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Effect of Malaria and Fever on Energy Metabolism in Gambian Children

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ABSTRACT. The aim of the present study was to measure the changes in resting energy expenditure (REE) induced by malaria and to assess to what extent they are related to fever and nutritional status. The REE of 19 Gambian children (mean age ± SEM, 9 ± 1 y; weight, 24 ± 2 kg; expected weight for height 86 ± 1%) were measured with a hood system at repeated intervals at the onset of malaria crisis (test A), 3 to 4 d after therapy (test B), and 14 to 21 d later (test C). Axillary temperature averaged 39.2 ± 0.1, 36.6 ± 0.1, and 36.7 ± 0.1°C in the tests A, B, and C, respectively. REE in test A was significantly higher than REE in test B (223 ± 10 *versus* 174 ± 8 kJ/kg·d, $p < 0.0001$), but in test C (169 ± 8 kJ/kg·d), it did not differ from that observed in test B. The percentage of increase in REE was significantly correlated with the difference in axillary temperature ($r = 0.46$, $p < 0.05$); the slope of the regression line indicated an increase of 6.9% in REE/°C of fever. Furthermore, the individual increase in REE/°C was correlated to the percentage of weight for height of the children ($r = 0.54$, $p < 0.05$), indicating that the child's nutritional status influences the magnitude of the hypermetabolism due to fever. We concluded that Gambian children suffering from an acute episode of malaria have an increase in REE averaging 30%; however, REE promptly returns to baseline value a few days after the beginning of therapy. (*Pediatr Res* 31: 102–106, 1992)

Abbreviations

BMI, body mass index, adiposity index (weight/height^2, *i.e.* kg/m^2)
REE, resting energy expenditure

It has been well established that recurrent infections (diarrheal diseases, respiratory tract infections, malaria) are, together with chronic insufficient or marginal nutrient intake, one of the main causes of the failure to thrive in children living in developing countries (1, 2). Although the impact of infection on growth has been the object of a number of studies (3), there is little quantitative information about the energy cost of specific infections, and the extent to which episodes of infection lead to a specific change in energy requirements is largely unknown in children.

During frequent episodes of acute illness, the increase in energy expenditure combined with a low energy intake due to anorexia is an important factor explaining the poor growth commonly observed in many children from developing countries. Surprisingly, Eccles *et al.* (4) showed a decrease in sleeping energy expenditure averaging 16% during episodes of malaria in children after correcting for the effect of body temperature, as compared with the period before the illness, whereas Duggan and coworkers (5–7) showed a negative energy balance but no increase in energy expenditure during measles in children. These results do not support the results of Du Bois (8), who showed an increase in energy expenditure that accompanies the fever episode.

These contradictory results have prompted us to assess the energy cost of an acute episode of malaria in Gambian children using indirect calorimetry. Another aim of the study was to investigate whether the changes in REE are related to the changes in body temperature, to nutritional state, or to parasitemia.

SUBJECTS AND METHODS

Environmental conditions. The study took place from October 1989 to September 1990 in Keneba, a rural village of the West Kiang district of The Gambia, West Africa. The life conditions of this village have been extensively described previously (9–11). During the rainy season (June to October), malaria is endemic with *Plasmodium falciparum* predominating. Because of the partial immunity of the children, most infections do not induce the well-known recurrent episodes of fever, but usually the symptomatology consists in a persistent fever with headaches, nausea, and vomiting. At that time of the year, the food available to the family is restricted because the previous year's food stores are almost exhausted.

Subjects (Table 1). Twelve girls and eight boys were recruited at the Keneba outpatient clinic to take part in the present study. Their acceptance to participate was obtained from their relatives after a detailed explanation of the procedure was given to them. The study did not interfere with the usual treatment of the children, which was immediately initiated after the diagnosis had been made. The study protocol was submitted to and approved by the Medical Research Council/Gambian Government Ethical Committee. One of the girls did not complete the study because she did not follow the treatment completely and, consequently, still had fever at the time of the second measurement.

