EPIDEMIC MALARIA IN THE HIGHLANDS OF PAPUA NEW GUINEA

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Abstract. As part of a larger study into the epidemiology of malaria in the highlands of Papua New Guinea, outbreak investigations were carried out at the end of the 2002 rainy season in 11 villages situated between 1,400 and 1,700 meters above sea level that had reported epidemics. Locations and timing of these epidemics corresponded largely to those reported in the pre-control era of the 1960s and 1970s. On average, 28.8% (range = 10.3–63.2%) of people in each of the 11 villages were found to be infected with malaria. Plasmodium falciparum accounted for 59% of all identified infections and P. vivax for 34%. The majority (53%) of infections were symptomatic. Although symptomatic infections were most common in children 2–9 years of age (36%), even in adults a prevalence of 20% was observed. A comparison with earlier non-epidemic data in three of the villages without easy access to health care showed markedly increased levels of morbidity, with 6–10-fold increases in parasite prevalence, a 3-fold increase in both measured and reported fevers, and a 12-fold increase in enlarged spleens. The average hemoglobin levels were reduced by 2.3–3.5 g/dL, with a concurrent increase in moderate to severe anemia (hemoglobin level < 7.5 g/dL) from 0.0–3.3% to 3.8–18.4%. These massive increases in morbidity have devastating impact on the affected communities and highlight that malaria epidemics are a serious and increasing public health problem in the highlands of Papua New Guinea.

INTRODUCTION

Traditionally, most tropical highlands have little or no malaria. However, the steady increase in malaria in the first half of 20th century, which was related primarily to increased movement of people between highlands and malarious lowlands,1 led to devastating epidemics in many highland areas.2 The consequent introduction of vector control in many highland areas reduced malaria transmission and protected people from such epidemics. Nevertheless, in the last 20 years malaria epidemics in highland areas have again become an urgent public health problem in many parts of the tropics, with recent major outbreaks occurring in the highlands of east Africa,3–6 Madagascar,7 and West Papua/Indonesia8 that affected large populations and had high levels of morbidity and mortality.

While short-term climatic variation9 and extreme weather events, such as El Niño-associated rain excess4,9 or drought,8 may trigger individual epidemics, it has so far proved difficult to link the increase in epidemics in the east African highlands to global climate change.10 Economic, social, and political factors such as failing health systems and control,11,12 an increase in drug resistance,3,13 migration, increased population densities and settlements,1,12 or changes in land use and deforestation associated with local warming and increase vector breeding,1,5,12,13 are likely to be more important contributors.

Prior to European contact, malaria in the highlands of Papua New Guinea was probably restricted to either lowland areas in steep inter-montane valleys, swampy valley bottoms, or communities with gardens in, or trading links to, the highly malarious lowlands.15 The opening up of the highlands by the Australian administration, especially after completion of a road link, changed this dynamic. Economic development, such as the establishment of coffee and tea plantations in drained areas, and mass recruitment of highland labor for work on lowlands plantations, resulted in the introduction of malaria into many formerly malaria-free areas and increased transmission in areas where it was already present.15

The first series of epidemics observed in the 1950s in plan-
tion lives at altitudes of 1,600–2,000 meters above sea level and have to be considered at risk of epidemics. To devise rational measures for control and possibly prevention of such epidemics, a better knowledge of the patterns underlying these local epidemics is needed. In this paper, we report the results of surveys conducted during a series of epidemics that occurred between March and June 2002 in the Eastern Highlands and Simbu provinces of Papua New Guinea.

