controls was virtually free from demonstrable parasites. Several of the biotin-deficient ducks infected with *P. cathemerium* died from the infection.

In both chickens and ducks, whether on a deficient or an adequate diet, the concentration of biotin in the plasma and red blood cells rose during the course of infection with P. lophurae. This rise can not be explained solely on the basis of the new red cells formed in response to the anemia produced by the parasites. In ducks, an increased biotin level was already apparent by the fourth day after inoculation, when there was as yet no large proportion of young red cells; the increase appeared in the plasma as well as in the red cells: and both plasma and red cells were back to a normal biotin level by the eighth day after inoculation, when a large proportion of young red cells was still present. Since P. lophurae multiplies to a greater extent in animals with a relatively low initial biotin level than in those with a higher initial biotin level, the increase in biotin which occurs during the course of the infection may well be concerned with the elimination of the parasites from the blood.

Whether these findings with avian malaria apply to simian or human malaria can be determined only by extended observations on body biotin levels in these species in relation to the degree of susceptibility to malarial infection. Certainly the results with chickens and ducks would indicate that biotin is one substance of known chemical nature which helps to determine the degree of resistance of the host to infection with malarial parasites. These results are also of interest in that they provide an example, in addition to the very few thus far discovered,<sup>7</sup> of a specific relation between a nutritional deficiency and susceptibility to an infectious disease. The full details of this work are to be published shortly.

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### RELATION OF FOOD INTAKE TO RESPONSE OF MICE INOCULATED WITH LANSING STRAIN OF MURINE POLIO-MYELITIS VIRUS<sup>1</sup>

In a recent preliminary communication<sup>2</sup> we reported that mice on a vitamin  $B_1$ -deficient diet showed increased resistance, over a period of 30 days, to the Lansing strain of murine poliomyelitis virus. Since then these observations have been confirmed, and in addition we have found that simple restriction of food intake will produce comparable results. In several trials, feeding of about 40 per cent. of the usual daily consumption definitely extended the time before the onset of paralysis and the time of death. To at least the twenty-first day after inoculation there was a statistically significant difference in deaths and cases of paralysis between the restricted groups and those fed ad libitum. This difference had disappeared by the twenty-seventh day.

In one experiment, 176 mice were divided into 6 groups. Group I received a synthetic diet (diet 100), Groups II and III a stock diet (diet 483) and Groups IV, V and VI a synthetic diet in which the relative amounts of all ingredients except carbohydrate were increased at the expense of the latter (diet 515). Groups I, II and IV were fed ad libitum and the other groups were given 1 gm of food per animal per day. On the third day of the experiment, Groups I to V inclusive were injected intracerebrally with a suspension of mouse brain infected with the Lansing strain of murine poliomyelitis virus. This amount of virus corresponded to between 500 and 1,000 fiftyper cent.-mortality doses. Group VI was injected with a suspension of normal brain. The cumulative percentages of animals dving and those showing paralysis by the tenth, fifteenth and twenty-first days after inoculation are given in Table 1. Any animals dying before the third day are not included in the totals.

Increasing the concentration of thiamin in the diet

TABLE	1
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Group No.	No. mice 3 days after inoc.	Diet No.	Amt. of diet	Inoculum	Days after inoculation					
					10		15		21	
					Par.1	Death	Par.1	Death	Par.1	Death
					Per cent.	Per cent.		Per cent.	Per cent.	Per cent.
II	$16 \\ 35 \\ 39 \\ 23 \\ 25 \\ 23 \\ 23 \\ 23 \\ 23 \\ 23 \\ 23$	100	ad lib ad lib	virus virus	88 80	$\begin{array}{c} 94 \\ 94 \end{array}$	88 80 33 96 32	100	88 80 56 96 52	$100 \\ 100 \\ 67$
III	39	$ar{483}{483}{515}$	$1  \mathrm{gm}$	virus	10	94 10	33	100 $28$	50 56	67
IV	23	515	ad lib	virus	91	10 87	96	100	9ĕ	100
vI	25	$515 \\ 515$	1  gm	virus	20	28	<b>32</b>	<b>48</b>	52	68
V I	23	919	$1 \mathrm{gm}$	normal brain	••	26	••	39	••	44

<sup>1</sup> Paralysis.

