# Technical Papers

## Transmission of *Plasmodium relictum* Grassi & Feletti by *Anopheles freeborni* Aitken[1]

Frances F. Mok

*Division of Entomology and Parasitology, University of California, Berkeley*

The successful transmission of *Plasmodium relictum* from canary to canary by *Anopheles freeborni* is described in this note. A number of culicine mosquitoes have been reported by previous workers to be capable of serving as hosts to *P. relictum*. More recently two species of anopheline mosquitoes, *Anopheles quadrimaculatus* and *Anopheles crucians* were added by Hunninen *et al.* (*1*) as susceptible hosts to *P. relictum*. The mosquito transmission of this parasite by *A. freeborni* has not thus far been reported.

The original sources of the *P. relictum* used in these experiments were 2 house finches caught in Berkeley, Cal., in September 1950. The infections were transferred from finches to canaries by the intravenous injection of citrated blood. The mosquitoes used were *A. freeborni* and *Culex pipiens molestus*; the latter was used as control. All the mosquitoes came from laboratory colonies.

Specimens of both species of mosquitoes were fed simultaneously on infected canaries having a gametocyte count of approximately 1/100 erythrocytes and kept in the insectary (74°–80° F and 80–92% humidity) during the development of the exogenous stages.

At intervals from 7 to 30 days after the initial meal, the mosquitoes were allowed to feed on parasite-free canaries. As soon as the mosquitoes had bitten, they were killed and dissected, the salivary glands examined for sporozoites, and the stomach for oocysts. The canaries were placed under observation after being bitten by mosquitoes with sporozoites in the glands.

Stomach dissections indicated that 7 (11.1%) of 62 *A. freeborni* were oocyst-positive from the 8th to the 21st day after biting. The number of oocysts per stomach ranged from 1 to 18, with an average of 4. Sporozoites were observed in the salivary glands of 5 (8.06%) *A. freeborni* on the 18th and 21st days. Three clean canaries were bitten by these sporozoite-positive mosquitoes, 2 of which were found to have a patent infection on the 9th and the 10th days, respectively. One did not become infected over a 33-day examination period.

Of the 94 *C. p. molestus* dissected, 26 (27.6%) were found to contain oocysts. The number of oocysts per stomach varied from 3 to 47, with an average of 15.3. Sporozoites were found in 14 instances in the period of 10–21 days after the infective feed. Five canaries were bitten by those that showed sporozoites in their glands. One bird became infected on the 8th day, 2 on the 9th, and 2 on the 10th day.

The findings described provide further evidence of the ability of anopheline mosquitoes to transmit avian plasmodia.

[1] This research was suggested by, and carried out under the direction of, Deane P. Furman, whose supervision is gratefully acknowledged.

### Reference

1. Hunninen, A. V., Young, M. D., and Burgess, R. W. *J. Natl. Malaria Assoc.*, **9**, 145 (1950).

## Inhibitory Effect of Cortisone on Dietary Necrotic Liver Degeneration in the Rat

Klaus Schwartz[1, 2]

*Experimental Biology and Medicine Institute, National Institutes of Health, USPHS, Bethesda, Maryland*

Degeneration of the liver leading to necrosis, hemorrhage, sudden coma, and death can be induced experimentally by a diversity of dietary means. In earlier experiments, liver degeneration was produced by 15% of a special alkali-treated casein, so-called casein VI, in synthetic diets (*1–4*). Many substances were tested for protective activity against this injury during 1940–43. This work led to the discovery of vitamin E as a liver-protecting agent (*2*). It was observed at that time that total lipoid extract from adrenals inhibited the development of the disease (*1*).

A severe but somewhat different type of dietary liver injury is caused by incorporation of large amounts of certain yeasts as protein source in synthetic diets, as shown by Hock *et al.* in 1943–4 (*5–7*) and Glynn and Himsworth in 1944 (*8*). It has been demonstrated that this condition is the result of the simultaneous inadequacy of the diet in at least two important respects. Cystine-methionine, on the one hand, and vitamin E, on the other, must be lacking at the same time in order to induce liver degeneration. Each of these substances alone can prevent the defect caused by yeast diets (*9*).

Disturbances of carbohydrate metabolism are under investigation in current experiments with young rats on a 30% yeast diet.[3] Lack of glyconeogenesis seems to be one of the essential features of liver degeneration (*10*). Cortisone (Kendall's Compound E, 17-

[1] Special research fellow, U. S. Public Health Service.

[2] The author acknowledges the technical assistance of Louis E. Lowman and Jerome H. Epstein.

[3] Composition of the diet: yeast 30%, sucrose 59%, lard (freed from vitamin E by molecular distillation, Distillation Prod., Inc.) 5%, salt mixture 5%, vitamin mixture in lactose 1%. One hundred-g diet contained the following supplemented vitamins: $B_1 \cdot HCl$ 400 $\gamma$, $B_2$ 250 $\gamma$, $B_6 \cdot HCl$ 200 $\gamma$, Ca Pantothenate 400 $\gamma$, choline chloride 1,000 $\gamma$, niacin 1,000 $\gamma$, vitamin K (2-methyl-1,4-naphthoquinone) 100 $\gamma$. 50 $\gamma$ crystalline vitamin A and 1 $\gamma$ vitamin $D_2$ were administered once weekly in 0.02 ml ethyl laurate.