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Highland malaria in Uganda: prospective analysis of an epidemic associated with El Niño

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Abstract

Malaria epidemics in African highlands cause serious morbidity and mortality and are being reported more frequently. Weather is likely to play an important role in initiating epidemics but limited analysis of the association between weather conditions and epidemic transmission parameters has been undertaken. We measured entomological variables before and during an epidemic of malaria (which began in February 1998) in a highland region of south-western Uganda and analysed temporal variation in weather data against malaria incidence (estimated from clinic records), mosquito density and entomological inoculation rates (EIR). Indoor resting density of *Anopheles gambiae s.l.* was positively correlated with malaria incidence ($r = 0{\cdot}68$, $P < 0{\cdot}05$) despite extremely low vector densities. EIR totalled only 0·41 infectious bites per person during the entire 8-month study period. Rainfall during and following the El Niño event in 1997 was much higher than normal, and rainfall anomaly (difference from the mean) was positively correlated with vector density 1 month later ($r = 0{\cdot}55$, $P < 0{\cdot}05$). Heavier than normal rainfall associated with El Niño may have initiated the epidemic; the relationship between temperature and transmission parameters remains to be defined. The results from this study indicate that, in this highland population, epidemic malaria may occur at extremely low inoculation rates.

Keywords: malaria, altitude, El Niño, epidemics, disease outbreaks, weather, *Anopheles*, Uganda

Introduction

Malaria epidemics in highland areas of Africa are being reported more frequently, including recent outbreaks reported from Madagascar (1200 m) (LEPERS *et al.*, 1988), Kenya (1780 m) (MALAKOOTI *et al.*, 1998; SOME, 1994), Burundi (1450 m) (MARIMBU *et al.*, 1993), and Uganda (1800 m) (MOUCHET *et al.*, 1998). Because the inhabitants of these areas have limited immunity to *Plasmodium* spp. owing to infrequent exposure, periods of increased transmission are likely to produce severe morbidity and mortality. To compound this problem, outbreaks generally take the health care sector by surprise, limiting a rapid and effective response to the diagnosis and treatment needs of those afflicted.

Weather conditions, principally rainfall, temperature and humidity, are considered to be the most important causes of epidemics in non-endemic areas (GILLES, 1993). Key malaria transmission parameters, such as mosquito population density and longevity, and the extrinsic incubation period of the parasite in the mosquito, are extremely sensitive to weather conditions. In areas of unstable transmission such as highlands, small changes in transmission parameters can have substantial impacts on human morbidity and mortality (ONORI & GRAB, 1980). Although not an epidemic investigation, LOEVINSOHN (1994) has shown malaria incidence in the highlands of Rwanda to be strongly related to both rainfall and minimum temperature. Simulation models of global-warming effects have predicted that malaria transmission will increase at high altitudes in Africa if temperatures rise (MARTENS *et al.*, 1995; JETTEN *et al.*, 1996; LINDSAY & BIRLEY, 1996; MCMICHAEL *et al.*, 1996).

Despite circumstantial and modelling evidence that weather conditions are likely to play an important role in highland malaria epidemics in Africa, no prospective analysis of associations between weather and malaria transmission parameters has been related to epidemic transmission. An understanding of the role that weather plays in initiating highland epidemics may be useful for forecasting and early warning.

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During the course of studies on environmental determinants of malaria in a highland region of Uganda, the people in our study area suffered a severe malaria epidemic beginning in February 1998. In this report we present the results of entomological investigations initiated before and continued during this epidemic, and analyses of how weather conditions affected transmission parameters.

Materials and Methods

Study area

Approximately 3% of Uganda's 20 million inhabitants live in the south-western highlands of Kabale District (1° 05′–1° 30′S, 29° 45′–30° 15′E) at an altitude of 1500–2400 m (Ministry of Finance and Economic Planning, 1992). Rainfall (850–1200 mm annually) is seasonally bimodal, whereas average daily minimum (9·8–12·6°C) and maximum temperatures (23·2–24·4°C) are fairly constant throughout the year. Whether malaria transmission occurred in the district during the early 1900s is debatable, but a World Health Organization (WHO) team which conducted control activities in Kabale from 1959 to 1963 identified a focus of endemic malaria around Lake Bunyonyi in which *Anopheles funestus* was the principal vector; sporadic small outbreaks of malaria occasionally associated with *An. gambiae s.l.* were also noted (DE ZULUETA *et al.*, 1964). Our study focused on an area of this district (1770–1920 m elevation) located within a 26-km radius of Kabale Town (Fig. 1).