**METHODOLOGY**

As part of a larger study into the epidemiology of malaria in the highlands of Papua New Guinea,21–23 cross-sectional malaria surveys were carried out between March and June 2002 in villages that reported being affected by epidemics directly to the malaria field team at the Papua New Guinea Institute of Medical Research. A total of 1,216 people were investigated during cross-sectional surveys carried out in 11 villages (Figure 1). Individual informed consent was obtained from all participants or in case of children their guardians prior to enrollment into the study. Five villages were surveyed during a reported epidemic in South Fore in the Eastern Highlands Province (EHP). An additional three Eastern Highlands villages were surveyed in May in the Lufa Valley. Three individual epidemic surveys were carried out in Simbu Province, Gena in the Kerowagi district, Maimag in the Chuave district, and Sul in the Gumini district. The villages are all situated at altitudes between 1,460 and 1,720 meters above sea level (interquartile range = 1,530–1,640 meters). A more detailed description of villages is reported elsewhere. In all villages, mass drug administration with chloroquine and sulfadoxine-pyrimethamine was carried out to control the epidemics and local health authorities were advised to conduct vector control activities. This study was reviewed and approved by the Papua New Guinea Medical Research Advisory Council (approval number 00.26).

In each village, households with a combined population of approximately more than 200 people were selected. From each selected household, every member who could be reached during their stay in the village was included in the survey. If the village had less than 200 inhabitants, sampling...
of every resident was attempted. This approach allowed us to sample approximately 80% of all selected household members. From each individual, thick and thin blood films were prepared, the spleen was palpated in the lying position, and an axillary temperature taken. In all but two villages (Paipindi and Palototoi), hemoglobin levels were measured using the Hemacue system (HemoCue AB, Angelholm, Sweden). A short questionnaire on current symptoms, past malaria episodes, and treatment was administered to each participant or their guardian.

One hundred fields on Giemsa-stained blood films were examined by microscopy with an oil-immersion lens before being declared negative. The parasite species in positive films were identified and densities were recorded as the number of parasites per 200 white blood cells. Densities were converted to the number of parasites per microliter of blood assuming 8,000 white blood cells/μL.

Statistical analyses were done using STATA 7.0 (Stata Corp., College Station, TX) statistical analyses software. Chi-square tests and logistic regression analyses were used for categorical variables. Continuous variables were investigated using the Student’s t-test, and analyses of variance. Regression splines were used to adjust hemoglobin values for age and sex effects.

RESULTS

A total of 1,216 people were investigated during cross-sectional surveys carried out in 11 villages (Figure 1) in five separate epidemics. The epidemic in the South Fore, EHP reportedly started in January 2002 (i.e., early in the rainy season), while an increase in presumptive malaria cases in Lufa, EHP and Maimagu, Simbu was first observed February and March. The other two epidemics in Simbu were only noted while carrying out a series of malaria surveys in May. The villages surveyed were situated at altitudes between 1,460 and 1,720 meters above sea level (interquartile range = 1,530–1,640 meters).

On average, 28.8% (range = 10.3–63.2%, Figure 1) of the people in each of the 11 villages surveyed were found to be slide positive for malaria the time of the surveys. An additional 12.2% of the villagers reported a successful antimalarial treatment, as confirmed by negative blood slide, in the two weeks prior to the surveys. Rates of enlarged spleens were relatively low (average = 12.6%, range = 2.1–25.9%), but measured fevers (axillary temperature > 37.5°C; average = 5.4%, range = 0.0–12.3%) and reports of fever illness in the three days preceding the survey (average = 33.3%, range = 16.7–57.9%) were common.

Overall, P. falciparum was the most common malaria species, accounting for 58.9% of all identified infections, followed by P. vivax with 34.2% (Table 1). Both P. malariae and P. ovale were rare. However, there were significant differences in species composition among villages, with the proportion of infection due to P. falciparum ranging from 23.9% to 91.7% (P < 0.001, by Fisher exact test). Plasmodium vivax predominated in only two villages (Auno = 63.0% and Maimagu = 65.0%) with a health center in their vicinity. Detailed village summary data are reported elsewhere by Mueller and others. Most infections were either scarce (39.2% < 500/μL) or light (34.2% 500–2,500/μL), with only 15.8% classified as moderate (2,500–10,000/μL) and 10.8% as heavy (>10,000/μL). Plasmodium falciparum and mixed infections had significantly higher geometric mean densities (F2,311 = 3.37, P = 0.01) (Table 1).

