

Technical Papers

Transmission of *Plasmodium relictum* Grassi & Feletti by *Anopheles freeborni* Aitken¹

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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

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

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.

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

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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 lipid 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 glycogenesis seems to be one of the essential features of liver degeneration (10). Cortisone (Kendall's Compound E, 17-

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³Composition 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: B₁·HCl 400 γ, B₂ 250 γ, B₆·HCl 200 γ, Ca Pantothenate 400 γ, choline chloride 1,000 γ, niacin 1,000 γ, vitamin K (2-methyl-1,4-naphthoquinone) 100 γ, 50 γ crystalline vitamin A and 1 γ vitamin D₂ were administered once weekly in 0.02 ml ethyl laurate.

INHIBITION OF DEATH FROM DIETARY LIVER DEGENERATION BY CORTISONE

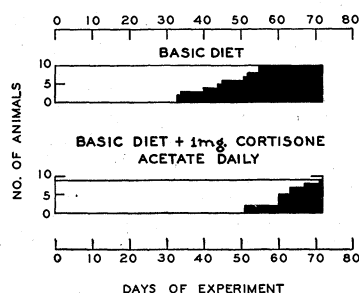


FIG. 1.

hydroxy-11-dehydrocorticosterone) is known to be a potent stimulator for glycogen formation in the liver (11). We have found that cortisone acetate has a delaying action on the development of liver degeneration in rats when given subcutaneously in daily doses of 1 mg/100 g of body weight (Fig. 1).

In 10 animals on a 30% yeast diet without cortisone, coma and death occurred after 33–60 days of the experiment, the average survival time being 45.2 days. The mortality was 100%. One out of 10 animals with cortisone died after 26 days with severe kidney damage; the liver was normal. The other 9 animals with cortisone survived the 50th day, whereas 7 out of 10 without cortisone were dead because of liver degeneration at this time (Table 1). Continued application of

TABLE 1
INHIBITION OF DIETARY LIVER DEGENERATION BY CORTISONE

	No. animals	Dead because of liver degeneration		Survival time (av)
		At 50th day	At end of experiment*	
Yeast diet	10	7	10	45.2 ± 3.50†
Yeast diet + cortisone acetate, 1 mg daily subcutaneously	9	0	9	60.0 ± 2.35†

* 71st day.

† Standard deviation of the mean.

the hormone, however, did not prevent the injury. The animals succumbed between the 51st and the 72nd day, the average survival being 60 days. Cortisone thus prolonged the average life span by about 15 days.

All rats on cortisone had an adrenal atrophy and an involution of the thymus. Two of them developed a marked symmetrical alopecia of the posterior body region. Separate analysis of the data for males and females showed no difference between the sexes in response to the hormone (Table 2); the number of ani-

TABLE 2
RESPONSE OF MALES AND FEMALES TO CORTISONE
(No. of Animals in Parentheses)

	Average survival time (in days)	
	Males	Females
Without cortisone	41.5 (6)	50.1 (4)
With cortisone	57.0 (5)	65.2 (4)
Difference due to cortisone	15.5 days	15.1 days

mals, however, is too small to draw significant conclusions. The finding that females live longer than males is customary in our experiments with liver degeneration resulting from yeast diets.

In Europe, adrenal cortical hormones have been used extensively for the treatment of parenchymal liver diseases, specially in cases of epidemic hepatitis, and have been claimed to be efficient (12). It will be of interest to learn whether the introduction of Compound E will be of additional value in this field.

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The Anterior Process and the Ossification of the Malleus in Mammals

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The view generally held regarding the ossification of the malleus in man (1, 2), as well as in other mammals (3), is that the bone ossifies independently of its anterior process. In man, the center of ossification of the malleus appears near the neck, whereas the anterior process ossifies separately in membrane and joins the main part of the bone about the sixth month of fetal life (2).

Studies on the temporal region in alizarin-stained preparations in a number of embryonic and post-embryonic stages of *Hemiechinus* (Insectivora), *Rousettus*, *Taphozous*, and *Tadarida* (Chiroptera), and *Mus* and *Gerbillus* (Rodentia) have not revealed