Changes in malaria incidence

Monthly reports from 5 rural health clinics in the study area were used to estimate changes in malaria incidence. These reports contained the number of people with new clinical malaria diagnoses and were used to estimate the number of new infections. The denominator for the incidence calculation was the total population size of the clinics' catchment areas as determined from the 1991 census records and adjusted for population growth (Ministry of Finance and Economic Planning, 1992). In October 1997, January 1998, and August 1998, 1 of the 5 clinic reports was missing; the catchment area for the missing clinic was therefore not considered in the denominator of the incidence rate for that month.

Malaria cases are not regularly slide-confirmed in these health units. Therefore, we monitored the accuracy of diagnosis across the study period by taking thick blood

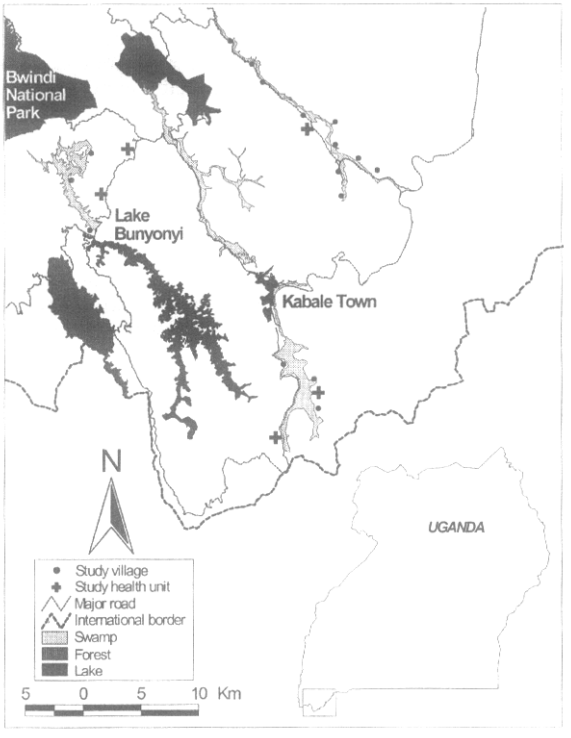

Fig. 1. Map of the study area in south-western Uganda and location of 16 study villages and 5 health units.

films from a sample of malaria patients at each health clinic from January through August 1998. The sample consisted of all patients residing in the clinic catchment area presenting with a temperature of ≥37·5°C who were diagnosed with malaria by the clinician. A brief questionnaire requesting demographic and travel history was administered to all participants. Blood films were dried, stained with Field's stain and examined by microscopy; a slide was declared negative if 100 fields were scanned at high power without finding parasites. Patients with positive blood smears were compared to patients with negative blood smears to identify characteristics associated with *Plasmodium* parasitaemia.

Entomological survey

We used the pyrethrum spray collection method as described by WHO (1975) to sample indoor resting mosquitoes in 16 villages twice per month from January through July 1998. (In December 1997, resting collections of mosquitoes were conducted on a limited basis with 1 collector using an aspirator to collect mosquitoes for 30 min.) Villages were selected along the valley bottoms at 2-km intervals. The 5 thatch houses situated closest to the valley bottom in each village were sampled throughout the duration of the study except when circumstances required a substitute house. We calculated the indoor resting density (IRD) as the average number of *An. gambiae s.l.* per house for each village and sampling period.

The presence of *P. falciparum* sporozoites in mosquitoes was determined using an enzyme-linked immunosorbent assay (ELISA) according to the method of WIRTZ *et al.* (1987). The sporozoite rate was calculated as the number of positive specimens divided by the total tested. Abdomens of captured anophelines were tested singly for the presence of human blood using a sandwich ELISA procedure (CHOW *et al.*, 1993). The human blood index (HBI) was calculated as the proportion of mosquitoes with partially digested or undigested blood meals testing positive for human blood.

Daily human biting rates (HBR) per house-sample were estimated using the following equation:

$$\text{HBR} = \frac{\text{Mosquitoes with undigested human blood} + (1/2)\ \text{Mosquitoes with partially digested human blood}}{\text{Number of people who slept in the house}}$$

Daily entomological inoculation rates (EIR) were calculated as the product of the HBR and the sporozoite rate for each house in each sampling period; mean EIR was calculated for each village as the arithmetic mean of all houses sampled during that period, and then for the sampling period as the mean of all villages.