Most infections were symptomatic (52.6% with fever in last three days) irrespective of type of malarial infections (P = 0.9, by Fisher’s exact test). Symptomatic infections were common in all age groups: 80% of infants (<2 years old) and 67% of toddlers (2–4 years old) with malarial infections reported symptoms of febrile illness. Symptoms were less frequent (38%) in older children (5–9 years old), adolescents (43%) and adults (57%, χ² = 17.2, degrees of freedom [df] = 4, P = 0.002). However, the prevalence of acute symptoms was strongly dependent on the density of infection: 83.8% of the infections with moderate or high densities (>2,500/μL) were associated with a reported fever over a three-day period prior to the surveys, but only 56.1% of scarce (<500/μL) and 47.2% of light (500–2,499/μL, χ² = 46.5, df = 2, P < 0.001) infections showed this association. Similarly, in comparison with uninfected people, age- and sex-adjusted mean hemoglobin levels were decreased by 0.8 g/dL, 1.2 g/dL, and 1.7 g/dL for scarce, light, and moderate/high infections, respectively. There were no differences in prevalence of febrile illness among different types of infections (χ² = 0.9, df = 2, P = 0.9), but there was a significantly bigger decrease in hemoglobin levels in infections with P. falciparum (1.4 g/dL, 95% confidence interval [CI] = 1.0–1.8) and mixed infections (1.5 g/dL, 95% CI = 0.7–2.3) compared other infections (0.4 g/dL, 95% CI = 0.0–0.9, F2,251 = 6.25, P = 0.002).

In comparison with surveys conducted in highland villages in all seasons (detailed data are presented elsewhere22,23), villages with reported epidemics had significantly higher parasite prevalences (Table 2) with infections more frequently due to P. falciparum. Villagers from epidemic-affected areas also presented significantly (P < 0.001) more often with fever, lower hemoglobin levels, and were more frequently anemic (hemoglobin level < 7.5 g/dL). There were no differences in demographic characteristics between epidemic and non-epidemic villages.

The prevalence of infections varied significantly among age groups (χ² = 31.1, df = 4, P < 0.001). Infections were most common in children, with 36.9% and 36.2% in those 2–4 and 5–9 years old, respectively, followed by adolescents (10–20 years old = 29.6%), infants (<2 years old = 25.9%), and adults (19.9%). Infections in children <10 years old had significantly higher densities than those in adolescents and adults (1.325/μL, 95% CI = 982–1,788/μL versus 656/μL, 95% CI = 500–863/μL, P < 0.001). However, symptomatic

| Table 1 |

| Density | Geometric mean | 95% CI |

| P. falciparum (P. f.) | 171 | 54.1 | 968 (710, 1,320) |
| P. vivax (P. v.) | 96 | 30.4 | 600 (431, 835) |
| P. malariae (P. m.) | 9 | 2.9 | 692 (289, 1,696) |
| P. ovale | 3 | 1.0 | 110 (11, 1,083) |
| Mixed infections | 37 | 11.7 | 1,694 (1,123, 2,555) |
| P. f. + P. v. | 36 | 1.725 | (1,131, 2,629) |
| P. f. + P. m. | 1 | 880 |

* CI = confidence interval; P. = Plasmodium.
infections were as common in adolescents or adults (32.9%) as in children < 10 years (31.3%). Similarly, there were no significant differences in the decrease in hemoglobin level with concurrent parasitemia (1.2 g/dL versus 1.1 g/dL < F_{2,105} = 0.01, P > 0.5).

A comparison with non-epidemic surveys done nine months earlier\(^2\) showed markedly increased levels of morbidity in the three villages (Anitoi, Kwale, and Ilasa) without easy access to health care. A 6–10-fold increase in parasite prevalence was paralleled by a three-fold increase in both measured and reported fevers, while enlarged spleens were 12 times more common (Table 2). The average hemoglobin levels were reduced by 2.3–3.5 g/dL, with a concurrent increase in moderate to severe anemia (hemoglobin level < 7.5 g/dL) from 0.0–3.3% to 3.8–18.4%. All villages also reported significant increases in both the occurrence of malaria illness and, with the exception of Anitoi, antimalarial treatment in the two weeks prior the survey (Table 3).

