

BRIEF NOTE

ACUTE MALARIA: EFFECTS OF *PLASMODIUM BERGHEI* ON THE METABOLIC RATE OF MICE¹

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Previous studies have shown that malaria parasites have detrimental effects on specific host organs involving tissue anoxia, phagocytosis of infected and uninfected erythrocytes and degeneration of liver, spleen, lymphoid and adrenal tissue (Maegraith 1966). Malaria parasites also affect host carbohydrate, lipid and protein metabolism (Siddiqui and Trager 1967; Angus *et al* 1971). Such changes may be accompanied by a modification of the metabolic rate of the host. The synchronous nature of paroxysms caused by most species of malaria preclude accurate measurements of the host metabolic rate. The present study examines changes in metabolic rate of mice infected with the asynchronous rodent malaria parasite, *Plasmodium berghei*.

Plasmodium berghei (Walter Reed Strain) was maintained in an outbred colony of swiss albino mice by intraperitoneal injection of blood from infected mice. Mice infected with *P. berghei* were provided by Dr. J. P. Kreier of The Ohio State University. Twenty-four 8-week-old female CF-1 mice (Carworth Farms) were randomly divided into 3 groups of 8 mice. One group consisted of uninjected controls, while a second group was injected with 0.2 ml of blood from uninfected mice and the third group received 0.2 ml of blood containing *P. berghei*. Between days 1 and 12 post-infection (PI), 4 infected mice were selected and their metabolic rates determined daily. Similar determinations were made for each control group (i.e., uninjected and injected mice) on days 1 and 12 PI. Previous studies

(Mayer and Pappas 1976) indicate little variation in metabolic rate of 8-week-old uninfected CF-1 mice over a period of 21 days. The metabolic rate of mice was determined according to the method of Lustick and Lustick (1972). Mice were fasted for 2 hr, weighed and placed in plastic chambers measuring 125 x 60 x 80 mm. The chambers were then placed in an Aminco temperature controlled water bath and the chamber temperature monitored continuously by means of a YSI Model 46 Telethermometer. The chamber was maintained at $30 \pm 0.5^\circ \text{C}$ and oxygen consumption monitored with a Beckman F-3 paramagnetic oxygen analyzer with an air flow of 1000 cc/min. An open circuit was used and CO_2 was not absorbed. The concentration of oxygen and metabolic rate was calculated according to Depocas and Hart (1957) using the value obtained when mice had reached a minimum steady state oxygen consumption. The rectal temperature of each mouse was taken at the end of each determination using a YSI Model 46 telethermometer fitted with a small animal probe. The percent parasitemia in infected mice was determined from Giemsa-stained tail vein blood. Experimental and control data were compared for significance of differences using Student's *t* test.

The metabolic rate of uninjected control mice was $1.88 \pm 0.1 \text{ ccO}_2/\text{g/hr}$ at day 1 PI, while mice injected with 0.2 ml of uninfected mouse blood showed a slight, but statistically insignificant increase to $1.94 \pm 0.4 \text{ ccO}_2/\text{g/hr}$. There was a statistically significant ($P < 0.05$) increase in metabolic rate of infected mice during the initial 24 hr after infec-

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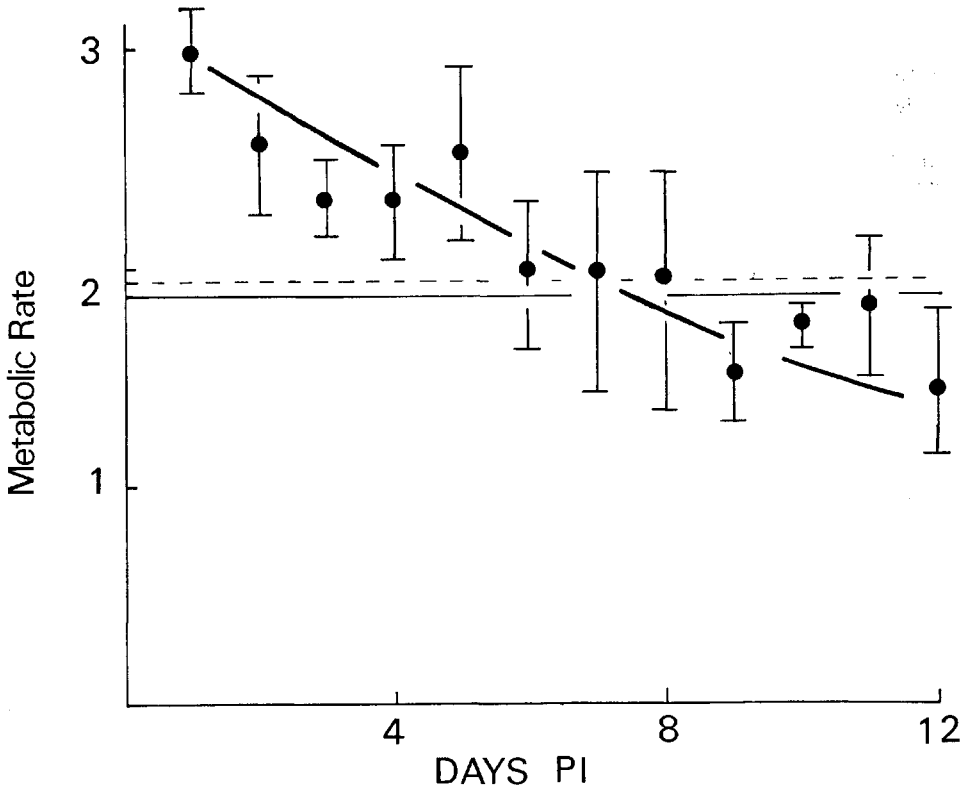


FIGURE 1. The metabolic rate (in $\text{ccO}_2/\text{g/hr}$) of female mice infected with *Plasmodium berghei* (solid circles), the mean of control mice on days 1 and 12 (solid line), and the mean of control mice injected with 0.2 ml of normal mouse blood (dashed line), as a function of days post-injection (PI). Vertical bars represent standard errors of the mean.

tion (fig. 1). The metabolic rate of infected mice remained significantly greater than that of the controls for 5 days PI and

TABLE 1
Percent parasitemia and internal body temperature
in mice infected with *Plasmodium berghei*
on days 1-12 post-infection.

Day	% Parasitemia	°C Temperature
1	2.5	37.3
2	2.7	37.6
3	3.3	38.0
4	14.5	37.2
5	19.5	37.7
6	21.0	37.9
7	24.0	37.5
8	31.0	38.0
9	42.0	35.0
10	55.0	33.0
11	58.0	32.0
12	63.0	32.1

declined to the level of control mice at day 6 PI. A decrease in the metabolic rate of infected mice occurred just before death and was accompanied by a corresponding decrease in internal body temperature (table 1). The parasitemia remained low during the first few days of infection (days 1-3 PI) but increased to a maximum of 63% just prior to death on day 12 PI; (table 1).

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