Anthropometry. Body weight was measured immediately before the calorimetric measurements in fasting conditions with an accuracy of 0.1 kg (Kipfer scale, model DPW 150, Jegendorf, Switzerland), with the child dressed with a light African cloth.

The initial physical characteristics of each child are given in Table 1. Body fat was assessed by measuring skinfold thickness at four different sites (biceps, triceps, subscapular, and suprailiac) (12) with a caliper (Holtain Ltd., Crymych, UK) allowing measurements with a precision of ±0.2 mm. The mean body fat, calculated using the equation of Brook (13), averaged 9.9 ± 0.6% of total body weight, and the mean fat-free mass was 21.7 ± 1.8 kg.

Experimental design. The children admitted to the Keneba

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Table 1. *Physical characteristics*

Subject	Sex	Age (y)	Weight A (kg)	Weight B (kg)	Weight C (kg)	Height (cm)	BMI (kg/m^2)	Weight for height* (%)	Parasites (no./μL)
1	F	10.0	27.0	27.7	28.3	138.0	14.2	85.6	35 400
2	F	12.4	26.2	25.5	25.7	144.0	12.6	70.9	30 100
3	M	15.0	43.8	44.2	46.1	163.5	16.4	86.3	818 000
4	F	13.7	38.7	37.1	38.7	153.0	16.5	93.2	85 200
5	F	3.7	12.1	12.2	12.4	94.5	13.5	85.7	69 400
6	F	12.4	32.6	31.1	32.6	147.0	15.1	83.1	61 400
7	F	5.8	15.7	15.9	15.8	109.0	13.2	87.5	60 900
8	F	6.8	22.3	22.9	22.5	124.5	14.4	92.6	7 880
9	M	7.0	17.9	18.3	19.2	116.0	13.3	86.3	9 200
10	F	7.7	17.4	16.7	16.8	117.5	12.6	84.3	18 100
11	M	9.4	27.3	27.0	27.4	138.5	14.2	84.7	51 400
12	M	13.2	30.6	30.8	31.0	142.5	15.1	86.8	38 000
13	M	7.6	18.6	19.1	19.4	121.5	12.6	82.2	45 300
14	F	7.6	19.1	19.5	19.2	120.5	13.2	85.8	†
15	F	9.2	21.9	21.6	22.0	131.0	12.8	79.9	7 600
16	F	4.8	15.1	15.5	14.8	101.5	14.7	96.5	228 000
17	M	13.2	24.6	25.1	25.1	134.0	13.7	84.9	16 800
18	M	15.0	33.5	33.2	32.8	147.5	15.4	89.4	25 800
19	M	3.7	14.2	14.3	14.3	99.0	14.5	93.9	42 800
Mean		9.4	24.1	24.1	24.4	128.5	14.1	86.3	91 800
SEM		0.9	2.0	1.9	2.1	4.5	0.3	1.3	43 100

* Weight relative to the mean weight for height according to the National Committee for Health Statistics standards.

† Missing value because of staining problems; estimated to be 45 000 by a parasite per high power field count.

outpatient clinic with a malaria crisis were candidates to be included in the study. They came for treatment within a day or two after the onset of malaria. Malaria was defined as a disease characterized by an axillary temperature higher than 37.5°C and a parasitemia higher than 5000 parasites/μL (14). For 14 children, this episode was their first malaria crisis during the year of the study, whereas for the five others, it was their 2nd to 5th episode. The treatment was started with an initial dose of 100 to 600 mg of chloroquine (depending on body weight), then three doses of 75 to 300 mg over 2 d. The children also received antipyretics [either paracetamol (initial dose 125 to 500 mg) or aspirin (initial dose 300 to 600 mg)]. Within 30 min after the administration of the treatment, each child's REE was measured over 30 min in the postabsorptive state by indirect calorimetry using a ventilated hood system (15, 16). This test was called test A. A detailed description of the method used in Keneba has been reported elsewhere (17). Axillary temperature was measured by a mercury thermometer before and after the REE measurement. Heart rate was estimated by counting the pulse over 30 s during the REE measurement. The ambient temperature was kept in a comfortable zone to avoid the occurrence of shivering or the induction of sweating.

