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MALARIA IN THE TAVETA AREA OF KENYA AND TANZANIA
Part V—Transmission eight years after the spraying period
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# MALARIA IN THE TAVETA AREA OF KENYA AND TANZANIA

## PART V

## TRANSMISSION EIGHT YEARS AFTER THE SPRAYING PERIOD

*By* A. SMITH AND G. PRINGLE*

Received for publication June 12, 1967.

THIS paper is a sequel to four earlier papers on "Malaria in the Taveta area of Kenya and Tanzania". The first described the epidemiology of malaria (Smith and Draper 1959a), the second the effect of three and a half years' treatment of huts with dieldrin on transmission of malaria (Smith and Draper 1959b), the third the entomological findings in the second and third years after the last cycle of residual spraying (Smith 1962) and the fourth the entomological findings in the fourth, fifth and sixth years afterwards (Smith 1966).

Taveta area is part of a larger area in which an experiment was made between 1954 and 1959 to find out whether or not malaria transmission could be broken, in an inland area of East Africa, by treatment of houses with the residual insecticide dieldrin. Intensive studies were made on the mosquito and human populations before and during the course of residual spraying. In Taveta area the first cycle of spraying was in August 1955 and the sixth and last in December 1958. The population of *Anopheles gambiae* Giles was reduced, at the most to one-fifth of its previous size by the residual spraying, but it recovered to its previous level in the second year after the last cycle i.e. in 1960, although the existing treated houses were still toxic. The recovery was attributed to some 30 per cent of the houses in the area, built since the cessation of residual spraying, providing enough untreated domestic resting places for the longevity of *A. gambiae* to become sufficient to restore the fecundity of the population. *Anopheles funestus* Giles, which had been the principal vector in the Taveta area before residual spraying, was eradicated from the area for eight years i.e. from the latter half of 1955 to the latter half of 1963. There were a number of interesting features about the return of *A. funestus* Giles. It returned two years after all traces of residual toxicity, in the remaining treated houses, had disappeared; it appeared in November 1963 and in the following year its densities in houses surpassed those before residual spraying; finally, its habits were identical with those of its predecessor.

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A great fall in human malaria indices during the spraying period showed that there had been a complementary reduction in malaria transmission due to eradication of *A. funestus* and the great reduction in longevity of the surviving *A. gambiae*. Even in 1961, three years after the last cycle of residual spraying, the risk of malaria infection in Taveta area was still low, being in the order of one-thirtieth of that before residual spraying. One of the main factors responsible for this low rate of transmission was almost certainly the absence of *A. funestus* type. The present paper is largely concerned with an assessment of the risk of malaria infection in Taveta area in 1965 and 1966 i.e. seven and eight years after the last cycle of residual spraying and two and three years, respectively, after the return of *A. funestus* type.

The entomological techniques employed at Kibalwa were (*a*) monthly pyrethrum spray-catches in ten local houses; (*b*) daily collections from ten box-shelters (Gillies 1954); (*c*) dissection and examination of salivary glands of *A. gambiae* and *A. funestus* for sporozoite infections.

The monthly rainfall at Taveta Sisal Estate and at Kibalwa village for 1965 and 1966 is shown in Table 1. The rainfall records indicate that in 1965 there was a slightly lower rainfall at Taveta Sisal Estate than the average of 23 inches for the previous ten years and a slightly higher than average rainfall in 1966. Kibalwa Outstation received slightly less rainfall than Taveta Sisal Estate during both years. The most significant aspect of the rainfall, during the two years, from the point of view of mosquito-production, was the failure of the short rains in 1966 shown by the low records for October and November.

## ENTOMOLOGICAL OBSERVATIONS

*Densities of* A. gambiae *and of the* A. funestus *complex in houses and outdoors.*

The average spray-catch density of *A. gambiae* per house was 35.0 in 1965 and dropped to 19.3 in 1966. Similarly, that of the *A. funestus* complex was 57.6 in 1965 and 18.6 in 1966. The *A. funestus* complex in houses in Kibalwa village was almost entirely composed of *A. funestus* Giles following the return of this species into the Taveta area (Smith 1966).

Densities of *A. gambiae* in box-shelters were low and similar, being five per 20 box-days, for both years: those of the *A. funestus* complex were higher, averaging 15.0 per 20 box-days for 1965, but dropping to 7.8 in 1966.

*The sporozoite rates of the vectors and estimated number of infective bites received by the Kibalwa population.*

Of 4,776 *A. gambiae*, from houses, examined for sporozoites in their salivary glands during 1965 and 1966, four were infected, giving a sporozoite rate of 0.08. Similarly, of 8,044 *A. funestus* complex from houses, 23 were infected giving a sporozoite rate of 0.29. The salivary glands of two *A. gambiae* and one individual of the *A. funestus* complex were found infected with *Piroplasma* sp.

