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Insecticide-treated curtains reduce the prevalence and intensity of malaria infection in Burkina Faso

A. Habluetzel[1,3], N. Cuzin[1], D.A. Diallo[1], I. Nebié[1], S. Belem[1], S. N. Cousens[2] and F. Esposito[3,4]

1 *Centre National de Lutte contre le Paludisme, Ouagadougou, Burkina Faso*
2 *Department of Infectious and Tropical Diseases, London School of Hygiene and Tropical Medicine, London, UK*
3 *Dipartimento di Biologia MCA, Università di Camerino, Camerino, Italy*
4 *Direzione Generale Cooperazione Sviluppo, Ministry of Foreign Affairs, Roma, Italy*

Summary A large, randomized controlled trial to investigate the impact of insecticide-treated curtains (ITC) on child mortality was conducted in an area of seasonal, holoendemic malaria in Burkina Faso. 158 communities totalling some 90000 people were censused and grouped into 16 geographical clusters, 8 of which were randomly selected to receive ITC in June – July 1994, just prior to the rainy season. In September – October 1995, at the peak period of malaria transmission, a cross-sectional survey was conducted in 84 of the villages. A random sample of 905 children aged 6–59 months was identified and visited. 763 children (84%) were present at the time of the visit and recruited into the study. Mothers were asked about fever in the past 24 h, the child's temperature was taken, and a sample of blood collected to identify and quantify malaria infections and to measure haemoglobin (Hb) levels. Children protected by ITC were less likely to be infected with *Plasmodium falciparum* (risk ratio = 0.92; 95% CI 0.86,0.98) or *P. malariae* (risk ratio = 0.42, 95% CI 0.19,0.95). The mean intensity of *P. falciparum* infections was lower among children protected by ITC (899 *vs*. 1583 trophozoites/μl; $P < 0.001$), while the mean Hb level was 0.4 g/dl higher ($P < 0.001$). While we found no evidence that ITC had an impact on the prevalence of malaria-associated fever episodes, the confidence intervals around our estimates of the impact of ITC on malaria morbidity were wide. We conclude that widespread implementation of ITC in this area of high malaria transmission led to a modest reduction in the prevalence of malaria infection and to a more substantial reduction in the intensity of these infections which caused increased Hb levels. We were unable to demonstrate any impact of ITC on malaria morbidity, but the wide confidence intervals around our point estimates do not preclude the possibility of a substantial impact.

keywords malaria, insecticide-treated curtains, RCT, Burkina Faso, infection, morbidity, anaemia

correspondence Annette Habluetzel, Dipartimento di Biologica MCA, Università di Camerino, Camerino 62032 (MC), Italy

Introduction

The spread of drug resistance and the practical problems faced in strengthening health systems in low-income countries present serious obstacles to the successful implementation of malaria control strategies based on prompt and proper case management. Preventive, vector control-based strategies have therefore attracted renewed interest in recent years, especially after the success of a pilot intervention in The Gambia, where insecticide-treated bednets combined with chemoprophylaxis were associated with a reduction in child mortality of more than 60% (Alonso *et al*. 1991). Following this study, four large, randomized, controlled trials of insecticide-treated netting (ITN), supported by the UNDP/World Bank/WHO Special Programme for Research and Training in Tropical Diseases (TDR) were conducted between 1992 and 1996. These studies in The Gambia (D'Alessandro *et al*. 1995), Ghana (Binka *et al*. 1996), Kenya (Nevill *et al*. 1996) and Burkina Faso (Habluetzel *et al*. 1997) demonstrated a positive impact of ITN on all-cause child mortality over the two years following implementation of the intervention.

Although a meta-analysis of published reports indicates that insecticide-treated bednets may reduce the incidence of

P. falciparum infection by 50% (Choi *et al.* 1995), relatively few data have been published relating to the impact of ITN on malaria morbidity. The precise nature of the relationship between malaria infection and morbidity remains unclear, especially in highly endemic areas (Greenwood 1997). Extrapolation from data on the impact of ITN on infection to their likely impact on morbidity is therefore very difficult. We report the findings of the trial conducted in Burkina Faso with respect to the prevalence and intensity of malaria infection and the prevalence of presumptive malaria episodes. This trial differed from those conducted in The Gambia, Ghana and Kenya in that the intervention involved the provision of insecticide-treated curtains (ITC) rather than bednets.

