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EFFECTS OF BLOOD-INDUCED INFECTIONS OF *Plasmodium hermani* ON DOMESTIC AND WILD TURKEY POULTS[1]

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Abstract: Experimental blood-induced infections of *Plasmodium hermani* were studied in young domestic and laboratory-reared wild turkey poults. Anemia, splenomegaly and decreased growth rates were observed, but no mortality due to the malarial infections occurred. It is suggested that malaria, acting in concert with other factors, may contribute to mortality of wild turkey poults in Florida during the first 3 to 4 weeks after hatching.

INTRODUCTION

Between 1964 and 1968 a marked decline in the populations of wild turkeys *(Meleagris gallopavo)* in Florida was detected by management biologists. In 1968 the statewide harvest of turkeys by hunters was the lowest on record. A study of the prevalence, distribution and impact of parasites and diseases in wild turkeys was initiated in 1969 in response to this situation. A number of reports on viruses,[1,2,7] helminths,[3,4,10,11] and blood protozoans[5,8,12,13] have been published. In 1972 a species of *Plasmodium* was isolated from 24 of 32 wild turkeys.[5] This malarial parasite was studied further and described as *P. hermani.*[12] The present study was undertaken to determine certain aspects of the course of infection and pathology in blood-induced infections of this malaria on domestic and wild turkey poults.

MATERIALS AND METHODS

Unsexed one-day-old Amerine broad-breasted-white poults were obtained from the Poultry Science Department, University of Florida. Wild turkey eggs were obtained from nests at Lykes Fisheating Creek Wildlife Management Area (Glades County, Florida) and incubated until hatching. The poults were housed in heated brooder cages and were provided with water and 22% protein ration *ad libitum.* After four weeks of age, they were transferred to unheated battery cages where they remained until the end of each experiment. The cages were maintained in an isolation room with an 18-hour photoperiod and temperature controlled at approximately 21 C.

A Palmdale strain (P-1) of *Plasmodium hermani* had been isolated from a wild turkey at Lykes Fisheating Creek Wildlife Management Area and had undergone four transfers (by blood inoculation) in domestic turkeys when used in Experiments 1 and 2, and 13 transfers when used in Experiment 3.

Each poult was weighed, a blood smear was prepared and the packed cell volume (PCV) was determined thrice weekly.

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Blood films were air-dried, fixed with absolute methanol and stained with Giemsa's stain. An index of anemia was obtained by determining the percentage of immature erythrocytes in a 300 cell sample for each smear. Parasitemia was determined by counting the number of asexual stages per 5,000 erythrocytes.

Three experiments were conducted to determine the effects of different dosage levels on domestic and wild poults up to 15 days of age.

Experiment 1

Fifty-four domestic poults were divided into three groups of 18 birds each. At 15 days of age, infections were initiated: one group of 18 was inoculated intramuscularly with 31×10^6 asexual stages (trophozoites, schizonts and segmenters) and another group of 18 with 3.1×10^6 asexual stages. A third group of 18 served as controls and received equivalent inoculations with blood from an uninfected turkey. Four or five birds from each group were killed on the following days post-infection: day 11 (when birds were estimated to become positive for *Plasmodium* stages in peripheral blood), day 18 (day of estimated peak parasitemia), day 25 (approximately one week after lowest PCV values) and day 53 (termination of experiment). At necropsy spleen weights were determined for each bird.

Experiment 2

Twelve 2-day-old domestic poults were divided into two groups of six birds each. Infections were initiated by intramuscular inoculations. One group received 4×10^5 asexual stages while the second group served as controls and received equivalent inoculations of blood from an uninfected turkey.

Experiment 3

Eight 12 to 18-hour-old wild poults (all from the same clutch) were divided into two groups of four. One group was inoculated intraperitoneally with 6.1×10^4 asexual stages, while the second group served as uninfected controls. An additional group of 11 three- to five-day-old wild turkey poults (all from another clutch) were divided into two groups. One group of six was inoculated with 6.1×10^4 asexual stages, while the other group of five poults served as uninoculated controls.

RESULTS AND DISCUSSION

No mortality occurred as a result of the induction of malaria. A few birds died, however, of causes unrelated to the *Plasmodium* infections.

Experiment 1

In Fig. 1 growth data are presented for each group of domestic poults for a 41-day period after infection. From about the seventh day post-infection onward, the uninfected control poults grew faster (by approximately 13%) than the infected birds. Growth of poults receiving 31×10^6 asexual stages was similar to that of poults receiving 3.1×10^6 asexual stages.