Weather data

The meteorological recording station in the study area which consistently recorded temperature and rainfall was at the Kabale District Offices located at 1980 m. Daily maximum and minimum temperatures and rainfall for October 1997 through July 1998 were obtained from this station as were monthly summaries for previous years.

Bimonthly (2-week period) rainfall and temperature anomalies were calculated from October 1997 through August 1998. Historical (1960–97) monthly average rainfall was halved to estimate the 2-week period amount, and then each 2-week period was smoothed by calculating a moving average to weight each period by the preceding and following monthly average; greater weight was given to the preceding month for the 2-week period at the beginning of the month of interest while more weight was given to the following month for the 2-week period at the end of the month of interest. Rainfall anomalies for each 2-week period were then calculated as the difference between observed bimonthly totals and the corresponding period's historical, smoothed average. Bimonthly maximum and minimum temperature anomalies were calculated similarly, except that the interval used for the historical average was taken from 1992–97 (because the longer time series shows an increase in mean maximum and minimum temperature over time) and the temperature for each historical 2-week period was taken to be the same as that for the month, and then smoothed as for rainfall.

Relative humidity (RH) was recorded at 1 house in each of the 16 study villages using a HOBO data logger (ONSET Computer Corporation, Pocasset, MA, USA). The RH data loggers were hung outdoors under the eaves of the house and RH was recorded hourly.

Saturation deficit, the difference between the maximum amount of water the atmosphere can hold at the current temperature (saturation vapour pressure) and the actual amount it holds (RH), was calculated for each day (CAMPBELL & NORMAN, 1998). A higher saturation deficit means greater evaporative water loss which generally decreases mosquito longevity (MOLINEAUX, 1988). Hourly temperatures were estimated from daily maximum and minimum temperatures using the method of CAMPBELL & NORMAN (1998), then combined with hourly RH readings at each village to estimate saturation deficit (D) using the following equation:

$$D = e_s(T)(1 - RH)$$

where $e_s(T)$ is the saturation vapour pressure at a given temperature (T). Hourly saturation deficit values were averaged by day within each village and an arithmetic mean of all villages was taken to give a daily mean saturation deficit for the study area.

Statistical analyses

Cross-correlations between time series data were computed and correlation coefficients were considered statistically significant ($P < 0{\cdot}05$) if they were greater than twice the standard error. A χ^2 test for trend was used to examine changes in categorical variables over time. Student's t-test was used to test for differences in means

of continuous variables. Odds ratios and 95% confidence intervals (95% CI) were calculated to identify factors associated with *Plasmodium* parasitaemia.

Human volunteers

Written, informed consent was obtained from all patients who provided blood samples; written permission was obtained from a parent or guardian of children aged <18 years. Oral permission was obtained from members of individual households for spray catches. This project was approved by the Health Sciences Institutional Review Board of the University of Michigan and the Uganda National Council for Science and Technology.

Results

The epidemic began in February 1998 (Fig. 2) and peaked in March with a malaria incidence almost 3 times greater than the mean of the previous 5 years. Incidence remained elevated through May and then returned to the historical monthly average. From January to June 1998, the proportion of clinical malaria patients positive for malaria parasites remained fairly constant (between 61·5% and 77·9%), indicating that increased incidence was not due to a change in diagnostic criteria or another febrile illness. There was a trend, however, toward lower slide positivity rates as the epidemic waned (trend $P < 0{\cdot}001$), with slide positivity rates remaining very low in August, suggesting that the increased incidence in that month may have included cases due to another cause.

Characteristics of malaria patients

A total of 36 112 new clinical malaria cases, representing 41·1% of the total catchment area population, was reported from January through August 1998. Of blood slides taken from 694 (1·9%) of these patients to confirm diagnosis, 61·5% were parasitaemic. Characteristics of slide-positive patients compared to slide-negative patients are presented in Table 1. Because other studies have found travel history to be associated with parasitaemia in African highlands (VAN DER STUYFT *et al.*, 1993) we examined differences in the proportion of slide-positive and slide-negative patients who had slept away from their home in the previous 30 days and found that the proportions were similar, indicating that few parasitaemias were acquired elsewhere.