Methods

Study area and population

The study area and population have been described in detail elsewhere (Habluetzel *et al.* 1997). Briefly, the trial was conducted in 158 villages, with a total population of about 90000, living in a zone of sudan savannah with seasonal, holoendemic malaria. The majority of the population belongs to the Mossi ethnic group and lives by subsistence farming. In communities unprotected by ITC it is estimated that, on average, inhabitants are exposed to 300–500 infective mosquito bites per year, though this figure varies substantially between communities (unpublished data). The major vectors are *A. gambiae* s.s., *A. arabiensis* and, to a lesser extent, *A. funestus*.

After obtaining appropriate ethical approval and community consent (Habluetzel *et al.* 1997), an initial census of the study communities was performed between January and August 1993 and annual censuses were performed subsequently in January-March 1994 and 1995. Details of the census procedure were published by Diallo *et al.* (1996).

Randomization

The study communities were grouped into 16 large geographical clusters which were then paired on the basis of their estimated baseline mortality rates. Within the 8 pairs thus formed, one cluster was randomly selected to receive ITC in 1994.

The intervention

In June and July 1994 all house openings (doors, windows, eaves) in the 78 villages in the intervention clusters were covered with mosquito netting (Polyester, 100 Denier, Siamdutch, Thailand) which had been treated by dipping in permethrin (Imperator 50% EC, ZENECA/ICI, UK). The target dose was 1 g/m^2. After initial treatment in June/July 1994 the mean permethrin concentration was 0.58 g/m^2. This subsequently increased to 1.38 g/m^2 after retreatment in November/December 1994 and was 1.23 g/m^2 following retreatment in June/July 1995. Bioassays performed at several time points confirmed the efficacy of the treatment (Habluetzel *et al.* 1997). Coverage was estimated to be 93% in November 1994 and 94% in August 1995. Use of the curtains, as assessed by random household visits conducted between 8 and 9 pm to record the position of door curtains, appeared to decline over time: from 78% of door curtains in place in September-November 1994 to 59% in October 1995. Despite this apparent decline in use, early morning spray catches conducted in randomly selected houses in September 1995 indicated a substantial reduction in indoor biting associated with ITC (an average of 13.5 bites/person/night in control areas *vs.* 0.6 bites/person/night in intervention areas; unpublished data).

Measurement of malaria parasitaemia and morbidity

Between 4th September and 6th October 1995 (at the end of the rainy season and during the peak period for malaria transmission), a cross-sectional survey was conducted in 84 average-sized villages (40 intervention and 44 control) in all 16 geographical clusters. A computer-generated random sample of 12% of children aged 6–59 months (approximately 900 children) was produced based on the most recent census data and the households of all these children were visited by 1 of 6 fieldworkers. All mothers were asked whether their child had had a fever during the past 24 h and informed consent was obtained from each to measure the child's axillary temperature and collect a blood sample by fingerprick. The child's temperature was taken twice, once under each arm. If a difference of > 0.5 °C was observed the measurements were retaken. Children reported febrile by the mother or with a recorded temperature $\geqslant 37.5$ °C were given presumptive malaria treatment (chloroquine and paracetamol). From the blood sample thick and thin films were prepared. Blood spots were also conserved on filter paper. Each field team was visited every other day by a supervisor to check that the correct procedures were being adhered to. This supervisor was also responsible for returning the completed forms, the blood slides and blood spots to the Centre National de Lutte contre le Paludisme (CNLP), located in Ouagadougou some 35–60 km from the study area.

Blood slides were stained with Giemsa and read by CNLP staff in Ouagadougou. Parasite density was estimated by counting 100 fields and equating this to 0.25 μl of blood. Suspected *P. vivax* infections were confirmed at the Swiss Tropical Institute in Basel. The children's haemoglobin (Hb) levels were estimated by eluting blood from spots stored on

filter paper and measuring light absorbance at a wavelength of 550 nm. A study of 83 individuals, who provided both spots to be processed by this method and blood samples which were analysed conventionally with a Coulter haemoglobinometer, found a correlation between the Hb level estimated from the blood spot and that recorded by the haemoglobinometer of $r = 0.86$. Details of the new method and its performance compared with the conventional method will be presented elsewhere.