7''Nutrition and Resistance to Disease," Nutrition Reviews, 1: 66, 1943.

<sup>1</sup> Aided by a grant from the National Foundation for Infantile Paralysis, Inc.

<sup>2</sup> Proc. Soc. Exp. Biol. and Med., 51: 215, 1942.

so that the amount consumed by the animals on the restricted intake was at least double that of the animals on the unrestricted intake did not increase the incidence of paralysis or death. The administration of 0.5 ml of 0.3 per cent. saline twice daily by stomach tube to the mice on restricted intake, likewise did not significantly alter the results.

From the data it appears that restricting the intake of either the complete ration or just the carbohydrate

delays the manifestation in mice of infection with the Lansing strain of poliomyelitis virus.

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# SCIENTIFIC APPARATUS AND LABORATORY METHODS

# A SIMPLIFIED PROPYLENE GLYCOL DIS-PENSER FOR FIELD USE<sup>1</sup>

A CONSIDERABLE amount of work is in progress at the present time on the effectiveness of propylene glycol as an air disinfectant.<sup>2, 3</sup> This agent is commonly employed in concentrations ranging from 1 gram per 5 million cc of air to 1 gram per 20 million cc of air<sup>3, 4, 5</sup> and is most conveniently introduced into the atmosphere by vaporization.

Because some of the suggested vaporizing equipment is rather elaborate and is not suited to largescale field experiments, we have developed a simple device requiring no special materials for construction. It consists of an ordinary electric light bulb dipping into a beaker or tin can filled with propylene glycol. Preferably the unit is insulated to diminish heat loss by setting it in a larger container and packing paper into the space between the sides. A 10-inch electric fan is placed one or two feet away so that it directs an air stream across the liquid surface. The large heating area of the bulb eliminates the danger of local super-heating with consequent decomposition of the propylene glycol, and the inexpensiveness of the equipment makes it feasible to install as many units as may be necessary in order to maintain a given concentration of vapor.

In practice the rate of evaporation of propylene glycol from the vaporizers should be great enough to bring all the fresh air coming into the room to the concentration level desired. It is usually estimated that a closed room has 2 to 10 air turnovers per hour under ordinary circumstances. Therefore, if a room has a volume of 2,000 cubic feet and there are 5 air turnovers per hour it would require the vaporization

of 14 grams of propylene glycol per hour to maintain a concentration of 1 part propylene glycol in 20 million parts of air throughout the room. A single 50watt bulb immersed in 700 cc of propylene glycol with a surface area of 18 square inches accomplishes this. The output of a vaporizing unit can readily be increased to 100 grams per hour by the selection of proper wattage and surface area.

For any given set of conditions the rate of evaporation of propylene glycol is a function of the temperature at the surface of the glycol. As an approximate figure for calculations we have found that an increase in vaporization amounting to 5 milligrams per minute per square inch of surface accompanies each degree  $(C.^{\circ})$  rise in temperature over the range 80° to 110° C. Since propylene glycol vapor has a fairly high specific gravity, vaporizers should be placed at least six feet from the floor and a sufficient number of fans should be installed to insure thorough mixing. Otherwise the vapor will sink to the floor and lead to erroneous interpretation of experimental data.

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<sup>&</sup>lt;sup>1</sup> The opinions advanced in this paper are those of the writers and do not represent the official views of the Navy Department.

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<sup>&</sup>lt;sup>5</sup>W. Henle, H. E. Sommer and J. Stokes, Jour. Pediatrics, 21: 577, 1942.