In endemic areas, children aged <5 years are at greatest risk of acquiring malaria infections; in this region of low and unstable transmission, however, both young and old people appeared equally at risk from malaria, although children aged 10–15 years were twice as likely to be parasitaemic compared to the oldest age-group. Peak infection would be predicted to occur in this age-group in areas with low levels of transmission (WOOLHOUSE, 1998) but it is not clear whether the increased odds of parasitaemia at these ages represented peak infection levels, an increased age-specific exposure rate or sampling bias.

Patients with a positive blood slide more often reported recent chloroquine usage, perhaps indicating that parasitaemic patients experienced more severe symptoms provoking increased use of chloroquine before clinic presentation. Alternatively, our sample may have been biased toward patients for whom chloroquine was not therapeutic. Mean axillary temperature was slightly higher for slide-positive (38·9°C) than slide-negative (38·4°C) patients and the difference was statistically significant ($P = 0{\cdot}001$).

Malaria transmission parameters

We conducted a total of 72 resting mosquito captures and 875 house-sprayings from December 1997 through July 1998 (Table 2). The number of adult, female anophelines captured was 287, of which 258 (90·0%) were identified as *An. gambiae s.l.* Other anophelines captured included *An. implexus* ($n = 11$), *An. coustani* ($n = 2$), *An. kingi* ($n = 2$) and a few too damaged for identification ($n = 14$). In addition, 1305 culicines were captured. IRD was low throughout the observation period; in the 2 months prior to the onset of the epidemic, the IRD averaged 0·40/house and peaked at 2·08/house in February, declining thereafter to negligible levels (Fig. 3).

The number of *An. gambiae s.l.* positive for *P. falciparum* sporozoites was 14 (6·1%) of 230 tested; none of the other *Anopheles* species tested ($n = 27$) was positive for sporozoites. The sporozoite rate peaked in February with 8·1% of mosquitoes positive; after March, no sporozoite-positive mosquito was found. Human blood was detected in 127 (66·1%) of 192 *An. gambiae s.l.* with partially digested or undigested blood meals. Only 4 (9·3%) of 43 mosquitoes with fully digested blood meals tested positive for human blood; no other anopheline tested ($n = 21$) was found positive for human blood.

EIR could only be detected by our methods in January and February: multiplying the average daily inoculation rates by the number of days in each sampling period and summing over the observation period resulted in a total inoculation rate of 0·41 infectious bites per person for the period December 1997 through July 1998.

Mosquito abundance measured as *An. gambiae s.l.* IRD was positively correlated with the incidence of malaria during the following month ($r = 0{\cdot}68$, $P < 0{\cdot}05$). HBR also was related to malaria incidence at a lag of 1 month ($r = 0{\cdot}66$, $P < 0{\cdot}05$). No temporal association between either EIR or sporozoite rates and malaria incidence was detected.

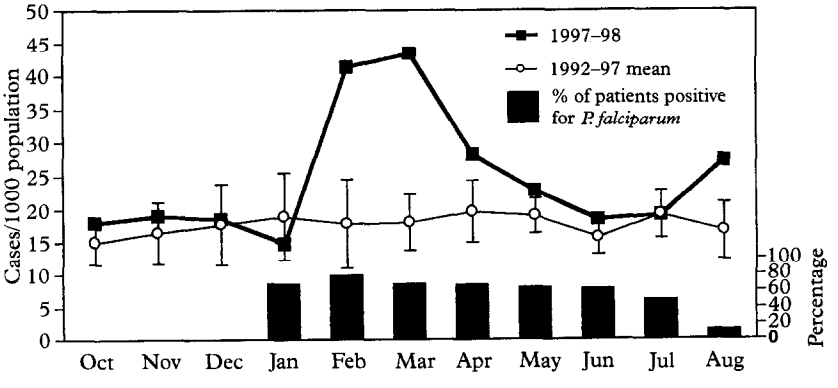

Fig. 2. Changes in the estimated incidence of malaria from October 1997 through August 1998 relative to the 5-year mean (1992–97), and the proportion of malaria patients positive for *Plasmodium* spp., Kabale, Uganda. Error bars are ±3 SE. Blood slides were not taken from October through December 1997.

