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Absence of association between *Plasmodium falciparum* malaria and human immunodeficiency virus infection in children in Kinshasa, Zaire

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The possible associations between Plasmodium falciparum *malaria and HIV (human immunodeficiency virus) seropositivity were investigated in 1986 at the Mama Yemo Hospital in Kinshasa, Zaire. No significant difference was found in the HIV seropositivity rate of 164 children presenting with* P. falciparum *malaria (1.2%) and 169 healthy controls (0.6%). Secondly, no association was found between* P. falciparum *slide positivity (51.6%) and HIV seropositivity (3.8%) among 1046 children presenting to the hospital with medical complaints. Infants less than 6 months old had the lowest slide-positivity rate, but among infected children the younger ones more frequently had high parasitaemias. HIV seropositivity rates were highest for children less than 6 months old. In older children, seropositivity was strongly associated with a history of blood transfusion. Thus, in Kinshasa children,* P. falciparum *malaria is a major public health problem; perinatal transmission and blood transfusions constitute important mechanisms of HIV infection; and* P. falciparum *does not appear to act as an opportunistic agent in children infected with HIV.*

Plasmodium falciparum malaria and the acquired immunodeficiency syndrome (AIDS) are now co-endemic in several areas of Africa where they present major but dissimilar public health problems. While malaria has been occurring since remote times and affects chiefly young children, AIDS is a relatively new epidemic affecting mainly the adult population (*1*). Both *P. falciparum* malaria and AIDS cause functional abnormalities of cell-mediated immunity (*2–4*) and the potential interactions between the two diseases have recently been investigated. Repeated stimulation of the immune system by malaria antigens has been hypothesized as one factor promoting the expression and spread of the human immunodeficiency virus (HIV), the causal agent of AIDS (*5*).

Of foremost public health concern is whether malaria acts as an opportunistic infection in AIDS patients, by occurring more frequently or causing more severe symptoms. A correlation between antibodies to human retroviruses and antibodies to *P. falciparum* has been reported from a survey in

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rural Zaire (*6*), and HIV antibodies have been found more frequently in Venezuelan patients with acute malaria than in healthy blood donors from the same area (*7*). In Kinshasa, Zaire, an HIV seroprevalence of 6% was found in children hospitalized with malaria, compared with 1% in control children (*8*).

To evaluate further the association between these two diseases, we undertook studies of *P. falciparum* malaria and HIV infection in a paediatric population in Kinshasa. The results of this investigation, which also examined blood transfusions, a known risk factor for paediatric HIV infection (*8*), are reported here.

PATIENTS AND METHODS

Kinshasa, the capital of Zaire (estimated population: 2 700 000), is located on the south bank of the Zaire river, 500 km south of the equator. Malaria, predominantly due to *P. falciparum*, is endemic and affects mainly children (*9*) who in tropical Africa suffer the most from malaria-associated morbidity and mortality (*10*). AIDS appeared in Kinshasa in the early 1980s, and the annual incidence rate was recently estimated at 55 to 100 cases per 100 000 (*11*). While most AIDS cases occur in adults and result from heterosexual HIV transmission, paediatric AIDS is increasingly being noted and attributed to parenteral and perinatal HIV transmission (*11*, *12*). The investigations reported here were conducted between May and July 1986 at the Mama Yemo Hospital (MYH), the major health facility for all 24 administrative zones of Kinshasa.

Patients

Two complementary studies were designed to evaluate the potential associations between *P. falciparum* malaria and HIV infection.

Study A tested the hypothesis, whether HIV infection increases the risk of severe malaria, by comparing the prevalence of HIV infection (as denoted by the presence of HIV antibodies) in selected children with symptomatic *P. falciparum* malaria of different degrees of severity, and in asymptomatic children. Children seen at the paediatric emergency ward of MYH were enrolled in this study if they had demonstrable *P. falciparum* parasitaemia accompanied by symptoms suggestive of malaria for which no other cause could be identified. Demographic data and information about the current illness were obtained from the accompanying adult, after which a physical examination was performed and a venous blood sample drawn. If the child was comatose, cerebrospinal fluid was examined to exclude bacterial and cryptococcal meningitis. Patients with symptomatic *P. falciparum* malaria were classified as having severe malaria (comatose with a blood smear positive for asexual forms of *P. falciparum*, or blood smear with parasitization of 5% or more of the erythrocytes), or non-severe malaria (signs and symptoms of malaria, without coma and with parasitaemia of less than 5%). In addition, a control group of asymptomatic children, of comparable sex and age distribution, were recruited from among healthy siblings of children attending the MYH outpatient clinic.

