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THE EFFECT OF SPRING RELAPSE IN ENGLISH SPARROWS ON INFECTIVITY OF MALARIA TO MOSQUITOES[1]

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Abstract: Previous work has shown that latent infections of *Plasmodium relictum* in English sparrows become patent in the spring of the year. This spring relapse phenomenon may be ecologically important in reestablishing transmission of the parasite if the increase in parasitemia is associated with an increased infectivity to mosquitoes. The present study compared laboratory transmission rates during winter and spring of chronically infected sparrows. Results of the study indicated that birds were significantly more infective to mosquitoes during the spring relapse period than during the winter latent period.

INTRODUCTION AND METHODS

The high prevalence of patent malarial infections in birds during the spring has been attributed to a general elevation of parasite populations in established infections rather than to new transmission.[1] Beaudoin *et al.* have suggested that this spring relapse phenomenon serves as a source of infection for newly emerging vectors in the temperate climate and is, therefore, an essential factor in the survival of avian malaria.[2] An assumption of their model is that elevated spring parasite populations result in an increased number of mosquitoes becoming infected. However, Huff and Marchbank failed to demonstrate a direct correlation between gametocyte density and infectivity to mosquitoes based on oocyst counts.[3] It was the purpose of this study to see if the renewal of patency in avian malaria in springtime is associated with increased infectivity to mosquitoes.

Plasmodium relictum was the experimental parasite, the vertebrate host was the English sparrow *(Passer domesticus),* and the mosquitoes were *Culex pipiens* from a colony maintained at the Naval Medical Research Institute, Bethesda, Maryland. The sparrows were part of an experimental field population at State College, Pa. Each bird was captured and infected by syringe transfer of parasitized whole blood during the summer of 1968.[1] Some natural transmission occurred in this population, but whether each bird was naturally infected prior to capture and injection was not determined. Following infection, all birds were banded and released into the original population. Thus, birds used in this study were exposed to ambient environmental condi-

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tions until the time each test began. For the experiments, six birds were recaptured and returned to the laboratory on 17 January 1969 (winter sample) and six were returned on 22 April 1969 (spring sample). Infections in these birds were ascertained by isodiagnosis. All experimental birds had been infected a minimum of 150 days previous to recapture. Control birds consisted of sparrows with primary infections of less than 30 days' duration.

Approximately 30 female mosquitoes were allowed to feed on each bird overnight. After eight days the mosquitoes which had taken blood meals were dissected and examined for the presence and numbers of oocysts.

RESULTS AND DISCUSSION

Thin blood films were made from each bird at the time of recapture. Examination of these films (10 minutes/slide) revealed that one of six birds in the winter sample had a patent infection, and in this bird, only a single schizont was found. In the spring sample four of six birds had patent infections consisting of one or two parasites per examination. Of the total of six parasites observed, two were mature gametocytes.

Results of the transmission experiment appear in Table 1. The difference between the spring sample and the winter sample is statistically significant by the X^2 test ($P < .001$). It should be noted, however, that primary infections (control) resulted in a significantly greater amount of transmission than either the winter or spring samples ($P < .001$). All infected mosquitoes from the spring sample had fed on the four sparrows with patent infections. This suggests that the observed enhancement of transmission in spring is probably related to the parasite population in the vertebrate host.

Data collected in this study indicate that spring relapse of malaria results in an increased potential for infection of mosquito vectors, and support the hypothesis of Beaudoin *et al.* that relapse is involved in the reintroduction of parasites to the temperate zone transmission cycle.[2]

Table 1. Effect of season on infectivity of chronic **Plasmodium relictum** infections to **Culex pipiens.**

	# Mosquitoes Examined	% Mosquitoes with Oocysts	$\bar{X}$ oocysts per Infected Mosquito
Winter	123	2.4	1.0
Spring	137	14.6	6.7
Control	52	71.2	38.6

LITERATURE CITED

1. APPLEGATE, J. E. 1971. Spring relapse of *Plasmodium relictum* in an experimental field population of English sparrows *(Passer domesticus).* J. Wildlife Dis. *7:*
2. BEAUDOIN, R. L., J. E. APPLEGATE, D. E. DAVIS and R. G. McLEAN. 1971. A model for the ecology of avian malaria. J. Wildlife Dis. *7:*
3. HUFF, C. G. and D. F. MARCHBANK. 1955. Changes in infectiousness of malarial gametocytes: I. Patterns of oocyst production in seven host-parasite combinations. Exptl. Parasitol. *4:* 256-270.

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