http://www.jstor.org

in parasitology, Vol. 16, W. H. R. Lumsden (ed.), Academic Press, London, p. 274). Normal, control, and anti-μ chain-suppressed (CBA × BALB/c) F_1 mice were infected with 200 infective larvae, orally. Twenty-three days later these animals were sacrificed and the gastrointestines removed, rinsed briefly in phosphate-buffered saline, and then suspended in physiological saline at 37 C for 3 hr, allowing for viable adult worms to migrate out of the small intestine. The gastrointestines of mice from both groups of 6 animals harboured fewer than 2 adults per animal (data not shown), which is certainly not consistent with the precept that B-cell suppression results in a marked delay in adult worm expulsion.

Thus, there is no evidence that anti-μ chain-treated B-cell-suppressed mice suffer an exacerbated primary infection despite their inability to mount an antibody response. Humoral responses seem to play little role in the control of a primary infection with *Trichinella spiralis* in the mouse. Perhaps this is not surprising, since survival of this parasite in an immunologically naive host may rely on remaining one step (i.e., parasite stage) ahead of the immune response (Almond and Parkhouse, 1986, loc. cit.). It should be reemphasised here that these experiments investigated the role of antibodies in controlling a primary infection alone. They do not indicate that antibodies per se cannot mediate protection against *T. spiralis,* as the passive transfer of a monoclonal antibody with specificity for the surface of newborn larvae has been found to be protective (Ortega-Pierres et al., 1984, Parasite Immunology **6:** 275–284). Further investigations are required to elucidate the role that antibodies may play during reinfection.

Effects of *Plasmodium relictum* on the Metabolic Rate and Body Temperature in Canaries (*Serinus canarius*)

Anita M. Hayworth, Division of Environmental Studies, University of California, Davis, California 95616; **Charles van Riper, III,** Department of Wildlife and Fisheries Biology and CPSU, University of California, Davis, California 95616; **and Wesley W. Weathers,** Avian Science Department, University of California, Davis, California 95616

Birds infected with malaria exhibit a variety of clinical signs, ranging from asymptomatic, to a fluffed or ruffled appearance, weight loss, and lethargy, depending on the severity and stage of the infection. The major pathology of avian *Plasmodium* involves the circulatory system with any one or all of the following contributing to death: severe anemia, dehydration, hemolysis, breakdown of the spleen and/or liver cells, and anoxemia (Seed and Manwell, 1977, *In* Parasitic protozoa, Vol. 3, J. P. Krier (ed.), Academic Press, New York, pp. 348–353). Anoxemia results from both red blood cell destruction and a change in the plasma pH, which reduces the hemoglobin's oxygen-binding capacity (Rigdon and Rostorfer, 1946, Journal of the National Malaria Society **5:** 253–262). Impaired O_2 transport may adversely affect physiological performance, and thereby indirectly jeopardize survival even in nonlethal malarial infections. For example, ducks infected with *P. lophurae* may show only 15–20% of the normal oxygen-carrying ability of the blood (Rigdon, 1946, Experimental Medical Surgery **4:** 156–164). Sustainable activity (running) was reduced 20% in *Plasmodium*-infected lizards that exhibited a 29% decrease in O_2-carrying capacity (Schall et al., 1982, Science **217:** 1057–1059). A reduction in metabolic rate leading to diminished ability to maintain body temperature, especially under thermally stressing conditions, could adversely affect survival of an infected host.