Study B tested the hypothesis, whether HIV infection increases the risk of *P. falciparum* infection, symptomatic or not, by examining the prevalence of *P. falciparum* infections and HIV seropositivity in an unselected, large sample of children presenting at MYH. The study design thus permitted an evaluation of the separate patterns of *P. falciparum* infections and of HIV seropositivity in the population investigated. This study enrolled all the children, irrespective of their presenting complaints, who came to the paediatric emergency ward or the paediatric outpatient clinic of MYH, during nonrandomly selected daytime periods varying in duration from 1 to 8 hours. (Trauma and surgical patients, in a separate surgical emergency ward, were not included.) Demographic data and information about past blood transfusions were obtained from the accompanying adult, axillary temperature was measured, and a fingerprick blood sample was obtained for malaria blood smear and HIV serology.

Laboratory studies

The presence of malaria parasites was ascertained on Giemsa-stained thick and thin blood smears. A slide was considered negative when no parasites were detected on examination of approximately 1000 leukocytes on the thick smear; parasites, when present, were counted on the thin smear, against 500 to 2000 erythrocytes (for parasitaemias of 1% or more) or at least 10 000 erythrocytes (for lower parasitaemias). In patients with very low parasite densities, parasites were counted against 300 leukocytes on the thick smear.

HIV serology was performed with commercially available kits by enzyme-linked immunosorbent assay (ELISA)[a] and by Western blot analysis.[b] A specimen was considered positive for HIV antibodies if it was repeatedly reactive on ELISA, with confirmation by a positive Western blot where the banding patterns of antibodies to p24, gp41, and p110 were compared with those of known HIV-seropositive serum samples. (In four samples, the confirmatory positive Western blot occurred after one ELISA.)

Statistical analyses: Statistical analyses were per-

[a] Organon-Teknika, Charleston, SC, USA.

[b] Du Pont Company, Wilmington, DE, USA.

formed using the χ^2, Fisher's exact, and Student's *t*-tests and a logistic regression analysis.

RESULTS

Study A (malaria severity study)

A total of 333 children, aged 9 months to 12 years, were enrolled (Table 1). Among the 164 symptomatic children infected with *P. falciparum*, 59 (36%) had severe malaria and 105 (64%) had non-severe malaria. The parasite densities in the symptomatic patients varied from 0.01% to 56%. In the control group of 169 healthy children, 21 (12.4%) were found to be asymptomatically infected with *P. falciparum*, the highest parasite density being 0.5%. The HIV seropositivity rates of the symptomatic patients (1.2%) and of the healthy children (0.6%) were not significantly different. The 3 HIV-seropositive children in this study were a 5-year-old boy with severe malaria and a parasitaemia of 39.8%, a 2-year-old boy with non-severe malaria and a parasitaemia of 2.5%, and an asymptomatic 1-year-old girl who was aparasitaemic. All 3 children had previously received blood transfusions.

Table 1. Characteristics of children in study A (malaria severity)

	Total ($n=333$)	Symptomatic children ($n=164$)	Asymptomatic controls ($n=169$)
No. of Kinshasa zones represented	22 (92)[a]	22 (92)	16 (67)
No. of males	170 (51.1)	91 (55.5)	79 (46.7)
Mean age (years)	5.3	4.8	5.7
Median age (years)	4.5	3.8	5.5
No. with fever[b]	91 (27.3)	91 (55.5)	0 (0)
No. with *P. falciparum* positive slide	185 (55.6)	164 (100)	21 (12.4)
Geometric mean parasitaemia:			
All slides	0.82%	2.35%	0.01%
Positive slides	1.94%	2.35%	0.07%
No. of HIV-seropositives	3 (0.9)	2 (1.2)	1 (0.6)

[a] Figures in parentheses are percentages.
[b] Axillary temperature >37.3 °C.

Study B (malaria infection study)

A total of 1046 patients, aged 1 month to 13 years, were enrolled (Table 2). While the emergency ward and outpatient clinic patients were comparable in their demographic characteristics, they differed in their fever rates, *P. falciparum* parasitological findings, and histories of blood transfusions (Table 2).