The development of splenomegaly is shown in Fig. 2. Both infected groups had enlarged spleens with very little difference between the two groups.

At about the seventh day post-infection PCV's began to decrease, and reached a low on day 15 for birds receiving 31×10^6 asexual stages (Fig. 3A) and on day 20 for birds receiving 3.1×10^6 asexual stages. Mean lowest PCV values for the two infected groups differed by only 5%.

The levels of parasitemia and the numbers of immature erythrocytes are presented for three of the six turkeys inoculated with 31×10^6 asexual stages (Fig. 3B and 3C). Results for turkeys receiving 3.1×10^6 asexual stages were similar. Peaks of parasitemia occurred on day 8 post-infection for the poults infected with 31×10^6 asexual stages, and on day 13 for poults which received 3.1×10^6 asexual stages. Concurrent with the peaking of parasitemia, PCV's dropped dramatically, and shortly there-

after the number of circulating immature erythrocytes increased and remained above normal during the remaining period of observation.

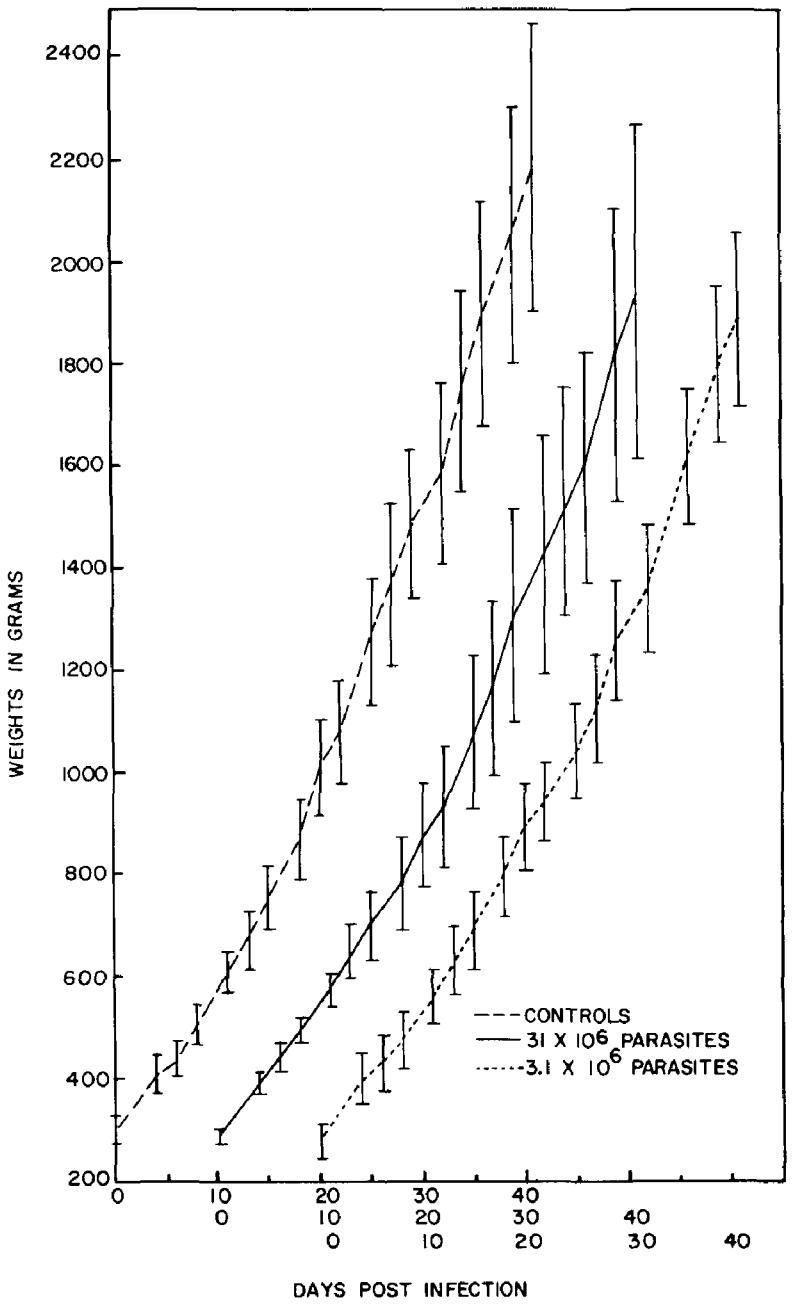

FIGURE 1. Growth of domestic turkey poults infected with 3.1×10^6 and 31×10^6 asexual stages of *Plasmodium hermani* at 15 days of age, Experiment 1. (Vertical bars designate standard deviations.)