In this experiment, we sought relationships between stages of malaria infection and changes in the bird's metabolic rate. Oxygen consumption ($\dot{V}O_2$) and body temperature (T_b) were measured in canaries (*Serinus canarius*) before and after subinoculation with schizonts of *Plasmodium relictum.* Six 2-yr-old nonbreeding canaries (3 male, 3 female) were used to measure $\dot{V}O_2$ and T_b at 2 air temperatures (T_a's): 33 C, which is within the canary's thermoneutral zone, and 5 C, which is well below the thermoneutral zone. Measure-
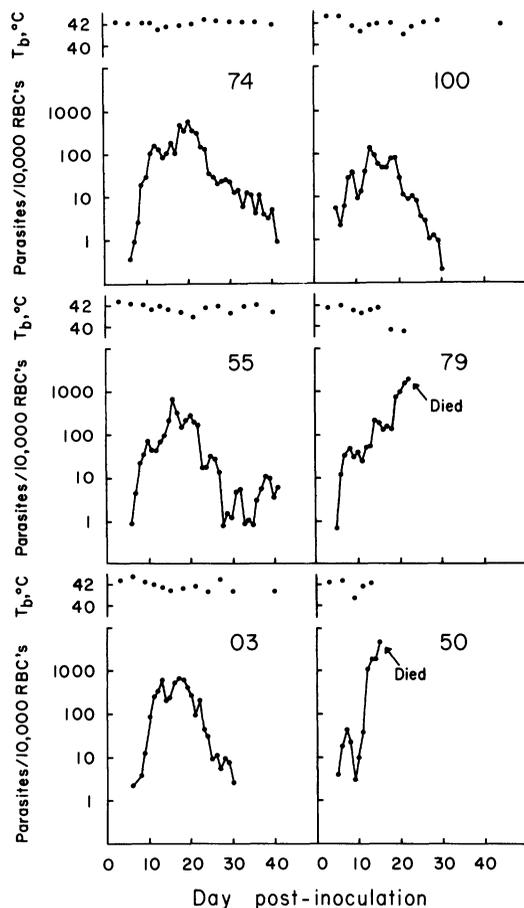
FIGURE 1. Parasitemia levels (parasites/10,000 RBC's) and body temperature at air temperature of 5 C in 6 canaries infected with *Plasmodium relictum.*
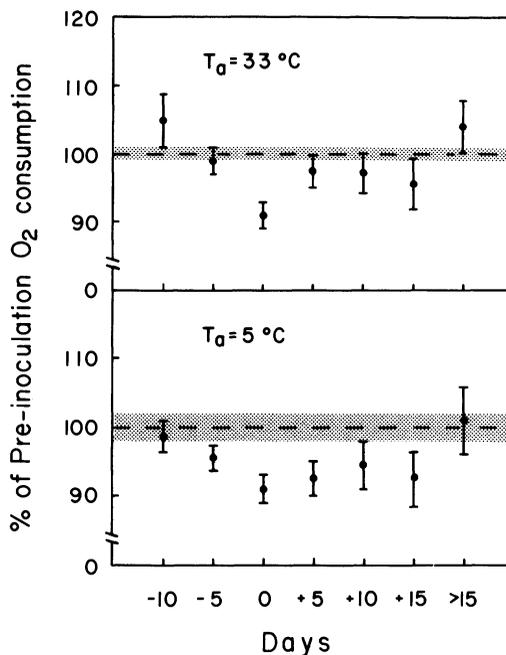

FIGURE 2. Oxygen consumption of canaries infected with *Plasmodium relictum* expressed as a percentage of the preinoculation (=control) value. Day 0 indicates day of peak parasitemia. Shaded area is the 95% confidence interval about the mean preinoculation O_2 consumption. Each point represents the mean; short vertical lines indicate SE.

ments at both T_a's were made on the same day on fasted birds during the nocturnal phase of their daily cycle. $\dot{V}O_2$ was measured in an open-circuit respirometer using an Applied Electrochemistry S-3A analyzer and N-22M sensor. Methods and details of equipment are presented elsewhere (Weathers et al., 1980, Comparative Biochemistry and Physiology **65A**: 235–238). T_b was measured using a YSI model 43 telethermometer with a fast response time probe, model 501, within 1 min of removal of the bird from the chamber. To establish mean control values, we measured each bird's $\dot{V}O_2$ and T_b 6–8 times on different days over a 2-wk period prior to challenge with *Plasmodium* parasites. Following subinoculation, measurements were made for all birds on days 3, 6, 9, 11, 13, 15, 18, 21, 24, 27, 30, 33, 36, 40, or until the bird died or had a very low parasitemia level.

The strain of *Plasmodium relictum* that we used was established from a red-winged blackbird (*Agelaius phoeniceus*) caught in Solano Co., California, and maintained in canaries by standard transmission methods (Garnham, 1966, Malaria parasites and other haemosporidia, Blackwell Scientific Publications, Oxford, 1114 p.). The 6 canaries used in this study were each subinoculated i.m. with 0.1 ml of blood containing an average of 16 schizonts. After day 5 postinoculation, parasitemia was determined for each bird by obtaining a thin blood smear from a clipped toe every 24 hr. Blood smears were fixed in acetone-free methanol and stained with Giemsa. Each slide was read under oil immersion for 10 min and parasites were classed as either gametocytes, schizonts, or trophozoites. The formula used to determine the number of parasites/ 10,000 red blood cells (RBC's) is that of van Riper et al. (1987, Ecological Monographs [in press]).

