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Epidemiology of *Plasmodium falciparum* in a rice field and a savanna area in Burkina Faso. Comparative study on the acquired immunoprotection in native populations

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A longitudinal study, including entomological, parasitological, immunological and clinical data, was carried out in a rice field and a savanna village in Burkina Faso. In this study, the authors followed the evolution of several parasitological parameters in order to compare the level of immunoprotection in children of these two areas. In particular, the percentages of recently 'infected' or 'recovered' children were calculated, during the interval separating two consecutive surveys. In both areas, parasite densities quickly increased in children from 0 to 14 years old, immediately after the beginning of the transmission period. In savanna, during the rainy season (May–October), parasite densities increased and the proportion of recently 'recovered' children from 0 to 4 years old (becoming parasitologically negative between two consecutive surveys) was very low. On the other hand, parasite densities decreased and the recovery rate was higher in children from 10 to 14 years old before the end of the rainy season, while the transmission was going on. In the rice field area, *Plasmodium falciparum* densities decreased only at the end of the transmission period (December) and had the same levels as those found in savanna, in spite of a lower inoculation rate. The second peak of transmission seemed neither to increase the proportion of recovered children, nor to boost the immunoprotection of these children.

Key words: Immunoprotection; West Africa; Rice field; Parasite; *P. falciparum*

Introduction

The development of rice field in Africa, is generally accompanied by an increase in malaria transmission and morbidity, when compared with the neighbouring savanna (Surtees, 1970; Chandler and Highton, 1975; Snow, 1983; Coosemans, 1985). No comparative study has been performed on immunoprotection acquired by individuals living in these epidemiological areas.

A longitudinal study, including entomological, parasitological, clinical and immu-

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nological data was conducted in a rice field and a savanna area in Burkina Faso, in 1985–86.

Entomological data have been described elsewhere (Robert et al., 1985; Robert et al., 1988). Briefly, there was a high anopheline density in rice field, 4-times higher than that observed in savanna. In savanna, the probability of survival through a day (p) was greater than 70% from April to November and, theoretically, allowed the complete development of the sporogonic cycle during 8 consecutive months. In the rice field village, p was greater than 70% only from May to June and from November to December. The malaria transmission presented two peaks in June and November and the inoculation rate was 6-times lower than in savanna. The epidemiological paradox of the Vallée du Kou could be explained by several associated factors as indicated by Robert (1989):

- a permanent use of bed nets by local populations, on account of mosquito nuisance,
- a lower survival rate of *A. gambiae* in rice fields, which did not allow the complete development of the sporogonic cycle of *Plasmodium*,
- a zoophilic deviation of the parous females (*A. gambiae*), that contrasts with the human blood preference of young *Anopheles*.

Poor susceptibility to infection of the rice-field strain of *A. gambiae* (Mopti cytotype), could be ruled out as previously showed by experimental infections on rearing mosquitoes (Boudin et al., 1989).

The aim of this study was to compare the consequences on malarial immunoprotection and parasite loads in local populations between both villages.

Material and Methods

Study area

The study was conducted near Bobo-Dioulasso (Burkina Faso). Two villages were selected: Karankasso, located in a savanna area, and Vallée du Kou N°4 (VK4) in a rice field. This geographical region is characterized by a rainy season from June to October, with an average rainfall of 1000 mm a year. In VK4, there are two harvests a year, in April and October. The fields are flooded from January to June and from July to November.

Methodology

In both villages, children (0–14 years old) coming from 31 volunteer families, were longitudinally studied. The families were selected according to the localization of their huts, near or far from the larval habitats; and their acceptance of the survey constraints. Parasitological surveys were organized every 2 months from March 1985 to February 1986.

Peripheral blood was taken from each child present at the time of surveys and thin and thick smears were prepared. After staining with Giemsa, 50 fields of the thick film were examined. The detection threshold was 10 parasites/mm³. A hundred fields of the thin film (about 20 000 red blood cells) were also examined to identify species and to evaluate parasite density. In this case, the detection threshold was 100 parasitized red blood cells/mm³.

P. falciparum blood stage and gametocyte prevalences were estimated on thick blood films, while the rate of different *Plasmodium* species and the parasite density were evaluated on thin smears. The mean parasite density was expressed as \log_e mean (corresponding to a geometric mean) of parasitized red blood cell number per microlitre in infected children.

