bers of worms were recovered from the cortisone-treated mice than from the untreated control mice. It is possible, but not likely, that worms may have ended up in other portions of the vascular system where they could have been overlooked; they did not accumulate in spleen, lungs or kidneys. This apparent strengthening of immunity stands in sharp contrast to findings with 2 nematode parasites, *Trichinella spiralis*(5,7) and *Nippostrongylus muris*(4), where cortisone treatment of the host weakens both natural and acquired resistances presumably because the treatment restrains the cellular phase of the immunity mechanism(8). By inference it is likely that in mice cellular defenses play little part in the initial phases of natural immunity to *S. mansoni*, that is during the period of the transient residence of the larva in the skin tissues. If the immunity strengthening effect observed here is a true one, then from the results of Experiment 3 where treatment ended on the 7th day after exposure to the parasite it would appear that the effect is exerted in the very early stage of infection and may be related to an inhibition of skin penetration and/or further migration by the larvae. Cercariae are known to exhibit hyaluronidase activity(9), and cortisone is known to inhibit hyaluronidase(10). According to Lewert and Lee the infective larvae of both *S. mansoni* and *Strongyloides ratti* possess collagenase activity, and cortisone treatment of the rat host seemed to inhibit this activity of *S. ratti* and to cause longer retention of the young worms in the skin(11). These facts may not necessarily be related to the present findings, but certainly further investigation is in order on the effects of cortisone in schistosomiasis.

Summary. In 3 separate experiments small groups of mice of 2 strains were exposed to infection with *Schistosoma mansoni* and half of them were treated with cortisone beginning shortly prior to infection and ending on the 29th, 15th and 7th days after infection, respectively. The various regimens of cortisone treatment employed did not increase susceptibility to the parasite, as judged by numbers of adult worms recovered from hepatic portal tracts of the mice 7-8 weeks after infection. The treatments may have enhanced the natural immunity slightly, since significantly fewer worms were recovered from the cortisone-treated mice.

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Received July 8, 1957. P.S.E.B.M., 1957, v96.

Paper Electrophoresis of Muscle Fractions from Vitamin E Deficient Rabbits.* (23378)

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These studies were undertaken in an attempt to correlate vit. E avitaminosis with changes in muscle proteins and excretion of urinary nitrogen components, such as creatine, allantoin and amino acids. The easily soluble

* This investigation was supported by National Muscular Dystrophy Research Foundation Inc., and Muscular Dystrophy Associations of America, Inc.

TABLE I. Average Daily Food Consumption and Weight Gain Data.

	Vit. E deficient (g)	Vit. E (g)
Food consumption	26.9	31.5
Wt gain	7.5	15.8

muscle proteins were studied by means of paper electrophoresis. This method was found to be sufficiently sensitive to differentiate muscle from dystrophic and control animals.

Experimental. Three pairs of male littermate California Giant rabbits weighing about 800 g were divided into 2 groups, one of which was fed a vit. E deficient diet and the other a vit. E supplemented diet. To this commercial diet† was added 2% codliver oil. Three milligrams of dl-α-tocopherol phosphate disodium salt per 25 g of the diet was fed on alternate days to the control group. The vit. E deficient group received the unsupplemented diet. The average daily food consumption and weight gain appears in Table I.

When the vit. E deficient animal of each pair showed typical signs of vit. E deficiency (1) the pair of animals was sacrificed by bleeding under anesthesia (ether). The eviscerated carcass was stored in the cold room and within 10 days striated muscle was removed from the right rear leg and ground in a small hand meat grinder. Twenty-five gram portions of ground muscle were extracted with 25 ml of distilled water, for 2 hours, at room temperature with occasional stirring. The mixture was then filtered through triple thickness gauze, centrifuged for 30 minutes at 450 x g. The supernatant fluid was recovered for the electrophoretic examination.

Filter paper electrophoresis was carried out using a glass plate horizontal electrophoresis apparatus and Whatman No. 3 filter paper with a barbiturate buffer pH 8.6 containing sodium diethyl barbiturate (20.6 g/l) and diethylbarbituric acid (3.68 g/l). Ten strips were run simultaneously using 20 lambda of the solution spotted at the midpoint of the strip. A potential of 450 volts with a current of 12 to 15 ma was applied for 3 hours, giving a separation of 5 to 6 cm between the more rapidly moving and the more slowly moving components. The strips were oven dried at 110°C for 30 minutes, then stained with a 1% solution of bromphenol blue in 95% ethyl alcohol saturated with mercuric chloride. The strips were placed in the dye bath for 5 minutes and washed free of excess dye by four 5 minute washings with methyl alcohol containing 1% mercuric chloride, with frequent agitation of the container. Finally, the strips were washed with methyl alcohol to remove the mercuric chloride and air dried on a clean dry cloth.