**Table 1**

*Rainfall in Taveta area, 1965 and 1966*

| Year | Jan. | Feb. | Mar. | Apr. | May | June | July | Aug. | Sept. | Oct. | Nov. | Dec. | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | *Taveta Sisal Estate* | | | | | | | |
| 1965 .. .. | 0.52 | 1.16 | 2.00 | 2.00 | 3.50 | 0 | 0 | 0.20 | 0.30 | 3.89 | 4.88 | 2.50 | 20.95 |
| 1966 .. .. | 1.15 | 1.35 | 9.04 | 7.45 | 4.55 | 1.85 | 0 | 0.25 | 0 | 0.10 | 0.20 | 2.05 | 27.99 |
| | | | | | | *Kibalwa Outstation* | | | | | | | |
| 1965 .. .. | 0.96 | 0.25 | 1.28 | 2.65 | 0.98 | 0.2 | 0.09 | 0.25 | 0.23 | 3.53 | 3.93 | 2.18 | 16.53 |
| 1966 .. .. | 1.98 | 1.19 | 6.89 | 3.83 | 2.80 | 0.98 | 0 | 0 | 0 | 0.10 | 1.26 | 1.46 | 20.49 |

**Table 2**

*Densities of* A. gambiae *and of the* A. funestus *complex in Kibalwa village as assessed by spray-catches in houses and collections from outdoor box-shelters*

| Year | Jan. | Feb. | Mar. | Apr. | May | June | July | Aug. | Sept. | Oct. | Nov. | Dec. | Average |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | *A. gambiae* | | | | | | |
| *Spray-Catch Densities in Houses* | | | | | | | | | | | | | |
| 1965 .. .. | 23.0 | 21.3 | 80.0 | 67.6 | 50.3 | 46.7 | 16.1 | 16.0 | 11.7 | 28.4 | 25.8 | 33.4 | 35.0 |
| 1966 .. .. | 6.9 | 18.0 | 16.1 | 39.3 | 36.2 | 41.7 | 19.5 | 10.4 | 15.0 | 3.1 | 4.3 | 21.6 | 19.3 |
| *Densities in Outdoor Box-Shelters (No. per 20 Box-Days)* | | | | | | | | | | | | | |
| 1965 .. .. | 6.3 | 1.3 | 10.7 | 11.8 | 3.1 | 2.7 | 2.1 | 2.0 | 0.8 | 5.8 | 10.2 | 5.7 | 5.2 |
| 1966 .. .. | 2.8 | 2.0 | 6.6 | 22.5 | 15.4 | 7.2 | 3.4 | 0.9 | 0.5 | 0.1 | 0.1 | 2.0 | 5.3 |
| | | | | | | | *A. funestus complex* | | | | | | |
| *Spray-Catch Densities in Houses* | | | | | | | | | | | | | |
| 1965 .. .. | 78.0 | 143.8 | 56.4 | 54.1 | 82.3 | 53.0 | 22.0 | 26.7 | 10.3 | 20.0 | 58.9 | 85.2 | 57.6 |
| 1966 .. .. | 22.2 | 41.6 | 15.3 | 21.1 | 31.0 | 38.6 | 26.9 | 4.4 | 14.7 | 0.6 | 1.6 | 5.8 | 18.6 |
| *Densities in Outdoor Box-Shelters (No. per 20 Box-Days)* | | | | | | | | | | | | | |
| 1965 .. .. | 7.3 | 3.0 | 5.2 | 7.8 | 3.6 | 7.2 | 7.5 | 24.6 | 15.5 | 45.7 | 29.5 | 23.0 | 15.0 |
| 1966 .. .. | 3.7 | 2.9 | 6.7 | 6.8 | 11.0 | 5.5 | 18.0 | 20.1 | 18.4 | 0.7 | 6.2 | 3.1 | 7.8 |

The estimated numbers of infective bites received by the human population and calculated by the method of Smith and Draper (1959a), are as follows:

Approximate Annual Malaria Inoculation Rates for Kibalwa Village

| | *A. gambiae* | *A. funestus* complex | Total |
|---|---|---|---|
| 1965 | 4 | 20 | 24 |
| 1966 | 2 | 6 | 8 |

The inoculation rates indicate that, with its return into the area, *A. funestus* was once more the principal vector of malaria in Kibalwa village and that in 1965 malaria transmission had returned to the same intensity as before the houses were treated with dieldrin ten years earlier (Smith and Draper, 1959b). The lower transmission in 1966 may be attributed to the lower densities of both vectors that year (Table 2) due to failure of the short rains (Table 1).