The proportions of individuals who became either 'infected' (parasitologically positive = a) or 'recovered' (parasitologically negative = b), were calculated as follows. Only individuals whose blood was taken in 2 consecutive surveys were included in the study. In each survey they were classified as *P. falciparum* carriers (+) or non-carriers (-). The proportion of negative subjects in the first survey who became positive in the second was given by $a = n(-+) / N-$. The proportion of positive subjects in the first survey who became negative in the second was given by $b = n(+ -) / N+$. The percentage of recently 'infected' children included both new infections and parasitological relapses. The percentage of recently 'recovered' children included true recovery and parasitological latency (under the detection threshold). These parameters were estimated for the three age-groups (group I = 0-4 years old, group II = 5-9 years and group III = 10-14 years).

Results

The most frequent species detected in the studied areas was *P. falciparum* (Table 1). *P. malariae* and *P. ovale* were commonly associated with *P. falciparum*. *P. ovale* was rarely detected (maximal prevalence = 0.3%). *P. malariae* was more frequently detected in savanna than in rice field. Prevalences fluctuated between 3.5 and 25% in savanna, and from 0 to 10.6% in the rice field area (Table 1). The maximal level was recorded during the dry season. More detailed information on *P. malariae* transmission and morbidity is indicated elsewhere (Boudin et al., 1991).

P. falciparum prevalences and densities varied in both villages according to both parameters, season and age.

In savanna

Overall prevalences of *P. falciparum* fluctuated between 35.4% in May at the end of the dry season, to 82.5% in October at the maximal level of the transmission (Table 1). The three age-groups (see Materials and Methods) presented the same evolution. The annual means were 63, 59.5 and 65.3% in the groups I, II, III respectively. They were not significantly different ($\chi^2 = 2.86$, DNS).

P. falciparum densities in infected children varied from 281 to 2980 parasites/mm³ (Fig. 1). The annual geometric means were 1675, 854 and 596 parasites/mm³ in the three groups. A significant decrease in densities was associated with age ($F = 7.12$, $P < 0.001$). Parasitological loads reached their maximal level in October in group I, and in August in the other two groups. In children from 5 to 14 years old, the densities decreased, while the transmission was going on (2.5 infected bites/person per night in September and 1.47 in October).

Gametocytes prevalences oscillated from 4 to 39.7% (Table 1). The annual means were not significantly different in the three age-groups (I = 27.1, II = 23.7 and III = 30.3%). Gametocyte densities (\log_e) in the three age-groups varied from 7 to 32

TABLE I
Species- and stage-specific plasmodial rates in savanna and rice field, during a cycle of malaria transmission

Sample	Index	Survey 1 (March)		Survey 2 (May)		Survey 3 (August)		Survey 4 (October)		Survey 5 (December)		Survey 6 (February)	
		R ^a	S ^b	R	S	R	S	R	S	R	S	R	S
Age-group I (0-4 years)	<i>n</i> ^c	77	57	81	65	79	62	66	40	76	60	70	62
	IPF ^d	29.8	58.4	16	35.4	22.7	72.5	33.3	82.5	28.9	73.3	35.7	54.8
	IPM ^e	0	3.5	0	9.2	0	8	3	25	0	25	4.3	14.5
	IGF ^f	16.9	37.3	3.7	9.2	8.8	32.2	6	20	15.8	31.6	11.4	22.5
Age-group II (5-9 years)	<i>n</i>	63	65	69	79	56	67	62	44	59	75	68	83
	IPF	49.2	56.9	31.9	48.1	33.9	64.1	58	75	45.7	57.3	29.4	47
	IPM	6.3	12.3	0	10.1	7.1	16.2	8	22.7	1.7	13.3	4.4	9.6
	IGF	23.8	30	7.2	22.7	8.9	28.3	17.7	4.5	18.6	25.3	7.3	16.8
Age-group III (10-14 years)	<i>n</i>	47	37	52	57	38	38	39	47	39	45	46	50
	IPF	34	61	30.7	59.6	31.5	78.9	46.1	72.3	38.4	53.3	23.9	54
	IPM	10.6	8.1	0	14	2.6	13.1	0	19.1	10.3	6.6	8.7	4
	IGF	19.1	39.7	0	29.8	2.6	39.4	5.1	8.5	10.2	31.1	0	28

^aR = rice field village.