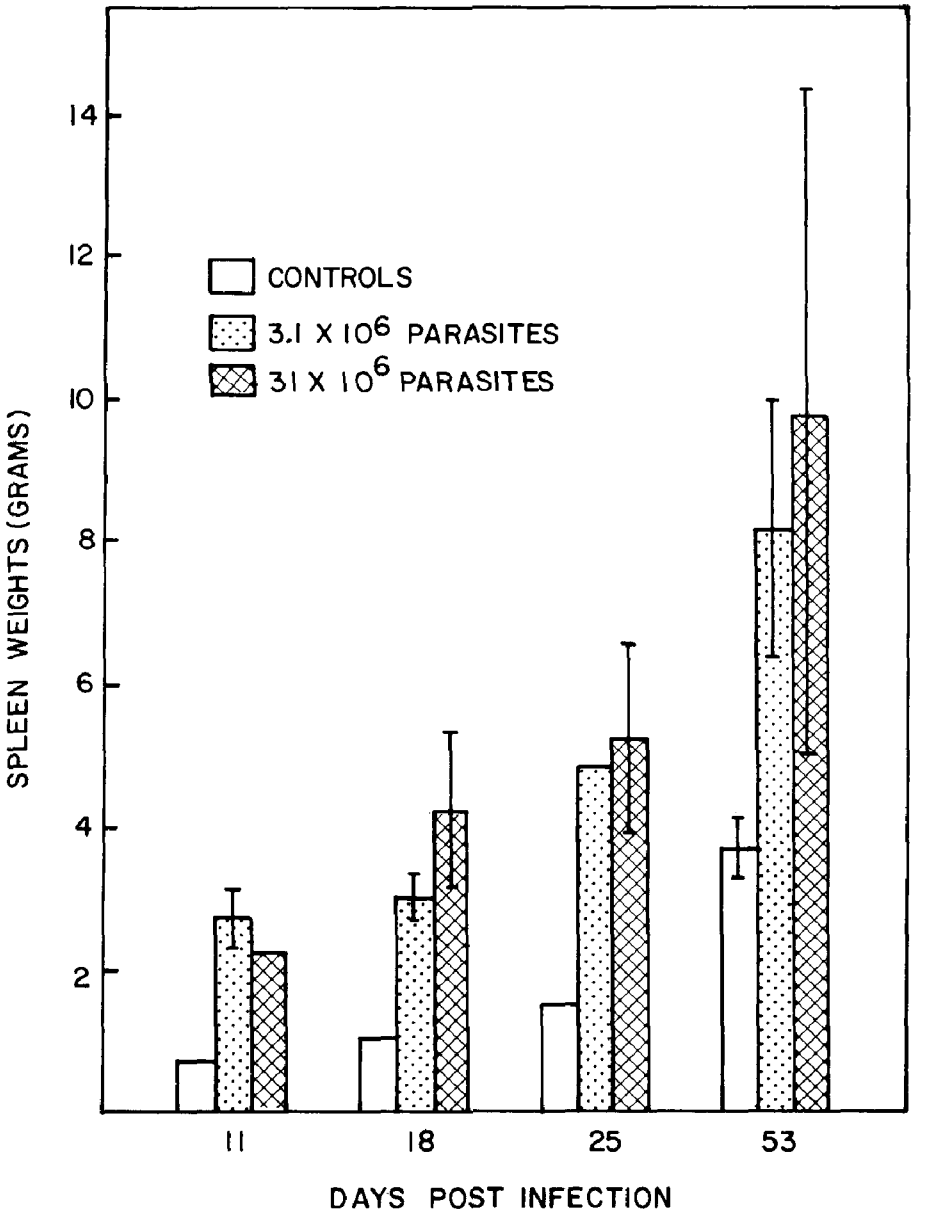

FIGURE 2. Weights of spleens from domestic turkey poults infected with 3.1×10^6 and 31×10^6 asexual stages of *Plasmodium hermani* at 15 days of age, Experiment 1. (Vertical bars designate standard deviations.)