Data entry and analysis

Data were double-entered onto microcomputer in Ouagadougou using Epi-Info version 5.0 and analysed using Stata versions 4.0 and 5.0. Statistical comparisons of intervention and control groups took account of the cluster randomization. For unadjusted analyses a summary measure was calculated for each outcome of interest for each cluster and these summary values were compared using the paired *t*-test. Adjusted analyses of the prevalence of infection and morbidity were performed by fitting logistic regression models including age and sex, but excluding any effect of treatment group. Observed and expected prevalences of infection and morbidity were calculated for intervention and control groups and compared to provide a point estimate of the risk ratio associated with the intervention. A summary 'residual' was then calculated for each outcome for each cluster and paired *t*-tests were performed on these summary residuals. Approximate 95% confidence intervals (CI) for these risk ratios were calculated using the standard error:

$$\text{SE}[\ln(\text{risk ratio})] = (1/\sqrt{8}) \times (\text{standard deviation of the ratio of paired residuals}).$$

Adjusted analyses of parasite density and Hb level were performed using linear regression and treating the pairs of clusters as random effects.

Results

905 children aged 6–59 months were selected for inclusion in the study. Of these, 763 (84%) were present on the day of the visit and were recruited. Absent children did not differ from those included in the study with respect to age (mean of absent children 33.1 months *vs*. 32.6 months for those present). A slightly higher proportion of girls (18%) than boys (13%) was absent. There was little difference in absenteeism between intervention and control communities (17% *vs*. 15%).

Table 1 shows the distribution of the children by age, sex and whether or not the mother reported the child to have fever. The age and sex distributions in the intervention and control clusters are broadly similar. A slightly higher proportion of children in the intervention clusters than the control clusters were in the oldest age group (48–59 months) (25% *vs*. 18%) and were male (52% *vs*. 46%). A larger proportion of children in the control clusters were reported febrile than in the intervention clusters (36% *vs*. 25%; $P = 0.01$).

Table 1 Distribution by age, sex and reported fever of children present on the day of the survey

	Number (%) of children	
	Intervention clusters ($n = 375$)	Control clusters ($n = 388$)
Age in months		
6–11	42 (11)	50 (13)
12–17	44 (12)	44 (11)
18–23	48 (13)	40 (10)
24–35	76 (20)	93 (24)
36–47	71 (19)	90 (23)
48–59	94 (25)	71 (18)
Sex		
Male	196 (52)	180 (46)
Female	179 (48)	208 (54)
Reported febrile		
No	282 (75)	250 (64)
Yes	93 (25)	138 (36)

Malaria infection

Blood slides were obtained from 761 children. 665 (87%) had evidence of *P. falciparum* infection, of whom 624 were infected with asexual blood stages. The prevalence of infection was lower among children in intervention clusters than among children in the control clusters (83% *vs*. 91%). Age-specific prevalence rates by treatment group are shown in Figure 1. After adjusting for age and sex, the estimated risk ratio for children in intervention clusters compared with those in control clusters was 0.92 (95% CI 0.86–0.98, $P = 0.02$). The distribution of *P. falciparum* trophozoite parasite densities is presented in Table 2. In the intervention clusters 39% of the children had parasite densities of 1000/μl of blood or more, while in the control clusters the corresponding figure was 55%. In the intervention clusters the geometric mean *P. falciparum* trophozoite density among positive children was 899/μl, compared with 1583/μl in the control clusters. After adjusting for age and sex, the estimated relative reduction in parasite densities among positive children living in the intervention clusters was 42% (95% CI 22% to 58%, $P < 0.001$). This reduction was most apparent in children ≥ 2 years (Figure 2). There was no statistically sig-