In Auno, which is less than a 15-minute walk from a health center, the increase in morbidity was less pronounced, despite a similar increase in prevalence (Table 3). While there was a similar increase in measured and reported fevers, hemoglobin levels decreased by only 1.1 g/dL and the increase in enlarged spleens was not significant (P = 0.14, by Fisher’s exact test). In striking contrast to the other villages, there was no shift from a predominance of *P. vivax* to one of *P. falciparum* (P = 0.6; Table 3) during the epidemic in Auno, and there was no significant increase in reports of malaria illness (P = 0.5) or antimalarial treatment (P = 0.6).

No differences in geometric mean density of infections prior and during epidemics was observed (635 versus 882, t = 1.0, df = 201, P = 0.3.). However, infections during epidemics were significantly more often symptomatic: 60.8% (95% CI = 52.6–68.6%) of the infections were associated with reported fever in the last three days compared with 32.6% (95% CI = 19.1–45.8%) in the non-epidemic surveys.

**DISCUSSION**

To differentiate properly between epidemic outbreaks and seasonal differences in malaria transmission, detailed longi-

### Table 2

<table>
<thead>
<tr>
<th>Village</th>
<th>No. surveyed</th>
<th>Epidemic</th>
<th>Non-epidemic surveys</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mar-Jun</td>
<td>Nov-Feb</td>
<td>Mar-Jun</td>
</tr>
<tr>
<td></td>
<td>Late wet-early dry</td>
<td>Early wet</td>
<td>Late-early dry</td>
</tr>
<tr>
<td>No. surveyed</td>
<td>1,216</td>
<td>2,535</td>
<td>1,292</td>
</tr>
<tr>
<td>Slide positive (%)</td>
<td>26.0</td>
<td>2.1</td>
<td>3.2</td>
</tr>
<tr>
<td>P. falciparum (%)</td>
<td>58.9</td>
<td>36.5</td>
<td>34.2</td>
</tr>
<tr>
<td>Enlarged spleen (%)</td>
<td>11.5</td>
<td>0.4</td>
<td>0.6</td>
</tr>
<tr>
<td>Temperature &gt; 37.5°C (%)</td>
<td>5.4</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Mean Hb (g/dL)</td>
<td>11.9</td>
<td>13.4</td>
<td>13.0</td>
</tr>
<tr>
<td>Hb &lt; 7.5 g/dL (%)</td>
<td>3.7</td>
<td>0.1</td>
<td>0.7</td>
</tr>
<tr>
<td>Reported malaria episode (%)‡</td>
<td>38.2</td>
<td>12.4</td>
<td>16.9</td>
</tr>
</tbody>
</table>

* Detailed data of individual surveys is reported in Mueller and others.\(^{2,23}\) P = *Plasmodium*; Hb = hemoglobin.
† Proportion of all species identified.
‡ During two weeks prior to the survey, the word malaria in the local perception is often assigned to any non-respiratory febrile illness.