Table 1. Characteristics of slide-positive malaria patients compared to slide-negative patients, Kabale, Uganda

Characteristic	Slide-positive ($n = 427$)	Slide-negative ($n = 267$)	Odds ratio (95% CI)[a]
Sex			
Male	180 (42·5%)	111 (41·9%)	1·02 (0·74–1·41)
Female	244 (57·5%)	154 (58·1%)	1·00
Age (years)			
0–5	82 (19·8%)	70 (26·9%)	0·88 (0·54–1·42)
5–10	58 (14·0%)	30 (11·5%)	1·45 (0·81–2·61)
10–15	61 (14·7%)	22 (8·5%)	2·08 (1·11–3·91)
15–30	130 (31·3%)	75 (28·8%)	1·30 (0·82–2·05)
30+	84 (20·2%)	63 (24·2%)	1·00
Slept away from home in the previous month			
Yes	42 (9·8%)	18 (6·7%)	1·51 (0·82–2·79)
No	385 (90·2%)	249 (93·3%)	1·00
Took chloroquine within the last 5 days			
Yes	123 (28·8%)	50 (18·7%)	1·76 (1·19–2·59)
No	304 (71·2%)	217 (81·3%)	1·00

[a]95% confidence interval.
The full information was not available for all patients.

Weather–transmission associations

Rainfall during the 4 months (October 1997–January 1998) before the onset of the epidemic was unusually heavy, ranging from 156 to 184 mm per month (Fig. 3). This represented more than 374 mm above normal for this period (Fig. 4). Maximum temperatures averaged around 24°C with minimum temperatures approximately 13°C and average daily temperatures between 16·8 and 19·6°C (Fig. 3). Minimum temperatures from mid-October through mid-May were 1·0–2·5°C higher than normal (Fig. 4). Saturation deficit, the difference between saturation and ambient vapour pressure, averaged approximately 0·4, with an observable increase in the deficit from May through July (Fig. 3).

Estimated incidence of malaria was not related to any weather variable at any lag period. IRD, however, was positively correlated with rainfall lagged by 1 month from December 1997 through April 1998 ($r = 0·75$, $P < 0·05$) although after April there was no significant, positive correlation ($r = -0·35$, $P > 0·05$). Similarly, no correlation between IRD and minimum temperature, maximum temperature, or saturation deficit at any lag period was found.

Rainfall anomaly was associated with IRD but not with malaria incidence. Rainfall anomaly was positively correlated with IRD 1 month later ($r = 0·55$, $P < 0·05$) throughout the entire study period.

Discussion

This study appears to be the first published report to demonstrate an association between weather variables and entomological transmission parameters during an African highland malaria epidemic. Our data suggest that this epidemic was precipitated by excesssive rainfall from mid-October 1997 through January 1998 which has been attributed to a strong El Niño event (World Meteorological Organization, 1997). KILIAN *et al.* (1999) found a similar association between rainfall and malaria case reports in a site to the north of our study area. In East Africa, El Niño, the warming phase of the Southern Oscillation, typically produces heavier than normal rainfall and higher temperatures from December through March (ROPELEWSKI & HALPERT, 1987). Through retrospective analyses, El Niño events have recently been associated with malaria epidemics in the Indian subcontinent and Latin America (BOUMA & VAN DER KAAY, 1996; BOUMA & DYE, 1997; BOUMA *et al.*, 1997). However, factors other than El Niño-related weather extremes undoubtedly impact on malaria risks in the highlands of south-western Uganda, as an earlier epidemic in Kabale and neighbouring districts in 1994 occurred in June and July after heavy rains in April and May (MOUCHET *et al.*, 1998), a period when El Niño does not affect precipitation (ROPELEWSKI & HALPERT, 1987; KILADIS & DIAZ, 1989).

In the present study, excessive rainfall was temporally associated with vector density while actual total rainfall was correlated only during the initial months of the epidemic. Simple total rainfall may not reflect adequately the amount of pooled water available for mosquito larval habitat. Similarly in Kenya, raw precipitation values were less predictive of entomological transmission parameters than a soil moisture model of surface-water availability which incorporated weather variables, landscape and soil features (PATZ *et al.*, 1998). Higher than normal rainfall anomaly may be more indicative of the extent of larval habitat than actual rainfall.