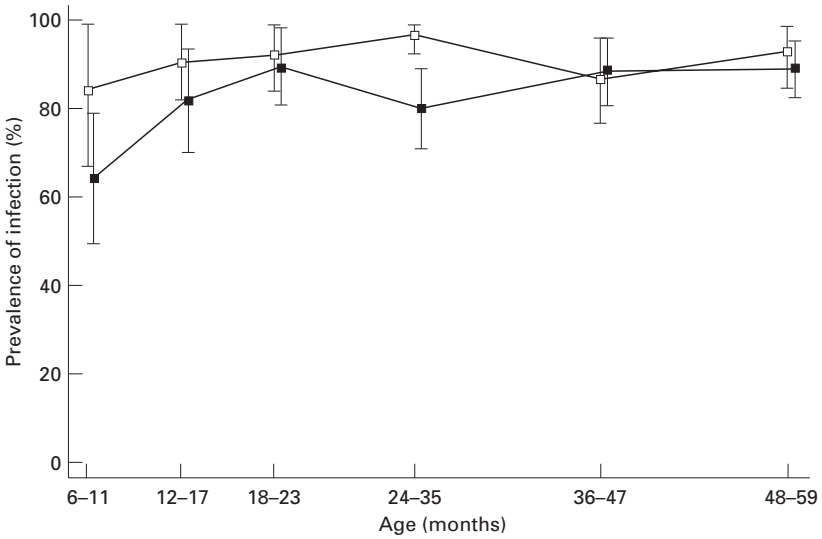

Figure 1 Prevalence (%) of *P. falciparum* infection by age and by treatment group. ■ Intervention; □ Control. (Vertical bars indicate the range of the 95% confidence interval for the measure being estimated).

nificant difference in the gametocyte index between intervention clusters and control clusters (39% *vs.* 43%; $P = 0.29$).

Infection with *P. malariae* was less common (22%) than infection with *P. falciparum*, and was usually (154/169 cases) associated with the latter. Infection was again more frequent in control than in intervention clusters (30% *vs.* 14%). After adjusting for age and sex, the estimated risk ratio for children in intervention clusters compared with those in control clusters was 0.42 (95% CI 0.19–0.95, $P = 0.04$). Only 7 and 1 children, respectively, had evidence of *P. ovale* and *P. vivax* infections, all concurrently with *P. falciparum* infection.

Malaria-associated fever

Table 3 shows the prevalence of measured fever (axillary temperature ≥ 37.5 °C and *P. falciparum* 'malaria' episodes in the intervention and control clusters for a range of parasite thresholds. Despite the higher reported fever rate among children in the control clusters and the lower prevalence and intensity of malaria infection among children in the intervention clusters, there was no evidence that the prevalence of malaria morbidity was lower in children in the intervention group. The prevalence of measured fever, with or without parasitaemia, was lower in the intervention clusters (risk ratio =0.85; $P = 0.06$), but there was no evidence that fever associated with *P. falciparum* infection was less common, irrespective of the threshold parasite density used. However, the confidence intervals around the risk ratio estimates are wide. Figure 3 shows the prevalence of fever by *P. falciparum* parasite density and by treatment group. In the range of parasite densities 2500–9999/μl the risk of fever appears, if anything, to be higher among children protected by curtains. We fitted various logistic regression models to examine whether the risk of fever, given a particular parasite density, differed between the two groups of children. None of the models fitted provided any statistical evidence that the relationship between parasite density and risk of fever differed between the two groups of children.

Table 2 Distribution of *P. falciparum* parasite densities by treatment group

Parasite density (trophozoites of *P. falciparum* per μl)	Number (%) of children	
	Intervention clusters ($n = 374$)	Control clusters ($n = 387$)
Negative	77 (21)	60 (16)
1–999	151 (40)	113 (29)
1000–2499	52 (14)	66 (17)
2500–4999	31 (8)	62 (16)
5000–9999	20 (5)	45 (12)
10000 ⩾	43 (11)	41 (11)

Haemoglobin levels

Estimated haemoglobin (Hb) levels were available for 533 children (70%) and ranged from 6.0 to 12.5 g/dl. Thirty-one children (6%) had Hbs below 7 g/dl, while 233 (44%) had Hbs below 9 g/dl. Only 28 children (5%) were estimated to have Hbs of 11 g/dl or more. Thus 95% of children were estimated to be anaemic according the international definition of anaemia. Hb levels increased with age, from a mean of 8.3 g/dl in children under 12 months to 10.0 g/dl in children aged 48–59 months. Anaemia was more prevalent in control than intervention clusters (98% *vs.* 91%), with the mean Hb