### Table 3

<table>
<thead>
<tr>
<th>Village</th>
<th>No. surveyed</th>
<th>Mean age (years)</th>
<th>Male (%)</th>
<th>Slide positive (%)</th>
<th>P. falciparum (%)</th>
<th>Mixed infections (%)</th>
<th>Enlarged spleen (%)</th>
<th>Temperature &gt; 37.5°C (%)</th>
<th>Reported fever in last 3 days (%)</th>
<th>Mean Hb (g/dL)</th>
<th>Hb &lt; 7.5 g/dL (%)</th>
<th>Reported malaria episode (%)</th>
<th>Reported antimalarial treatment (%)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anitoi Lufa/EHP</td>
<td>Prior (124)</td>
<td>23.9</td>
<td>53.9</td>
<td>10.1</td>
<td>33.3</td>
<td>–</td>
<td>4.0</td>
<td>18.4</td>
<td>12.6</td>
<td>3.2 (2.5, 4.0)</td>
<td>3.3</td>
<td>17.7</td>
<td>11.3</td>
</tr>
<tr>
<td>Kwale Lufa/EHP</td>
<td>Epidemic (38)</td>
<td>25.4†</td>
<td>50.0†</td>
<td>63.2</td>
<td>67.9</td>
<td>33.3</td>
<td>18.9</td>
<td>57.9</td>
<td>9.3</td>
<td>2.3 (1.7, 2.9)</td>
<td>18.4</td>
<td>15.3</td>
<td>15.8</td>
</tr>
<tr>
<td>Ilasa South Fore/EHP</td>
<td>Prior (138)</td>
<td>22.7</td>
<td>50.0</td>
<td>4.4</td>
<td>0.0</td>
<td>–</td>
<td>–</td>
<td>13.1</td>
<td>13.5</td>
<td>2.5 (2.2, 2.9)</td>
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<td>17.7</td>
<td>5.1</td>
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<td>Anitoi Lufa/EHP</td>
<td>Epidemic (105)</td>
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<td>47.6‡</td>
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<td>48.3</td>
<td>16.0</td>
<td>1.5</td>
<td>13.1</td>
<td>11.2</td>
<td>13.6</td>
<td>0.0</td>
<td>3.8</td>
<td>11.4‡</td>
</tr>
<tr>
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<td>Epidemic (176)</td>
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<td>50.3</td>
<td>2.3</td>
<td>0.0</td>
<td>–</td>
<td>0.0</td>
<td>18.2</td>
<td>13.6</td>
<td>2.5 (2.2, 2.9)</td>
<td>0.0</td>
<td>0.0</td>
<td>7.4</td>
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<tr>
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<td>Epidemic (179)</td>
<td>19.0†</td>
<td>46.1‡</td>
<td>21.2</td>
<td>67.5</td>
<td>–</td>
<td>6.0</td>
<td>51.2</td>
<td>10.8</td>
<td>29.0</td>
<td>6.0</td>
<td>23.9§</td>
<td>4.6</td>
</tr>
<tr>
<td>Auno Lufa/EHP</td>
<td>Prior (128)</td>
<td>25.7</td>
<td>52.6</td>
<td>6.0</td>
<td>67.5</td>
<td>–</td>
<td>1.7</td>
<td>15.6</td>
<td>13.6</td>
<td>25.7</td>
<td>1.7</td>
<td>23.9§</td>
<td>4.6</td>
</tr>
<tr>
<td>Auno Lufa/EHP</td>
<td>Epidemic (128)</td>
<td>24.4†</td>
<td>41.9‡</td>
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<td>1.7</td>
<td>15.6</td>
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<td>25.7</td>
<td>1.7</td>
<td>23.9§</td>
<td>4.6</td>
</tr>
</tbody>
</table>

* All differences are significantly different (P < 0.05) except where indicated. EHP = Eastern Highlands Province; P = *Plasmodium*; Hb = hemoglobin.
† P > 0.1.
‡ P < 0.1.
§ During two weeks prior to the survey, the world malaria in the local perception is often assigned to any non-respiratory febrile illness.
tudinal data as well as clear definitions of epidemics are needed. However, the remoteness of the areas affected by malaria in the highlands of Papua New Guinea makes longitudinal studies unfeasible. Available records from health facilities in both Eastern Highlands and Simbu provinces do show seasonal increases in admissions with malaria that correspond with late rainy and early dry season and parasite prevalence in areas without reported outbreaks exhibit corresponding fluctuations (Table 2). However, the massive increases both in prevalence of infections and levels of morbidity (Tables 2 and 3) and the limited geographic and temporal extent of reported outbreaks (Figure 1) that were subject of this study indicated that intensity of transmission and levels of morbidity are exceeding the usual seasonal increases in malaria in highlands areas of Papua New Guinea.