Experiment 2

Poults infected at two days of age showed similar effects as the 15 day old poults of Experiment 1 including a lag in growth (22%) compared to uninfected controls of the same age. The mean PCV for the infected poults dropped to 17% by day 21 and returned to the normal range

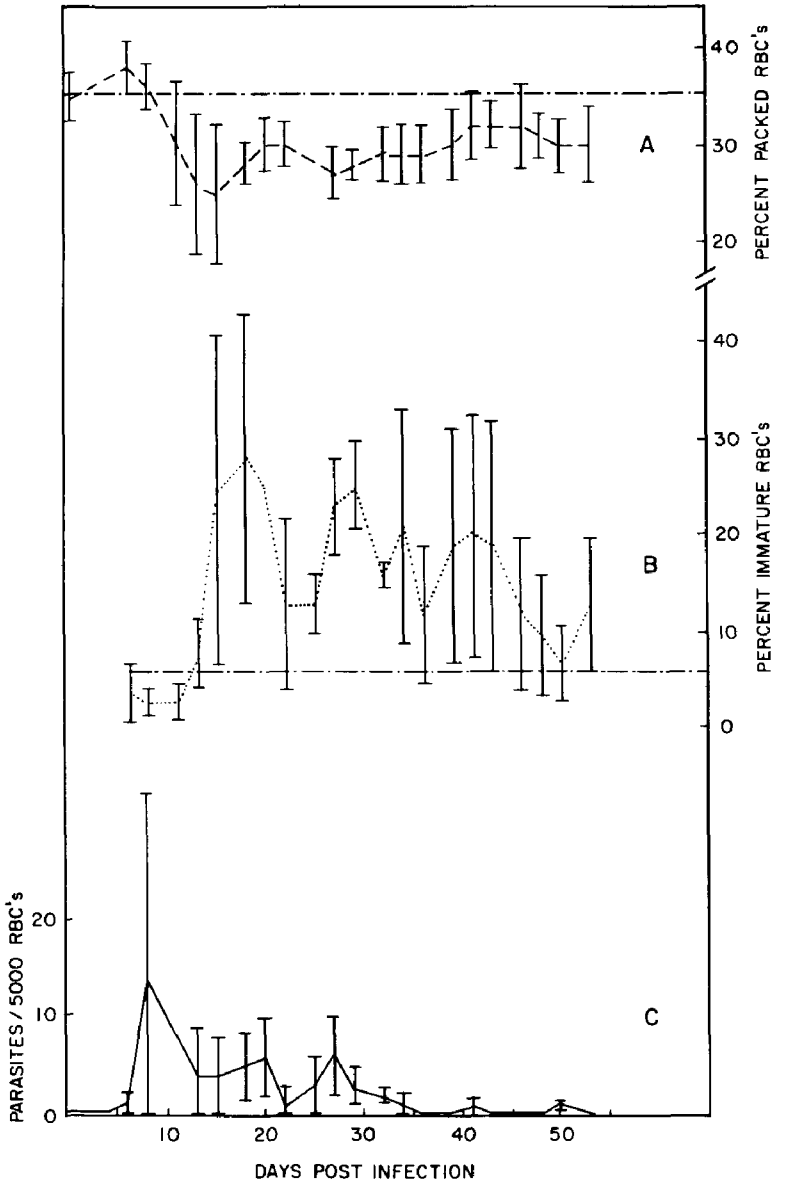

FIGURE 3. Hematocrits (A), percent immature RBC's (B), and parasitemias (C) of domestic turkey poults infected with 31×10^6 asexual stages of *Plasmodium hermani* at 15 days of age, Experiment 1. (Vertical bars designate standard deviations; _._ designates control grand mean.)

by day 30. The peak parasitemia occurred on day 14 and was followed by a drop in PCV and a subsequent increase in the number of circulating immature erythrocytes.

Experiment 3

Results of studies with wild poults were similar to those for the domestic poults. Growth was slower in infected wild poults (15% for those infected at 3 to 5

days of age and 26% for those infected at 12 to 18 h of age) compared to their uninfected controls. The mean PCV dropped to 20% on day 25 post-infection and the peak of parasitemia occurred on day 18 for poults infected at 12 to 18 h of age (Fig. 4). The results were similar in poults infected at 3 to 5 days of age.

