethyl selenide) per kg of body weight. That for the rats was 1.6 g of Se as dimethyl selenide (2.2 g dimethyl selenide) per kg of body weight. It will be noted that these figures are several times greater than the minimum fatal doses for either sodium selenate (5.25 to 5.75 mg Se per kg)(2), or selenium-cystine (4 mg Se per kg of body weight)(4).

In relation to the selenium detoxification mechanism, it is of particular interest to point out that after the administration of sodium selenate, which is a relatively toxic compound, selenium rapidly appears in the respiratory gases(15) in the form of dimethyl selenide (10), which is a sparingly toxic compound. Thus, it would appear that the animal organism when treated with a toxic form of selenium, converts it in part to a less toxic compound which is readily excreted via the lungs.

Summary. The median lethal dose (L.D. 50) of dimethyl selenide was determined for the mouse and rat. It was found, after intraperitoneal injection of dimethyl selenide, that the median lethal dose for the mouse was 1.3 g of Se as dimethyl selenide (1.8 g dimethyl selenide) per kg of body weight, and 1.6 g Se (2.2 g dimethyl selenide) per kg of body weight for the rat.

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Leucocytosis in Vitamin E Deficient Rabbits.* (19334)

JAMES S. DINNING.

From the Department of Biochemistry, School of Medicine, University of Arkansas, Little Rock.

Previous reports from this laboratory have indicated an effect of vit. E deficiency on nucleic acid metabolism(1,2). It was also noted that E-deficient monkeys exhibited a leucocytosis which responded to therapy with alpha tocopherol(1). This paper reports the results of a study of the effect of vit. E deficiency on the peripheral leucocytes of rabbits. The data to be presented show that E-deficient rabbits exhibit a leucocytosis which

TABLE I. Peripheral Leucocytes in Control and Vitamin E Deficient Rabbits.

Diet	No. of rabbits	Peripheral leucocytes, thousands/μl, mean $\pm$ stand. error		
		Total WBC	Lymphocytes	Granulocytes
Chow	10	10 $\pm$ 1.1	5.8 $\pm$ .7	3.5 $\pm$ .6
Ferric chloride treated — E	4	25.9 $\pm$ 6.9	4 $\pm$ .8	19.5 $\pm$ 7
" " " + E	3	5.8 $\pm$ .5	2.8 $\pm$ .3	2.6 $\pm$ .2
Purified — E	7	22.4 $\pm$ 1	3.7 $\pm$ .7	17.1 $\pm$ 1.1
" + E	5	10 $\pm$ 1.3	5.3 $\pm$ .7	3.8 $\pm$ 1.3

is the result of a greatly increased number of circulating granulocytes.

Materials and methods. White New Zealand rabbits of both sexes and weighing approximately one kilogram were placed on the experimental diets. In one series the rabbits were given the ferric chloride treated diet described by Mackenzie and McCollum(3). In another series the animals were fed the purified diet previously described(2). Controls for both series were given the same diet plus supplements of 2 mg of alpha tocopherol acetate daily per kilo body weight. The supplement was administered orally from a corn oil solution by means of a syringe with a blunt needle. Additional controls were given a diet of commercial rabbit chow. *Blood counts* were taken after the rabbits receiving the deficient diets were unable to right themselves when placed on their sides. Controls were bled at the same time. Rabbits exhibited this symptom of muscular dystrophy after 2 to 4 weeks on the deficient diets. Blood counts were made by standard hematological procedures on blood taken from the marginal ear vein. There were no differences in red cell counts, hemoglobin, or hematocrit values and the data are not presented.

Results and discussion. The data in Table I show the circulating granulocytes are greatly elevated in vit. E deficient rabbits when compared either to their respective controls or to rabbits receiving the chow diet. In recovery experiments it has been found that when the deficient rabbits are recovered with alpha tocopherol the peripheral blood picture returns to normal.

These experiments do not prove that the leucocytosis which is exhibited by rabbits suffering from nutritional muscular dystrophy is a direct result of vit. E deficiency. The peripheral leucocytosis may merely reflect a leucocytic infiltration of the dystrophic muscle and not be specific for E deficiency. The elevated peripheral white cell count is of some interest however since it has been shown that the deficient rabbits synthesize extra amounts of creatine(4) and data from this laboratory have suggested a relationship between creatine and white blood cell production(5,6).

Summary. The peripheral granulocytes are greatly elevated in vit. E deficient rabbits.

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