Table 2. Characteristics of patients seen in study B (malaria infection)

	Total ($n=1046$)	Emergency ward (EW) ($n=646$)	Outpatient clinic (OC) ($n=400$)
No. of Kinshasa zones represented	24 (100)[a]	24 (100)	24 (100)
No. of males[b]	540 (51.8)	322 (50.1)	218 (54.5)
Mean age (years)	3.3	3.1	3.6
Median age (years)	2.0	2.0	2.0
No. with fever[c]	472 (45.8)	336 (53.2)	136 (34.2)[d]
No. with *P. falciparum* positive slide	540 (51.6)	403 (62.4)	137 (34.3)[d]
Geometric mean parasitaemia:			
All slides	0.88%	1.38%	0.29%[e]
Positive slides	2.41%	3.03%	1.10%[e]
No. of HIV-seropositives	40 (3.8)	28 (4.3)	12 (3.0)
No. with a history of blood transfusion	147 (14.1)	107 (16.6)	40 (10.0)[f]

[a] Figures in parentheses are percentages.
[b] Sex unavailable in 3 children in EW.
[c] Axillary temperature >37.3 °C; temperature unavailable in 16 children (14 in EW and 2 in OC).
[d] EW vs OC: χ^2 ($P<10^{-6}$).
[e] EW vs OC: Student's *t*-test ($P<0.01$).
[f] EW vs OC: χ^2 ($P<0.001$).

A total of 540 (51.6%) children had blood smears positive for asexual forms of *P. falciparum*. Slide positivity was not associated with sex but was associated with fever (defined as an axillary temperature above 37.3 °C). Infected children were more frequently febrile (54.2%) than uninfected ones (37%) (χ^2, $P<10^{-6}$); 204 (37.8%) of the slide-positive children had parasitaemias of less than 1%, 155 (28.7%) had parasitaemias of 5 % or more, and in 25 (4.6%) the parasite density reached 25% or more. The highest parasitaemia, 60%, was in a 3-month-old child seen in the emergency ward.

The patterns of parasitaemia varied with age (Table 3). Children aged less than 6 months had a significantly lower slide-positivity rate than older children. When infected, the younger children had more frequently elevated parasitaemias. The proportion of infected children with parasitaemias of 5% or more was significantly higher in younger children when the 4 successive age groups were compared, or when children under 5 years old were compared with older children. The same results were found for parasitaemias of 10% or more.

A total of 40 (3.8%) children in study B were HIV seropositive (Table 2). The seropositivity rates of children seen in the emergency ward and in the outpatient clinic were not significantly different. While no association was found with sex, the HIV seropositivity rates varied with age. Children under 6 months of age had a significantly higher prevalence of HIV antibodies than older children (Table 3). Infants 6–11 months old had the lowest seropositivity rate (1.5%).

Seropositive children were less frequently febrile than seronegative ones (fever rates of 28.2% and 46.5%, respectively; χ^2, $P=0.04$). This negative association was no longer observable when the children were stratified into slide-positive and slide-negative groups.

HIV-seropositive children had a lower malaria slide-positivity rate than the seronegative ones (37.5% vs 52.2%). This difference, which was not significant (χ^2, $P=0.1$), persisted when children seen in the emergency ward and in the outpatient clinic were considered separately. No significant difference was found between the geometric mean parasitaemias of HIV-seropositive and seronegative children, both when the mean parasitaemias for all children (0.81% vs 0.89%; Student's *t*-test, $P=0.8$) and when only those of infected children (3.9% vs 2.4%; Student's *t*-test, $P=0.2$) were considered. Parasitaemic children had a lower HIV seropositivity rate than non-parasitaemic ones (2.8% vs 4.9%; χ^2, $P=0.1$).

Of the 40 HIV-seropositive children, 23 (57.5%) (12 boys and 11 girls) had received a blood transfusion in the past. A strong association was found between HIV seropositivity and history of blood transfusion (χ^2, $P<10^{-6}$; OR=9.6; 95% confidence interval, 4.8–19.5), which persisted when considering separately children seen in the emergency ward (χ^2, $P<10^{-8}$; OR=10.7) and those in the outpatient clinic (χ^2, $P=0.001$; OR=7.2). This association was not demonstrable in the youngest children, whether considering children under 6 months old or under 1 year old. Conversely, the association between HIV seropositivity and history of blood transfusion was

Table 3. Distribution of various characteristics among children in study B, by age group

	Age group				
	<6 months (*n* = 69)	6–11 months (*n* = 134)	1–4 years (*n* = 574)	5–13 years (*n* = 269)	Total (*n* = 1046)
No. infected with asexual forms of *Plasmodium falciparum*[a]	20 (29)[b]	60 (44.8)	309 (53.8)	151 (56.1)	540 (51.6)
No. of those infected having 5% parasitaemia or more[c]	7 (35)	22 (36.7)	102 (33)	24 (15.9)	155 (28.7)
No. of those infected having 10% parasitaemia or more[d]	6 (30)	14 (23.3)	59 (19.1)	10 (6.6)	89 (16.5)
No. of HIV-seropositives[e]	7 (10.1)	2 (1.5)	22 (3.8)	9 (3.3)	40 (3.8)
No. with a history of blood transfusion[f]	4 (5.8)	11 (8.2)	95 (16.6)	37 (13.8)	147 (14)
No. of seropositives with a history of blood transfusion	1 (14.3)	0 (0)	15 (68.2)	7 (77.8)	23 (57.5)