It is well established that temperature modifies mosquito development rates, biting rates and the extrinsic incubation period of malaria parasites within their mosquito host (MOLINEAUX, 1988). Additionally, malaria incidence in the highlands of Rwanda has been shown to be sensitive to minimum temperature (LOEVINSOHN, 1994). In our study, no statistically significant association was found between actual mean bimonthly minimum or maximum temperatures and malaria incidence or transmission parameters including IRD, HBR and sporozoite rates. Nevertheless, temperature may have played a role in this epidemic. Minimum bimonthly temperatures from mid-October 1997 through mid-May 1998 ranged from 1·0 to 2·5°C above normal, consistent with the regional effects of El Niño (KILADIS & DIAZ, 1989) although variation in actual temperature during this period was slight. Higher than normal minimum temperatures may have accelerated larval development, parasite development or biting rates and thus contributed to the increase in transmission, but multi-year data on mosquito abundance and EIR might be needed to detect any effect of this variable on malaria transmission.

Our prospective investigation demonstrated that in a human population with little immunity, malaria epidemics can suddenly appear despite very low inoculation rates. Other studies have shown a positive linear association between malaria incidence and EIRs less than 1 infectious bite per person per night (BEIER *et al.*, 1994; CHARLWOOD *et al.*, 1998). Our results are consistent with these reports as the 3 sampling periods in which an EIR greater than zero was detected immediately pre-

Table 2. Summary of entomological transmission parameters by sampling period, December 1997 through July 1998, Kabale, Uganda

	December		January		February		March		April		May		June		July	
	First period	Second period	First period	Second period	First period	Second period	First period	Second period	First period	Second period	First period	Second period	First period	Second period	First period	Second period
Sampled houses (*n*)	42	30	53	61	59	60	63	56	63	59	60	69	70	66	66	70
An. gambiae s.l. (*n*)	4	8	31	38	110	15	23	10	6	1	0	3	1	4	3	1
Other anophelines (*n*)	0	0	1	2	1	0	1	6	1	1	2	2	2	3	3	3
IRD[a]	0·08	0·27	0·61	0·64	2·08	0·37	0·42	0·17	0·08	0·02	0	0·05	0·01	0·06	0·05	0·02
Sporozoite rate[b]	0/4	1/8	1/27	2/35	8/99	0/14	0/17	2/10	0/6	0/1	0/0	0/2	0/1	0/3	0/3	0/0
HBI[c]	2/3	3/3	12/20	16/33	66/85	10/12	9/15	5/8	2/4	0/0	0/0	2/2	0/1	0/3	3/3	0/0
HBR (10^{-2})[d]	0·40	3·13	8·56	3·64	19·60	3·74	3·74	1·63	0·49	0	0	0·69	0	0	1·62	0
EIR (10^{-2})[e]	0	0	0·21	0·27	2·44	0	0	0	0	0	0	0	0	0	0	0

[a]Indoor resting density (IRD) in each period is the mean of 16 village means of the number of *An. gambiae s.l.* per house.
[b]Indicates the number positive/number tested for *An. gambiae s.l.* only.
[c]Human blood index (HBI) indicates the number positive/number tested in each period for *An. gambiae s.l.* only, excluding those mosquitoes with fully digested blood meals.
[d]Human biting rate (HBR) is calculated from the number of *An. gambiae s.l.* testing positive for human blood as the number with undigested blood meals plus half the number with partially digested blood meals divided by the number of household residents. The value in each period is the mean of 16 village means.
[e]Entomological inoculation rate (EIR) is calculated as the product of the sporozoite rate and HBR for each household. The value in each period is an average daily rate calculated as the mean of 16 village means.