^bS = savanna village.

^c*n* = number of children.

^dIPF = *P. falciparum* rate.

^eIPM = *P. malariae* rate.

^fIGF = *P. falciparum* gametocyte index.

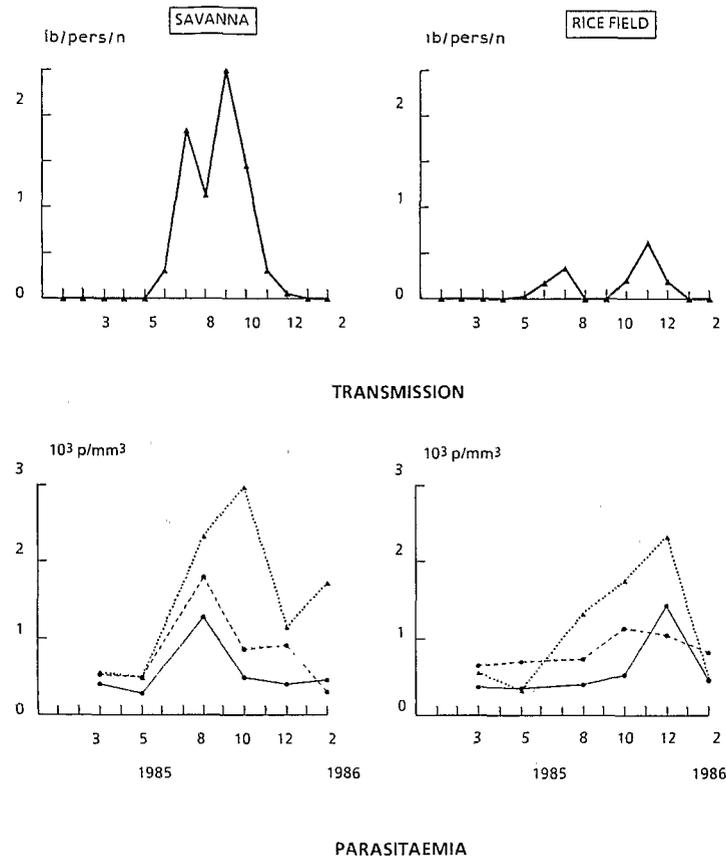


Fig. 1. Evolution, in a savanna and a rice-field area, of inoculation rates (Robert et al., 1988) and parasite densities, in three age-groups: 0-4 (.....), 5-9 (— —), and 10-14 (—) years old (for effectives, at each survey, see Table 1). ib/pers/n (infected bites/person per night)=inoculation rate; 10^3 p/mm³ (10^3 parasites/mm³)=geometric mean densities.

gametocytes/mm³ according to both age and season. The annual geometric means were significantly different (I=21.6, II=14.6 and III=11.6 gametocytes/mm³) ($F=3.57$, $P<0.05$). The first group presented more gametocytes than the third one ($t=2.63$, $P<0.02$). There was no relationship between gametocyte prevalences or densities and the transmission variations.

The percentages of recently 'infected' or 'recovered' children varied in an opposite way. These indexes respectively reached their maximal or minimal levels in August-October.

At the beginning of transmission period (May/August), inoculation rate increased progressively (from 0.3 to 1.83 infected bites/person per night). In all groups, the percentage of children who became 'infected' was high, while that of children who 'recovered' was low. Parallely, parasitaemia quickly rose.

At the maximal level of transmission (August/October), a high percentage of the children belonging to group I became 'infected' (60%), while only a few of them 'recovered' (13%) (Fig. 2). *P. falciparum* densities reached their highest levels at this