Three to 4 d after the therapy onset (test B), REE and anthropometry measurements were again carried out in the fasting child in the same environmental conditions. If the axillary temperature was higher than 37.5°C during test B or if the child presented any complications, he or she was then excluded from the study. On this basis, one child was excluded. A last REE and anthropometric measurement (test C) was carried out 14 to 21 d (mean duration 16.6 ± 0.6 d) after the treatment onset.

Analysis of results. The results are expressed as mean ± SEM. Statistical differences were assessed by using a paired t test when comparing two situations or an analysis of variance of repeated measurements when comparing more than two tests. The level of significance was chosen as $p < 0.05$.

RESULTS

Anthropometry, temperature and heart rate. As shown in Table 1, body weight, height, BMI, fat-free mass, and the relative body fat of the children were not significantly different among the three experimental situations, A, B, and C.

In test A, axillary temperature measured just before REE measurement was 39.4 ± 0.2°C; it decreased slightly to 39.0 ± 0.2°C at the end of REE measurement ($p < 0.05$). The mean of these two values was used as the axillary temperature (T) during test A (39.2 ± 0.1°C). In test B, axillary temperature decreased to 36.6 ± 0.1°C, a highly significant change ($p < 0.0001$). In test C, axillary temperature was 36.7 ± 0.1°C, a value similar to that observed in test B (Table 2).

In test A, heart rate was 121 ± 4 min^{-1}; it decreased to 84 ± 4 min^{-1} ($p < 0.0001$) in test B and averaged 85 ± 4 min^{-1} in test C, a value similar to that of test B.

REE and RQ. REE was positively correlated with body weight ($r = 0.948$, $p < 0.0001$; $r = 0.924$, $p < 0.0001$; and $r = 0.895$, $p < 0.0001$, respectively, in test A, B, and C). For the analysis of the results, the values of REE were normalized for body weight and expressed in kJ/kg·d. REE averaged 223 ± 10, 174 ± 8, and 169 ± 8 kJ/kg·d in tests A, B, and C, respectively (Table 2 and Fig. 1).

REE in test A was 19.2 ± 1.9% ($p < 0.0001$) higher than the basal metabolic rate predicted by the Schofield equation (18) (predicted basal metabolic rate = 187 ± 8 kJ/kg·d), whereas REE in test B and in test C were 7.6 ± 1.6% ($p < 0.0005$) and 10.0 ± 1.5% ($p < 0.0001$) lower than the predicted values, respectively.

REE in test A was 29.5 ± 2.6% ($p < 0.0001$) higher than REE in test B and 32.8 ± 1.7% ($p < 0.0001$) higher than REE in test C. However, the values of REE in tests B and C were not significantly different from each other (Fig. 1). Expression of the REE results per unit fat-free mass or body surface area did not change the degree of significance among tests A, B, and C. In test A, heart rate was positively correlated with REE ($r = 0.576$, $p < 0.01$).

The parasitemia measured at the study onset (mean value = 91 800 parasites/μL, ranging from 7600 to 818 000 parasites/μL) did not show any correlation with REE, axillary temperature, or heart rate.

RQ in test A (0.804 ± 0.013) was significantly lower ($p <$

Table 2. *Axillary temperature (T), REE, and RQ in children measured in tests A, B, and C*