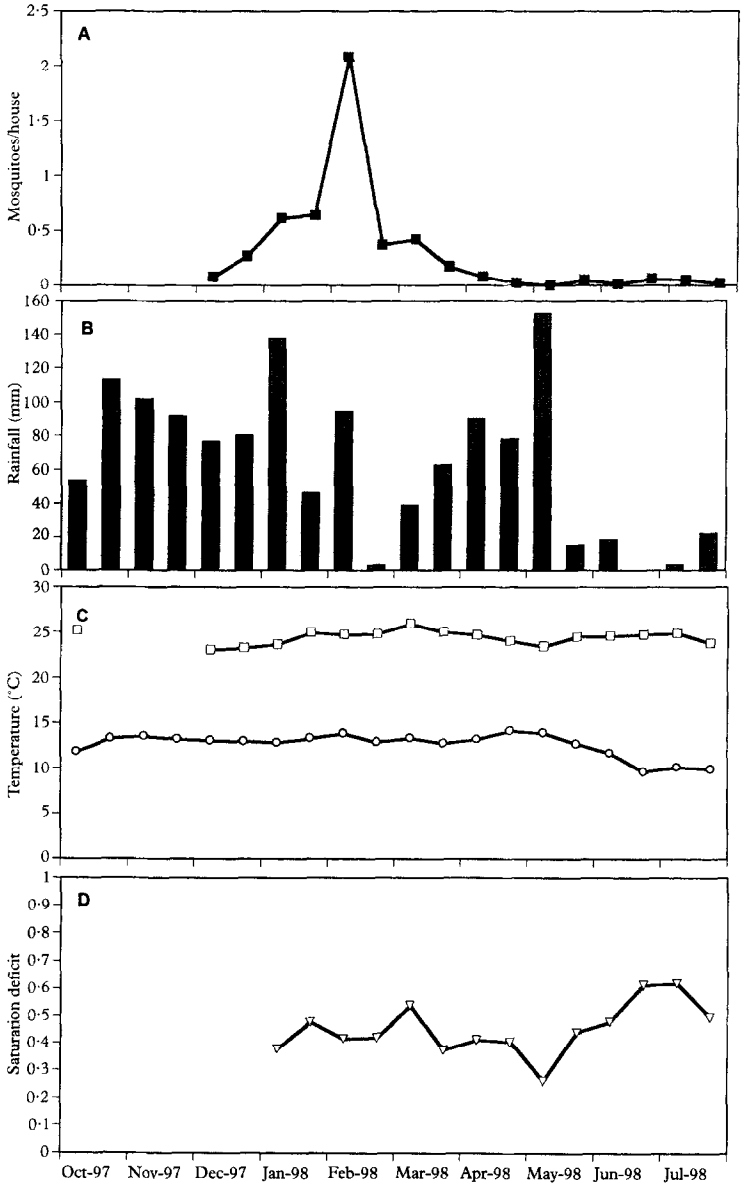

Fig. 3. Variation in (A) mean indoor resting density, (B) total rainfall, (C) mean maximum and minimum temperature, and (D) mean saturation deficit for 2-week periods from October 1997 through July 1998, Kabale, Uganda. Maximum temperatures were missing from mid-October through November 1997. IRD was not measured in October and November and saturation deficit was not measured prior to January 1998.

ceded the interval of increased malaria incidence. Total EIR over the study period was estimated to be 0·41 infectious bites per person, which suggests up to 41 of every 100 people may have received an infectious bite, assuming homogeneity of transmission. Even though some infectious bites will not result in infection, it is clear that an outbreak of malaria would result if the proportion of the human population receiving an infectious bite increased from none in December 1997 to near 41% by mid-February 1998.

An epidemic arising from such few vectors and low inoculation rates may seem surprising but is not unprecedented. HEISCH & HARPER (1949), who obtained prospective entomological data during a highland malaria epidemic in Kenya in which *An. funestus* was implicated, found an overall sporozoite rate of 2·5% and an IRD in the month preceding the epidemic of only 1·73 mosquitoes per house, values which are very similar to those reported in this study. MACDONALD (1953) used mathematical models to demonstrate that a 5·3-fold increase in vector density could precipitate an epidemic that would peak ~80 days later. This prediction is also consistent with our results: the increase in IRD from December 1997 to January 1998 showed a similar magnitude (3·7 times greater in January) and the peak malaria incidence occurred in March, i.e., 60–90 days after the initial increase in vectors.

Speculation abounds that the frequency of highland malaria epidemics may increase as global temperatures rise, exposing vulnerable populations to the parasite with calamitous consequences. However, unusual weather events over shorter periods of time may be more proximate causes of outbreaks, particularly where transmission is unstable. Our results suggest that, with careful analysis of local conditions, many of these outbreaks can be predicted, allowing proactive responses. To achieve