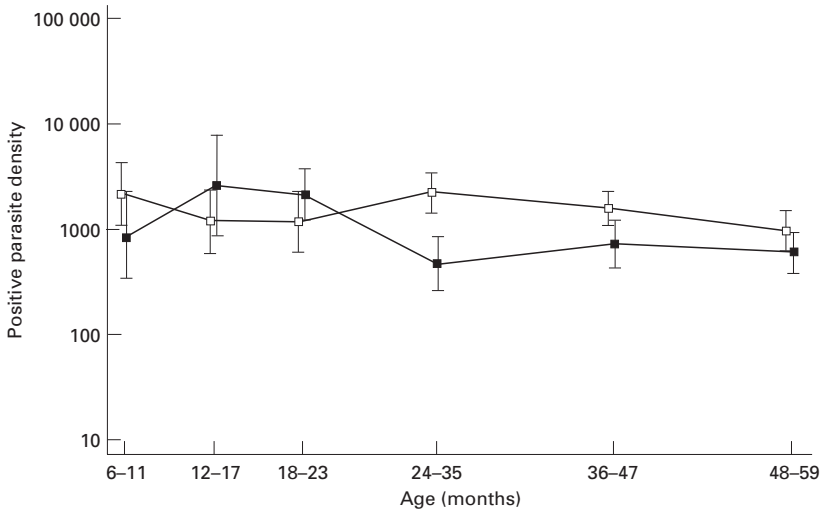

Figure 2 Geometric mean density of *P. falciparum* infections (trophozoites/μl) by age and by treatment group. ■ Intervention; □ Control. (Vertical bars indicate the range of the 95% confidence interval for the measure being estimated).

level 0.45 g/dl higher in children living in intervention clusters than in children living in control clusters (9.4 g/dl *vs.* 8.9 g/dl). This difference remained statistically significant after adjusting for age and sex (adjusted difference = 0.41 g/dl; 95% CI (0.23,0.58); $P < 0.001$), but was only apparent among children ≥ 2 years (Figure 4). There was a weak negative correlation between Hb level and parasite (trophozoite) density ($r = -0.15$). Although adjusting for parasite density reduced the estimated mean difference in Hb between intervention and control groups (from 0.41 to 0.37 g/dl), this difference remained statistically significant ($P < 0.001$).

Discussion

We observed a modest reduction in the overall prevalence of malaria infection and almost a halving of parasite densities among children protected by ITC. We found no evidence of a reduction in the prevalence of fever associated with malaria infection. Haemoglobin levels were higher in intervention than in control clusters. Entomological data indicate that children protected by ITC are likely to be exposed to 20–30 infective bites per year, while children unprotected by ITC are likely to be exposed to several hundred such bites each year (unpublished observation).

Our study, based on the randomization of 16 clusters of communities, was able to demonstrate an impact, albeit small, of ITC on the prevalence of malaria infection during the peak transmission season in Burkina Faso. In a smaller, earlier study, also performed in Burkina Faso, Procacci *et al*. (1991) observed a substantial reduction in the prevalence of infection during the dry season among children protected by ITC, but were not able to demonstrate any such reduction during the rainy season. In Tanzania, where vector control measures had an impact on malaria transmission of a similar order of magnitude to that achieved in the current study, modest reductions in the prevalence of infection were observed, with the greatest reductions occurring during the period of lower transmission (Curtis *et al*. 1998). Thus, our results may indicate the minimum impact of ITC on the prevalence of infection.

Table 3 Distribution of measured fever and *P. falciparum* 'malaria episodes' in intervention and control clusters

Definition of episode	Children with fever/'malaria episode'% (number) Intervention clusters ($n = 374$)	Control clusters ($n = 387$)	Adjusted* risk ratio (95% CI)	*P*-value
Fever (temperature ≥ 37.5 °C)	18 (67)	20 (79)	0.85 (0.71,1.01)	0.06
Fever with parasitaemia	15 (57)	17 (64)	0.88 (0.67,1.16)	0.32
Fever with 1000 + parasites/μl	11 (42)	12 (46)	0.92 (0.51,1.66)	0.75
Fever with 2500 + parasites/μl	8 (31)	9 (33)	0.90 (0.43,1.85)	0.73
Fever with 5000 + parasites/μl	6 (23)	6 (23)	0.98 (0.41,2.35)	0.96

* Adjusted for age and sex.