Each of the 6 birds that we studied showed differences during the course of infection in both timing and height of peak parasitemia. For 4

TABLE I. *Relationship of parasitemia at the peak of the infection to the magnitude of change in the metabolic rate of individual birds at 33 C and 5 C.*

Bird number	Rank of para-sitemia	Parasites per 10,000 RBC's at crisis peak	% Decrease in $\dot{V}O_2$	
			33 C	5 C
50	1	5,000	−3.9	−17.6
79	2	2,020	−12.5	−9.7
03	3	749	−7.3	−6.8
55	4	714	−12.1	−14.2
74	5	552	−1.8	−3.0
100	6	132	+1.2	−3.2
Mean			−6.1	−9.1
SE			2.27	2.42
"r"			0.60	−0.77*

* Indicates significant correlation ($P < 0.05$) as determined by Spearman's coefficient of rank correlation.

birds, peak parasitemia was reached on days 12–16. Two birds (nos. 50 and 79) showed a continuous rise in parasite numbers until death at days 15 and 22, respectively. Peak parasitemias ranged from 132 to 5,000 parasites/10,000 RBC's (Fig. 1).

Preinoculation $\dot{V}O_2$ for all 6 birds averaged 59.6 ± 2.4 ml/hr (mean ± SE) at 33 C and 116.0 ± 8.8 ml/hr at 5 C. During the crisis period, the corresponding values were 56.0 ± 3.0 ml/hr at 33 C and 105.3 ± 8.6 ml/hr at 5 C. Significant decreases in $\dot{V}O_2$ occurred during the crisis for 3 birds at 33 C (nos. 55, 03, and 79) and 4 birds at 5 C (nos. 55, 03, 79, and 50). Generally, $\dot{V}O_2$ decreased during the peak parasitemia, especially as measured at 5 C, but then returned to preinoculation levels after the crisis period (Fig. 2). At 5 C there was a significant correlation ($r = 0.77$; $P < 0.05$) between the level of parasitemia at peak infection and relative magnitude of the decline in $\dot{V}O_2$ (Table I). The correlation was not significant at 33 C ($r = 0.60$; $0.05 < P < 0.10$).

Individuals varied in the degree to which T_b decreased during the crisis period (Fig. 1). All birds, except one at 5 C, showed some decline but, due to a large degree of T_b variation within individuals, not all decreases were statistically significant. T_b in birds has been found to have a high degree of lability (Huff, 1939, American Journal of Hygiene **29**: 149–154), which makes the detection of a change difficult. At 33 C, T_b significantly decreased in birds nos. 74, 55, 100, and 79 during the crisis period. For these individuals, the decrease ranged from 0.3 C to 1.6 C, with a mean drop of 0.6 C. Only bird no. 79 showed a significant change in T_b at 5 C: a 2.4
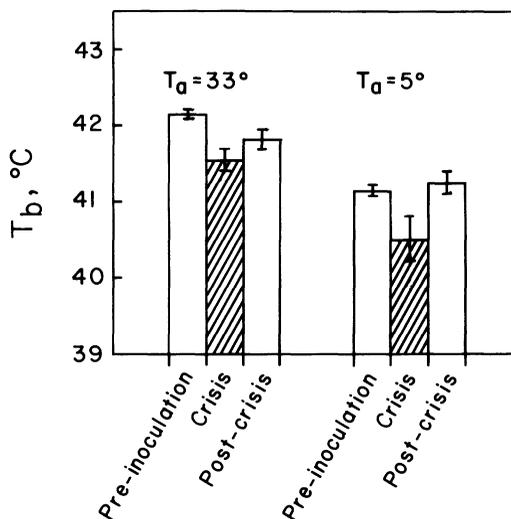

FIGURE 3. Mean body temperature of 6 canaries during preinoculation, crisis, and post crisis periods of infection with *Plasmodium relictum*. Crisis is that period where parasitemia level is greater than 100 parasites 10,000 RBC's. Bars indicate mean; short vertical lines indicate SE.