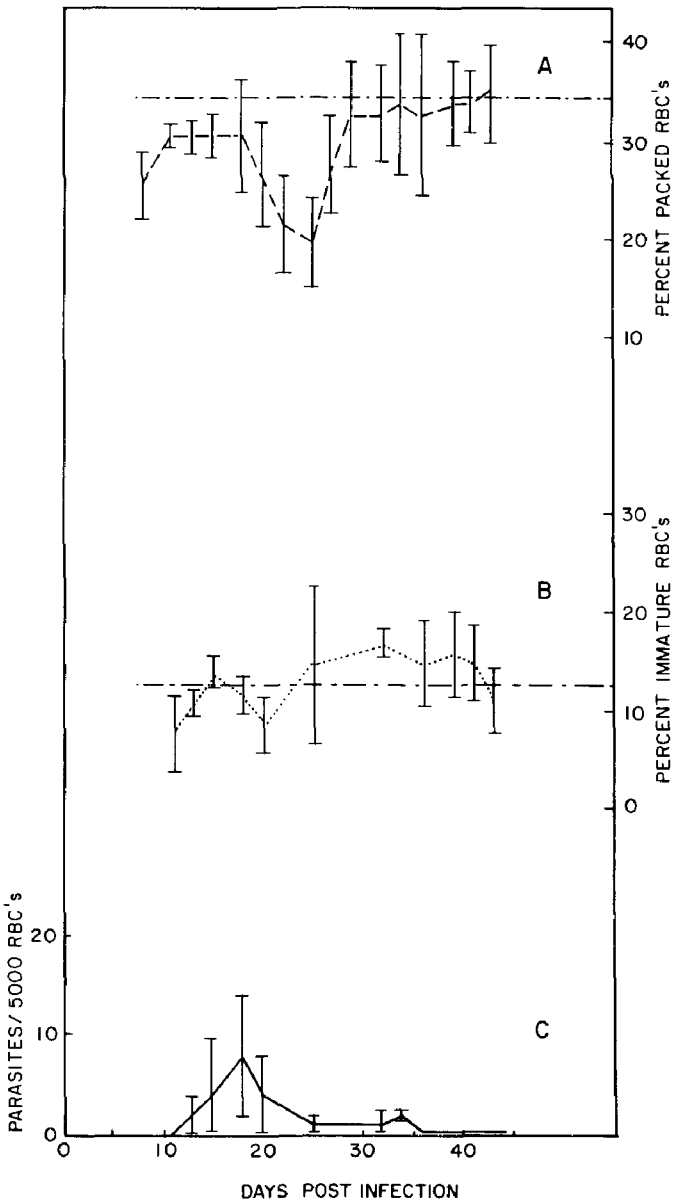

FIGURE 4. Hematocrits (A), percent immature RBC's (B), and parasitemias (C) of wild turkey poults infected with 6.1×10^4 asexual stages of *Plasmodium hermani* 12 to 18 hours after hatching, Experiment 3. (Vertical bars designate standard deviations; _._ designates control grand mean.)

The experiments described in this paper showed that domestic and wild turkey poults exposed to *P. hermani* at 15 days of age or younger were affected similarly, regardless of the size of the infecting dose. One exception was that low infecting doses produced delayed peaks of parasitemia. Although no mortality occurred, it was demonstrated that infected poults experienced anemia, splenomegaly, and depressed growth rates. The latter were more pronounced in poults infected at 12 to 18 h of age than in those infected at 15 days of age. Anemia and splenomegaly are common effects of various malarias on birds[6] and decreased rates of weight gain have been observed also in chicks infected with *P. lophurae*.[9]

Several behavioral effects were noted in infected birds in all three experiments. The infected poults were unthrifty and showed apathy, lethargy, postural changes (*i.e.*, drooped wings and retracted necks), ruffled feathers and they emitted shrill, recurrent distress calls. These effects were seen mainly during the second and third weeks post-infection.

These observations may be significant in understanding the impact of parasitism and disease on wild turkey populations in Florida. From 1968 to 1976, wild turkey poults at Fisheating Creek experienced about 50% mortality during the first 3 to 4 weeks after hatching (Williams and Austin, 1979, unpubl.). Close to 90% of the wild turkeys in that population, from which the Palmdale strain of *P. hermani* was obtained, were infected.[5] Although the poults infected experimentally in the present study did not die from their infections, these birds had the benefit of only minimal stress, *i.e.*, unlimited amounts of water and high quality feed, protection from inclement weather, predators, insect pests and other diseases, etc. We feel that the observed effects of the malaria would contribute to the death of poults in nature where more serious stresses are present. Poults showing such weakness might well be left behind by the brood as it foraged and, in any case, would probably not be able to escape an attack by a predator. This population of wild turkeys at Fisheating Creek is known to have a variety of other protozoan,[5] helminth,[10] viral,[1,7] and bacterial diseases (White and Forrester, 1979, unpubl.), which in concert with malaria, could also act as important factors of poult mortality.

Acknowledgements

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