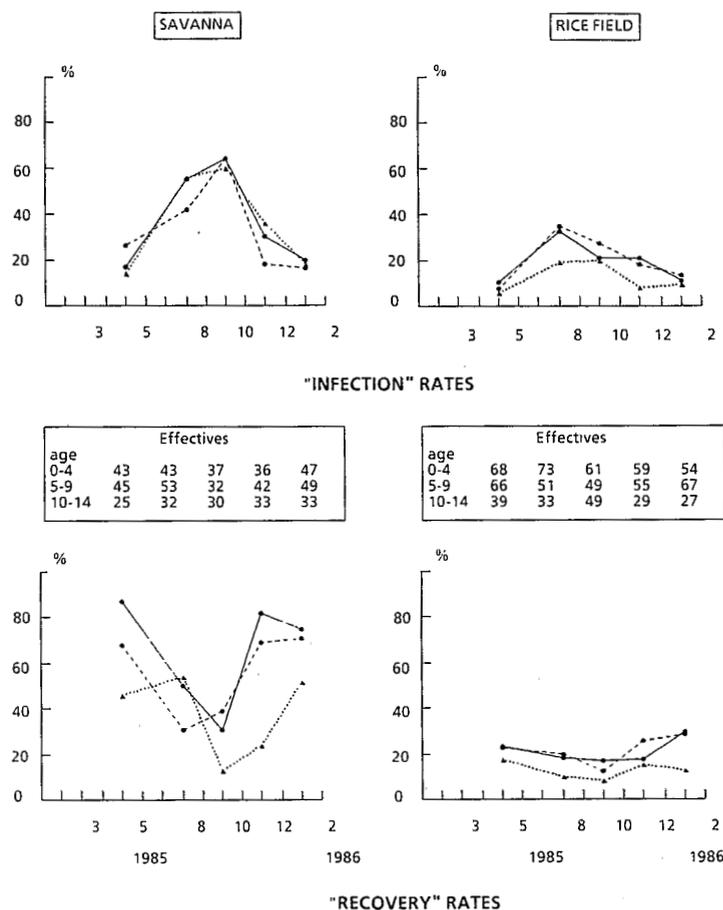


Fig. 2. Evolution, in a savanna and a rice-field area, of the percentages of children who become parasitologically positive or negative, between two consecutive surveys, in three age-groups: 0-4 (.....), 5-9 (---), and 10-14 (—) years old. Infection rate = $n(-+)/N-$ between two surveys (see text); recovery rate = $n(+ -)/N+$ between two surveys (see text).

time (Fig. 1). 64% of children from group III also became 'infected' during this period, but 31% 'recovered' (twice as many as in group I). *P. falciparum* densities decreased in group III, in spite of a high inoculation rate.

At the end of transmission period (October/December), 36% of the children from group I became 'infected' and 24% of them 'recovered'. *P. falciparum* densities began to decrease parallelly to the inoculation rate. Only 30% of the children from the third group became 'infected', while 82% of them 'recovered' (Fig. 2). The parasitaemia was stable at a low level.

In rice field

P. falciparum prevalences fluctuated between 16% in May (the end of the dry season) to 58% in October (Table 1). The annual means were not significantly different (27.4,

41.1 and 33.7% for groups I, II and III respectively). The prevalences tended to be lower than in savanna, but the difference was not statistically significant ($\chi^2=3.12$, DNS).

P. falciparum densities in infected children fluctuated from 337 to 2345 parasites/mm³, with a maximum in December, during the second peak of transmission (Fig. 1). In contrast to savanna, they increased throughout the transmission period, from May to December in all groups. Geometric mean densities in infected children presented the same levels as those obtained in savanna (I=1273, II=918 and III=604 parasites/mm³). These levels decreased significantly with age ($F=6.38$, $P<0.05$).

Prevalences of *P. falciparum* gametocytes fluctuated from 0 to 23.8% (Table 1). The annual means were 10.4, 13.8 and 6.1% in groups I, II and III respectively. These prevalences were lower than in savanna ($\chi^2=4.48$, $P<0.05$) and gametocyte densities varied from 0 to 119 gametocytes/mm³, according to age and season. The annual geometric means were 22, 30 and 8.5 gametocytes/mm³ and the gametocyte density in group III was the lowest ($t=2.51$, $P<0.05$).

At the beginning of the transmission period, we observed an increase of *P. falciparum* densities from May to August, only in group I. By contrast with savanna, groups II and III showed stable levels of parasitaemia.

During the first period of transmission (May/August), the entomological inoculation rate fluctuated from 0.18 to 0.34 infected bite/person per night. In group I, 19.6% of the children became 'infected', while only 9.6% 'recovered' (Fig. 2). Geometric mean parasite densities increased from 337 p/mm³ at the end of the dry season to 1326 p/mm³ (Fig. 1). In group III, practically the same rate of children became 'infected' (21.2%) but 18.2% 'recovered' (twice more than in group I). Children of 10–14 years old had a stable parasitaemia.