A line was drawn through the center of the strip lengthwise and was ruled off every ⅛ of an inch and optical density was determined at each intersection of the lines in a Photovolt Densitometer 52c without a filter. The results of such an analysis appear in Table II. These data show a significant difference in distribution of protein between the dystrophic and the control groups in the fast moving component and a significant difference in the slow moving component.

The electrophoresis pattern of the water extracts of a similarly treated pair of animals was stained and cut each ⅛", and was eluted with 3 ml of 4% sodium carbonate in 50% methyl alcohol for 2 hours. The optical density of the eluate was read at 540 mμ and optical density x 1000 was plotted on the ordinate and the fraction number on the abscissa in Fig. 1.

Discussion. The findings reported here, using a water extraction procedure, showed that vit. E deficiency caused a change in the electrophoretic pattern of the easily soluble muscle proteins. Amount of nitrogen extracted from the muscles was the same in the control and the dystrophic group. Haan(2),

TABLE II. Optical Density of Water Extract of Muscle Electrophoretic Pattern.

	Vit. E deficient	Vit. E
Total optical density	5.14	5.17
% of total		
Fast moving	31.1	23.3
Slow "	68.8	76.6
2P (comparison of slow moving)=	.025	
2P (" " fast ")=	.02	

† Nutritional Biochemicals, Cleveland, Ohio.

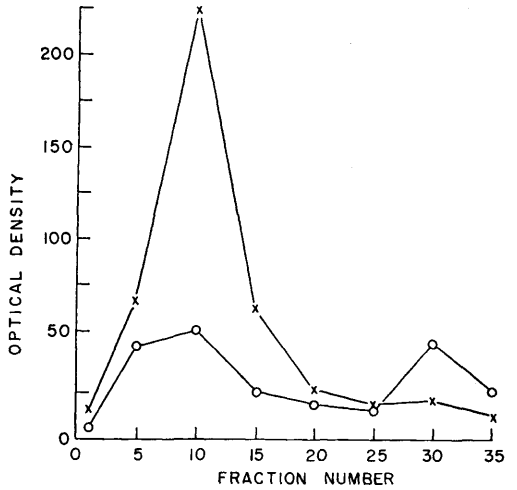

FIG. 1. Electrophoresis of aqueous extract of muscle from vit. E supplemented (×) and vit. E deficient (○) animal.

employing a dilute salt extraction procedure found that there was an increase in percentage of the most rapidly moving component peak 11 and also that the amount of the fast moving component peak 11 was constant in both groups of animals.

Summary. Three pairs of male littermate rabbits were fed a commercial vit. E deficient and a vit. E supplemented diet. When the vit. E deficient member of each pair showed typical vit. E deficient symptoms, the pair of animals was sacrificed and the water soluble muscle proteins extracted from the striated muscle. The electrophoretic pattern of the deficient animals showed a significant change in distribution of water soluble proteins as compared to the control group. This change was an increase in the fast moving peak and a decrease in the slow moving peak.

The authors wish to thank Mrs. B. A. Sullivan for assistance in the care of the animals.

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Received July 2, 1957. P.S.E.B.M., 1957, v96.

Ethanol Metabolism in Vitamin Deficiencies. I. Niacin.* (23379)

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Westerfeld(1) recently reviewed the pathways of ethanol† metabolism, and more recently Schulman and Westerfeld(2) have proposed an additional possible pathway. Since the route of metabolism has not been definitely established, it seemed worthwhile to study the effects of various vitamin deficiencies on the rate of alcohol metabolism in an effort to evaluate the role of vitamins. The first step in alcohol metabolism appears to be its conversion to acetaldehyde probably by means of alcohol dehydrogenase with DPN acting as hydrogen acceptor. Since DPN is a niacin-containing coenzyme, this study was begun with niacin.

Methods. Weanling male rats of the Sprague-Dawley strain, 24 days old when received in this laboratory, were used throughout the experiments.

Since the rat can synthesize niacin from abundant dietary tryptophan, the tryptophan content of the diet was limited. Preliminary work in this field indicated that a true niacin deficiency could be induced in rats with the diet shown on the next page.

The control diet contained, in addition, 50 mg niacin. Some rats were maintained on a diet similar to the above except the casein-casein hydrolysate ratio was 80-120 instead of 100-100.

Immediately upon arrival of the rats in this laboratory, the animals were paired and individually caged, and the deficient group was allowed to eat the niacin deficient diet

* This investigation was supported by a research grant from the U. S. Public Health Service.

† Hereafter referred to as alcohol.