*Malaria in the human population of the Kimorigo/Kibalwa Area*

Before the first round of dieldrin residual spraying malaria transmission around Kimorigo and Kibalwa was less intense than in the interior of Taveta Forest. Nevertheless, Smith and Draper (1959a) reported that the malaria parasite rate in young children among communities living near the edge of the Forest was over 60 per cent; the rate rising to a higher figure towards the end of the rainy season. By 1959, following three years of virtually complete interruption of malaria transmission, the parasite rate in these children had subsided to around five per cent (Smith and Draper, 1959b).

Subsequently, the rate of return of malaria to this district was tentatively traced by the periodic examination of children attending Kimorigo School, as well as of random samples of the population that live in the eastern fringes of the Forest. The results of these surveys are summarized in Tables 3 and 4.

**Table 3**

*Malaria parasite rates in children aged 5-9 years, attending Kimorigo School*

| *Date* | *Number examined* | *Number positive* | *Parasite rate* | *Mean parasite rate for each year:* |
|---|---|---|---|---|
| 1960: February | 31 | 2 | 6 | |
| ,, November | 32 | 8 | 25 | Mean for 1960: 16 |
| 1961: March | 25 | 8 | 32 | |
| ,, June | 20 | 9 | 45 | |
| October | 150* | 77 | 51 | Mean for 1961: 48 |
| 1962: May | 125* | 72 | 58 | |
| ,, November | 20 | 8 | 40 | Mean for 1962: 55 |
| 1963: February | 19 | 11 | 57 | |
| ,, June | 52* | 28 | 54 | Mean for 1963: 55 |
| 1964: February | 36 | 10 | 28 | |
| ,, October | 36 | 13 | 36 | Mean for 1964: 32 |
| 1965: November | 34 | 7 | 21 | Mean for 1965: 21 |
| 1966: June | 120* | 64 | 53 | Mean for 1966: 53 |

*including data obtained in the same area from other children, during random surveys of local population.

**Table 4**

*Malaria parasite rates obtained during random surveys of the population of Taveta Forest and particularly the communities around Kimorigo and Kibalwa*

| Date | 0–2 yrs | | | 2–9 yrs | | | 10–19 yrs | | | Over 19 yrs | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Exd. | Pos. | PR. | Exd. | Pos. | PR. | Exd. | Pos. | PR. | Exd. | Pos. | PR. |
| 1961 Oct. | 19 | 10 | 53 | 150 | 77 | 51 | 108 | 40 | 37 | 91 | 18 | 20 |
| 1962 May | 25 | 12 | 48 | 125 | 72 | 58 | 104 | 49 | 47 | 78 | 16 | 21 |
| 1963 June | 5 | 2 | 40 | 52 | 28 | 54 | 18 | 5 | 28 | 29 | 5 | 17 |
| 1964 Feb. | 0 | — | — | 36 | 10 | 28 | 16 | 4 | 25 | 42 | 2 | 5 |
| 1966 June | 8 | 1 | 13 | 59 | 34 | 58 | 25 | 4 | 16 | 56 | 2 | 4 |

## DISCUSSION

The present results of dissection for sporozoite infections in *A. gambiae* and *A. funestus* complex from houses in Kibalwa village are in conformity with observations, obtained before residual spraying, that *A. funestus* Giles is responsible for the greater part of malaria transmission in Taveta area (Smith and Draper, 1959a). This is due to its higher sporozoite rate and generally higher annual densities in houses, in conjunction with its highly anthropophilic feeding habits (Smith 1966). It thus seems that *A. funestus* that returned to Taveta area in 1963, after being absent for eight years, has not only the same habits as its predecessor (Smith 1966) but also the same vector-capacity.

Changes observed in parasite rates since 1959 suggest that malaria transmission did not recur to a significant degree around Taveta Forest until the latter half of 1960—two years after the final round of dieldrin spraying. Subsequently, further increases in infection rates were recorded between then and 1963. Since 1963, however, despite the greater malaria inoculation risk created by the re-establishment of *A. funestus* Giles in and around the Forest, local parasite rates have tended to decline. The discordance between the increased malaria infection risk and the concomitant fall in infection rates is similar to the sequence of events noted during parallel circumstances in the Mkomazi Valley of South Pare. Here, as described by Pringle (1967) an increase in malaria risk from the previous, persistently low level was also marked by a fall in parasite rates among the human population.

These observations suggest that, nowadays, the extent of malarial parasitism in a community reflects the degree of public awareness of the disease as much as it does the frequency of infection. In such communities, any sudden exacerbation of malaria transmission may provoke such a widespread exhibition of antimalarial drugs that the net human infection rate falls, instead of rising. During this time, an estimation of malaria infection risk through human malariometry would be totally misleading.

Recent experience in Taveta and South Pare, therefore, suggests the precaution, when measuring malaria endemicity in sophisticated African communities, that the information gained from examination of the human population is confirmed by a measurement of the local biting frequency of soporozoite-positive malaria vectors. The validity of this concept was recently tested by Pringle and Avery-Jones (1966).

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