During the interval between the two peaks of inoculation rate (August/October), the transmission was very low or non-existent. In group I, the percentages of children 'infected' or 'recovered', did not change. The parasitaemia continued to increase. In group III, the percentage of 'infected' children increased, while that of 'recovered' children remained stable. However, the parasite densities and the prevalences increased.

During the second peak of transmission (October/December), the inoculation rates were higher than in the first one (from 0.61 to 0.19 infected bite/person per night). In group I, only 8.4% of children became 'infected' and 15.2% 'recovered'. In spite of this inversion of the percentages, the geometric mean parasitaemia continued to increase from 1754 to 2345 p/mm³. In group III, the proportion of children who became 'infected' decreased, while that of children who 'recovered' remained stable. The parasitaemia increased to its maximal level.

Discussion

In the African savanna village, malaria transmission is probably permanent, with important seasonal variations. Each individual living in the village can receive about 260 infected bites of *Anopheles* per year (Robert et al., 1988). Parasitological prevalences in children are high during the transmission period, while parasite densities in infected children are moderate. These data confirm the previous observations of Choumara et al. (1959), Molineaux and Gramiccia (1980) and Gazin et al. (1985).

In children of groups II and III, parasite densities decreased from August to October, while the inoculation rate was still high. This phenomenon could reflect the acquisition of an effective immunoprotection during the first half of the transmission period. We did not observe this phenomenon in children belonging to group I with a low level of malaria experience. The chloroquine self-medication was rare and could no more explain this paradoxical decrease of parasitaemia in group II and III. On the contrary, parasite loads quickly increased at the beginning of transmission. Children could have lost their partial immunoprotection during the dry season, in parallel with the decrease of antigenic stimulus.

In the rice-field village malaria endemicity is lower, in spite of a high anopheline density throughout the year. Each individual can receive about 60 infected bites of *Anopheles* per year (Robert et al., 1985). In fact, the observed inoculation rate (h) is overestimated, because it was evaluated on non-protected catchers. Therefore, children sleeping under bed nets probably receive less than 60 infected bites per year. *Plasmodium* prevalences are lower than in savanna. This fact could reflect the different levels of transmission between these two areas, and possibly a higher consumption of chloroquine. As a matter of fact, people are generally richer than in savanna and the village is nearer Bobo-Dioulasso than Karankasso. In contrast to our findings, Coosemans (1985) found, in the Rusizi-rice field (Burundi), that anopheline densities, inoculation rates and parasitological prevalences were higher than in a nearby savanna village. In this rice field area, the survival rate of *Anopheles* and the antropophilic index of *A. arabiensis* (the local vector) were higher than in VK4, and individuals did not sleep under bed nets.

In VK4, parasite densities in infected children showed the same levels as in savanna, in spite of a lower transmission. The second peak of inoculation rate (November) did not seem to boost the immunological resistance, since parasite densities were increasing from October to December. Contrasting with savanna, some young adults developed malarial attacks (Pazart, personal communication). These observations could reflect a level of immunity lower than in the surrounding savanna. But the evolution of 'infected' or 'recovered' rates is difficult to interpret in this area, probably because of the significant consumption of chloroquine. The difference between the levels of transmission in both areas could explain the apparent lower level of immunoprotection acquired by the rice-field populations, although other limiting factors could interact, such as use of bed nets, socio-economic factors and genetic differences.

In conclusion, the development of rice-field areas in Africa can be accompanied by an increase or a decrease of the malarial transmission according to ecological parameters, consumption of antimalarial drugs, use of bed nets and susceptibility of the local strains of *Anopheles*.

In stable endemic malaria areas, the equilibrium between infection and immunity, characterizes host-vector-parasite relationships. It is essential that this equilibrium, in different epidemiological situations, be carefully investigated in order not to destabilize ecological systems by introducing inappropriate modifications or control measures. Until now, there has been no parameter to estimate and follow levels of immunoprotection acquired by different age-groups. However, a better understanding of host-parasite relationships, based upon longitudinal and multi-disciplinary studies on malaria transmission and morbidity, could solve this problem.

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