	Test A (d 0)			Test B (d 3–4)			Test C (d 14–21)		
Subject	T (°C)	REE (kJ/kg·d)	RQ	T (°C)	REE (kJ/kg·d)	RQ	T (°C)	REE (kJ/kg·d)	RQ
1	39.6	209	0.775	36.6	158	0.802	36.7	155	0.968
2	38.9	203	0.825	36.8	174	0.824	36.8	150	0.974
3	39.3	189	0.796	35.9	121	0.896	37.2	137	0.901
4	38.7	165	0.734	37.0	124	0.808	36.9	122	0.833
5	39.8	317	0.782	36.0	270	0.844	37.2	264	0.824
6	40.2	185	0.971	36.3	134	0.832	36.7	142	0.902
7	37.8	222	0.765	36.3	179	0.872	37.3	184	0.870
8	38.1	204	0.785	36.5	175	0.901	37.0	174	0.860
9	39.7	271	0.809	36.5	199	0.844	36.7	187	0.895
10	39.3	245	0.914	37.2	214	0.819	36.1	183	0.813
11	39.5	209	0.817	36.9	170	0.868	36.8	164	0.874
12	39.4	180	0.773	37.0	150	0.806	35.9	132	0.876
13	39.4	236	0.815	35.9	180	0.871	35.6	166	0.912
14	38.9	222	0.785	36.9	182	0.827	36.5	171	0.918
15	39.3	209	0.789	36.5	166	0.806	36.0	158	0.876
16	39.2	251	0.742	37.2	190	0.818	36.5	189	0.839
17	39.0	209	0.792	36.5	149	0.819	36.7	156	0.824
18	39.1	194	0.773	36.5	142	0.818	36.5	140	0.872
19	39.7	319	0.825	36.5	220	0.832	37.3	232	0.923
Mean	39.2	223	0.804	36.6	174	0.837	36.7	169	0.882
SEM	0.1	10	0.013	0.1	8	0.007	0.1	8	0.010

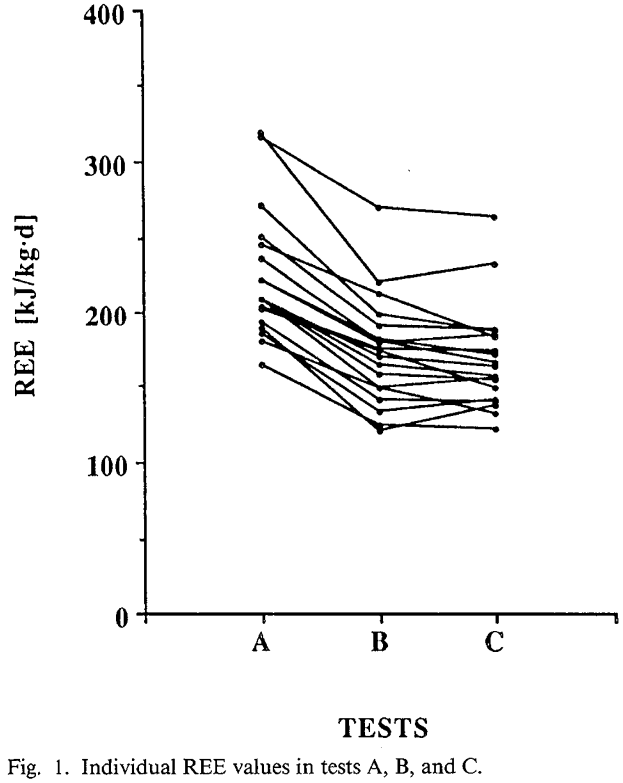

Fig. 1. Individual REE values in tests A, B, and C.

0.05) than in test B (0.837 ± 0.007); the latter was lower ($p < 0.005$) than in test C (0.882 ± 0.010) (Table 2).

Relationship between REE and axillary temperature. The individual difference in temperature between tests A and B (ΔT) was positively correlated with the difference in REE between these two tests ($r = 0.515$, $p < 0.05$). When this difference was expressed relative to the value of REE in test B (considered as a baseline), this relative difference (ΔREE) was also correlated with ΔT ($r = 0.457$, $p < 0.05$) (Fig. 2). The slope of the regression line (6.9% change in REE/°C) represents an estimate of the increase in REE/°C of fever. The intercept of the regression line described above at ΔT = 0 gives a value of a change in REE of 11.5%.