[a] χ^2 test for trend, $P<0.0001$; and for <6 months vs ⩾6 months, $P<0.001$.
[b] Figures in parentheses are percentages.
[c] χ^2 test for trend, $P<0.001$; and for <5 years vs ⩾5 years, $P<0.0001$.
[d] χ^2 test for trend, $P=0.0001$; and for <5 years vs ⩾5 years, $P<0.001$.
[e] <6 months vs ⩾6 months, $P=0.01$ (Fisher's exact test).
[f] χ^2 test for trend, $P=0.042$.

Table 4. Association between the number of blood transfusions and HIV seropositivity in children aged 1–13 years in study B[a]

No. of blood transfusions	No. of HIV seropositives	No. of HIV seronegatives	Odds of seropositives	Crude odds ratio	Adjusted odds ratio[b]
0	9	702	0.013	1.00	1.00
1	10	83	0.120	9.40	2.32
2	3	14	0.214	16.71	5.39
⩾3	9	13	0.692	54.00	12.50

[a] χ^2 test for trend, $P<10^{-6}$.

[b] Calculated using logistic regression analysis for children of all ages, correcting for age, malaria slide-positivity, and time since last transfusion ($P=0.008$).

highly significant in both the 6-month to 13-year age group ($P<10^{-8}$, Fisher's exact test) and the 1-year to 13-year age group ($P<10^{-8}$, Fisher's exact test). These findings persisted when histories of single transfusions only were considered.

A direct association was found between the number of transfusions received and HIV seropositivity (Table 4). A trend was also noted between the date of last blood transfusion and the HIV seropositivity rate, the latter being higher in children whose transfusions were more recently administered (Table 5). In addition to these calculations of crude odds ratios, logistic regression analysis was used to assess the effect of blood transfusions on HIV seropositivity while controlling for the potential confounding effects of slide positivity, age, and time since last transfusion. While the adjusted relative odds of HIV seropositivity for the number of transfusions were somewhat lower than the crude odds ratio, the association remained highly significant (Table 4). Similarly, when the time interval since last blood transfusion was taken as the variable of interest, the adjusted odds ratios were similar to the crude estimates (Table 5).

DISCUSSION

This study confirms that *P. falciparum* malaria is a major cause of morbidity in Kinshasa children, and demonstrates that antibodies to HIV can be found in a substantial proportion of children presenting to the Mama Yemo Hospital. No direct, facilitating interaction was found between HIV seropositivity and *P. falciparum* malaria. HIV seropositivity was highest in children less than 6 months old, and in the older children was associated with a history of blood transfusion, thus confirming two major mechanisms of HIV transmission in children.

That *P. falciparum* malaria is a public health problem in the children of Kinshasa is evident from the fact that half of all children coming to MYH (study B) were infected with *P. falciparum*. In many of these children, who presented with a wide spectrum of complaints, the parasitaemia was no doubt a concomitant infection. However, in more than one quarter of the infected children, the parasite density was equal to or higher than 5%, a level likely to be associated with symptomatic, and potentially fatal, *P. falciparum*

Table 5. Association between date of latest blood transfusion and HIV seropositivity in children of study B[a]

Date of last transfusion[b]	No. of HIV seropositives	No. of HIV seronegatives	Odds of seropositives	Crude odds ratio	Adjusted odds ratio[c]
⩾24 weeks ago	4	47	0.09	1.00	1.00
12–23 weeks ago	5	25	0.20	2.35	1.64
1–11 weeks ago	7	33	0.21	2.49	2.69
Less than 1 week ago	4	10	0.40	4.70	4.41

[a] χ^2 test for trend, $P=0.048$.

[b] The date of last blood transfusion was not available for 3 of the 23 HIV-seropositive children and for 9 of the 124 HIV-seronegative children with histories of transfusion.

[c] Calculated using logistic regression analysis, correcting for age, malaria slide-positivity, and number of blood transfusions ($P=0.053$).

malaria.