C decrease. The preinoculation T_b (mean ± SE) was 42.2 ± 0.04 C at 33 C, and 41.2 ± 0.05 C at 5 C. During the crisis period, T_b was 41.5 ± 0.13 C and 40.5 ± 0.30 C at 33 C and 5 C, respectively. There was a significant decrease of 0.7 C in mean T_b at both 33 C and 5 C. After the crisis period, T_b approached the preinoculation levels with values of 41.8 ± 0.08 C and 41.2 ± 0.14 C at 33 C and 5 C, respectively. The mean change in T_b at the 2 T_a's is shown in Fig. 3.

Our results reveal that bird malarial parasites do not act as pyrogenic agents. In fact, due to their already high T_b, birds reverse the process characteristic of human malaria and lower their T_b, thus further compounding the problem of O_2 assimilation during the crisis period. The lower T_b and inability to increase T_b at T_a's below thermoneutral corresponds to the decrease in O_2 consumption.

In general, hematozoan infections are common in wild avian populations. In North America, a survey of 57,026 birds of 388 species revealed 36.9% to be infected with some form of hematozoa; 3.8% of the birds had at least one form of *Plasmodium* (Greiner et al., 1975, Canadian Journal of Zoology **53**: 1762–1787). Bennett et al. (1976, *In* Wildlife diseases, L. A. Page (ed.), Plenum Press, New York, pp. 25–33) felt

that, except for special cases such as that found in Hawaii (i.e., Warner, 1968, Condor **70:** 101–120; van Riper et al., 1987, loc. cit.), hematozoan infections are not major regulators of avian populations. However, our results point to the fact that hematozoan infections may be more important than originally suspected, particularly if peak parasitemia occurs at a thermally stressful time.

If a bird is able to survive the initial crisis, this does not ensure that the individual will not be impacted by the malarial parasite at a later date. Relapses have been brought about by treatment with UV radiation or injection with adrenalin, and have been associated with different seasons of the year and breeding condition of the host (Ben-Harel, 1923, American Journal of Hygiene **3:** 652–685; Applegate, 1971, Journal of Wildlife Diseases **7:** 37–42). The one common denominator in all of these situations is that relapse occurs following some form of stress to the infected host. This fact is especially important for wild birds that must deal with environmental vagrancies. If a previously infected bird is placed in a stressful situation which is severe enough to cause a relapse, the chances of its survival are greatly reduced by the added impact of its decreased ability to thermoregulate and adequately supply oxygen to the tissues. Especially severe thermal environments, such as an unseasonable cold spell, could thus cause an exceptionally high mortality in birds that had survived an initial malaria infection. Impaired thermoregulation at peak parasitemia has important implications for survival of newly infected as well as relapsed wild avian hosts, especially when exposed to thermally stressing conditions.

A Model of the Cerebral Ganglion in
Macracanthorhynchus hirudinaceus (Acanthocephala)

T. T. Dunagan and Donald M. Miller, Department of Physiology and Pharmacology, Southern Illinois University, Carbondale, Illinois 62901

The brain (CG, cerebral ganglion) of Acanthocephala was discovered by von Siebold according to Kaiser (1893, Bibliotheca Zoologica 7: 1) in *Macracanthorhynchus hirudinaceus.* However, it was Schneider (1868, Archiv für Anatomie, Physiologie and Wissenschaftliche Medicin **5:** 583–596) and Brandes (1899, Abhandlungen Deutsches Naturforschendo Gesellschaft (Halle) **21:** 271–299) who described the ganglion. The model presented by Brandes was subsequently adopted by investigators and generally copied by reviewers of these worms. The simplicity of the illustration as well as its apparent completeness appealed to zoologists.We, too, were impressed with the small number of cells shown in this model particularly considering the fact that this is thought to be the only ganglion in female worms. Thus, in 1968 we began a series of studies on the nervous system of *M. hirudinaceus* in an attempt to verify Brandes' observations. As a result, we published our version of a 3-dimensional model of the cerebral ganglion of *M. hirudinaceus* (Dunagan and Miller, 1970, Comparative Biochemistry and Physiology 37: 235–242), which was much different from the one by Brandes. We have since published a number of papers including an atlas of cells in the cerebral ganglion of this species. We confirmed the small number of ganglion cells present (86) although this number was considerably larger than Brandes originally described. We have also published a series of papers concerning the nerves originating from the cerebral ganglion. The results of these more recent studies on the nerves require a modification in our 1970 model. Figure 1 is our present interpretation of the nerves originating from this ganglion.

The discovery (Miller and Dunagan, 1983,