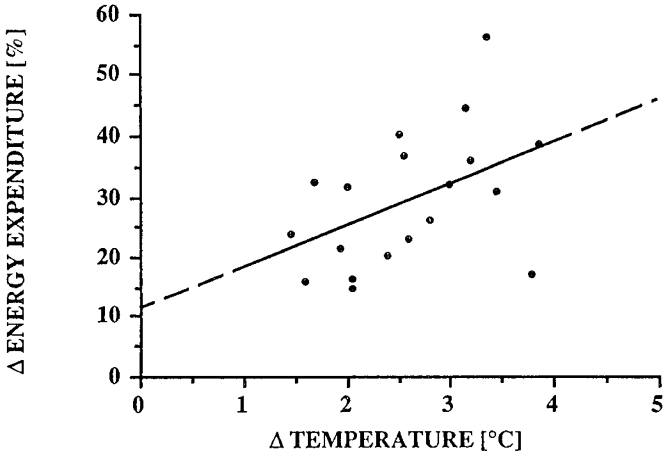

Fig. 2. Correlation between the difference in temperature (T) between tests A and B [ΔT = T(A) − T(B)] and the relative augmentation in REE (ΔREE = {[REE (A) − REE (B)]/REE B}·100) ($r = 0.457$, $p < 0.05$).

This value can be considered as the increase in REE during the acute episode of malaria that is not related to fever.

Another way to assess the global energy cost of fever is to calculate for each child the increase in REE/°C of fever (*i.e.* ΔREE/ΔT) between test A and B; this approach gives an average "cost of fever" of 11.7% of REE/°C, *i.e.* a 29.5 ± 2.6% rise in REE for a change in temperature of 2.6 ± 0.2°C. The rise of REE/°C of fever was positively correlated with BMI ($r = 0.573$, $p < 0.05$) and with the weight for height expressed as a percentage of the expected values (19) ($r = 0.539$, $p < 0.05$; Fig. 3).

DISCUSSION

The deleterious impact of malaria on growth in children (1–3) is probably explained by the occurrence of negative energy and nitrogen balances (20). The negative energy balance stems from the diminution of food intake induced by anorexia and from the hypermetabolism due to the acute episode of malaria. In the present study, the changes in energy metabolism were investigated without attempting to delineate the effect of malaria

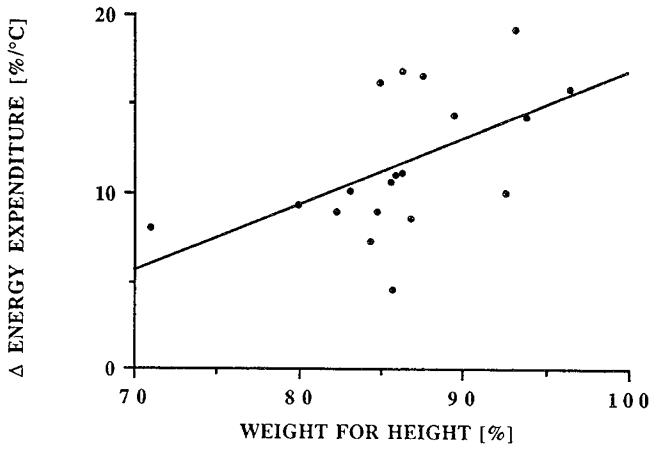

Fig. 3. Correlation between the children's percentage of weight for height (according to the National Committee for Health Statistics standards) and the relative augmentation in REE per degree of fever (ΔREE/ΔT) ($r = 0.539$, $p < 0.05$).

on food intake. Therefore, it was not possible to assess the degree of energy imbalance caused by the illness.

Anthropometry. By using the National Committee for Health Statistics standards (19), the weight of the children averaged 86.3 ± 1.3% relative to the expected weight for height (Table 1). In addition, the percentage of body fat of all the children was lower than the Fomon's standards (21). These results illustrate the poor nutritional state of these rural Gambian children.

It is interesting to note that the mean value of body weight and body composition of the children did not change significantly throughout the 14 to 21 d of study. The duration of the fever episode was less than 4 d. However, most children from developing countries do not have access to immediate therapy when they suffer from malaria. Therefore, the febrile period may last longer than in this study, which can result in a weight loss (3).