The prevalence of parasitaemia was lowest in children under 6 months old. In this age group, malaria might occupy a lesser share of the spectrum of diseases necessitating a hospital visit. On the other hand, these children might be relatively protected from malarial infection, either because of decreased contact with mosquito vectors or from passive transfer of maternal malarial antibodies. When infected, however, the younger children have more frequently high, potentially dangerous levels of parasitaemia. These findings confirm that in Africa malaria tends to be more severe in younger children and justify the targetting of children less than 5 years old in efforts to reduce the impact of malaria as a disease.

The higher HIV seroprevalence found in all children coming to the hospital (study B), compared with the rate found in healthy children and in children suffering only from malaria (study A), indicates that HIV infections contribute substantially to paediatric morbidity in Kinshasa. The association of HIV seropositivity with blood transfusions confirms and quantifies previous findings (*11*, *12*). Transfusion is thus an important mechanism for HIV transmission in children in Kinshasa, which is amenable to public health intervention.

Another mode of HIV transmission was evident in children under 6 months old, who had the highest HIV seroprevalence, but in whom no association was detectable between HIV seropositivity and transfusion. In these children, vertical transmission was a likely contributor, since a previous survey of Kinshasa mothers found an HIV seroprevalence of 8% (*12*). The lower HIV seroprevalence in children aged 6–11 months, followed by an increase in older children, might reflect a loss of passively transferred maternal antibodies, followed by true infections. The failure of the seroprevalence rates in the older children to reach the same levels as in the youngest children is of interest. This might reflect a relatively low rate of HIV infection in infants born to HIV-positive mothers, or a lower survival in these children. Conversely this might indicate an increasing incidence of HIV infection in children born more recently in Kinshasa. A similar question is raised by the finding that among children with histories of blood transfusion, HIV seropositivity was higher in those whose last transfusion was received in the more recent past.

Among children coming to the hospital, a negative trend, which was not statistically significant, was found between HIV seropositivity and *P. falciparum* malaria. HIV-infected children presenting to the hospital might constitute a symptomatic group independent from children presenting with malaria-related complaints. This hypothesis is favoured by the fact that fever, which was associated with malaria, was negatively associated with HIV infection. Alternatively, the HIV-infected children might have received antimalarial drugs more frequently than HIV-negative children, for the treatment of HIV-related symptoms. Administration of antimalarial drugs for various symptoms, on the assumption that they are caused by malaria, is a practice frequently encountered in hyperendemic areas.

Neither of the two approaches adopted in this investigation demonstrated a positive association between HIV seropositivity and *P. falciparum* malaria. In study A, the prevalence of HIV seropositivity in symptomatic malaria was comparable to that found in an asymptomatic population. Thus, within this paediatric population during the period of the study, no evidence was found that *P. falciparum* malaria acts as an opportunistic infection in HIV-infected children. These findings pertain to children in whom the prevalence of malaria is high and that of HIV infection is low. Whether the same conclusions are applicable in adults, in whom symptomatic malaria is uncommon while HIV infection is more prevalent, remains to be ascertained.

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RÉSUMÉ

ABSENCE D'ASSOCIATION ENTRE LE PALUDISME À *PLASMODIUM FALCIPARUM* ET L'INFECTION PAR LE VIRUS DE L'IMMUNODÉFICIENCE HUMAINE CHEZ LES ENFANTS DE KINSHASA, ZAÏRE

En 1986, on a étudié au Mama Yemo Hospital de Kinshasa, Zaïre, les éventuelles associations entre paludisme à *Plasmodium falciparum* et séropositivité pour le VIH. On n'a trouvé aucune différence significative dans le taux de séropositivité pour le VIH entre 164 enfants atteints de paludisme à *P. falciparum* (1,2%) et 169 témoins en bonne santé (0,6%). Par ailleurs, aucune association n'a été mise en évidence entre la positivité pour *P. falciparum* observée sur lame (51,6%) et la séropositivité pour le VIH (3,8%) chez les 1046 enfants conduits à l'hôpital pour des soins médicaux. Les nourrissons de moins de 6 mois avaient le taux de positivité pour le paludisme le plus faible, mais, parmi les enfants infestés, les plus jeunes présentaient plus souvent des parasitémies élevées. Les plus forts taux de séropositivité pour le VIH ont été rencontrés chez les enfants de moins de 6 mois. Chez les autres enfants, la séropositivité était fortement associée à des antécédents de transfusion sanguine. Ainsi, chez les enfants de Kinshasa, le paludisme à *P. falciparum* constitue un gros problème de santé publique; l'infection par le VIH, elle, a surtout lieu par transmission périnatale et transfusion sanguine; enfin, *P. falciparum* ne semble pas agir comme agent opportuniste chez les enfants infectés par le VIH.

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