All the epidemics investigated in this report, as well as most other epidemics reported in the highlands of Papua New Guinea in recent years, occurred between February and June, i.e., towards the end of the wet season and the start of the dry season, in villages situated between 1,400 and 1,700 meters above seal level. Locations and times of epidemics correspond largely to those observed in the 1950s and 1960s. The current situation in Eastern Highlands and Simbu province is therefore similar to that observed in Enga in 1980, with P. vivax dominant even during times of peak transmission, despite the increasing incidence of P. falciparum at that time. The current situation in Eastern Highlands and Simbu province is therefore similar to that observed in Enga in 1980, with P. vivax dominant even during non-epidemic periods. The observed shift from P. vivax to P. falciparum is likely to have significantly increased the severity of epidemics, as indicated by significantly higher parasite densities and more pronounced decrease in hemoglobin levels associated with infections with P. falciparum.

Although children were at higher risk of malarial infections and had higher parasite densities, this is unlikely to reflect significant levels immunity in these highland populations. In stark contrast to the situation in highly endemic areas, most infections were symptomatic and febrile symptoms occurred at significantly lower parasite densities irrespective of age. As a result, in epidemic villages febrile illness was a common feature in all age groups and the prevalence of measured fevers (axillary temperature >37.5°C) was more the five times that found in the Wosera, a highly endemic lowland community in Papua New Guinea (5.4 versus 1.0%; Genton B and others, unpublished data). The higher prevalence of infections in children is therefore more likely to be related to differences in infection risk or health-seeking behavior than significant levels of acquired immunity in older age groups.

The comparison of morbidity prior to and during the epidemics further highlights the devastating effect such epidemics have on local communities. The significant increase in spleen rates decrease in and hemoglobin levels of up to 3 g/dL are indications that these epidemics have been persisting for some time, perhaps several months, and that most people, even those not infected at the time of the surveys, had been exposed to malarial infections at some time in the recent past. In one village, Anitoi, the level of morbidity reached such dimensions that the inhabitants decided to abandon the village and rebuild the houses further up the hill. At the time of the survey only 38, two-thirds of whom had malarial infections, of the 130 original inhabitants were still residing in the village. Subsequent to the survey, the village was completely abandoned.

Easy access to treatment may prevent some of the severe morbidity during epidemics. In Auno, which is adjacent to a well-functioning health center and where antimalarial drugs are readily prescribed for any presumptive fever even outside epidemics (Table 3), morbidity levels were significantly lower than in the other two villages in the Lufa epidemic. Nevertheless, prevalence rates in Auno remained high, but P. vivax instead of P. falciparum was the clearly dominant parasite. It is very likely that many P. vivax cases are relapses of earlier infection rather than new infections, while treatment efficiently reduced P. falciparum infections. Despite reduced morbidity, the high prevalence of P. vivax indicates that presumptive treatment with chloroquine and sulfadoxine-pyrimethamine alone may not be sufficient to control epidemic outbreaks of malaria in the highlands of Papua New Guinea. Effective control needs to combine effective case management, mass drug administration, and vector control measures. In light of increasing levels of resistance to commonly used antimalarials, the use of artemisinin combination therapy for the control of epidemics should be considered. Besides being highly effective in clearing blood stage infections, artemisinin drugs have the added advantage of reducing gametocytemia and thus further reduce the risk of transmission.

Many factors may conspire to create the observed increase in frequency of malaria epidemics in the highlands of Papua New Guinea. Most soils in the highlands are fast draining, leaving only few breeding sites during the drier parts of the year. However, during the rainy season, water tables rise and many highland rivers flood. As rivers recede towards the end of the rainy season, standing pools of water are common, leading to increases in mosquito numbers and malaria transmission consequently increases dramatically during that time of the year. Traditionally, many lower-lying valley bottoms were not settled because they were known to be disease infested. However, pacification, drainage of swamps, establishment of coffee and tea plantations, and subsequent vector control after World War II led to the establishment of many settlements in these areas, and areas such as the Waghi Valley now have some of the highest population densities in all of Papua New Guinea. After the collapse of vector control, these communities are now at risk of epidemic malaria.