REE and RQ. In the present study, REE measured in test C (14 to 21 d after the treatment onset, when the children had recovered from the acute episode of malaria) was 10.0 ± 1.5% lower than the basal metabolic rate predicted from Schofield's equations (18), which confirms previous findings in adults from The Gambia (22). Even in the early recovery phase (test B, 3 to 4 d after the treatment onset), REE was lower than Schofield's prediction. This low metabolic rate might result from an adaptive response to the marginal nutrient intake during the rainy season, as previously shown in Gambian adults (22, 23) and children (24). This adaptive response appears to decrease the energy expenditure in individuals subjected to a chronic lack of food.

The REE due to the acute malaria episode was 29.5 ± 2.6% greater than that observed in the early recovery period (test B). Eccles *et al.* (4), however, showed a decrease in the sleeping energy expenditure of two children during the acute phase of malaria after correcting for the effect of body temperature. The reasons for these discrepant results remain unclear. An interesting observation is the early normalization in REE after the acute episode of malaria that parallels the return to a normal axillary temperature. As emphasized by McGregor (20), the situation may be quite different when the infection is not immediately treated; it is likely that energy expenditure remains elevated as long as fever is present. Inasmuch as the acute phase of malaria is characterized by anorexia, food intake is reduced. Although REE is elevated, energy expenditure due to physical activity is reduced in these children, who usually remain supine during the acute episode. If the child does not eat when febrile, the energy deficit due to malaria can be estimated to be 130% of his usual REE. For example, a 5-yr-old fasting child would expend about 5000 kJ/d during an acute episode of malaria, corresponding to a weight loss of 250 to 300 g/d (15 to 20 g/kg·d) (25).

During the acute phase, it was observed that the RQ was lower than during the early recovery phase, and then it increased again in test C. This could be explained by the fact that at the time of the first measurement the child was in postabsorptive conditions because of the anorexia related to the infection; it is likely that the child uses endogenous body fat mainly for covering his or her energy expenditure. The increase in RQ in the late recovery phase is in keeping with resuming food intake and shows that there is an increase in carbohydrate oxidation.

Relationship between fever and REE. As shown in Figure 2, the relative increase in REE was significantly correlated with the increase in axillary temperature ($r = 0.457$, $p < 0.05$). The fraction of the increase in REE explained by fever is defined by the slope of the regression line (6.9%/1°C), whereas the intercept at 0°C change in temperature can be considered as the increase in REE that is independent of fever. This corresponds to an elevation of REE of 11.5% attributed to factors other than fever, such as the increased protein turnover (26). The anemia usually observed with malaria is accompanied by an increase in cardiac output to maintain oxygen delivery to the tissues; this is accompanied by a rise in cardiac oxygen consumption.

Another way of calculating the rise in energy expenditure related to fever is to divide the individual increase in REE by the individual increase in temperature, thus assuming that all of the rise in REE can be attributed to fever. With this approach, the caloric cost of fever is 11.7% per unit rise (°C) in axillary temperature. This value is similar to that calculated by Du Bois (8).

The fact that the increase in REE for each degree of increase in temperature was positively correlated to BMI and to the percentage of the expected weight for height (Fig. 3) indicates that a child with an impaired nutritional state responds with a smaller hypermetabolism than a better nourished child for the same degree of fever. This could be explained by an adaptive mechanism to the marginal nutritional status of the child or by lower immune and metabolic responses to the infection process in the presence of malnutrition (20, 27). This observation may have important implications for saving energy in a child whose nutritional status is compromised.

In conclusion, in Gambian children suffering from an acute episode of malaria, REE was increased by 30%. There was a prompt return of REE to baseline values within 4 d after onset of therapy. These data show that the important hypermetabolism induced by the acute phase of the disease is abolished by effective therapy. When such therapy is not available, the hypermetabolism associated with anorexia may aggravate energy deficit. These observations emphasize the need to begin the treatment of malaria without any delay to reduce the duration of the severe hypermetabolism associated with the acute phase of the disease.

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