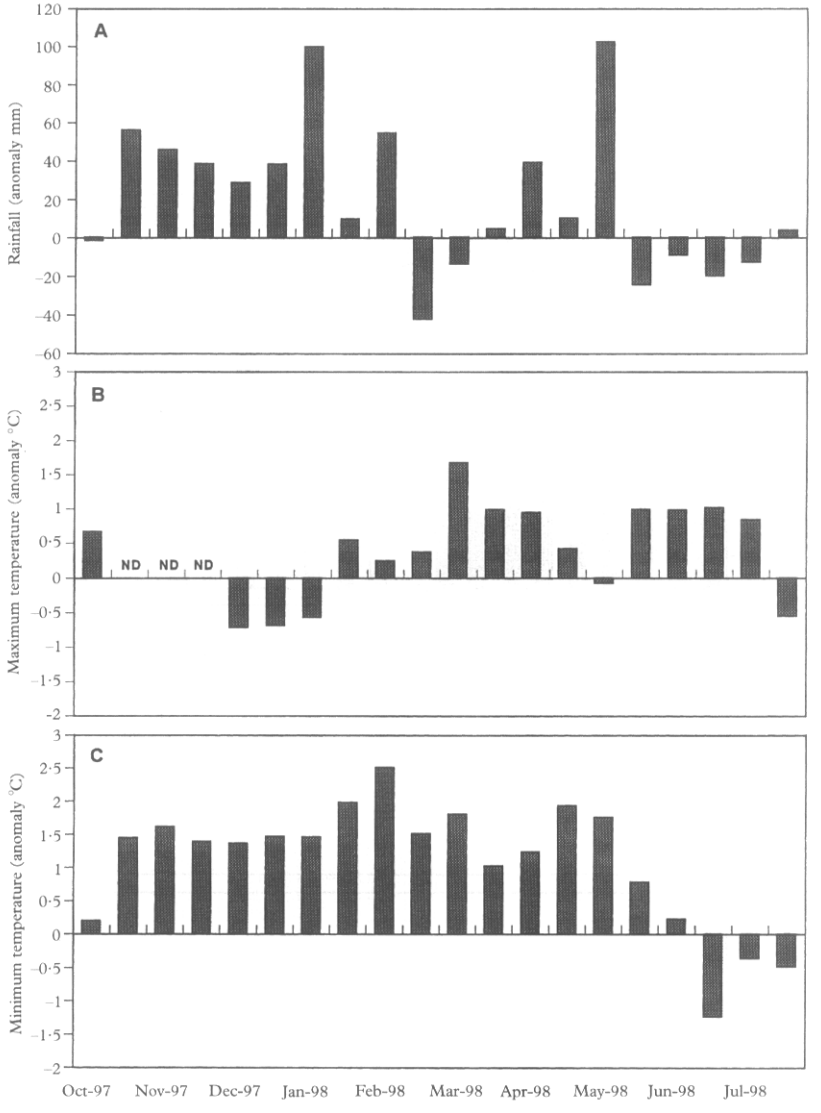

Fig. 4. Two-week period differences between (A) total rainfall, (B) average daily maximum, and (C) average daily minimum temperatures and the bimonthly historical mean, October 1997 through July 1998, Kabale, Uganda. Rainfall anomaly is measured against the 1960–97 historical mean; temperature anomalies are calculated using the 1992–97 average.
ND, no data.

such goals, however, extended time series of entomological transmission parameters are needed for analysis of the role precipitation and temperature variation plays in initiating epidemics.

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Announcement

2000 Award in honour of Fred L. Soper (1893–1976) for publications in the field of Inter-American Health

This is an announcement and call for submission of nominations for the 2000 award in honour of Fred L. Soper, former Director of the Pan American Health Organization (the World Health Organization Regional Office for the Americas) from 1947 to 1958.

The Award is presented annually to the author or authors of an original scientific contribution comprising new information on, or new insights into, the broad field of public health, with special relevance to Latin America or the Caribbean or both. This may consist of a report, an analysis of new data, experimental or observational, or a new approach to analysing available data. Preference is given to studies involving more than one discipline and to papers related to infectious diseases, a life-long concern of Dr Soper.

Only papers already published in scientific journals listed in the Index Medicus or in the official journals of the Pan American Health Organization are eligible for consideration. Furthermore, the award is limited to contributions by authors whose principal affiliation is with teaching, research or service institutions located in the countries of Latin America and the Caribbean (including the Centers of the Pan American Health Organization).

The Award consists of a certificate and a prize of US$ 1000.

Papers submitted by or on behalf of their authors may be considered for the Fred L. Soper Award. For the 2000 Award, only papers published during the calendar year 1999 will be considered; all submissions must be received by 31 March 2000 at the following address: Fred L. Soper Award Program, PAHEF, 525 23rd Street NW, Washington, DC 20037, USA.