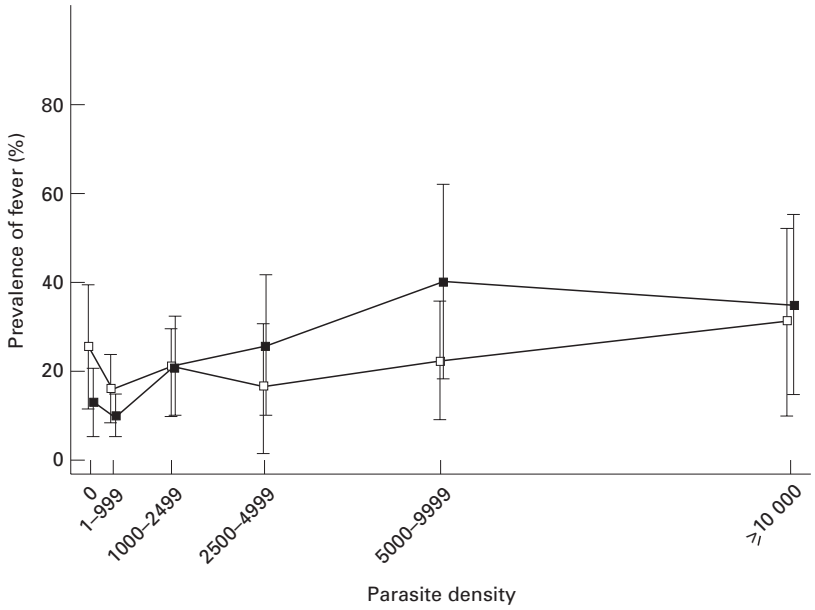

Figure 3 Prevalence (%) of measured fever by intensity of *P. falciparum* infection (trophozoites/μl) and by treatment group. ■ Intervention; □ Control. (Vertical bars indicate the range of the 95% confidence interval for the measure being estimated).

The impact on the prevalence of *P. malariae* infection (30% *vs.* 14%) appears to have been more substantial than that on *P. falciparum* infection (91% *vs.* 84%). This finding is not unexpected given the anticipated impact of ITC on mosquito life expectancy and the longer sporogonic cycle of *P. malariae*. The relatively modest reduction in the prevalence of *P. falciparum* infection and the reduction in its intensity occurred in a setting with previously high transmission, in which the average number of infective bites per person per year is estimated to have been reduced by more than 95% from several hundred to tens of bites (unpublished data). That a very substantial reduction in transmission results in a less dramatic reduction in the prevalence and intensity of malaria infection is not surprising. Although the precise natures of the relationships between transmission, infection and disease are poorly understood, it is known that they are complex and nonlinear (Snow & Marsh 1995; Trape & Rogier 1996). Nevertheless, although the confidence intervals are wide and do not preclude a substantial impact on morbidity, the absence of any apparent reduction in the prevalence of fever associated with parasite densities above 1000/μl, following a large reduction in transmission, is a disappointing finding. It is, however, consistent with the findings of the Tanzanian study previously mentioned (Curtis *et al.* 1998), where no impact on malaria morbidity was detectable during the peak transmission season although the authors