The seasonal nature of epidemic risk may be reinforced by local patterns of subsistence. In many inter-montane valleys, settlements are high on ridges but food and coffee gardens, as well as hunting areas, extend far down into the valleys. Harvesting of coffee or collection of marita (Pandanus sp.) at lower altitude, which both coincide with the main malaria transmission period, have been associated with malaria epidemics even in communities whose main settlements are situated at altitudes where temperature precludes malaria transmission. The shift from traditional to a modern, western-style of housing, as well as the construction of infrastructure such as road or airfields, created additional vector breeding sites and increased malaria transmission in many highlands areas. In the highlands of West Papua/Indonesia, the replacement of traditional village huts with more modern, housing coincided with an upsurge of malaria transmission. Anopheles punctatus was found to readily breed in drainage ditches and to
bite preferably in new compared with traditional style houses. Similarly, a recent study in a highlands village in Papua New Guinea found *An. punctulatus* to be the dominant vector, with biting rates significantly higher than in the 1950s.

The increase in travel to the highly malarious lowland ensures a steady importation of malaria infections into the highlands. In non-epidemic areas, more than 80% of the episodes of clinical malaria were found to be directly linked to recent traveling. Even a single introduction from outside may in certain circumstances be enough to trigger an local epidemic.

Although, as in Africa, socioeconomic effects seem to be primarily responsible for resurgence of highlands epidemics, long-term climatic trends may also play a part. In the last 40 years, there was a significant increase of 0.6–0.9°C in mean temperature (National Agricultural Research Institute, Ayura, EHP) and a shift to a longer, but less pronounced rainy season, which may prolong the length of epidemics season. If these trends continue, the most densely populated areas in the highlands at 1,600–1,800 meters above sea level are likely to see a significant increase in risk of malaria epidemics.

However, the most important reasons for the resurgence of epidemic malaria in the highlands of Papua New Guinea are to be found in the complete cessation of vector control activities in the early 1980s and the progressive failure of the curative health system in many rural areas of this country. The extent of chronic morbidity seen in some of the reported surveys is proof of delayed or inadequate response of local health authorities to these epidemics. The highly diverse geography and generally higher elevation of the highlands of Papua New Guinea prevented the kind of large-scale epidemics that devastated the highlands of Madagascar after vector control ended there.

Compared with most epidemics observed elsewhere, epidemics in Papua New Guinea generally affect only small populations in restricted geographic areas. The exception was the large malaria epidemic during 1997 El Niño that was observed throughout the New Guinea Highlands on both sides of the Papua New Guinea/Indonesia border. The dynamic of this epidemic was quite distinct from the regularly observed epidemics described earlier since it occurred at the end of a severe dry spell. The prolonged failure of rains resulted in decreases in water levels of major rivers that were usually fast flowing, leaving numerous small pools in river beds that acted as mosquito breeding sites. Moreover, due to water shortages in their villages on the ridges, people moved to the lower-lying rivers, which were the only water sources, thus concentrating the population in areas of increased mosquito breeding. Similar dry season epidemics were observed in Sri Lanka.

Malaria epidemics are once again a serious and increasing public health problem in the highlands of Papua New Guinea. Unfortunately, the current health system in this country is ill-equipped to deal even with small localized outbreaks. To protect highlands populations and the economy from the potentially devastating effect of malaria epidemics, a concerted effort is needed, to build up efficient epidemic surveillance and control systems and improve the capacities of rural health clinics to diagnose and treat malaria cases. As the experience from Madagascar proves, reintroduction of vector control by indoor residual insecticide spraying should be seriously considered, at least in easily accessible, densely populated areas with substantial risk of malaria outbreaks. Indoor residual insecticide spraying is likely to be more cost effective in preventing malaria in very low transmission settings than the distribution of insecticide-treated bed nets, as shown in a recent study from the Kenyan highlands. We continue to develop evidence-based recommendations for integrated malaria controls on a province by province basis.

Received December 17, 2003. Accepted for publication April 9, 2004.

Acknowledgments: We thank all the villages for their help and participation in the surveys. Special thanks are given to Jerome Whitfield and Gimana Poigeno for help with the field work.

Financial support: This study was supported by the World Health Organization through the Regional Office for the Western Pacific and the Global Roll Back Malaria Initiative.

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