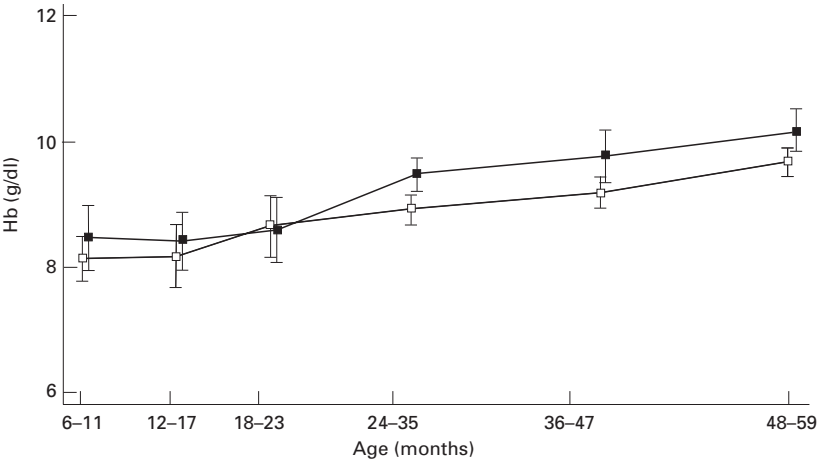

Figure 4 Mean haemoglobin level (g/dl) by age and by treatment group. ■ Intervention; □ Control. (Vertical bars indicate the range of the 95% confidence interval for the measure being estimated).

report 'some evidence' of a benefit subsequently during a period of lower transmission.

Nevill *et al*. (1996), working with insecticide-treated bednets in an area of low transmission in Kenya, detected a substantial (44%) reduction in the number of severe malaria cases presenting to hospital over a two-year period. This reduction was even more substantial than that observed for mortality (33%) over the same period. We observed a reduction of around 30% in the number of febrile episodes reported by mothers, with a smaller (15%) reduction in the prevalence of febrile episodes measured by the study team. However, when attention was restricted to measured fever associated with *P. falciparum* infection, there was no evidence of an impact of the curtains. This finding is consistent with the earlier work of Procacci *et al*. (1991) in Burkina Faso, who reported an impact of ITC on morbidity during the dry season but not during the rainy season. A conservative interpretation of our results might be that, during the peak transmission season in Burkina Faso, ITC have no impact on malaria morbidity.

However, the confidence intervals around our point estimates for the impact of ITC on malaria morbidity are wide. For example, using as our definition of a malaria episode measured fever with *P. falciparum* infection, we estimate a 12% reduction due to ITC (with a 95% CI from − 16% to 33%). This point estimate is not dissimilar to the overall estimated reduction in mortality (15%; 95% CI–4% to 30%) over two years (Habluetzel *et al*. 1997). Any such comparison of cross-sectional morbidity data with longitudinal mortality data should be treated with circumspection, especially since during the period when the cross-sectional survey reported here was performed, mortality in the control clusters was unusually low, with fewer deaths in control than in intervention clusters (Habluetzel *et al*. 1997).

It has been suggested that, while ITN may protect children initially against malaria mortality, they may delay or diminish the acquisition of immunity against malaria (Snow *et al*. 1994). This hypothesis, which has been and remains a subject of heated debate (e.g. Greenwood 1997), together with our observation of reduced parasitaemia without any apparent concomitant reduction in morbidity, led us to investigate whether there was any evidence that ITC-protected children tended to develop fever at lower parasite densities than unprotected children. Such a difference might indicate reduced tolerance of infection among ITC-protected children and hence, perhaps, reduced immunity. While visual inspection of the data (Figure 3) suggested a higher prevalence of fever among ITC-protected children at parasite densities between 2500 and 9999/μl, we were unable to demonstrate any statistically significant difference.

In addition to the reduction in the prevalence and intensity of *P. falciparum* infection among children protected by ITC, we also observed an increase in Hb levels of about 0.4 g/dl. Although controlling current parasite density at the individual level did not remove the association between ITC and improved Hb, it is likely that the impact of ITC on Hb is through a reduction in parasite density. This mechanism is biologically plausible and, at the population level, the improvement in Hb was most apparent in children aged ≥ 2 years, the same age range in which the reduction in parasite density was most evident. The low correlation between parasite density and Hb at the individual level may reflect random error in the measurement of both Hb and parasite density or that Hb levels depend not only on current parasite density but also on intensity of infection in the past.

In summary we conclude that the widespread implementation of ITC in a high transmission area led to a modest reduction in the prevalence of malaria infection and a more substantial reduction in the intensity of these infections, in turn increasing Hb levels. We were unable to demonstrate any impact of ITC on malaria morbidity, but the wide confidence intervals around our point estimates do not preclude the possibility of a substantial impact.

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